ORIGINAL ARTICLE

Can Surface Neuromuscular Electrical Stimulation of the Wrist and Hand Combined With Routine Therapy Facilitate Recovery of Arm Function in Patients With Stroke?

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ABSTRACT. Rosewilliam S, Malhotra S, Roffe C, Jones P, Pandyan AD. Can surface neuromuscular electrical stimulation of the wrist and hand combined with routine therapy facilitate recovery of arm function in patients with stroke? Arch Phys Med Rehabil 2012;93:1715-21.

Objective: To investigate whether treatment with surface neuromuscular electrical stimulation to the wrist extensors improves recovery of arm function in severely disabled patients with stroke.

Design: Single blinded randomized controlled trial.

Setting: Acute stroke unit and stroke rehabilitation wards of a university hospital.

Participants: Patients with no upper limb function (Action Research Arm Test [ARAT] score 0) (N=90; mean age ± SD, 74±11y; 49% men) were recruited to the study within 6 weeks of stroke. Only 67 participants were alive at the end of the study and data from 66 of these people were analyzed.

Interventions: Participants were randomized to surface neuromuscular electrical stimulation using surface electrical stimulators for 30 minutes, twice in a working day for 6 weeks in addition to standardized upper limb therapy or just standardized upper limb therapy.

Main Outcome Measure: The primary outcome measure was the ARAT score. Assessments were made at baseline and at 6, 12, 24, and 36 weeks after recruitment.

Results: There were statistically significant improvements in measures of wrist extensor (mean difference 0.5; 95% confidence interval [CI], 0.0–1.0) and grip strength (mean difference 0.9; 95% CI, 0.1–1.7) over the treatment period. Arm function (ARAT score) was not significantly different between the groups over the treatment period at 6 weeks (mean difference 1.9; 95% CI, −2.9 to 6.8) or over the study period at 36 weeks (mean difference 6.4; 95% CI, −1.8 to 14.7), and the rate of recovery was not significantly different (mean difference 0.7; 95% CI, −0.2 to 1.6).

Conclusions: In patients with severe stroke, with no functional arm movement, electrical stimulation of wrist extensors improves muscle strength for wrist extension and grip, and larger studies are required to study its influence on arm function.

Key Words: Arm; Electrical stimulation; Rehabilitation; Stroke.

MOST PATIENTS who survive a stroke will regain the ability to walk independently, but only less than 50% will recover arm function.[1,2] Recovery of arm function after a stroke follows a predictable pattern, yet the time course of recovery is variable.[3] Delays in the recovery of function increase the risk of secondary complications such as spasticity, contractures, and pain,[3] and these affect normal movement and further interfere with rehabilitation.[3,4]

There is a growing body of evidence to suggest that some adjunct therapies such as neuromuscular electrical stimulation (NMES), biofeedback, and constraint-induced therapy have the potential to either facilitate recovery of arm function or prevent the formation of secondary complications.[5-10] Among these, NMES has been the most widely researched.[6,8] In spite of encouraging results from randomized controlled trials,[6,9] there is still no definitive evidence to support the use of NMES as routine adjunct treatment.[5]

It was previously shown that treatment with surface NMES (sNMES) of the wrist extensors for 8 weeks leads to a transient improvement in arm function, not maintained 24 weeks after the cessation of treatment.[9] However, secondary analysis of the data from this randomized controlled trial suggests that results may have been confounded by heterogeneity in the study population at recruitment. Subgroup analyses showed that patients with no upper limb function at recruitment had a greater chance of regaining arm function when treated with sNMES, and these benefits were maintained until the end of the study, 24 weeks after the discontinuation of sNMES.[4,11]

This phase II study was set up to investigate whether treatment with sNMES to the wrist extensors in combination with

List of Abbreviations

ARAT	Action Research Arm Test
BI	Barthel Index
NMES	neuromuscular electrical stimulation
sNMES	surface neuromuscular electrical stimulation

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standardized rehabilitation therapy improves the recovery of arm function in people with poor prognostic indicators of arm function.

