Background and Purpose—It has been suggested that cyclic neuromuscular electrical stimulation (ES) may enhance motor recovery after stroke. We have investigated the effects of ES of the wrist extensors on impairment of wrist function and on upper-limb disability in patients being rehabilitated after acute stroke.

Methods—We recruited 60 hemiparetic patients (mean age, 68 years) 2 to 4 weeks after stroke into a randomized, controlled, parallel-group study comparing standard rehabilitation treatment with standard treatment plus ES of wrist extensors (3 times 30 minutes daily for 8 weeks). Isometric strength of wrist extensors was measured using a device built for that purpose. Upper-limb disability was assessed with use of the Action Research Arm Test (ARAT). Observations were continued for 32 weeks (24 weeks after the finish of ES or the control intervention phase).

Results—The change in isometric strength of wrist extensors (at an angle of 0° extension) was significantly greater in the ES group than the control group at both 8 and 32 weeks (P=0.004, P=0.014 by Mann Whitney U test). At week 8 the grasp and grip subscores of the ARAT increased significantly in the ES group compared with that in the control group (P=0.013 and P=0.02, respectively); a similar trend was seen for the total ARAT score (P=0.11). In the subgroup of 33 patients with some residual wrist extensor strength at study entry (moment at 0° extension >0), the ARAT total score had increased at week 8 by a mean of 21.1 (SD, 12.7) in the ES group compared with 10.3 (SD, 9.0) in the control group (P=0.024, Mann Whitney U test); however, at 32 weeks the differences between these 2 subgroups were no longer statistically significant.

Conclusions—ES of the wrist extensors enhances the recovery of isometric wrist extensor strength in hemiparetic stroke patients. Upper-limb disability was reduced after 8 weeks of ES therapy, with benefits most apparent in those with some residual motor function at the wrist. However, it is not clear how long the improvements in upper-limb disability are maintained after ES is discontinued. (Stroke. 1999;30:1384-1389.)

Key Words: arm ■ electric stimulation ■ hand strength ■ randomized controlled trials ■ rehabilitation ■ stroke

Approximately half of all stroke survivors are left with major functional problems in their hand and arm. Therapists in the United Kingdom mainly use either the Bobath or Movement Sciences approach to encourage recovery of upper-limb function. Adjunctive methods include splinting and plastering, feedback therapies, and the use of restraint as a method of forcing the use of the affected arm. The best methods of treatment to encourage maximal recovery of the upper limb after stroke remain uncertain. If a low-cost treatment were found that reduced functional impairment and disability, this could benefit many stroke patients.

It has been proposed that cyclic electrical neuromuscular stimulation enhances motor recovery after stroke, with claims that it can reduce spasticity, strengthen muscles, and increase the range of movement of joints with prevention or correction of contractures. However, many of the studies of ES at the wrist are difficult to interpret because of the small numbers of patients, variable timing of the intervention in relation to the stroke, and lack of longer-term follow-up. Late after stroke, ES at the wrist in combination with other rehabilitation strategies can result in increased grip strength and improved motor function; however, it is not clear whether the ES component of therapy contributes to these beneficial effects. The effects of applying ES at the wrist during the recovery phase after stroke are also uncertain. In a recent randomized controlled study, a significant improvement in motor function after ES was found; however, a high proportion of the patients (39%) dropped out of this study, raising questions about the validity of the results. The aims of our study were to determine whether ES at the wrist, administered during the recovery phase after hemiplegic stroke, reduces neuromuscular impairment and upper-limb disability, and to determine whether any improvements are maintained after ES is discontinued.

Received November 12, 1998; final revision received January 29, 1999; accepted March 12, 1999.

From the Department of Bioengineering, University of Strathclyde (A.D.P., M.G.), and Academic Section of Geriatric Medicine, University of Glasgow (J.P., M.C., D.J.S.), Glasgow, Scotland.

