Can treatment with upper limb electrical stimulation can be justified in the severely disabled acute stroke patient?

Pandyan AD [1], Granat MH [2]

[1] Institute of Ageing/Department of Physiotherapy Studies, Keele University, Keele, UK – ST5 5NA
[2] School of Health and Social Care, Glasgow Caledonian University, Glasgow, UK – G4 0BA

Email: a.d.pandyan@physio.keele.ac.uk

Abstract

Background: Disability following a stroke is common and loss of arm function significantly reduces participation and independence. Patients who do not to recover hand function in the acute stages of their stroke may be at an increased risk of developing contractures.

Design: Secondary analysis of data from a previous randomised controlled trial.

Method: Subjects were selected for the secondary analysis if they had no upper limb functional movement at recruitment (measured using the Action Research Arm test and the nine hole peg test). Subjects in the control group received routine physiotherapy and additional therapist contact time. In addition to routine therapy, subjects in the treatment group received treatment with surface electrical stimulation to the wrist extensors for a period of eight weeks after recruitment. Following a baseline measurement at recruitment, range of movement, stiffness and strength were measured at four, eight, twenty and thirty-two weeks from baseline. The primary end-points were changes over the study period analysed using an independent sample t-test.

Result: The control group had 14 patients and treatment group 16. Adaptive shortening was seen in both groups over the study period, it appeared to be less in the treatment group, but the deterioration was not significantly between groups (p > 0.10). Grip strength improved in both groups but the improvements were not significantly between groups (p > 0.10). The change in the ability to actively extend (i.e. active isometric extension strength) was greater in the treatment group (p < 0.01).

Conclusion: Electrical stimulation of the wrist extensors may reduce the rate of contracture formation in severely disabled stroke patient. Treatment may also facilitate improvements in wrist and finger extensor control.

1 Introduction

Despite routine conventional therapy, reported levels of arm recovery following stroke are poor [1] [2], although evidence suggests recovery could be better [3]. Patients who tend not to recover hand function, in the acute stages of their stroke, have a poor prognosis for recovery of arm function [2] [4]. Motor deficits, following a stroke, have often been associated with contractures [5]. The joints most prone to contractures are the wrist and ankle [6], with upper limb contractures being more common [5]. The aim of this study was to investigate whether treatment with electrical stimulation (ES) had the potential to prevent the formation of wrist flexion contractures in the severely disabled stroke patient.

2 Methods

This was a secondary analysis of data from a previously published randomised controlled trial aimed at investigating the effects of surface ES of the wrist on recovery of hand function [7]. Subjects were included to the original trial if they had arm weakness following a stroke two to four weeks previously, and could provide informed consent [7]. Subjects, from the original cohort, were selected for the secondary analysis if they were incapable of upper limb functional movement at recruitment (measured using the Action Research Arm Test (ARAT) and the nine hole peg test (NHPT))[2]. A person was classified as not having arm function, at recruitment, if they could not "Pickup and place" at least one peg in the NHPT and "Pickup and reach out and place a wooden cube ($1cm^3$)" in the ARAT.

Subjects in the control group (CG) received routine physiotherapy and had additional therapy contact time to compensate for treatment time associated with ES treatment [7]. Subjects in the treatment group (TG) received routine physiotherapy and received additional treatment with surface ES to the wrist extensors for a period of eight weeks after recruitment [7]. The surface electrodes were positioned for

balanced wrist extension, i.e. wrist extension with no ulnar or radial deviation [8].

Contractures were measured using a custom built device [8]. The primary measures were the resting posture of the wrist, resistance to passive extension at the wrist and passive range of wrist movement from flexion to extension [2][8]. Other impairments measured were grip strength and isometric wrist extension strength at neutral [2][8]. After an initial base line assessment, prior to randomisation, repeated measurements were taken at four, eight, twenty and thirty-two weeks after recruitment. The primary end-point was changes over the study period analysed using an independent sample t-test. Changes over the study period were analysed using the repeated measures analysis of variance. SPSS^{TM} (version 11.5) was used for all the analyses.

3 Results

From the original cohort of 60 patients, 14 CG patients and 16 TG patients were selected for the secondary analysis (Table 1). Treatment compliance, although not formally measured, was approximately 50%.

