Article title:

Amount of therapy matters in very early aphasia rehabilitation after stroke: A clinical prognostic model.

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Conflict of Interest Statement
None declared.

Abstract

Background and Aim

The effects of very early aphasia therapy on recovery are equivocal. This paper examines predictors of very early aphasia recovery through statistical modelling.

Methods: This study involved a secondary analysis of merged data from two randomized, single-blind trials conducted in Australian acute and subacute hospitals. Study 1(N=59) compared daily therapy to usual ward care (UC) for up to four weeks post-stroke, in patients with moderate-severe aphasia. Study 2(N=20) compared daily *group* therapy to daily *individual* therapy for 20 one-hour sessions over five weeks, in patients with mild-severe aphasia. The primary outcome measure was the WAB Aphasia Quotient (AQ) at therapy completion.

This analysis used regression modelling to examine the effects of age, baseline AQ and baseline modified Rankin Scale (mRS), average therapy amount, therapy intensity and number of therapy sessions on aphasia recovery.

Results: Baseline AQ (p=.047), average therapy amount (p=.030) and baseline mRS (p=.043) were significant predictors in the final regression model, which explained 30% (p<.001) of variance in aphasia recovery.

Conclusion: The amount of very early aphasia therapy could significantly affect communication outcomes at 4-5 weeks post stroke. Further studies should include amount of therapy provided to enhance reliability of prognostic modelling in aphasia recovery.

Keywords:

Very Early Rehabilitation, aphasia, stroke.

Background

Aphasia is a devastating condition that affects hundreds of thousands of stroke survivors around the world each year[1-3]. Aphasia has well known negative impacts on the stroke survivor, family and community. These include increased mortality[4] and reduced functional outcomes[5], higher levels of depression and lower return to work[6] rates when compared to stroke survivors without aphasia[7]. Predicting aphasia recovery after stroke and the effect of aphasia on stroke recovery have long been the focus of discussion in the literature.[1-5] Variability in the nature, severity and type of post-stroke aphasia[1] and subsequent enormous variability in the amount, speed and extent of recovery that occurs in the months following stroke[8] makes selecting the most appropriate amount, type and timing of aphasia therapy treatment for individuals with aphasia a difficult task.

Complete aphasia recovery is reported to range from 11-74%[2,3,8,9] of cases depending on the sensitivity of the outcome measure used and the length of recovery time post-stroke. Predicting aphasia recovery after stroke has received renewed attention in recent literature with papers examining early aphasia diagnosis and the factors believed to affect recovery, whilst providing a description of natural language

recovery across time,[2,3] using patient performance on language and cognitive tests to predict therapy gain in the chronic recovery phase[5,8,10] and providing a comprehensive literature review of the variables considered important in aphasia recovery.[11]

The ability to more reliably predict improved aphasia outcomes in individual stroke survivors may allow the benefit of limited aphasia rehabilitation services in clinical settings to be maximised. Predictors of aphasia recovery including vascular risk factors, stroke type, infarct volume/size and location, initial aphasia severity and type, age, gender, handedness, level of education, performance on language assessments and intelligence have all been investigated in an attempt to determine both stroke[4,5] and aphasia outcomes[1-3,8-10] at various points in the recovery journey. To date, however, there are no stroke related or personal characteristics that reliably predict aphasia recovery in an individual[8] and a combination of the above factors has only accounted for between 32 and 41% of variability in recovery[8]. This suggests that other factors, critical in recovery, have not been accounted for, potentially including the type and timing of aphasia treatment.