Correspondence to Prof David J Stott, Academic Section of Geriatric Medicine, 3rd Floor Centre Block, Glasgow Royal Infirmary, Glasgow G4 0SF, Scotland. E-mail d.j.stott@clinmed.gla.ac.uk

© 1999 American Heart Association, Inc.

Stroke is available at http://www.strokeaha.org
Subjects and Methods
We studied 60 hemiplegic stroke patients in a randomized, controlled, parallel-group study comparing standard treatment with standard treatment plus ES. Randomization was by computer-generated random numbers held in sealed envelopes by an individual not involved with the study. Study entrants were randomized after their baseline characteristics had been determined and logged into a computer database.

Consecutive stroke patients admitted to Glasgow Royal Infirmary were assessed for their suitability for the study. Inclusion criteria were hemiparesis due to acute stroke with Medical Research Council power of wrist extension grade 4/5 or worse at 2 to 4 weeks after stroke onset, and no previous wrist problem. Patients were excluded if they had a previous wrist problem (including previous hemiplegia or arthritis) or were unable to understand the study (due to impaired consciousness level, dysphasia, or cognitive impairment). Written informed consent was obtained from the patient or a close relative. The study was approved by the local hospital ethics committee.

All patients had a CT brain scan. The stroke type was categorized as intracerebral hemorrhage or infarction, and also by the Oxfordshire Community Stroke Project classification system (total anterior cerebral syndrome, partial anterior cerebral syndrome, lacunar syndrome, and posterior cerebral syndrome).1 Premorbid Rankin scale12 and Barthel Index13 scores were determined from interview of the patient or a reliable witness.

The treatment group received 3 half-hour periods of ES daily (a total of 90 minutes per day) for 8 weeks. Wrist and finger extensors (extensor carpi radialis longus and brevis, extensor carpi ulnaris, and extensor digitorum communis) were stimulated with a pair of self-adhesive electrodes. One electrode was placed proximally over the forearm below the elbow, and the other was placed distally on the forearm (positioned for optimally balanced joint movement). ES had the forearm below the elbow, and the other was placed distally on the forearm. ES was generated random numbers held in sealed envelopes by an individual

Assessment of Upper Limb Disability
(1) The total score in the Action Research Arm Test (ARAT), a validated and reproducible measure of upper limb function,16 was used as the primary measure of upper-limb disability in the hemiparetic arm. The maximal score is 57; the higher the score the less the upper-limb disability. All components of the ARAT were performed, and the subscores were recorded as the best performance for each part (grip, pinch, and gross movement). The total score is a summation of the grasp, grip, pinch, and gross movement subscales.

(2) Grip strength was measured as the best of 3 measurements with a Jamar hand dynamometer (Smith and Nephew). A purpose-built device was developed to measure the isometric strength of wrist extension and the active and passive ranges of motion at the wrist.9 This device consisted of a fixed base, clamped to a specially designed table, and a moveable arm with a handle at its free end. A potentiometer was coupled to the axis of the moveable arm (to measure angular displacement), and the handle was strain-gauged (to measure passively applied and actively generated moments). The table was designed so that the measurement device could quickly and easily be moved into the correct position around the patient, who was supported in an upright sitting position on a wide height-adjustable plinth. For all measurements at the wrist the forearm was midpronated to eliminate the effect of gravity on wrist extension and clamped to the apparatus. A mark on the apparatus was used to standardize the position of the ulnar head. The elbow was held at 90° of flexion and the shoulder in approximately 30° of abduction. The strain-gauge handle and electrotomometer were connected via an amplifier to a 12-bit A/D converter in an IBM-compatible PC. A computer program, developed in Turbo Pascal (Borland International) was used to collect data from the strain-gauge arm and the goniometer. Sampling frequency was set at 50 Hz. The program displayed angular velocity in the form of a moving horizontal bar to enable the assessor to passively move the wrist at a consistent angular velocity. Data were not displayed during the assessment but were stored in a coded file for later analysis.

