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Combined arm stretch positioning and neuromuscular electrical stimulation during rehabilitation does not improve range of motion, shoulder pain or function in patients after stroke: a randomised trial

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Question: Does static stretch positioning combined with simultaneous neuromuscular electrical stimulation (NMES) in the subacute phase after stroke have beneficial effects on basic arm body functions and activities? Design: Multicentre randomised trial with concealed allocation, assessor blinding, and intention-to-treat analysis. Participants: Forty-six people in the subacute phase after stroke with severe arm motor deficits (initial Fugl-Meyer Assessment arm score ≤ 18). Intervention: In addition to conventional stroke rehabilitation, participants in the experimental group received arm stretch positioning combined with motor amplitude NMES for two 45-minute sessions a day, five days a week, for eight weeks. Control participants received sham arm positioning (ie, no stretch) and sham NMES (ie, transcutaneous electrical nerve stimulation with no motor effect) to the forearm only, at a similar frequency and duration. Outcome measures: The primary outcome measures were passive range of arm motion and the presence of pain in the hemiplegic shoulder. Secondary outcome measures were severity of shoulder pain, restrictions in performance of activities of daily living, hypertonia, spasticity, motor control and shoulder subluxation. Outcomes were assessed at baseline, mid-treatment, at the end of the treatment period (8 weeks) and at follow-up (20 weeks). Results: Multilevel regression analysis showed no significant group effects nor significant time × group interactions on any of the passive range of arm motions. The relative risk of shoulder pain in the experimental group was non-significant at 1.44 (95% CI 0.80 to 2.62). Conclusion: In people with poor arm motor control in the subacute phase after stroke, static stretch positioning combined with simultaneous NMES has no statistically significant effects on range of motion, shoulder pain, basic arm function, or activities of daily living. Trial registration: NTR1748. [de Jong LD, Dijkstra PU, Gerritsen J, Geurts ACH, Postema K (2013) Combined arm stretch positioning and neuromuscular electrical stimulation during rehabilitation does not improve range of motion, shoulder pain or function in patients after stroke: a randomised trial. Journal of Physiotherapy 59: 245–254]

Keywords: Stroke, Upper extremity, Muscle stretching exercises, Electrical stimulation, Activities of daily living, Randomized controlled trial

Introduction

Annually, 15 million people worldwide suffer a stroke (Mackay and Mensah 2004). About 77–81% of stroke survivors show a motor deficit of the extremities (Barker and Mullooly 1997). In almost 66% of patients with an initial paralysis, the affected arm remains inactive and immobilised due to a lack of return of motor function after six months (Sunderland et al 1989, Wade et al 1983). Over time, the central nervous system as well as muscle tissue of the arm adapt to this state of inactivity, often resulting in residual impairments such as hypertonia (de Jong et al 2011, van Kuijk et al 2007), spasticity (O’Dwyer et al 1996) or contractures (Kwaal et al 2012, O’Dwyer et al 1996, Pandyan et al 2003). In turn, these secondary impairments are associated with hemiplegic shoulder pain (Aras et al 2004, Roosink et al 2011) and restrictions in performance of activities of daily living (Lindgren et al 2007, Lundström et al 2008).

Several interventions improve arm function after stroke and prevent secondary impairments, eg, bilateral arm training (Coupar et al 2010) or constraint-induced movement therapy (Sirtori et al 2009). However, these interventions are not suitable for people with severe motor deficits because they require ‘active’ residual arm motor capacity. For these people ‘passive’ interventions may be needed to prevent secondary impairments and optimise long-term handling.
and assistive use of the affected arm. It is also important to elicit muscle activity if at all possible, and to improve arm function. To prevent the loss of passive range of joint motion as a result of contracture of at-risk muscles in the shoulder (eg, internal rotators, adductors) and forearm (eg, pronators, wrist and finger flexors) in particular, the application of arm stretch positioning alongside regular physiotherapy was deemed important (Ada and Canning 1990), especially because contractures are associated with shoulder pain (Araš et al 2004, de Jong et al 2007, Wanklyn et al 1996). However, in general, passive stretch does not produce clinically important changes in joint range of motion, pain, spasticity, or activity limitations (Katalinic et al 2011). One explanation for the lack of effect of passive stretch of the shoulder muscles could be the inadequate duration of stretch, with clinical trials using a dose of 20 or 30 minutes only (Borisova and Bohannon 2009). However, it is questionable whether stretch of the shoulder muscles for much more than 60 minutes per day during intensive rehabilitation programs is feasible (Turton and Britton 2005).

