Sources of phoneme errors in repetition: perseverative, neologistic and lesion patterns in jargon aphasia

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Sources of Phoneme Errors in Repetition: Perseverative, Neologistic and Lesion Patterns in Jargon aphasia

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Abstract

This study examined patterns of neologistic and perseverative errors during word repetition in fluent Jargon aphasia. The principal hypotheses accounting for Jargon production indicate that poor activation of a target stimulus leads to weakly activated target phoneme segments, which are outcompeted at the phonological encoding level. Voxel-lesion symptom mapping studies of word repetition errors suggest a breakdown in the translation from auditory-phonological analysis to motor activation. Behavioural analyses of repetition data were used to analyse the target relatedness (Phonological Overlap Index: POI) of neologistic errors and patterns of perseveration in 25 individuals with Jargon aphasia. Lesion-symptom analyses explored the relationship between neurological damage and jargon repetition in a group of 38 aphasia participants. Behavioural results showed that neologisms produced by 23 jargon individuals contained greater degrees of target lexico-phonological information than predicted by chance and that neologistic and perseverative production were closely associated. A significant relationship between jargon production and lesions to temporoparietal regions was identified. Region of interest regression analyses suggested that damage the posterior superior temporal gyrus and superior temporal sulcus in combination was best predictive of a Jargon aphasia profile. Taken together these results suggest that poor phonological encoding secondary to impairment in sensory-motor integration alongside impairments in self-monitoring result in jargon repetition. Insights for clinical management and future directions are discussed.

Introduction

Neologistic Jargon aphasia is an acquired language disorder characterised by severely distorted speech output. Production in Jargon aphasia is fluent but underspecified and contains numerous nonword errors, rendering it hard to comprehend. Prognosis in Jargon aphasia is poor, with reports of declining vocabulary size and mixed therapeutic outcomes (e.g., Bose, 2013; Eaton et al., 2011; Panzeri et al., 1987; Robson et al., 1998a; 1998b). Perseveration, repeated patterns of phonological distortion, frequently co-occurs with Jargon aphasia and is particularly evident during elicitation tasks such as serial repetition.

Neurobiologically, the repetition of a word requires the transformation of sensory information into motor activation. Traditional neurological accounts of impaired repetition posit damage to the white matter tracts—particularly the arcuate fasciculus—connecting posterior and anterior language areas as the source of breakdown (Geschwind, 1965). Recent neuroimaging and stimulation work has expanded this dorsal network to include cortical regions; namely the inferior supramarginal gyrus (SMG) and posterior superior temporal gyrus (pSTG) (Anderson et al., 1999; Quigg & Fountain, 1999) including area Spt at the boundary of the inferior parietal and superior temporal lobes, which includes portions of the planum temporale (Hickok et al., 2003; 2009; Hickok & Poeppel, 2004). In repetition, the pSTG plays a perceptual role analysing phonetic and phonemic information in the speech stream (Buchsbaum et al., 2001; Deschamps & Tremblay, 2014; McGettigan et al., 2010). This phonological information is transformed into motor responses for articulatory processes, a function proposed to be supported by area Spt (Buchsbaum et al., 2011; Hickok, 2009; Hickok et al., 2011; Hickok & Poeppel, 2004; Warren et al., 2005). Area Spt has direct structural connectivity with motor and frontal regions, including the pars opercularis and premotor cortex which are associated with the articulatory components of speech production (Basilakos et al., 2015; Isenberg et al., 2012; Itabashi et al., 2016). The SMG is also proposed to support encoding for production (Mesgarani et al., 2014; Ravizza et al., 2004; Trébuchon et al., 2013) but is more prominently associated with auditory short-term memory/working memory functions (Henson et al., 2000; Paulesu et al., 1993) which support the temporary maintenance of phonological information during the repetition process.

Convergent with the neurobiological account, cognitive neuropsychological and psycholinguistic models highlight a phonological pathway for repetition. In addition, many models allow a further repetition route via a semantic pathway (Dell et al., 2007; Hanley et al., 2004; Hanley & Kay, 1997; Hillis & Caramazza, 1991; McCarthy & Warrington, 1984; Nozari et al., 2010). Word repetition is commonly impaired in aphasia, and has classically been used as a diagnostic screening test (Kaplan, 1983). However, repetition errors do not occur in all aphasic conditions. For example, individuals with isolated semantic impairment such as those with transcortical sensory aphasia or semantic dementia have preserved repetition abilities (Boatman et al., 2000; Hodges et al., 2008; Jefferies & Lambon Ralph, 2006). Where repetition errors do occur they tend to be phonological in nature, with a comparative scarcity of purely semantic errors (Hanley et al., 2002; Martin, 1996; Martin et al., 1994). These behavioural patterns are consistent with a neurobiological mechanism predominantly engaging sensory-motor integration functions with relatively less weight on semantic processes (Moritz-Gasser & Duffau, 2013). Nonwords are one form of phonological repetition error which are particularly frequent in individuals with Jargon aphasia. Nonwords can range from mild phonemic substitutions of acoustically or articulatory similar phonemes (e.g. village - /vɪlti:/), typically referred to as phonological paraphasias, to severe distortions which bear little resemblance to target phonology (e.g. rocket - /waɪæpəl/), typically referred to as neologisms. Perseverative errors, the repeated intrusion of phoneme strings or syllabic patterns, have been noted to occur alongside neologistic production in Jargon aphasia (Buckingham et al., 1978; Moses et al., 2004). A fourth type of error commonly observed is referred to as a formal error, which occurs when an alteration in the phonological structure of a word results in a real word error (e.g. cot - /kəʊt/). There has been considerable research into the underlying causes of nonword and perseverative errors in repetition and other production modalities. Much evidence points to a single impairment source for paraphasias, neologisms, and perseverative errors, with different error types reflecting a range of severity (Buckingham & Buckingham, 2011; Dell et al., 1997; Martin & Dell, 2007; Olson et al., 2007; 2015; Schwartz et al., 2004). The predominant hypothesis indicates a disruption in lexical and phonological processes, during which weak and aberrantly spreading activation can result in non-target phonology being selected for production. Nonword production is modulated by word length and word frequency, suggestive of a single lexico-phonological source generating errors with a range of severity (Nozari et al., 2010; Olson et al., 2007; 2015). Nonword accuracy range adheres to a normal distribution, thereby suggesting that a single underlying source generates errors of varying severity (Olson et al., 2007). An alternative hypothesis is that paraphasic and neologistic nonwords are independent error types whereby neologisms are produced when lexical retrieval fails, and a random or idiosyncratic phoneme string is generated for output (Buckingham, 1990; Butterworth, 1979; Eaton et al., 2010; Moses et al., 2004). Such production would give rise to two separate error populations; one with very limited target relatedness and the other with high target overlap, thereby conforming to a bimodal distribution.