(1) The resting angle of the wrist was measured 4 times (if the posture of the wrist was in flexion, the wrist was passively extended to an angle of 0° and then allowed to return to the resting position before the resting posture was remeasured). Results are given for the first and last measurements.

(2) Passive range of movement at the wrist was recorded by the assessor extending the subject’s wrist from a fully flexed position until the point of “end feel,” aided by 2-finger pressure.

(3) Active range of movement was recorded by asking the subjects to maximally extend their wrist. The angle of maximal wrist extension was taken for analysis (best of 3 measurements for both passive and active range of movement).

(4) Measurement of isometric strength of wrist extensors was made with the wrist held in neutral (0°), 15°, and 30° of extension. Three measurements were taken at each wrist angle; the maximum result at each angle was used in the analyses.

(5) Muscle tone during passive extension of the wrist was assessed using the modified Ashworth scale, which evaluates global resistance to imposed movement (scale range, 0 to 5; increased scores indicate increased resistance, which can be due to spasticity, spastic dystonia, and passive tissue resistance caused by muscle shortening or joint contractions).14

Other Measurements
Local discomfort at the wrist at rest and on passive extension was assessed with a 6-point rating scale and a 10-cm visual analog scale. Visual-spatial neglect was assessed with use of the star cancellation test.16 The Rankin12 and Barthel13 scores were recorded as measures of global handicap and disability.
Statistical Analysis

At the time this study was designed, very limited data were available to perform power calculations to determine the optimal sample size. A previous study claimed that ES improves passive range of movement at the wrist by approximately 7 ± 5 (mean ± SEM) degrees.17 It was calculated that 25 patients per group were required for 80% power (at P < 0.05) to detect a difference between ES and the control group of 4° range of movement at the wrist (assuming a standardized difference of 0.8). Therefore, the study size was 30 patients per group, allowing for several dropouts. The primary end points were changes in upper-limb impairment and disability from baseline to the end of treatment (weeks 0 to 8) and to the end of follow-up (weeks 0 to 32) in the ES group compared with the control group. Statistical analysis was by unpaired Student t test for normally distributed data and Mann-Whitney U test for nonparametric data. Results were accepted as statistically significant at P ≤ 0.05.

Results

In total, 621 hospital admissions with clinical acute stroke were assessed for their suitability for the study. Sixty patients were suitable and agreed to take part; of them, 48 (80%) completed the study. The reasons that patients were deemed unsuitable for the study and reasons for study dropouts are shown in Figure 1. Of the 12 patients who dropped out of the study, 7 were in the control group and 5 in the ES group. Fourteen patients in the ES group and 14 controls remained in the hospital throughout the intervention phase of the study, 7 were in the control group and 5 in the ES group. The components of the ARAT score that showed improvement after ES (change from baseline to week 8) were the grasp and grip subscores (P = 0.013 and P = 0.02, respectively, compared with changes in controls) (Table 3). These differences were no longer statistically significant at 32 weeks. There were no significant improvements in the pinch or gross movement subscores after ES compared with those in the control group. There was a tendency for an increase from baseline to week 8 in total ARAT score in the treatment group compared with that in the control group (P = 0.11; Table 3). There remained a tendency for total ARAT score to be improved in the ES group at the end of follow-up; however, this difference again was not statistically significant. In the subgroup of 33 patients (15 ES, 18 control) with measurable residual wrist extensor strength at study entry (moment at 0° extension > 0) who were followed up at 8 weeks, the ARAT total score had increased by a mean of 21.1 (SD, 12.7) in the ES group compared with 10.3 (SD, 9.0)

