ORIGINAL ARTICLE

Neuromuscular Electric Stimulation Effect on Lower-Extremity Motor Recovery and Gait Kinematics of Patients With Stroke: A Randomized Controlled Trial

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ABSTRACT. Yavuzer G, Geler-Külcü D, Sonel-Tur B, Kutlay S, Ergin S, Stam HJ. Neuromuscular electric stimulation effect on lower-extremity motor recovery and gait kinematics of patients with stroke: a randomized controlled trial. Arch Phys Med Rehabil 2006;87:536-40.

Objective: To evaluate the effects of neuromuscular electric stimulation (NMES) of the tibialis anterior muscle on motor recovery and gait kinematics of patients with stroke.

Design: Randomized, controlled, assessor-blinded trial.

Setting: Rehabilitation ward and gait laboratory of a university hospital.

Participants: A total of 25 consecutive inpatients with stroke (mean age, 55y), all within 6 months poststroke and without volitional ankle dorsiflexion.

Intervention: Both the NMES group (n=12) and the control group (n=13) participated in a conventional stroke rehabilitation program, 5 days a week for 4 weeks. The NMES group also received 10 minutes of NMES to the tibialis anterior muscle of the paretic limb.

Main Outcome Measures: Brunnstrom stages of motor recovery and kinematic characteristics of gait.

Results: Brunnstrom stages improved significantly in both groups ($P<.05$). In total, 58% of the NMES group and 61% of the control group gained voluntary ankle dorsiflexion. Between-group difference of percentage change was not significant ($P>.05$). Gait kinematics was improved in both groups, but the difference between groups was not significant.

Conclusions: NMES of the tibialis anterior muscle combined with a conventional stroke rehabilitation program was not superior to a conventional stroke rehabilitation program alone, in terms of lower-extremity motor recovery and gait kinematics.

Key Words: Cerebrovascular accident; Electric stimulation; Gait; Rehabilitation.

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DESPITE UNDERGOING REHABILITATION, many people are left with a walking deficit after stroke.[1] Motor weakness, poor motor control, and spasticity result in an altered gait pattern, poor balance, risk of falls, and increased energy expenditure during walking.[2-4] Ineffective ankle dorsiflexion during swing (drop foot) and failure to achieve heel strike at initial contact are common problems that disturb gait pattern after stroke.[5,6] Voluntary ankle dorsiflexion in the lower extremity is a standpoint indicating the achievement of selective motor control.[7] Once voluntary movement is achieved (Brunnstrom stages II or higher), synergistic patterns are then modified to selective (out-of-synergy) patterns. Many treatments are prescribed to increase gait efficiency of chronic stroke patients who cannot perform voluntary ankle dorsiflexion, such as 1- or 2-channel peroneal nerve stimulators,[8-10] functional electric stimulation (FES),[11-15] and solid ankle-foot orthosis.[16]

FES refers to the regular use of electric stimulation to achieve overall functional improvement for the patient.[13] Studies of subjects late after stroke (>6mo) have shown that FES has a positive orthotic effect on walking ability.[8,13-15] Thompson and Stein[17] reported that increased activation of the tibialis anterior muscle during FES-aided walking increased afferent inputs to the central nervous system and thereby influenced plasticity in healthy subjects. Khaslavskaia et al[18] have shown that repetitive electric stimulation of the common peroneal nerve leads to long-standing sensorimotor cortical reorganization in healthy subjects. It is possible that more benefit could be gained by applying neuromuscular electric stimulation (NMES) early after stroke.[19]

In this study, we hypothesized that repetitive dorsiflexion of the ankle by NMES may enhance selective motor control and improve gait kinematics during the first 6 months after stroke. Our purpose was to determine whether combining NMES with a conventional stroke rehabilitation program is more effective than a conventional program alone in facilitating recovery of selective motor control in the lower extremity, and in improving gait kinematics after stroke.