People with severe motor deficits after stroke have a higher risk of developing increased resistance to passive muscle stretch (hypertonia) and spasticity of the muscles responsible for an antigravity posture (de Jong et al 2011, Kwah et al 2012, Urban et al 2010). These muscles are also at risk of developing contracture. As a result, the passive range of the hemiplegic shoulder (external rotation, flexion and abduction), elbow (extension), forearm (supination) and wrist (extension) can become restricted.

Stretching hypertonic muscles is difficult when they are not sufficiently relaxed. Cyclic neuromuscular electrical stimulation (NMES) (Chae et al 2008), another example of a ‘passive’ intervention, can not only be used to improve pain-free range of passive humeral lateral rotation (Price and Pandyan 2000), but also to reduce muscle resistance (King 1996) and glenohumeral subluxation (Pomeroy et al 2006, Price and Pandyan 2000). From these results we hypothesised that NMES of selected arm muscles opposite to muscles that are prone to the development of spasticity and contracture might facilitate static arm stretching both through reciprocal inhibition (‘relaxation’) of antagonist muscles (Alfieri 1982, Dewald et al 1996, Fujiwara et al 2009) and the imposed (cyclic) stretch caused by motor amplitude NMES. Consequently, static arm stretch positioning combined with NMES could potentially result in larger improvements of arm passive range of motion and less (severe) shoulder pain compared to NMES or static stretching alone. From these hypotheses we developed the following research questions:

1. Does eight weeks of combined static arm stretch positioning with simultaneous NMES prevent the loss of shoulder passive range of motion and the occurrence of shoulder pain more than sham stretch positioning with simultaneous sham NMES (ie, transcutaneous electrical stimulation, TENS) in the subacute phase of stroke?

2. Does the experimental intervention have any additional effects on timing and severity of shoulder pain, restrictions in daily basic arm activities, resistance to passive stretch (hypertonia) and spasticity, arm motor control, and the degree of shoulder subluxation?

Method

Design

A multicentre, assessor-blinded, randomised controlled trial was conducted. After inclusion, participants were randomised in blocks of four (2:2 allocation ratio) in two strata (Fugl-Meyer Assessment arm score 0–11 points and 12–18 points) at each treatment centre. Opaque, sealed envelopes containing details of group allocation were prepared by the main co-ordinator (LDdJ) before trial commencement. After a local trial co-ordinator had determined eligibility and obtained a patient’s consent, the main co-ordinator was contacted by phone. He instructed an independent person to draw an envelope blindfolded and to communicate the result back to the local trial co-ordinator. The local trial co-ordinator then made arrangements for the baseline measurement after which the allocated intervention was initiated. Mid-treatment, end-treatment, and follow-up measurements took place at 4, 8, and 20 weeks after baseline measurement by two independent assessors (physiotherapists), who were unaware of group allocation and not involved in the treatment of participants. To keep the assessors blinded, participants were reminded before each measurement not to reveal the nature of their treatment. Participants were considered to be unaware of group allocation because they were informed about the existence of two intervention groups but not about the study hypothesis. The participants’ and assessors’ beliefs regarding allocation were checked at the eighth-week (ie, end of treatment) assessment using a three-point nominal scale (I suspect allocation to experimental/control group, I have no clue of group allocation). All investigators, staff, and participants were kept blinded with regard to the outcome measurements.