The source of perseveration errors is also controversial. The predominant hypothesis states that weak target activation or phonological encoding allows recently used, and therefore the most active representations, to override the current target (Ackerman & Ellis, 2007; Buckingham & Buckingham, 2011; Eaton et al., 2010; Hirsh, 1998; Moses et al., 2007a). As such, perseverative, paraphasic and neologistic errors are hypothesised to have a common source. The co-occurrence of perseverative and non-perseverative nonword errors supports this hypothesis (Martin & Dell, 2007; Moses et al., 2007b). An alternative hypothesis posits that errors arise from disruption of inhibitory processes, and a failure of post-activation suppression (Papagno & Basso, 1996; Sandson & Albert, 1984; Santo Pietro & Rigrodsky, 1986; Stark, 2007; Yamadori, 1981). Concurrent inhibition and encoding deficits have been identified in some dysgraphic individuals indicating that these mechanisms are not mutually exclusive (Fischer-Baum & Rapp, 2012). However, it is unclear whether such inhibitory mechanisms are a specific feature of the phonological encoding system or a domain-general cognitive function, and whether different mechanisms operate more strongly in different subtypes of aphasia. A significant challenge in distinguishing between nonword error and perseveration hypotheses within the neologistic Jargon aphasia population comes from the relative rarity of the condition, which has resulted in small scale case-series investigations or single case studies. This results in difficulty applying psycholinguistic patterns to the wider Jargon aphasia population.

Despite this, evidence from lesion-symptom mapping is currently consistent with the proposed impairment in phonological encoding put forward by computational modelling and neuropsychological investigations. Repetition errors in chronic aphasia have been associated with lesions affecting the left inferior parietal lobe (Fridriksson et al., 2010), the left posterior temporo-parietal cortex (Baldo et al., 2012), and area Spt (Rogalsky et al., 2015) similarly interpreted as a disruption to sensory-motor integration (including phonological encoding). However, lesion-symptom mapping, modelling and neuropsychological evidence is not currently directly comparable. Lesion-symptom mapping repetition studies currently contain few or no individuals with jargon-type repetition impairments, and predominantly include those with conduction-like repetition deficits (Baldo et al., 2012; Rogalsky et al., 2015), reducing the applicability of these results to the jargon population. As such the possibility remains that more "peripheral" aspects of the repetition system, such as perceptual auditory-phonological or articulatory processing, may contribute to jargon repetition. An impairment in perceptual analysis is consistent with the majority of individuals with Jargon aphasia also displaying Wernicke's-type aphasia associated with auditory-phonological processing impairments (Robson et al., 2012; 2013; 2014) and the association of neologistic production and impairments in self-monitoring (Kinsbourne & Warrington, 1963; Maher et al., 1994; Marshall et al., 1998). Perceptual and articulatory processes are also not captured in computational modelling which focuses on core linguistic components of semantic, lexical and phonological processing. A further possibility is that no single process or neural region results in the deficit. Rather, jargon repetition may occur following damage to multiple components of the repetition network, resulting in the severe distortions observed in the condition. Investigating the lesion profiles associated with nonword and perseverative errors in a large cohort is required to explore these hypotheses.

In the current study we use a combination of psycholinguistic and lesion-symptom mapping analyses to explore the cognitive and neurobiological underpinnings of jargon repetition deficits. The target relatedness and distribution of nonword errors are analysed to distinguish the default generation and phonological encoding hypotheses. Patterns of perseveration are examined, and the co-occurrence of perseveration and non-perseveration errors is explored to determine whether these error types share a common source. Whole brain and region of interest lesion-symptom mapping analyses are used to explore the contribution of the wider dorsal repetition network to neologistic Jargon aphasia.

Method

Ethical approval for the current study was given by the Multicenter NHS Research Ethics Committee, the NHS East of England Research Ethics Committee and the University of Reading School of Psychology Research Ethics Committee.

Participants

We report data from 46 individuals with aphasia (female n=15), mean age 69.7 years (σ = 12.24; range = 31-93), mean time post onset 35 months (σ = 47.63; range = 5-204), see Table 1. Aphasia profile was assessed with the Boston Diagnostic Aphasia Examination – Short Form (Goodglass et al., 2001). Percentile scores for auditory comprehension, repetition (word and sentence) and fluency subtests are presented in Table 1. Twenty individuals presented with Wernicke's aphasia, four with conduction aphasia, four with anomic aphasia, and two with transcortical sensory aphasia. In the nonfluent categories, four participants were classified as Broca's type aphasia, with one individual classified as transcortical motor aphasia. Four individuals were classified as mixed aphasic, and the remaining six were unable to be classified as the necessary BDAE data were unavailable. Different individuals were entered into behavioural and neuroimaging analyses based on analysis criteria discussed below.

Neuroimaging

Neuroimaging data were available for 38 participants (see Table 1). 3T T1w research MRI scans were collected for 27 individuals. Scans were collected across different studies and, as a result, protocols varied. Clinical imaging scans were available for the remaining 11 participants. Only scans which were carried out after 24 hours post stroke onset were included in the analysis to avoid significant underestimation of the extent of the stroke. Lesions were manually delineated by lesion drawing in native space. The native lesion masks were used for cost-function masking during normalisation. Normalisation was implemented in the SPM Clinical toolbox (Rorden et al., 2012). Normalisation parameters were applied to the native lesion masks which were subsequently manually checked for normalisation accuracy. Lesion overlap maps for the whole aphasia group and for the Jargon aphasia subgroup are presented in Figure 1A & 1B. Lesions were observed throughout the entire left MCA territory in the aphasia group as a whole with peak lesion overlap in the temporoparietal junction including the superior temporal gyrus and sulcus and supramarginal gyrus, in both the whole group and Jargon subgroup.