METHODS

Participants

The study included 25 consecutive inpatients with hemiparesis resulting from stroke. Their mean ages and time since stroke ± standard deviation (SD) were 55.3±8.2 years and 2.4±1.1 months, respectively. Stroke was defined as an acute event of cerebrovascular origin causing focal or global neurologic dysfunction lasting more than 24 hours,[20] as diagnosed by a neurologist and confirmed by computed tomography or magnetic resonance imaging. Patients were required to meet the following criteria for inclusion in the study: (1) first episode of unilateral stroke with hemiparesis during the previous 6 months, (2) a score between 1 and 3 inclusive on the Brunnstrom stages for the lower extremity, (3) ability to understand and follow simple verbal instructions, (4) ambulatory before stroke, (5) no medical contraindication to walking or to electric stimulation, and (6) ability to stand with or without assistance and to take at least 1 or more steps with or without assistance. The protocol was approved by the Ankara University Ethics Committee.

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0003-9993/06/8704-10317$32.00/0
doi:10.1016/j.apmr.2005.12.041

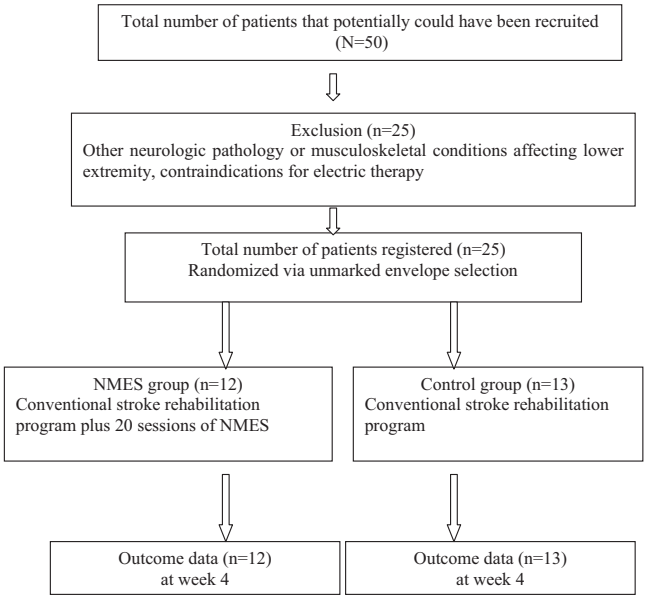

Fig 1. Flow diagram for randomized subject assignment.

Sample Size

The required sample size was determined by using the pooled estimate of within-group SDs obtained from pilot data. The minimal effect size for NMES in motor recovery has been reported as .54 for stroke patients.[21] Power calculations indicated that a sample of 25 subjects would provide an 80% (β=.20) chance of detecting a 20% (α=.05) difference in improvement between groups.

Design

We used an assessor-blinded, randomized, controlled design in this study. The physician who performed the gait analysis was blinded to the use of NMES; however, neither the patients nor the physiotherapist who delivered the NMES were blinded because it was impossible to do so given the obvious muscle contraction produced. Patients were randomized after initial evaluation by selecting a sealed, unmarked envelope containing a letter that informed them of their group allocation.[22] The blinded physician prepared the envelopes and the physiotherapist who delivered the NMES held them. After randomization, 13 patients were assigned to the control group (conventional rehabilitation program) and the remaining 12 were assigned to the NMES group (conventional rehabilitation program plus NMES) (fig 1). The control group did not receive sham stimulation.

Intervention

All 25 subjects participated in a conventional stroke rehabilitation program, 5 days a week, 2 to 5 hours a day, for 4 weeks. The conventional program is patient-specific and consists of neurodevelopmental facilitation techniques, physiotherapy, occupational therapy, and speech therapy (if needed). The NMES group also received 10 minutes of NMES to the tibialis anterior muscle of the paretic limb once daily, 5 days a week for 4 weeks.[23] Two sponge-type electrodes with rubber carriers were placed on the target muscle close to the insertion points (bipolar placement). Transcutaneous NMES was given with the Sonopuls 992,[a] and a surge-alternating current was used at a frequency of 80Hz to stimulate muscle contraction. The stimulator on time of 10 seconds consisted of 2 seconds of ramp up and 1 second of ramp down. The off time was 50 seconds. The amplitude was adjusted to produce muscle contraction without affecting the patient's comfort.[23,24] We did not ask patients to volitionally contract their muscles during the NMES application because any volitional effort may stimulate flexor synergy and spastic co-contraction.

