ORIGINAL ARTICLE

# Effect of Sensory-Amplitude Electric Stimulation on Motor Recovery and Gait Kinematics After Stroke: A Randomized Controlled Study

*Gunes Yavuzer, MD, PhD, Öznur Öken, MD, Mesut B. Atay, MD, Henk J. Stam, MD, PhD*

ABSTRACT. Yavuzer G, Öken Ö, Atay MB, Stam HJ. Effect of sensory-amplitude electric stimulation on motor recovery and gait kinematics after stroke: a randomized controlled study. Arch Phys Med Rehabil 2007;88:710-4.

**Objective:** To evaluate the effects of sensory-amplitude electric stimulation (SES) of the paretic leg on motor recovery and gait kinematics of patients with stroke.

**Design:** Randomized, controlled, double-blind study.

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

**Participants:** A total of 30 consecutive inpatients with stroke (mean age, 63.2y), all within 6 months poststroke and without volitional ankle dorsiflexion were studied.

**Intervention:** Both the SES group ($n=15$) and the placebo group ($n=15$) participated in a conventional stroke rehabilitation program 5 days a week for 4 weeks. The SES group also received 30 minutes of SES to the paretic leg without muscle contraction 5 days a week for 4 weeks.

**Main Outcome Measures:** Brunnstrom stages of motor recovery and time-distance and kinematic characteristics of gait.

**Results:** Brunnstrom stages improved significantly in both groups ($P<.05$). In total, 58% of the SES group and 56% of the placebo group gained voluntary ankle dorsiflexion. The between-group difference of percentage change was not significant ($P>.05$). Gait kinematics was improved in both groups, but the between-group difference was not significant.

**Conclusions:** In our patients with stroke, SES of the paretic leg was not superior to placebo in terms of lower-extremity motor recovery and gait kinematics.

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

SENSORY INPUT CAN MODULATE reorganization of the motor cortex,[1-3] which may be beneficial in therapeutic interventions to improve motor function in stroke rehabilitation.[4] Increased inflow of signals from sensory modalities could enhance plasticity of the brain and may partly explain beneficial effects of this treatment.[5] Afferent stimulation can be achieved in various ways.[6-12] Golaszewski et al[13] studied the effect of cutaneous stimulation of the hand in 6 healthy subjects in the immediate poststimulation period during simple motor tasks with magnetic resonance imaging; they reported that the afferent stimulation, delivered below the sensory threshold, was associated with increased signals in the primary and secondary motor and somatosensory areas, including the supplementary motor area.

Peripheral electric nerve stimulation enhances corticomotoneural excitability by activating group Ia large muscle afferents, group Ib afferents from Golgi organs, group II afferents from slow and rapidly adapting skin afferents, and cutaneous afferent fibers.[1,3,14] Long-term reorganization of the motor cortex has been reported for repetitive electric stimulation of peripheral nerves of swallowing[1] and hand[3] muscles. Median nerve stimulation has been used for neuroresuscitation of coma patients.[15] Khaslavskaia et al[14] showed in healthy subjects that reorganization can be elicited for lower-limb muscles via repetitive stimulation of common peroneal nerve. They concluded that changes in neural excitability related to lower-limb muscle can be increased by using afferent input. In a case study, Sullivan and Hedman[16] described a home program combining sensory amplitude electric stimulation and neuromuscular electric stimulation to the paretic arm, which increased upper-extremity function even 5 years after a stroke. Peurala et al[12] investigated the effects of cutaneous electric stimulation of the paretic limb by using glove or sock electrodes in patients with chronic stroke. They reported that subthreshold sensory stimulation may improve limb function late after stroke. Conforto et al[11] reported an improvement of pinch muscle strength during a 2-hour period of median nerve stimulation; they suggested that somatosensory stimulation may be a promising adjuvant to rehabilitation of the motor deficits in stroke patients. These studies suggest that ascending sensory information can have an influence on cortical motor circuits and their descending pathways.

In a recent meta-analysis, Robbins et al[17] reported that there was insufficient research to make conclusions regarding the effectiveness of sensory-threshold electric stimulation on improvement in walking after stroke and suggested further controlled studies. In this study, we hypothesized that sensory-amplitude electric stimulation (SES) of the paretic leg may enhance selective motor control and improve gait kinematics during the first 6 months after stroke. The aim was to investigate whether SES combined with a conventional stroke rehabilitation program is more effective than the conventional program and sham SES in facilitating recovery of selective motor control in the lower extremity and in improving gait kinematics after stroke.

