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Contractures in the post-stroke wrist: a pilot study of its time course of development and its association with upper limb recovery

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Background: Contractures are common in a stroke population, yet there is little information on the time course of development.
Objectives: Investigate quantitatively changes associated with contracture formation in an acute stroke population.
Study design: Longitudinal study on 22 subjects who were 2–4 weeks post stroke.
Outcome measures: Contractures were assessed by quantifying the resting posture, resistance to passive movement and passive range of movement. Upper limb function was measured using the Action Research Arm Test and the Nine-Hole Peg Test. Active range of extension, wrist extension strength (isometric), grip strength and neglect were also measured.
Repeated measures: Following an initial assessment, repeated measurements were taken at 4, 8, 20 and 32 weeks after recruitment.
Results: Two distinct subgroups, one capable of some functional movement (F group; 8 subjects) and another which was not (NF group; 14 subjects), were identified at the start of the study. The NF group showed changes associated with contracture formation at the wrist, i.e., reduction in the passive range of movement, an increase in resistance to passive movement and a worsening of the flexion posture. Changes were observed from the time of recruitment even though neglect improved. The F group showed improvements in upper limb function and there was no evidence to support contracture formation.
Conclusions: Subjects most prone to contracture formation were those who showed no signs of early functional recovery (2–4 weeks after the stroke). Changes consistent with adaptive shortening were seen from week 4 of the study period.

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Introduction

Stroke is a major cause of disability in the developed world.[1,2] Upper limb disabilities adversely affect the quality of life in more than half this population in spite of intense therapeutic efforts.[3–5] Motor deficits following a stroke have often been associated with contractures and spasticity (as defined by Lance[6]).[7–10] The joints most prone to contractures are the wrist and ankle,[11] with upper limb contractures being more prevalent.[7] Although contractures are reported to be common,[7–11] there is no information on its time course of development and incidence in a stroke population.

In general, prolonged immobilization of a joint in a shortened position causes contractures.[10] In the stroke population spasticity is also reported to be a cause of contractures[8–10] but evidence supporting this claim is, at best, tenuous.[12,13] Other suggested causes of immobilization are paresis, rigidity/spastic dystonia, neglect and pain.[3,13–18] However, the association between these factors and contracture formation is unclear.

The aim was to identify the time course of contracture formation at the wrist in an acute stroke population, and to study its relationship to changes in spasticity, neglect, pain, and motor and functional recovery. The hypothesis was that poor upper limb functional recovery would lead to the development of wrist flexion contractures.

Methods

Subjects for this longitudinal study were selected from the control group of a randomized controlled trial investigating the effects of electrical stimulation at the wrist.[20] Inclusion criteria were hemiparesis due to a unilateral stroke between two and four weeks previously, power of wrist extension in the impaired arm grade 4/5 or worse on the MRC scale (Medical Research Council Scale[19]), and no previous wrist problems.[20] Patients with impaired cognition were excluded.[20] Contractures, spasticity, neglect, pain, motor and functional recovery were quantified. Measurements were taken on the day of recruitment (2–4 weeks following the stroke) and were repeated at 4, 8, 20 and 32 weeks from this date. Twenty-two subjects who had not missed any of the assessment sessions were selected from the control group of 30 subjects. No other selection criteria were used.

Outcome measures

Wrist flexion contractures were characterized by measuring changes in the posture, the resistance to passive extension and the passive range of movement at the wrist, using a custom-built system.[21] When quantifying contractures the assessor ensured that the velocity of passive movement was less than 10 degrees/s using an on-line velocity display.[13] The following procedure and outcome measures were used:

- The forearm and wrist were positioned in the measuring system[21] with the wrist in its natural resting posture and the forearm in mid pronation–supination. The wrist was passively extended to neutral and released to reach a new resting position. This procedure was repeated three times. The final resting position of the wrist was measured as the resting posture. If this was negative the wrist was flexed. The maximum moment used to extend the wrist to neutral during the third stretch was used as a measure of resistance to passive movement.
- The wrist was then passively extended, within a pain-free range of movement, from full flexion to maximum possible extension. This was repeated three times. Passive range of movement and the maximum moment used to extend the wrist were measured during the third repetition.

The modified Ashworth Scale[22] was used to grade the resistance to a rapid stretch.[23] The presence or absence of pain was measured using a vertical visual-analogue scale and then converted to a dichotomous variable (i.e., '1' subject had pain; '0' subject did not have pain) as these scales have limited reliability in quantifying the intensity of pain over time.[24] Neglect was quantified using the Star Cancellation Test.[25] Although attempts to measure oedema were made the results were discarded due to limitations in the measurement procedures.[6,13]

Motor recovery was quantified by measuring

active movement and strength using the best of three repeated measures (a 15-second rest was given between repeated measures).

