Electromyographically Triggered Electric Muscle Stimulation for Chronic Hemiplegia

R. Wayne Fields, PhD
Department of Physiology and Pharmacology, Oregon Health Sciences University, S.D., Portland, OR 97201

ABSTRACT. Fields RW: Electromyographically triggered electric muscle stimulation for chronic hemiplegia. Arch Phys Med Rehabil 68:407–414, 1987.

• Electromyographically triggered electric muscle stimulation (EMS) was evaluated in combination with conventional treatment in 69 consecutive postcerebrovascular accident outpatients whose onset of hemiplegia was four months to 14 years earlier. Six subjects initially exhibited no residual volitional activity in targeted muscles, and all patients had undergone conventional therapy with little or no functional recovery. Electromyographic (EMG) recordings and EMS directed to prime movers of impaired movements were accomplished by way of skin-surface electrodes. Prescribed treatment (patient compliance was frequently substandard) involved several months of four to five sessions per week, focusing on wrist extension and/or ankle dorsiflexion initially, and often other movements later. During 30 to 300 movement attempts per session, EMGs that exceeded a preset threshold triggered immediate stimulation to force movement completion. Over sessions, patients commonly realized substantially improved increases in voluntary EMG capabilities generally proportionate to the frequency of treatment sessions. Parallel improvements were also found for subjectively scaled functional measures of range-of-motion and ambulation. Motivation was important to success, but side and nature of stroke, age, and poststroke interval were not. Progress often far exceeded that of previous conventional therapy (each patient served as his/her own control). Regarding mechanisms, impaired proprioceptive feedback is considered central to stroke-disrupted sensorimotor control. EMG-triggered EMS is intended to improve brain relearning by reinstating proprioceptive feedback time-locked to each attempted movement. Clinical results were consistent with this theory; further assessment of the new EMG-triggered EMS modality intergrated into conventional treatment regimens seems warranted.

KEY WORDS: Cerebrovascular disorders; Electrotherapy; Physical therapy; Proprioception

It is widely accepted that rehabilitation offers the only means to address the residual deficits of stroke.[30] Nevertheless, underlying mechanisms of conventional physical rehabilitation are poorly understood,[30] and the limitations of stroke therapy are well documented.[12,17,23]

In general, a brain area damaged by stroke does not regenerate significantly,[3] so that observed recovery is due to plasticity phenomena[7,13] invoking other areas of the brain to assume some level of function originally managed by the lost tissue.[3,11] In the process of physical rehabilitation, at least three types of physiologic information must converge concurrently on key areas of the brain: central recurrent derivatives of motor output encoding the goal of movement;[25,26] afferent input to provide the means to monitor movement progress;[1,9,22,25,26] and relevant data from motor memory.[9] The importance of feedback input from the periphery is emphasized by the poorer prognosis of patients exhibiting sensory deficits compounding hemiplegia.[8,21,27,31] Recovery is also highly dependent on learning and attendant requirements such as attention and suitable motivation.[2,10,15,17,29]

One of the most fundamental deficits common to many strokes is impaired afferent input,[8,15] especially proprioception.[11,24] Significantly, proprioception is considered the most important afferent input in motor control.[1,9,18] The multiple, complex, and highly integrated feedback loops of the normal sensorimotor control system[9,16,19] are interrupted in various ways by stroke, depriving the recovering brain of essential real-time data on unfolding posture and movement. In an attempt to augment afferent input in stroke patients,

Table 1: Translation of Quantitative Range-of-Motion Data into Nonparametric Scale Values

SCORE	
0	0% active motion (100% deficit); joint excursion can be accomplished passively through the available range. The joint may range from severe flaccidity to marked spasticity.
1	1% to 20% active motion (80% to 99% deficit); active excursion can be carried out through a small fraction of the available range of passive motion.
2	20% to 40% active motion (60% to 80% deficit); active excursion can be achieved through a small but significant portion of the available range of passive motion.
3	40% to 60% active motion (40% to 60% deficit); within the available range of passive motion, active excursion capabilities are moderate.
4	60% to 80% active motion (20% to 40% deficit); active excursion can be completed through a substantial portion of the available range of passive motion.
5	80% to 100% active motion (0% to 20% deficit); within the available range of passive motion, active excursion can be nearly or fully completed.

EMG-biofeedback has been employed to provide substitute audio and/or visual indices of movement and its progress.[3,11] EMG-biofeedback has been successful under certain circumstances[4,6,11,32] but has its limitations,[20,29] perhaps

Preliminary results of this study were presented at the combined annual meeting of the American Academy of PM&R and the American Congress of Rehabilitation Medicine, Baltimore, October 22, 1986.

Submitted for publication October 15, 1986. Accepted in revised form December 30, 1986.

due to the fact that the modalities involved are merely substitutes[3,11,12] for lost proprioception.

Conceptually, first-order logic would argue for reinstating the missing or deficient proprioceptive feedback. To this end, the present study concerns stabilized stroke patients treated with a new modality, EMG-triggered electric muscle stimulation (EMS), integrated with conventional treatment. In EMG-triggered EMS, as a patient attempts to initiate an involved movement, relevant muscles are stimulated to force movement completion if (and only if) the patient achieves a therapist-set EMG threshold. The goal is to reinstate more normal movement and thus augment proprioception time-locked to the patient's movement attempt. This is intended to restore sensorimotor interaction believed essential to the highly learning-dependent processes reestablishing control of posture and movement.