Table 1. Baseline Characteristics of the Study Groups

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Treatment Group</th>
<th>Control Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male:female ratio</td>
<td>14:16</td>
<td>14:16</td>
</tr>
<tr>
<td>Age, y</td>
<td>69.0 (10.8)</td>
<td>66.4 (12.2)</td>
</tr>
<tr>
<td>Prestroke Barthel Index score, median (range)</td>
<td>23.9 (7.7)</td>
<td>22.9 (5.5)</td>
</tr>
<tr>
<td>Time after stroke to study entry, d</td>
<td>9 (6–10)</td>
<td>9 (6–10)</td>
</tr>
<tr>
<td>AMT score, median (range)</td>
<td>(n=24)</td>
<td>(n=23)</td>
</tr>
<tr>
<td>Left:right hemiparesis</td>
<td>18:12</td>
<td>20:10</td>
</tr>
<tr>
<td>Oxfordshire Community Stroke Project classification</td>
<td>PACI</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>TACI</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>LACI</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>ICH</td>
<td>4</td>
</tr>
</tbody>
</table>

Results are mean (SD) unless otherwise stated. PACI indicates partial anterior cerebral infarct; TACI, total anterior cerebral infarct; LACI, lacunar infarct; and ICH, intracranial haemorrhage.

In terms of age, sex, neurological impairment, and other clinical characteristics, with no significant baseline differences between the groups in any of the variables presented in Tables 1 and 2.

There was no significant difference in change in resting wrist angle, the range of passive extension, or the end angle of active wrist extension between the treatment and control groups. There were no significant differences between the treatment and control groups in change in handgrip strength, star cancellation (number of stars cancelled), Ashworth score (Table 2), or performance in the 9-hole peg test (Table 2).

The components of the ARAT score that showed improvement after ES (change from baseline to week 8) were the grasp and grip subscores (P = 0.013 and P = 0.02, respectively, compared with changes in controls) (Table 3). These differences were no longer statistically significant at 32 weeks. There were no significant improvements in the pinch or gross movement subscores after ES compared with those in the control group. There was a tendency for an increase from baseline to week 8 in total ARAT score in the treatment group compared with that in the control group (P = 0.11; Table 3). There remained a tendency for total ARAT score to be improved in the ES group at the end of follow-up; however, this difference again was not statistically significant. In the subgroup of 33 patients (15 ES, 18 control) with measurable residual wrist extensor strength at study entry (moment at 0° extension > 0) who were followed up at 8 weeks, the ARAT total score had increased by a mean of 21.1 (SD, 12.7) in the ES group compared with 10.3 (SD, 9.0)
in the control group ($P = 0.024$, Mann Whitney $U$ test); however, at 32 weeks the differences between these 2 subgroups were no longer statistically significant. There were no significant differences between the ES and control groups in change in Barthel or Rankin scores from weeks 0 to 8 or weeks 0 to 32 (Table 3). There were no significant between-group differences in local discomfort at the wrist at rest or on passive movement as assessed by the 6-point rating scale and the 10-cm visual analog scale.

Of the 27 patients who were allocated to ES and completed the study intervention period, including the 8-week assessment (end of treatment), 19 complied well, 3 missed occasional treatment sessions, and 5 complied poorly ($<50\%$ of sessions received). Of the 28 patients who were allocated to the control group and completed the study intervention period, including the 8-week assessment, 11 received between 12 and 16 additional physiotherapy contacts, 10 received 8 to 11 contacts, and 7 had $<8$ contacts (each up to 10 minutes in duration) between weeks 0 and 8. There was a trend for an increased total number of physiotherapy contacts during the 8-week study intervention phase in the ES group (median number of contacts, 12 to 16) compared with the control group (median, 8 to 12); however, this difference was not statistically significant ($P = 0.49$, Mann-Whitney $U$ test). The median proportion of total physiotherapy time spent in upper-limb activities was estimated at 26\% to 50\% for both the ES and the control groups. There were no significant differences between the ES and control groups in total physiotherapy plus occupational therapy contact time as an inpatient or after discharge from hospital; 28 of the 30 in the ES group had 1 hour of inpatient contact per day compared with 27 of 30 in the control group.

