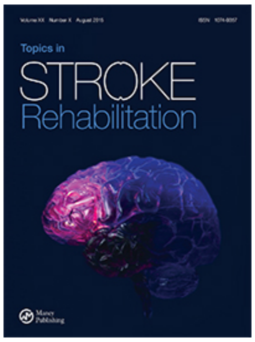

Topics in Stroke Rehabilitation

ISSN: 1074-9357 (Print) 1945-5119 (Online) Journal homepage: http://www.tandfonline.com/loi/ytsr20

Cognitive performance and aphasia recovery

José Fonseca, Ana Raposo & Isabel Pavão Martins

TOPICS IN STROKE REHABILITATION, 2017
https://doi.org/10.1080/10749357.2017.1390904

Cognitive performance and aphasia recovery

José Fonseca[a,b], Ana Raposo[c] and Isabel Pavão Martins[a,b]

[a]Language Research Laboratory, Faculty of Medicine, University of Lisbon, Lisbon, Portugal; [b]UNIC, Instituto de Medicina Molecular, Lisbon, Portugal; [c]Faculdade de Psicologia, Universidade de Lisboa, Lisbon, Portugal

ABSTRACT

Objectives: This study assessed cognitive performance of subjects with aphasia during the acute stage of stroke and evaluated how such performance relates to recovery at 3 months.
Materials & methods: Patients with aphasia following a left hemisphere stroke were evaluated during the first (baseline) and the fourth-month post onset. Assessment comprised non-verbal tests of attention/processing speed (Symbol Search, Cancelation Task), executive functioning (Matrix Reasoning, Tower of Hanoi, Clock Drawing, Motor Initiative), semantic (Camel and Cactus Test), episodic and immediate memory (Memory for Faces Test, 5 Objects Memory Test, and Spatial Span. Recovery was measured by the Token Test score at 3 months. The impact of baseline performance on recovery was evaluated by logistic regression adjusting for age, education, severity of aphasia and the Alberta Stroke Program Early CT (ASPECT) score.
Results: Thirty-nine subjects (with a mean of 66.5 ± 10.6 years of age, 17 men) were included. Average baseline cognitive performance was within normal range in all tests except in memory tests (semantic, episodic and immediate memory) for which scores were ≤−1.5sd. Subjects with poor aphasia recovery ($N = 27$) were older and had fewer years of formal education but had identical ASPECT score compared to those with favorable recovery. Considering each test individually, the score obtained on the Matrix Reasoning test was the only one to predict aphasia recovery (Exp(B)=24.085 $p = 0.038$).
Conclusions: The Matrix Reasoning Test may contribute to predict aphasia recovery. Cognitive performance is a measure of network disruption but may also indicate the availability of recovery strategies.

ARTICLE HISTORY
Received 23 March 2017
Accepted 2 September 2017

KEYWORDS
Aphasia; assessment; cognition; recovery

Introduction

Aphasia affects up to 42% of first-ever stroke survivors[1,2] and has wide-range of social and financial implications with few patients regaining full independence.[3] Recovery of aphasia is related to initial severity, lesion size and brain plasticity for reorganization.[4] Yet most models of recovery can only predict 50–60% of the variance. Several studies have shown that aphasia may be accompanied by changes in memory, attention and executive functions[5] and subjects with aphasia tend to present lower cognitive scores than controls, although their performance may be within normal range.[6,7] Vascular lesions follow vascular territories that encompass multiple brain networks involved in different cognitive functions. It is therefore not surprising that other cognitive domains may be affected. Yet, the pattern of cognitive performance is not uniform and its relation with verbal comprehension or aphasia severity is not linear.[6,8] Cognitive functions such as motivation, inhibitory control, attention or memory are often impaired after stroke[9] but are necessary to effective speech and language therapy, and it is possible that a marked or multi-domain cognitive impairment may be associated with a poor recovery. However, little is known about the contribution of those abilities to the recovery processes and outcome of aphasia.

The aim of this study is to evaluate the cognitive abilities of subjects with acute aphasia (besides the domain of language), by using a battery of non-verbal cognitive tests, and to evaluate the impact of these abilities on aphasia recovery at 3 months.