METHODS

This single blind randomized controlled trial with an independent assessor was carried out at a university hospital between 2004 and 2008.

All adult patients with a first stroke who had no arm function (defined as a score of 0 in the Grasp subsection of the Action Research Arm Test [ARAT])[11] within 6 weeks of onset and who had no contraindications to sNMES were considered for trial inclusion. Participants were excluded if they were medically unstable, if they had a history of osteoarthritis, rheumatoid arthritis, or soft-tissue injuries resulting in contractures or a reduced range of movement in the wrist and fingers, and if informed consent or relatives' assent could not be obtained.

The study was approved by the North Staffordshire Local Research Ethics Committee (Ref no. 04/Q2604/1) and conducted to the principles of Good Clinical Research Practice.

Participants were randomized into 2 groups, that is, a control group and a treatment group, using a method of concealed random allocation (a pseudo random computed sequence in blocks was generated, and the codes were stored by an independent person not involved in recruitment or measurement). Baseline measures were taken by an independent assessor who was blinded to the allocation of participants. Participants in both groups were given a defined module of upper limb physiotherapy that reflected current local clinical practice[12] for a period of 6 weeks in addition to the routine treatment on the stroke unit.

Intervention

Participants in the treatment group were treated with sNMES for 6 weeks. sNMES was delivered by Conformite Europenne (CE)-marked electrical stimulators developed by Odstock Medical Limited[b] customized for the study. Patients received 30-minute sessions of sNMES to the wrist and finger extensors at least twice a day for 5 days a week. Treatment was delivered by surface electrodes positioned on the dorsal surface of the forearm (inactive electrode placed slightly inferior to the common extensor origin below the elbow and the active electrode posterior and few inches above the wrist).[10] The stimulation parameters required to produce slow movement through the full range at maximum patient comfort were as follows: Pulse width=300 microseconds; ON time=15 seconds; OFF time=15 seconds.[13] The ON time included a ramp-up time of 6 seconds and a ramp-down time of 6 seconds[13] to provide smooth movements. The frequency of stimulation was set to 40Hz.[9,13] The intensity of stimulation was adjusted to obtain maximum possible range of wrist and finger extension with an intensity that was tolerated by the patient and without inducing fatigue. On completing the initial treatment session, the patient or his or her carer (relative) was trained on using the sNMES system and delivering treatment. Treatment compliance in both groups was monitored using a patient record.

Assessments were done at baseline, at the end of the treatment period (6 weeks), and at 3, 6, and 9 months after stopping treatment. The primary outcome measure was recovery of arm function (ARAT score).[14] Secondary outcomes were independence in activities of daily living assessed by the Barthel Index (BI),[15] active range of movement in wrist flexion and extension, wrist flexor and extensor strength, and grip strength. Basic demographic data and details of the stroke were taken from the case notes.

Sample Size Estimation

A sample size of 72 participants (36 in each arm) is required to reject the null hypothesis, that is, treatment with sNMES will not facilitate recovery of arm function, with 80% power and a 2-tailed significance level of 5%. For sample size estimation, return of useful arm function was defined as a 9-point improvement in the ARAT score; SD was 8 and 17 in the control and treatment arms, respectively.[4] Allowing for attrition, 45 patients were recruited in each arm. However, at the end of this study, a full data set was obtained only in 66 participants (31 in the treatment arm and 35 in the control arm). Poststudy power calculation was done to examine the internal validity of the study, and it yielded a power of 75%.

Statistical Analysis

The data collected were analyzed using SPSS[a] version 15. Missing values were imputed in 2 ways; when an intermediate assessment was missed, the mean of the 2 adjacent values was used, and when someone dropped out of the study, the last value was carried over. We adopted a conservative approach, carrying forward last value/means for middle missing values, even though it tends to suppress the slope because other methods (eg, regression analysis for predicting the missing values) were found to overestimate the level of recovery (eg, giving scores higher than maximum possible in ARAT). One patient improved between consent and baseline assessment and no longer met the inclusion criteria, and he was therefore excluded from the inferential analysis.

