

The brief executive language screen: sensitivity and specificity in acute to early sub-acute stroke

Mia R. Phillips, Jessica Byrne, Emily C. Gibson, Casey Gilbert, Lucy Ford, Georgia Marsh, Jessica Chow, Gail A. Robinson & Neuropsychology Research Unit

To cite this article: Mia R. Phillips, Jessica Byrne, Emily C. Gibson, Casey Gilbert, Lucy Ford, Georgia Marsh, Jessica Chow, Gail A. Robinson & Neuropsychology Research Unit (04 Jun 2024): The brief executive language screen: sensitivity and specificity in acute to early sub-acute stroke, Topics in Stroke Rehabilitation, DOI: [10.1080/10749357.2024.2356412](https://doi.org/10.1080/10749357.2024.2356412)

To link to this article: <https://doi.org/10.1080/10749357.2024.2356412>



© 2024 The Author(s). Published with license by Taylor & Francis Group, LLC.



[View supplementary material](#)



Published online: 04 Jun 2024.



[Submit your article to this journal](#)



[View related articles](#)



[View Crossmark data](#)

The brief executive language screen: sensitivity and specificity in acute to early sub-acute stroke

Mia R. Phillips^a, Jessica Byrne^b, Emily C. Gibson^b, Casey Gilbert^b, Lucy Ford^{a,b}, Georgia Marsh^b, Jessica Chow^b, Gail A. Robinson ^{a,b}, and Neuropsychology Research Unit

^aQueensland Brain Institute, the University of Queensland, St Lucia, QLD, Australia; ^bNeuropsychology Research Unit, School of Psychology, the University of Queensland, St Lucia, QLD, Australia

ABSTRACT

Introduction: Propositional language and underlying executive functions can be impaired post-stroke and affect communication and quality of life. Current stroke screening tools are largely tailored to patients with aphasia, being either non-verbal or focussed on core language skills such as naming and repetition. The Brief Executive Language Screening Test (BELS) is a newly developed cognitive screening tool that assesses memory, oral apraxia, core language, as well as propositional language and associated executive functions that can be impacted and overlooked in stroke patients without aphasia. This study examines BELS sensitivity and specificity, and performance in acute to early sub-acute stroke relative to controls.

Method: Cross-sectional BELS data from 88 acute left and right hemisphere stroke patients (within 7 weeks of stroke) and 116 age-matched healthy controls were compared using independent samples t-tests. ROC Curve Analysis was performed to determine a cutoff score for the BELS.

Results: Left and right stroke patients were reduced on all propositional language subtests, and executive function subtests of inhibition, strategy, and selection. Differences were also observed for Oral Apraxia, Naming, and Memory. By contrast, Word Comprehension and Repetition, and Sentence Completion Initiation (after corrections applied) did not differ between groups. A total BELS score of 79.25/100 was highly sensitive (.89) and specific (.89) when classifying stroke patients and healthy controls.

Conclusion: The BELS is brief, sensitive, suitable for bedside administration, and can aid in detection and rehabilitation of subtle executive language impairments. This in turn will help improve relationships and quality of life post-stroke.

ARTICLE HISTORY

Received 11 September 2023




Accepted 23 April 2024

KEYWORDS

Cognitive screen; language; executive functions; sensitivity; stroke

Post-stroke cognitive impairments occur in up to 70% of patients and frequently persist long-term (> 1 year), which contributes to disability, social isolation, and reduced quality of life^{1-4 5-7} Executive functioning and language are cognitive domains commonly affected in acute stroke patients that also predict long-term functional, neuropsychological, and emotional outcomes^{8-11,5,12,13} Current stroke screening tools are largely tailored to patients with aphasia, being either non-verbal or focussed on core language skills [e.g. Cognitive Assessment Scale for Stroke Patients; CASP,¹⁴ Cognitive Linguistic Quick Test; CLQT,¹⁵ Oxford Cognitive Screen; OCS,¹⁶ Oxford Cognitive Screen Plus; OCS-Plus,¹⁷ Quick Aphasia Battery;^{18,19} Western Aphasia Battery – Revised.²⁰ Further, other cognitive

screening tools used in the early stages of stroke only capture severe global impairment [e.g. Montreal Cognitive Assessment; MoCA,²¹ Mini-Mental State Examination; MMSE,²²⁻²⁴ or minimally assess executive functions, which are known to be impacted in early stroke (e.g. CASP, CLQT, OCS, OCS-Plus, MoCA, MMSE). Consequently, subtle communication impairments (e.g. initiation impairment resulting in diminished connected speech) in stroke patients without clear aphasia go undetected, and therefore do not receive appropriate intervention and rehabilitation, which impacts relationships, daily living, and quality of life.⁹ This study investigates the sensitivity and specificity of the Brief Executive Language Screening Test [BELS;¹³]; a recently developed cognitive screening tool that

CONTACT Gail A. Robinson  gail.robinson@uq.edu.au  Queensland Brain Institute, the University of Queensland, St Lucia, QLD 4072, Australia
 Supplemental data for this article can be accessed online at <https://doi.org/10.1080/10749357.2024.2356412>

© 2024 The Author(s). Published with license by Taylor & Francis Group, LLC.

This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0/>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. The terms on which this article has been published allow the posting of the Accepted Manuscript in a repository by the author(s) or with their consent.

assesses memory, oral apraxia, executive functions, and both core language (nominal) skills and spontaneous (propositional) language.

Impairment to executive functions (e.g. initiation, inhibition, planning, problem solving) occur in ~40% of acute stroke patients, and impact successful daily living and mental health^{25,8,26} Additionally, it is the strongest predictor of functional and cognitive outcomes one-year post-stroke.^{5,10,11} In addition, stroke patients often demonstrate impaired propositional language; namely, voluntary, spontaneous, and connected speech that is novel to a context, and crucial for communicating ideas, thoughts, instructions, and feelings.^{9,13,27–30} Impairment presents as diminished quantity and quality of connected speech,³¹ and consequently individuals can experience less meaningful communication and strained relationships with others.⁹

Propositional language impairments highlight the interface of language and executive functioning, which occurs at an early or higher level of language generation.^{13,31–35} Executive functions are integral to the pre-verbal conceptualization stage of language, and include planning, idea generation, selection (i.e. choosing from competing ideas, inhibiting irrelevant ideas), sequencing ideas, initiation, task-setting and monitoring.^{31–38} These executive functions have been associated with both left frontal [e.g. initiation, selection; ^{37,39–43} and right frontal [e.g. inhibition,^{44,45} regions. Importantly, patients with the acquired language disorder of dynamic aphasia demonstrate that disruption to conceptualization processes and severely reduced propositional language can occur despite intact core or nominal language [i.e. naming, repetition, comprehension.^{46–48,37,43,49} Non-aphasic stroke patients have also shown propositional language impairment, highlighting these deficits are subtle and can be easily missed without sufficient assessment; contributing to a lack of or insufficient intervention and rehabilitation.⁹

Assessment of propositional language and executive functions in the acute stage of stroke is critical for early detection and rehabilitation. Brief cognitive screens are more practical than large neuropsychology batteries for acute patients who tire quickly; however, tests such as the MMSE and MoCA^{21,22} lack sensitivity and specificity in this population, and underestimate cognitive impairment due to absence or limited measurement of complex, multifaceted

domains like language and executive functioning, known to be affected in stroke.^{23–25,50,51,16,52–56}

Recently there has been a shift toward stroke-specific cognitive screening tools (e.g. OCS, OCS-Plus, CASP, QAB, WAB-R), which are tailored to stroke patients with aphasia.^{14,16–20} Although extremely valuable, these screens either largely remove spoken language, or tap core language only (e.g. naming), meaning higher level language impairments are not captured. Importantly, current screening tools (both aphasia screens and cognitive screens) only very minimally assess the executive function domain, that underpin the pre-verbal message-generation stage of propositional language (e.g. OCS – Trails; MoCA – brief Trails and Verbal Fluency; CASP – Motor Go No-Go; QAB – connected speech topic; CLQT – Verbal Fluency and Design Generation; WAB-R – connected speech topic). Thus, a cognitive screen that more completely captures executive functions and propositional language deficits in non-aphasic stroke patients (in addition to articulation and core language skills) is necessary to improve rehabilitation services, quality of life and long-term stroke outcomes.