METHODS

Patient Selection and Orientation

Sixty-nine hemiplegic outpatients aged 29 to 83 years (median 63 years), who presented consecutively for treatment and produced data sufficient for analysis, were included in this study. The length of time since onset of hemiplegia ranged from four months to 14 years (median three years). The hemiparesis was on the right side in 26 and on the left side in 43 subjects; 45 subjects were men and 26 were women.

The relatively unrestrictive acceptance criteria for inclusion in the subject program were that patients (1) were suitably alert to be able to follow instructions, and (2) had significant motivation for rehabilitation.[12,17] No other patient preselection was imposed. All patients had previously undergone one or more conventional stroke therapy programs and in their judgment had achieved negligible or unsatisfactory functional recovery. In general, the long poststroke interval (many years for most patients; less than nine months for two patients, and nine to 11 months for six patients) and poor response to previous treatment permitted the consideration of each patient as his/her own control.[11] Since neither the long-term course of rehabilitation nor the response to therapy has been found strongly affected by the type or location of the stroke lesion,[23] the present study did not distinguish these parameters.

Initially, each patient was given a detailed nontechnical description of the present EMG-triggered EMS technique (described in more detail below), especially emphasizing EMG-biofeedback and EMS, including the unique individual and collective purposes and interactions of these techniques in the EMG-triggered EMS procedure. The patient was informed that the therapy does not provide a cure, that in most CVA cases there will always be some residual deficit, and that sustained program participation and recommended home exercises were essential to realize the full potential of physical rehabilitation. No commitments were made about the eventual degree of recovery or regarding the elimination of any supportive devices; the therapists merely agreed to work with each subject to help him/her regain as much function as possible. Many patients had transportation, financial, and other problems preventing rigid adherence to scheduling or full clinic and/or home participation. This accounted for significant variance in attendance, the fact that a number of patients (over and above the 69 presented here) produced records considered inadequate for reasonable evaluation, and it may mean that the results underestimate gains achievable under closer adherence to prescribed treatment.

Treatment Program

Desired treatment programs spanned one to six months at a rate of four to five clinic treatment sessions per week (weekends were excluded). An average treatment session involved approximately 45 minutes for a target muscle group in the upper or lower limb alone. Most patients received both upper and lower limb treatment during each session; therefore, sessions typically lasted 90 minutes.

The treatment programs were composed of the EMG-triggered EMS protocol integrated into conventional physical therapy, with both regimens specially tailored to the needs and goals of each patient. The spectrum of traditional therapy included elements of neuromuscular reeducation, range-of-motion (ROM) exercises (passive stretching, active assistive), strengthening and neuromuscular facilitation procedures, functional training (progressive mobility, gait training), and heat modalities to address stiffness and pain. Conventional therapy followed normal strategies, which usually emphasize proximal rather than distal limb activities initially. In contrast, EMG-triggered EMS focused on wrist extensors and ankle dorsiflexors (distal limb segments) at first, and often other functions later.

The EMG-triggered EMS procedure was structured as follows: (1) attempt—the patient was asked to attempt an involved movement; (2) detection—an ultrasensitive EMG recording system detected the attempted movement; (3) threshold—if the EMG exceeded a threshold level preset by the therapist, EMS of the same muscle or group was triggered; and (4) stimulus—the EMS, preset by the therapist to augment significantly the patient's unaided voluntary capabilities, forced the involved movement to completion through the full available ROM. As patients advanced through the program, the EMG threshold was progressively raised, as permitted by acquired voluntary capabilities. The program goal was to bring the patient to a maximal unaided performance level, at which time the assistive EMG-triggered EMS would no longer be required.

Techniques and Instrumentation

A commercial EMG stimulator[a] device with patient-initiated response, which automated the EMG-triggered EMS procedure outlined above, was used. The ultrasensitive recording section derived EMG signals from commercial, conductive gel-coated, skin-surface electrodes (approximately 10 square cm in area), held in place with adhesive tape. The raw EMG signal (maximum resolution approximately 0.1μV) was rectified and integrated for comparison to a therapist-preset threshold. Suprathreshold EMG trials triggered 1.0sec of EMS at therapist-preset parameters (asymmetrical, bi-phasic pulse train, 0 to 90ma peak constant power output, 20 to 100pps frequency, and 0.3 or 1.0ms pulse width), selected to drive the involved movement beyond the patient's limited voluntary

Table 2: Definition of Nonparametric Values for Characterizing Ambulation

SCORE	
0	Unable to ambulate; must be transported by others. May be transferred from the sitting to standing position with maximal assistance. No voluntary hip or knee flexion or dorsiflexion of the involved lower extremity.
1	Scant ambulation; supportive and assistive devices required. Transfers with assistance; ambulates less than 15 steps with maximum assistance. May "hip hike" but does not demonstrate voluntary knee or hip flexion or dorsiflexion of the involved lower extremity.
2	Meager ambulation; must use supportive and assistive devices to ambulate. Transfers without assistance; ambulates with minimal assistance more than 15 (but less than 50) steps. Shows some hip and knee flexion and dorsiflexion of the involved lower extremity.
3	Moderate ambulation; uses a supportive or assistive device to ambulate, but does not need both. Ambulates and transfers unassisted; ambulates 15 to 100 steps without undue fatigue. Exhibits fair hip and knee flexion and dorsiflexion of the involved lower extremity.
4	Substantial ambulation; may ambulate with or without supportive aids or assistive devices. Has voluntary control of knee and hip flexion and dorsiflexion of the involved lower extremity; ambulates 100 to 1,000 steps without undue fatigue.
5	Complete or nearly complete ambulation: control of hip and knee flexion and dorsiflexion of the involved lower extremity. Does not require assistance of any kind in transfers or ambulation. May ambulate distances exceeding 1,000 steps with at least a substantially correct gait pattern.