The analyses included the following:

1. The differences between the groups in ARAT scores (primary outcome) and other secondary outcome measures over the study period for participants who were alive till the end of the study (study completers) using the independent sample *t* test. The results from the study completers only has been reported and discussed within the main text because there were significant baseline differences in age and functional ability (BI) between them and those who died. However, intention-to-treat analysis for all including those who died (n=89) is reported in supplemental table 1 (available online only at http://www.archives-pmr.org/). The mean differences have been reported to show the effectiveness of the treatment.
2. The rate of recovery of outcome measures[16,17] for each individual for the treatment period (0–6wk), the follow-up period (12–36wk), and the entire study period (0–36wk). This was done to assess whether there is any corresponding improvement in the recovery rate with treatment.

No corrections were made for multiple testing despite the possibility of alpha inflation, because this was considered to add to the limitations in the study power.

Statistical test results for the description and analysis of baseline differences in data are given in the text and tables. The data have been rounded up and reported to a single decimal point because these outcomes cannot be clinically measured to an accuracy of 2 decimal points.

RESULTS

Of the 90 participants recruited, 23 patients died during the course of the study because of study-unrelated causes such as respiratory infections, recurrent stroke, and cardiac arrest (recalculated power is reported in the sample size estimation section). This resulted in the study having 5 patients fewer than originally calculated. The data from 1 participant who

