

Grammatical Comprehension, Aphasic Syndromes and Neuroimaging

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ABSTRACT

This paper presents grammatical comprehension, computerized tomography (CT) and positron emission tomography (PET) data from a group of 43 aphasic patients. Comparison of comprehension performance with structural lesion (CT) revealed a correlation between comprehension of syntax and one inferior frontal lobe region (Broca's area) and one temporal lobe region (Wernicke's area). In contrast, comparison between comprehension performance and metabolic data demonstrated strong correlations between morphology and syntax comprehension and regional measures throughout the temporal, parietal and occipital lobes. The second aspect of the study looked at performance differences between diagnostic groups of aphasic patients. Very few behavioral differences were observed between aphasic groups (Broca's, Anomic, Conduction and Wernicke's), other than differences in severity. On all measures, Broca's and Wernicke's patients performed similarly. Overall, the findings support a model of brain-behavior relations in which language comprehension is represented by more widespread regions of the left hemisphere than is traditionally thought, and the aphasic syndromes differ from each other less than is traditionally believed.

INTRODUCTION

Throughout the history of aphasiology a central focus of research has been to utilize the clinical symptomatology of the aphasias to better understand the relationship

between the brain and language. However, until the advent of computerized tomography (CT), attempts to map behavioral patterns onto the brain could be approached only through post-mortem examination. Since that time, there have been remarkable advances in the technology of neuroimaging, making possible for the first time the study of the neurology of language and language impairments in the living brain.

Over the past nine years, our group has reported on number of studies of cerebral glucose metabolism in aphasia (Kempler *et al.* 1988, 1990; Metter *et al.* 1981, 1983, 1984, 1985, 1986, 1987a, 1989). The early studies considered approximately 15 subjects imaged on a positron emission tomography (PET) scanner with $1.6 \times 1.6 \times 1.0$ cm resolution. Over the past five years, the subjects were drawn from a group of 50 aphasic patients who had a history of a single cerebrovascular event, and underwent neuroimaging (CT and PET) studies and behavioral evaluations more than one month post onset. They were studied on a PET scanner with improved resolution of $1.1 \times 1.1 \times 1.0$ cm.

Several general comments can be made about glucose metabolism and brain damage and about its relationship to aphasia in particular. Firstly, and most generally, **it has become apparent that the areas of metabolic abnormality (hypometabolism) following either cerebral infarction or haemorrhage, as measured by PET, are much larger than the areas of structural damage measured by CT.** This finding is expected immediately post onset due to local inflammation and residual ischaemia. The more surprising finding is that large metabolic changes are observed (1) many months post onset, suggesting that it is not entirely due to an acute effect of regional inflammation; and (2) in regions very distant from the site of injury, suggesting that it is not a regional effect at all. For instance, we have demonstrated that subcortical lesions have a direct effect on metabolic function of specific cortical regions (Metter *et al.* 1988), and a number of investigators have found a metabolic deficit in cerebellar regions contralateral to the structural lesion (Metter *et al.* 1987a). These findings have been interpreted as a reflection of disrupted systems which are represented in widespread but specific regions throughout the brain. In addition to the basic finding that metabolic lesions are larger than structural lesions, we have found that those cases where this does not hold (i.e. structural lesions in which the metabolic abnormality was limited to the region of structural damage) were not associated with clinical symptomatology (Metter *et al.* 1987b; Metter 1987). This suggests that it is the interaction of structural damage and its functional consequences elsewhere in the brain that are critical in determining behavioral sequelae of cerebral lesions. In summary, it appears that the metabolic effects of local damage give us information about how the structurally damaged regions interact (or fail to interact) with other, crucial, but sometimes quite distant, regions.

