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Spasticity and contractures at the wrist after stroke: time course of development and their association with functional recovery of the upper limb

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Objective: To investigate the time course of development of spasticity and contractures at the wrist after stroke and to explore if these are associated with upper limb functional recovery.
Design: Longitudinal observational study using secondary data from the control group of a randomized controlled trial.
Setting: The Acute Stroke Unit at the University Hospital of North Staffordshire.
Subjects: Patients without useful arm function (Action Research Arm Test – ARAT) score of 0 within 6 weeks of a first stroke.
Main measures: Spasticity was measured by quantifying muscle activity during passively imposed stretches at two velocities. Contractures were measured by quantifying passive range of movement and stiffness. Upper limb functional movement was assessed using the ARAT. All assessments were conducted at baseline, and at 6, 12, 24 and 36 weeks after recruitment.
Results: Thirty patients (43% male, median age 70 (range 52–90) years, median time since stroke onset 3 (range 1–5) weeks) were included. Twenty-eight (92%) demonstrated signs of spasticity throughout the study period. Participants who recovered arm function ($n = 5$) showed signs of spasticity at all assessment points but did not develop contractures. Patients who did not recover useful arm function ($n = 25$) had signs of spasticity and changes associated with contracture formation at all time points tested.
Conclusion: In this group of patients who had no arm function within the first 6 weeks of stroke, spasticity was seen early, but did not necessarily hinder functional recovery. Contractures were more likely to develop in patients who did not recover arm function.

Introduction

Stroke is a leading cause of death and severe adult disability. Approximately 110,000 strokes occur in England every year[1] and around half of all those

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who survive have impairments that lead to loss of upper limb function.[2] Spasticity and contractures are two common impairments that affect the muscle and joints of the upper limb[3,4] and may significantly contribute to this functional loss and restrict social participation.[5]

Spasticity is defined as disordered sensorimotor control, resulting from an upper motor neuron lesion and presenting as intermittent or sustained involuntary activation of muscles.[6,7] It is a common neurological impairment which may develop within a week following a stroke.[8] Post-stroke spasticity may be maladaptive and interfere with a person's ability to perform functionally useful movement.[9,10] Contractures are more likely to develop if the abnormal muscle activity, resulting from spasticity, holds a joint in either shortened position and/or prevents active movement.[7]

Contracture is a pathological condition of soft tissues characterized by stiffness. It is usually associated with loss of elasticity and fixed shortening of the involved tissues resulting in both loss of range of movement and increased stiffness around a joint.[11] Many authors report the development of contractures in hemiplegic limbs following a stroke.[12–15] However, there is little information on the prevalence of contractures in the hemiplegic population. The two joints most prone to contractures are the wrist and ankle[3] with a higher incidence in the upper limb.[3,4] Contractures, in the upper limb joints, can lead to significant problems with cosmesis, hygiene and active movement capabilities, thereby resulting in significant participation restrictions. Spasticity may contribute to contracture formation[11] and clinical texts suggest that such a causal association exist.[10,12–15] However, there is little evidence to prove either a clinically important association between spasticity and contractures exists or that spasticity interferes with functionally useful movement.

The first steps in the exploration of these relationships are:

- to study the time course of development of both spasticity and contractures at the wrist in patients with stroke who do not have arm function at recruitment
- to assess whether spasticity impedes function and contributes to contractures.

Methods

Secondary anonymous data for this longitudinal analysis were obtained from the control group of a randomized controlled trial (RCT) conducted between 2004 and 2008. This study had full approval from the local research ethics committee (LREC approval 04/Q2604/1). Only those patients from the control group with a complete set of relevant measures associated with spasticity, contractures, pain and arm function were selected for this secondary analysis.

Patients within 6 weeks of a first stroke were eligible to participate in the RCT if they had a score of 0 in the Action Research Arm Test (ARAT).[16,17] Patients were excluded if they were medically unstable, had a previous medical history of osteoarthritis, rheumatoid arthritis or soft tissue injuries that resulted in contractures or had a reduced range of movement in the wrist and fingers. The control group received routine physiotherapy for 30 minutes each day for 6 weeks from recruitment (5-day week). The study therapist provided standardized routine upper limb therapy to all the participants and this therapy was a reflection of local practice.[18] Overnight splints were not used.