**Discussion**

The randomization procedure resulted in groups that were reasonably well matched at baseline in terms of patient age, sex, stroke type, and upper-limb disability. Patients selected as being

---

**TABLE 2. Neurological Impairments at Study Entry (Baseline, Week 0) and Changes From Baseline to End of Treatment (Week 8) and End of Follow-Up (Week 32)**

<table>
<thead>
<tr>
<th></th>
<th>Baseline (Week 0)</th>
<th>Change From Baseline (Weeks 0–8)</th>
<th>Change From Baseline (Weeks 0–32)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Treatment Group</td>
<td>Control Group</td>
<td>Treatment Group</td>
</tr>
<tr>
<td>Resting wrist angle, degree of extension: 1st measurement</td>
<td>$-9.8 (9.5)$</td>
<td>$-8.8 (11.3)$</td>
<td>$-1.4 (10.3)$</td>
</tr>
<tr>
<td>Resting wrist angle, degree of extension: 4th measurement</td>
<td>$-4.9 (5.3)$</td>
<td>$-4.4 (7.8)$</td>
<td>$-1.2 (7.7)$</td>
</tr>
<tr>
<td>End range passive extension, degree</td>
<td>$59.4 (14.9)$</td>
<td>$59.3 (13.9)$</td>
<td>$-8.3 (16.0)$</td>
</tr>
<tr>
<td>End active extension angle, degree</td>
<td>$14.7 (18.9)$</td>
<td>$14.8 (24.4)$</td>
<td>$4.6 (18.3)$</td>
</tr>
<tr>
<td>Moment of wrist extension at 0°, nm, median (interquartile range)</td>
<td>$0.1 (0.0, 1.0)$</td>
<td>$0.7 (0.0, 2.0)$</td>
<td>$0.9 (0.0, 1.5)$</td>
</tr>
<tr>
<td>Moment at 15°, nm, median (interquartile range)</td>
<td>$0.0 (0.0, 0.3)$</td>
<td>$0.1 (0.0, 1.3)$</td>
<td>$0.6 (0.0, 1.2)$</td>
</tr>
<tr>
<td>Moment at 30°, nm, median (interquartile range)</td>
<td>$0.0 (0.0, 0.1)$</td>
<td>$0.0 (0.0, 0.4)$</td>
<td>$0.0 (0.0, 0.7)$</td>
</tr>
<tr>
<td>Grip, kg, median (interquartile range)</td>
<td>$2 (0, 5)$</td>
<td>$0 (0, 8.5)$</td>
<td>$2 (0, 3)$</td>
</tr>
<tr>
<td>Star cancellation, no. of stars cancelled</td>
<td>$36.2 (10.3)$</td>
<td>$41.1 (17.7)$</td>
<td>$7.7 (15.7)$</td>
</tr>
<tr>
<td>Ashworth score, median (interquartile range)</td>
<td>$0 (0, 1)$</td>
<td>$1 (0, 2)$</td>
<td>$0 (0, 1)$</td>
</tr>
</tbody>
</table>

Results are mean (SD) unless otherwise stated.

Statistical analyses by Mann Whitney $U$ test; comparisons are change from baseline in control compared with change from baseline in treatment group.

---

**Figure 2.** Moment of wrist extension (at an angle of wrist extension of 0°) in the treatment (ES) and control groups (median, nm). Week numbers are indicated on the bars.
TABLE 3. Disability at Study Entry (Baseline, Week 0) and Changes From Baseline to End of Treatment (Week 8) and End of Follow-Up (Week 32)