Outcome Measures

Lower-extremity motor recovery. We assessed lower-extremity motor recovery using the Brunnstrom stages for the lower extremity.[7] The 6 stages of the Brunnstrom scale for the lower extremity are: (1) flaccidity, (2) synergy development (minimal voluntary movements), (3) voluntary synergistic movement (combined hip flexion, knee flexion, and ankle dorsiflexion, both sitting and standing), (4) some movements deviating from synergy (knee flexion >90° and ankle dorsiflexion with the heel on the floor in the sitting position), (5) independence from basic synergies (isolated knee flexion with the hip extended and isolated ankle dorsiflexion with the knee extended in the standing position), and (6) isolated joint movements (hip abduction in the standing position, knee rotation with inversion and eversion of the ankle in the sitting position). We used the Brunnstrom scale because it reflects underlying motor control based on clinical assessment of movement quality. Brunnstrom stages I through III indicate more synergistic and mass movements, whereas stages IV through VI indicate isolated and selective movements.[25] Patients were classified into 2 subgroups in terms of motor stage, that is, those with no selective motor control (Brunnstrom stage ≤III) versus those with some (Brunnstrom stage ≥IV) control.

Gait kinematics. Our outcome parameters were walking velocity, step length, percentage of stance phase at the paretic side, sagittal plane kinematics of pelvis, hip, knee, and ankle, maximum ankle dorsiflexion angle at swing, and maximum ankle plantarflexion angle at initial contact. Three-dimensional gait data were collected with the Vicon 370 system[b] and processed by the Vicon Clinical Manager (version 3.2) software.[b] Anthropometric data collected included height, weight, leg length, and joint width of the knee and ankle. Fifteen passively reflective markers were placed on standard and specific anatomic landmarks: sacrum, bilateral anterior superior iliac spine, middle thigh, lateral knee (directly lateral to axis of rotation), middle shank (the middle point between the knee

Table 1: Characteristics of the 2 Study Groups

Variable	NMES (n=12)	Control (n=13)	*P*
Age (y)	56.3±7.5	54.2±8.1	.25
Sex (women/men)	5/7	4/9	.69
Type of injury (ischemia/hemorrhage)	10/2	10/3	.54
Paretic side (right/left)	5/7	7/6	.69
Time since stroke (mo)	2.4±1.7	2.3±1.3	.17
Height (cm)	163.2±9.6	162.0±8.9	.11
Weight (kg)	74.5±11.2	75.2±9.4	.16
Brunnstrom stages (II/III)	3/9	3/10	.59
Modified Ashworth Scale score	3.2±2.1	3.3±2.2	.31
FIM admission score	69.2±27.4	67.2±19.4	.21
Walking velocity (m/s)			.02
Mean	.18±.03	.45±.26	
Median	.20	.39	

NOTE. Values are mean ± SD or as indicated.

Table 2: Outcome Measures in the NMES Group and the Control Group

Outcome Measures	Pretreatment		Posttreatment	
	NMES	Control	NMES	Control
Brunnstrom stage for lower extremity	2.7±1.1	2.9±1.2	4.8±1.3	4.1±1.1
Walking velocity (m/s)	0.18±0.03	0.45±0.26*	0.23±0.11	0.51±0.22
Step length (m)	0.24±0.11	0.29±0.12	0.28±0.12	0.35±0.11
% of stance phase (paretic side)	58.7±3.5	59.1±2.5	59.9±4.7	58.6±3.8
Pelvis (deg)†	11.2±6.7	6.02±3.3	10.0±5.2	4.7±2.9
Hip (deg)†	15.6±9.6	27.3±10.0*	16.3±7.8	28.0±9.8
Knee (deg)†	21.2±11.2	35.7±14.9*	22.9±15.7	36.6±9.9
Ankle (deg)†	14.4±13.7	16.3±4.6	16.5±5.9	20.9±16.3
Maximum ankle DF at swing (deg)	−6.2±2.3	−5.9±2.4	−4.5±3.1	−5.1±1.2
Maximum ankle PF at initial contact (deg)	−12.8±0.9	−13.0±1.4	−11.2±4.5	−12.4±5.1

NOTE: Values are mean ± SD.
Abbreviations: DF, dorsiflexion; PF, plantarflexion.
*$P<.05$.
†Sagittal plane total excursion.

marker and the lateral malleolus), lateral malleolus, and heel and forefoot between the second and third metatarsal head.[2] After subjects were instrumented with retroreflective markers, they were instructed to walk at a self-selected speed over a 10-m walkway, during which data were captured. Five cameras recorded (at 60Hz) the 3-dimensional spatial location of each marker as the subject walked. We used the best data of 3 trials in our analysis. The trial in which all the markers were clearly and automatically identified by the system was accepted as providing the best data.

