Would Wernicke's Aphasic Speech Vary with Auditory Comprehension Abilities and/or Lesion Loci?

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ABSTRACT

Speech characteristics of Wernicke's aphasia are characterized by such errors as empty speech, jargon, paraphasia, filler and others. However, not all the errors can be observed in each patient presumably due to diverse auditory comprehension (AC) abilities and/or lesion loci. The purpose of this study was, thus, to clarify the speech characteristics of Wernicke's aphasics according to the AC levels (i.e., better vs. worse) and lesion loci (i.e., Wernicke's area, WA vs. non-Wernicke's area, NWA). The authors divided 21 Wernicke's aphasic patients into four patient groups based on their AC levels and the lesion loci. The results showed that the four groups differed only in CIU (Correct Information Unit) rate. The patient groups with a better AC ability had higher CIU rates than the groups with a worse AC regardless of the lesion loci (e.g., WA or NWA). Therefore, it was concluded that CIU rate, the differentiating speech variable was most likely related to the AC levels, but not to lesion loci.

Keywords: Wernicke's aphasia, speech characteristics, auditory comprehension, Wernicke's area, CIU (Correct Information Unit) rate

I. Introduction

Wernicke's aphasia, as defined by Goodglass and Caplan (1972), is characterized by poor auditory comprehension (AC), fluent spontaneous speech, poor repetition, and speech deficits

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such as empty speech, jargon, and neologism. Not all Wernicke's aphasics, however, present the aforementioned speech characteristics— their speech errors may be diverse. For example, some patients might present more paraphasias than others whereas others might predominantly show empty speech. Would this dissimilarity differ with different degrees of AC deficits? Or, would it be reflective of different lesion loci [e.g., Wernicke's area (WA) as a core comprehension locus vs. non-Wernicke's area (NWA)]?

Regarding the relation between AC level and speech characteristics, Vermeulen et al., (1989) reported that AC ability has significant correlations with syntactic ability, vocabulary, and neologistic paraphasia. That is, patients with better AC ability generally produced fast rate of speaking; longer mean length of utterance (MLU); and large number of conjuctions, auxiliaries, and words in spontaneous speech. On the other hand, the researchers observed fewer speech errors such as irrelevant paraphasias and neologism in patients with better AC compared to those with a worse AC. It has been argued that individuals with high comprehension ability would generally edit out such erroneous productions as neologistic paraphasia.

Ever since Wernicke claimed the "center" of AC, the so-called WA has been considered as a core area for AC function (Saffran, 2002). However, brain mapping studies using brain computerized tomography (CT), magnetic resonance imaging (MRI), functional MRI (fMRI), and positron emission tomography (PET) indicated that this might not be the case (Hanlon et al., 1999; Kim et al., 1998; Kreisler et al., 2000; Radanovic & Scaff, 2003; Willmes & Poeck, 1993). Although the primary areas responsible for Wernicke's aphasia were declared to be superior posterior temporal gyrus, Wernicke's aphasia might occur from lesions of the brain areas other than the WA. In fact, lesions in the NWAs such as insula, anterior part of temporal gyrus, posterior frontal lobule, and inferior parietal lobule were also related to Wernicke's aphasia.

However, it is of particular interest to explore if characteristics of Wernicke's aphasia from the WA lesion might differ from those of Wernicke's aphasia from the NWA. WA usually refers to the area in the first temporal gyrus of left hemisphere (Bogen & Bogen, 1976) or more specifically the posterior one-third of the superior temporal gyrus (Kertesz, 1979). A work by Damasio and Damasio (1989) indicated on a lesion in the Brodmann area 22 (i.e., WA), presented a patient hyperfluent speech and numerous phonemic and semantic paraphasias. According to Hillis et al. (2001), aphasic patients with WA hypoperfusion show lexical-semantic impairments in such speech tasks as repetition and oral picture naming. However, these speech characteristics can also be observed in patients with NWA (Kim et

al., 1998). According to the aforementioned study, Wernicke's aphasics with NWA lesions of parietal lobe, basal ganglia, or occipital lobe had speech errors such as paraphasia, neologism, repetition and others. The extent of the lesion might also be an important issue regarding its influence on patterns of speech characteristics. However, up to date, the authors could not find a study dealing with the relation between WA lesion size and speech characteristics. The purpose of this study is, then, to investigate if different speech characteristics of Wernicke's aphasics are influenced by different levels of AC deficits or reflective of different lesion loci (e.g., WA vs. NWA).