Brief executive language screening test (BELS)

The BELS¹³ is a valid, recently developed brief cognitive test (~15-20 minutes) that can be administered at bedside. It has an Oral Apraxia subtest to screen for articulation and motor speech difficulties, as well as a Nominal Language subsection (Object and Action Naming, Word Comprehension, and Word and Sentence Repetition). A novel feature of the BELS is the Propositional Language and Executive Function subsection, which includes two Spontaneous Speech Scene Descriptions, Phonemic and Semantic Verbal Fluency (with two “Goal” conditions), Sentence Completion (Initiation and Inhibition), and a Motor Go No-Go subtest. This section taps executive functions (initiation, selection, inhibition, and strategy use) known to impact connected speech.¹³

Aims

This study aimed to determine stroke patients’ performance on the BELS relative to healthy age- and gender-matched controls, as well as sensitivity and specificity of a total BELS score. We also aimed

to explore a BELS cutoff score for left and right hemisphere patients (LHS and RHS, respectively). Our first hypothesis was that healthy controls would perform significantly better than stroke patients on the BELS. Secondly, we hypothesized a Receiver Operating Characteristic (ROC) curve analysis would provide a cutoff score on the BELS that was highly sensitive (high true positive rate) and specific (low false positive rate) when classifying stroke and healthy cases. We expected cutoff scores to be similar for LHS and RHS, due to both left and right frontal patients demonstrating impairment to executive processes⁴⁴ Burgess & Shallice, 1996.^{37,41,43,45}

Method

Data used in this cross-sectional study was obtained via convenience sampling by supervised clinical neuropsychology registrars and doctoral candidates in hospital stroke wards (Princess Alexandra Hospital, Royal Brisbane and Women's Hospital, Surgical Treatment and Rehabilitation Service) as part of a longitudinal project between 2016 and 2023. This study was approved by the Metro South and Metro North Queensland Health Human Research Ethics Committees (approval number HREC/16/QPAH/793). All participants provided informed written consent. Data and BELS study materials are available on the Open Science Framework (<https://osf.io/473g6/>). This study conforms to STARD Guidelines.

Participants

Stroke patients

Inclusion criteria were first-time stroke confirmed by brain imaging, over 18 years old and fluent English-speakers. Exclusion criteria were Transient Ischemic Attack, or diagnosis of another neurological disorder. Patients and controls who did not complete the BELS were excluded from analyses. One-hundred-and-nine patients were recruited, with a final sample of 88 stroke patients tested on average 17.39 days (ranging from 2–49 days) post-stroke; acute to early subacute stage⁵⁷, 43% female; handedness: 90% right, 1% forced right, 9% left; $M_{AGE} = 62.88$, $SD_{AGE} = 14.10$; $M_{EDUCATION} = 12.03$,

$SD_{EDUCATION} = 2.71$]. There were 80 ischemic stroke patients, and eight hemorrhagic patients, 29 left hemisphere patients (LHS), 57 right hemisphere patients (RHS), and two bilateral patients.

Controls

Healthy controls were recruited through the University of Queensland (UQ) networks and included if they spoke fluent English and were 18 years or older. Exclusion criteria was any neurological disorder or performing in the impaired range (<5th percentile) on standard neuropsychology tests. The final control group consisted of 116 healthy adults (47% female; handedness: 92% right, 1% right/ambidextrous, 7% left; $M_{AGE} = 63.15$, $SD_{AGE} = 13.52$; $M_{EDUCATION} = 15.12$, $SD_{EDUCATION} = 3.56$).

Materials

Due to the verbal nature of the BELS, stroke patients were screened for aphasia via independent neuropsychological tests. These included the National Adult Reading Test to assess reading [NART – 2nd Edition,⁵⁸ normative data for cutoffs from⁵⁹; the Boston Naming Test to assess naming [BNT – Short Form,⁶⁰ normative data for cutoffs from,⁶¹ and⁶² and the Hayling Sentence Completion Test (Section A) to assess comprehension [HSCT.⁶³ Visual perception was also assessed via the Visual Object and Space Perception Battery Incomplete Letters subtest⁶⁴ Patients were classified as aphasic if they performed below the 5th percentile on either NART, BNT, or HSCT A.

BELS

The BELS has four subsections.

Oromotor functioning

Oromotor functioning is assessed via the Oral Apraxia subtest, which involves execution of five facial/mouth movements (e.g. whistling).

Nominal language

Ten items (e.g. tiara) are used for Object Naming, Word Repetition and Word Comprehension. Action Naming includes five illustrated actions obtained from Druks and Masterson's⁶⁵ Object and

Action Naming Battery (e.g. “shooting”), which are subsequently transformed to past tense (e.g. “shot”). There are five Sentence Repetition items.

Propositional language and executive functions.

Spontaneous speech is assessed via a complex scene (Australian Beach Scene), with participants instructed to describe the scene for one minute. The Cookie Theft Scene⁶⁶ is then presented as a “goal” condition, with participants instructed to speak continuously for one minute about the scene.^{13,67} Word Fluency tasks involve naming as many words as possible in one minute under two conditions⁶⁸: phonemic (i.e. words beginning with S and B) and semantic (i.e. animals and fruits/vegetables). “B” and “Fruit/Vegetables” are “goal” conditions, in which participants have a goal of speaking 20% more words than “S” and “Animals,” respectively, which increases language generation.⁶⁷ Motor Go-No-Go is based on,⁶⁹ tapping task and requires participants to copy finger tapping sequences performed by the assessor (e.g. assessor taps once, participant taps once), and then reverse tapping rules (e.g. assessor taps once, participant taps twice). Sentence Completion (SC) was developed based on the Hayling Sentence Completion Test,⁶³ and requires participants to complete ten sentences read aloud by the assessor by producing one word, under two conditions: Initiation (the sentence must be meaningful, e.g. “The lecture should last about one ... ” *hour*) and Inhibition (the sentence must be non-meaningful, e.g. “The lecture should last about one ... ” *plant*). Low Constraint Initiation items increase selection demands due to multiple competing responses becoming available, compared to High Constraint items which have a dominant response.^{13,37,43}

Memory. Incidental Verbal Memory involves participants recalling the ten items from Object Naming, Word Repetition and Word Comprehension subtests.

BELS scoring

A total BELS score out of 100 can be calculated, as well as a total score for each subsection. See Supplementary Materials for BELS score sheets,

instructions, and scoring manual (including how to calculate a total score).