Statistical Analysis

We analyzed the data using SPSS[c] for Windows. The group means between the NMES and the control groups were compared using nonparametric paired and unpaired *t* tests. We preferred nonparametric statistics because of the abnormal distribution of the data. The percentage change between pre- and posttreatment data for both groups was calculated as 100 × (pretreatment minus posttreatment)/pretreatment. We used the chi-square test to compare the groups in terms of the number of patients with Brunnstrom stages I through III or IV through VI. Significance was set at .05.

RESULTS

Initial and final evaluations were made 1 to 3 days before and after the 4 weeks of the treatment period. None of the patients missed more than 1 scheduled session during the study, and all completed the study. Demographic and clinical characteristics of the groups are presented in table 1. Age, sex, height, weight, injury characteristics, time since stroke, baseline Modified Ashworth Scale score of ankle plantarflexor muscles, Brunnstrom stages in the lower extremity, FIM instrument scores, and walking velocity were all similar in both groups.

Lower-Extremity Motor Recovery

Brunnstrom stages improved significantly in both groups ($P<.05$) after the treatment. The difference between groups in terms of the percentage change, however, was not significant (table 2). In total, 7 patients (58%) in the NMES group and 8 (61%) in the control group gained voluntary ankle dorsiflexion. The between-group difference of percentage change was not significant ($P>.05$) (table 3).

Gait Kinematics

The 2 groups' mean values ± SD of assessed parameters at pre- and posttreatment are presented in table 2. There was no significant difference between the groups in terms of all initial clinical characteristics except for walking velocity. Pretreatment mean walking velocity values of the NMES group were significantly lower than those in the control group ($P=.02$). Time-distance and sagittal plane gait kinematics were improved in both groups, but the difference between pre- and posttreatment data for each group, and the percentage of change between the groups, was not significant (table 3).

DISCUSSION

This study revealed that in our group of stroke patients, NMES of the tibialis anterior muscle combined with a conventional rehabilitation program does not provide additional benefit in terms of lower-extremity motor recovery and gait kinematics.

Lower-Extremity Motor Recovery

The primary outcome parameter of this study was achievement of voluntary ankle dorsiflexion at the paretic side, representing selective motor control. Ankle dorsiflexion is an important kinematic aspect of the swing and initial stance phase

Table 3: Percentage Change After Treatment in the NMES Group and the Control Group

Outcome Measures	NMES Group (n=12) (%)	Control Group (n=13) (%)	*P*
ΔBrunnstrom stage for lower extremity	48*	41*	.25
Brunnstrom stages from I–III to IV–VI	58	61	.51
ΔWalking velocity (m/s)	16	15	.89
ΔStep length (m)	17	19	.34
Δ% of stance phase (paretic side)	2	1	.56
ΔPelvis (deg)†	11	14	.86
ΔHip (deg)†	4	3	.75
ΔKnee (deg)†	8	3	.42
ΔAnkle (deg)†	15	18	.45
ΔMaximum ankle DF at swing (deg)	17	14	.62
ΔMaximum ankle PF at initial contact (deg)	13	11	.71

Abbreviation: Δ, percentage change between pre- and posttreatment.
*$P<.05$.
†Sagittal plane total excursion.

of the gait cycle. Ankle movement training facilitates brain reorganization, and the angle paradigm may serve as an ongoing physiologic assay of the optimal type, duration, and intensity of rehabilitative gait training.[26] Dobkin et al[27] demonstrated that the supraspinal sensorimotor network for the neural control of walking can be assessed indirectly by ankle dorsiflexion.

Because none of our patients had voluntary ankle dorsiflexion at baseline evaluation, we did not ask them to participate in electric stimulation in order not to stimulate flexor synergy of the lower extremity. Because repeated, task-specific exercise protocols induce brain reorganization,[28-31] we hypothesized that repetitive dorsiflexion of the ankle by NMES may induce use-dependent brain reorganizations responsible for selective motor control of the ankle. It has been reported, however, that active, repetitive, or triggered movement trainings that require skill acquisition[32,33] facilitate the motor recovery of stroke survivors.[17,34-37] Because our patients were cognitively inactive during the NMES therapy, electrically evoked ankle movements in dorsiflexion did not create any cognitive effort or investment. Khaslavskaia et al[18] used a similar repetitive electric stimulation of the common peroneal nerve and observed a significant increase in motor cortical excitability that was more pronounced when agonistic voluntary exercise was coupled with electric stimulation.