2. Methods

2.1 Subjects

The authors examined 95 Wernicke's aphasic patients who admitted to the department of neurology in a general hospital due to a cerebral vascular accident (CVA). Among them, the authors selected 21 patients (10 men and 11 women) based on the following criteria: those with a brain-MRI, and those with AC subtest scores between 0 and 3.9, or between 5 and 6.9. AC scores were derived from the subtest for auditory comprehension in the Paradise · the Korean version- Western Aphasia Battery (Paradise-K-WAB) (Kim & Na, 2001, 2004). All patients were diagnosed as having a Wernicke's aphasia based on the Kertesz's criterion of aphasia classification (Kertesz, 1979).

Table 1 presents information on the 21 patients such as sex, age, years of education, post onset time (POT), AC scores, naming subtest scores, and lesion loci. The age of the patients ranged from 37 to 80 (M = 62.62, SD = 10.08) and the mean years of education ranged from 0 to 16 (M = 8.05, SD = 4.67). POT at the time of language testing ranged from 15 to 760 days for 18 out of 23 subjects. The authors also had to include 3 subjects with relatively short POTs of 9, 4, and 1 days respectively because we could hardly find any subject with high AC scores and WA involvement unless they were on acute stages. MRI scans were acquired within 2 days prior to the language testings. The patients did not present concomitant speech disturbances. All subjects were right-handed, and all patients had a left hemisphere lesion except for 3 patients (S10, S13, S21) with bilateral lesions in the perisylvian regions. The lesion loci were determined by two clinical neurologists (C-S. Chung & K-H. Lee). They were blinded to the results of the language tests.

Table 1. Information on 21 Wernicke's aphasic patients

Subj.	Sex/ Age	Years of Edu.	POT (day)	AQ	AC Scores (out of 10)	Naming Scores (out of 10)	Lesion loci
S1	M/67	9	455	20.6	0	2.5	Lt. temporal lobe
S2	M/59	12	15	13.8	0.3	0.5	Lt. temporal lobe, Lt. occipital lobe
S3	F/56	3	760	29.4	1.9	1.8	Lt. temporal lobe
S4	F/58	6	60	28.6	2.1	1.1	Lt. temporo-fronto-parietal lobes
S5	F/52	0	17	49.0	2.2	3.4	Lt. sup. & mid. temporal gyri
S6	F/60	3	30	32.2	2.4	1.5	Lt. sup. temporal gyrus, Lt. inf. parietal gyrus, Lt. cerebellum, Lt. BG
S7	F/78	6	30	30.9	2.75	0.6	Lt. temporal & parietal lobes
S8	M/63	12	73	39.8	2.9	2.7	Lt. sup. temporal gyrus
S9	F/67	0	106	36.6	3.0	0.9	Lt. temporal lobe
S10	M/49	12	639	32.6	1.05	1.05	Lt. BG, Bi. sup. frontal gyrus
S11	M/80	16	25	37.9	2.25	2.2	Lt. parietal lobe
S12	M/38	9	28	39.3	3.55	0.3	Lt. parietal & occipital lobe
S13	F/70	6	38	57.7	3.9	1.9	Bi. perisylvian region
S14	M/76	6	9	50.2	5.4	4.1	Lt. temporal & parietal lobes
S15	F/61	12	1	54.8	5.55	3.4	Lt. sup. & mid. temporal gyri
S16	M/61	6	4	50.1	6.8	0.9	Lt. parietal lobe & sup. temporal gyrus
S17	M/58	14	240	59.1	5.25	7.5	Lt. inf. frontal gyrus
S18	F/66	9	25	77.0	5.9	8.0	Lt. perisylvian region
S19	F/51	6	22	64.4	5.9	7.1	Lt. supramarginal gyrus
S20	M/66	16	365	54.2	6.3	4.2	Lt. cerebellum
S21	F/69	6	30	68.3	6.45	5.83	Bi. perisylvian, Lt. supramarginal gyrus