Table 1: Demographic, imaging and BDAE information

					BDAE centiles		
Pt code	Age (years)	Time post stroke (months)	Gender	Imaging	Comprehension	Fluency	Repetition
1	55	24	M	3T	n/a	n/a	n/a
2	70	96	M	3T	n/a	n/a	n/a
3	80	8	F	Clinical CT	30	n/a	n/a
4	54	145	F	n/a	38	n/a	45
5	56	22	M	3T	77	n/a	15
6	75	132	F	3T	58	7	60
7	63	144	M	3T	48	13	40
8	31	15	M	n/a	48	20	20
9	68	108	M	3T	87	30	65

10	81	8	M	Clinical CT	15	30	25
11	59	14	M	3T	10	38	13
12	68	24	M	3T	9	42	7
13	65	108	F	3T	50	48	45
14	74	6	M	n/a	12	51	13
15	69	15	F	n/a	33	55	10
16	72	204	M	3T	100	62	60
17	73	6	M	3T	3	63	<1
18	62	84	M	3T	n/a	63	n/a
19	78	72	F	3T	5	68	10
20	53	7	M	n/a	15	68	<1
21	64	6	M	n/a	10	68	15
22	66	10	M	3T	5	70	25
23	49	24	F	3T	70	70	60
24	81	7	F	Clinical CT	18	75	15
25	85	9	F	n/a	<1	75	<1
26	86	13	M	3T	10	80	7.5
27	88	9	M	Clinical MRI	42	80	65
28	73	13	F	3T	10	83	10
29	60	5	M	3T	7	84	8
30	77	24	M	3T	40	90	25
31	71	72	M	3T	7	90	1
32	70	42	M	3T	45	100	28
33	59	6	M	3T	17	100	20
34	75	12	M	Clinical MRI	28	100	5
35	78	9	F	3T	73	100	80
36	83	9	F	Clinical CT	48	100	60
37	93	9	F	Clinical CT	67	100	80
38	68	9	M	Clinical CT	55	100	50
39	80	9	F	n/a	25	100	20
40	71	9	M	Clinical MRI	50	100	80
41	82	9	M	Clinical MRI	64	100	30
42	76	14	M	3T	13	100	1
43	74	9	M	3T	57	100	50
44	57	9	M	3T	15	100	10
45	86	13	F	Clinical CT	3	100	15
46	49	5	M	3T	67	100	60

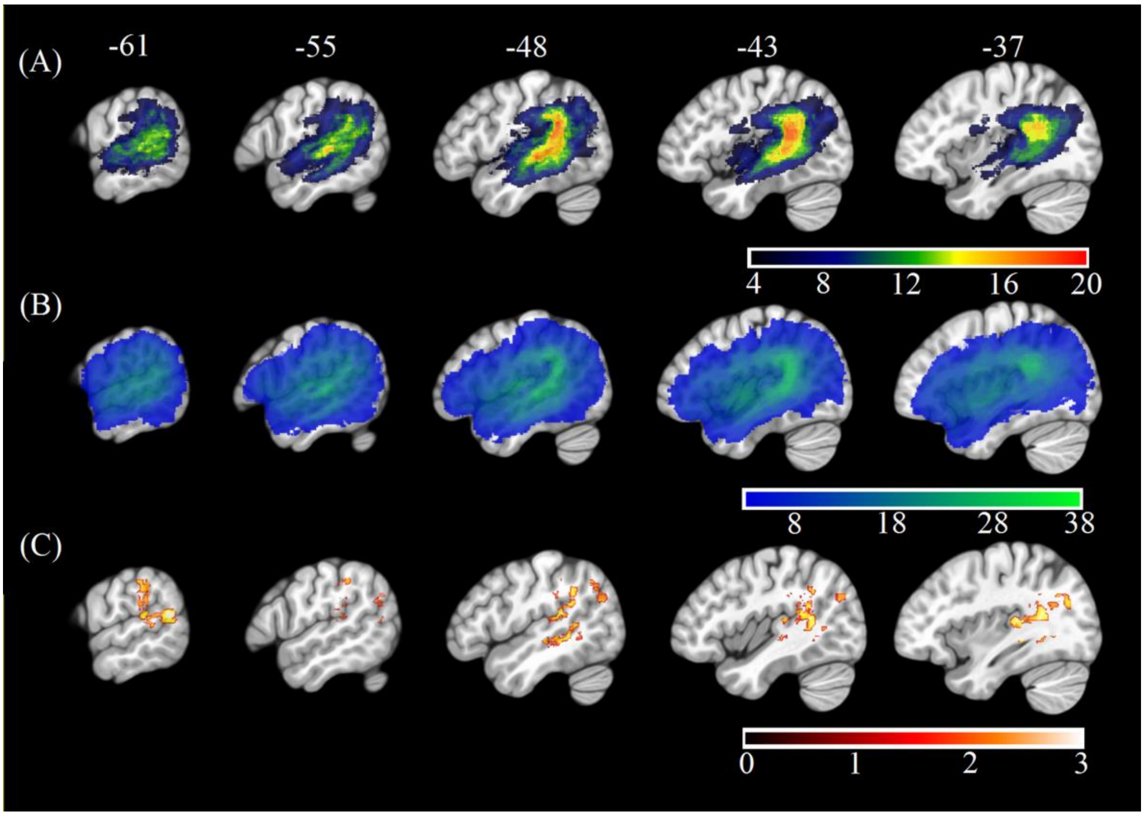

Figure 1: Lesion Overlap Maps and VLSM Results

Repetition tasks

All participants completed an 80 item word repetition task. Sixteen participants completed the word repetition test from the PALPA (Psycholinguistic Assessment of Language Processing in Aphasia, subtest 9: Kay et al., 1996) and 30 participants completed an in-house 80 item repetition test. The 80 items were administered either continuously or in shorter blocks if a participant was perceived to require a break. The experimenter provided repetitions when requested.

Recording and error coding

All response data were transcribed into broad phonemic transcription. When multiple responses were given per item, the final stressed response was accepted. All transcriptions were then converted into DISC symbols (1:1 phoneme: symbol correspondence, e.g. IPA = [i:], DISC = [i]); to enable automated data extraction via Microsoft excel and MATLAB. Responses were categorised following criteria used by Moses, Nickels, and Sheard (2004). Non-lexical responses were classified as nonwords. Lexical errors were labelled according to their target relationship, and were classed as either formal (either identical first phoneme or fifty percent phonology overlap with target), semantic (semantically related to target), mixed (semantically and phonologically related to target word form), unrelated (real word error that did not share an obvious relationship to target), no response (individual indicated they could not provide an answer or did not respond); or circumlocution (individual provides information about the item by talking around it but not naming it).

Analysis summary

Four different analyses were undertaken to explore behavioural patterns in jargon production. Phonological accuracy of nonwords was explored using the Phonological Overlap Index measure (POI: Schwartz et al., 2004), and nonword accuracy distributions were examined using the Kolmogorov-Smirnov test. Perseverative patterns were analysed using the Intrusion Perseveration Probability (IPP) measure, adapted from Cohen and Dehaene (1998), and the relationship between perseverative and non-perseverative nonwords was explored, using a correlation analysis. Voxel-lesion symptom mapping and follow-up region-of-interest (ROI) analyses were used to investigate the relationship between jargon production and lesion profiles.