Statistical analyses

Using IBM SPSS Statistics 27,⁷⁰ an alpha level of .05 was set for all analyses unless otherwise stated for Bonferroni corrections for multiple comparisons. Differences between stroke and control groups were investigated via independent samples t-tests (and Mann-Whitney U tests for non-parametric data). ROC curve analysis was performed to determine sensitivity and specificity of a total BELS score. To ensure aphasic patients were not driving significant results, inferential statistics and ROC analyses were conducted with and without aphasic patients. ROC curve analyses were also conducted with RHS and LHS separated to determine most appropriate cutoffs.

Results

Descriptive statistics for baseline neuropsychology and BELS subtests are presented in [Table 1](#). Patients and controls were matched in age ($U = 4927$, $p = .672$). In total, 15 stroke patients (17%; $n = 7$ LHS, 7 RHS, and 1 bilateral patient) showed signs of aphasia on independent reading ($n = 3$; 1 LHS, 1 RHS, 1 Bilateral), naming ($n = 11$; 6 LHS, 5 RHS), and comprehension ($n = 2$; 1 LHS, 1 RHS) measures. One patient was impaired on both reading and comprehension.

The percentage of patients impaired (i.e. performance <5th percentile) on BELS subsections (and proportion of LHS and RHS impaired) were similar when aphasic patients were included and excluded (see [Table 2](#)). The largest change was a 5% decrease in impaired patients for Nominal Language, Propositional Language and Executive Functions, and Memory subsections. LHS and RHS were relatively equally impaired (except for Oral Apraxia and Memory, where more RHS were impaired).

Inferential statistics comparing patients and controls were conducted with and without aphasic patients (see [Table 3](#)). Stroke and control groups did not significantly differ on Word Comprehension, Word Repetition, or SC Initiation number correct (after correction for multiple comparisons; see [Table 3](#)). On all other BELS

Table 1. Descriptive Statistics for BELS Subtests: Controls and Stroke (LHS & RHS).

	Stroke (N = 88)													
	Controls (N = 116)					ALL			LHS			RHS		
	N	M(SD)	Range	5 th % ile	N	M(SD)	Range	% < 5 th % ile (N LHS: N RHS: N Bilateral)	N	M(SD)	Range	N	M(SD)	Range
Age	116	63.15 (13.52)	18–88		88	62.88 (14.10)	18–91	-	29	62.34 (14.55)	28–91	57	63.56 (13.01)	18–86
Education	116	15.12 (3.56)	9–25		88	12.03 (2.71)	7–20	-	29	12.03 (2.31)	8–16	57	12.12 (2.87)	7–20
Reading (NART errors /50)	112	16.13 (6.55)	3–35		83	22.11 (7.29)	3–42	4 (1:1:1)	27	22.78 (5.81)	12–35	54	21.39 (7.73)	3–42
Naming (BNT-15 SF4 /15)	47	14.34 (0.89)	11–15		78	13.31 (2.04)	5–15	14 (6:5:0)	28	13.11 (2.47)	6–15	49	13.45 (1.78)	5–15
Visuospatial (VOSP IL /20)	102	19.57 (0.65)	17–20		85	19.39 (0.87)	16–20	1 (0:1:0)	29	19.48 (0.74)	17–20	54	19.31 (.95)	16–20
Comprehension (HSCT A.55)	80	5.80 (0.68)	3–7		68	5.10 (1.62)	1–7	3 (1:1:0)	24	5.08 (1.44)	1–7	43	5.09 (1.27)	1–6
Articulation														
Oral Apraxia /10	116	9.75 (0.64)	7–10	8	88	9.09 (1.08)	4–10	7 (0:6:0)	29	9.28 (0.75)	8–10	57	8.98 (1.22)	4–10
Nominal Language														
Word Comprehension /10	116	10.00 (0.00)	10–10	10	88	9.99 (0.11)	9–10	1 (1:0:0)	29	9.97 (0.19)	9–10	57	10.00 (.00)	10–10
Word Repetition /10	116	10.00 (0.00)	10–10	10	88	9.99 (0.11)	9–10	1 (0:1:0)	29	10.00 (0.00)	10–10	57	9.98 (.13)	9–10
Sentence Repetition /5	116	4.97 (0.16)	4–5	5	88	4.81 (0.48)	3–5	16 (4:10:0)	29	4.79 (0.56)	3–5	57	4.81 (.44)	3–5
Oral Naming /10	116	9.05 (1.01)	6–10	7	88	7.90 (1.73)	3–10	19 (7:9:1)	29	7.72 (1.80)	3–10	57	8.04 (1.71)	3–10
Action Naming /10	116	9.63 (0.67)	7–10	8	88	8.81 (1.67)	2–10	14 (4:8:0)	29	8.76 (1.48)	5–10	57	8.84 (1.79)	2–10
<i>Nominal Language Total /20</i>	116	19.31 (0.68)	17.50–20.00	17.93	88	18.15 (1.81)	10.50–20.00	28 (8:16:1)	29	18.03 (1.75)	12.75–20.00	57	18.24 (1.88)	10.50–20.00
Propositional Language and Executive Functions														
Beach WPM	114	124.82 (31.50)	38–228	73.75	82	90.50 (32.30)	31–189	29 (8:15:1)	27	92.07 (33.17)	37–189	53	90.34 (35.60)	31–186
GOAL Cookie WPM	114	151.45 (29.31)	79–256	106.50	82	115.16 (37.12)	38–214	45 (7:28:2)	27	123.15 (35.75)	65–208	53	111.70 (38.10)	38–214
Spontaneous Speech /20														
S WPM	111	14.72 (2.75)	8–20	10	81	10.74 (3.47)	4–18	30 (15:9:0)	27	8.52 (3.22)	4–16	52	11.81 (3.10)	4–18
GOAL B WPM	116	17.11 (4.65)	9–30	10	87	11.97 (4.65)	2–24	32 (7:19:2)	29	12.28 (4.94)	4–24	56	11.93 (4.58)	2–22
Animal WPM	114	22.55 (4.33)	12–36	16.75	88	15.63 (5.18)	2–30	44 (13:24:1)	29	9.76 (4.08)	0–16	56	10.93 (4.53)	2–20
GOAL Fruit/Veg WPM	116	21.91 (4.46)	13–36	14	88	15.82 (5.52)	2–30	52 (13:33:0)	29	15.79 (6.36)	2–30	57	15.44 (4.58)	5–25
								35 (10:21:0)	29	15.59 (6.40)	2–30	57	15.93 (5.18)	6–28

(Continued)

Table 1. (Continued).