capabilities to achieve the full available ROM. Electrodes were positioned over the central belly of the prime mover of the movement targeted for treatment. Through a special convenience feature of the EMG stimulator, the stimulation and recording functions shared the same pair of electrodes. During a trial, the peak EMG was extracted by the instrumentation and presented to the patient for 2sec as a visual reinforcement. The procedure also incorporated several other forms of motivation, including visual feedback, goal achievement (attainment of threshold), stimulus-induced kinesthetic sensation, visual observation of the stimulated movement itself, and, of course, therapist reinforcement.

Measures of Progress

As a patient progressed through the treatment program, the peak, unassisted, voluntary EMG recorded from the prime movers of targeted movements were used as a quantitative index of achievement. For any one EMG recording trial, only a single attempt was allowed, to optimize intertest comparability and minimize the effects of fatigue. Pretreatment EMGs at the beginning of each therapy session were found the most useful, because readings recorded at various times during a session were difficult to normalize temporally and intermediate or end-session EMGs were variably complicated by fatigue. Both intrasession and intersession variations in electrode placement and skin resistance almost certainly existed, but this inconsistency did not compromise the utility of the EMG measure because of the large and progressive EMG changes commonly encountered.

The above quantitative EMG data supplemented selected measures of functional relevance; specifically, limb active ROM, and, for the lower limb, ambulation. ROM measurements were comprehensive relative to the motor deficit and planned therapy for each particular patient. Thus, reported changes in ROM represent averages over involved areas of the whole limb. Both ROM and ambulation measurements were recorded as nonparametric values on scales of 0 to 5 (tables 1 and 2, respectively; see also reference 5). Such data were recorded for each patient at intervals of several weeks. The EMG and functional data on progress were informally supplemented with patient and family interviews and, when available, information from the patient's physician.

RESULTS

Peak, Unassisted, Voluntary, Pretreatment EMGs

In general, the pretreatment EMGs used as a quantitative index of progress for each patient were found to increase significantly over sessions. Figure 1 illustrates session-by-session pretreatment EMGs selected to exemplify several characteristics noted overall: (1) progress was typically evident very early, usually in the first or second session; (2) notable progress was commonly evident when averaged over several consecutive sessions at any point during the program; and (3) considerable intersession variability was encountered (prominent downswings were often informally correlated with episodes of poor patient health or motivation). Based on accumulated experience, it is now felt that patients unsuitable for EMG-triggered EMS can be identified within the first few treatments as the result of both substandard voluntary EMG capabilities and negligible functional improvements; overall, the success rate (measurable functional progress) exceeded 90% of consecutively admitted patients who met the relatively unrestrictive program entrance requirements.

Figure 2 shows that most patients exhibited progress as early

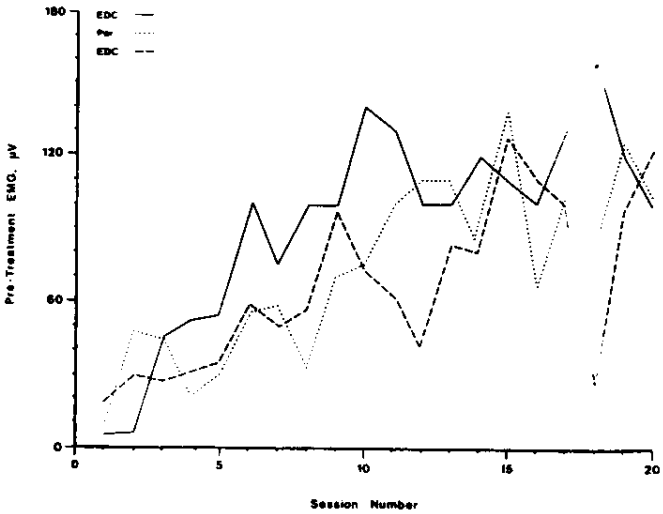

Fig 1—Single-trial, peak, unassisted, voluntary pretreatment EMGs, as a function of session number, for three examples from different patients. The total duration for each patient differs in the data of this and other figures because all patients did not adhere to the same treatment schedule. EDC = extensor digitorum communis; Per = peroneals.

as the first session. (Note that figs 2, 3, 4 have logarithmic axes, so that even small plot displacements frequently represent pronounced changes.) Many patients improved in peak, voluntary, unaided EMG capabilities after only a few EMG-triggered EMS trials, and some achieved dramatic first-session increases in this EMG index ranging to many times the initial value. Even though the data of fig 2 demonstrate significant first-session progress, the posttreatment data are biased toward lower values, due to their collection at the end of sessions when fatigue was most pronounced. As is qualitatively apparent from the figure, no significant difference was found for the results derived from different muscles. In fig 2 (and figs 3 and 4 as well), the data just adjacent to the vertical axis represent a special class of patients who were initially incapable of generating any EMG in the involved muscle. In these instances, a special feature of the EMG stimulator was employed, which allowed EMG triggering by remotely located normal musculature but with stimulation still applied to the involved muscle. This remote recording feature was only required for a short time, since the patients quickly became able to generate voluntarily measurable EMGs in the involved muscle. Subsequently, the normal combined recording-stimulation configuration of the EMG stimulator was routinely used.