POT, Post onset time

AQ, Aphasia quotient

AC scores, Auditory comprehension scores

sup., superior; mid., middle; inf., inferior; ant., anterior; post., posterior; SC, striato-capsule; BG, basal ganglia

2.2 Speech Collection and Analyses

Two speech-language pathologists (H. Kim & Y.M. Lee) administered the K-WAB to each patient. The experimental speech task was to describe the stimulus picture "Seashore" in a subtest. The speech samples were recorded on a tape-recorder (Marantz PD221) and transcribed by the two speech-language pathologists. The transcribed data of 21 patients were analyzed using 10 speech variables shown in Table 2. The 10 speech variables were under 5 categories: syntactic, vocabulary, articulatory, phonological, and neologistic. The syntactic category includes the number (#) of utterance, utterance duration, # of syllables per utterance, and # of reversal. The vocabulary category includes the Correct Information Unit (CIU) rate and # of empty words. The articulatory category includes # of articulatory errors such as repetition and jargon. The phonological category consists of the # of self-corrections whereas the neologistic category includes the # of paraphasia. The 10 variables were selected because they were described as clinically relevant variables (Vermeulen et al., 1989).

Some variables, however, were excluded because either they were rarely produced among our patients or were not existent in Korean language. For instance, auxiliary verbs (e.g., is, will, can) are not used as separate sentence constituent in Korean syntactic realms. We included 'number of utterances' that a patient produces from the picture stimulus given. A time-constraint was not given because it was not necessary to reflect a patient's fluency/syntactic ability (Kim et al., 1998). An utterance was usually defined as a sentence. If there was a pause that lasts more than 2 seconds within a sentence, however, we considered the two segments as separate utterances (Kim et al., 1998). 'Utterance duration' was measured from the beginning to the end of an utterance. We counted number of syllables for each utterance and divided them by number of utterance to get an average number of syllables per utterance. A 'reversal' was indicated if an utterance contains unconventional word order (for example if 'A boat is floating' was produced as 'is floating, a boat')

CIU was originally proposed by Yorkston and Beukelman (1980) and defined as "a grouping of information that is always expressed as a unit by normal speaker." In their sentence example, "The little boy is on the stool and reaching up for a cookie," the words 'little' and 'boy' were considered as content units whereas 'on' was not. Because the word 'on' usually followed by noun phrase (here, 'the stool'), it cannot represent as independent content or information unit. CIU rate was computed as a proportion of total number of

different CIUs in the total number of words. 'Empty words' were regarded as meaningless syllables and words such as /ə/, /dʒə/, /ʉm-/ and /dʒəgɪ/. It was also regarded as an empty word when a noun (e.g., ball) replaces a pronoun (e.g., this). For 'repetition', we included repetitions of syllables, words, phrases, and sentences. A 'jargon' was counted when an incomprehensible segment of speech was observed. A 'self-correction' was indicated when a patient revised uttered word. Paraphasia included phonemic as well as semantic paraphasias and neologism.