There remains a dearth of evidence around large-scale best-practice aphasia intervention. The authors of the recent Cochrane Review of Speech and Language Therapy following Stroke,[12] concluded there was “some benefit of speech and language therapy following stroke in relation to functional communication, reading, comprehension, expressive language and writing”[12(pp 39)]. The authors went on to warn of the dangers of over-interpreting this finding as the results in the meta-analysis were highly dependent on a single trial where there was “very limited information on the nature of the speech and language therapy intervention and the quality of research undertaken”[12(pp 39)]. The results of the Cochrane Review[12] do little to assist speech pathology practice in relation to the everyday application of the right amount of the right type of therapy for the right person at the right time in recovery, particularly in relation to acute care settings. It would be a logical step then, to look for other levels of

evidence to guide clinical practice in aphasia therapy after stroke. It is here that we find an abundance of high quality single-case design[13-15] and small group studies[16-18] to support the provision of different types of aphasia therapy provided at various phases in the recovery journey. The vast majority of this research, however, does not include people with aphasia in the very early (within two weeks) and early (two to six weeks) post-stroke recovery phases (See examples of studies investigating very early and early intervention).[13-18]

Given the constant high demand for limited speech pathology services during (very) early recovery combined with the need to comply with health-funding regulators and the potential for therapy-induced recovery,[19,20] it is crucial that speech pathologists deliver evidence-based efficient and efficacious aphasia therapy interventions. Unfortunately, current evidence based documents used to guide speech and language therapy practices[21-23] in very early and early aphasia intervention after stroke raise more questions than they answer. A prime example of this is the question of "How much direct therapy?" particularly when considering the delicate nature of very early stroke recovery[1,4] and limitations in service delivery that many speech pathology services report.[24]

Positive effects have been found from very early aphasia therapy in two small clinical randomised controlled trials[19,20] and from two trials in early recovery[25,26]. Yet few if any studies have included the amount of aphasia therapy in early aphasia recovery as a possible predictor in models of improved communication outcomes in post-stroke aphasia. This paper specifically examines the role of the amount of very early aphasia therapy within the first five weeks after stroke in predicting aphasia recovery.

Methods

Design

This secondary analysis merged data from two independent randomized,

single-blind controlled trials which were conducted in Perth, Australia. The trials compared communication outcomes for two independent cohorts in the very early post-stroke recovery phase. The primary endpoint for this analysis was at therapy completion, which was a mean of 24 days (SD=12.3) post-stroke. These studies have been previously reported[19,20] and the merged data are outlined below.

Participants

Participants were admitted to an acute care teaching hospital in metropolitan Perth, Western Australia and were recruited to the trials within 14 days following stroke if they met the following criteria:

I. Diagnosed with an acute stroke by a stroke physician or neurologist
II. The diagnosis was confirmed by computer tomography and/or magnetic resonance imaging within 48 hours of hospital admission
III. Medical stability (Glasgow Coma Score > 10)
IV. The patient could remain awake for at least 30 minutes and
V. The patient scored less than 93.8 on the Aphasia Quotient (AQ) of the Western Aphasia Battery (WAB).[27]

Patients were excluded from the trials if they:

I. Had a previous diagnosis of aphasia
II. Were unable to participate in therapy in English
III. Had a mental illness or dementia
IV. Had a previous history of sub-arachnoid and/or sub-dural haemorrhage or
V. Had neurosurgical intervention and
VI. uncorrected hearing or vision impairment.

Assessments and baseline data

The data in this analysis is from the baseline assessment and assessment immediately following treatment. This period ranged from 21 days to 51 days post stroke, depending on the therapy regimen undertaken by each participant. Baseline

participant and stroke characteristics for the 79 participants are outlined in Table 1.

Amount and type of very early aphasia rehabilitation

The amount of intervention provided to the participants in these trials varied greatly and has been outlined in detail[19,20]. All direct therapy was provided by a trained speech and language therapist.

Trial 1 investigated therapy intensity and randomised individuals with moderate to severe aphasia to either daily therapy (DT) or standard ward based usual care (UC) for the duration of their inpatient stay (mean = 22 days). Those participants in the daily therapy group (N = 32)[19] received a mean 7.5 sessions of 45 minutes of therapy over a period of 22 days. Twenty-three of the 27 participants randomised to UC (N = 27)[19] received no therapy during their acute hospital stay (mean = 22 days). The remaining four participants receiving UC in this trial received a collective total of 295 minutes (4.9 hours) over a total of 7 sessions during the 22 days of the intervention period which equates to an average of 11 minutes of therapy.