Figures 3 and 4 compare first-session pretreatment EMGs recorded for each patient to analogous pretreatment EMGs obtained before the eighth and last sessions, respectively. Inspection of fig 3 reveals pronounced additional progress after eight sessions over that realized after the first-session results of fig 2; data for this eighth session (chosen to represent two to four weeks of typical therapy in this study) was selected for presentation because, especially early in the program, pretreatment EMGs subjectively appeared to correlate well with functional improvement. Figure 4 indicates that even further elevation of pretreatment EMGs is apparent at the time of the patient's last session. Nevertheless, it was felt that pretreatment EMGs became less representative of functional progress toward the end of the program. In fact, some patients exhibited a late decline in pretreatment EMGs from higher values achieved

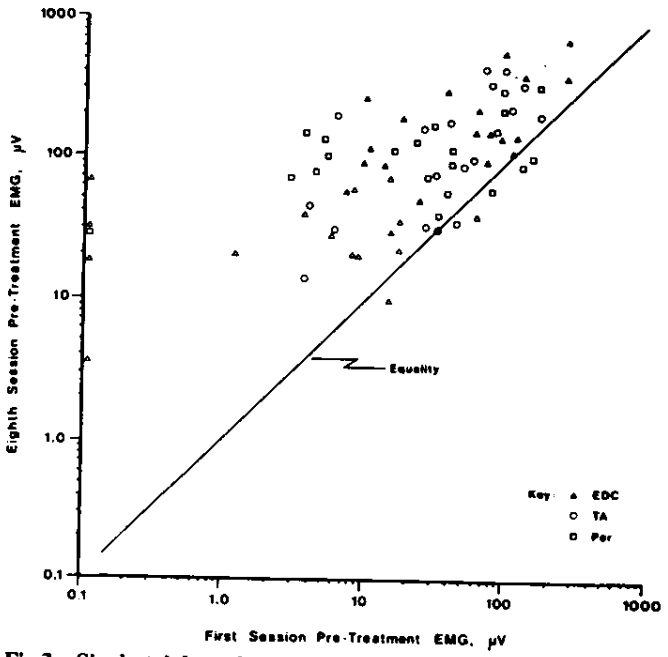

Fig 3—Single-trial, peak, unassisted, voluntary EMGs by patient: eighth session pretreatment vs first session pretreatment. (See fig 2 legend for further details.)

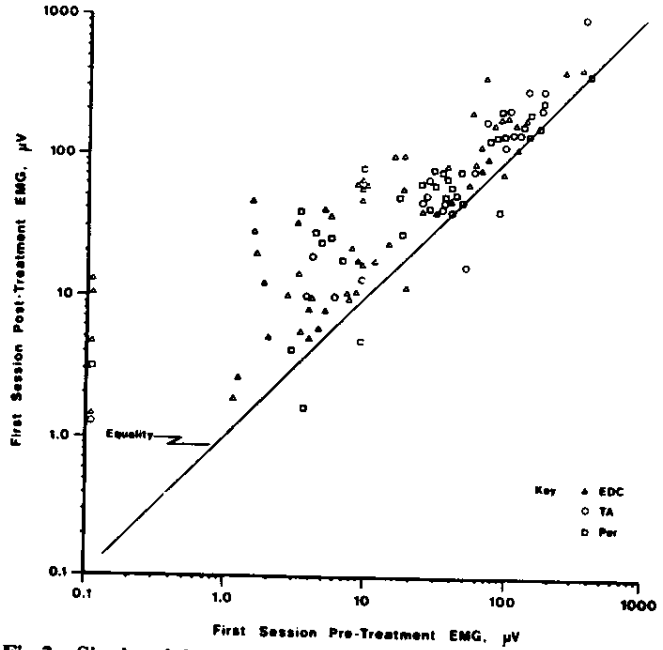

Fig 2—Single-trial, peak unassisted, voluntary EMGs by patient: first session posttreatment vs first session pretreatment. The origin was chosen as 0.1μv for both axes to recognize that value as the approximate maximum resolution of the EMG recording circuitry. EDC = extensor digitorum communis; TA = tibialis anterior; Per = peroneals.

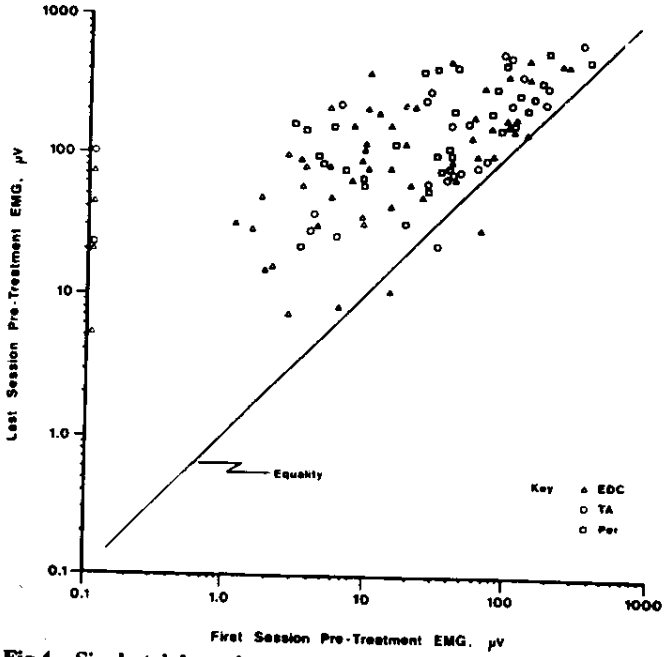

Fig 4—Single-trial, peak, unassisted, voluntary EMGs by patient: last session pretreatment vs first session pretreatment. (See fig 2 legend for further details.)