Phonological accuracy in neologistic Jargon

The degree to which neologistic errors are produced with reference to target phonology was investigated using the POI measure (Bose, 2013; Schwartz et al., 2004). The POI for each non-word repetition response was calculated using the formula:

$$\boldsymbol{POI} = (\boldsymbol{n\ phonemes\ shared\ between\ target\ and\ response})\boldsymbol{x\ 2/} (\boldsymbol{n\ phonemes\ in\ target} + \boldsymbol{n\ phonemes\ in\ response}).$$

A value of 0 indicates no overlap with target phonology and a value of 1 indicates complete overlap between the target and response. Non-word responses were then assigned to a paraphasic (>0.51 POI) or neologism (≤0.5 POI) error category (Schwartz et al., 2004). The target relatedness of neologistic errors was compared to a chance rate derived from null distributions. In each null distribution, all non-word errors from all participants were randomly reassigned to a new target and a new POI calculated. To statistically compare individual and chance accuracy an equal number of resampled responses as neologistic errors were randomly extracted for each participant. The observed POI mean was compared against each resampled POI mean to derive a level of significance.

Nonword accuracy distributions

The accuracy (POI) distribution of both nonword error types (paraphasias and neologisms) was examined using the one sample Kolmogorov-Smirnov (KS) test of normality, in order to examine whether distributions adhered to a normal curve and conformed to the single source hypothesis.

Perseveration

The Intrusion Perseveration Probability (IPP) measure, adapted from Cohen and Dehaene (1998), calculates how often a phonological error occurs in each of the previous ten responses. To calculate it, every intruded/erroneous phoneme was identified. Then, how often each of these intruded phonemes was present (matched) in each of the previous ten responses was measured. The probability was calculated by dividing the number of matched phonemes at each lag by the total number of intruded phonemes. The average IPP across the ten lags was calculated so as to assign each individual with a perseveration value, representative of persistent patterns of phoneme intrusions. To account for breaks in administration, data were split into blocks of twenty responses, and only responses eleven to twenty were analysed in relation to the previous ten responses. This method provided 40 trials per individual for analysis. Both correct and incorrect responses were included in the analysis. Six individuals (4, 12, 26, 41, 43, 44) were excluded from this analysis because their data could not be split into blocks of twenty.

Chance perseveration

To interpret the prevalence of perseveration within the Jargon aphasia group, observed IPP values were compared against a chance rate. In the current study, all responses from all participants were randomly reassigned to a new target to create a null distribution, and forty trials were randomly selected to undergo IPP analysis. This process was repeated 1000 times. The observed IPP score was compared against each resampled IPP score to derive a level of significance.

Relationship between perseverative and non-perseverative nonwords

For individuals who presented with fluent Jargon aphasia, the number of perseverative nonword errors was calculated using criteria from Martin and Dell (2007). A nonword was identified as a perseveration when a phoneme error was present in the previous response. Otherwise, the nonword was labelled as a non-perseveration. To accommodate administration breaks, the initial response in each subset was discounted. The association between perseverative and non-perseverative nonwords was examined using Spearman's rank correlation.

Voxel-lesion symptom mapping

All participants with an available clinical or 3T T1w image were included in an exploratory voxel-lesion symptom mapping analysis implemented in the vlsm2 matlab toolbox (version 2.3; Bates, et al., 2003). This analysis uses a mass univariate general linear model approach to determine the relationship between the presence of lesion and behaviour at each voxel while accounting for total lesion volume. The analysis was constrained to the left hemisphere grey and white matter regions. Results were obtained at thresholds of 0.05 and 0.01 and compared to those obtained from 1000 permutations/null distributions. The VLSM analysis was extended using an ROI analysis. VLSM clusters significant at $p<0.05$ and greater than 200 voxels were identified and the percentage lesion overlap with each cluster was extracted in each participant. ROI data were used to identify the consistency of lesion-behaviour associations and the strongest predictors of jargon repetition.

Results

Overall accuracy and error patterns

All but 4 individuals (participants 7; 38; 40; 46) displayed a repetition impairment ($\bar{x}$ = 35 σ = 23.24, range = 1-73; see Table 2). Individuals with anomic aphasia were the most accurate as a group ($\bar{x}$ = 54; σ= 28.62), followed by those with Broca's aphasia ($\bar{x}$ = 49.7; σ = 19.40), then conduction aphasia ($\bar{x}$ = 43.3; σ = 25.16). Those with Wernicke's aphasia were the least accurate as a group ($\bar{x}$ = 21.45; σ = 16.30). Across all participants, the predominant error types were nonwords (1288, 35%) and formal errors (304, 8%). The remaining four error categories (unrelated, semantic, circumlocution, no response) contributed just over 7% of the overall response rate. POI analysis indicated roughly equal numbers of neologistic and paraphasic errors (medians; paraphasias = 14; neologisms = 8.5; Mann Whitney U = 875.5; p = .153). Participants who presented with fluent speech and produced 5 or more neologistic errors during repetition were considered to present with neologistic Jargon aphasia; 25 participants met these criteria.

Table 2: Number of each response type on single word repetition task.

			Nonwords					
Pt code	Test	Correct	Paraphasia	Neologism	Formal	Unrelated	No response	Other
17	Palpa	1	8	71	0	0	0	1
44	DV	3	12	59	3	3	0	0
45	DV	4	20	38	8	8	1	2
20	Palpa	6	18	44	4	6	2	2
21	Palpa	6	17	51	1	4	1	2
19	Palpa	8	25	30	3	6	7	0
22	Palpa	8	38	22	7	3	2	0
26	Palpa	8	19	33	9	11	0	1
12	DV	10	21	28	12	9	0	1
28	Palpa	16	18	32	9	1	2	2
11	Palpa	17	19	19	7	3	15	2
31	DV	19	8	1	7	1	43	1
42	DV	19	15	23	13	9	0	1
41	DV	20	30	16	11	2	0	0
25	Palpa	21	34	17	5	1	0	0
34	DV	23	10	30	13	4	0	0
10	DV	25	16	18	16	4	0	0
30	Palpa	25	26	17	6	4	0	0
29	Palpa	26	9	14	6	6	18	0
5	DV	33	30	6	6	4	0	0
9	DV	36	25	9	7	3	0	0
6	DV	37	14	4	21	0	3	0
14	Palpa	37	21	9	7	3	1	1
15	DV	39	15	9	16	1	0	0
8	DV	40	6	0	3	2	29	1
32	Palpa	40	21	8	6	3	1	1
43	DV	40	14	11	11	1	1	0
3	Palpa	42	19	6	10	2	0	0
33	Palpa	42	14	5	2	1	15	0
39	DV	49	17	1	11	1	0	1
24	Palpa	50	12	13	3	0	0	0
27	DV	60	8	0	11	1	0	0
1	DV	61	8	2	8	0	0	0
4	DV	62	11	0	6	1	0	0
2	DV	66	5	1	6	0	0	1
13	DV	67	7	0	4	1	1	2

35	DV	69	4	1	4	2	0	0
23	DV	70	7	0	2	1	0	0
36	DV	71	4	1	4	0	0	1
16	DV	72	4	0	3	0	1	1
37	DV	72	2	1	5	0	0	0
18	DV	73	3	1	3	0	0	1
40	DV	76	1	0	3	0	0	0
7	DV	78	2	0	0	0	0	2
38	DV	78	0	0	2	0	0	1
46	DV	80	0	0	0	0	0	0
Total (#)		**1805**	**637**	**651**	**304**	**112**	**143**	**28**
			1288					

Ordered by fewest correct responses

Phonemic content of neologisms

Chance POI was calculated as 0.18 (± 0.01) independent of the number of samples extracted from each null distribution (see methods). The mean POI of neologisms produced by 23 Jargon individuals was greater than the chance prediction ($p \leq .007$; see Table 3). Two individuals (33, 44) could not be differentiated from chance ($p \geq .066$; see Figure 2).