Trial 2 investigated the nature of very early aphasia therapy by comparing group versus individual therapy provided each day for up to 20 one-hour sessions over five weeks in patients (N=20) with mild-severe aphasia[20]. Participants in this trial were randomised to daily group therapy or daily 1:1 therapy and received a total of 356.75 hours of therapy over 373 sessions. Participants received a mean of 18.65 sessions, which equated to a mean of 57 mins per session. On average, each participant in this trial received a total of 17.71 hours of therapy over a mean of 38.5 days.

Therapy in Trial 1 and 2 aimed to increase verbal production of connected speech and was impairment based. Further description of the therapy provided to participants in each trial is outlined previously.[19,20]

Trial 1: The treating therapists determined which therapy approach would be used and all therapies were provided as per published instructions. The therapies for the daily therapy group and the usual care group were Lexical-semantic (BOX)

therapy,[28] Mapping Therapy[29] and Semantic Feature Analysis (SFA)[30] and adhered to principles of neurorehabilitation, incorporating repetitious trained activity together with facilitation of error-free learning. Additionally, all participants who received therapy in this trial attempted a picture description task at each session during the intervention phase.

Trial 2: Group therapy was based on the Constraint Induced Language Therapy (CILT) outlined by Pulvermuller and colleagues.[16,31] It took place in small groups of 2-4 participants and one speech pathologist who provided language support appropriate to each participant's needs. The stimuli and language support were designed to accommodate all levels of aphasia severity in the same group.

Individual therapy (1:1) was tailored to suit the individual needs of the participant. Based on the participant's assessment results, the treating therapist selected the appropriate therapy from SFA[30], phonological feature therapy[32], or Lexical-semantic (BOX) therapy[28] or Mapping therapy.[29] As per Trial 1,[19] the treating therapists determined which therapy approach would be used and all 1:1 therapies were provided as per published instructions. Participants received either a single therapy, or a combination of therapy types such as cued naming therapy and semantic feature therapy.

Outcome assessment and Primary Outcome Measure

Participants in both trials were assessed by qualified speech language therapists blinded to group allocation. Assessments were completed at acute hospital admission (baseline) and immediately following intervention (between four to five weeks post-stroke). The primary outcome measure was the AQ score of the Western Aphasia Battery (WAB)[27] at therapy completion.

Recording intervention sessions and time

Duration of aphasia therapy was recorded by the treating therapists via the Allied Health System (AHS), a software package which records intervention in five minute units. Therapists recorded the amount of time spent on each intervention with each participant, including all time on communication related issues and on swallowing issues (as appropriate). The data presented here relate only to direct aphasia therapy provided to each participant. The data presented here do not include time spent on assessment, information provision, carer education, discharge planning or any other non-direct speech therapy intervention as described by Leff and Howard.[33]

Average Therapy and Therapy Intensity Measures

Average therapy, the average amount of therapy each participant received, was calculated by dividing the total therapy amount (in minutes) for all participants by the number of days in therapy (study duration). For both studies, the number of days in therapy reflected the total length of stay (seven days per week) regardless of the fact that rehabilitation was provided on five day per week regimen Therapy intensity, calculated by dividing the total therapy amount for all participants by the number of therapy sessions. We believed the average therapy amount and therapy intensity to be potential predictors of aphasia recovery and consequently both were included in the statistical modelling.

Statistical analyses

A linear regression model was developed to analyse the impact of amount and intensity of aphasia therapy on aphasia recovery.

Predictors:

The impact of the amount and intensity of aphasia therapy were investigated by including average therapy amount, therapy intensity and number of therapy sessions as predictors in the model. This included the data from participants who received no

input, as per the Usual Care group in Trial 1. Other predictors added to the model were: baseline AQ to control for initial aphasia severity, the baseline mRS[34] to control for stroke related disability and age as it is thought to be a predictor of aphasia recovery.