Table 3: Increase in Pretreatment EMG vs Treatment Frequency

Increase in pretreatment EMG; highest recorded value ÷ 1st session value	Treatment frequency; sessions ÷ days of treatment duration			
	.00–.24	.25–.49	.50–.74	≥.75
> 32x	1	5	3	0
10x – 31x	1	9	4	0
3.2x – 9.9x	2	6	2	0
1.0x – 3.1x	2	7	1	0
Total	6	27	10	0

earlier in the program; this was frequently accompanied by improved functional capabilities, not performance decrements, and therefore appeared indicative of enhanced movement efficiency. As is qualitatively apparent in both figs 3 and 4, no significant difference was found for the results derived from different muscles.

Treatment Frequency

Table 3 addresses the important issue of treatment frequency by displaying degrees of pretreatment EMG improvement vs successive ranges of treatment frequency. Inspection of the table reveals a pronounced shift to higher achieved pretreatment EMG multiples as the treatment frequency progressively increased from the lowest to the second highest range. Significantly, more than half the patients exhibited an EMG multiple exceeding a factor of ten. It is pertinent that none of the present patients equaled or exceeded a frequency of 0.75 treatments per day averaged over their entire treatment program. Given the fact that weekend treatments were not available, a patient would have had to receive treatments almost every weekday for the entire therapy program to achieve a treatment frequency of 0.75, and none of the patients accomplished that

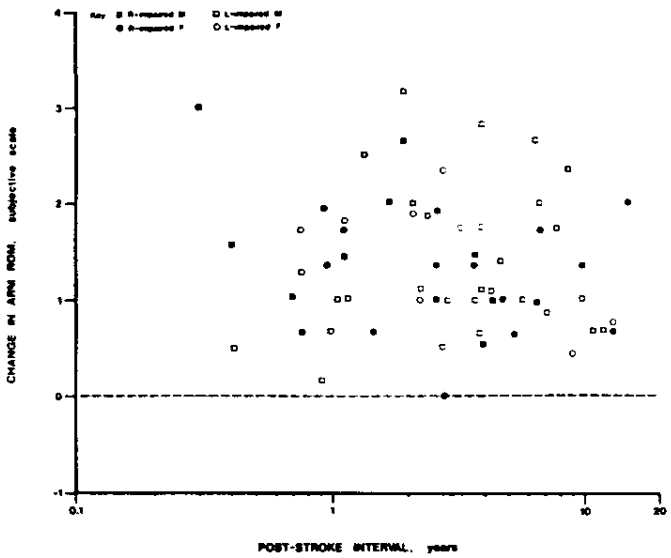

Fig 5—Change in arm active ROM (based on a subjective scale where 0 = no movement, and 5 = full movement) vs poststroke interval (years) over the entire treatment program for each patient. L = left-side stroke; R = right-side stroke; M = male; F = female. Positive ROM change values represent positive improvement.

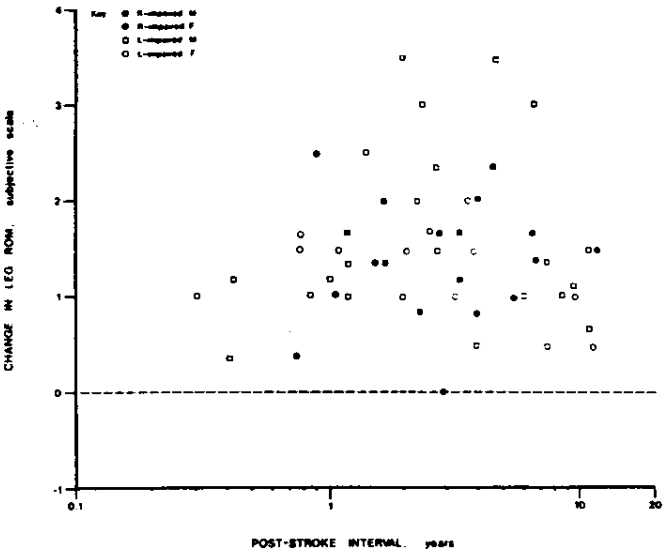

Fig 6—Change in leg active ROM vs poststroke interval. (Details analogous to fig 5.)

level of participation. Nevertheless, extrapolation from the data would suggest that higher treatment frequency is worthy of serious consideration.