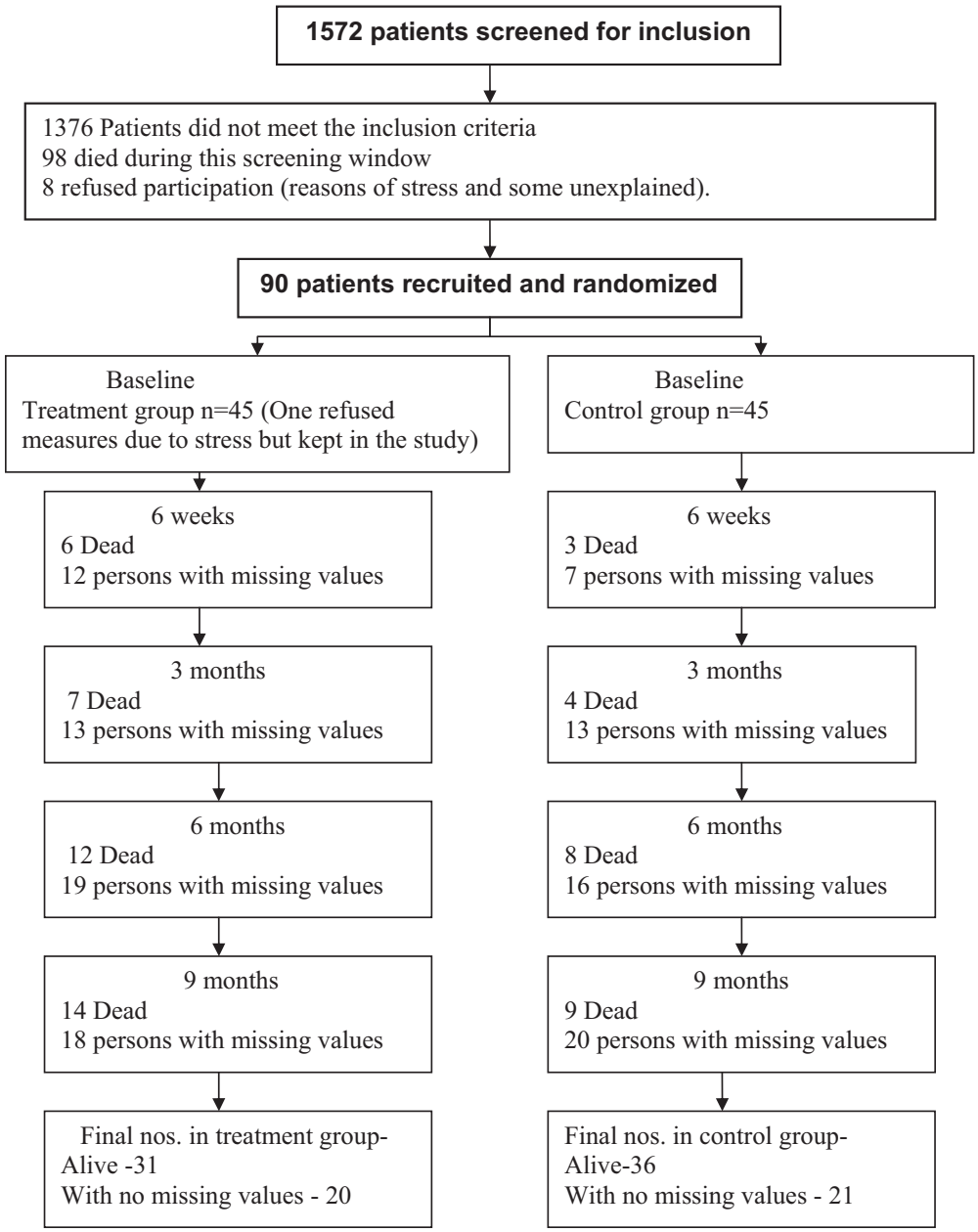

Fig 1. Flow of participants in the study.

refused baseline measurements (because of stress) after randomization were still included in the analysis. The reasons for loss of follow-up data were inability to contact participants, participants moving away from the accessible geographical area, or refusal of repeat measurements. The Consolidated Standards of Reporting Trials flowchart (fig 1) for the study gives further detail of the progression of participant numbers.

The descriptive demographic data, baseline characteristics of all 90 participants, of those who were alive till the end of the study (n=67—treatment and control group differences), and of those who died during the course of the study (n=23) are shown in table 1. Patients who died within the first 9 months were older (P=.003) and more disabled (P=.004). Because there were potentially confounding differences at the baseline between those who survived and those who died, the results and the discussion will focus on those who were alive at the end of the study.

Primary Outcome Measures

The ARAT measure was not significantly different between the groups over the treatment period at 6 weeks or over the study period at 36 weeks (P=0.4 and 0.1, respectively). Although ARAT scores improved more in the treatment group than in the control group over the treatment period and over the whole study period, this was not significant (table 2). The difference in the rate of recovery was not statistically significant between the groups during the treatment phase (P=0.1) and over the entire study period (P= 0.2). The rate of recovery in the treatment group was higher than in the control group, during the treatment phase and over the entire study period

Table 1: Baseline Characteristics of Participants

Characteristics and Outcome Measures	The Entire Sample (N_{total}=90)	Participants Who Died Before the End of the Study (n_{dead}=23)	Participants Who Were Alive at the End of the Study (n_{alive}=67)	
			Treatment	Control
Sex: men, n (%)	44 (49)	13 (57)	15 (48)	16 (52)
Total anterior circulation syndrome, n (%)	61 (68)	17 (74)	19 (62)	25 (69)
Partial anterior circulation syndrome, n (%)	19 (21)	5 (22)	5 (16)	9 (25)
Lacunar syndrome, n (%)	9 (10)	1 (4)	6 (19)	2 (6)
Posterior circulation syndrome, n (%)	1 (1)	0	1 (3)	0 (0)
Hemiparesis, n (% right)	46 (51)	13 (57)	17 (25)	16 (23)
Age (y)	74.6 (11.0)	80.4* (9.3)	72.4 (12.1)	72.7 (9.9)
ARAT	0.2 (2.3)	0.0 (0.0)	0.0 (0.0)	0.6 (3.5)
BI	2.8 (3.3)	1.1 (1.4)*	4.4†(3.9)	2.5 (2.9)
AROM-Wrist maximum flexion (deg)	3.1 (13.8)	−1.5 (13.8)	5.9 (13.1)	3.5 (14.2)
AROM-Wrist maximum extension (deg)	0.8 (8.2)	0.3 (1.5)	2.2 (12.3)	0.0 (6.1)
Isometric muscle strength wrist flexors (N)	0.1 (0.5)	0.0 (0.1)	0.3 (0.8)	0.06 (0.2)
Isometric muscle strength wrist extensors (N)	0.1 (0.4)	0.0 (0.0)	0.2 (0.6)	0.06 (0.2)
Grip strength (N)	0.1 (1.0)	0.0 (0.0)	0.47 (1.7)	0.0 (0.0)