Outcome Variable:

The outcome variable for the regression model was the proportion of the potential maximal gain. This measure has previously been used by Lambon-Ralph et al[10] and developed by Lazar et al.[35] who called it percent of maximal achievable recovery. It is obtained by calculating the ratio of the achieved improvement in AQ score to the maximum attainable improvement at baseline. The ratio was then multiplied by 100 to convert it to a percent. This is represented in Figure 1 and is expressed as:

$$\%\mathrm{MPR} = \frac{AQ\ at\ therapy\ completion - Baseline\ AQ}{100 - Baseline\ AQ} \times 100$$

This measure is preferred to raw AQ scores because it accounts for initial aphasia severity and has better statistical properties.[36] Furthermore, it addresses the issue of ceiling effects of raw AQ scores.

Model Development:

The model was developed through a forward selection process. The selection protocol ensured that the predictor with the largest effect on the outcome variable was entered into the model first. Other variables were successively selected into the model based on the size of their effect on the outcome variable, relative to other predictors. The process stopped when entering additional variables to the model did not have a statistically significant impact on the outcome.

The final model obtained through the forward selection process was verified by developing a model using backward elimination. Both forward selection and backward elimination converged to the same final model.

Results

Seventy-nine cases were used in this study. Their baseline stroke and demographic characteristics are shown in Table 1. Nine participants (11%) were not included in the final regression model as they did not complete the end of therapy assessment. Seven of these participants died during the intervention period and two suffered significant stroke related medical complications. The mean (SD) participant age was 69.5 years (14.0); baseline AQ score was 31.7 (27.6) and mRS was 4.03 (.97). The mean (SD) total therapy amount was 392 minutes (468.3). The mean percent of maximal potential recovery for the group was 33.81 (30.95). Seventy cases were loaded into the final model.

The Regression Model:

The forward selection process yielded a regression model which explained 30% of the aphasia recovery (R^2=0.294, p<0.001). Baseline AQ (B=0.29, p=0.047), initial stroke severity (B=-7.5, p=0.043) and average therapy amount (B=0.63, p=0.030) were found to be significant predictors aphasia recovery and were included in the final model. Conversely, therapy intensity, frequency of service and age did not have a significant effect, and did not feature in the final model. Therapy intensity and average therapy amount were highly correlated (r = 0.928, p < 0.001). Therefore, average therapy amount may be considered to be a surrogate measure for therapy intensity.

Explaining the model:

Details of the regression model are presented in Table 3. From a practical perspective, the model may be interpreted from the unstandardized coefficients. The unstandardized coefficient of 0.63 for average therapy amount suggests that after controlling for baseline AQ and initial stroke disability (mRS), a one minute increase in the average therapy amount would result in a 0.63% improvement in aphasia recovery. Equivalently, after controlling for baseline AQ and initial stroke disability, a 10 minute

increase in average therapy amount, can be expected to improve recovery by 6.3%. In other words, if two patients present with the same baseline AQ and the same initial mRS, the patient who receives a higher amount of therapy per day in the very early rehabilitation phase, can be expected to have significantly better recovery. The model predicts that for every 10 minutes increase in therapy per day during the very early recovery period, the patient's prognosis improves by 6.3%. Similarly, after controlling for initial stroke related disability, and average therapy amount, a person who scored 10 points better on the WAB AQ at baseline had 2.9% better prognosis. Stroke related disability had a negative effect on aphasia recovery; an increase in stroke disability of one point on the mRS scale resulted in 7.5% poorer prognosis, after controlling for baseline aphasia and average therapy amount.

The unstandardized coefficients provide a clinically useful and practical interpretation of the regression model. However, since *each predictor is measured on a different scale*, the sizes of the unstandardized coefficients are not indicative of the relative impact of each predictor on recovery. This information can be ascertained by looking at the standardised coefficients which are also presented in Table 3. The standardised coefficients are obtained by standardising the variables in the model to unit-less quantities with a standard deviation of one. These standardised coefficients are directly comparable and therefore provide better insight into the relative importance of each predictor in the model.[36] As seen from Table 3, the standardised coefficient for the predictors in the model are $\beta = 0.252$ for baseline AQ, $\beta = -0.243$ for initial mRS and $\beta = 0.245$ for average therapy amount. The fact that three standardised coefficients are of approximately the same size (ignoring the negative sign of the coefficient for initial mRS) suggests that all three predictors have approximately the same impact on aphasia recovery.