Participants

Between August 2008 and September 2010, consecutive newly admitted patients on the neurological units of three rehabilitation centres in the Netherlands (Beetsterzwaag, Doorn, and Zwolle) were approached for participation. Willing patients were initially screened by a physician for the following inclusion criteria: first-ever or recurrent stroke (except subarachnoid haemorrhages) between two and eight weeks poststroke; age > 18 years; paralysis or severe paresis of the affected arm scoring 1–3 on the recovery stages of Brunnstrom (1970); and no planned date of discharge within four weeks. Subsequently, a local trial co-ordinator excluded patients with: contraindications for electrical stimulation (eg, metal implants, cardiac pacemaker); pre-existing impairments of the affected arm (pre-existing contracture was not an exclusion criterion); severe cognitive deficits and/or severe language comprehension difficulties, defined as < 3/4 correct verbal responses and/or < 3 correct visual graphic rating scale scores on the AbilityQ (Turner-Stokes and Rusconi 2003); and moderate to good arm motor control (> 18 points on the Fugl-Meyer Assessment arm score).

Interventions

All participants received multidisciplinary stroke rehabilitation, ie, daily training in activities of daily living by rehabilitation nurses, occupational therapists, physiotherapists, and speech therapists. These interventions were not standardised, but generally administered in a way that was consistent with the recommendations of
Participants were requested to undergo the additional allocated treatment twice daily for 45 minutes on weekdays for 8 weeks. Participants from the experimental group received arm stretch positioning (presented in Figures 1a and 1b) with simultaneous four-channel motor amplitude NMES. Participants from the control group received a sham stretch positioning procedure (presented in Figure 1c) with simultaneous sham conventional TENS with minimal sensory sensation by using a similar treatment protocol, electrical stimulator and electrode placement (but on the forearm only) as the experimental group. A detailed description of the experimental and control group procedures can be found in Appendix 1 (see the eAddenda for Appendix 1).

Treatment was planned to result in 60 hours of positioning and 51 hours of NMES/TENS. All procedures were performed by the local trial coordinator or instructed nursing staff. Nursing staff monitored compliance to the intervention by logging each session on a record sheet, which was always kept in the vicinity of the participant’s bed. During the first 8 weeks of the trial, prescription of pain and spasticity medication as well as content of physical and occupational therapy sessions for the arm were also monitored.

Outcome measures

The primary outcome measures were passive range of arm motion and pain in the hemiplegic shoulder. All goniometric assessments were performed by two observers using a fluid-filled goniometer. Inter-observer reliability of this technique was high (de Jong et al 2012). The presence of shoulder pain was checked using the first (yes/no) question of the ShoulderQ (Turner-Stokes and Jackson 2006). The secondary outcome measures were timing and severity of poststroke shoulder pain, performance of real-life passive and basic daily active arm activities, hypertonia and spasticity, arm motor control and shoulder subluxation. All measurements were carried out in the same fixed order by the same two trained assessors. Every effort was made to motivate participants to undergo all planned measurements even after withdrawal from the study.