With regard to aphasia, we have analyzed PET data from Broca's, Wernicke's and Conduction aphasic patients in order to investigate the relationship between the major

Gramma
syndromes and glucose metabolism (Metter *et al.* 1989). Previous structural damage and classic aphasic syndromes (e.g. Kertesz 1979) behavioral syndromes can be associated with structural damage in the left hemisphere. For example, Broca's aphasia (non-fluent speech, preserved comprehension) is associated with posterior frontal and subcortical left hemisphere lesions while Wernicke's aphasia (fluent speech, impaired comprehension) is typically associated with posterior temporal and subcortical left hemisphere lesions. While evaluation of the structural damage associated with these syndromes has contributed greatly to a specific model of cerebral organization (Kertesz 1979; Kertesz 1979), the glucose metabolic data give us a new picture of the regional deficits which distinguish these behavioral syndromes. Specifically, we have found that all aphasic patients that we have studied show abnormalities in the temporoparietal regions. The most common abnormality was the angular gyrus which had abnormal glucose metabolism (Metter *et al.* in press)¹. A second important observation is that the prefrontal cortex, including prefrontal areas, is metabolically abnormal in about 50% of cases. In examining the glucose metabolic activity of the left hemisphere, the supplementary motor area responds in a manner distinct from the prefrontal, Broca's area and primary motor cortex to structural damage (Metter *et al.* 1990). Therefore, the emerging picture of aphasic syndromes and glucose metabolism emphasizes both similarities and differences between these groups. Although temporal lobe metabolism appears uniform in all these aphasic patients, the behavioral syndromes can be distinguished from one another by the degree of hypofunction in other regions. The primary motor cortex distinguished the Conduction from Wernicke's and Broca's (relatively preserved in Conduction aphasia), while frontal lobe damage distinguished Broca's from Wernicke's aphasia (significantly more impaired in Broca's). We have argued that common features in these groups of aphasic patients are the metabolic changes in the temporal lobe, while unique features are the frontal and parietal changes (Metter *et al.* 1989).

In summary, the relationship between aphasia and brain damage is different from that observed for aphasia and structural brain damage. The metabolic lesions are much larger than structural lesions, and involve multiple systems throughout the brain. In addition, we have observed that these aphasic syndromes can be distinguished from one another by their behavioral disturbance, it is not in the same way that aphasic syndromes are distinguished with structural lesion data. That is, all aphasic syndromes show temporal lobe hypofunction, regardless of syndrome or structural damage. They appear to differ from one another in degree of metabolic impairment in the undamaged prefrontal and parietal cortex.

These provocative findings from the use of PET have been accompanied by a growing interest in aphasia by linguists and significant advances in linguistic theory. For instance these developments in the field of linguistics have produced grammatical characterizations of aphasia which differ in content from classical clinical diagnostic typology. Clinical diagnoses have relied largely on characteristics of speech such as fluency, incidence of paraphasias, and relative performance across task modalities, such as comprehension, production, repetition, writing and reading. In contrast, linguistic analyses of aphasic symptoms have tried to characterize aphasic impairments in terms of the grammatical system. Such theory-based analyses have already made important contributions to our understanding of aphasia, offering unified grammatical explanations for aspects of aphasic performance which had previously appeared to be unrelated (e.g. Caplan *et al.* 1985; Caplan and Futter 1986; Grodzinsky 1984, Kean 1977, 1985; Linebarger *et al.* 1983).

Linguistic analyses of aphasic symptoms, however, have to date not been related to results from currently available imaging techniques. The goal of this paper is to extend the enterprise of relating behavioral and neurological data by examining the relationships between aphasic syndromes, neuroimaging, and specific linguistic impairments. The specific questions we address in this paper are: (1) what is the relationship between CT findings and aspects of the comprehension of morphology and syntax? (2) what is the relationship between PET findings and comprehension performance? and (3) what is the relationship between clinically diagnosed aphasic syndrome and linguistic performance?