The specific values of the standardised coefficients may be interpreted as follows:

- An increase of one standard deviation in baseline AQ results in an increase of

0.252 standard deviations in percent of maximal recovery achieved.

- An increase of one standard deviation in initial mRS results in a decrease of 0.243 standard deviations in percent of maximal recovery achieved.
- An increase of one standard deviation in average therapy amount per day results in an increase of 0.245 standard deviations in percent of maximal recovery achieved.

Table 2 shows the standard deviations for these variables. Since the standard deviation for the percent of maximal potential recovery is 30.95, a 0.252 standard deviation increase corresponds to 7.8% increase in percent of potential maximal recovery. Since the standard deviation of baseline AQ is 27.3, this means that a 27.3 point increase in AQ is associated with a 7.8% improvement in prognosis of recovery. Similarly, since the standard deviation of mRS is 1, a one category change in mRS is associated with a 0.243 standard deviation change in percent of maximal potential recovery, which corresponds to a 7.6% change in prognosis. Since the sign of the coefficient is negative, an increase in mRS is associated with poorer prognosis. Finally, the standard deviation for average therapy amount is 12.1 and the standardised coefficient is 0.245. This suggests that an increase of 12.1 minutes of therapy per day in the very early rehabilitation period will result in a 7.6% improvement in prognosis. In summary, this may be interpreted to mean that an approximately 8% difference in prognosis may be associated with either a 27 unit difference in baseline AQ of a 1 category difference in mRS or a difference of 12 minutes of therapy per day, in the very early rehabilitation period.

The partial and part correlation coefficients presented in Table 3 also confirm the fact that the relative importance of the three predictors in the model is approximately the same. The partial correlation coefficients represent the relationships between each predictor and the outcome variable, while controlling for the effects that the other predictors have on the relationship the part correlation coefficients represent

the relationships between each predictor and the outcome variable, while controlling for the effect that each of the other predictors has on the outcome variable[39]. As seen from Table 3, the partial correlations for baseline AQ, initial mRS and average therapy amount are 0.242, -0.247 and 0.263 respectively, while the corresponding part correlations are 0.210, -0.214 and 0.229 respectively. Once again, the fact that these are of approximately the same size (ignoring the negative sign of the coefficients for mRS) suggests that after "factoring out" the effects of the other predictors in the model, each predictor has approximately the same correlation with the prognosis of recovery. It should be noted that the partial and part correlations of average therapy amount with recovery are marginally higher than those of baseline AQ and of initial mRS. Although these small differences are not likely to be statistically significant, they provide a promising direction for future investigation of the intensity of very early aphasia therapy.

The clinical interpretation of this model is presented in Table 4 which demonstrates *case examples* for specific values of baseline mRS, and AQ scores. This model predicts the amount of change in the AQ score when no therapy, (spontaneous recovery) 30 and 60 minutes of therapy are provided within the very early aphasia recovery phase. For example the highlighted section in Table 4 gives the example of a person with a mRS of 4 and a baseline AQ score of 16 indicating significant stroke related disability and severe aphasia. Using this model to interpret the regression information presented in Tables 2 and 3, the person in this case example could expect spontaneous recovery to account for a 31 point increase (47-16) on the AQ after 22 – 25 days of intervention. If the same person received 30 minutes of therapy, five days per week for the same period, they could expect to gain an extra 11 points on the AQ score (58-16) over and above what could be expected from spontaneous recovery. If that same person was to receive 60 minutes of direct aphasia therapy, for the same intervention period, they could anticipate a gain of 22 points over and above what

could be expected from spontaneous recovery. A twenty-two point gain on the AQ is both a statistically and clinically significant improvement which could be explained in a clinical setting as the difference between a person speaking in single words with poor comprehension requiring full assistance to communicate to talking independently in sentences with mild word finding difficulties.