- Active range of extension and isometric wrist extension strength (with the wrist in its natural resting position and forearm in mid pronation–supination) were measured using the custom-built measuring device.[21]
- Grip strength was measured using a JAMAR Hand Dynamometer (JAMAR, JA Preston Co., Michigan, USA) with minimum elevation at the shoulder, elbow flexed at 90 degrees, forearm in mid pronation–supination, and wrist in neutral. The dynamometer was fully supported by the assessor to enable consistent positioning.

Upper limb function and dexterity was measured using the Action Research Arm Test (ARAT)[27] and a normalized Nine-Hole Peg Test score (NHPT; see Appendix),[28,13] respectively.

Subgroup analysis

In order to investigate the association between contracture formation and functional recovery the study population was divided into two groups. Subjects with some functional movement, at the time of recruitment, were identified and assigned to an F group using the following rule:

Pick up and place at least one peg in the Nine-Hole Peg Test (NHPT >0)
and
Pick up and reach out with a wooden cube (1 cm^3) in the ARAT (grasp subscore >0).

A person with minimal functional recovery, not just return of active movement, was expected to succeed in completing both tasks. All other subjects were assigned to an NF group.

Data analysis

The mean and standard error were used to summarize the results. As the study population was small and many of the tests provided, at best, an ordinal level of measurement (ARAT, NHPT, modified Ashworth Scale, Star Cancellation Test, and Pain) the Friedman's test was used to determine if changes occurred over the study period. The Mann–Whitney *U*-test was used to determine if any differences existed between the groups at the start of the study (week 0). All statistical procedures were carried out using SPSS for Windows (version 10.0).

Results

Ten male and 12 female subjects (mean age 64.9 years (range 40–93); mean time post stroke 22.7 days (range 14–34)) were selected. Fourteen subjects had strokes that affected the left upper limb and eight the right. All patients had CT brain scans. Two were classified as intracerebral haemorrhage and 20 as cerebral infarction. Five were classified as total anterior circulation syndrome (TACS), 11 as partial anterior circulation syndrome (PACS), four as lacunar syndrome (LACS).[29] All subjects received routine medical treatment and therapy.

Over the study period, in the group as a whole, the resting posture of the wrist worsened (i.e., became more flexed) and the resistance to passive movement increased significantly ($p < 0.01$) but the passive range of movement did not ($p > 0.1$). The modified Ashworth score increased significantly ($p < 0.01$). Neglect tended to improve ($p = 0.064$). Half the population had pain at the start of the study and there was no significant change ($p > 0.1$) over the study period. The active range of extension and wrist extension strength showed no significant change ($p = 0.064$ and $p > 0.1$). Grip strength increased significantly ($p < 0.01$). The NHPT and total ARAT scores improved significantly ($p < 0.01$).

Subgroup analysis

Eight subjects had some function at the time of recruitment (F group) and 14 did not (NF group). In the NF group, one subject regained some function at week 4 and another at week 8. The early functional gains made by the former were lost at the end of the study.

At the start of the study, the resting posture and the passive range of movement were identical in both groups (Table 1) but the resistance to passive movement was higher in the NF group (Table 1). In the F group, the resting posture, the resistance to passive movement and the passive range of movement did not change significantly over the study period (Table 1). However, in the NF group, over the same time period, the resting

posture became more flexed, the resistance to passive movement increased and passive range of movement tended to decrease (Table 1). (Note: The moment required to maximally extend the wrist did not show any significant change over the entire study – Table 1.)

At the start of the study, the modified Ashworth score and neglect were significantly higher in the NF group (Table 1). The modified Ashworth score subsequently increased and neglect decreased in this group (Table 1). These scores did not change significantly in the F group (Table 1). The proportion of subjects with pain in both groups did not change over the study period ($p > 0.1$).

At the start of the study, active range of extension was significantly better in the F group and it increased significantly (Table 2). These subjects were capable of extending the wrist and fingers appropriately when asked. Subjects in the NF

Table 1 A summary of the results from the measures used to quantify contractures, modified Ashworth score and neglect. Although the *M*±SE were used to describe the data the Friedman's test was used to test for significant differences