Table 2. Ten speech variables and definitions

Speech variables	Definition
1) Number of utterance	Total number of utterances
2) Utterance duration	Duration of an utterance in second
3) Number of syllables per utterance	Number of syllables per utterance
4) Reversal	Total number of phrase reversals per utterance
5) CIU rate	Total number of different CIUs ÷ total number of words
6) Empty word	Meaningless syllables (e.g., /ə/, /dʒə/, / ʉm-/, /dʒəgɪ/) and substituted pronouns per utterance
7) Paraphasia	Number of phonemic, semantic paraphasia and neologism
8) Repetition	Repetitions per utterance
9) Jargon	Number of jargons per utterance
10) Self-correction	Number of self-corrected words per utterance

3. Results

3.1 Subject Grouping according to AC Levels and Lesion Loci

Table 3 summarizes four groups of 21 Wernicke's aphasic patients according to their AC levels and lesion loci. Two AC levels were determined based on Kertesz's (1982) study. When AC score ranged from 0 to 6.9, he or she was regarded as a Werncke's aphasic. If

AC score was as low as 0 to 3.9, the Wernicke's aphasic patient was regarded as having more severe AC deficit (i.e., ^{lower}AC group). The range of score (i.e., 0 to 3) was derived from the Kertesz's criterion of global aphasia. On the other hand, the better AC group (i.e., ^{higher}AC group) included patient cases with AC levels between 5 and 6.9. Patients with AC scores between 4 and 4.9 were excluded in order to decrease the borderline effect.

Two types of lesion loci (i.e., WA vs. NWA) were determined. The WA was referred as Brodmann area (BA) 22 as shown in the work by Damasio and Damasio (1989) (see Fig 1). The 'A' section of Figure 1 presents BA 22 designated by arrows on the original figure and 'B' section shows the corresponding areas marked with dark shades. The WA lesion was observable in the three axial image cuts (i.e., lower, middle, and upper) along the brain neuraxis. If a patient had a lesion within the WA, we considered him or her as having WA lesion. On the other hand, if a patient had a lesion outside the WA, he or she was regarded as a NWA patient.

Thus, the four patient-groups were as follows: 1) WA+ ^{lower}AC consists of 9 patients with worse AC scores ($\leq$3.9) as well as WA lesion; 2) NWA+ ^{lower}AC consists of 4 patients with worse AC scores ($\leq$3.9) as well as NWA lesion; 3) WA+ ^{higher}AC comprises 3 patients with better AC scores ($5<AC\leq6.9$) as well as WA lesion; and 4) NWA+ ^{higher}AC consists of 5 patients with better AC scores ($5<AC\leq6.9$) as well as NWA lesion.

Table 3. Four patient-groups formed based on their AC levels and lesion loci

		AC levels		Total
		AC≤3.9	5<AC≤6.9	
Lesion loci	WA	WA+ ^{lower}AC (n=9)	WA+ ^{higher}AC (n= 3)	n=12
	NWA	NWA+ ^{lower}AC (n= 4)	NWA+ ^{higher}AC (n=5)	n=9
Total		n=13	n=8	n=21

AC levels, Auditory comprehension levels; WA, Wernicke's area; NWA, Non-Wernicke's area

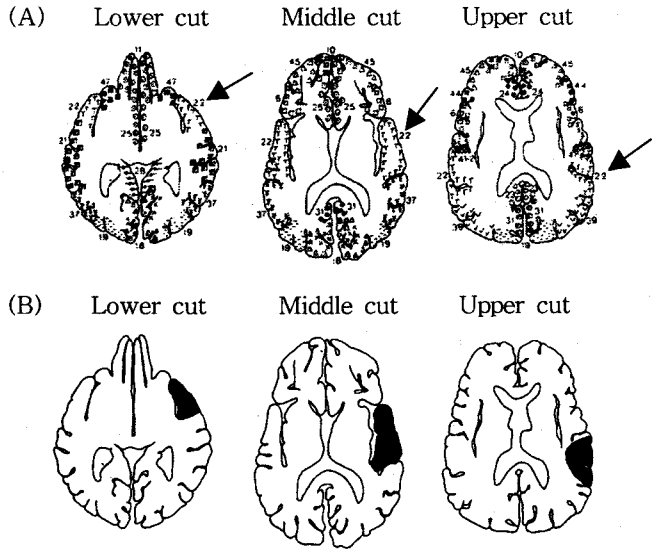


Figure 1. Wernicke's area referred as Brodmann area 22 designated by arrows (A) (Damasio & Damasio, 1989) and corresponds WA marked with dark shades (B). Both (A) and (B) with three (i.e., lower, middle, & upper) axial image cuts.

