Quantitative speech production profiles and focal left hemisphere lesion

Introduction

The clinical differentiation between apraxia of speech (AOS) and aphasia with phonemic paraphasia is based on impressionistic consideration of a varying list of speech properties. The diagnosing clinician is challenged with determining the presence or absence of these disorders by considering the extent to which characteristic features are evident in the speech output and by determining how much relative weight to assign to each. Predictably, the subjective nature of the diagnostic process can translate to limited agreement, even among experienced clinicians (Haley, Jacks, de Riesthal, Abou-Khalil, & Roth, 2012), and the risks of misdiagnosis and diagnostic uncertainty are substantial (Wambaugh, 2006). Additionally it is likely that the adherence to a strictly dichotomous classification system overlooks theoretically and clinically important heterogeneity.

The purpose of this study was to identify a preliminary set of speech production profiles based on naturally occurring variations in individuals with acquired focal brain lesions. To avoid classification circularity, assessments were conducted without consideration of clinical speech diagnosis, and metrics were selected to represent diverse and robust observations about speech properties associated with left hemisphere lesions.

Method

To date, sixteen participants have been enrolled in this study, based on a diagnosis of aphasia at least three month prior and difficulty producing accurate speech sounds (table 1). The etiology was stroke in all but one case, which was due to gunshot trauma. Speech samples were recorded from all participants based on the repetition of syllables, words, and sentences according to a standard motor speech evaluation protocol (Duffy, 2013; Wertz, LaPointe, & Rosenbek, 1984). In addition, 50 monosyllabic words were recorded to assess speech intelligibility.

Speech qualities associated with apraxia of speech and phonemic paraphasia (McNeil, Robin, & Schmidt, 2009; Wambaugh, Duffy, McNeil, Robin, & Rogers, 2006; Ziegler, 2008) were quantified, using a combination of auditory perceptual and acoustic measures. Three metrics were selected to capture phonemically salient sound errors. These included: speech intelligibility, defined as the proportion of monosyllabic words identified correctly by a panel of ten listeners that were blinded to the target sample; accuracy of multisyllabic words, defined as the proportion of attempts produced without phonemic errors; and phonetic complexity ratio, defined as rated complexity of the speech output (Stoel-Gammon, 2010) relative to the target. Three measures of sound distortion were obtained from narrow phonetic transcription of the speech sample. A comprehensive set of diacritic marks (Shriberg & Kent, 1995) was used to denote distortions outside the normal range of variation. Transcriptions were then quantified as the proportion of word attempts produced with distortion of tongue placement, voicing, and sound prolongation. Segmental and inter-segmental prolongation of multisyllabic utterances was operationalized as the mean syllable duration in single three- and four-syllable words. For participants who were able to repeat sentences, temporal prosody was also expressed in syllables per second and as the proportion of the sentence duration that included an acoustically defined pause. Two measures of fluency were included. Self-corrections were defined as the proportion of items that were repeated or corrected by the participant without prompting from the clinician, and rejections

were defined as the proportion of trials that the participant did not attempt. Finally, variability upon sequential attempts to produce the same word was expressed as the number of produced variants relative to the number of trials attempted (Marquardt, Jacks, & Davis, 2004), and sequential motion rate was coded based on at least three consecutive repetitions of the triad syllable sequence "puh-tuh-kuh."

For ten participants, clinical brain MRI scans were of sufficient quality to allow systematic documentation of lesion location. Ten perisylvian regions of interest were identified in each scan, using key anatomical landmarks. Degree of lesion was rated in each region from 0 (no lesion) to 5 (complete lesion).

Results and Discussion

Speech quantification results for each participant are presented in table 2. We divided the dataset into two groups based on the frequency of phonemic errors. P01-P09 displayed moderately to severely impaired speech sound production. Their speech intelligibility for monosyllabic words ranged from 1% to 77%, and their ability to repeat multisyllabic words with full accuracy never exceeded 10%. Some of these individuals, all with intelligibility scores in the lower range, demonstrated a reduction in the phonetic complexity of the speech output. Most participants in the low phonemic accuracy group also displayed prominent distortion errors and abnormal temporal prosody for both multisyllabic words and sentences. There were two exceptions. P01 was unable to repeat multisyllabic words and P09 produced them at a normal rate, indicating that a diagnosis of AOS was uncertain. The seven remaining participants in this group (P02-P08) display the combination of salient sound production errors, phonetic distortions, and slow articulation that is typically associated with AOS.