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From the Department of Physical Medicine & Rehabilitation, Ankara University Faculty of Medicine, Ankara, Turkey (Yavuzer, Atay); Physical Medicine & Rehabilitation Clinic of Ankara State Hospital, Ankara, Turkey (Öken); and Erasmus University Medical Center, Department of Rehabilitation Medicine, Rotterdam, The Netherlands (Stam, Yavuzer).

Reprint requests to Gunes Yavuzer, MD, PhD, Mustafa Kemal Mahallesi, Barış Sitesi 87. sok No: 24, Ankara 06800, Turkey, e-mail: *gunesyavuzer@hotmail.com.*

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## METHODS

### Participants

The trial included 30 consecutive inpatients with hemiparesis after stroke who met the study criteria. 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 Brunnstrom stages of 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 (having pacemaker or venous thrombosis at the paretic leg), and (6) ability to stand with or without assistance and to take at least 1 or more steps with or without assistance. The mean age ± standard deviation (SD) was 63.2±9.7 years, and the mean time since stroke was 3.4±2.1 months. Stroke was defined as an acute event of cerebrovascular origin causing focal or global neurologic dysfunction lasting more than 24 hours and diagnosed by a neurologist and confirmed by computed tomography scan or magnetic resonance imaging. The protocol was approved by the Ankara University Ethics Committee.

### Design

A double-blind, randomized, controlled design was used. The patients and the physician (GY) who performed the outcome measures were blinded to the use of SES but not the therapist who delivered the electric stimulation. Patients were randomly assigned to 1 of the 2 groups after initial evaluation. We used the block randomization method to ensure an equal number of patients in each group. Blocks were numbered, and then a random-number generator program was used to select numbers that established the sequence in which blocks were allocated to one or the other group. A medical resident who was blinded to the research protocol and was not otherwise involved in the trial operated the random-number program. After randomization, 15 patients were assigned to the placebo group (conventional rehabilitation program plus sham SES), and the remaining 15 were assigned to the SES group (conventional rehabilitation program plus SES) (fig 1).

### Intervention

All subjects participated in a conventional stroke rehabilitation program delivered by multiple therapists 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 SES group also received 30 minutes of SES once daily 5 days a week for 4 weeks to the common peroneal nerve of the paretic leg. Two 6×8mm "sponge"-type electrodes with rubber carriers were placed on the anatomic localization of the common peroneal nerve (just below the capitulum fibulae of the lower leg) and on the belly of the tibialis anterior muscle while the patients were in supine position.[18] A Sonopuls 992[a] was used to deliver the asymmetric biphasic rectangular stimulation at a frequency of 35Hz with a pulse width of 240μs. The stimulation amplitude was adjusted at each session to the point in which the patient perceived a mild tingling sensation (≈10mA) but below an observable or palpable muscle contraction. A duty cycle of 10 seconds on and 10 seconds off was used to minimize sensory habituation.[16] The same setup was used for the placebo group without any stimulation. The machine was turned on so that there was a light to indicate that it was in operation. Patients in the placebo group were not told that they would feel the stimulation. The same therapist delivered the SES or sham stimulation.

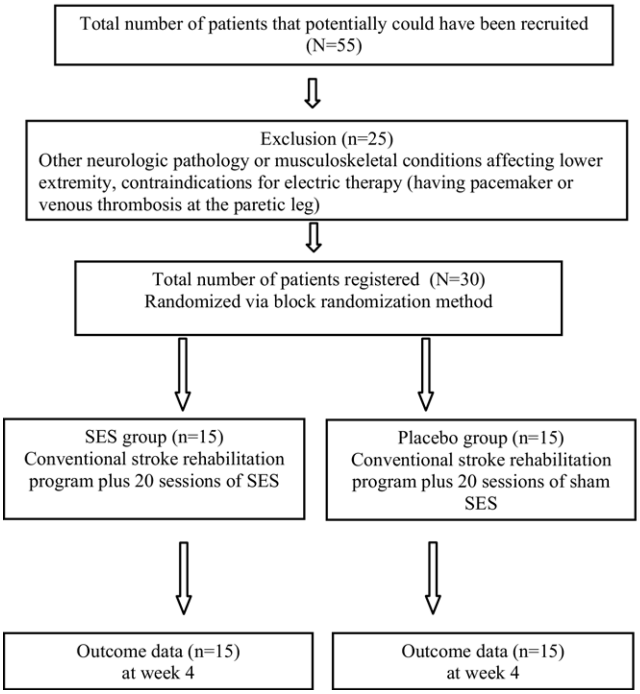

**Fig 1. Flow diagram for randomized subject assignment in this study.**

### Outcome Measures

Outcome measures were performed by the same investigator (GY) 1 to 3 days before and after the 4 weeks of the treatment period in the rehabilitation ward.