Nonword accuracy distributions

The POI of all nonwords (paraphasias and neologisms) produced by 20 neologistic individuals adhered to a normal distribution ($p \geq .067$). Nonword POI distributions exhibited by individuals 41, 30, 22, 12, and 44, violated the normal distribution ($0.124 \leq KS \leq 0.211$; $p \leq .05$; see Table 3). Individual 12 produced a bimodal distribution and individual 44 exhibited a left skew (see supplementary materials). Histograms for these five individuals are presented in supplementary materials.

Table 3: Test statistics for Phonological Overlap Index (POI) and distribution analyses

Pt code	Mean POI	*p* value	KS stat[a]
33	0.29	0.066	0.148
3	0.39	≤0.001	0.146
32	0.44	≤0.001	0.154
14	0.34	≤0.001	0.154
15	0.39	≤0.001	0.127
43	0.32	0.007	0.149
24	0.42	≤0.001	0.18
29	0.37	≤0.001	0.16
41	0.35	≤0.001	0.211***
25	0.34	≤0.001	0.109
30	0.35	≤0.001	0.163**

10	0.32	0.002	0.102
11	0.33	≤0.001	0.12
22	0.36	≤0.001	0.124*
42	0.35	≤0.001	0.121
12	0.27	0.003	0.212***
19	0.35	≤0.001	0.12
34	0.26	0.006	0.096
28	0.32	≤0.001	0.09
26	0.30	≤0.001	0.09
45	0.26	0.004	0.11
20	0.29	≤0.001	0.093
21	0.27	≤0.001	0.072
44	0.20	0.149	0.159***
17	0.24	≤0.001	0.09

Note. * = $p \leq .05$; ** = $p \leq .01$; *** = $p \leq .001$.
[a]*Kolmogorov-Smirnov test statistic*

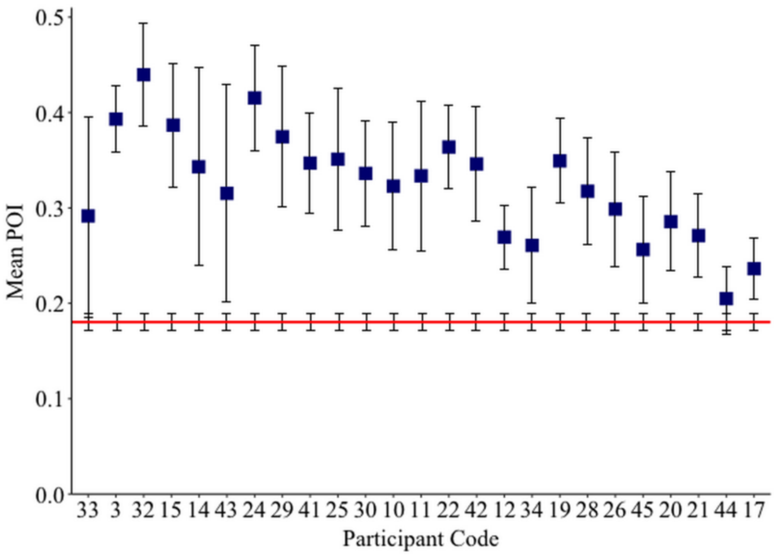

Figure 2: Mean neologism Phonological Overlap Index (POI) score per Jargon individual (squares), and the mean chance POI estimate (red line). Error bars show 95% confidence intervals.

Perseveration results

The IPP measure quantifies how frequently intruded phonemes occur over the previous ten responses. This analysis was applied to individuals with fluent Jargon aphasia for whom suitable data were available (n=25). The perseveration probability scores observed across lags one to ten were averaged to derive a single IPP (perseveration) score. Individual IPP scores were compared against the null chance distributions. Thirteen individuals (3, 28, 39, 22, 20, 25, 34, 30, 42, 45, 19, 21, 17) produced perseveration at significantly greater rates than the chance prediction ($p \leq 0.039$; see Table 4). The remaining twelve individuals did not perseverate at above the chance prediction ($p \geq 0.054$; see Figure 3).

Table 4: Test statistics for Intrusion Perseveration Probability (IPP) analysis.

Pt code	IPP mean	*p* value
31	0.01	1
27	0.13	0.963
36	0.13	0.949
10	0.14	0.905
15	0.16	0.527
14	0.17	0.467
33	0.17	0.395
37	0.17	0.337
29	0.18	0.269
32	0.18	0.219
11	0.19	0.068
24	0.20	0.054
3	0.20	0.039
28	0.21	0.029
39	0.21	0.028
22	0.21	0.007
20	0.24	≤0.001
25	0.24	≤0.001
34	0.25	≤0.001
30	0.26	≤0.001
42	0.28	≤0.001
45	0.28	≤0.001
19	0.29	≤0.001
21	0.29	≤0.001
17	0.57	≤0.001

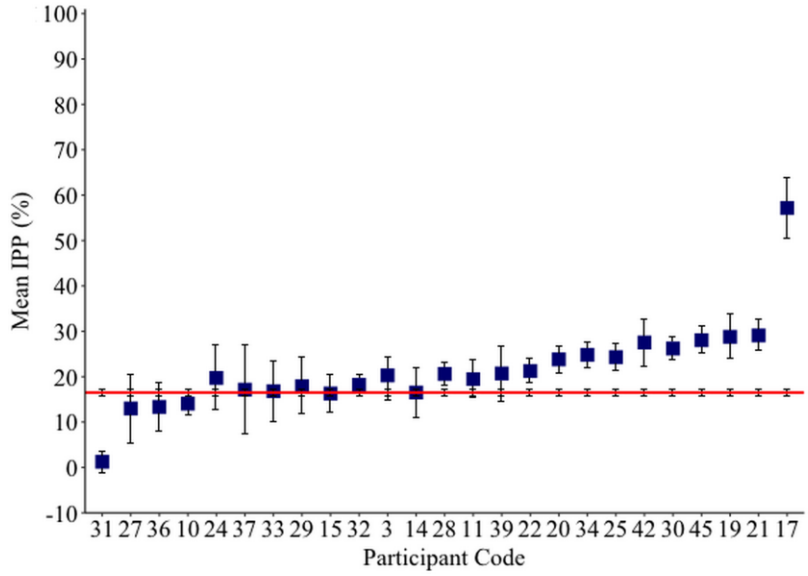

Figure 3: Mean Intrusion Perseveration Probability (IPP) score per Jargon individual (square), and IPP chance estimate (red line). Error bars show 95% confidence intervals.