Following a baseline assessment, repeated measurements were taken at 6, 12, 24 and 36 weeks after recruitment. Measurements were taken at the patient's bedside on the acute stroke unit and the stroke rehabilitation ward. Follow-up measures were also done in the community e.g. in the patient's own home, sheltered housing, and in nursing or residential homes.

Clinical measures

Demographic details including age, gender, affected side of the body and stroke subtype were taken at recruitment. Patients were examined neurologically and classified as total anterior circulation syndrome (TACS), partial anterior circulation syndrome (PACS), lacunar syndrome (LACS) and posterior circulation syndrome (POCS).[19]

Spasticity was quantified neurophysiologically by measuring the muscle activity during passive extension of the wrist.[8] Wrist contractures were characterized biomechanically by measuring the

passive range of movement and stiffness at slow stretch at the wrist.[20] These measurements were taken using a custom-built device.[20] The measurement procedure, in brief, was as follows.

The participant's forearm was fully supported and positioned in a direction parallel to the ground, with forearm in mid pronation-supination, the elbow flexed to approximately 90° and the shoulder slightly abducted (<10° estimated visually) during the tests. The wrist was first flexed as far as comfortable for the subject. Applying a force transducer (to measure force used for stretching the forearm manually) on the palmar surface of the hand, the wrist was passively extended using a slow stretch from maximum flexion into maximum extension (manual count for 3 seconds).The wrist was once again returned into flexion and the movement was repeated using a brisk stretch as per guidance for modified Ashworth scale (duration of stretch being one second).[21] Force (measured in Newtons), passive range of movement (measured in degree) and muscle activity (measured in millivolts (mV)) were simultaneously taken during the externally imposed passive extension. The data from the transducers were sampled at 1000 Hz and stored in a personal computer for analysis.

Muscle activity was quantified from surface electromyography recordings using a customized programme (MathCAD 12, Mathsoft, USA). The raw electromyography data was notch filtered (50 Hertz) and smoothed using a root mean square procedure (window width 20 millisecond).[20] For each individual, wrist angles and muscle activity data were graphed as an XY scatterplot to classify muscle action.[8] The area under the angle muscle activity plot was then calculated to quantify muscle activity.[8] To be consistent with current definitions, the assumption was that greater spasticity was associated with greater electromyographic (EMG) activity.

As force (in Newtons) (applied to produce the displacement), range of movement (in degrees) and duration of displacement (in seconds) were measured, it was possible to quantify stiffness (as Newtons/degree) and velocity (as degrees/second). The angle versus force data were also presented as an XY scatter plot to determine the stiffness (resistance to passive extension) of muscle. The resistance to passive extension was calculated as the slope of the force angle curve between 10–90% available range of movement using standard linear regression techniques and the coefficient of determination (r^2). Contractures are associated with an increase in stiffness and a reduction in range of movement. Instantaneous velocity for slow and fast movement was calculated using the first difference approximation. From this the 'average velocity' was calculated.

Severity of disability was measured using the Barthel Index (BI).[22] Upper limb functional movement was assessed using the ARAT.[16,17] Pain was measured using a five-point verbal rating scale (ranging from 0 (no pain) to 5 (pain that could not be any worse)).

Statistical methods

Data are reported for the whole group and for two predefined subgroups. Patients who recovered arm function (defined as ARAT score of ≥6) at any time during the study were allocated to the functional group and those who did not were allocated to the non-functional group. The mean and the standard error (standard deviation divided by square root of the sample size) were used to summarize the results at each time point.

Change over time within the sample and the respective subgroup was studied (using the Friedman's test). The differences between the functional group and non-functional group were studied using the Mann–Whitney *U* test. Mean differences and 95% confidence intervals (CI) are reported where appropriate. In addition, the change over time was analyzed using an approach recommended by Matthews *et al.*[23] All the statistical procedures were carried out using SPSS version 15.

