Broca's Aphasia: Comparison to Metabolically Matched Aphasic Subjects

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Anatomic aphasia classifications suggest that definable lesions result in specific types of aphasia. With the presence of severe nonfluent speech that is sparse and telegraphic with relative preservation of comprehension, Broca's aphasia is perhaps the best described of these syndromes (Benson, 1979). The anatomic lesions that result in this syndrome include damage to the posterior inferior frontal lobe and to deeper structures. Though such lesions characteristically occur in Broca's aphasia, the presence of such lesions do not specify the aphasia. Recently, it has been shown using (F-18)-fluorodeoxyglucose (FDG) and positron emission tomography (PET) that the structural lesions in Broca's aphasia are associated with alterations in glucose metabolism throughout much of the cortex in the dominant damaged hemisphere, a pattern that differed from other types of aphasia. The distinction between Broca's and other aphasias included the extent of metabolic abnormalities in the dominant prefrontal cortex and the extent of subcortical structural damage (Metter et al., 1989).

To further examine the role of structural damage and glucose metabolism in Broca's aphasia, Broca's aphasic subjects were compared to other aphasic patients who had similar structural and metabolic patterns. In previous studies, we have found that the most striking metabolic and structural differences in this group of aphasic patients are in the degree of prefrontal hypometabolism (Regions 1 to 4, Figure 1) and the extent of subcortical structural damage. For this reason, we elected to identify other aphasic subjects who had the same degree of prefrontal hypometabolism. Our goal was to match the lesions as closely as possible, basing the study on either structural damage or the pattern of metabolic abnormality in the damaged left hemisphere (Metter et al., 1987). We asked what structural or metabolic factors other than the prefrontal metabolism would account for the behavioral differences.