	Stroke (N = 88)													
	Controls (N = 116)					ALL			LHS			RHS		
	N	M(SD)	Range	5 th %ile	N	M(SD)	Range	% < 5 th %ile (N LHS: N RHS: N Bilateral)	N	M(SD)	Range	N	M(SD)	Range
Verbal Fluency /16	114	14.67 (1.86)	8–16	10	87	8.87 (4.84)	0–16	51 (14:29:1)	29	9.03 (4.95)	0–16	56	8.79 (4.91)	0–16
<i>Initiation Total /10</i>	109	9.22 (1.56)	2.00–10.00	5.90	88	7.55 (2.87)	1–10	30 (9:15:1)	29	7.62 (2.73)	1.80–10.00	57	7.58 (2.89)	1.40–10.00
<i>Inhibition /10</i>	115	8.02 (1.86)	3–10	4	86	4.36 (3.24)	0–10	43 (7:28:1)	29	5.59 (3.11)	0–10	55	3.80 (3.17)	0–10
<i>Strategy /2</i>	115	1.81 (0.46)	0–2	1	86	1.02 (0.87)	0–2	36 (8:21:2)	29	1.28 (0.88)	0–2	55	0.93 (.84)	0–2
Sentence Completion /22	108	19.16 (2.41)	13–22	15	86	12.98 (5.56)	1.60–22.00	57 (13:34:2)	29	14.48 (4.75)	5.40–21.80	55	12.38 (5.75)	1.60–22.00
<i>Motor Go No-GO /3</i>	115	2.94 (0.27)	1–3	2	88	2.76 (0.48)	1–3	2 (2:0:0)	29	2.72 (0.59)	1–3	57	2.79 (.41)	2–3
<i>Propositional Language and Executive Function /60</i>	105	50.35 (4.78)	39.00–58.80	41.24	78	33.84 (10.07)	11.40–51.80	72 (20:34:2)	27	33.63 (10.23)	11.40–51.80	49	34.08 (10.17)	13.80–51.00
Memory														
<i>Incidental Verbal Memory /10</i>	116	6.46 (1.77)	2–10	3.85	81	4.74 (2.27)	0–9	26 (5:14:2)	29	4.96 (2.62)	0–9	53	4.70 (2.12)	0–8
<i>Total BELS /100</i>	105	85.82 (5.25)	73.50–96.00	75.95	73	65.77 (12.66)	37.40–87.80	78 (18:37:2)	24	66.19 (13.61)	37.40–87.80	47	65.77 (12.45)	37.70–86.50

Table 2. Percentage of patients (and Proportion of LHS: RHS) Impaired on BELS Subsections.

BELS Subsection	Whole Stroke Group		Stroke Group Without Aphasia	
	%<5 th (N LHS: N RHS: N Bilateral)	% of LHS: % of RHS	%<5 th (N LHS: N RHS: N Bilateral)	% of LHS: % of RHS
Oral Apraxia	7% (0:6:0)	0:11	8% (0:6:0)	0:12
Nominal Language	28% (8:16:1)	28:28	23% (5:12:0)	23:24
Propositional Language & Executive Functions	72% (20:34:2)	74:69	67% (14:28:1)	70:65
Memory	26% (5:14:2)	17:26	21% (2:11:1)	10:24
Total BELS	78% (18:37:2)	75:79	77% (13:32:1)	72:78

subtests, controls performed significantly better than stroke patients. When aphasic patients were removed from comparisons, the only changes to results were Sentence Repetition and Motor Go No-GO, which became non-significant after correcting for multiple comparisons ($U = 3818.50$, $p = .007$; and $U = 3732.50$, $p = .012$, respectively). Additionally, to ensure BELS Oromotor Function and Nominal Language impairments were not

driving significantly poorer total BELS scores, a second t-test was conducted with controls ($N = 105$) and patients who were not impaired on these subsections ($N = 49$). Stroke patients' total BELS scores remained significantly lower than controls, $t(56.89) = 9.87$, $p < .001$, $d = 2.20$ (equal variances not assumed). The percentage of patients impaired on overall BELS scores also remained high, at 73%.

Table 3. Inferential statistics for BELS subtests: Comparison of controls and stroke groups.

	Whole Group				Without Aphasia			
	N	t/U	p	d/r	N	t/U	p	d/r
Oromotor Functioning								
Oral Apraxia/10	204	3114.50	<.001	.41	189	2516.50	<.001	.42
Nominal Language								
Word Comprehension/10	204	5046.00	.251	-	189	4234.00	1.000	-
Word Repetition/10	204	5046.00	.251	-	189	4176.00	.207	-
Sentence Repetition/5	204	4419.50	<.001	.24	189	3818.50	.007	-
Oral Naming/10	204	3010.00	<.001	.36	189	2773.00	<.001	.30
Action Naming/10	204	3520.50	<.001	.30	189	3022.50	<.001	.28
<i>Nominal Language Total /20</i>	204	2788.50	<.001	.40	189	2506.00	<.001	.35
Propositional Language and Executive Functions								
Beach WPM	196	7.44	<.001	1.08	181	6.38	<.001	.98
GOAL Cookie WPM	196	7.36	<.001	1.11	181	7.13	<.001	1.10
Spontaneous Speech Total /20	192	8.52	<.001	1.29	177	1500.00	<.001	.50
S WPM	203	2173.00	<.001	0.49	188	1977.50	<.001	.44
GOAL B WPM	203	10.40	<.001	1.48	188	9.01	<.001	1.35
Animal WPM	202	1454.50	<.001	0.61	187	1243.00	<.001	.59
GOAL Fruit/Veg WPM	204	8.47	<.001	1.23	189	7.48	<.001	1.12
Verbal Fluency Total /16	201	1459.50	<.001	.62	186	1347.50	<.001	.59
Initiation Raw/10	204	4227.50	.009	-	189	3670.50	.049	-
HC/5	204	4974.00	.195	-	189	4212.50	.740	-
LC/5	204	4245.50	.009	-	189	3693.50	.056	-
Initiation RT (seconds)	197	-5.64	<.001	.84	182	5412.50	<.001	.31
HC RT	197	6255.50	<.001	.26	182	5161.00	<.001	.25
LC RT	197	-5.55	<.001	.80	182	5424.00	<.001	.31
<i>Initiation Total/10</i>	197	3095.00	<.001	.32	182	2826.00	<.001	.26
Inhibition/10	201	1802.50	<.001	.55	187	1513.00	<.001	.54
HC/5	201	1822.00	<.001	.56	187	1514.50	<.001	.55
LC/5	201	2104.50	<.001	.50	187	1818.50	<.001	.48
Strategy/2	201	6941.50	<.001	.42	187	2178.50	<.001	.48
Sentence Completion /22	194	1510.50	<.001	.58	180	1398.00	<.001	.54
Motor Go No-GO/3	203	4235.00	<.001	.25	188	3732.50	.012	-
<i>Propositional Language and Executive Function /60</i>	183	13.40	<.001	2.20	169	448.50	<.001	.73
Memory								
Incidental Verbal Memory /10	197	5.70	<.001	.86	183	2457.00	<.001	.31
TOTAL BELS								
<i>Total BELS /100</i>	178	12.79	<.001	2.22	165	11.41	<.001	2.22

Note. Where data is non-parametric and unable to be transformed, Mann-Whitney U statistics are reported. Bonferroni correction was applied for control and stroke comparisons ($\alpha = .002$). "r" indicates effect sizes for Mann-Whitney U tests (.10 = small.30 = medium.50 = large), and "d" indicates effect sizes for t-tests (.20 = small.50 = medium.80 = large).

Table 4. ROC curve statistics and cutoffs for BELS total score.