Relationship between perseverative and non-perseverative nonwords

Nonword errors were coded as a perseveration if an intruded phoneme was present in the previous response. Remaining nonwords were coded as non-perseverative errors. A correlation analysis revealed a significant positive relationship between rates of perseverative and non-perseverative nonwords (ρ = 0.557, p = .001; see Figure 4). The size of this effect increased from moderate to large when the two outlying individuals (17 and 44) were removed (ρ = 0.749, $p \leq .001$).

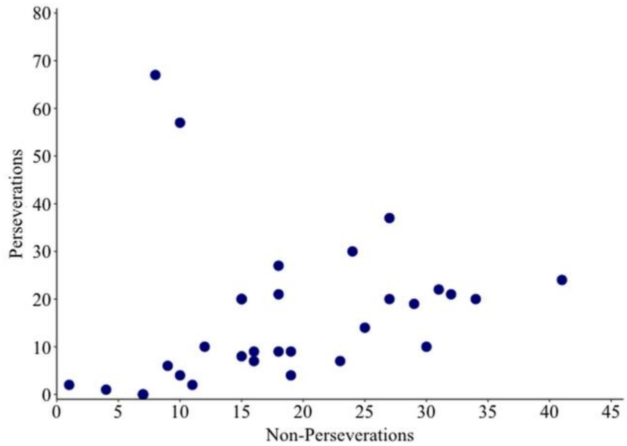

Figure 4: Scatter plot showing the relationship between numbers of perseverative and non-perseverative nonwords.

Lesion-Symptom mapping

The Jargon aphasia group were combined with a wider aphasia group for whom neuroimaging data were available to explore the relationship between lesion and jargon repetition. All but four participants (7; 38; 40; 46) in the wider aphasia group displayed a degree of repetition impairment; however, these impairments were only considered Jargon in

25 participants. As well as the significant relationship between perseverative and non-perseverative errors, Pearson correlation analyses displayed strong to medium relationships between overall repetition accuracy, number of neologistic errors, number of paraphasic errors and total number of intruded phonemes, see Table 5. Principal component analysis was used to derive a summary score representing number of neologisms, paraphasias and intruded phonemes (jargon score) which was entered into the VLSM analysis as the continuous dependent variable.

VLSM analysis identified lesion clusters associated with the jargon score in the posterior temporal and inferior parietal lobe, Figure 1C. These regions included the grey and white matter of the posterior superior temporal gyrus (STG), including areas Spt, the posterior superior temporal sulcus (STS), grey matter of the inferior parietal lobe (IPL) including the supramarginal gyrus (SMG) and white matter at the temporal-parietal boarder. These clusters remained significant at p = .01, (see Table 5), however did not survive permutation correction.

Table 5: Correlations coefficients displaying medium-strong relationships between jargon score components

		Number Neologisms	Number Paraphasias	Total Intruded Phonemes
Repetition Accuracy	r value	-0.799	-0.709	-0.671
	p value	<0.001	<0.001	<0.001
Number Neologisms	r value		0.363	0.851
	p value		.023	<0.001
Number Paraphasias	r value			0.324
	p value			.044

Table 6: Peak VLSM results, threshold ≤0.01

Region	MNI Coordinate
Posterior Superior Temporal Gyrus	-42 -50 15 -50 -36 17 -58 -57 16
Superior Temporal/Inferior Parietal Lobe	-33 -37 15
Supramarginal Gyrus	-50 -46 33

Posterior Superior Temporal Sulcus	-45 -50 9

Significant clusters occurred in regions of high lesion overlap, (see Figure 1B). Therefore, follow-up ROI analyses were used to explore consistency of the VLSM results across the aphasia group. Four neuroanatomically constrained clusters were identified from the VLSM analyses: 1) White matter of the STG and IPL; 2) STS; 3) Grey matter of IPL including SMG and 4) Grey matter of the STG, (see Figure 5). The number of lesion voxels in each ROI was identified for each participant and participants were separated into low overlap (<30% ROI voxels lesioned) or high overlap (>30% ROI voxels lesioned). T-tests were used to compare the jargon score between the high and low overlap groups in each ROI. There was no significant difference in jargon score for clusters 1 and 2. There was a significant difference in jargon scores between the high and low overlap groups in cluster 3, IPL ($t_{(36)} = 2.0$, p = .049), and a borderline significant difference in cluster 4, STG grey matter ($t_{(36)} = 1.77$, p = .085), (see Figure 5).

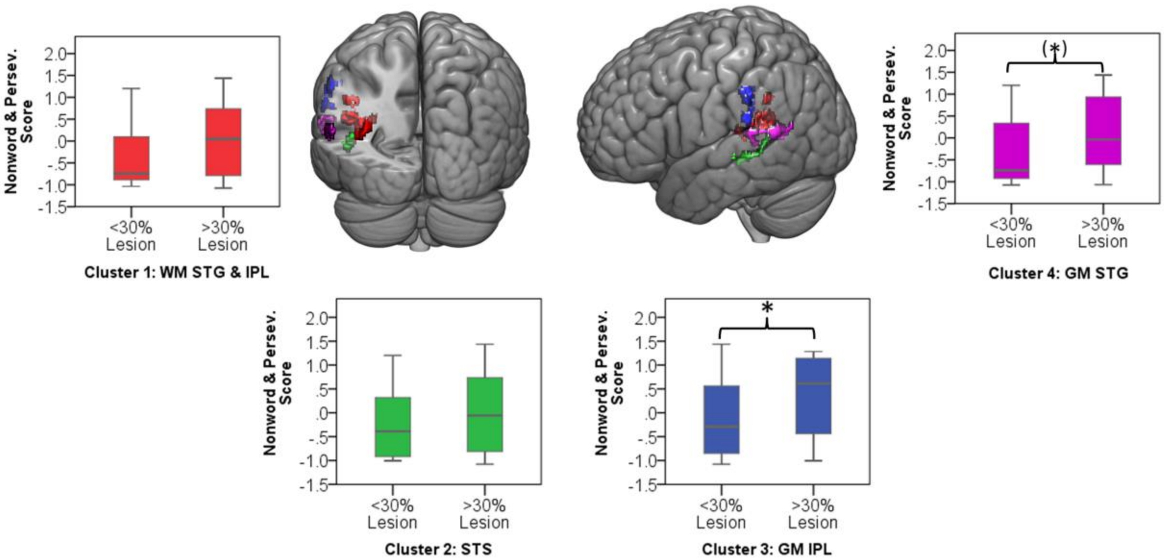

*Figure 5: Colour areas display four regions of interest derived from VLSM clusters. Graphs indicate jargon score for low and high lesion overlap group in each ROI. Ceiling performance on jargon score = -1.07. * = significant group difference; (*) = borderline significant group difference. WM = white matter; GM = grey matter; STG = superior temporal gyrus; IPL = inferior parietal lobe; STS = superior temporal sulcus.*