Material & methods

Study design

In this observational, prospective, longitudinal study, we evaluated the predictive value of a baseline cognitive assessment on aphasia recovery at 3 months in a sample of aphasic participants (AP) with single ischemic lesions of the left hemisphere. The Token test score was used as the main recovery outcome.

Patients

Patients with aphasia due to a first ischemic stroke of the left hemisphere were recruited from six Hospitals. Patients were invited to participate by their neurologist or speech and language therapist, after explaining the purpose and procedures of the study. Patients or their families (whenever patients were unable to write) signed a written consent.

CONTACT José Fonseca jfonseca@medicina.ulisboa.pt

The inclusion criteria were: (a) age ≥50 years; (b) a minimum of 4 years of education; (c) single ischemic stroke of the left hemisphere based on clinical examination and imaging exams; (d) time post stroke ≤30 days and (e) no evidence of previous dementia (clinical diagnosis). The exclusion criteria were a history of alcohol or drug dependency, other neurological or psychiatric disease and severe medical illness.

The protocol was approved by the Institutional Joint Ethics Committee for the Faculty of Medicine, University of Lisbon and North Lisbon Hospital Centre.

Material

First language and cognitive evaluation took place within the first 30 days of stroke. The cognitive assessment consisted of a battery of neuropsychological tests that did not require language production. The battery included 10 tests (producing 13 measures) directed to three cognitive domains: memory, executive functions, and attention and speed processing. The memory domain, included the 5 Objects Memory Test (which assesses episodic memory – immediate and delayed recall),[10] Spatial Span of Wechsler Memory Scale III (a measure of immediate memory),[11] Memory of Faces both immediate and delayed recall;[11] and Camel and Cactus Test (a semantic memory test).[12] To evaluate executive functions, the Tower of Hanoi (a measure of planning and problem-solving), was used [13] Matrix Reasoning of WASI (which evaluates abstract thinking),[14] Clock Drawing (visual and constructive ability and planning) and Motor Initiative of the Lisbon Battery for Assessment of Dementia, BLAD (a graphical switching test).[15] Attention and speed processing was evaluated by the Symbol Search of the Wechsler Adult Intelligence Scale – WAIS (a test for sustained attention and speed processing)[16] and the Cancelation Task of BLAD (to assess sustained attention).[15] Test description is presented in Table 1.

The tests selected did not require language production or involved only a minimum component of language. All tests had been previously used in aphasic populations[5] and age and education adjusted norms for the population were available. Each test was preceded by two training items to guarantee that the patient understood the procedure. Only subjects that passed those items proceeded to the evaluation. Tests were administered in the same order to all participants. The obtained scores were converted to standard scores (Z scores), adjusted for age and education according to normative values. A composite cognitive score (CCS) was calculated by averaging the standard values obtained in all cognitive tests applied.

Language was assessed by a comprehensive language battery (Lisbon Aphasia Assessment Battery, BAAL)[17] with tests of fluency, object naming, word and sentence comprehension, word repetition, Aphasia Quotient (AQ), and the Token test.[18]

Neuroimaging data were retrieved from medical records. Lesions were analyzed on the first CT or MRI scan where the lesion was visible. Lesion analysis followed the Alberta Stroke Program Early CT Score (ASPECTS).[19] Two independent examiners, blind to the clinical information, evaluated the presence or absence of a lesion in ten different areas of the left MCA territory. Affected regions were summed and the total was subtracted from 10, producing a score ranging between 0 (lesion in all MCA territory) and 10 (no visible lesion). Whenever there was no complete agreement the images were reviewed and scored by consensus.

Aphasia recovery was evaluated three months later (on the 4th month post-stroke). This time interval corresponds to the greater recovery of language in vascular aphasia.[20] Aphasia recovery was dichotomized according to Token test score[18] obtained in the second evaluation. A score of ≥17 (in a maximum of 22) was considered a favorable recovery since it denotes a normal score for individuals with 4 or more years of education.

Table 1. Neuropsychological battery and language tests.