In a similar study, Yan et al[38] reported that 15 sessions of simple FES, given 30 minutes per session along with standard rehabilitation 5 days a week, improved motor recovery and functional mobility in acute stroke subjects, more than did placebo stimulation and standard rehabilitation, or standard rehabilitation only. In that study, Yan applied simple FES using surface electrodes on quadriceps, hamstring, tibialis anterior, and medial gastrocnemius muscles mimicking normal gait, while the affected lower extremity was supported in a sling. They measured isometric voluntary contraction of ankle dorsiflexor and plantarflexor muscles by joint torque and surface electromyography, and found that percentage increases in maximum isometric voluntary contraction torque and integrated electromyographic signals of the FES group were significantly larger than those of the control group. Although purpose of our study was similar to that of Yan (ie, to enhance neuroplasticity and remind patients how to perform the movement properly during electric stimulation), the 2 studies differ both in patient characteristics and in treatment intervention and outcome parameters. Yan found a significantly larger percentage of voluntary ankle dorsiflexion in the FES group at the end of the first week. In that study, electric stimulation (with 0.3-ms pulses at 30Hz) was applied, starting at 8.7 ± 5.8 days after stroke, to the quadriceps, hamstring, tibialis anterior, and medial gastrocnemius muscles, for 15 sessions of 60 minutes each. In our study, we applied electric stimulation (with 0.1-ms pulses at 80Hz) only to the tibialis anterior muscle, for 20 sessions of 10 minutes each about 2.4 months after stroke, as suggested elsewhere.[23,24] There are no uniform guidelines that specify a certain number of NMES sessions or the duration of the daily stimulation times. Although duration, intensity, and selected mode of the electric stimulation were not found to be associated with stroke outcome,[13] the timing of the intervention is important. Natural recovery of walking function occurs within the first 11 weeks after stroke, and early and intensive treatment significantly improves motor and functional outcome.[39] Although most of the overall improvement in motor functions occurs within the first month after stroke, modulation of motor networks may still be possible in some patients up to 6 months later. The reliability of outcome studies of specific treatments during the early poststroke rehabilitation is, however, limited by the variables of spontaneous recovery.[34] Thus, we included patients during the 2 to 6 months after stroke in order to avoid the variability of spontaneous recovery.

Gait Kinematics

Both of our groups achieved an improvement in gait characteristics of the paretic side; however, the between-group difference was not significant. Walking velocity is the most suitable temporal stride variable for measuring gait performance.[40,41] Burridge et al[5] reported that a 10% improvement in walking velocity was considered to be functionally relevant. In our study, although walking velocity increased both in the NMES (16%) and the control group (15%), the difference between pre- and posttreatment data was not significant, which may have been because of our small sample size. Unfortunately, there was a significant difference between the groups in baseline walking velocity. It is well known that lower-extremity motor recovery[25] and functional status[42] are the main determinants of walking velocity. One may expect this difference to cause bias in the investigation; however, walking velocity is positively correlated with motor stages of the proximal lower extremity, but not with the motor stages of the ankle and foot.[25]

We did not use placebo (sham) stimulation together with the conventional stroke rehabilitation program in the control group. This was mainly because of the short period of the stimulation (10min), which was unlikely to cause a bias between the groups in terms of treatment intensity. Moreover, it has been reported that even the placement of electrodes on the skin is likely to stimulate mechanosensitive nerve fibers.[43] Thus, it has been suggested that in designing trials after stroke, a control group with no intervention except conventional rehabilitation could provide better information.[44]

CONCLUSIONS

NMES of the tibialis anterior muscle combined with a conventional rehabilitation program was not superior to the conventional stroke rehabilitation program alone, in terms of selective motor control and gait kinematics of our group of patients with stroke.