Measures of Functional Improvement

Patient changes in averaged active limb ROM is displayed in figs 5 and 6 for the involved arm and the involved leg, respectively. In both figures, the functional data are plotted against poststroke interval. As is subjectively apparent from the figures, no relationship was found between the ROM data and the elapsed time since the stroke event. The mean improvements in ROM for the arm and leg were 1.4 (s = 0.7, n = 63) and 1.4 (s = 0.7, n = 53) subjective units, respectively (changes were derived against a full-scale range of 0 to 5; see Methods). No obvious relationships were evident between changes in ROM and either the sex of the patient or side of stroke.

Figure 7 illustrates ambulation scores as a function of poststroke interval. As with the ROM data above, no relationship was found between ambulation and the elapsed time since the stroke event. Ambulation improved an average of 1.6 subjective units (s = 0.5, n = 55; changes were derived against a full-scale range of 0 to 5; see Methods). No obvious relationships between ambulation and either the sex of the patient or side of stroke were revealed.

Although not assessed quantitatively, information gathered informally from patients, family, and their physicians (as available) revealed for most subjects enhanced feelings of well-being, self-esteem, and functional improvements in activities of daily living. The general impression of most family members was that the combined conventional and EMG-triggered EMS treatments produced improved and more rapid results compared to their previous conventional therapy alone.

Increased EMGs vs Functional Improvements

The subjective sense of a correlation between pretreatment EMGs and functional improvements was tested by comparing

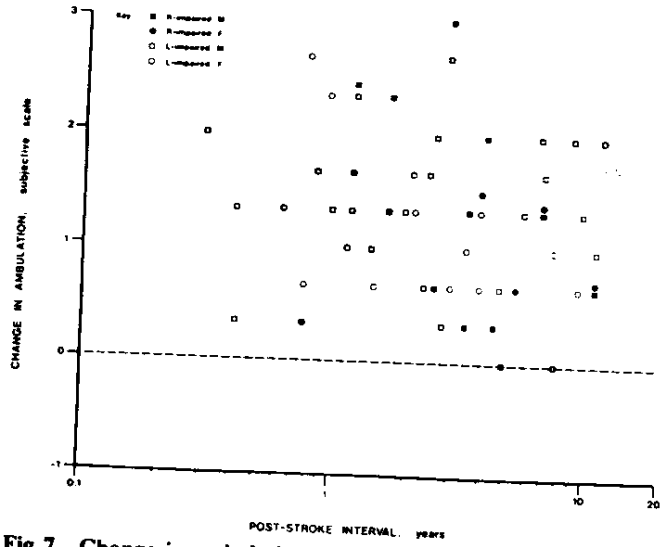

Fig 7—Change in ambulation vs poststroke interval. (Details analogous to fig 5.)

last vs first session data (fig 8). It is important to recall that the hypothesized correlation was considered primarily limited to the initial phases of treatment. Nevertheless, the need for adequately coordinated functional data for such an initial-phase analysis (eg, eighth vs first sessions) had not been anticipated in advance, and the data in fig 8 were the best available. In spite of the suboptimal records, a relationship of intermediate strength was revealed, having a Pearson correlation coefficient of 0.5. Two regression lines were also calculated, one minimizing the sum-of-errors-squared parallel to the EMG and the other parallel to the wrist ROM axis. The two regression lines intersect at the mean values of 175μv for the change in EMG and 1.51 scale units for the wrist ROM data, respectively.

DISCUSSION

There were three aspects of the preliminary EMG-triggered EMS results that are significant in the perspective of general physical rehabilitation of stroke: (1) the majority of patients were judged by both staff and family as having achieved more meaningful and rapid recovery in the present program than in their previous conventional therapy; (2) poststroke intervals ranging to 14 years were successfully managed; and (3) improvements were clearly measurable in spite of minimal patient preselection. Elements of these points are discussed in more detail below.

In this study, every patient had undergone significant conventional stroke therapy without notable functional improvement before the EMG-triggered EMS program. All but two patients were nine months or more poststroke and were considered stabilized, so that each patient was legitimately considered as his/her own control.[11] While renewed therapy of any kind may induce some improvement,[5] it was found, based on the totality of patient interviews and assessments, that the present results were superior, in spite of the fact that our therapy had the distinct comparative disadvantage of occurring after previous treatment. Progress was frequently apparent during the first therapy session after only a few trials, and, as the EMG results show, was quite significant after only eight sessions.

Historically, stroke physical rehabilitation has been characterized as declining to generally ineffective levels by six to 18 months poststroke.[14] Some success in dealing with stabilized patients has been achieved using EMG-biofeedback techniques.[4,12,17,20] This success has been attributed to augmentation of sensorimotor interactions[3,11] but limited by dependence on substitute feedback modalities.[2,3,11,12] The present EMG-triggered EMS-based treatments were deemed generally superior to previous conventional therapy regardless of poststroke interval or patient/stroke characteristics, and dramatic improvements were achieved in many cases after only a few trials in the first session. These results are consistent with our reasoning that even stronger attention to sensorimotor interactions and learning dependence might afford improved achievement. Our immediate objective was to restore more normal proprioceptive feedback from the prime movers of target movements to reinstate the dynamic (real-time) sensorimotor interactions normally time-locked to movement initiation and progress. In this regard, the nervous system learns by doing; it has been repeatedly shown that active involvement is superior to passive participation for learning, maturation, and maintenance.[28] It seems plausible that this would be true for relearning also, and that the best approach would be to use natural rather than substitute feedback modalities.