Cognitive domain	Test
Memory	5 Objects Memory Test (immediate and delayed recall)
	Spatial span of Wechsler Memory Scale III (WMS)
	Memory of Faces of WMS (Immediate and delayed recall)
	Camel and Cactus Test
Executive functions	Tower of Hanoi
	Matrix reasoning of Wechsler Abbreviate Scale of Intelligence (WASI)
	Clock drawing of Lisbon Battery for Assessment of Dementia (BLAD)
	Motor initiative of BLAD
Attention and speed processing	Symbol Search of Wechsler Adult Intelligence Scale (WAIS)
	Cancelation task of BLAD
Language	Lisbon Battery for Assessment of Aphasia (BAAL):
	Speech fluency
	Object naming
	Verbal comprehension (Object identification and Sentence comprehension
	Word repetition
	Token test (22 -item short-version)

Statistical analyses

Statistical analyses were performed using the software Statistical Package for Social Sciences (version 21.0). Descriptive statistics were used to characterize the sample. An intraclass correlation was computed to estimate interrater reliability in the neuroimaging analysis. Related-Samples Wilcoxon signed rank tests were used to compare within subjects scores in the two evaluation moments.

A logistic regression was carried out to investigate the relation between performance in each cognitive test and the recovery of aphasia at 3 months. To control for the extent of the lesion (as indexed by ASPECTS), age, education and the severity of aphasia (AQ), we included these variables as covariates in the analysis.

Results

Fifty patients were enrolled and completed the baseline evaluation. The revaluation was performed in 39 of them. Two patients were excluded because of new stroke and medical complications, 8 for lost contact and in one case imaging data was not available.

Thirty-nine subjects (17 men) were included in the analysis. They had an average age of 66.5 (± 10.6) years, ranging between 50 and 87 years old and an average number of years of formal education of 7.7 (± 5.0). Baseline assessment and revaluation took place at 14.1 ± 10.4 (2–30) and 98.1 ± 6.8 (87–122) days post onset, respectively.

The majority (29 subjects, 74.4%) received rtPA in the acute stage and 10 subjects (25.6%) received conventional treatment.

All participants except four (89.7%) received speech therapy, with an average of three hours per week. In the first evaluation, all patients presented aphasia syndromes, notably global and anomic aphasia (Table 2). These syndromes were changed at 3 months due to language recovery.

Brain imaging analysis was carried out on CT (36 cases) or MRI[3] performed 0.69 ± 1.03 days (range 0 to 3 days) post onset. Average ASPECT score was 7.08 ± 1.74 (3–10). The interrater reliability was 0.89, 95% CI [0.81, 0.94], $p < 0.001$. In 8 cases (20.5%), lesion localization had to be decided by consensus. Most of the lesions were located on insula ribbon, lateral MCA cortex, and lentiform nucleus. The less affected region was the caudate nucleus (Table 3).

Cognitive assessment

Average scores obtained in the first (baseline) and second evaluation (3 months later) are presented in Table 4. Cognitive performance at baseline was within average range in most cognitive domains, except in the memory domain. Performance in the semantic memory test (Camel and Cactus Test), short term memory test (Spatial Span) and episodic memory test (immediate and delayed recall of the 5 Objects Memory Test) was below −1.5sd of the mean.

There was a significant improvement in all language tests from the first to the second evaluations. Score improvement was also observed in all cognitive tests except for the Tower of Hanoi. The difference between the two moments was statistically significant in 9 out of 13 measures.

Aphasia recovery

Twelve (30.8%) patients had a favorable recovery from aphasia (final Token test score ≥17) at 3 months (Table 5). These patients were younger, had more years of formal education, and had significantly better scores in all language tests at baseline compared to individuals with poor recovery. In contrast, both groups had a similar ASPECT score ($M = 7.04$ for patients with favorable recovery and $M = 7.17$ for patients with poor recovery), and an identical lesion localization, considering the 10 sub-regions of the ASPECTS score, except for the M4 region (corresponding to the middle frontal gyrus, in the anterior branches of the middle cerebral artery territory) which was significantly more affected in those with poor recovery ($\chi^2 = 5.394$ $p = 0.043$). Likewise, patients' performance in the cognitive battery at baseline was similar across the two recovery groups in most tests. Significant differences between the two groups were only found for the attention/speed processing tests (Symbol Search and Cancelation task), Memory for Faces and Matrix Reasoning. Performance in these tests was worse in the group with poor recovery, resulting in a worse global composite cognitive score (CCS).