Discussion

This study is the first of its kind to include the amount of aphasia therapy as a factor in predicting aphasia recovery. Importantly, the data only include the amount of time spent in direct therapy,[33] which allows for some interpretation of the results regarding the intensity of aphasia therapy in very early aphasia recovery. Of great interest in this cohort are the stroke survivors (N=23; 29%) with aphasia who received no direct aphasia therapy in the first 22 days of their recovery (from Trial 1).[19] This cohort and any change made in AQ scores between their baseline assessment and the end of the intervention period can be reliably attributed to spontaneous recovery.

The selection criteria for these trials were designed to be broadly inclusive to allow for reasonable external validity when interpreting the results. We believe the participants in these trials were representative of a typical stroke related aphasia caseload in very early and early recovery phases. Over half (N=44 or 56%) of the cohort experienced severe aphasia (score of between 0 – 32.2 on the AQ) and the age of the cohort in this study (69 years) sits within the age range (59[35] to 76[1,9]) of previous studies of aphasia recovery. However a natural variation in age is evident, the reason for the differences is unknown and may have some relationship with the region in which the studies were undertaken.

Lazar et al,[35] outline a distinctive relationship between initial aphasia severity and the predicted amount of change at 90 days post-stroke in people with mild to moderate

aphasia. The authors[35] report the proportion of maximal potential recovery in people with aphasia is similar to the amount of predicted and proportional recovery in motor impairment suggesting that "spontaneous recovery may have similar biological mechanisms, related to initial severity, across modalities."[(35pp1487)] Due to the high predictability of acute stroke recovery, Lazar et al raise three alternative hypotheses regarding early treatment, these being: i) treatment induces a predictable relation with therapists providing intervention in direct proportion to impairment; ii) treatment has no effect on language recovery and iii) treatment acts to trigger or enable spontaneous, biological recovery mechanisms.

We believe there are several elements from this study that support Lazar et al's alternative hypotheses ii) and iii). Primarily, this study adds information regarding recovery in people with severe aphasia and that the model we have presented includes various amounts of direct aphasia therapy provided in the very early phase when the mechanisms of spontaneous recovery are said to be their greatest.[37]

From the model presented in this study, we can see that the expected effects of spontaneous recovery are significant. Given the data presented in this study, and the trend towards the positive effects of aphasia therapy[12] hypothesis ii) "treatment has no effect on language recovery" may prove incorrect. Conversely, evidence to support Lazar et al's[35] hypothesis iii) "that treatment acts to trigger or enable spontaneous, biological recovery mechanisms" especially in the very early and early recovery phases is growing. This is evidenced by the data in this study which showed that the standardised coefficients for baseline AQ, initial mRS and average amount of therapy have approximately the same impact on aphasia recovery.

In view of the fact that initial aphasia severity and stroke disability are two factors to consistently predict aphasia recovery and that amount of aphasia therapy has not previously been included in predictive models, it is interesting to see that amount of aphasia therapy has a similar impact on aphasia recovery as the universally accepted

factors of aphasia severity and stroke disability.

Study Limitations

As a preliminary attempt at modelling, the current model has a number of limitations. The number of factors used in this analysis includes only the major clinical predictors (not AQ subtest scores, gender or handedness) as there was insufficient power in the sample to include more. In addition, the end point of this analysis was calculated at therapy completion which does not allow for further interpretation of the possible long -term effects of the amount of very early intervention.

Evidence for aphasia therapy effectiveness in the very early stage after onset is equivocal, despite the recent trend toward positive results. This study has drawn data from only the two positive studies completed in the very early recovery phase and as such the results should be interpreted with due caution. Due to difficulties with data-pooling, differences in outcome measures and lack of reporting of data regarding direct aphasia intervention, studies that did not show a therapy effect[38,39] were not considered.

This model gives a scientifically sound estimation of factors that contribute to aphasia recovery, the model presented here accounts for approximately 30% of the variability seen in overall aphasia recovery, suggesting there is still much work to be done to identify the remaining undeclared factors that that contribute to aphasia recovery. Nonetheless, this type of modelling is a first attempt at providing systematic prediction of outcomes incorporating the important notion of treatment intensity. Numerous factors potentially contribute to recovery as previous studies have alluded to. It is proposed that future studies could systematically include many of these variables in a model such as the one used in this study to complete the picture.