Outcome measure	Group	Week 0 *M*(SE)	Week 4 *M*(SE)	Week 8 *M*(SE)	Week 20 *M*(SE)	Week 32 *M*(SE)	*p*-value
Resting wrist angle (degrees) was used to measure the resting posture of the wrist	F	–2.0 (1.5)	–0.4 (1.2)	–1.5 (1.8)	–5.0 (2.2)	–0.6 (0.7)	0.068
	NF	–6.8 (2.7)	–9.4 (3.5)	–10.5 (3.8)	–16.4 (3.9)	–16.1 (4.3)	<0.01
Moment to extend wrist to neutral (N m) was used to measure resistance to passive movement	F	0.1 (0.0)	0.1 (0.0)	0.2 (0.1)	0.2 (0.1)	0.1 (0.1)	>0.1
	NF	1.0* (0.5)	0.9 (0.4)	1.3 (0.8)	1.9 (0.8)	1.7 (0.8)	0.015
Passive range of movement (degrees)	F	56.9 (3.0)	59.3 (3.6)	51.3 (4.3)	61.3 (4.1)	59.6 (2.9)	<0.1
	NF	60.1 (4.8)	50.0 (5.7)	46.0 (6.5)	41.8 (6.8)	46.4 (6.7)	0.061
Moment to maximally extend (N m)	F	4.0 (0.5)	4.0 (0.5)	4.2 (0.6)	5.0 (0.7)	4.3 (0.4)	<0.1
	NF	5.1 (0.7)	4.3 (0.5)	4.2 (0.5)	4.3 (0.7)	4.8 (0.6)	>0.1
Modified Ashworth Score was used to quantify resistance to a brisk stretch	F	0.4 (0.2)	0.5 (0.2)	0.4 (0.2)	0.3 (0.2)	0.4 (0.3)	>0.1
	NF	1.6* (0.3)	1.9 (0.3)	2.3 (0.3)	2.6 (0.3)	2.6 (0.3)	<0.01
Star Cancellation Test was used to quantify neglect[a]	F	53.4 (0.3)	53.4 (0.4)	53.6 (0.2)	52.6 (0.6)	52.6 (0.8)	>0.1
	NF	30.0* (5.7)	40.6 (5.0)	37.6 (5.6)	42.8 (4.6)	39.6 (5.0)	<0.01

M, Mean; SE, standard error; F, subgroup some functional movement; NF, subgroup no functional movement.
*Significant differences between F group and NF group at week 0 ($p < 0.01$).
[a]Non-stroke-related visual and auditory impairments and poor compliance resulted in the three missing values.

Table 2 A summary of the results from the measures used to quantify motor and functional recovery. Although the *M*±SE were used to describe the data Friedman's test was used to test for significant differences

Outcome measure	Group	Week 0 *M*(SE)	Week 4 *M*(SE)	Week 8 *M*(SE)	Week 20 *M*(SE)	Week 32 *M*(SE)	*p*-value
Active range of wrist extension (degrees)	F	36.3 (3.4)	42.6 (4.7)	35.5 (3.5)	42.0 (2.9)	49.1 (3.6)	<0.01
	NF	−0.3* (5.5)	−1.5 (5.9)	−0.3 (6.2)	−6.2 (6.3)	−5.8 (6.1)	>0.1
Active wrist extension strength (isometric) (N m)	F	2.9 (1.0)	4.0 (1.0)	4.1 (1.0)	4.9 (0.9)	4.6 (1.0)	0.012
	NF	0.4* (0.2)	0.5 (0.2)	0.3 (0.2)	0.2 (0.1)	0.2 (0.1)	0.059
Grip strength (kg)	F	10.6 (2.3)	15.6 (3.9)	17.6 (3.5)	20.4 (3.9)	22.1 (3.8)	<0.01
	NF*	0.6* (0.0)	1.4 (0.8)	1.1 (0.6)	2.2 (0.8)	2.4 (1.0)	0.057
NHPT	F	0.39 (0.1)	0.70 (0.1)	0.76 (0.1)	0.84 (0.1)	0.88 (0.0)	<0.01
	NF	0.00* (0.0)	0.00 (0.0)	0.00 (0.0)	0.00 (0.)	0.01 (0.0)	>0.1
ARAT – total	F	36.4 (5.2)	45.0 (3.6)	49.0 (3.2)	53.6 (2.1)	53.3 (2.5)	<0.01
	NF	0.6* (0.6)	3.0 (2.0)	4.1 (2.7)	3.7 (2.6)	3.1 (2.3)	0.037

M, mean; SE, standard error; F, subgroup functional movement; NF, subgroup no functional movement.
*Significant differences between F group and NF group at week- 0 ($p < 0.01$).

group showed no significant improvement in the active range of extension over the study period even though some were capable of active, yet uncontrolled, movement (Table 2 – the negative values suggest that many subjects were flexing the wrist and fingers when asked to extend). The F group was also capable of generating a larger wrist extension force and grip force at the start of the study (Table 2). Grip strength increased significantly in both groups (Table 2). Wrist extension strength improved in the F group and tended to deteriorate in the NF group (Table 2).