A logistic regression (Table 6) was applied to create a model capable of predicting language recovery based on the independent variables identified as significant in the univariate analysis (age, years of formal education, initial aphasia severity (AQ) and Z-scores obtained in tests of immediate Memory for Faces, Matrix Reasoning, Cancelation Task and Symbol Search, as illustrated in Table 5). ASPECT score was also included as a covariate. The analysis showed that the overall model explained 0.76 of the variance. The most important predictor of recovery, and the only one that was statistically significant, was the Matrix Reasoning score (odds ratio: 24.085; 95% CI [1.185, 489–627], $p < 0.001$). This shows that as the score obtained in the Matrix Reasoning at baseline increases, so do the odds of language recovery. None of the other variables contributed significantly to the predictive value of the language recovery model.

Table 2. Aphasia diagnosis at baseline and at 3 months.

Diagnosis	1st evaluation (Baseline) N (%)	2nd evaluation (at 3 months) N (%)
Global	11 (28.2)	6 (15.4)
Anomic	8 (20.5)	12 (30.8)
Transcortical Motor	5 (12.8)	8 (20.5)
Broca's	4 (10.3)	5 (12.8)
Wernicke's	4 (10.3)	0
Conduction	3 (7.7)	4 (10.3)
Mixed Transcortical	2 (5.1)	0
Transcortical Sensory	2 (5.1)	0
Sequelae	0	4 (10.3)

Discussion

This study aimed to evaluate how cognitive performance of subjects with aphasia at the acute stage of stroke influences their subsequent recovery process.

Patients presented low scores in nonverbal tests of semantic, episodic and immediate memory at the acute stage of stroke, but performed within the normal range in 8 of the 12 measures applied, notably in tests of executive functioning, attention, and processing speed. Moreover, one cognitive measure improved the predictive model of recovery.

Few studies have assessed the cognitive profile of patients with acute aphasia beyond the language domain and these have revealed contradictory findings,[5] which may result from different selection criteria, follow-up time, the type of evaluation performed and the pattern of lesions.[8,21] Studies on chronic populations[6] reported preserved cognitive abilities in memory and executive functions but impairments in semantic and short-term memory related to deficits in comprehension.[22]

El Hachioui *et al.*,[23] studied 147 subjects with aphasia in the acute phase, at 3 months and at one year. Memory was the most impaired cognitive domain in all the evaluations. Our results are in agreement with this finding. The Camel and Cactus Test presented one of the most severe impairments. The poor performance in semantic memory tests may stem from different mechanisms namely an executive dysfunction rather than a disruption of semantic memory.[24]

About 30% of patients presented a favorable recovery at 3 months as measured by a normal score in the token test. Baseline aphasia severity, patients' age, and ASPECTS score had small and statistically non-significant contributions. The score obtained in a Matrix Reasoning task was the single best predictor of recovery, even when controlling for the other variables.

Most models of aphasia recovery explain about 60% of recovery variance. This is the case of the model described by Pedersen et al.,[20] which included aphasia severity, neurological stroke severity, age and sex as variables. In the series reported by Lazar *et al.*,[25] 83% of recovery variance was predicted by the aphasia composite mean, lesion volume and patients age. However, this

Table 3. Local of lesion by ASPECTS scale.