	Sens.	Spec.	Cutoff	AUC	<i>p</i>	95%CI		<i>N</i> Stroke	<i>N</i> Controls
						Upper	Lower		
Whole group	.89	.89	79.25	.94	.000	.91	.98	73	105
Without aphasia	.87	.89	78.75	.94	.000	.89	.98	60	105
LHS & Controls	.88	.81	81.05	.92	.000	.85	.99	24	105
Without aphasia	.83	.81	81.05	.90	.000	.81	.99	18	105
RHS & Controls	.92	.89	79.25	.95	.000	.92	.99	47	105
Without aphasia	.90	.89	78.75	.95	.000	.91	.99	41	105

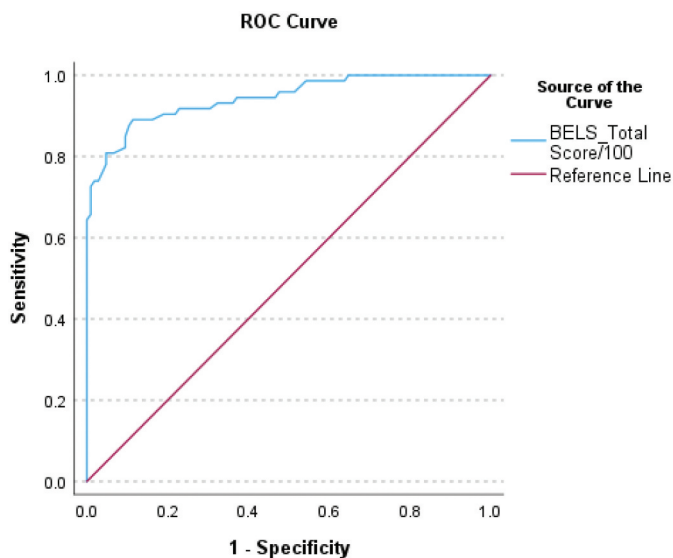
Note. "Sens." = Sensitivity (true positive rate; high sensitivity indicates few false negatives). "Spec." = 1-Specificity (true negative rate; high value indicates few false positives). AUC = Area Under Curve (indicates ability of BELS to differentiate healthy and stroke groups).

Six ROC curve analyses were conducted (see Table 4). With all stroke and control participants included, AUC was .94, $p < .001$, 95%CI [.91-.98] (see Figure 1). A cutoff score of 79.25/100 on the BELS had .89 sensitivity and .89 specificity. The likelihood ratio indicated a score below 79.25 was 8.1 times more likely to be that of a stroke patient than a control. With aphasic patients removed, specificity did not change, and sensitivity reduced slightly (by .02). Sensitivity and specificity were higher for RHS without aphasia (.90 and .89) compared to LHS without aphasia (.83 and .81).

Discussion

Early intervention and rehabilitation of language post-stroke is reliant on administration of sensitive cognitive assessment tools during the early stages of stroke. Screening tools used in stroke are typically either language screens designed to detect aphasia, or non-verbal cognitive screens

designed for patients with aphasia (which is undoubtedly crucial). However, patients without aphasia may still have subtle propositional language impairments that go undetected on language screens, and current widely used cognitive screens only minimally assess the executive functions that underpin the pre-verbal message-generation of connected speech. Following stroke, propositional language deficits and executive dysfunction impacts interpersonal relationships and quality of life.^{8,9,26} The BELS is a recently developed, valid screening tool which – in addition to articulation and memory – measures two cognitive domains (language and executive functions) that predict long-term neuropsychological, functional, and emotional outcomes post-stroke.^{5,8,10–13} We aimed to investigate how acute to early sub-acute stroke patients performed on BELS subtests relative to controls, and to determine sensitivity and specificity of a total BELS cutoff score.

**Figure 1.** ROC Curve for BELS Total Score.

Patients (whole group) and controls did not differ on Word Comprehension or Word Repetition subtests, or on total correct SC Initiation items (after Bonferroni corrections); however, patients performed significantly worse on all other BELS subtests. Impairment on individual subtests ranged from 1% to 52%. Despite largely intact articulation and core nominal language skills, most patients (73%) were impaired on the Propositional Language and Executive Function subscale. With aphasic patients removed from analyses, results remained largely unchanged – the exceptions being Sentence Repetition and Motor Go No-Go becoming non-significantly different between groups (after Bonferroni corrections). Percentages of patients impaired on BELS subsections (and proportions of LHS and RHS groups impaired) remained similar. This aligns with prior evidence that stroke patients without aphasia can experience language impairment at the executive level [i.e. initiation, selection, inhibition, strategy].^{9,37,43} For the SC Initiation subtest, stroke patients demonstrated greater difficulty than controls with selection of multiple competing responses (SC Initiation Low Constraint), compared to selection of a single dominant response (SC Initiation High Constraint). However, this became non-significant after correcting for multiple comparisons. Notably, controls were significantly faster than patients to initiate responses on both high and low constraint items, highlighting difficulties with both selection, and speed at which speech is initiated in acute to early sub-acute stroke. Deficits to idea selection have also been linked to reduced spontaneous speech in dynamic aphasia patients, meaning assessment of these executive processes are necessary to aid in detection and rehabilitation of connected speech.^{13,37,43,46,71} Deficits to selection and initiation of speech can be subtle, and although they may be detected in more extensive testing by speech and language therapists, there are currently no cognitive screening tools (to our knowledge) that capture these processes in an acute stroke setting. Importantly, these deficits can arise in the absence of aphasia which means these patients are not referred to speech and language therapists. For instance, one study found that only 10% of acute stroke patients with cognitive communication disorder were referred for community-based rehabilitation when discharged home, compared to 53% of acute stroke patients with aphasia.⁷²

The BELS was highly sensitive (.89) and specific (.89) when classifying stroke patients and healthy controls, particularly compared to the MoCA which is widely used [sensitivity.81, specificity.70].⁷³ When excluding patients with aphasia, specificity remained the same, and sensitivity remained high at.87. Sensitivity and specificity were higher for RHS (without aphasia) and controls (.90 and.89, respectively), compared to LHS (without aphasia) and controls (.83 and.81, respectively), which aligns with research indicating right hemisphere lesions are more susceptible to executive impairments.^{44,45,74–78} However, we acknowledge the smaller sample of LHS compared to RHS patients, and that with more patients, sensitivity and specificity may increase. For clear first-time lateralized stroke patients, we recommend the LHS and RHS without aphasia cutoffs, and the whole-group cutoff for bilateral or 2nd time stroke patients, or patients with preexisting neurological conditions (e.g. dementia).

The BELS can identify articulation and nominal language impairments, in addition to subtle impairments to executive processes underlying more complex language, making it suitable for a wide range of stroke patients (i.e. non-aphasic patients¹³). This is crucial due to subtle language impairments going undetected on other cognitive and non-verbal stroke screens. The BELS therefore compliments tools like the OCS,¹⁶ OCS-Plus,¹⁷ QAB^{18,19} and CASP,¹⁴ by providing a more complete understanding of patients' propositional language and executive functioning. Consequently, speech and language therapists can intervene early in the rehabilitation process to ensure best possible outcomes and recovery.

Social communication is crucial for interpersonal relationships and the ability to perform social or occupational roles.^{72,79–81} Reduction or impairment of communication post-stroke can impact reintegration into these roles, leading to social isolation and associated negative impacts on general health and wellbeing.^{79,82–87} While the impact of post-stroke aphasia is well-established, the impact of subtle, executive-level communication deficits (e.g. Cognitive Communication Disorder) on wellbeing and quality of life are less-researched, potentially due to a paucity of tools that screen or assess

more complex language production in an acute stroke setting.^{72,88,89}

The BELS is a valid and valuable bedside screening tool that is sensitive to language and executive impairments experienced by stroke patients in the acute to early sub-acute phase, even in the absence of significant aphasia. A limitation to the current study was that if any BELS subtests were not completed, a total score could not be calculated. This meant 15 stroke patients and 11 controls were excluded from the ROC curve analysis. However, total scores for each BELS subsection can still be calculated, and each subsection (except Memory) is standalone, meaning only the section of interest may be administered. It would therefore be beneficial for future studies to determine a cutoff for each BELS subsection. Due to non-parametric data, we were unable to use ANCOVAs to control for Articulation and Nominal Language scores when examining Propositional Language and Executive Functions, and total BELS scores. However, when patients impaired on Articulation and Nominal Language were excluded, the percentage of patients impaired on Propositional Language and Executive Functions and total BELS scores remained high (~75%), and group comparisons remained significant. Similar results were found when patients indicating aphasia on independent measures were removed. Finally, LHS patients may be underrepresented in our sample, likely because they would be expected to have more core language deficits (e.g. non-fluent aphasia) that impact skills required for consent processes (e.g. comprehension).