Subjects in the F group were all able to attempt the NHPT and they showed significant improvements over the study period (Table 2). The ARAT consists of four subtests, i.e., grasp, grip, pinch and gross movement. In all but one of the subtest, i.e., 'gross movement', the increase in the ARAT scores was exclusively due to an increase in the scores of the F group. In the 'gross movement' section of the ARAT, only subjects in the NF group showed significant improvement. The total scores are reported in Table 2.

Discussion

Prolonged immobilization of a joint, in a shortened position, results in contracture formation.[8–10,13–15,30,31] Therefore, the study population was divided into two groups based on signs of early functional recovery, the rationale being that subjects showing functional recovery would be more mobile and hence less prone to joint fixa-

Clinical messages

- Wrist flexion contractures develop rapidly (within 6–8 weeks post stroke) in subjects who have no early functional upper limb recovery.
- Strategies designed to prevent contractures should focus on this high-risk subgroup and begin early in rehabilitation (within four weeks of the stroke).
- Intervention aimed at preventing contractures will need to incorporate protocols involving movement about the joints and may need to be continued until functional recovery occurs.

tion in a shortened position. The subgroup analysis identified two distinct groups within the study population. The group profiles did not alter significantly over the study period.

Analysis of results from the study population as a whole suggested that contractures were worsening in spite of significant improvements in function. As a result of this ambiguity, which could be explained by the influences of the NF group on contractures and F group on function respectively, the discussion that follows will focus on the results from the subgroup analysis.

Changes consistent with developing wrist flexion contractures were seen in the NF group. The increase in resistance to passive movement was seen prior to observing changes in the wrist posture and range of movement.[11] The changes appeared within 6–8 weeks following the stroke, despite subjects receiving routine treatment. These changes are consistent with models of contracture formation based on animal experiments.[10,18,30,31] The most likely cause for contractures may have been immobilization caused by loss of functional movement and paresis[31] as similar changes were not observed in the F group.[13,20,21,31]

Although spasticity (as measured by the modified Ashworth Scale) increased, it may not have influenced contracture formation at the wrist. Spasticity is a dynamic condition that is not associated with fixed positioning of the limb segments[23,32] or loss of functional movement.[23,33–36] It is also possible, due to inherent limitations in this outcome measure, that the changes in the modified Ashworth score may have resulted from changes in the soft tissue and joint compliance associated with developing contractures.[23] Rigidity/spastic dystonia could have caused fixed positioning of the limb segments in a shortened position and accelerated contracture formation.[18,32] These may have also influenced the outcome measures used to quantify contractures.

Contracture formation may have been independent of changes in neglect as this improved in the NF group. The influence of pain on contractures still remains unclear as there were no differences in the pain profiles between both groups.

Functional improvements in the F group supports the findings that signs of early functional recovery is a predictor of long-term functional recovery.[39] The improvements in grip strength and 'ARAT – Gross Movement subtest', seen in the NF group, may have resulted from the reinforcement of synergistic movement patterns in the upper limb.[11,40]

This was a small sample study in which the EMG activity from the wrist flexors and extensors was not measured or monitored. Therefore the effects of muscle tone on contractures was not quantified.[8–10,32]

Conclusion

This study demonstrates that subjects most prone to wrist flexion contractures were those who showed no upper limb function within four weeks of their stroke and that contractures could develop rapidly. The most probable cause of contractures, despite some subjects showing signs of motor recovery and receiving routine therapy, may have been immobilization resulting from reduced functional use. Rigidity, if present, could also have significantly influenced contracture formation. It was not possible to comment on the influence of spasticity on contractures and contracture formation appeared to have been independent of pain and neglect. Further work is now required to elucidate these relationships.

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Appendix – Procedure used to calculate the normalized Nine-Hole Peg Test (NHPT) score

The subjects were given 120 s ($T = 120$) to place all 9 pegs ($P = 9$) in the slots. The number of pegs placed correctly in the given time was N.

Scaled NHPT score $NHPT_s = (N \div P) + ((T-t) \div T)$

The nonimpaired population is expected to complete the test in 18 s ($N = 9$ and $t = 18$ s).[28]

$\therefore$ $NHPT_s$ for nonimpaired subject = $NHPT_{ni} = 1.85$

Normalized NHPT score = NHPT = $NHPT_s \div NHPT_{ni}$

If the $NHPT_{ns} = 0$ a subject could not attempt the test.
If the $NHPT_{ns} \geq 1$ a subject could complete the test as fast as or faster than a nonimpaired person.