Localization	Frequency	%
C – Caudate	4	10.3
M3 – Posterior MCA cortex	5	12.8
IC – Internal capsule	8	20.5
M1 – anterior MCA cortex	9	23.1
M6 – posterior immediately superior to M3	9	23.1
M4 – anterior immediately superior to M1	10	25.6
L – Lentiform (putamen)	12	30.8
M5 – lateral immediately superior to M2	15	38.5
M2 – Lateral MCA cortex	17	43.6
I – Insular ribbon	25	64.1

study intends to simply assess the difference between the initial WAB tests and their performance 90 days later, not intending to measure almost complete recovery. El Hachioui *et al.*,[2] model explained 55.7% of recovery including a phonological score, Barthel index score, age, educational and stroke subtype as variables. Finally, Forkel *et al.*,[26] showed that 62% of the variance could be explained by age, sex, lesion size and volume of the arcuate fasciculus. Some authors have suggested that the remaining variance of recovery models (about 40%) may be explained by individual factors namely the pattern of language organization and the degree of expertise attained in some language functions.[27] None of the above-mentioned models used any cognitive abilities beyond language as predictors. In the present study, we were able to improve the predictive variance to 76% by adding the Matrix Reasoning Test, a simple "paper and pencil test" that does not require expensive technology and is easily used in a clinical/therapeutic setting.

The impact of cognitive functions on aphasia recovery can be explained by different mechanisms.

First, cognition may be mediated by language, and be an indirect measure of language ability. In this study, we found a correlation between AQ and matrix reasoning task at the baseline ($r = 0.347$ $p = 0.030$) and at the 3-month evaluation ($r = 0.464$ $p = 0.003$), somehow supporting this hypothesis

Second, cognitive performance can be an indirect measure of lesion size. However, in the present study, there was no significant correlation ($r = 0.179$ $p = 0.275$) between Matrix reasoning test and ASPECTS scores, which suggests that this effect was not mediated by lesion extension in the middle cerebral artery territory.

Third, aphasia recovery depends upon the use of alternative networks or strategies to perform the same function. The impact of damage to one system depends on the integrity of another.[27] While the recovery of speech production depends on slowly evolving activation changes in the left hemisphere of peri-infarct tissue, the recovery of speech comprehension appears to depend on both left right temporal lobe activation.[28] In this study, we do not have imaging data to evaluate this hypothesis. In addition, these abilities are necessary to fully involve the patients in speech therapy.

Fourth, the importance of learning ability for successful rehabilitation had been emphasized by Ferguson,[29] as well as by Fillingham *et al.*,[30] Fillingham *et al.*,[31] reported a relation between episodic memory, good attention and naming ability in 11 patients with aphasia. Lambon Ralph *et al.*,[32] found a relationship between the treatment of anomia and tests of semantic and spatial memory, visuospatial capacities and attention. Although the results reported are rather heterogeneous, these and our findings suggest that nonverbal abilities should be better explored and may be used as independent predictors of aphasia recovery.

Finally, it is possible that the performance in Matrix Reasoning Test can be an indirect measure of cognitive reserve. Reserve is the ability to optimize performance, for any given degree of lesion load,[33] by using brain structures or networks not engaged in the intact brain.[34] It is usually associated with previous cognitive stimulation and development as measured by literacy and vocabulary performance. However, in a subject with aphasia vocabulary abilities cannot be measured and other measures of cognitive reserve need to be found. The finding that patients with a worse recovery were older and had fewer years of education

Table 4. Language and cognitive assessment at baseline and at 3 months.