Future directions include investigation into whether the BELS (particularly the Propositional Language and Executive Function subsection) is sensitive to left versus right hemisphere stroke impairments, and whether BELS scores can predict long-term neuropsychological and functional outcomes. This will better equip rehabilitation teams to identify language and executive impairments, tailor appropriate interventions, and predict and track improvements post-stroke.⁹⁰

Acknowledgments

We wish to thank all participants for their time, especially stroke patients. We also thank Amelia Ceslis, Amelia Hobson, and Katie Veretennikoff for assisting with controls and data collation. This study was supported by the Brazil Family

Program for Neurology and an Australian National Health and Medical Research Council Boosting Dementia Research Leadership Fellowship (APP1135769) awarded to GAR.

Disclosure of Interest

The authors report there are no competing interests to declare.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Funding

This work was supported by the National Health and Medical Research Council [APP1135769].

ORCID

Gail A. Robinson  <http://orcid.org/0000-0002-0078-0364>

References

1. Barker-Collo S, Feigin V. The impact of neuropsychological deficits on functional stroke outcomes. *Neuropsychological Review*. 2006;16(2):53–64. doi:10.1007/s11065-006-9007-5.
2. Hackett ML, Anderson CS. Health outcomes 1 year after subarachnoid hemorrhage: an international population-based study. *Neurology*. 2000;55(5):658–662. doi:10.1212/WNL.55.5.658.
3. Hommel M, Miguel ST, Naegle B, Gonnet N, Jaillard A. Cognitive determinants of social functioning after a first ever mild to moderate stroke at vocational age. *J Neurol Neurosurg Psychiatry Res*. 2009;80(8):876–880. doi:10.1136/jnnp.2008.169672.
4. Hochstenbach JB, Anderson PG, van Limbeek J, Mulder TT. Is there a relation between neuropsychologic variables and quality of life after stroke?. *Arch Phys Med Rehabil*. 2001;82(10):1360–1366.
5. Nys GMS, van Zandvoort MJE, de Kort PLM, Jansen BP, De Haan EH, Kappelle L. Cognitive disorders in acute stroke: prevalence and clinical determinants. *Cerebrovascular Dis*. 2007;23(5–6):408–416. doi:10.1159/000101464.
6. Wagle J, Farner L, Flekkøy K, et al. Early post-stroke cognition in stroke rehabilitation patients predicts functional outcome at 13 months. *Dement Geriatr Cogn Disord*. 2011;31(5):379–387. doi:10.1159/000328970.
7. Warlow CP. Epidemiology of stroke. *Lancet*. 1998;352: S1–S4. doi:10.1016/S0140-6736(98)90086-1.
8. Chahal N, Barker-Collo S, Feigin V. Cognitive and functional outcomes of 5-year subarachnoid

- haemorrhage survivors: comparison to matched healthy controls. *Neuroepidemiology*. 2011;37(1):31–38. doi:10.1159/000328647.
9. Law B, Young B, Pinsker D, Robinson A. Propositional speech in unselected stroke: the effect of genre and external support. *Neuropsychological Rehabilitation: An International Journal*. 2015;25(3):374–401. doi:10.1080/09602011.2014.937443.
 10. Lesniak M, Bak T, Czepiel W, Seniow J, Czlonkowska A. Frequency and prognostic value of cognitive disorders in stroke patients. *Dement Geriatr Cogn Disord*. 2008;26(4):356–363. doi:10.1159/000162262.
 11. Nys GMS, van Zandvoort MJE, de Kort PLM, et al. The prognostic value of domain-specific cognitive abilities in acute first-ever stroke. *Neurology*. 2005a;64(5):821–827. doi:10.1212/01.WNL.0000152984.28420.5A.
 12. Robertson IH, Ridgeway V, Greenfield E, Parr A. Motor recovery after stroke depends on intact sustained attention: a 2-year follow-up study. *Neuropsychology*. 1997;11(2):290–295. doi:10.1037/0894-4105.11.2.290.
 13. Robinson G, Shi L, Nott Z, Ceslis A. A brief executive language screen for frontal aphasia. *Brain Sci*. 2021a;11(3):1–15. doi:10.3390/brainsci11030353.
 14. Benaim C, Wauquiez G, Pérennou D, et al. Cognitive assessment scale for stroke patients (CASP): a multicentric validation study. *Ann Phys Rehabil Med*. 2022;65(3):101594. doi: 10.1016/j.rehab.2021.101594.
 15. Helm-Estabrooks N. *Cognitive Linguistic Quick Test: CLQT*. San Antonio, TX: Psychological Corporation; 2001.
 16. Demeyere N, Riddoch MJ, Slavkova ED, Bickerton WL, Humphreys GW. The Oxford cognitive screen (OCS): validation of a stroke-specific short cognitive screening tool. *Psychol Assess*. 2015;27(3):883. doi:10.1037/pas0000082.
 17. Demeyere N, Haupt M, Webb SS, et al. Introducing the tablet-based Oxford cognitive screen-plus (OCS-Plus) as an assessment tool for subtle cognitive impairments. *Sci Rep*. 2021;11(1):8000. doi:10.1038/s41598-021-87287-8.
 18. Wilson SM, Eriksson DK, Schneck SM, Lucanie JM. Correction: a quick aphasia battery for efficient, reliable, and multidimensional assessment of language function. *PloS One*. 2018;13(6):e0199469–e0199469. doi:10.1371/journal.pone.0199469.
 19. Wilson SM, Entrup JL, Schneck SM, et al. Recovery from aphasia in the first year after stroke. *Brain*. 2023;146(3):1021–1039. doi:10.1093/brain/awac129.
 20. Kertesz A. Western aphasia battery–revised. 2007.
 21. Nasreddine ZS, Phillips NA, Bédirian V, et al. The Montreal Cognitive Assessment, MoCA: a brief screening tool for mild cognitive impairment. *J Am Geriatr Soc*. 2005;53(4):695–699. doi: 10.1111/j.1532-5415.2005.53221.x.
 22. Folstein MF, Folstein SE, McHugh PR. “Mini-mental state”: a practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res*. 1975;12(3):189–198. doi:10.1016/0022-3956(75)90026-6.
 23. Chan E, Khan S, Oliver R, Gill SK, Werring DJ, Cipolotti L. Underestimation of cognitive impairments by the Montreal Cognitive Assessment (MoCA) in an acute stroke unit population. *J Neurol Sci*. 2014;343(1–2):176–179. doi:10.1016/j.jns.2014.05.005.
 24. Chan E, Altendorff S, Healy C, Werring DJ, Cipolotti L. The test accuracy of the Montreal Cognitive Assessment (MoCA) by stroke lateralisation. *J Neurol Sci*. 2017;373:100–104. doi:10.1016/j.jns.2016.12.028.
 25. Blackburn DJ, Bafadhel L, Randall M, Harkness KA. Cognitive screening in the acute stroke setting. *Age Ageing*. 2013;42(1):113–116. doi:10.1093/ageing/afs116.
 26. Pohjasvaara T, Leskelä M, Vataja R, et al. Post-stroke depression, executive dysfunction and functional outcome. *Eur J Neurol*. 2002;9(3):269–275. doi:10.1046/j.1468-1331.2002.00396.x.
 27. Barker MS, Nelson NL, Robinson G. Idea formulation for spoken language: the interface of cognition and language. *J Int Neuropsychol Soc*. 2020;26(2):226–240. doi:10.1017/S1355617719001097.
 28. Levelt WJM. *Speaking from Intention to Articulation*. Cambridge, MA: MIT Press; 1989. doi:10.7551/mitpress/6393.001.0001.
 29. Levelt WJM. Producing spoken language: a blueprint of the speaker. In: Brown CM, Hargroot P, eds. *The Neurocognition of Language*. Oxford, UK: Oxford University Press; 1999: 83–122. doi:10.1093/acprof:oso/9780198507932.003.0004.
 30. Sherratt S. Multi-level discourse analysis: a feasible approach. *Aphasiology*. 2007;21(3–4):375–393. doi:10.1080/02687030600911435.
 31. Barker MS, Young B, Robinson GA. Cohesive and coherent connected speech deficits in mild stroke. *Brain Lang*. 2017;168:23–36. doi:10.1016/j.bandl.2017.01.004.
 32. Alexander MP. Impairments of procedures for implementing complex language are due to disruption of frontal attention processes. *J Int Neuropsychol Soc*. 2006;12(2):236–247. doi:10.1017/S1355617706060309.
 33. Barker MS, Ceslis A, Robinson GA. Idea selection for propositional language production. *Aging, Neuropsychology, and Cognition*. 2022;29(2):260–283. doi:10.1080/13825585.2020.1862044.
 34. Robinson G, Shallice T, Cipolotti L. Dynamic aphasia in progressive supranuclear palsy: a deficit in generating a fluent sequence of novel thought. *Neuropsychologia*. 2006;44(8):1344–1360. doi:10.1016/j.neuropsychologia.2006.01.002.
 35. Robinson GA, Spooner D, Harrison WJ. Frontal dynamic aphasia in progressive supranuclear palsy: distinguishing between generation and fluent sequencing of novel thoughts. *Neuropsychologia*. 2015;77:62–75. doi:10.1016/j.neuropsychologia.2015.08.001.
 36. Barker MS, Robinson GA. Selection for sentence generation in the context of severe anomia: a case series of left temporal patients. *J Clin Exp Neuropsychol*. 2019;41(4):353–363. doi:10.1080/13803395.2018.1562050.