### Lower-Extremity Motor Recovery

Lower-extremity motor recovery was assessed by using the Brunnstrom stages for the lower extremity.[19] The 6 grades of the Brunnstrom stages for the lower extremity are as follows: (1) flaccity, (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 exceeding 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 and knee rotation with inversion and eversion of the ankle in the sitting position). The Brunnstrom stages were chosen because it reflects underlying motor control based on clinical assessment of movement quality. In the lower extremity, voluntary ankle dorsiflexion is a stand point indicating the achievement of selective motor control.[19] Once voluntary movement is achieved, synergistic patterns are then modified to selective (out of synergy) patterns. Brunnstrom stages I through III indicate more synergistic and mass movements, whereas stages IV through VI indicate isolated and selective movements. Patients were classified into 2 subgroups in terms of motor stage (ie, those with none [Brunnstrom stages ≤ III] versus those with some [Brunnstrom stages ≥ IV] selective motor control).[20]

**Table 1: Characteristics of the 2 Study Groups**

| Variable | SES (n=15) | Placebo (n=15) | *P* |
|---|---|---|---|
| Age (y) | 61.9±10.01 | 64.4±9.8 | .51 |
| Sex (women/men) | 8/7 | 6/9 | .20 |
| Type of injury (ischemia/ hemorrhage) | 12/3 | 10/5 | .86 |
| Paretic side (right/left) | 8/7 | 9/6 | .67 |
| Time since stroke (mo) | 3.5±2.1 | 3.4±2.3 | .18 |
| Height (cm) | 161.5±11.7 | 163.0±9.9 | .13 |
| Weight (kg) | 81.5±9.7 | 79.2±11.4 | .24 |
| Brunnstrom stages (II/III) | 3/12 | 2/13 | .17 |
| FIM admission score | 72.3±18.3 | 73.4±16.6 | .75 |
| Walking velocity (m/s) | 0.31±0.18 | 0.36±0.22 | .85 |

NOTE. Values are mean ± SD or n.

### Gait Kinematics

Walking velocity; step length; percentage of stance phase at the paretic side; sagittal plane kinematics of the pelvis, hip, knee, and ankle; maximum ankle dorsiflexion angle at swing; and maximum ankle plantarflexion angle at initial contact were selected as outcome parameters. Three-dimensional gait data were collected with the Vicon 370 system[b] and processed by the Vicon Clinical Manager (version 3.2) software. Anthropometric data including height, weight, leg length, and joint width of the knee and ankle were collected. Fifteen passively reflective markers were placed on the following 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 marker and the lateral malleolus), lateral malleolus, heel, and forefoot between the second and third metatarsal head.[21] Subjects were instructed to walk at a self-selected speed over a 10-m walkway during which data capture was completed. Five cameras recorded (at 60Hz) the 3-dimensional spatial location of each marker as the subject walked. The best data of 3 trials were used in analysis. The trial in which all the markers were clearly and automatically identified by the system was determined as providing the best data.

### Statistical Analysis

Data were analyzed by using SPSS[c] for Windows. The percentage change between pre- and posttreatment data for both groups was calculated as: 100 × (pretreatment − posttreatment)/pretreatment.

The group means and percentage changes were compared between the SES and the placebo group by using nonparametric paired and unpaired *t* tests. The chi-square test was used to compare the groups in terms of the number of patients with Brunnstrom stages for lower extremity I through III or IV through VI. We preferred nonparametric statistics because of the abnormal distribution of the data. 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 of them finished the study. Demographic and clinical characteristics of the 2 groups are given 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 did not differ statistically between the groups.