The approach recommended by Matthews *et al.*[23] is briefly described below. The change in each individual was modelled using a method of linear regression ($y = a + bx$), minimizing for least square error, with the outcome measure as the dependent variable (y) and time (in weeks) of measurement as the independent variable. The slope (b) from this equation was used to quantify change over time. The comparisons between slopes of the functional and non-functional groups were studied using the Mann–Whitney *U* test.

Results

Thirty patients were eligible (13 males and 17 females) to participate in the study. The median age was 70.5 years (range 52–90) and the median time from stroke onset was 3 weeks (range 1–5). Fourteen (47%) patients had right and 16 (53%) patients had left hemiparesis. Twenty (67%) patients had TACS, 8 (26%) PACS and 2 (7%) LACS. The baseline characteristics for individual groups are presented in Table 1.

The descriptive data obtained from the whole group analysis are presented in Table 2. There was a significant decrease in the passive range of movement ($P<0.01$) (Table 2). The mean rate of decrease in passive range of movement was −0.5 degrees/week (95% CI = −0.9 to −0.16). There was no significant increase in resistance to passive movement ($P>0.1$) (Table 2). The mean rate of increase in joint stiffness was 0.002 N/degrees/week (95% CI = 0 to 0.005). There was no significant change in the EMG activity during a slow or fast stretch ($P>0.1$) over the study period (Table 2). The testing protocol was carried out as planned (i.e velocity during the fast movement was always faster than the slow movement). The mean difference in the average velocity over the study period was 76 degrees/s (SD = 39; range = 10–190). There was a significant increase in pain ($P=0.01$) (Table 2), the mean rate of increase was 0.1 units/week (95% CI = − 0.01 to 0.3). There was significant increase in the BI ($P<0.01$) (Table 2), the mean rate of improvement was 0.2 units/week (95% CI = 0.15 to 0.27).

The descriptive data obtained from the subgroup group analysis (i.e. with the group split as functional and non-functional) are presented in Table 3. Out of 30 control subjects, five subjects had recovered arm function by the end of the study and 25 did not. The 95% CI showed that between 7% and 34% of people who had no arm function at 6 weeks after a stroke, are likely to start recovering within 12 to 24 weeks after a stroke.

In the functional group, both the passive range of movement and stiffness did not change significantly ($P>0.1$) (Table 3). The mean rate of increase in passive range of movement was 0.9 degrees/week (95% CI = −0.06 to 1.77) and stiffness was <0.0001 N/degrees/week (95 % CI = −0.002 to 0.001). However, in the non-functional group the passive range of movement deteriorated significantly ($P<0.01$) but stiffness did not change significantly ($P>0.1$). The mean rate of decrease in passive range of movement was −0.8 degrees/week (95% CI = −1.1 to −0.49) and mean rate of increase in stiffness was 0.002 N/degrees/week (95 % CI = 0 to 0.005).

The EMG activity during slow and fast stretches remained unchanged over time in both the functional and non-functional groups ($P>0.1$) (Table 3). The mean rate of change of EMG activity in the functional group during both stretches was 0.02 mV/week (95% CI = −0.03 to 0.07). The mean rate of decrease of EMG activity in the non-functional group was −0.01 mV/week (95% CI = −0.03 to 0.07) and −0.08 mV/week (95% CI = −0.2 to 0.006) respectively. Abnormal muscle activity was evident in 29 out of 30 (24/25 in the non-functional group and 5/5 in the functional group) patients at recruitment. At the end of the study abnormal activity was seen in 28 of the 30 patients (23/25 in the non-functional group and 5/5 in the functional group).