<table>
<thead>
<tr>
<th></th>
<th>Baseline (Week 0)</th>
<th>Change From Baseline (Weeks 0–8)</th>
<th>Change From Baseline (Weeks 0–32)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Treatment Group</td>
<td>Control Group</td>
<td>Treatment Group</td>
</tr>
<tr>
<td>ARAT grasp subscore</td>
<td>0 (0, 6)</td>
<td>0 (0, 15)</td>
<td>4 (0, 11)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>P=0.013</td>
</tr>
<tr>
<td>ARAT grip subscore</td>
<td>0 (0, 3)</td>
<td>0 (0, 8)</td>
<td>2 (0, 8)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>P=0.02</td>
</tr>
<tr>
<td>ARAT pinch subscore</td>
<td>0 (0, 1)</td>
<td>0 (0, 4)</td>
<td>1 (0, 9)</td>
</tr>
<tr>
<td>ARAT gross movement subscore</td>
<td>0 (0, 8)</td>
<td>0 (0, 9)</td>
<td>1 (0, 3)</td>
</tr>
<tr>
<td>ARAT total</td>
<td>6 (0, 18)</td>
<td>0 (0, 39)</td>
<td>10 (0, 29)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>P=0.001</td>
</tr>
<tr>
<td>No. of pegs per second</td>
<td>0 (0, 0.01)</td>
<td>0 (0, 0.35)</td>
<td>0 (0, 0.13)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>P=0.02</td>
</tr>
<tr>
<td>Barthel Index score</td>
<td>7.5 (6, 11)</td>
<td>7.5 (7, 14)</td>
<td>5 (3, 7)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>P=0.003</td>
</tr>
<tr>
<td>Rankin scale score</td>
<td>4 (4, 5)</td>
<td>4 (4, 5)</td>
<td>−1 (−1, 0)</td>
</tr>
</tbody>
</table>

Results are median (interquartile range).

Statistical analyses by Mann Whitney U test; comparisons are change from baseline in control compared with change from baseline in treatment group.

suitable for the study complied well with our study protocol, with few dropping out before the end of the intervention period. ES was not associated with any significant local discomfort, and this may have been due to the particular care that was taken in setting the amplitude of stimulation at the minimum level required to produce full-wrist extension.

We found that ES enhanced the recovery of isometric wrist extensor strength, as shown by a greater increase in the moment at the wrist (at an angle of 0°) at the end of the treatment phase compared with the increase in the control group. Enhanced recovery in the treatment group was still apparent at the end of follow-up, 24 weeks after ES was stopped. Similar findings have been reported in a systematic review of randomized controlled trials of ES applied to various other skeletal muscle groups after stroke and in a recent study of ES of wrist extensors.

We found that ES applied early after stroke did not help to maintain the range of wrist extension movement. However, these results do not exclude the possibility that ES may have beneficial effects on range of joint movement later after stroke, when contractures have developed. We have previously shown that in such patients ES of wrist extensors can significantly increase the range of movement at the wrist; however, benefits may not be maintained when treatment is discontinued.

ES reduced upper-limb disability as assessed by the grip and grasp subscores in the ARAT. However, the differences between treatment and control were no longer statistically significant at week 32; it is therefore not certain how long benefits are maintained when treatment is discontinued. No significant differences in improvement in pinch or gross movement were seen. Although there was a tendency for the total ARAT score to be increased after ES, this was not statistically significant. However, in the subgroup of patients with measurable residual motor function at baseline, there was a significant increase in total ARAT score at week 8.

ARAT scores tended to deteriorate between weeks 8 and 32, indicating an increase in upper-limb disability, even though the improvement in isometric wrist extensor strength appeared to be maintained. This is likely due to a combination of factors, including patients’ regression in functional abilities after the program of therapy had stopped; reduction in range of movement at the wrist late after stroke may also have played a part. It is recognized that there is not necessarily a close relationship between neurological impairments and disability in stroke patients.

Most previous studies of ES of the upper limb after stroke have concentrated on the effects on neurological impairments. Where studies have included functional assessments, many subjects have been unable to attempt the tasks chosen as methods of measurement. A recent study failed to find any beneficial effect of ES of wrist extensors on disability as measured by the self-care component of the Functional Independence Measure. However, this assessment may not be sensitive enough to detect modest but clinically worthwhile changes. In our study, the 9-hole peg test proved to be uninformative, because many patients were unable to insert any pegs. This test is recognized to have a floor effect, with an inability to discriminate between moderate and severe upper-limb disability. In contrast, most of our patients were able to perform at least a part of the ARAT, our primary measure of upper-limb disability.