37. Robinson G, Blair J, Cipolotti L. Dynamic aphasia: an inability to select between competing verbal responses. *Brain*. 1998;121(1):77–89. doi:10.1093/brain/121.1.7.
38. Stuss DT, Alexander MP. Is there a dysexecutive syndrome? *Philosophical Transactions of the Royal Society of London. Series B. Biological Sciences*. 2007;362(1481):901–915. doi:10.1098/rstb.2007.2096.
39. Burgess PW, Shallice T. Response suppression, initiation and strategy use following frontal lobe lesions. *Neuropsychologia*. 1996;34(4):263–272.
40. Madden DL, Sale MV, Robinson GA. Age-related differences in idea generation and selection for propositional language. *Aging, Neuropsychology, and Cognition*. 2019;26(4):486–506. doi:10.1080/13825585.2018.1476668.
41. Robinson GA. Primary progressive dynamic aphasia and Parkinsonism: generation, selection and sequencing deficits. *Neuropsychologia*. 2013;51(13):2534–2547. doi:10.1016/j.neuropsychologia.2013.09.038.
42. Robinson G, Shallice T, Bozzali M, Cipolotti L. Conceptual proposition selection and the LIFG: neuropsychological evidence from a focal frontal group. *Neuropsychologia*. 2010;48(6):1652–1663. doi:10.1016/j.neuropsychologia.2010.02.010.
43. Robinson G, Shallice T, Cipolotti L. A failure of high level verbal response selection in progressive dynamic aphasia. *Cogn Neuropsychol*. 2005;22(2005):661–694. doi:10.1080/02643290442000239.
44. Aron AR, Robbins TW, Poldrack RA. Inhibition and the right inferior frontal cortex. *Trends Cogn Sci*. 2004;8(4):170–177. doi:10.1016/j.tics.2004.02.010.
45. Robinson GA, Cipolotti L, Walker DG, Biggs V, Bozzali M, Shallice T. Verbal suppression and strategy use: a role for the right lateral prefrontal cortex? *Brain*. 2015b;138(4):1084–1096. doi:10.1093/brain/awv003.
46. Crescentini C, Lunardelli A, Mussoni A, Zadini A, Shallice T. A left basal ganglia case of dynamic aphasia or impairment of extra-language cognitive processes? *Neurocase*. 2008;14(2):184–203. doi:10.1080/13554790802108380.
47. Gold M, Nadeau SE, Jacobs DH, Adair JC, Rothi LJG, Heilman KM. Adynamic aphasia: a transcortical motor aphasia with defective semantic strategy formation. *Brain Lang*. 1997;57(3):374–393. doi:10.1006/brln.1997.1750.
48. Luria AR. *The Working Brain*. London, UK: Penguin; 1973.
49. Warren JD, Warren JE, Fox NC, Warrington EK. Nothing to say, something to sing: primary progressive dynamic aphasia. *Neurocase*. 2003;9(2):140–155. doi:10.1076/neur.9.2.140.15068.
50. Blake ML, Duffy JR, Myers PS, Tompkins CA. Prevalence and patterns of right hemisphere cognitive/communicative deficits: retrospective data from an inpatient rehabilitation unit. *Aphasiology*. 2002;16(4–6):537–547. doi:10.1080/02687030244000194.
51. Chiti G, Pantoni L. Use of montreal cognitive assessment in patients with stroke. *Stroke*. 2014;45(10):3135–3140.
52. Dong Y, Sharma VK, Chan BP-L, et al. The Montreal Cognitive Assessment (MoCA) is superior to the Mini-Mental State Examination (MMSE) for the detection of vascular cognitive impairment after acute stroke. *J Neurol Sci*. 2010;299(1):15–18. doi:10.1016/j.jns.2010.08.051.
53. Mancuso M, Demeyere N, Abbruzzese L, et al. Using the Oxford cognitive screen to detect cognitive impairment in stroke patients: a comparison with the minimal state examination. *Front Neurol*. 2018;9:101. doi:10.3389/fneur.2018.00101.
54. Nys GMS, van Zandvoort MJE, de Kort PLM, Jansen BPW, Kappelle LJ, de Haan EHF. Restrictions of the mini-mental state examination in acute stroke. *Arch Clin Neuropsychol*. 2005b;20(5):623–629. doi:10.1016/j.acn.2005.04.001.
55. Salvadori E, Pasi M, Poggesi A, Chiti G, Inzitari D, Pantoni L. Predictive value of MoCA in the acute phase of stroke on the diagnosis of mid-term cognitive impairment. *J Neurol*. 2013;260(9):2220–2227. doi:10.1007/s00415-013-6962-7.
56. Shen Y-J, Wang W-A, Huang F-D, et al. The use of MMSE and MoCA in patients with acute ischemic stroke in clinical. *International Journal of Neuroscience*. 2016;126(5):442–447. doi:10.3109/00207454.2015.1031749.
57. Bernhardt J, Hayward KS, Kwakkel G, et al. Agreed definitions and a shared vision for new standards in stroke recovery research: the Stroke Recovery and Rehabilitation Roundtable taskforce. *Int J Stroke*. 2017;12(5):444–450. doi:10.1177/1747493017711816.
58. Nelson HE, Willison J. *National Adult Reading Test (NART) (Pp. 1–26)*. Windsor: Nfer- Nelson; 1991.
59. Bright P, Hale E, Gooch VJ, Myhill T, Van der linde I. The national adult reading test: restandardisation against the Wechsler adult intelligence scale-fourth edition. *Neuropsychol Rehabil*. 2018;28(6):1019–1027. doi:10.1080/09602011.2016.1231121.
60. Mack WJ, Freed DM, Williams BW, Henderson VW. Boston Naming Test: shortened versions for use in Alzheimer’s disease. *Journal of Gerontology (Kirkwood)*. 1992;47(3):154–158. doi:10.1093/geronj/47.3.P154.
61. Abeare K, Cutler L, An KY, Razvi P, Holcomb M, Erdodi LA. BNT-15: revised performance validity cut-offs and proposed clinical classification ranges. *Cognitive and Behavioral Neurology*. 2022;35(3):155–168. doi:10.1097/WNN.0000000000000304.
62. Fastenau PS, Denburg NL, Mauer BA. Parallel short forms for the Boston Naming Test: psychometric properties and norms for older adults. *J Clin Exp Neuropsychol*. 1998;20(6):828–834. doi:10.1076/jcen.20.6.828.1105.
63. Burgess P, Shallice T. *The Hayling and Brixton Tests: Test Manual*. London, UK: Thames Valley Test Company; 1997.
64. Warrington EK, James M. *The Visual Object and Space Perception Battery*. Bury St Edmunds, England: Thames Valley Test Company; 1991.