Passive range of shoulder external rotation, flexion and abduction, elbow extension, forearm supination, wrist extension with extended and flexed fingers were assessed because these movements often develop restrictions in range as a result of imposed immobility, with muscle contractures causing a typical flexion posture of the hemiplegic arm. The (entire) ShoulderQ was administered in participants who indicated that they had shoulder pain. This questionnaire assesses timing and severity of pain by means of eight verbal questions and three vertical visual graphic rating scales. We were primarily interested in the answer to the (verbal) question How severe is your shoulder pain overall? (1 = mild, 2 = moderate, 3 = severe, 4 = extremely severe) and pain severity measured at rest, on movement, and at night using the 10-cm vertical visual graphic rating scales. The ShoulderQ is sensitive (Turner-Stokes and Jackson 2006) and responsive to change in pain experience (Turner-Stokes and Rusconi 2003). Performance of basic functional activities of daily life involving the passive arm was assessed using the Leeds Adult/Arm Spasticity Impact Scale (Ashford et al 2008). Using this semi-structured interview, participants were asked to indicate whether they or their carer(s) experienced difficulty performing 12 different tasks involving the hemiplegic arm (cleaning the palm/elbow/armpits, cutting fingernails, putting the arm through a sleeve/in a glove, rolling over in bed, doing exercises, balancing while standing/walking, and holding objects). The scores on the separate items (1 point = no difficulty, 0 = difficulty or activity not yet performed) were summed, divided by the total number of items performed and multiplied by 100, resulting in a summary score (0 = severe disability, 100 = no disability). Hypertonia and spasticity of the shoulder internal rotators, elbow flexors, and long finger flexors were assessed using a detailed version (Morris 2002) of the Tardieu Scale (Held and Pierrot-Deseilligny 1969). The Tardieu Scale can differentiate spasticity from...
Figure 2. Design and flow of participants through the trial. aAll reasons for exclusion are listed where patients were ineligible for multiple reasons. bIncluding multiple sclerosis, Alzheimer's disease, locked-in syndrome, recurrent stroke, and participation in another trial. NMES = neuromuscular electrical stimulation. cOne participant from each group dropped out after randomisation but before receiving any intervention. dUnrelated to stroke. eOne participant missed the Week 4 assessment due to poor weather. fOne participant missed the Week 8 assessment due to recurrent stroke but was subsequently available for the Week 20 follow-up assessment.
Table 1. Baseline characteristics of participants and centres.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Exp (n = 23)</th>
<th>Con (n = 23)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr), mean (SD)</td>
<td>56.6 (14.2)</td>
<td>58.4 (9.6)</td>
</tr>
<tr>
<td>Time post-stroke at baseline (days), mean (SD)</td>
<td>43.7 (13.3)</td>
<td>43.3 (15.5)</td>
</tr>
<tr>
<td>MMSE(*), median (IQR)</td>
<td>27 (23 to 28.25)</td>
<td>28 (26 to 29.5)</td>
</tr>
<tr>
<td>Gender, n males (%)</td>
<td>15 (65)</td>
<td>12 (52)</td>
</tr>
<tr>
<td>Stroke type, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ICVA</td>
<td>19 (83)</td>
<td>18 (78)</td>
</tr>
<tr>
<td>HCVA</td>
<td>4 (17)</td>
<td>5 (22)</td>
</tr>
<tr>
<td>Affected hemisphere, n right (%)</td>
<td>12 (52)</td>
<td>8 (35)</td>
</tr>
<tr>
<td>Aphasia, n (%)</td>
<td>5 (22)</td>
<td>6 (26)</td>
</tr>
<tr>
<td>Initial FMA arm score, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0–11 points</td>
<td>19 (83)</td>
<td>17 (74)</td>
</tr>
<tr>
<td>12–18 points</td>
<td>4 (17)</td>
<td>6 (26)</td>
</tr>
<tr>
<td>Centres, participants treated, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beetsterzwaag</td>
<td>7 (30)</td>
<td>8 (35)</td>
</tr>
<tr>
<td>Doorn</td>
<td>4 (17)</td>
<td>4 (17)</td>
</tr>
<tr>
<td>Zwolle</td>
<td>12 (52)</td>
<td>11 (48)</td>
</tr>
</tbody>
</table>

Exp = experimental group, Con = control group, FMA = Fugl-Meyer Assessment arm score, HCVA = haemorrhagic cerebrovascular accident, ICVA = ischaemic cerebrovascular accident, MMSE = Mini Mental State Examination. *Not administered in subjects with aphasia.

data contracture (Haugh et al 2006, Patrick and Ada 2006) and has fair to excellent test-retest reliability and inter-observer reliability (Paulis et al 2011). The mean angular velocity of the Tardieu Scale’s fast movement was standardised (see the eAddenda for Appendix 2). Muscle reaction quality scores ≥ 2 were considered to be clinically relevant hypertonia. Spasticity was deemed present if the angle of catch was present and occurred earlier in range than the maximal muscle length after slow stretching (ie, spasticity angle > 0 degs). Arm motor control was assessed using the 66-point arm section of the Fugl-Meyer Assessment (Fugl-Meyer et al 1975, Gladstone et al 2002). Shoulder inferior subluxation was diagnosed by palpation (Bohannon and Andrews 1990) in finger breadths (< ½, < 1, ≥ 1, > 1½) and considered present if it was one category higher than on the nonaffected side.