NOTE. Values with asterisk indicate that *t* test showed significant difference for these variables at baseline. Values are n, %, or mean ± SD or as otherwise noted.
Abbreviation: AROM, active range of movement.
*$P \leq .05$ *t* test for comparison between alive and dead.
†$P = .03$ *t* test for comparison between treatment and control group.

(table 3). Patients in the treatment group were more likely to recover (clinically important ARAT score ≥6) than were those in control group (odds ratio 2.3; 95% confidence interval 0.7–7.2), but this was not statistically significant.

Secondary Outcome Measures

Results for secondary outcome measures are shown in table 2. The BI improved in both groups during the treatment and the follow-up periods, but there was no difference in the level of improvement between the treatment groups. Active range of movement at the wrist (flexion and extension) improved more in the treatment group than in the control group, but the difference in improvement was not statistically significant at any point ($P>0.2$). Wrist extension strength and grip improved significantly in the treatment group over the study period ($P=.04$ and .03, respectively). Although the treatment group remained stronger at the end of the study, the difference was not statistically significant ($P=0.2$ and 0.1 for wrist extension strength and grip strength, respectively).

The rate of improvement was 3 times faster for active range of movement in extension ($P=.04$) and 6 times faster for wrist strength in extension ($P<.01$) in the treatment group than in controls during the treatment period (see table 3). There were no significant differences in the rate of recovery for any of the other secondary outcomes at any other time point.

DISCUSSION

The main findings of this study are that surface neuromuscular stimulation of forearm muscles significantly improves wrist extension strength and grip strength in patients with stroke who had no active movement at the start of treatment. We also found nonsignificant improvements in complex functional arm movements (ARAT). Wider activities of daily living (BI) did not improve. The effect of treatment ceased after the discontinuation of the intervention. This could be due to reduced focus on upper limb therapy in routine stroke care[18] and loss of translation of this effect of treatment into activities of function in daily life. Significant improvement of direct measures of function (muscle strength, grip strength) suggests that the treatment is effective at reducing impairment. It is likely that a larger study, with routine therapy as control, would have shown significant improvements in complex arm function.

Normally, the focus of routine therapy in patients with severe levels of disability (as recruited for this trial) will focus on retraining trunk control and mobility. However, in this study, patients in both groups, that is, the control group and the treatment group, received 30 minutes of physiotherapy focused on the rehabilitation of the upper limb. This additional therapy may have led to a greater than normal level of improvement in the control group. As a result, the differences between the groups would not have been significant. There is some evidence from the literature,[9,19] and data from our secondary analysis (mean improvements in the control group were 2.0 ARAT units, SD 8.0) that support the argument that the improvements in the control group of this study were better than in patients who get an undefined form of routine treatment. More work will be required to test the hypothesis that a daily structured program of upper limb rehabilitation in acute stroke will lead to improvements in hand function of a severely disabled stroke population.

The duration of treatment followed in this study was limited to 6 weeks. The evidence was that the rate of recovery in the relevant impairments and the recovery of function were highest during the period when active treatment was applied. However, as soon as this therapy was discontinued, the rate of recovery between groups almost equalized. It is possible that in such a severely disabled group of patients, the duration of treatment may need to be longer than that followed in this trial. While it is not possible to comment on how long the duration would normally be required, it can be hypothesized that any treatment should be continued until the patient achieves a threshold of function that can be built on by the patient and the therapist. Again, here is a need for much more work to elucidate the minimum duration of treatment. It is possible that the high attrition rate (nearly 30%) and the resultant reduction in the sample size may have also contributed in part to the lack of significance.