References

1. Duncan PW, Goldstein LB, Matchar D, Divine GW, Feussner J. Measurement of motor recovery after stroke: outcome assessment and sample size requirements. Stroke 1992;23:1084-9.
2. De Quervain IA, Simon SR, Leurgans S, Pease WS, McAllister D. Gait pattern in the early recovery period after stroke. J Bone Joint Surg Am 1996;78:1506-14.
3. Lehmann JF, Condon SM, Price R, deLateur BJ. Gait abnormalities in hemiplegia: their correction by ankle-foot orthoses. Arch Phys Med Rehabil 1987;68:763-71.
4. Tyson SF. Trunk kinematics in hemiplegic gait and the effect of walking aids. Clin Rehabil 1999;13:295-300.
5. Burridge JH, Wood DE, Taylor PN, McLellan DL. Indices to describe different muscle activation patterns, identified during treadmill walking, in people with spastic drop-foot. Med Eng Physics 2001;23:427-34.
6. Roth EJ, Harvey RL. Rehabilitation of stroke syndromes. In: Braddom RL, editor. Handbook of physical medicine and rehabilitation. 2nd ed. Philadelphia: WB Saunders; 2004. p 727-56.
7. Sawner K, Lavigne J. Brunnstrom's movement therapy in hemiplegia: a neurophysiological approach. Philadelphia: JB Lippincott; 1992.
8. Burridge JH, Taylor PN, Hagan SA, Wood DE, Swain ID. The effects of common peroneal stimulation on the effort and speed of walking: a randomized controlled trial with chronic hemiplegic patients. Clin Rehabil 1997;11:201-10.