Data analysis

Sample size calculation was based on a reliably assessable change in passive shoulder external rotation range of motion of ≥ 17 degs (de Jong et al 2012). The clinically relevant difference between the experimental and control intervention was therefore set at a minimum of 20 deg. The standard deviation was considered to be 21.5 deg (Ada et al 2005). Alpha was set at 5% (two-sided), beta at 80%. Thus, the required number of participants in each group was 18. Anticipating a 10% drop-out rate and requiring 36 complete datasets, we aimed to recruit at least 20 participants per group.

All participants minus two premature dropouts were analysed as randomised (intention-to-treat). Arm passive range of motion was analysed using a multilevel regression analysis. As main factors time (baseline, 4, 8, and 20 weeks), group allocation (2 groups) and time × group interaction were explored using the −2log-likelihood criterion for model fit, as well as random effects of intercept and slope. For completeness, this analysis was repeated using the data of the participants including the two premature dropouts (n = 48) using the last observation carried forward approach. Nominal outcome measures (presence of hypertonia/spasticity and subluxation) at eight weeks were analysed using a Chi-square test. Ordinal outcome measures (Fugl-Meyer Assessment, Leeds Adult/Arm Spasticity Impact Scale, ShoulderQ) were first analysed for time effects within subjects using the Friedman test. If differences over time (from baseline to follow-up) were found, these were further explored using the Wilcoxon signed-rank test with Bonferroni-Hochberg correction (Norman and Streiner 2000). Between-group differences were analysed using a Mann-Whitney U test only at 8 weeks to avoid multiple testing.

Results

Flow of participants through the trial

The flow of participants through the trial is presented in Figure 2. Forty-eight patients met all eligibility criteria. One participant from the experimental group (a 68-year-old female with a right-sided ischaemic stroke who regretted participation) and one from the control group (a 62-year old male with a left-sided ischaemic stroke who was rehospitalised due to acute liver and kidney failure) dropped out the day after baseline measurement and before receiving any intervention. These participants were not included in the analyses because their data were missing due to unavailability for further measurements.

Of the 11 patients who were lost to follow-up or discontinued their prescribed intervention during the 8-week treatment period, four (36%) complained of pain. Baseline characteristics of the 46 participants analysed are shown in Table 1. Twenty-two participants (51%, n = 43) had no clue as to which group they were allocated, but 17 participants (40%) were correct in their belief regarding allocation. The three participants who were lost to follow-up before 8 weeks did not provide data about allocation beliefs. The two assessors had no clue regarding group allocation. The three participants who were lost to follow-up before 8 weeks did not provide data about allocation beliefs. The two assessors had no clue regarding group allocation in 67% and 72% of the cases. They were correct in their belief regarding allocation in 9 (21%) and 4 (9%) of the participants, respectively.

Co-interventions and compliance with trial method

In the experimental group more participants were prescribed pain and spasticity medication, as presented in Table 2. They also received slightly more conventional therapy for the arm and adhered less to the prescribed intervention protocol. Overall, compliance in the experimental group was 68% (stretch positioning) and 67% (NMES), compared to 78% (sham positioning) and 75% (TENS) in the control group. Non-compliance was mainly caused by drop-out and
Table 2. Mean (SD) or number of participants (%) for co-interventions and compliance to the intervention protocol during the eight-week intervention period and mean difference (MD) or percentage risk difference (RD) between groups, with 95% confidence intervals (95% CI).

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Exp (n = 23)</th>
<th>Con (n = 23)</th>
<th>Difference between groups (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prescription of pain medication, n (%)</td>
<td>16 (73)a</td>
<td>11 (48)</td>
<td>RD 25% (~4% to 50%)</td>
</tr>
<tr>
<td>Prescription of spasticity medication, n (%)</td>
<td>5 (23)a</td>
<td>2 (9)</td>
<td>RD 14% (~8% to 36%)</td>
</tr>
<tr>
<td>Upper limb occupational therapy (hr), mean (SD)</td>
<td>5 (4)a</td>
<td>4 (4)a</td>
<td>MD 1 (~2 to 3)</td>
</tr>
<tr>
<td>Upper limb physiotherapy (hr), mean (SD)</td>
<td>3 (5)</td>
<td>2 (3)a</td>
<td>MD 1 (~2 to 3)</td>
</tr>
<tr>
<td>Total of positioning (hr), mean (SD)</td>
<td>41 (17)a</td>
<td>47 (16)</td>
<td>MD –6 (~15 to 4)</td>
</tr>
<tr>
<td>Total of electrical stimulation (hr), mean (SD)</td>
<td>34 (16)a</td>
<td>38 (14)</td>
<td>MD –4 (~13 to 5)</td>
</tr>
</tbody>
</table>

Exp = experimental group, Con = control group. aData missing for one participant.