### Lower-Extremity Motor Recovery

There was a statistically significant improvement in pre- to posttreatment mean Brunnstrom scores in both groups ($P<.05$). However, the difference between the 2 groups in terms of the percentage change was not significant (table 2). In total, 60% (n=9) of the patients in the SES group and 53% (n=8) of the patients in the placebo group improved from Brunnstrom stages (I−III) to Brunnstrom stages (IV−VI). The between-group difference of the percentage change was not significant ($P>.05$) (table 3).

### Gait Kinematics

The mean values ± SD of assessed parameters of the groups at pre- and posttreatment are given in table 2. There was no significant difference between the groups in any of the initial clinical characteristics. Time-distance and sagittal plane gait kinematics were improved in both groups. However, neither the difference between pre- and posttreatment data for each group nor the percentage of change between the groups was significant (see table 3).

**Table 2: Outcome Measures in the SES Group and the Placebo Group**

| Outcome Measures | Pretreatment | | Posttreatment | |
|---|---|---|---|---|
| | SES | Placebo | SES | Placebo |
| Brunnstrom stages of lower extremity | 3.2±1.6 | 3.3±1.2 | 4.1±1.4* | 3.5±0.9* |
| Walking velocity (m/s) | 0.31±0.18 | 0.36±0.22 | 0.34±0.11 | 0.37±0.20 |
| Step length (m) | 0.29±0.11 | 0.28±0.16 | 0.32±0.12 | 0.30±0.12 |
| % of stance phase (paretic side) | 59.1±3.5 | 58.0±5.5 | 58.7±4.7 | 58.9±5.7 |
| Pelvis (deg)† | 13.2±5.7 | 12.2±3.3 | 10.3±6.2 | 11.0±3.1 |
| Hip (deg)† | 16.6±8.6 | 17.3±10.0 | 16.9±7.8 | 17.7±7.9 |
| Knee (deg)† | 25.4±10.2 | 27.7±14.9 | 27.9±11.7 | 28.1±9.8 |
| Ankle (deg)† | 17.1±12.7 | 16.1±3.8 | 18.5±4.9 | 16.6±9.9 |
| Maximum ankle DF at swing (deg) | −5.9±2.3 | −5.8±2.6 | −4.6±4.1 | −4.3±1.5 |
| Maximum ankle PF at initial contact (deg) | −1.8±0.9 | −3.0±1.5 | 1.2±7.5 | −2.1±1.2 |

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

**Table 3: Percentage Change After Treatment in the SES Group and the Placebo Group**

| Outcome Measures | SES Group (%) (n=15) | Placebo Group (%) (n=15) | *P* |
|---|---|---|---|
| Δ Brunnstrom stages of lower extremity | 46 | 44 | .31 |
| Brunnstrom stages from I–III to IV–VI | 60 | 53 | .09 |
| ΔWalking velocity (m/s) | 13 | 13 | .97 |
| ΔStep length (m) | 18 | 19 | .34 |
| Δ% of stance phase (paretic side) | 2 | 1 | .60 |
| ΔPelvis (deg)* | 12 | 14 | .89 |
| ΔHip (deg)* | 4 | 3 | .75 |
| ΔKnee (deg)* | 7 | 3 | .44 |
| ΔAnkle (deg)* | 16 | 19 | .47 |
| ΔMaximum ankle DF at swing (deg)* | 22 | 25 | .44 |
| ΔMaximum ankle PF at initial contact (deg)* | 14 | 12 | .70 |

Abbreviation: Δ, percentage change between pre- and posttreatment.
*Sagittal plane total excursion.

## DISCUSSION

This study reveals that SES of the paretic leg in addition to a conventional rehabilitation program does not provide additional benefit in terms of lower-extremity motor recovery and gait kinematics in our group of patients with stroke. The primary goal of this study was achievement of voluntary motor control in the lower extremity and consequently improve gait pattern after stroke. Dobkin et al[22] showed that assessment of ankle dorsiflexion gives information about neural control of walking. We followed the isolated ankle dorsiflexion of the patients by both clinical examination (Brunnstrom stages) and by using quantitative gait analysis. Clinically, more than 50% of our patients in both groups gained selective ankle dorsiflexion with similar percentage changes. However, changes in gait kinematics were not significant in any of the groups. Walking velocity is the most suitable temporal stride variable for measuring gait performance.[16] Burridge et al[23] reported that a 10% improvement in walking velocity was considered to be functionally relevant. In the present study, although walking velocity increased both in the SES (13%) and in the placebo group (13%), the difference between pre- and posttreatment data was not significant.