Table 1 Baseline characteristics of the study group (30 patients)

Characteristics	Non-functional group ($n=25$)	Functional group ($n=5$)
Gender (male:female)	11 : 14	2 : 3
Side of body affected - Left : Right	15 : 10	1 : 4
Median age in years (range)	70 (52–88)	78 (67–90)
Median time post stroke in weeks (range)	3.0 (1–5)	4.0 (2–5)
Oxfordshire Community Stroke Project Classification System		
Total anterior circulation syndromes (TACS)	17	3
Partial anterior circulation syndrome (PACS)	7	1
Lacunar syndrome (LACS)	1	1
Posterior circulation syndrome (POCS)	0	0

Table 2 This table shows a summary of results for the whole group, where mean+/− standard error (SE) is used to describe the data. Friedman's test was used to determine significant differences in the group

Outcome measure	wk 0 M (SE)	wk 6 M (SE)	wk 12 M (SE)	wk 24 M (SE)	wk 36 M (SE)	*P*-value for the change over time	Mean slope (i.e. b) (95%CI)
PROM at slow stretch	99.0 (3.6)	79.6 (4.6)	77.2 (3.7)	72.5 (4.9)	75 (5.3)	<0.01	− 0.5 deg/wk (−0.9 to −0.16)
Stiffness at slow stretch	0.047 (0.12)	0.08 (0.02)	0.05 (0.02)	0.08 (0.02)	0.13 (0.04)	0.14	0.002 N/deg/wk (−0.0001 to 0.005)
EMG at slow stretch	1.1 (0.19)	0.99 (0.24)	0.85 (0.16)	0.75 (0.95)	0.87 (0.15)	0.68	− 0.08 mV/wk (−0.2 to 0.006)
EMG at fast stretch	1.2 (0.21)	1.1 (0.23)	0.95 (0.2)	0.78 (0.1)	1.0 (0.16)	0.36	− 0.01 mV/wk (−0.3 to 0.07)
Pain	0.43 (0.18)	1.4 (0.29)	1.3 (0.29)	1.2 (0.28)	1.1 (0.29)	0.01	0.1 units/wk (−0.01 to 0.3)
Barthel Index range: 0–20	2.6 (0.55)	6.5 (0.93)	8.2 (1.0)	9.5 (1.1)	10.3 (1.2)	0.00	0.2 units/wk (0.15 to 0.27)

CI, confidence interval; EMG, electromyography; deg, degree; mV, millivolts; N, Newton; PROM, passive range of movement; Wk – week.

Pain remained unchanged in the functional group ($P>0.1$), while it significantly increased in the non-functional group ($P=0.01$) (Table 3). The mean rate of change of pain was 0.003 units/week (95% CI = 0 to 0.003) in the functional group and 0.12 units/week (95% CI = −0.01 to 0.03) in the non-functional group. The BI significantly increased in both the groups ($P<0.01$), the mean rate of improvement was 0.4 units/week (95% CI = 0.3 to 0.5) in the functional group and 0.2 units/week (95% CI = 0.1 to 0.2) in the non-functional group. The mean rate of improvement was 1 unit/week (95% CI = 0.5 to 1.6) in the functional group and 0.03 units/week (95% CI = 0.01 to 0.04) in the non-functional group. Out of the five patients in the functional group, three recovered some arm function by week 6 and the remaining two between week 6 and week 12 (Figure 1).

Discussion

Spasticity was quantified using passive testing protocols, in a way congruent to current understanding of spasticity.[6,8,11] Almost the entire sample, even those who recovered arm function, demonstrated signs of spasticity at all time points of measurement. The presentation of spasticity varied with time. All those who recovered function always showed some form of position-dependent spasticity.[8] The data suggest that spasticity, as measured using passive testing protocols, may not interfere with recovery of useful functional movement contrary to the general perception that it does.[10]

The functional group, demonstrating position-dependent spasticity showed an increase in muscle activity as the muscles were passively stretched and even continued when the movement was stopped (at end range of movement). It has been previously hypothesized that the position-dependent spasticity may be a marker for activity in the long latency cortical pathways[8] and, if true, then one possible reason for functional recovery may be the existence of activity in the pathways connecting the muscles of the arm to the cortex. If this can be proved then position-dependent spasticity, early after stroke, may be a prognostic marker for functional recovery. Further research needs to be conducted to verify this hypothesis.

Changes consistent with contracture formation were observed in the study population as a whole. Contractures mainly developed in those who did not recover arm function and were not evident in those who recovered function. Significant reduction in passive range of movement was seen prior to observing increase in joint stiffness. Contractures were completely established between 6-weeks and 12-weeks following a stroke despite the subjects receiving routine treatment.