METHODS

Subjects

The patients were 43 right-handed, monolingual speakers of Standard American English with radiological evidence of a single (left hemisphere) infarct or haemorrhage and aphasia. All patients were studied more than one month post onset of the stroke. None was receiving therapy with anticonvulsants, antidepressants, or sedatives. Each subject was administered the Western Aphasia Battery (Kertesz 1982), which was used to classify the type of aphasia. Of the 43 patients sampled, 11 (26%) had Broca's aphasia, six (14%) had Wernicke's aphasia, eight (19%) had Conduction aphasia, and 18 (42%) had Anomic aphasia. See Table 1 for additional subject information. Each patient also received additional tests of auditory comprehension, and 38 of the patients also received a non-contrast CT scan and a resting state (F18)-fluorodeoxyglucose PET scan. Control subjects for the PET were 22 healthy volunteers with no known neurological disorder.

TABLE 1
Subject Information

ID number	Sex	Age	Mon
<u>Anomic aphasia</u>			
151	Male	66	
144	Male	57	
138*	Male	56	
129	Male	63	
135	Male	62	
125	Female	72	
110	Male	61	
109	Male	55	
112	Male	57	
111	Male	63	
103	Male	71	
108	Male	63	
104	Male	60	
116*	Male	60	
119	Male	73	
124	Male	58	
118*	Female	49	
122	Male	67	
Anomic means:		61.833	29
(n = 18) SD:		6.219	28
<u>Broca's aphasia</u>			
114	Male	59	
115*	Male	65	
142	Male	43	
136	Male	64	
128*	Male	57	
133	Male	62	
131	Male	59	
106	Male	62	
105	Male	64	
152	Male	56	
130	Male	63	
Broca's means:		59.455	20
(n = 11) SD:		6.219	16

TABLE 1—continued

ID number	Sex	Age	Months post onset	Aphasia quotient
Wernicke's aphasia				
121	Male	69	1	45.5
148	Male	74	32	57.0
132	Male	73	1	45.1
117	Male	63	5	30.3
154	Male	65	1	16.4
143	Male	63	2	38.0
Wernicke's means:		67.833	7	38.717
(n = 6) SD:		4.916	12.345	14.074
Conduction aphasia				
101	Male	60	6	74.1
102	Male	66	10	88.4
126	Male	55	35	84.5
123	Female	37	79	76.1
127	Female	67	10	80.7
147	Male	70	2	65.5
146	Male	58	3	76.7
139	Male	62	2	62.2
Conduction means:		59.375	18.375	76.025
(n = 8) SD means:		10.309	26.774	8.887
Total means:		61.605	22.093	67.393
(n = 43) SD:		7.32	24.245	24.522

* CT and PET data are not available for these patients.

PET—Functional Imaging

Each subject underwent a resting state PET scan using (F18)-2-fluoro-2-deoxy-D-glucose (NeuroECAT, CTI, Knoxville, TN). Subjects lay on the scanner bed in a darkened room, listening to ambient room noise (eyes and ears unoccluded), and had (F18)-2-fluoro-2-deoxy-D-glucose injected intravenously, and serial arterialized venous blood samples were drawn over time. Scanning was begun 40 min later, with the head positioned using the techniques described by Mazziotta *et al.* (1985), and the distance from the auditory meatus to the vertex was measured and used as a reference to determine plane localization. Values of local cerebral metabolic rates for glucose (LCMRGlc) were calculated as previously described (Phelps *et al.* 1979). Fifteen cerebral regions from each hemisphere (Fig. 1) were outlined on a video monitor using an interactive