Despite encouraging results of afferent stimulation of hand after stroke in terms of voluntary motor control,[11,12,16,24] there is no evidence yet for lower extremity.[17] In a randomized controlled trial (RCT) by Chen et al,[25] stroke patients received SES via electrodes placed over the Achilles' tendon and gastrocnemius for 20 minutes 6 times a week for 1 month. They reported significant improvement in gait speed. Peurala et al[12] treated subjects with SES on the foot and ankle for 30 minutes twice a day for 3 weeks and reported significant improvement in motor recovery but not in gait speed. Both studies used a 10-m walk test, and subjects were in the chronic stage of recovery.

In a recent meta-analysis, Robbins et al[17] reported that motor threshold electric stimulation improves gait speed and can be an effective tool in the rehabilitation of patients after stroke. In motor stimulation, the current intensity is high enough to exceed motor threshold and evoke muscle contractions that are associated with cutaneous, muscle, and joint proprioceptive afferent feedback. However, in sensory stimulation, the low-current intensity evokes a sensory reaction without muscle contraction associated only with cutaneous afferents. In our study, both the SES and the placebo group achieved an improvement in gait characteristics of the paretic side; however, the between-group difference was not significant.

In a Cochrane review,[26] the results of 24 RCTs of electrostimulation delivered to the peripheral neuromuscular system, which was designed to improve voluntary movement control, functional motor ability, and activities of daily living, was reported. In this review, Pomeroy et al[26] reported that the majority of findings in favor of electric stimulation were found when it was compared with a group of stroke patients who were not receiving any treatment. There were no differences either between electrostimulation and placebo or between electrostimulation and another type of physical therapy. For the placebo group, we used sham stimulation together with the conventional rehabilitation program. Because it has been reported that even the placement of electrodes on the skin is likely to stimulate mechanosensitive nerve fibers,[27] we might have caused an iatrogenic afferent sensory input in our placebo group.

We delivered the electric stimulation without any active involvement of the patients. However, it has been shown that active repetitive movements are a key factor in recovery after stroke, but beyond simple repetition, an element of problem solving is also required.[28] A recent review[29] reported that triggered stimulation was more likely to yield improvements in motor control than nontriggered stimulation after stroke. They did not detect a relationship between stimulation parameters, duration of stimulation, and subject characteristics and clinical outcome. The same group suggested that the behavioral experiences that induce long-term plasticity in humans are likely to be those activities that are important and meaningful and require cognitive investment and effort. Thus, repetitive movement therapy in which the subject is cognitively involved in generating the movement is more likely to be important and meaningful.

There are no uniform guidelines concerning the overall duration of electric stimulation or for the daily stimulation time. Although it is reported that duration, intensity, and selected mode of the electric stimulation are not associated with stroke outcome, the timing of the intervention is important.[30] In the present study, we included patients during the first 2 to 6 months after stroke. Natural recovery of walking function occurs within the first 11 weeks after stroke, and early and intensive treatment significantly improve motor and functional outcome. Although most of the overall improvement in motor functions occur within the first several months after stroke,[31] modulation of motor networks may still be possible in chronic stroke patients.

## CONCLUSIONS

SES of the paretic leg in addition to a conventional rehabilitation program is not superior to the conventional rehabilitation program and placebo in terms of selective motor control and gait kinematics of our group of patients with stroke.

### References

1. Hamdy S, Rothwell JC, Aziz Q, Singh KD, Thompson DG. Long-term reorganization of human motor cortex driven by short-term sensory stimulation. Nat Neurosci 1998;1:64-8.
2. Chen R, Corwell B, Hallett M. Modulation of motor cortex excitability by median nerve and digit stimulation. Exp Brain Res 1999;129:77-86.