9. Voigt M, Sinkjaer T. Kinematic and kinetic analysis of the walking pattern in hemiplegic patients with drop-foot using a peroneal nerve stimulator. Clin Biomech (Bristol, Avon) 2000;15:340-51.
10. Kottink AI, Buschman HP, Kenney LP, et al. The sensitivity and selectivity of an implantable two-channel peroneal nerve stimulator system for restoration of dropped foot. Neuromodulation 2004; 7:277-83.
11. Bogataj U, Gros N, Klajic M, Acimovic R, Malezic M. The rehabilitation of gait in patients with hemiplegia: a comparison between conventional therapy and multichannel functional electrical stimulation therapy. Phys Ther 1995;75:490-502.
12. Daly JJ, Ruff RL. Electrically induced recovery of volitional gait components for older stroke patients. Am J Phys Med Rehabil 2000;79:349-60.
13. Kottink AI, Oostendorp LJ, Buurke JH, Nene AV, Hermens HJ, IJzerman MJ. The orthotic effect of functional electrical stimulation on the improvement of walking in stroke patients with a dropped foot: a systematic review. Artif Organs 2004;28:577-86.
14. Kenney L, Bultstra G, Buschman R, et al. An implantable two channel drop foot stimulator: initial clinical results. Artif Organs 2002;26:267-70.
15. Waters RL, McNeal D, Perry J. Experimental correction of electrical stimulation of the peroneal nerve. J Bone Joint Surg Am 1975;57:1047-54.
16. Gök H, Küçükdeveci A, Altınkaynak H, Yavuzer G, Ergin S. Effects of ankle-foot orthoses on hemiparetic gait. Clin Rehabil 2003;17:137-9.
17. Thompson AK, Stein RB. Short-term effects of functional electrical stimulation on motor-evoked potentials in ankle flexor and extensor muscles. Exp Brain Res 2004;159:491-500.
18. Khaslavskaia S, Ladouceur M, Sinkjaer T. Increase in tibialis anterior motor cortex excitability following repetitive electrical stimulation of the common peroneal nerve. Exp Brain Res 2002; 145:309-15.
19. Chae J, Bethoux F, Bohinc T, Dobos L, Davis T, Friedl A. Neuromuscular stimulation for upper extremity motor and functional recovery in acute hemiplegia. Stroke 1998;29:975-9.
20. Foulkes MA, Wolf PA, Price TR, Mohr JP, Hier DB. The stroke data bank: design, methods, and baseline characteristics. Stroke 1988;19:547-54.
21. Chae J, Yu D. Neuromuscular stimulation for motor relearning in hemiplegia. Crit Rev Phys Med Rehabil Med 1999;11:279-97.
22. Johnson CA, Burridge JH, Strike PW, Wood DE, Swain ID. The effect of combined use of botulinum toxin type A and functional electric stimulation in the treatment of spastic drop foot after stroke: a preliminary investigation. Arch Phys Med Rehabil 2004; 85:902-9.
23. DeVahl J. Neuromuscular electrical stimulation in rehabilitation. In: Gersh MR, editor. Electrotherapy in rehabilitation. Philadelphia: FA Davis; 1992. p 218-68.
24. Burridge JH, Swain ID, Taylor PN. Functional electrical stimulation: a review of the literature published on common peroneal nerve stimulation for the correction of dropped foot. Rev Clin Geriatr 1998;8:155-61.
25. Chen CL, Chen HC, Tang SF, Wu CY, Cheng PT, Hong WH. Gait performance with compensatory adaptations in stroke patients with different degrees of motor recovery. Am J Phys Med Rehabil 2003;82:925-35.
26. Carey JR, Anderson KM, Kimberley TJ, Lewis SM, Auerbach EJ, Ugurbil K. fMRI analysis of ankle movement tracking in subject with stroke. Exp Brain Res 2004;154:281-90.
27. Dobkin BH, Firestine A, West M, Saremi K, Woods R. Ankle dorsiflexion as an fMRI paradigm to assay motor control for walking during rehabilitation. Neuroimage 2004;23:370-81.
28. Asanuma H, Keller A. Neurobiological basis of motor relearning and memory. Concepts Neurosci 1991;2:1-30.
29. Taub E, Wolf SL. Constraint induced movement techniques to facilitate upper extremity use in stroke patients. Top Stroke Rehabil 1997;3:38-61.
30. Hesse SA, Bertelt C, Jahnke MT, et al. Treadmill training with partial body weight support compared with physiotherapy in nonambulatory hemiparetic patients. Stroke 1995;26:976-81.
31. Cunha IT, Lim PC, Qureshy H, Henson H, Monga T, Protas EJ. A comparison of regular rehabilitation and regular rehabilitation with supported treadmill ambulation training for acute stroke patients. J Rehabil Res Dev 2001;38:245-55.
32. Plautz EJ, Milliken GW, Nudo RJ. Effects of repetitive motor training on movement representations in adult squirrel monkeys: role of use versus learning. Neurobiol Learn Mem 2000;74:27-55.
33. Kleim JA, Barbay S, Nudo RJ. Functional reorganization of the rat motor cortex following motor skill learning. J Neurophysiol 1998; 80:3321-5.
34. Ring H, Rosenthal N. Controlled study of neuroprosthetic functional electrical stimulation in sub-acute post stroke rehabilitation. J Rehabil Med 2005;37:32-6.
35. Landers M. Treatment-induced neuroplasticity following focal injury to the motor cortex. Int J Rehabil Res 2004;27:1-5.
36. Chae J. Neuromuscular electrical stimulation for motor relearning in hemiparesis. Phys Med Rehabil Clin N Am 2003;14(Suppl): 93-109.
37. Kroon JR, IJzerman MJ, Chae J, Lankhorst GJ, Zilvold G. Relation between stimulation and clinical outcome in studies using electrical stimulation to improve motor control of the upper extremity in stroke. J Rehabil Med 2005;37:65-74.
38. Yan T, Hui-Chan CW, Li LS. Functional electrical stimulation improves motor recovery of the lower extremity and walking ability of subjects with first acute stroke. Stroke 2005;36:80-5.
39. Jorgensen HS, Nakayama H, Raaschou HO, Olsen TS. Recovery of walking function in stroke patients: the Copenhagen stroke study. Arch Phys Med Rehabil 1995;76:27-32.
40. Bohannon RW, Walsh S. Nature, reliability, and predictive value of muscle performance measures in patients with hemiparesis following stroke. Arch Phys Med Rehabil 1992;73:721-5.
41. Roth EJ, Merbitz C, Mroczek K. Hemiplegic gait: relationships between walking speed and other temporal parameters. Am J Phys Med Rehabil 1997;76:128-33.
42. Dettman MA, Linder MT, Sepic SB. Relationships among walking performance, postural stability, and functional assessments of the hemiplegic patient. Am J Phys Med 1987;66:77-90.
43. Vallbo AB, Olausson H, Wessberg J. Unmyelinated afferents constitute a second coding tactile stimuli of the human hairy skin. J Neurophysiol 1999;81:2753-63.
44. Johansson BB, Haker E, von Arbin M, et al. Acupuncture and transcutaneous nerve stimulation in stroke rehabilitation. Stroke 2001;32:707-13.

Suppliers

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