Table 3 This table shows a summary of the results for individual groups, where mean+/− standard error (SE) is used to describe the data. Friedman's test was used to determine significant differences in each group. Mann–Whitney U test was used to determine significant differences between the functional (F) and non-functional (NF) group over time and also for comparing the slopes between groups

Outcome measure	Group NF = 25 F = 5	Wk 0 M(SE)	Wk 6 M (SE)	Wk 12 M (SE)	Wk 24 M (SE)	Wk 36 M (SE)	*P*-value for change over time	Mean slope (i.e. b0) (95% CI)
PROM at slow stretch	NF	100.3 (4)	80.8 (4.8)	74 (4.1)	65.1 (4.4)	67.7 (5.3)	<0.01	−0.8 deg/wk (−1.1 to −0.49)
HV: better movement	F	92.9 (9.5)	73.7 (15)	93.1 (5.1)	110 (7.6)	112 (2.4)	0.12	0.9 deg/wk (−0.06 to1.77)
LV: worse movement	*p-value comparing groups*	*0.55*	*0.67*	*0.03*	*0.01*	*0.00*	Not applicable	*0.00*
Stiffness at slow stretch	NF	0.05 (.02)	0.08 (.02)	0.06 (.02)	0.09 (.03)	0.15 (.05)	0.12	0.002 N/deg/wk (0.000 to 0.005)
LV: better movement	F	0.046 (.01)	0.09 (.03)	0.025 (.02)	0.03 (.03)	0.04 (.02)	0.3	−0.0006 N/deg/wk (−0.002 to 0.001)
HV: worse movement	*p-value comparing groups*	*0.55*	*0.50*	*0.50*	*0.40*	*0.28*	Not applicable	*0.12*
Stiffness at fast stretch	NF	0.07	0.08	0.10	0.12	0.2	0.00	0.002 N/deg/wk (0.00 to 0.008)
LV: better movement	F	0.05	0.05	0.09	0.09	0.01	0.8	0.002 N/deg/wk (−0.005 to 0.001)
HV: worse movement	*p-value comparing groups*	*0.66*	*0.70*	*0.66*	*0.12*	*0.00*	Not applicable	*0.5*
EMG at slow stretch	NF	1.1 (0.2)	0.97 (0.3)	0.73 (0.2)	0.74 (0.2)	0.7 (0.1)	0.9	0.01mV/wk (−0.03 to 0.07)
HV: more muscle activity	F	1.1 (0.4)	1.1 (0.5)	1.4 (0.6)	0.82 (0.2)	1.7 (0.6)	0.6	0.02mV/wk (−0.03 to 0.07)
LV: less muscle activity	*p-value comparing groups*	*0.96*	*0.60*	*0.25*	*0.60*	*0.03*	Not applicable	*0.4*
EMG at fast stretch	NF	1.2 (0.3)	1.1 (0.3)	0.9 (0.2)	0.7 (0.1)	0.8 (0.1)	0.5	− 0.08mV/wk (−0.2 to 0.006)
HV: more muscle activity	F	1.0 (0.4)	1.3 (0.6)	1.3 (0.7)	1.1 (0.3)	1.9 (0.7)	0.6	0.02 mV/wk (−0.03 to 0.07)
LV: less muscle activity	*p-value comparing groups*	0.83	0.90	0.60	0.20	0.07	Not applicable	*0.2*
Pain	NF	0.52 (0.3)	1.7 (0.3)	1.4 (0.3)	1.4 (0.3)	1.3 (0.3)	0.01	0.12 units/wk (−0.01 to 0.03)
LV: improved	F	0.0 (0.0)	0.0 (0.0)	0.8 (0.8)	0.0 (0.0)	0.2 (0.2)	0.4	0.003 units/wk (0 to 0.003)
HV: worsened	*p-value comparing groups*	0.4	0.02	0.3	0.05	0.2	Not applicable	*0.7*
Barthel Index range: 0–20	NF	2.8 (0.6)	5.8 (1.0)	7.1 (1.0)	7.9 (1.2)	8.6 (1.3)	0.00	0.2 units/wk (0.1 to 0.2)
HV: better functionality	F	1.4 (1.4)	10.0 (2.1)	13.6 (1.9)	17.2 (0.58)	18.4 (0.50)	0.00	0.4 units/wk (0.3 to 0.5)
LV: worse functionality	*p-value compare groups*	*0.20*	*0.12*	*0.03*	*0.00*	*0.00*	Not applicable	*0.00*