A different profile emerged for participants P10-P16, who displayed considerably lower frequency of phonemic errors (single word intelligibility was 90% or greater and repetition of multisyllabic words was 69% or greater). Two participants in this group, P11 and P12, produced a low frequency of distortion errors, normal temporal prosody for both multisyllabic words and sentences, yet evidenced notable errors on repetition of multisyllabic words. Thus, a diagnosis of aphasia with phonemic paraphasia seemed appropriate for these individuals. Three participants (P14-P16) displayed such low error rates on all tasks that their profile warranted assignment to a separate group with minimal impairment.

Other profiles were even more clearly outside the range of traditional diagnostic categories. For example, consistent with her diagnosis of Broca's aphasia, P10 displayed reduced rate for sentence repetition at about one syllable per second. However, her mean syllable duration in multisyllabic words was normal (280 ms). Similarly, monosyllabic speech intelligibility was normal (99%), and multisyllabic word repetition was only mildly reduced (90%). The only indication of a presentation consistent with AOS was a relatively high rate of distortions affecting voicing (10%).

Fluency and production variability varied across participants and were most clearly related to overall degree of impairment. Severely affected individuals were likely to reject items, whereas moderately affected individuals were more likely to self-correct. Similarly, token variability was greater for participants with the most frequent sound production errors. Most individuals in our sample were unable to reproduce the SMR sequence.

Results of the clinical brain MRI analysis are presented in table 3. Scans were available for four individuals who displayed prominent speech sound errors. They showed lesions affecting critical perisylvian areas in frontal, parietal, as well as temporal cortices. In contrast, individuals with limited sound production errors evidenced relative or complete sparing of at least one critical area in this circuitry, and participants with very mild sound production impairments showed extensive sparing in the perisylvian region. Although posterior inferior frontal cortex, in particular pars opercularis, was affected in all three participants with a performance pattern indicative of AOS, it was also prominently affected in P10, who presented with Broca's aphasia but only very mildly affected articulation, and it was partially affected in another two participants without signs of AOS. Similarly, the anterior insula was lesioned not only in individuals with performance profiles indicative of AOS, but also in P10, in one participants with a profile indicative of aphasia with phonemic paraphasia (P11), and partially in one participant with only minimal sound production difficulties (P15).

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Table 1. Participant demographics.

	Sex	Age	TPO	Handedness	Education	Aphasia type
P01	F	57	13 years	R	18 years	Mixed nonfluent
P02	F	66	2 years	R	12 years	Broca
P03	M	63	2 years	R	16 years	Global
P04	F	41	7 years	L+R	14 years	Broca
P05	F	55	10 years	R	14 years	Broca
P06	M	43	3 years	R	16 years	Broca
P07	F	46	2 years	R	16 years	Borderline fluent
P08	M	46	2 years	R	16 years	Broca
P09	F	66	5 months	R	12 years	Broca
P10	F	61	3 years	L+R	21 years	Broca
P11	F	52	8 years	L+R	18 years	Anomic
P12	F	70	4 years	R	18 years	Anomic
P13	M	64	3 months	R	21 years	Anomic
P14	F	65	12 years	R	16 years	Not aphasic
P15	M	47	1 year	R	12 years	Anomic
P16	M	52	4 years	R	23 years	Anomic

TPO=time post onset. Handedness=self-reported premorbid handedness. Aphasia type based on administration of either the Western Aphasia Battery (Kertesz, 2006) or the Aphasia Diagnostic Profiles (Helm-Estabrooks, 1992).

Table 2. Quantitative speech metrics for each participant. Grey cells indicate performance outside the expected normal range.