There are a variety of possible reasons that our patients who were given ES showed benefit. ES may have a direct effect leading to increased muscle strength and improved motor control, resulting in reduced upper-limb disability. It has been claimed that improvement in muscle strength after ES may be due to a reduction in muscle tone and any beneficial effects are short lived. Our results do not support this latter hypothesis, because tone appeared to be unaffected while improvements in muscle strength were maintained after ES was discontinued. ES may simply be a convenient method of repetitive contraction and stretching of muscle groups, and it is possible that simple, passive range-of-motion or active, assisted range-of-motion exercises would lead to the same results. However, it is possible that ES has a combination of effects, including those at the level of the muscle, and also a central effect associated with improved motor relearning. As
with any treatment there may be a placebo effect: a general increase in awareness of the hemiparetic limb may occur as a nonspecific effect of ES, encouraging the patient to use the upper limb with therapy activities. We attempted to control for the additional clinical contact that inevitably is associated with applying ES by providing additional visits from the study physiotherapist to the control group to discuss progress in rehabilitation. However, it is far from clear whether the level of input that we provided resulted in the same level of nonspecific contact for both of our study groups. Last, it has been suggested that ES may have a specific effect on the recovery of neglect and sensory deficits; however, we could find no evidence of effects on visuospatial neglect, as measured by the star cancellation test.

One of the key difficulties in studying the effects of ES is in devising an appropriate control group. When ES is being administered to cause muscle contraction and joint movement, it is clearly not feasible to blind the patients to their treatment allocation. In contrast, this may be possible when ES is administered at a low intensity, causing cutaneous stimulation but without muscle contraction and joint movement; in this circumstance it may be worth considering use of an inactive dummy or placebo stimulator in the control group. The choice of the control group intervention also will depend on whether the study is pragmatic or exploratory. In a pragmatic study such as ours, it is appropriate to establish the effects of adding ES to the usual treatment. This will then give valuable information about the likely clinical impact of the intervention, although it may be more difficult to determine the mechanism of any effect.

There remain a number of unresolved issues for future studies to address. It is not certain that the improvements we have seen in selected impairments and upper-limb disability will lead to improved quality of life. Larger-scale studies will be required to determine whether ES of specific muscle groups after stroke will lead to improved self-care. The mechanism of effect of ES is probably multifactorial and requires further study for definition. Properly controlled studies are also required of cutaneous ES (at an intensity below that required for muscle contraction) in patients with motor deficit and in those with neglect.

Conclusions
In carefully selected patients with acute stroke, cyclic ES of the wrist extensors enhances motor recovery and reduces upper-limb disability. Although the reduction in upper-limb disability may not be maintained after treatment is stopped, the above benefits may still be clinically worthwhile. ES is a relatively low-cost intervention, at approximately $350 per system with only minor consumables costs. This treatment should be considered for highly motivated patients with moderate motor deficit persisting beyond 2 weeks. It is unlikely to be beneficial in patients with very mild deficits or in those with profound weakness in whom there is little prospect of useful functional recovery.

Acknowledgments
The authors would like to thank the stroke rehabilitation team at Glasgow Royal Infirmary and Ward 1 Lightburn Hospital for their help and support. Financial support was received from the Scottish Office Home and Health Department (Disability and Continuing Healthcare Committee).

References
Electrical Stimulation of Wrist Extensors in Poststroke Hemiplegia
Joanna Powell, A. David Pandyan, Malcolm Granat, Margaret Cameron and David J. Stott

Stroke. 1999;30:1384-1389
doi: 10.1161/01.STR.30.7.1384

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/30/7/1384

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/