			1st evaluation (Baseline)	2nd evaluation (at 3 months)		
	N	Max score	Mean ± SD	Mean ± SD	U	*p*
5 objects memory Test						
Immediate recall	38		−2.63 ± 3.51*	−1.41 ± 2.44	184.000	0.062 *ns*
Delayed recall	37		−1.61 ± 3.48*	−0.46 ± 1.89	371.000	0.044
Spatial span of WMS	39		−1.56 ± 1.49*	−1.34 ± 1.22	336.000	0.178 *ns*
Memory of faces of WMS						
Immediate recall	31		−0.48 ± 1.63	0.01 ± 1.53	348.000	0.005
Delayed recall	29		−0.31 ± 0.98	0.09 ± 1.30	255.500	0.002
Camel and Cactus Test	27		−3.19 ± 2.24*	−1. 93 ± 2.74*	314.000	<0.001
Tower of Hanoi	21		0.40 ± 1.97	−0.04 ± 1.17	40.500	0.267 *ns*
Matrix reasoning of WASI	39		−1.18 ± 0.86	−0.84 ± 0.92	455.000	0.007
Clock drawing of BLAD	26		0.47±1.02	0.85 ± 0.70	15.000	0.041
Motor initiative of BLAD	24		−0.13 ± 0.98	0.16 ± 0.86	18.500	0.084 *ns*
Symbol search of WAIS	19		−0.65 ± 1.04	0.06 ± 1.68	90.500	0.016
Cancelation task of BLAD	28		−0.04 ± 1.15	0.47 ± 1.26	326.500	0.001
CCS	39		−1.39 ± 1.61	−0.59 ± 0.94	657.000	<0.001
Speech fluency (NF/F)	39		(22/17)	(16/23)	$\chi^2 = 20.964$	<0.001
Fluency (rating)	39	5	2.67 ± 1.38	3.44 ± 1.29	$\chi^2 = 32.389$	0.001
Naming	39	16	5.21 ± 5.12	9.44 ± 6.05	491.000	<0.001
Comprehension	39	24	19.54 ± 5.75	22.56 ± 3.76	627.500	<0.001
Word repetition	39	30	14.46 ± 13.45	19.72 ± 12.68	228.000	<0.001
Token test	35	22	5.5 ± 4.84	12.36 ± 6.32	528.000	<0.001
AQ	39	100	49.01 ± 25.96	67.71 ± 27.12	741.000	<0.001

*Test with scores below – 1.5 sd of the mean.

Table 5. Differences in baseline language and cognitive performance between groups with poor or favorable language recovery.

		Poor recovery (*N* = 27)	Favorable recovery (*N* = 12)		
	N	Mean ± SD	Mean ± SD	U	*P*
Age	39	69.3 ± 10.66	60.33 ± 7.89	81.000	0.013
Education	39	6.19 ± 3.05	11.08 ± 6.68	226.000	0.052
Speech fluency (NF/F)	39	1.41 ± 0.50	1.50 ± 0.52	$\chi^2 = 0.290$	0.730 *ns*
Fluency (rating)	39	2.30 ± 1.41	3.50 ± 0.91	$\chi^2 = 7.899$	0.048
Naming	39	3.89 ± 4.79	8.17 ± 4.75	243.000	0.013
Comprehension	39	18.06 ± 6.37	22.88 ± 0.68	248.500	0.007
Word repetition	39	11.70 ± 13.24	20.67 ± 12.28	245.000	0.011
Token test	35	3.37 ± 3.54	9.58 ± 4.42	239.000	<0.001
AQ	39	41.33 ± 25.11	66.30 ± 19.15	251.000	0.006
ASPECTS	39	7.04 ± 1.83	7.17 ± 1.59	$\chi^2 = 3.307$	0.855 *ns*
5 objects memory test					
Immediate recall	38	−3.04 ± 3.77	−1.75 ± 2.80	191.500	0.269 *ns*
Delayed recall	37	−2.12 ± 4.02	−0.56 ± 1.58	140.500	0.761 *ns*
Spatial span of WMS	39	−1.80 ± 1.50	−1.02 ± 1.36	204.500	0.199 *ns*
Memory of faces of WMS					
Immediate recall	31	−0.90 ± 1.79	0.20 ± 1.07	167.000	0.032
Delayed recall	29	−0.52 ± 0.91	−0.02 ± 1.05	138.500	0.107 *ns*
Camel and Cactus test	27	−2.71 ± 2.26	−3.88 ± 2.13	57.500	0.134 *ns*
Tower of Hanoi	21	0.10 ± 1.20	1.00 ± 3.04	49.500	1.000 *ns*
Matrix reasoning of WASI	39	−1.57 ± 0.54	−0.30 ± 0.81	−5.785	*<0.001*
Clock drawing of BLAD	26	0.36 ± 1.04	0.63 ± 1.02	87.500	0.799 *ns*
Motor initiative of BLAD	24	−0.32 ± 1.06	0.09 ± 0.87	89.000	0.331 *ns*
Symbol search of WAIS	19	−1.11 ± 1.01	−0.24 ± 0.92	75.500	0.010
Cancelation task of BLAD	28	−0.54 ± 0.87	0.74 ± 1.14	153.000	0.004
CCS	39	−1.76 ± 1.72	−0.56 ± 0.91	250.000	0.006

Table 6. Logistic regression analysis.