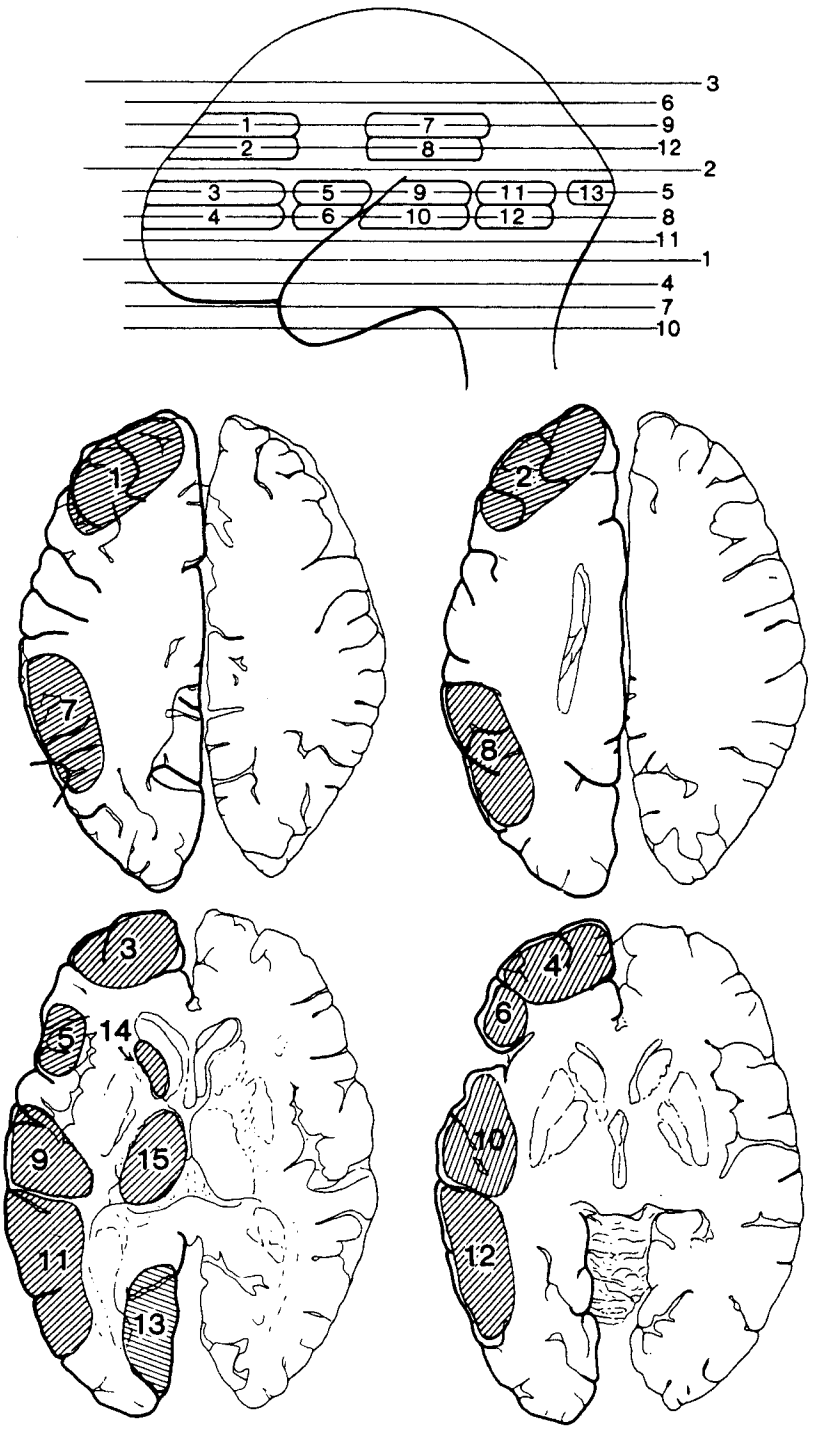

Figure 1. Regions of interest used for the quantitative measurements. Regions 1–4 are prefrontal, 5–6 are Broca's area, 7–8 are parietal, 9–10 are Wernicke's area, 11–12 are posterior middle and inferior temporal, 13 is occipital, 14 is the head of the caudate, and 15 is the thalamus.

METHOD

Forty-five aphasic patients who had a history of a single cerebrovascular (CV) event were studied more than 1 month post onset of the aphasia. These subjects have been reported previously in a number of studies. None were on medications that would interfere with their testing. Each subject was administered the Western Aphasia Battery (WAB) (Kertesz, 1981) to classify the nature of their aphasia.

Subjects were studied in a resting state by FDG PET with their eyes and ears unoccluded while lying quietly on the tomograph bed. They were injected with 5–10 millicuries of FDG and periodic serial arterialized venous blood samples were drawn over a 40-minute period. Scanning was then completed that yielded 12 tomographic sections. Regions of interest were determined using a standard approach, and local cerebral metabolic rates for glucose (LCMRglc) were reported in mg/100 gm tissue/minute using the methods developed by Phelps and colleagues (Phelps et al., 1979) for 15 brain regions (Figure 1). Left-to-right ratios were calculated for each region, and this value was used in the analyses described below.

Each subject had a computed tomographic scan at approximately the time of the other testing. The 16 brain regions measured on the PET images were identified, as well as the anterior and posterior internal capsules, lenticular nuclei and insula. Each region was graded on a 5-point scale: 0 = normal, 1 = atrophy, 2 = damage but no loss of tissue, 3 = partial tissue loss, 4 = complete tissue loss. Each scan was read by two physicians and the two readings were averaged. An 88% agreement (kappa = .71) was found between the two ratings (Metter et al., 1987).

RESULTS

Eleven Broca's aphasic patients were identified by the WAB. The range of prefrontal hypometabolism in the Broca's subjects was .12 to .79, with 10 subjects ranging from .40–.79. The patient with the prefrontal level of .12 was distinctly different from the other 10 Broca's aphasic subjects and could not be matched with any of the other aphasic subjects. Nine other aphasic patients were identified who had metabolic ratios in this latter range (Table 1, prefrontal). The resulting comparison was then for 10 Broca's and 9 other aphasic patients. The nine aphasic patients included three with Wernicke's, one with conduction, and five with anomic aphasias. The average age for the two groups was 59.2 years for the Broca's patients and 64.4 years for the other aphasic patients.

TABLE 1. REGIONAL METABOLIC COMPARISONS BETWEEN APHASIC GROUPS

Region	*Broca Group*	*Comparison Group**	*P*
Prefrontal	.65	.69	.43
Broca	.44	.56	.27
Parietal	.49	.50	.85
Wernicke	.49	.55	.68
Post. Temp.	.64	.60	.77
Occipital	.94	.89	.38
Caudate	.37	.46	.26
Thalamus	.46	.53	.33

**Note:* The prefrontal region was used to classify the comparison group of aphasic subjects. Regional measures were compared using *t* test.

TABLE 2. REGIONAL COMPARISON OF STRUCTURAL DAMAGE BETWEEN APHASIC GROUPS

Region		*Broca Group*	*Comparison Group*	*P*
Prefrontal	1	0.55	0.72	.68
	2	0.45	0.83	.40
	3	0.45	0.77	.47
	4	0.35	0.44	.81
Broca	5	1.65	1.00	.33
	6	1.45	1.06	.59
Parietal	7	2.45	2.11	.58
	8	2.65	2.61	.95
Wernicke	9	2.25	2.11	.82
	10	2.25	1.94	.62
Posterior	11	1.50	1.28	.71
Temporal	12	1.10	0.78	.61
Occipital	13	0.05	0.55	.29
Caudate	14	1.90	1.11	.25
Thalamus	15	1.10	0.38	.11
AIC		2.90	2.05	.22
PIC		2.15	1.22	.06
LN		3.20	2.94	.68
Insula		3.30	1.94	.04

Note: The numbers are the regional localizations from Figure 1.
AIC = anterior internal capsule, PIC = posterior internal capsule, LN = lenticular nuclei.