65. Druks J, Masterson J. *Object & Action Naming Battery*. Hove, U.K: Psychology Press; 2000.
66. Goodglass H, Kaplan E. *Boston Diagnostic Aphasia Examination Booklet*. Lea & Febiger; 1983.
67. Robinson GA, Campbell L, Ceslis A. A goal intervention improves language fluency: evidence from Parkinson's disease and healthy aging. *Medicines*. 2021b;8(3):15. doi:10.3390/medicines8030015.
68. Strauss E, Sherman EM, Spreen O. A compendium of neuropsychological tests: administration, norms, and commentary. *American Chemical Society*. 2006.
69. Luria AR. *Higher Cortical Functions in Man*. New York: Basic Books; 1966.
70. IBM Corp. Released. *IBM SPSS Statistics for Windows, Version 27.0*. Armonk, NY: IBM Corp; 2020.
71. Costello A, Warrington EK. Dynamic aphasia: the selective impairment of verbal planning. *Cortex*. 1989;25(1):103–114. doi:10.1016/S0010-9452(89)80010-3.
72. Heweston R, Cornwell P, Shum D. Cognitive-communication disorder following right hemisphere stroke: exploring rehabilitation access and outcomes. *Top Stroke Rehabil*. 2017;24(5):330–336. doi:10.1080/10749357.2017.1289622.
73. Potocnik J, Ovcar Stante K, Rakusa M. The validity of the Montreal cognitive assessment (MoCA) for the screening of vascular cognitive impairment after ischemic stroke. *Acta Neurol Belg*. 2020;120(3):681–685. doi:10.1007/s13760-020-01330-5.
74. Aron AR, Robbins TW, Poldrack RA. Inhibition and the right inferior frontal cortex: one decade on. *Trends Cogn Sci*. 2014;18(4):177–185. doi:10.1016/j.tics.2013.12.003.
75. Kawashima R, Satoh K, Itoh H, et al. Functional anatomy of GO/NO-GO discrimination and response selection — a PET study in man. *Brain Res*. 1996;728(1):79–89. doi:10.1016/0006-8993(96)00389-7.
76. Konishi S, Nakajima K, Uchida I, Sekihara K, Miyashita Y. No-go dominant brain activity in human inferior prefrontal cortex revealed by functional magnetic resonance imaging. *Eur J Neurosci*. 1998;10(3):1209–1213. doi:10.1046/j.1460-9568.1998.00167.x.
77. Volle E, De Lacy Costello A, Coates LM, et al. Dissociation between verbal response initiation and suppression after prefrontal lesions. *Cereb Cortex*. 2012;22(10):2428–2440. doi:10.1093/cercor/bhr322.
78. Wilson BA, Evans JJ, Emslie H, Alderman N, Burgess P. The development of an ecologically valid test for assessing patients with a dysexecutive syndrome. *Neuropsychol Rehabil*. 1998;8(3):213–228. doi:10.1080/713755570.
79. Abusamra V, Côté H, Joannette Y, Ferreres A. Communication impairments in patients with right hemisphere damage. *Life Span and Disability*. 2009;12:67–82.
80. Brady MC, Clark AM, Dickson S, Paton G, Barbour RS. The impact of stroke-related dysarthria on social participation and implications for rehabilitation. *Disabil Rehabil*. 2011;33(3):178–186. doi:10.3109/09638288.2010.517897.
81. Cruice M, Worrall L, Hickson L. Quantifying aphasic people's social lives in the context of non-aphasic peers. *Aphasiology*. 2006;20(12):1210–1225. doi:10.1080/02687030600790136.
82. Christiansen J, Qualter P, Friis K, et al. Associations of loneliness and social isolation with physical and mental health among adolescents and young adults. *Perspect Public Health*. 2021;141(4):226–236. doi:10.1177/17579139211016077.
83. Evans M, Fisher EB. Social isolation and mental health: the role of nondirective and directive social support. *Community Ment Health J*. 2022;58(1):20–40. doi:10.1007/s10597-021-00787-9.
84. Holt-Lunstad J, Steptoe A. Social isolation: an underappreciated determinant of physical health. *Curr Opin Psychol*. 2022;43:232–237. doi:10.1016/j.copsyc.2021.07.012.
85. Mukherjee D, Levin RL, Heller W. The cognitive, emotional, and social sequelae of stroke: psychological and ethical concerns in post-stroke adaptation. *Top Stroke Rehabil*. 2006;13(4):26–35. doi:10.1310/tsr1304-26.
86. Rohde D, Gaynor E, Large M, et al. The impact of Cognitive Impairment On Poststroke Outcomes: a 5-year follow-up. *J Geriatr Psychiatry Neurol*. 2019;32(5):275–281. doi:10.1177/0891988719853044.
87. Smith KJ, Victor C. Typologies of loneliness, living alone and social isolation, and their associations with physical and mental health. *Ageing and Society*. 2019;39(8):1709–1730. doi:10.1017/S0144686X18000132.
88. Blake ML. Right Hemisphere Strokes. *Perspectives of the ASHA Special Interest Groups*. 2016;1(2):63–65. doi:10.1044/persp1.SIG2.63.
89. Le K, Mozeiko J, Coelho C. Discourse analyses: characterizing cognitive-communication disorders following TBI. *The ASHA Leader*. 2011;16(2):18–21. doi:10.1044/leader.FTR4.16022011.18.
90. Robinson GA, Butterworth B, Cipolotti L. “my mind is doing it all”: no “brake” to stop speech generation in Jargon Aphasia. *Cognitive and Behavioral Neurology*. 2015c;28(4):229–241. doi:10.1097/WNN.0000000000000080.