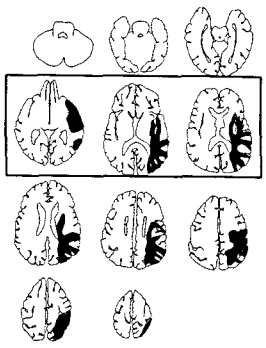
Table 4 presents mean and standard deviations of AQs and AC scores of each group. The WA groups (i.e., WA+ ^{lower}AC and WA+^{higher}AC) and NWA groups (i.e., NWA+ ^{lower}AC and NWA+ ^{higher}AC) did not statistically differ in the AQs and AC scores. Figure 2 shows lesion loci of patients in each group: (A) shows 3 selective patients (S2, S5, S7) among 9 patients of WA+ ^{lower}AC; (B), 3 selective patients (S10, S11, S12) of NWA+ ^{lower}AC; (C), all 3 patients (S14, S15, S16) of WA+ ^{higher}AC ; and (D), 3 selective patients (S17, S18, S19) among 5 patients of NWA+ ^{higher}AC. We examined the correlation index between naming performance (i.e., confrontation naming) and AC scores of our 21 Wernicke's patients and it was high (r= .704).

Table 4. Comparing the mean, SDs of each group of AQ and AC scores

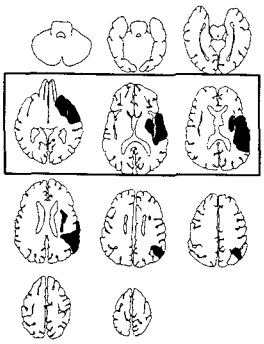
	Group	M (SD)	F	Sig.
AQs	WA+ ^{lower}AC	31.21 (10.28)	.025	.877
	NWA+ ^{lower}AC	41.87 (10.92)		
	WA+ ^{higher}AC	51.70 (2.69)	2.149	.193
	NWA+ ^{higher}AC	64.61 (8.75)		
AC scores	WA+ ^{lower}AC	1.95 (1.09)	.148	.708
	NWA+ ^{lower}AC	2.76 (1.23)		
	WA+ ^{higher}AC	5.92 (0.77)	1.624	.250
	NWA+ ^{higher}AC	5.74 (0.54)		

AQ, Aphasia quotient; AC score, auditory comprehension score

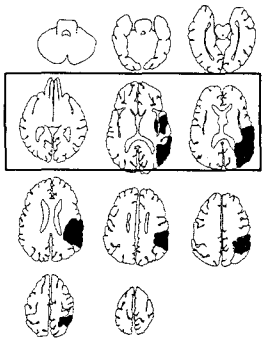
(A)



S2

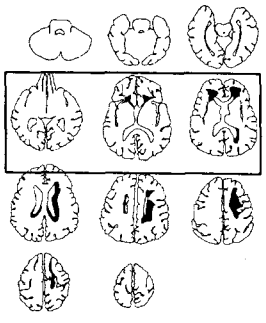


S5

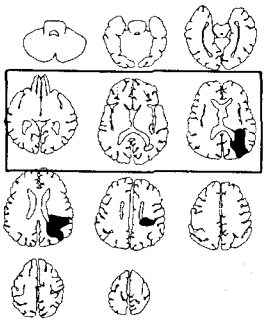


S7

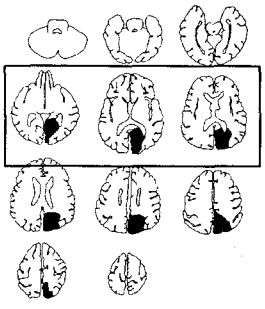
(B)