							95% CI for Exp(B)	
	B	SE	Wald	df	*p*	Exp (B)	Lower	Upper
Age	−0.107	0.090	1.437	1	0.231	0.898	0.753	1.071
Education	0.064	0.207	0.095	1	0.757	1.066	0.711	1.599
AQ	0.046	0.028	2.589	1	0.108	1.047	0.990	1.107
ASPECTS	−0.034	0.457	0.006	1	0.940	0.966	0.395	2.366
Matrix Reasoning Test	3.182	1.537	4.286	1	0.038	24.085	1.185	489.627
Memory of Faces	0.391	0.639	0.374	1	0.541	1.478	0.422	5.171
Cancelation Task	2.074	1.252	2.745	1	0.098	7.959	0.684	92.606
Symbol Search	−2.992	2.268	1.740	1	0.187	0.050	0.001	4.279
Constant	6.476	7.906	0.671	1	0.413	649.673		

supports this hypothesis. These factors could eventually indicate less cognitive reserve or a more extensive functional impairment since there were no significant differences in ASPECTS score.

We acknowledge some limitations in this study namely, the relatively small sample size with a large variety of aphasia diagnosis, the short follow-up time, and the fact that the complete cognitive battery could not be applied to all patients. In addition, the measure used for lesion extension is semi-quantitative and does not identify specific areas. A very strict measure of recovery was used and did not evaluate partial language improvement.

Despite these limitations, this study suggests that non-verbal cognitive assessment may provide additional information about aphasia recovery, evidence that may be worth exploring more systematically in other studies. Furthermore, cognitive assessment may assist speech therapists in the preparation of the treatment plan taking in consideration possible limitations. More studies are necessary to understand the intervention of cognitive abilities in language recovery strategies and the effect of rehabilitation.

Disclosure statement

All author states that there is no conflict of interest.

References

1. Engelter ST, Gostynski M, Papa S, et al. Epidemiology of aphasia attributable to first ischemic stroke incidence, severity, fluency, etiology, and thrombolysis. *Stroke*. 2006;37:1379–1384.
2. El Hachioui H, Lingsma HF, Van De Sandt-Koenderman MW, et al. Long-term prognosis of aphasia after stroke. *J Neurol Neurosurg Psychiatry*. 2013;84:310–315.
3. Davidson B, Howe T, Worrall L, Hickson L, Togher L. Social participation for older people with aphasia: The impact of communication disability on friendship. *Top Stoke Rehabil*. 2008;15(4):325–340.
4. Saur D, Lange R, Baumgaertner A, et al. Dynamics of language reorganization after stroke. *Brain*. 2006;129:1371–1384.
5. Fonseca J, Ferreira JJ, Martins IP. Cognitive performance in aphasia due to stroke – a systematic review. *Int J Disability Human Dev*. 2016;16:127–139. doi: 10.1515/ijdhd-2016-0011.