Table 2: Mean ± SD and *t* Test Results for Participants Who Were Alive Over the Weeks With Intention to Treat

Outcome	Week 6					Week 12					Week 24					Week 36				
	T (n=39)	C (n=41)	MD	95% CI	*P*	T (n=38)	C (n=40)	MD	95% CI	*P*	T (n=33)	C (n=36)	MD	95% CI	*P*	T (n=31)	C (n=35)	MD	95% CI	*P*
ARAT total score	5.0±11.7	3.1±10.2	1.9	−2.9 to 6.8	0.4	7.7±14.6	3.3±12.6	4.3	−1.8 to 10.5	0.2	10.1±17.1	4.8±13.9	5.4	−2.1 to 12.8	0.2	11.6±18.9	5.2±14.3	6.4	−1.8 to 14.7	0.1
BI total	5.4±4.0	5.8±5.2	−0.4	−2.5 to 1.7	0.7	7.3±5.3	6.9±5.7	0.4	−2.1 to 2.9	0.8	9.4±5.9	8.1±6.7	1.3	−1.7 to 4.3	0.4	10.5±5.8	8.9±7.1	1.5	−1.7 to 4.7	0.4
Wrist flexion AROM (deg)	10.1±16.8	8.2±13.4	1.9	−4.8 to 8.7	0.6	9.8±15.1	8.9±11.9	0.9	−5.1 to 7.1	0.8	17.0±21.3	16.6±20.9	0.4	−9.8 to 10.6	0.9	15.7±18.9	13.6±19.3	2.2	−7.3 to 11.6	0.7
Wrist extension AROM (deg)	10.2±16.2	6.3±10.3	3.9	−2.1 to 9.9	0.2	8.7±15.5	5.8±12.8	2.9	−3.5 to 9.4	0.4	10.2±18.9	7.5±19.1	2.7	−6.4 to 11.8	0.6	16.3±22.8	10.6±19.0	5.7	−4.6 to 15.9	0.3
Isometric muscle strength wrist flexors (N)	1.0±2.6	0.4±0.9	0.6	−0.3 to 1.5	0.2	1.1±2.2	0.6±1.1	0.5	−0.2 to 1.3	0.2	1.7±2.7	1.1±1.6	0.6	−0.5 to 1.7	0.3	1.4±1.9	1.3±1.7	0.2	−0.7 to 1.1	0.7
Isometric muscle strength wrist extensors (N)	0.7±1.5	0.2±0.5	0.5	0.0 to 1.0	0.04	0.9±1.7	0.5±1.0	0.5	−0.2 to 1.1	0.2	1.2±1.9	0.7±1.1	0.6	−0.2 to 1.2	0.12	1.4±1.9	0.9±1.4	0.5	−0.3 to 1.4	0.2
Grip strength (N)	1.0±2.5	0.2±0.7	0.9	0.1 to 1.7	0.03	1.5±3.2	0.7±2.5	0.7	−0.5 to 2.1	0.2	2.2±3.9	1.5±3.7	0.8	−1.0 to 2.7	0.4	3.2±5.3	1.4±3.2	1.7	−0.4 to 3.9	0.1

Abbreviations: AROM, active range of movement; C, control group; CI, confidence interval; MD, mean difference at each point of measurement; T, treatment group.

Table 3: Mean ± SD and *t* Test for Difference Between the Groups in Rate of Recovery, for the Participants Who Were Alive With Intention to Treat