A regression analysis was performed to investigate whether a combination of lesions was most predictive of jargon production. The centred percentage lesion overlap of each ROI and the two-way interaction between ROIs were added as predictor variables alongside age, time post onset at testing and total lesion volume into a linear regression; jargon score was the dependent variable. Interaction terms were calculated by multiplying percentage of lesion in each cluster e.g. percentage overlap in cluster 1 x percentage overlap in cluster 2. Predictors in the model displayed sufficient collinearity tolerance; the minimum tolerance value outside interaction predictors was 0.2. The regression returned a borderline significant model ($F_{12,25} = 2.05$, adjusted $R^2 = 0.253$, p = .063). Time post onset was a significant predictor (t = -2.3, p = .03) indicating that the greater time post onset the less jargon production. Lesions in isolated clusters did not significantly contribute to the model, however the interaction between cluster 2 (STS GM) and cluster 4 (STG GM) was a significant predictor (t = 2.3, p = .03) indicating that jargon was more severe when lesions affected both the STS and STG.

Discussion

The aim of the current study was to explore, side by side, the behavioural and neurological patterns associated with repetition deficits in Jargon aphasia. Behavioural analyses identified the target relatedness of phonological distortions, and explored the effect of phoneme perseveration in Jargon repetition. Correlation analyses exposed the relationship between perseverative and non-perseverative errors. Lesion analyses were used to identify neurological regions and patterns of damage associated with jargon repetition. Results support the hypothesis that weak activation of target phonology results in neologistic production. Individuals with increasingly severe production deficits showed greater degrees of perseveration, and there was a clear association between the occurrence of perseverative and non-perseverative nonwords, suggesting that both error types arise from a common mechanism. Lesion analyses converge with this interpretation and, additionally, implicate a contribution of impairments in analysis and maintenance of auditory information to jargon repetition.

Psycholinguistic models account for nonword errors in Jargon aphasia through a breakdown in phonological encoding, whereby activation is not effectively transferred from the lexical to the phonological level (Dell, 2014; Marshall, 2006; Olson et al., 2007; 2015; Schwartz et al., 2004). Therefore, phonological and neologistic errors are accounted for by the same mechanism with differing degrees of breakdown severity. However, some evidence has pointed towards a random or default phonological activation pattern for some individuals, hypothesised to arise when lexical retrieval fails, (Butterworth, 1979; Eaton et al., 2010; Moses et al., 2004). Phonological Overlap Index (POI) analysis of the neologisms produced by the 25 participants with neologistic Jargon aphasia in the current study is largely consistent with the phonological encoding hypothesis and does not provide direct support for the default phonology hypothesis. The phonological overlap between neologisms and targets, although by definition low, was significantly above chance for 23 of the 25 neologistic Jargon aphasia participants, indicating a post-lexical retrieval breakdown. However, a large cluster of nonword errors with very limited target overlap occurring alongside errors with greater target relatedness would provide evidence for an additional lexical retrieval failure and default phonological production source. To investigate this hypothesis, the POI distribution across all nonword errors was analysed. Only five individuals violated the normal distribution and only one of these participants (individual 12) displayed evidence of a separate cluster of nonword errors with limited target overlap, thus conforming to the two deficit account. However, caution must be taken in this interpretation in that the pattern could be accounted for by a large number of perseverative responses which were not distinguished within the POI or accuracy distribution analyses. For example, individual 44 exhibited a left skew indicating that most of their nonwords had very limited target overlap, and the POI analysis identified the accuracy of individual 44 as at chance. However, the correlation analysis indicated that individual 44 was highly perseverative, thus it is probable that their skewed POI distribution and neologistic accuracy is contaminated by perseveration.

A perseveration error is thought to occur when poor activation of target phonology allows recently used segments to compete and intrude. Therefore, perseveration errors are proposed to share a source with other nonword jargon errors (Buckingham & Buckingham, 2011; Martin & Dell, 2007). In the current study 25 individuals with Jargon aphasia had suitable data for IPP perseveration analysis. Thirteen of these individuals displayed perseveration at a significantly greater level than the chance prediction, demonstrating that perseveration was a common but not universal feature of Jargon aphasia. Correlation analysis conformed to previous data (e.g. Martin & Dell, 2007) showing that nonword perseverative and non-perseverative error rates are strongly associated, indicating a common error source. Taken together, these results illustrate that perseverative errors occur at moderate to severe levels of phonological encoding impairment. One interpretation is that when phonological encoding is sufficiently impaired, a dearth of target activation results in the availability of only previously active phonological units. These results do not, however, preclude a breakdown of within-network inhibitory processes contributing to perseverative error production. Indeed, if the existence of both excitatory and inhibitory processes are presumed to occur within a cognitive system, it would be highly unlikely that one is impaired and the other spared.

Failure of inhibition as a dominant impairment is hypothesised to result in a qualitatively different error pattern than impairments in activating new target information, with consistent perseverative responses occurring without a correspondingly high level of non-perseverative nonword errors (Fischer-Baum & Rapp, 2012). Two individuals in the current study (participants 17 and 44) displayed this pattern, producing extremely high proportions of errors classified as perseverative with a comparatively low number of errors classified as non-perseverative nonword responses. This may indicate a greater contribution of inhibitory breakdown in these two individuals. Again, however, caution must be taken in this interpretation. The perseverative errors produced by these two individuals were blended perseverations in which responses contained both perseverated phonemes and non-perseverated phonemes. Non-perseverated phonemes were, for the most part, not related to the target item, suggestive of additional phonological encoding breakdown. Extreme breakdown in phonological encoding would cause consistent failure of target phonology activation and an over-reliance on previously encoded phonology resulting in the majority of responses being identified as perseverative. This would also account for the error patterns produced by participants 17 and 44. Further testing of dissociating individuals would provide useful information on the nature and consistency of production patterns, and is crucial for better understanding Jargon aphasia and the heterogeneity within the population (Nickels et al., 2011).