S10

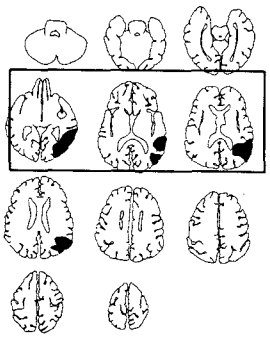


S11

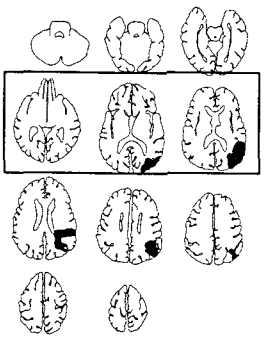


S12

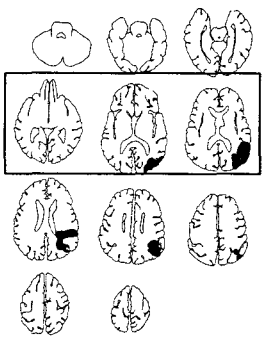
(C)



S14

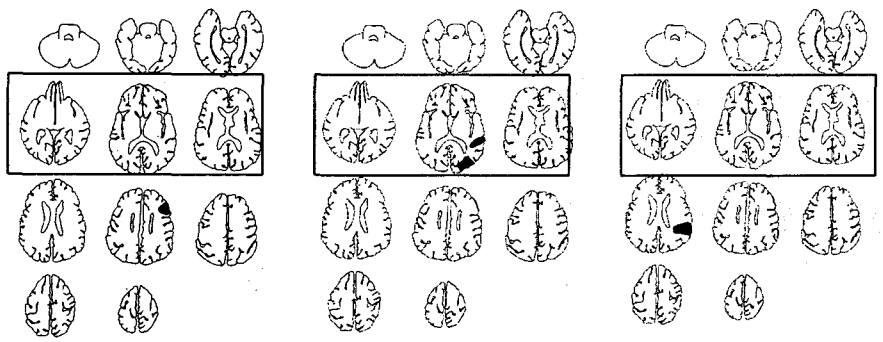


S15



S16

(D)



S17 S18 S19

Figure 2. Brain MRIs of selective patients of four Wernicke's aphasic groups: (A) WA+ ^{lower}AC (S2, S5, S7); (B) NWA+ ^{lower}AC (S10, S11, S12); (C) WA+ ^{higher}AC (S14, S15, S16); and (D) NWA+ ^{higher}AC (S17, S18, S19). The three axial image cuts inside the windows, containing the BA22 as described in Figure 1.

3.2 Speech Characteristics of Four Patient-groups

We used two-way ANOVA to analyze the data produced by four patient-groups. The dependent variables were lesion loci and auditory comprehension. As seen in Table 5, CIU rate was the only speech variable that differentiated among the four groups. The other nine speech characteristics were not different.

Table 5. Mean, SDs and two-way ANOVA results of CIU rate

AC	WA	n	CIU rate		F	Sig.
			Mean	SD		
^{lower}AC	WA	9	0.04	0.05	20.299	.000*
	NWA	4	0.02	0.02		
^{higher}AC	WA	3	0.13	0.07		
	NWA	5	0.15	0.05		

AC, Auditory comprehension; WA, Wernicke's area; NWA, Non-Wernicke's area; CIU rate, correct information unit rate; n=number of patients

*p<.001

4. Discussion

This study confirmed that the speech characteristics of Wernicke's aphasic patients were strongly related to AC ability, not to lesion loci. We observed that WA+ ^{higher}AC or NWA+ ^{higher}AC group presented significantly higher CIU rates than WA+^{lower}AC or NWA+ ^{lower}AC group regardless of their lesion loci. Likewise, a previous study by Vermeulen et al. (1989) reported that the patients with better AC ability had shown higher occurrence of TTRC (Type/token ratio of content words). In their term, TTRC was interpreted as 'proportion of different content words in total number of content words'. In our study, however, we utilized CIU rate referring to 'a proportion of total number of different CIUs (i.e., content words) in the total number of content as well as function words'. Lower CIU rates might indicate reduced ability in vocabulary usage.