Outcome T (n=31) C (n=35)	Week 0–6					Week 12–36					Week 0–36				
	T	C	MD	95% CI	*P*	T	C	MD	95% CI	*P*	T	C	MD	95% CI	*P*
ARAT total score	1.1±2.1	0.4±1.6	0.7	−0.2 to 1.6	0.1	0.1±0.5	0.1±0.2	0.04	−0.1 to 0.2	0.7	0.3±0.5	0.1±0.4	0.2	−0.1 to 0.4	0.2
BI total	0.3±0.5	0.6±0.7	−0.2	−0.5 to 0.1	0.1	0.1±0.1	0.1±0.1	0.1	−0.1 to 0.1	0.8	0.2±0.1	0.2±0.2	0.01	−0.1 to 0.1	0.9
Wrist flexion AROM (deg)	1.2±2.5	0.7±2.8	0.5	−0.8 to 1.8	0.5	0.2±0.6	0.2±0.6	−0.01	−0.3 to 0.28	0.9	0.3±0.4	0.3±0.5	−0.1	−0.3 to 0.2	0.6
Wrist extension AROM (deg)	1.7±2.9	0.6±1.2	1.1	0.03 to 2.2	0.04	0.2±0.6	0.2±0.6	0.04	−0.3 to 0.3	0.8	0.3±0.5	0.2±0.5	0.04	−0.2 to 0.3	0.8
Isometric muscle strength wrist flexors (N)	0.2±0.4	0.1±0.2	0.1	−0.03 to 0.3	0.1	0.002±0.1	0.02±0.05	−0.02	−0.05 to 0.01	0.2	0.03±0.05	0.03±0.04	−0.01	−0.03 to 0.02	0.6
Isometric muscle strength wrist extensors (N)	0.1±0.2	0.02±0.1	0.11	0.03 to 0.2	0.0	0.01±0.04	0.01±0.03	−0.003	−0.02 to 0.02	0.7	0.03±0.05	0.02±0.05	0.01	−0.01 to 0.03	0.5
Grip strength (N)	0.1±0.4	0.02±0.1	0.12	−0.02 to 0.3	0.1	0.06±0.1	0.03±0.1	0.03	−0.02 to 0.1	0.2	0.06±0.1	0.1±0.1	0.0	−0.1 to 0.1	0.9

Abbreviations: AROM, active range of movement; C, control group; CI, confidence interval; MD, mean difference at each point of measurement; T, treatment group.

It is not clear whether these improvements that we have observed in this study are associated with systemic effects of electrical stimulation, in particular effects associated with increased cortical excitability and the resultant neural plasticity[20] and/or effects on muscle physiology,[21] or the additional therapy time in the treatment group. There is some suggestion that the effects in this study could have been attributed to the effects of sNMES on muscle physiology because there is clear evidence that patients in the treatment group had improved extensor strength following treatment when compared to the control group patients. Improved extensor strength can lead to more efficient gripping, which is essential for activities of daily living.[22] It is also possible that the systemic effects associated with increased cortical excitability may have improved the long-term functional outcome in some of the patients, and this could explain the continued long-term functional benefits seen in the treatment group.[23] More research is required to tease out the time effects from the systemic effects of treatment.

This study has demonstrated that for a homogenous group of severely disabled patients small but meaningful improvement is possible. The improvement reported in this study is likely to be clinically relevant because it has included a full data set of patients who were alive at 9 months after recruitment to the study and included patients who had both left and right hemispheric strokes. In this regard, this is probably one of the largest, clinically relevant, studies that has been conducted exploring the effects of an acute upper limb rehabilitation protocol in severely disabled patients with stroke.

Study Limitations

A significant limitation in this study protocol was that the electrical stimulation was limited to a cyclical movement of one single limb segment (the wrist). There can be criticism that the movement used in this study may not be functionally relevant and could explain the small effect size. There is some pilot evidence in the published literature that simultaneous stimulation of multiple limb segments may have a bigger treatment effect.[24] Again, more work will be needed to elucidate the relative merits of multiple channel stimulation.