Conclusion and future directions

The major contribution of this study is that the amount of aphasia therapy has been included in a predictive model investigating factors in aphasia recovery *and* it was shown to be a significant predictor with a similar impact on recovery as baseline aphasia severity and initial stroke related disability. The amount of direct aphasia therapy provided in the very early recovery phase was tolerated by the majority of participants and is thought to be a clinically appropriate amount of therapy in the acute recovery phase.

In order to better address the issues around sample size and the prognostic value of individual factors in aphasia recovery, the aphasia research community require further international collaboration and data sharing initiatives such as VISTA-Rehab.[40] Researchers and clinicians should consider ways in which to record various aphasia related interventions that allow for the analysis of amount of direct aphasia therapy to enable the question of aphasia related treatment intensity to be more fully explored.

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Figure 1. Diagrammatic representation of percent of maximal potential recovery.

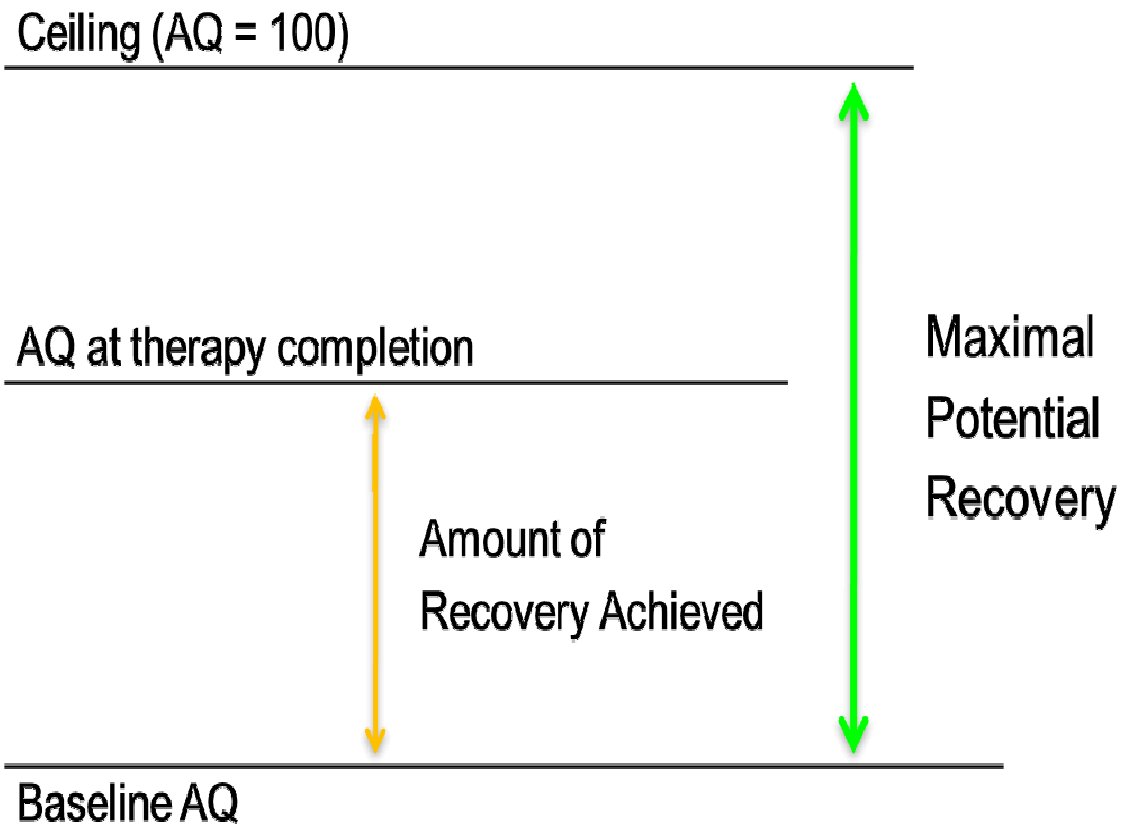

Table 1. Baseline demographic and stroke characteristics for the cohort

N = 79		Number (%)
Age		
Mean (SD)		69.5 (14.0)
Minimum		25
Maximum		91
Female		38 (48)
Previous Stroke:		
Yes		9 (11)
Stroke type		
Ischaemic		71 (90)
Haemorrhagic		8 (10)
Stroke classification		
PACs		22 (28)
TACs		48 (61)
PoCs		1 (1)
LACs		0
Non-classified[41]		8 (10)
Stroke Hemisphere		
Left		76 (96)
Right		3 (4)
Admission mRS score		
2		6 (8)
3		18 (23)
4		23 (29)
5		32 (40)
Baseline raw AQ Scores		
Mean (SD)		31.7 (27.6)
Minimum		0.0
Maximum		88.3
Baseline AQ severity		
Mild	(62.6 – 93.6)	12 (15)
Moderate	(32.3 – 62.5)	23 (29)
Severe	(0 – 32.2)	44 (56)

PACs: partial anterior circulation stroke; TACs: total anterior circulation stroke; PoCs: posterior circulation stroke LACI: Lacunar stroke; Non-classified = haemorrhage[x]

Table 2. Descriptive Statistics for Aphasia Therapy and Outcomes