	Phonemic errors			Distortion errors			Speech rate			Fluency			
	Int	MS	PC	D-ton	D-voc	D-prol	SyD	S-rate	S-pp	SC	Rej	TTV	SMR
P01	0.01	0.00	0.41	0.08	0.19	0.13	cnp	cnp	cnp	0.00	0.51	cnp	0
P02	0.20	0.00	0.63	0.11	0.09	0.16	427	2.63	0.33	0.04	0.00	0.83	0
P03	0.29	0.00	0.76	0.10	0.08	0.24	756	cnp	cnp	0.00	0.10	0.75	0
P04	0.37	0.00	0.73	0.04	0.07	0.01	362	1.20	0.67	0.00	0.00	0.50	0
P05	0.56	0.10	0.94	0.03	0.07	0.03	429	0.91	0.54	0.08	0.36	cnp	0
P06	0.63	0.00	1.00	0.05	0.05	0.04	326	cnp	cnp	0.00	0.10	1.00	0
P07	0.76	0.06	0.91	0.00	0.07	0.01	372	cnp	cnp	0.02	0.11	0.67	0
P08	0.77	0.03	0.94	0.02	0.06	0.06	605	0.72	0.60	0.11	0.00	0.75	0
P09	0.55	0.03	0.76	0.02	0.06	0.00	262	1.03	0.65	0.00	0.02	0.25	0
P10	0.99	0.90	1.01	0.00	0.10	0.01	280	1.19	0.67	0.08	0.00	0.25	0
P11	0.95	0.69	0.93	0.01	0.04	0.00	256	3.20	0.34	0.07	0.00	0.13	1
P12	0.90	0.79	0.97	0.00	0.02	0.01	246	3.74	0.25	0.05	0.00	0.25	1
P13	0.91	0.97	0.98	0.01	0.03	0.02	284	2.39	0.35	0.12	0.00	0.08	0
P14	0.92	1.00	0.96	0.00	0.01	0.00	256	3.24	0.31	0.00	0.00	0.00	1
P15	0.93	0.98	0.98	0.01	0.02	0.00	258	3.40	0.26	0.09	0.00	0.08	0
P16	0.95	0.97	1.00	0.00	0.00	0.00	252	3.58	0.23	0.00	0.00	0.00	1

Int = Single word intelligibility; MS=accuracy on multisyllabic words; PC=phonetic complexity ratio; D-ton=distortion of tongue placement; D-voc=distortion of voicing; D-prol=distortion of prolongation. SyD=mean syllable duration in multisyllabic words; S-rate = sentence production rate in syllables per second, S-pp = sentence pause proportion, TTV=total token variability; SC=percent of attempts with self corrections; Rej=percent of attempts rejected, SMR=ability to produce sequential motion rate

Table 3. Rated magnitude of lesion in perisylvian regions of interest for ten participants for whom clinical brain MRI scans were available.

	pTr	pOp	aIns	pIns	iPrCG	iPoCG	iSMG	aAG	pSTG	mSTG
P03	4.0	5.0	3.0	5.0	5.0	3.0	3.0	3.5	5.0	5.0
P07	4.5	5.0	5.0	5.0	5.0	4.0	5.0	5.0	5.0	3.0
P08	2.0	5.0	4.0	4.0	2.5	3.0	2.0	5.0	5.0	3.0
P09	4.0	5.0	5.0	5.0	4.5	4.0	4.5	2.0	4.0	5.0
P10	4.0	5.0	5.0	5.0	4.0	1.0	0.0	0.0	5.0	5.0
P11	0.0	3.0	5.0	5.0	5.0	3.5	3.0	2.0	5.0	5.0
P13	0.0	0.0	0.0	4.0	3.0	5.0	5.0	0.0	4.0	2.0
P14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	4.0	0.0	0.0
P15	2.0	3.5	3.0	2.0	2.0	3.0	3.0	0.0	2.0	1.0
P16	0.0	1.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Regions of interest: pTr = pars Triangularis, pOp = pars Opercularis, aIns = anterior insula, pIns = posterior insula, iPrCG = inferior precentral gyrus, iPoCG=inferior post central gyrus, iSMG = inferior supramarginal gyrus, aAG=anterior angular gyrus, pSTG = posterior superior temporal gyrus. mSTF = middle superior temporal gyrus.
Lesion rating: 0.0=no lesion; 1.0=equivocal lesion; 2.0=small patchy lesion; 3.0=half of area lesioned; 4.0=more than half but not all area lesioned; 5.0=total area lesioned. Cells with lesions rated as 3.0 or greater are shaded in grey.