Voxel-lesion symptom mapping (VLSM) analyses were used to explore the relationship between lesion distribution and a sensitive measure of jargon repetition. Results parallel those obtained in previous VLSM studies and revealed a significant relationship between jargon production and lesion in the posterior temporoparietal region. Four significant clusters were identified in the grey matter of the posterior superior temporal gyrus (pSTG), supramarginal gyrus (SMG) and superior temporal sulcus (pSTS) and the white matter at the border of the superior temporal and inferior parietal lobes. These regions are commonly observed to activate during functional imaging studies of speech production and repetition, although the precise roles remain under discussion. The pSTG region identified included area Spt at the border between the temporal and parietal lobe. Area Spt is proposed to be a hub region supporting the translation of auditory into motor information (Buchsbaum et al., 2011; Hickok et al., 2011; Hickok & Poeppel, 2004; Warren et al., 2005). These posterior auditory and phonological processes are thought to interact with frontal motor and articulatory processes via dorsal stream white matter tracts associated with the regions of white matter lesion identified in the current study. This finding converges with the phonological encoding impairment interpretation from the current and previous psycholinguistic analyses in that, in the context of repetition, phonological encoding requires the translation of auditory information into phonological patterns that can interface with articulatory processes. The SMG and pSTS regions identified in the VLSM analysis are associated with other processes. The SMG is frequently found to be active during tasks which require the temporary storage of phonological information, leading to the interpretation of this area as a phonological short term memory store. An impairment in phonological short term memory is likely to exacerbate difficulties with phonological encoding through a difficulty in maintaining phonological strings during production and, indeed, those with a greater degree of lesion in the SMG region displayed significantly more severe jargon repetition (Figure 5). The pSTS may play a role in maintaining auditory targets during repetition (Markiewicz & Bohland, 2016; Tourville et al., 2008). This converges with traditional hypotheses which implicate an impairment in self-monitoring in Jargon aphasia (Kinsbourne & Warrington, 1963; Maher et al., 1994); difficulties in holding auditory targets may result in limited information with which to monitor production. The VLSM analysis did not identify regions associated with articulatory processes, therefore indicating limited involvement of articulatory impairment in jargon repetition.

ROI analyses were used to explore whether combinations of lesions across the posterior temporal-parietal region were predictive of jargon repetition. Regression analysis found that combined lesions to the STG and STS region were associated with jargon production. This indicates that jargon is more likely to occur when impairments in phonological encoding and self-monitoring occur in combination. The STG and STS clusters were proximal and consequently there was a medium correlation between percentage lesion overlap in these clusters across the group ($r = 0.54$). However, over 1/3 of the group displayed high lesion overlap in the STG or STS but not in the other region, therefore this pattern is not fully accounted for by a lesion to a single region.

The VLSM analyses in the current study converge with previous lesion studies undertaken with a smaller proportion of severely impaired individuals. Therefore, these results indicate that jargon repetition may be a more severe manifestation of milder conduction-like repetition deficits. However, ROI analyses in the current study found that individuals with mild or no impairments still presented with lesions in regions identified by the VLSM analysis. These individual differences may be a consequence of post-stroke reorganisation, which was also a significant predictor of jargon production, and are of interest for neuroscientific studies of stroke recovery. These results should, however, be treated as exploratory. Although the results parallel previous VLSM studies of repetition in aphasia (Baldo et al., 2012; Fridriksson et al., 2010; Rogalsky et al., 2015), the results did not remain significant following permutation testing. This is likely to be a consequence of high lesion overlap in the aphasia group as a whole and the high prevalence of repetition impairment, Figure 1, Table 1. Additionally, caution must be taken in interpreting mass-univariate lesion-symptom mapping analyses which suffer from spatial distortion because of constraints of the vascular architecture (Mah et al., 2014) and do not account for regions which have limited functional capacity but remain structurally intact (Robson et al., 2016).

Insights for therapy

Current findings highlight several possible therapeutic strategies that may aid clinical management of Jargon aphasia. Weak activation of target segments at the phonological encoding level dictates that therapy and management should maximise the degree of activation feeding through to the phonological level. According to cognitive-neuropsychological models of word repetition, this is achieved via two converging avenues; lexical (via semantics) and sub-lexical (auditory-phonological analysis and translation into motor instructions). To fully utilise and maximise activation via lexical and sub lexical avenues, clinical tasks should include stimuli in multiple modalities, administering a written and verbal model of the stimuli, and imagery where possible. Phonological awareness training could be adapted to include post phonological processing tasks – an area of comparative strength in this patient population (Romani & Galluzzi, 2005; Romani et al., 2002). Jargon aphasia therapy studies are scarce and further research is crucial to enhance understanding of the Jargon impairment, and thus support development of targeted treatments.

Conclusions

This study explored behavioural and neurological patterns associated with neologistic and perseverative word repetition errors in Jargon aphasia. Results from the behavioural and lesion analyses converge and support an impairment in encoding target phonology, possibly secondary to impairments in sensory-motor integration. Region of interest lesion analysis extended behavioural findings by indicating that impairments in maintaining auditory information in combination with phonological encoding impairments are particularly detrimental for repetition and were the most predictive of jargon responses in the current study. Behavioural analysis found that nonword and perseverative production are for the most part closely associated, paralleling previous psycholinguistic investigations and supporting the interpretation that perseverative and nonword errors can be accounted for by the same impairment source. These results imply that strengthening auditory-phonological integration and supporting self-monitoring would support speech production in Jargon aphasia.

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Figure 1: MNI X-coordinate displayed above. Panel A: Lesion overlap map for Jargon aphasia participants for whom imaging was available. Colour bar indicates number of individuals with lesion at each voxel. Panel B: Lesion overlap Lesion overlap map for all aphasia participants for whom imaging was available. Colour bar indicates number of individuals with lesion at each voxel. Panel C: VLSM results (threshold ≤0.05) showing voxels significantly associated with jargon score. Colour bar indicates t-statistic.

Figure 2: Mean neologism Phonological Overlap Index (POI) score per Jargon individual (squares), and the mean chance POI estimate (red line). Error bars show 95% confidence intervals.

Figure 3: Mean Intrusion Perseveration Probability (IPP) score per Jargon individual (square), and IPP chance estimate (red line). Error bars show 95% confidence intervals.

Figure 4: Scatter plot showing the relationship between numbers of perseverative and non-perseverative nonwords.

Figure 5: Colour areas display four regions of interest derived from VLSM clusters. Graphs indicate jargon score for low and high lesion overlap group in each ROI. Ceiling performance on jargon score = -1.07. * = significant group difference; (*) = borderline significant group

difference. WM = white matter; GM = grey matter; STG = superior temporal gyrus; IPL = inferior parietal lobe; STS = superior temporal sulcus.

Conflict of Interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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