	Entire Cohort N = 79	Assessed at Therapy End N=70
Amount of very early therapy (mins)		
Mean (SD)	392.0 (468.3)	406.6 (466.1)
Minimum	0.0	0.0
Maximum	1415	1200
Number of therapy sessions		
Mean (SD)	7.9 (8.2)	8.3 (8.3)
Minimum	0.0	0.0
Maximum	21.0	21.0
Length of Stay (days)		
Mean (SD)	23.9 (11.3)	24.4 (11.8)
Minimum	5	5
Maximum	49	49
Therapy Intensity (min/session)		
Mean (SD)	29.5 (24.7)	30.1 (23.6)
Minimum	0.0	0.0
Maximum	88.4	75.0
Average Therapy Amount (min/day)		
Mean (SD)	13.5 (14.7)	13.2 (12.1)
Minimum	0.0	0.0
Maximum	83.2	34.3
Initial mRS		
Mean (SD)	4.0 (1.0)	4.0 (1.0)
Minimum	2	2
Maximum	5	5
Baseline AQ Scores		
Mean (SD)	31.7 (27.6)	32.0 (27.3)
Minimum	0.0	0.0
Maximum	88.3	88.3
AQ at Therapy End		
Mean (SD)		51.2 (33.7)
Minimum		0.0
Maximum		98.0
Percent Max Potential Recovery		
Mean (SD)		33.81 (30.95)
Minimum		-25.20
Maximum		96.82

Table 3. Final Regression Model showing the relationship between the dependent variable %MPR and predictors: Baseline AQ, Initial mRS and Average Therapy Amount

	Unstandardized Coefficients		Standardized Coefficients			95.0% Confidence Interval for B		Correlati	
Predictors	B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound	Zero-order	Parti
Intercept	46.698	17.335		2.694	.009	12.087	81.309		
Baseline AQ	.285	.141	.252	2.026	.047	.004	.566	.450	.242
Initial mRS	-7.466	3.610	-.243	-2.068	.043	-14.674	-.259	-.385	-.24
Average Therapy Amt	.627	.283	.245	2.216	.030	.062	1.193	.356	.263

Table 4. Predictive model with case examples of stroke disability (baseline mRS), aphasia severity (baseline AQ), and 0 minutes (spontaneous recovery), 30 minutes and 60 minutes of very early aphasia therapy.*

Baseline mRS	Baseline AQ	Average Therapy (min/session)	Predicted AQ at therapy end	Average Therapy (min/session)	Predicted AQ at therapy end	Average Therapy (min/session)	Predicted AQ at therapy end
3	16	0	57	30	67	60	76
	48		79		86		92
	80		93		97		99
4	16	0	47	30	58	60	69
	48		72		80		87
	80		89		94		97
5	16	0	35	30	48	60	60
	48		63		73		81
	80		84		90		95

* n.b. Therapy was provided five days per week for the 22-25 day intervention period