6. Helm-Estabrooks N, Bayles K, Ramage A, Bryant S. Relationship between cognitive performance and aphasia severity, age, and education: Females versus males. *Brain Lang*. 1995;51:139–141.
7. Erikson RJ, Goldinger SD, Lapointe LL. Auditory vigilance in aphasic individuals: Detecting nonlinguistic stimuli with full or divided attention. *Brain Cogn*. 1996;30:244–253.
8. Helm-Estabrooks N. Cognition and aphasia: A discussion and a study. *J Commun Disorders*. 2002;35(2):171–186.
9. Sun J-H, Tan L, Yu J-T. Post-stroke cognitive impairment: Epidemiology, mechanisms and management. *Ann Transl Med*. 2014;2(8):80–96.
10. Papageorgiou SG, Economou A, Routsis C. The 5 objects test: A novel, minimal-language, memory screening test. *J Neurol*. 2014;261(2):422–431.
11. Wechsler D. *Wechsler Memory Scale*. 3rd ed. San Antonio, TX: Pearson; 1997.
12. Bozeat S, Lambon Ralph MA, Patterson K, Garrard P, Hodges JR. Non-verbal semantic impairment in semantic dementia. *Neuropsychologia*. 2000;38:1207–1215.
13. Shallice T. Specific impairments of planning. *Philos Trans Royal Soc London B*. 1982;298:199–209.
14. Wechsler D. *Wechsler abbreviated scale of intelligence (WASI)* [The Psychological Corporation: Harcourt Brace & Company]. 1999; New York: Psych Corp.
15. Garcia C. *Doença de Alzheimer. Problemas de diagnóstico clínico* Tese de Doutoramento. Lisboa: Faculdade de Medicina de Lisboa; 1984.
16. Wechsler D. *Wechsler adult intelligence scale*. San Antonio, TX: Pearson; 1997.
17. Ferro JM. *Neurologia do comportamento*. Estudo de correlação com a tomografia axial computorizada. Doctoral thesis. Lisbon Faculty of Medicine; 1986.
18. De Renzi E, Vignolo LA. The Token Test. A sensitive test to detect receptive disturbances in aphasics. *Brain*. 1962;85:665–678.
19. Pexman JW, Barber PA, Hill MD, et al. Use of the Alberta Stroke program early CT score (ASPECTS) for assessing CT scans in patients with acute stroke. *Am J Neuroradiol*. 2001;22:1534–1542.
20. Pedersen PM, Jorgensen HS, Nakayama H, Raaschou HO, Olsen TS. Aphasia in acute stroke: Incidence, determinants and recovery. *Ann Neurol*. 1995;38:659–666.
21. Majerus S, Attout L, Artielle MA, Van Der Kaa MA. The heterogeneity of verbal short-term memory impairment in aphasia. *Neuropsychologia*. 2015;77:165–176.
22. Martin RC, He T. Semantic short-term memory deficit and language processing: A replication. *Brain Lang*. 2004;89:76–82.
23. El Hachioui H, Vish-Brink EG, Lingsma HF, et al. Nonlinguistic cognitive impairment poststroke aphasia: A prospective study. *Neurorehabilitation Neural Repair*. 2014;28(3):273–281.
24. Mares I, Martins IP. Contribuição do estudo de casos na compreensão da hierarquização da memória semântica. *Revista Portuguesa de Psicologia*. 2015;44:31–38.
25. Lazar RM, Minzer B, Antoniello D, Festa JR, Krakauer JW, Marshall RS. Improvement in aphasia scores after stroke is well predicted by initial severity. *Stroke*. 2010;41(7):1485–1488.
26. Forkel SJ, De Schotten MT, Dell'acqua F, et al. Anatomical predictors of aphasia recovery: A tractography study of bilateral perisylvian language networks. *Brain*. 2014;137:2027–2039.
27. Ferguson A. Learning in aphasia therapy: It's not so much what you do, but how you do it!. *Aphasiology*. 1999;13(2):125–150.
28. Seghier ML, Patel E, Prejawa S, et al. The PLORAS database: A data repository for predicting language outcome and recovery after stroke. *Neuroimage*. 2016;124:1208–1212.
29. Price CJ, Crinion J. The latest on functional imaging studies of aphasic stroke. *Curr Opin Neurol*. 2005;18(4):429–434.
30. Fillingham JK, Hodgson C, Sage K, Lambon Ralph MA. The application of errorless learning to aphasic disorders: A review of theory and practice. *Neuropsychol Rehabil*. 2003;13:337–363.
31. Fillingham JK, Sage K, Lambon Ralph MA. The treatment of anomia using errorless learning. *Neuropsychol Rehabil*. 2006;16(2):129–154.
32. Lambon Ralph MA, Snell C, Fillingham JK, Conroy P, Sage K. Predicting the outcome of anomia therapy for people with aphasia post CVA: Both language and cognitive status are key predictors. *Neuropsychol Rehabil*. 2010;20(2):289–305.
33. Satz P. Brain reserve capacity on symptom onset after brain injury: A formulation and review of evidence for thresh-old theory. *Neuropsychology*. 1993;7:273–295.
34. Stern Y. What is cognitive reserve? Theory and research application of the reserve concept. JINS. 2002;8:448–460.