	CG	TG
Total anterior circulation syndromes (TACS)*	5	3
Partial anterior circulation syndrome (PACS) *	7	9
Lacunar syndrome (LACS) *	1	1
Intra-cerebral haemorrhage (ICH)	1	3
Side of body affected Left : Right	10:4	11:5
Time post stroke Mean (range) days	23 (14 – 34)	25 (12 – 39)
Age Mean (range) Years	64.2 (40-93)	69.2 (44 – 91)
Gender Female : Male	6 : 8	8 : 8

*Table 1: Clinical details related to the study groups. * Bamford J et al 1991 Classification and natural history of clinically identifiable subtypes of cerebral infarction. Lancet 337: 1521 – 1526.*

The resting posture deteriorated in both groups and the deterioration was not significantly different between the groups (p > 0.10: mean difference 4°: 95% CI:-3 to 11). The resting posture did not deteriorate significantly in the TG (p > 0.10: F = 1.67 with 4 df), however,

there was a significant deterioration in the CG (p < 0.01: F = 6.04 with 4 df).

The resistance to passive extension deteriorated in both groups but the deterioration was not significantly different between the groups (p > 0.10: mean difference -0.46 Nm: 95% CI:-1.40 to 0.45). The resistance to passive extension increased in the treatment group (p = 0.062: F = 2.38 with 4 df) and significantly increased in the control group (p < 0.05: F = 2.89 with 4 df).

The passive range of movement significantly deteriorated in both groups (p < 0.01: F = 7.24 & 3.81 respectively for treatment and control group with 4 df) but the deterioration was not significantly different between the groups (p > 0.1: mean difference -4°: 95% CI: -19 to 11).

Grip strength improved in both groups but the improvement was not significantly different between the groups (p > 0.10: mean difference 1.5kg and 95% CI:-1.1 to 4). The improvements for the treatment group (p < 0.01: F = 9.34 with 4 df) and the control group (p < 0.05: F = 3.6 with 4 df) were significant.

The improvement in the ability to actively extend the wrist (measured as the active isometric extension strength at neutral) was significantly better in the treatment group (p < 0.01: mean difference 1.6Nm and 95% CI: 0.66 to 2.63) when compared with the control group. The treatment group significantly improved (p < 0.01: F = 7.57 with 4 df) and the control group showed no change (p > 0.1: F = 0.87 with 4 df). Some subjects in the control group flexed their wrists when asked to extend.

4 Discussion and Conclusions

Adaptive shortening (dynamic contractures) was seen in both groups and the deterioration was less in the treatment group. Such a change can be primarily attributed to the increased mobilisation at the wrist associated with ES [9]. If one were to accept that contractures resulted from spasticity, then it could be argued that the reduction in contractures resulted from a reduction of spasticity. However, any reduction in spasticity associated with ES treatment has limited carry over [10] and, therefore, it is unlikely to have influenced contracture prevention.

Contrary to the initial expectations grip strength improved in both groups. If one were to accept previous findings that improvements in grip strength was a predictor of return in arm function [3] then functional improvements would have been expected in both groups.

However, this was not the case [11] and there are two possible explanations for this anomaly. Firstly grip strength of the treatment group was greater than the control group at recruitment, although not significantly. This could suggest that grip strength has to exceed a certain threshold in order to be functionally useful. Secondly it is possible grip strength is not a good prognostic indicator, as improvements in grip strength could be confounded by synergistic upper limb movement patterns [12] It is also possible that the reduction in grip strength could be associated with the changes in the resting position of the wrist (i.e. passive insufficiency).

There is evidence that treatment with ES can prevent atrophy and improve muscle strength in people with upper motor neurone lesions [13] and this alone may explain the improvements in the ability to actively extend. However, it is also possible that the length of the extensor muscles could have influenced the improvements in isometric extension strength. In this study, wrist extension strength was measured at neutral. As the resting posture in the control group significantly worsened, their ability to produce a moment in extension may have also reduced secondary to a lengthening in the extensors.

This was a small sample study that was not sufficiently powered, therefore, there is a risk of a Type I error. In the absence of EMG data it was not possible to draw any inference on changes in the levels of spasticity and "spastic hypertonia" per se [14]. However, as the measurement protocol involved a combination of slow passive movements and static measurements it is less likely to have been confounded by changes in spasticity [2].

The evidence suggests that there may be important therapeutic benefits associated with ES treatment such as prevention/reduction of adaptive shortening, improvement in strength and improvement in motor control. Treatment with ES is relatively cost effective and has few documented side effects. Therefore future studies should explore the therapeutic potential associated with ES treatment.

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