Discussion

To our knowledge this is the first study to analyse the effects of a daily arm stretch positioning procedure combined with simultaneous NMES in patients with a poor prognosis for functional recovery in the subacute phase after stroke. The 8-week high-intensity multimodal intervention did not result in any significant differences in arm passive range of motion (contractures), shoulder pain, basic arm activities, hypertonia/spasticity, arm motor control or shoulder subluxation compared to a control group receiving a similar amount of sham positioning combined with TENS in addition to conventional rehabilitation.

Previous attempts to maintain hemiplegic arm joint range of motion using static muscle stretching procedures could not prevent considerable loss of shoulder passive range of motion (Ada et al 2005, Gustafsson and McKenna 2006, de Jong et al 2006, Turton and Britton 2005). Our participants showed similar reductions in mean passive range of motion across most arm joints. Overall, there were no significant differences in passive range of motion between the two groups. At baseline (on average, six weeks post-stroke), 57% of the participants reported (shoulder) pain. During the intervention period, the prevalence increased to 52% and decreased to 36% three months later. These findings are in line with reports that post-stroke shoulder pain is common, affecting 22–64% of cases, particularly patients with poor arm function (Aras et al 2004, Gamble et al 2002, Lindgren et al 2007). Overall, pain severity also increased, particularly on movement and at night. This adverse effect was also noted in other trials (Gustafsson and McKenna 2006, Turton and Britton 2005). Although there were no significant between-group differences regarding shoulder pain, worrisome observations were that in the experimental group some participants reported that they considered the intervention to be very arduous, pain and spasticity medication were prescribed more frequently, and protocol compliance was lower. Combined with the finding that shoulder pain was more likely to occur in participants in the experimental group than in the control group (relative risk 1.44), these findings may indicate that for some participants the intervention was not well tolerated.

During the eight weeks of intervention our participants showed increased Leeds Adult/Arm Spasticity Impact Scale sum scores and Fugl-Meyer Assessment arm motor scores – changes that were probably not clinically relevant.
Table 3. Mean (SD) for passive range of motion in degrees for each group, mean (SD) difference within groups, and mean (95% CI) difference between groups. The multi-level regression analysis identified significant time effects for the three shoulder movements and for forearm supination. There was no significant group effect nor a significant group \times time interaction. A random intercept results in the best fit for the data (–2log-likelihood criterion).

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Groups</th>
<th>Difference within groups</th>
<th>Difference between groups</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Week 0</td>
<td>Week 4</td>
<td>Week 8</td>
</tr>
<tr>
<td></td>
<td>Exp (n = 23)</td>
<td>Con (n = 23)</td>
<td>Exp (n = 21)</td>
</tr>
<tr>
<td>Shoulder external rotation</td>
<td>29 (20)</td>
<td>34 (19)</td>
<td>20 (28)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shoulder flexion</td>
<td>130 (33)</td>
<td>122 (29)</td>
<td>111 (37)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shoulder abduction</td>
<td>110 (48)</td>
<td>93 (41)</td>
<td>93 (51)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Elbow extensiona</td>
<td>3 (8)</td>
<td>3 (7)</td>
<td>2 (9)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Forearm supination</td>
<td>77 (13)</td>
<td>78 (11)</td>
<td>68 (16)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wrist extension I</td>
<td>58 (18)</td>
<td>54 (17)</td>
<td>55 (20)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wrist extension II</td>
<td>66 (12)</td>
<td>60 (14)</td>
<td>59b (17)</td>
</tr>
</tbody>
</table>