Table 1 presents a comparison of the metabolic ratios between the two groups with no differences found in regional glucose metabolism between the two groups. Table 2 gives a comparison between the structural lesions in the two groups. Nonparametric analysis found significant differences for the insula and the posterior internal capsule. A stepwise regression analysis to predict the aphasic groups based on structural damage (several regions were excluded from the analysis including regions 1, 3, 4, 6, 10, 12 in Figure 1) demonstrated that only the insula and posterior internal capsule contributed significantly to the equation accounting for 36% of the variance. This analysis was consistent with the region-by-region nonparametric analysis. The sample size is too small to create reliable equations. The purpose of the stepwise analysis was to explore whether other regions would reduce the importance of damage to the insula and posterior internal capsule. This was not the case. When these two regions were included with 7 metabolic regional measures to explain the aphasic group, only the 2 structural lesions were included in the final equation. In this instance the structural damage better explained differences between the two groups than did the metabolic data, but it should be remembered that the groups were selected based on comparability of the metabolic ratios.

Table 3 presents the behavioral comparison between the two groups. The Broca's aphasic subjects performed more poorly on overall WAB-AQ,

TABLE 3. COMPARISON OF PERFORMANCE ON THE WESTERN APHASIA BATTERY BETWEEN APHASIC GROUPS

Subtest	*Broca's Group*	*Comparison Group*	*P*
IC	5.00	5.22	.86
F	2.70	6.89	.00
YN	54.3	53.8	.89
AWR	49.5	48.6	.89
SeC	45.0	51.1	.62
Rep	3.11	6.33	.02
OBJN	23.7	36.4	.17
WF	2.20	4.33	.25
SC	5.20	7.11	.28
Resps	2.80	6.89	.03
Read	52.6	61.6	.45
Write	36.9	33.9	.85
AQ	43.3	63.2	.04

IC = information content, F = fluency, YN = yes/no questions, AWR = auditory word recognition, SeC = sequential commands, Rep = repetition, OBJN = object naming, WF = word fluency, SC = sentence completion, Resps = responsive speech

fluency, repetition and responsive speech. In a stepwise regression analysis only fluency added significantly to the equation to explain subject grouping and accounted for 67% of the variance.

DISCUSSION

Two groups of aphasic subjects were identified. The first, based on performance on the WAB, was defined as having features of Broca's aphasia. The second was defined based on metabolic similarities to the first group to match the overall changes as measured by glucose metabolism. None of the subjects in the second group had Broca's aphasia but had several other varieties of aphasias and as a group were less severely aphasic than the Broca's subjects based on the WAB-AQ. Previously, we found that all right-handed aphasic patients have metabolic abnormalities in the left temporoparietal region, while about half have left prefrontal changes. For this reason, we matched subjects primarily based on the prefrontal measure where metabolic differences tended to occur. The goal was to compare structural lesions that are common to Broca's aphasia. Until now, PET studies in aphasia had primarily emphasized defining the unique contribution of changes in brain metabolism. We focused on examining what is unique about the structural damage in Broca's aphasia when the metabolic changes are controlled. We found that the two groups differed on the presence of structural damage to the insula and posterior internal capsule. The behavioral differences are reflected primarily in speech fluency.

Such observations are consistent with the literature on the localization of Broca's aphasia (Levine & Sweet, 1983). Lesions in a number of cortical and subcortical regions can result in Broca's aphasia. This led to the hypothesis that an anatomic structure complex is fundamental to Broca's aphasia and that disrupting part of the circuitry will result in the syndrome.

Strikingly, when groups were controlled for degree of prefrontal metabolic changes, the key differences were structural in relation to subcortical structures and particularly the insula and the posterior internal capsule. The resulting behavioral difference was restricted to speech fluency. In previous reports, we have emphasized the metabolic consequences of structural damage and the importance of the distant effects of the lesion (Metter et al., 1987; Metter et al., 1989). With regard to aphasia, metabolic changes in the left temporoparietal region appear most important in explaining the language abnormalities.

This study points to the fact that the structural lesion needs to be understood and considered. The pattern of speech abnormality in Broca's aphasia appear to be dependent on the functional alterations in the left frontal region and the subcortical structural damage that alters this function. In

Broca's aphasia, the subcortical lesion has a greater degree of damage in the insula and posterior internal capsule than in other patients not having the same pattern of speech output. This fits well with our previous analyses showing that subcortical lesions directly affect speech fluency and indirectly affect it through the frontal lobes (Metter et al., 1988). These findings suggest that in Broca's aphasia, subcortical structural damage is critical for the distinction rather than the prefrontal changes. It is unclear whether this represents the presence of more than one functional system or differential damage to a single system.

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