CONCLUSIONS

In patients with severe stroke and no functional arm movement, electrical stimulation of the wrist extensors improves extensor muscle strength and grip strength, but there were no significant improvements in terms of improvements in the range of movement. There is some evidence that this treatment facilitated recovery of arm function. It is not clear as to whether this functional improvement was a direct result of plasticity or was secondary to strength gains. The functional improvement, although clinically important, did not reach levels of statistical significance. There are 3 potential reasons for not achieving statistical significance: (1) the sample size was inadequate, (2) the treatment duration was inadequate, and (3) the control group received additional treatment lasting between 30 minutes a day (this is not equivalent to conventional therapy). To address the first point, a larger sample study will need to be carried out. To address the latter 2 points, more fundamental work is needed to identify the optimal duration of treatment and also the interaction effects between treatment with electrical stimulation and other potential concomitant therapies.

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Suppliers

a. SPSS Inc, 233 S Wacker Dr, 11th Fl, Chicago, IL 60606.
b. Odstock Medical Limited, The National Clinical FES Centre, Salisbury District Hospital, Salisbury, Wiltshire, SP2 8BJ, United Kingdom.

Supplemental Table 1: Mean ± SD, *t* Test Results for All Participants With Intention to Treat (Excluding Data From Participant Who Did Not Meet Inclusion Criteria)

Outcome T (n=45) C (n=44)	Week 6					Week 12					Week 24					Week 36				
	T	C	MD	95% CI	*P*	T	C	MD	95% CI	*P*	T	C	MD	95% CI	*P*	T	C	MD	95% CI	*P*
ARAT total score	4.3±10.9	2.9±9.9	1.5	−2.9 to 5.9	0.5	6.5±13.6	3.8±12.8	2.7	−2.9 to 8.3	0.3	7.4±15.2	4.6±13.4	2.8	−3.2 to 8.9	0.4	8.0±16.6	4.8±13.6	3.2	−3.2 to 9.6	0.3
BI total	4.7±4.1	5.4±5.2	−0.7	−2.6 to 1.3	0.5	6.3±5.4	6.5±5.8	−0.2	−2.6 to 2.1	0.8	7.3±6.2	7.4±6.6	−0.1	−2.8 to 2.6	0.9	7.6±6.5	7.9±7.0	−0.4	−3.2 to 2.5	0.8
Wrist flexion AROM (deg)	8.8±16.0	7.6±13.1	1.2	−5.0 to 7.4	0.7	8.9±14.6	8.3±11.5	0.6	−4.9 to 6.1	0.8	13.1±19.7	14.4±19.8	−1.3	−9.6 to 7.1	0.8	11.5±17.4	11.5±17.9	−0.1	−7.5 to 7.4	0.9
Wrist extension AROM (deg)	8.8±15.4	5.8±10.0	2.9	−2.5 to 8.5	0.3	7.7±14.6	5.9±12.8	1.8	−4.0 to 7.5	0.6	7.8±16.7	7.3±17.9	0.4	−6.9 to 7.7	0.9	11.5±20.2	9.6±17.8	1.9	−6.2 to 9.9	0.6
Isometric muscle strength wrist flexors (N)	0.9±2.4	0.4±0.9	0.5	−0.3 to 1.3	0.2	0.9±2.0	0.6±1.1	0.4	−0.3 to 1.1	0.3	1.3±2.4	0.9±1.5	0.3	−0.5 to 1.2	0.4	0.9±1.8	1.0±1.5	−0.0	−0.7 to 0.7	0.9
Isometric muscle strength wrist extensors (N)	0.6±1.5	0.2±0.5	0.5	−0.01 to 0.9	0.1	0.8±1.6	0.4±0.9	0.4	−0.2 to 0.9	0.2	0.9±1.8	0.6±1.0	0.4	−0.3 to 0.9	0.2	0.9±1.8	0.7±1.3	0.3	−0.4 to 0.9	0.4
Grip strength (N)	0.9±2.3	0.2±0.7	0.8	0.0 to 1.5	0.04	1.3±2.9	0.6±2.4	0.7	−0.5 to 1.8	0.2	1.7±3.5	1.1±3.4	0.6	−0.9 to 1.9	0.5	2.3±4.6	1.1±2.9	1.1	−0.5 to 2.7	0.2

Abbreviations: AROM, active range of movement; C, control group; CI, confidence interval; MD, mean difference at each point of measurement; T, treatment group.