CI, confidence interval; deg, degree; EMG, electromyography; F, functional; HV, higher values; LV, lower values; mV, millivolts; N, Newton; NF, non-functional; PROM, passive range of movement; wk, week.

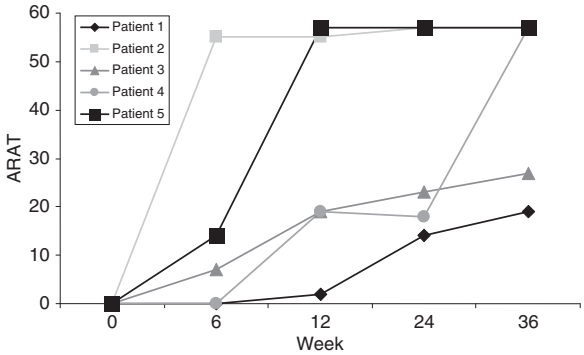

Figure 1 A summary of time course of change in the upper limb function (ARAT, action research arm test) in all 5 patients of the F group.

The people who developed contractures had both spasticity and no function. From first principles, the primary hypothesis would suggest that immobilization caused due to lack of functionally useful movement was the most likely cause of contractures.[8,11,24] Spasticity may not have contributed to contracture formation, as people who developed arm function did not develop contracture. The one anomaly in this study was a patient in the functional group who appears to have developed stiffness despite not losing range of movement. The most probable cause for the increase in stiffness is likely to be reduced use of the hand or oedema but more work is required to explore this behaviour.

Although less likely, contracture formation may be dependent on pain as the pain profiles differed between the groups and pain significantly worsened in the non-functional group. Pain can be a barrier to active movement and this loss of movement could exacerbate the formation of contractures. This would further encourage fixed positioning and thereby lead to the formation of contractures.

This is a novel study exploring the time course of development of both spasticity and contractures, but it lacks statistical power. The sampling frame was limited to a homogenous sample that was not fully representative of the stroke population; however, this was intentional as there is evidence that people who show early signs of functional recovery get better naturally,[11] so there was a need to explore the time course of change associated with the two significant barriers of recovery in stroke. For findings to be generalizable, a more comprehensive longitudinal study is required.

The 15 patients who were unable to complete the assessments may have demonstrated different patterns with respect to functional recovery, spasticity and contractures. It was not possible to identify what was different in the people who regained function when compared to those who did not. The lack of premorbid data on status of joints was also likely to be a confounding factor in this study. It was not possible to confirm whether those who recovered function had joints that were normal nor was it possible to confirm if those who developed contractures had pre-existing problems that exacerbated the formation of contractures. Incorporating information on premorbid status in any prospective longitudinal study will be recommended.

Two methods were used to analyse the repeated measure data. The application of Matthews *et al.*[23] approach in studying time course of change in stroke is relatively new. This method[23] is superior, as there is a possibility that some of the serial measures in stroke-related impairments are not strictly independent and the data can be analyzed in a way that are appropriate to the question. A further advantage of the Matthews *et al.*[23] approach is that repeated serial measures can be reduced to a single variable that can then be analysed using a single test – this is likely to reduce errors associated with multiple comparison. Even though it might be more labour intensive, it is recommended for future use.

EMG can vary over time but the consistency within the data would suggest that the signal to noise ratio is sufficiently high so as not to change the interpretation. Therefore, despite the limitations, the key findings need to be considered within clinical practice.

Clinical messages:

- Spasticity appears not to be a barrier to functional recovery.
- Wrist contractures develop rapidly after a stroke.
- Loss of function, and not spasticity, may be the primary contributor to contracture formation.

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