Then, would there be any relationship between anomic deficits and AC dysfunction? According to the previous studies, anomic phenomenon is usually related to auditory comprehension disorders regardless of types of aphasia (Gainotti et al., 1986; Ilmberger et al., 2001; Silver & Halpern, 1992). However, auditory comprehension disorders in a Wernicke's aphasic patient may be ascribed to a larger degree of vocabulary deficits compared to Broca's aphasia with possible syntactic deficits causing auditory comprehension dysfunction (Caramazza, et al., 2001). In fact, when we scrutinized the correlation between naming performance (i.e., confrontation naming) and AC scores of our 21 Wernicke's patients, the index was high. This indicated that lower naming scores are linked to poorer AC functions.

Even though the results of our study did not show any significant difference in speech characteristics depending on lesion loci, some previous studies have reported anatomical correlates of speech errors. For example, different lesion sites were responsible for various types of paraphasias. In particular, phonemic paraphasia was caused by lesions of NWAs as external capsule (Kreisler et al., 2000) and insula or putamen (Knopman et al., 1984). However, semantic paraphasia was observed mainly after temporal lobe lesions (Kreisler et al., 2000) or more specifically WA (i.e., posterior superior temporal lobe) lesions or such NWA as inferior parietal lobe lesions (Knopman et al., 1984). These results are contrastive with ours that reported phonemic paraphasia due to WA in addition to NWA lesions. This dissimilarity might partly, if not all, attribute to the different language assessment materials of the studies. Whereas our observation was based on the patients' spontaneous speech of picture description, Kreisler et al. (2000) utilized several subtests of the Boston Diagnostic

Aphasia Examination but did not use a picture description task to determine aphasic disorders. In a similar token, Knopman et al. (1984) analyzed patients' speech errors based on the confrontation-naming subtest.

Conventionally, many researchers have believed that WA lesion produces Wernicke's aphasia characteric of poor AC ability and speech errors such as jargon, semantic paraphasia, and anomia (Leonard, 1999; Saffran, 2002). Likewise, our study showed patients with widespread WA lesions presented lower AC as seen in Figure 2 (A). However, it seems that if one exhibits an NWA lesion that is extensive enough, he or she might show significant AC deficit and consequently such speech characteristics as low CIU rate (Selnes et al., 1983). In Figure 2 (B), the NWA+ ^{lower}AC group showed lower AC ability despite lesion type. For example, patient S10 showed bilateral striato-capsular lesion. Likewise, S11 had extensive lesions in left parietal lobe. S12 exhibited massive lesions on left parietal and occipital lobes.

On the other hand, a smaller WA lesion might spare AC ability and would not induce typical speech errors that patients with extensive WA lesions. Naeser et al. (1987) reported from their earlier computed tomographic (CT) scan study of 10 Wernicke's aphasic patients that AC could be dependant upon the size of WA lesion. They argued that Wernicke's aphasic patients with damage in more than half of the WA tended to have worse AC ability whereas patients with damage in half or less than half of WA had better AC ability. As seen in Fig. 2 (C), three patients (S14, S15, S16) of the WA+ ^{higher}AC group presented little invasion of the WA when compared to the WA+ ^{lower}AC patients. In fact, S14 had a partial WA lesion on only upper temporal cut among the three brain-cuts (inside of windows), preserving lower, and middle cuts. In the case of S15, the middle and upper cuts were partially invaded. Similarly, S16 had a partial lesion only on the upper cut. Therefore, it is feasible to say that lesion size impacts AC function and speech characteristics. An extensive lesion in neural pathways would disconnect functional neuraxis for a particular language function (Nadaeau & Crosson, 1997).

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