Exp = experimental group, Con = control group, I = wrist extension with extended fingers, II = wrist extension with flexed fingers. aElbow extension values indicate deviation from the neutral position, ie, degrees of elbow flexor contracture with negative values representing hyperextension. bData missing for one participant.
and caused by a mix of spontaneous post-stroke recovery of function, learned capacity to use compensatory movement strategies of the nonaffected arm and/or increased involvement of the carer. Overall, the prevalence of elbow flexor hypertonia and spasticity jointly increased up to 55% at the end of the treatment period, roughly corresponding to three months post-stroke for our participants. These results are in concordance with previous work (de Jong et al 2011, van Kuijk et al 2007, Urban et al 2010). The unexpected high prevalence of hypertonia and spasticity (62%) and a decreasing prevalence of shoulder subluxation (31%) at follow-up in our sample may be explained by the fact that patients with relatively poor arm motor control have a higher risk of developing hypertonia (de Jong et al 2011).

Although we performed an intention-to-treat analysis (ie, using any available data from all randomised subjects), we did not use forward imputation of missing data representing a clinical variable (eg, shoulder passive range of motion) that is worsening over time (de Jong et al 2007), as this might increase the chance of a Type I error. However, for completeness, this stricter intention-to-treat analysis using the data of all randomised subjects (n = 48) was performed. This analysis was similar in outcome to the original analysis but revealed an additional time effect of wrist extension with flexed fingers. A per protocol analysis would also have resulted in similar results because no patients crossed over to the other group. We also refrained from performing a sensitivity analysis based on compliance because meaningful conclusions could not be drawn from the resulting limited sample sizes. We furthermore acknowledge that the Leeds Adult/Arm Spasticity Impact Scale lacks psychometric evaluation and our method to standardise the Tardieu Scale’s stretch velocity (V3) using a metronome was not validated and tested for reliability. Therefore, our data regarding basic arm activities, hypertonia, and spasticity should be interpreted with caution. Finally, because overall compliance to both protocols was only about 70%, an underestimation of the treatment effect may also have occurred. Nevertheless, the combined administration of 43 hours of static stretching and 36 hours of NMES was more than administered during any previous trial (Borisova and Bohannon 2009).

A recent study produced inconclusive evidence about the effectiveness of a combined intervention of electrical stimulation in conjunction with prolonged muscle stretch (using a splint) to treat and prevent wrist contracture (Leung et al 2012). Similarly, our results also showed no added benefit of electrical stimulation during static stretching of the shoulder and arm. The results of these multimodal approaches to the problem of post-stroke arm contracture development are in line with the conclusion of the most recent meta-analysis (Katalinic et al 2011) that static stretch positioning procedures have little, if any, short or long term effects on muscle contracture (treatment effect ≤ 3 deg), pain, spasticity, or activity limitations. Although pooled data from studies investigating the effects of electrical stimulation suggested some treatment effects on functional motor ability (Pomeroy et al 2006) and pain-free range of passive humeral lateral rotation in patients with residual arm motor capacity (Price and Pandyan 2000), we found no such results in our sample of patients without residual arm motor capacity. As the combined procedure did not result in any meaningful treatment effects, it suggests that application of muscle stretching or NMES alone as a monotherapeutic intervention will not have a clinically relevant impact in this subgroup of patients either.

Research to date suggests that it is not possible to control or overcome (the emergence of) contractures and hypertonia using the current static arm muscle stretching procedures. Similarly, NMES of the antagonists of the muscles prone to shortening does not seem to provide additional benefits either. We therefore argue that these techniques should be discontinued in the treatment of patients with a poor prognosis for functional recovery. In this subgroup of patients it is becoming an increasingly difficult challenge to find effective treatments that can prevent the development of the most common residual impairments such as contractures, hypertonia, and spasticity and its associated secondary problems such as shoulder pain and restrictions in performance of daily life activities. Further research is required to investigate what renders these interventions ineffective. The efficacy of other approaches, such as transcranial magnetic stimulation, NMES of the muscles prone to shortening (Goldspink et al 1991), or other combinations of techniques, could also be investigated.

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