Central to the present approach is the hypothesis that, for effective relearning to occur, crucial data on both the goal and outcome of movement must converge temporally as well as spatially in key brain centers. Since most of the stroke-

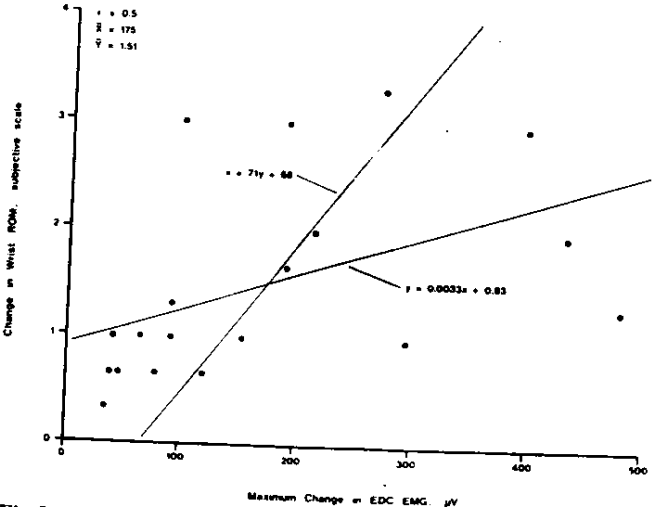

Fig 8—Change in wrist-active ROM (based on a subjective scale where 0 = no movement, and 5 = full movement) vs change in EDC (extensor digitorum communis) pretreatment EMG (μv) over the entire treatment program for each patient. The ROM data were restricted to that of the wrist for this analysis, rather than the whole arm as in previous figures, to focus more precisely on the specific movement associated with EDC EMGs. The EMG value used for each patient was the highest pretreatment value recorded for any session in the program. A patient's data were accepted for analysis given completion of at least 20 treatment sessions at a mean frequency of at least 0.33 treatments/day.

damaged area is considered permanently disabled,[3] previously participating brain centers must modify their function and/or new centers must join the system for reorganization to take place. One detailed mechanism is probably not sufficient to explain the present findings, because the patient population was quite heterogeneous in nature, location, and extent of stroke. Nevertheless, this very patient diversity, combined with consistent clinical success, suggests that a broad-based, nonspecific principle (such as our working hypothesis) may hold. It is pertinent that an anatomic-physiologic substrate consistent with our phenomenologic model has recently been receiving substantial development and growing acceptance. The existence of significant brain plasticity has been described after injury, exhibiting properties (in addition to learning, of course) such as the formation of new connections and modification of old pathways,[3,7] which would be fundamental to any meaningful brain reorganization.

There are three aspects of the present EMG-triggered EMS approach that require further comment. First and foremost is the reinstatement (or, for the less severely impaired, at least augmentation) of proprioception, time-locked to the movement attempt. It is believed that the main effect of the present muscle stimulation was the physiologic activation of relevant proprioceptors due to restored movement (nevertheless, the direct electric activation of proprioceptive and cutaneous afferents must also occur, to some degree, due to nonspecific stimulation). Second, the reinstated proprioceptive feedback was made to occur physiologically concurrent with the attempted movement, a feature believed central to the dynamics of relearning.[7,28,30] Exploratory tests (unpublished data) qualitatively suggested that delays in EMS imposed after movement attempts degraded voluntary, unaided EMG progress. Finally, that the primary purpose of the present muscle stimulation was to restore timely proprioception is quite novel in the routine clinic environment; it stands in stark contrast to conventional muscle stimulation, which is used for muscle strengthening and nondynamic reeducation, atrophy counteraction, circulatory augmention, and other benefits. The present approach almost certainly induces some of the benefits of conventional stimulation as well, but as a fortuitous accompaniment to the primary purpose of reconnecting the disrupted sensorimotor feedback interrelationships that underlie voluntary movement.

CONCLUSION

Preliminary results indicate that EMG-triggered EMS is a promising new modality for treating stroke and perhaps other forms of focal brain damage. EMG-triggered EMS is intended to intervene directly in the dynamics of sensorimotor control, reinstating proprioceptive feedback time-locked to movement attempts. The technique was envisioned to be judiciously integrated into existing programs for the physical rehabilitation of brain-injured patients, and was used in this way in the present study. The encouraging results, while preliminary, argue for further consideration of EMG-triggered EMS as an additional modality for incorporation into stroke therapy regimens.

Acknowledgments: We hereby acknowledge Karen Fiechtner, PTA, Georgina Draeger, RPT, Grazyna Wojiechowski, PTA, Janice Jockers, PTA, Bill Iwen, RPT, Peter Pakulis, RPT, Kathy Thome, PTA, Donette Doherty, PTA, John Marshall, PTA, and Peter Rigby, RPT, who conducted various therapy sessions and/or assisted in data collection. A portion of the data were also collected by Carl J. Ylvisaker, RPT.