3. Ridding MC, Brouwer B, Miles TS, Pitcher JB, Thompson PD. Changes in muscle responses to stimulation of the motor cortex induced by peripheral nerve stimulation in human subjects. Exp Brain Res 2000;131:135-43.
4. Aziz Q, Thompson DG, Ng VW, et al. Cortical processing of human somatic and visceral sensation. J Neurosci 2000;20:2657-63.
5. Lo YL, Cui SL. Acupuncture and the modulation of cortical excitability. Neuroreport 2003;14:1229-31.
6. Sonde L, Gip C, Fernaeus SE, Nilsson CG, Vitnanen M. Stimulation with low frequency (1.7 Hz) transcutaneous electric nerve stimulation (low-tens) increases motor function of the post-stroke paretic arm. Scand J Rehabil Med 1998;30:95-9.
7. Van Nes IJ, Geurts AC, Hendricks HT, Duysens J. Short-term effects of whole-body vibration on postural control in unilateral chronic stroke patients: preliminary evidence. Am J Phys Med Rehabil 2004;83:867-73.
8. Kawahira K, Higashihara K, Matsumoto S, et al. New functional vibratory stimulation device for extremities in patients with stroke. Int Rehabil Res 2004;27:335-7.
9. Weingarden H, Ring H. Functional electrical stimulation-induced neural changes and recovery after stroke. Eura Medicophys 2006; 42:87-90.
10. Lo YL, Cui SL, Fook-Chong S. The effect of acupuncture on motor cortex excitability and plasticity. Neurosci Lett 2005;384: 145-9.
11. Conforto AB, Kaelin-Lang A, Cohen LG. Increase in hand muscle strength of stroke patients after somatosensory stimulation. Ann Neurol 2002;51:122-5.
12. Peurala SH, Pitkänen K, Sivenius J, Tarkka IM. Cutaneous electrical stimulation may enhance sensorimotor recovery in chronic stroke. Clin Rehabil 2002;16:709-16.
13. Golaszewski S, Kremser C, Wagner M, Felber S, Aichner F, Dimitrijevic MM. Functional magnetic resonance imaging of the human motor cortex before and after whole-hand afferent electrical stimulation. Scand J Rehabil Med 1999;31:165-73.
14. 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.
15. Cooper JB, Jane JA, Alves WM, Cooper EB. Right median nerve electrical stimulation to hasten awakening from coma. Brain Inj 1999;13:261-7.
16. Sullivan JE, Hedman LD. A home program of sensory and neuromuscular electrical stimulation with upper-limb task practice in a patient 5 years after a stroke. Phys Ther 2004;84:1045-54.
17. Robbins SM, Houghton PE, Woodbury MG, Brown JL. The therapeutic effect of functional and transcutaneous electric stimulation on improving gait speed in stroke patients: a meta-analysis. Arch Phys Med Rehabil 2006;87:853-9.
18. DeVahl J. Neuromuscular electrical stimulation in rehabilitation. In: Gersh MR, editor. Electrotherapy in rehabilitation. Philadelphia: FA Davis; 1992. p 218-68.
19. Sawner K, Lavigne J. Brunnstrom's movement therapy in hemiplegia: a neurophysiological approach. Philadelphia: JB Lippincott; 1992.
20. 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.
21. 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.
22. 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.
23. Burridge JH, Swain ID, Taylor PN. Functional electric stimulation: a review of the literature published on common peroneal nerve stimulation for the correction of dropped foot. Rev Clin Gerontol 1998;8:155-61.
24. Dimitrijevic MM, Dobrivoje SS, Wawro AW, Wun CC. Modification of motor control of wrist extension by mesh-glove electrical afferent stimulation in stroke patients. Arch Phys Med Rehabil 1996;77:252-8.
25. Chen SC, Chen YL, Chen CJ, Lai CH, Chiang WH, Chen WL. Effects of surface electrical stimulation on the muscle-tendon junction of spastic gastrocnemius in stroke patients. Disabil Rehabil 2005;27:105-10
26. Pomeroy VM, King L, Pollock A, Baily-Hallam A, Langhorne P. Electrostimulation for promoting recovery of movement or functional ability after stroke. Cochrane Syst Rev 2006;(2):CD003241.
27. 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.
28. Kimberley TJ, Lewis SM, Auerbach EJ, Dorsey LL, Lojovich JM, Carey JR. Electrical stimulation driving functional improvements and cortical changes in subjects with stroke. Exp Brain Res 2004;154:450-60.
29. 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.
30. 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.
31. 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.

## Suppliers

a. Enraf-Nonius BV, Röntgenweg 1, PO Box 810, 2600 AV Delft, The Netherlands.
b. Vicon; Oxford Metrics, 14 Minns Estate, West Way, Oxford, OX2 OJB, UK.
c. Version 9.0; SPSS Inc, 233 S Wacker Dr, 11th Fl, Chicago, IL 60606.