ADDRESS REPRINT REQUESTS TO:
R. Wayne Fields, PhD
Department of Physiology and Pharmacology
Oregon Health Sciences University, S.D.
611 S.W. Campus Drive
Portland, OR 97201

References

1. Adams JA: Closed-loop theory of motor learning. J Motor Behav 3:111–149, 1971
2. Bach-y-Rita P: Brain plasticity as a basis for therapeutic procedures. *In* Bach-y-Rita P (ed): Recovery of Function: Theoretical Considerations for Brain Injury Rehabilitation. Baltimore, University Park Press, 1980, pp 255–262
3. Bach-y-Rita P: Rehabilitation versus passive recovery of motor control following central nervous system lesions. *In* Desmedt JE (ed): Motor Control Mechanisms in Health and Disease. New York, Raven Press, 1983, pp 1085–1092
4. Baker M, Regenos E, Wolf SL, Basmajian JV: Developing strategies for biofeedback; applications in neurologically handicapped patients. Phys Ther 57:402–408, 1977
5. Basmajian JV, Kukulka CG, Narayan MG, Takebe K: Biofeedback treatment of foot-drop after stroke compared with standard rehabilitation technique: effects on voluntary control and strength. Arch Phys Med Rehabil 56:231–236, 1975
6. Basmajian JV, Regenos EM, Baker MP: Rehabilitating stroke patients with biofeedback. Geriatrics 32:85–88, Jul 1977
7. Bishop B: Neural plasticity: part 4 lesion-induced reorganization of CNS: recovery phenomena. Phys Ther 62:1442–1451, 1982
8. Bobath B: Observations on adult hemiplegia and suggestions for treatment. Physiotherapy 45:279–289, 1959
9. Brooks VB: Motor control: how posture and movements are governed. Phys Ther 63:664–673, 1983
10. Bowman BR, Baker LL, Waters RL: Positional feedback and electrical stimulation: automated treatment for hemiplegic wrist. Arch Phys Med Rehabil 60:497–502, 1979
11. Brudny J, Korein J, Grynbaum BB, Friedmann LW, Weinstein S, Sachs-Frankel G, Belandres PV: EMG feedback therapy: review of treatment of 114 patients. Arch Phys Med Rehabil 57:55–61, 1976
12. Brudny J, Korein J, Levidow, L, Grynbaum BB, Lieberman A, Friedmann LW: Sensory feedback therapy as modality of treatment in central nervous system disorders of voluntary movement. Neurology (Minneap) 24:925–932, 1974
13. Cotman CW (ed): Neuronal Plasticity. New York, Raven Press, 1978
14. Davies PM: Steps to Follow: Guide to Treatment of Adult Hemiplegia. New York, Springer-Verlag, 1985
15. De Souza LH: Effects of sensation and motivation on regaining movement control following stroke. Physiotherapy 69:238–240, 1983
16. Evarts EV: Brain control of movement: Possible mechanisms of functional reorganization. *In* Bach-y-Rita P (ed): Recovery of Function: Theoretical Considerations for Brain Injury Rehabilitation. Baltimore, University Park Press, 1980, pp 173–186
17. Flom RP, Quast JE, Boller JD, Berner M, Goldberg J: Biofeedback training to overcome poststroke foot-drop. Geriatrics 31:47–51, 1976
18. Garraway WM, Whisnant JP, Drury I: Continuing decline in incidence of stroke. Mayo Clin Proc 58:520–523, 1983

19. Houk JC: Motor control processes: New data concerning motoservo mechanisms and tentative model for stimulus-response processing. *In* Talbott RE, Humphrey DR (eds): Posture and Movement. New York, Raven Press, 1979, pp 231–241
20. Houk J, Henneman E: Feedback control of movement and posture. *In* Mountcastle VB (ed): Medical Physiology. Ed 12, St Louis, Mosby, 1968, pp 1681–1696
21. Katz S, Ford AB, Chinn AB, Newill VA: Prognosis after strokes: part II. Long-term course of 159 patients. Medicine **45**:236–246, 1966
22. Leo KC, Soderberg GL: Relationship between perception of joint position sense and limb synergies in patients with hemiplegia. Phys Ther **61**:1433–1437, 1981
23. Liberson WT, Holmquest HJ, Scot D, Dow M: Functional electrotherapy: stimulation of peroneal nerve synchronized with swing phase of gait of hemiplegic patients. Arch Phys Med Rehabil **42**:101–105, 1961
24. Lind K: Synthesis of studies on stroke rehabilitation. J Chronic Dis **35**:133–149, 1982
25. Luriia AR: Restoration of Function after Brain Injury. New York, Pergamon Press, 1963
26. Matthews PBC: Muscle afferents and kinaesthesia. Br Med Bull **33**:137–142, 1977
27. McCloskey DI: Kinesthetic sensibility. Physiol Rev **58**:763–820, 1978
28. Michels E: Motor behavior in hemiplegia. Phys Ther **45**:759–767, 1965
29. Moore J: Neuroanatomical considerations relating to recovery of function following brain lesions. *In* Bach-y-Rita P (ed): Recovery of Function. Theoretical Considerations for Brain Injury Rehabilitation. Baltimore, University Park Press, 1980, pp 9–90
30. Mroczek N, Halpern D, McHugh R: Electromyographic feedback and physical therapy for neuromuscular retraining in hemiplegia. Arch Phys Med Rehabil **59**:258–267, 1978
31. Stein DG, Rosen JJ, Butters N (eds): Plasticity and Recovery of Function in Central Nervous System. New York, Academic Press, 1974
32. Winchester P, Montgomery J, Bowman B, Hislop H: Effects of feedback stimulation training and cyclical electrical stimulation on knee extension in hemiparetic patients. Phys Ther **63**:1096–1103, 1983

Supplier

a. Electronic Medical Instruments, 1800 112th Avenue N.E., Suite 340E, Bellevue, WA 98004-2939