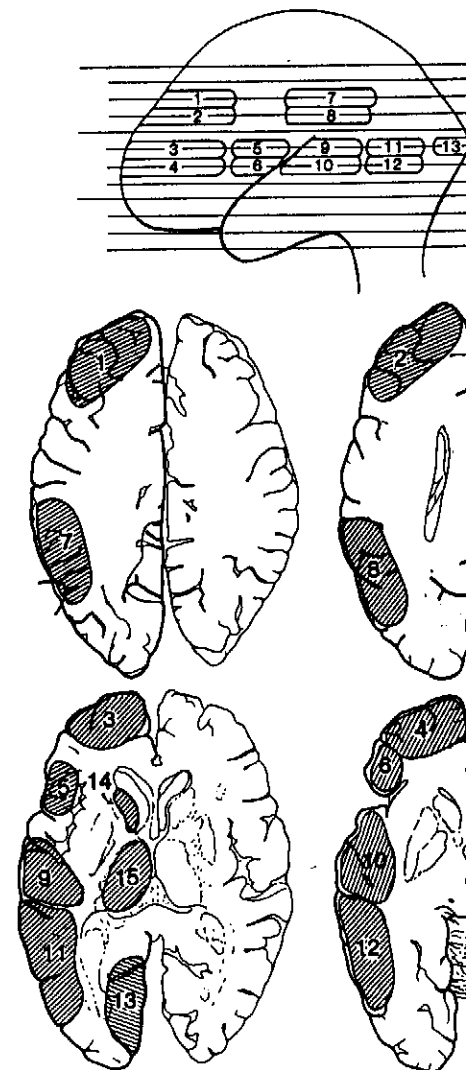


Figure 1. Schematic Illustration of Cerebral Region Damage and Glucose Metabolic Rates. Regions 1 and 2 are High Frontal; 3 and 4 are Low Frontal; 5 and 6, 9 and 10, Wernicke's; 11 and 12, Temporal; and 13, 14, 15, Thalamus. (Reprinted from Kempler *Cerebral Metabolism*, Archives of Neurology, 1988, American Medical Association.)

program, and regional LCMRGlc values were derived in milligrams of glucose per 100 g of tissue per min. Region of localization was based on the data of Matsui and Hirano (1978). The LCMRGlc values of aphasic patients' right hemispheres differ little from those of healthy controls but may show interindividual variation; therefore, left-right ratios for homologous regions were calculated and used in the analyses.

CT—Structural Imaging

Each subject had a computed tomographic scan (1200SX [Picker, Cleveland, OH] or 8800 Scanner [General Electric, Schenectady, NY]) at approximately the same time and in the same scanning plane as PET. The same regions as measured for glucose metabolism were rated. In addition, the anterior internal capsule, posterior internal capsule, insula and lenticular nuclei were rated. The regions of interest were rated using a five-point scale (0, normal; 1, atrophy; 2, structural damage with no tissue loss; 3, structural damage with partial tissue loss; 4, structural damage with complete tissue loss) by a neuroradiologist who was naive to the project and by one of the authors (EJM). Regional scores showed a 90% agreement between raters, and the two ratings were averaged to obtain an estimate of the degree of structural damage.

Grammatical Comprehension

The linguistic comprehension measures were comprised of a set of 45 comprehension items, assessing 15 syntactic and morphological structures (three items assessed understanding of each structure). All items followed a picture-matching format in which the subject selected one (of two, three or four) pictures which best matched an auditorily presented sentence. The distractor pictures (wrong answers) represented different permutations of the grammatical relations involved in the test sentence. Therefore, it was not possible for a subject to consistently select the correct answer by virtue of lexical knowledge alone. The subtests were divided into those which assessed syntax vs those which assessed morphology, and all items were drawn from the *CYCLE-R* (Curtiss—Yamada Comprehensive Language Evaluation—Receptive Measures) (Curtiss and Yamada 1987). The morphology items assessed two distinct types of morphology—items which express grammatical inflection (e.g. marking of number, person, tense) by a change in the word form; and items which express grammatical relationships by the use of an individual word from "closed classes" such as prepositions (e.g. "with") or modals (e.g. "will"). Syntactic structures (sentences) are also assessed in two distinct type of sentences, those which involve only one clause (simple structures) and those which involve two or more clauses (complex structures which involve embedding, such as relative clauses).

A list of structures tested, with examples, is presented in the Appendix, and a

sample item is presented in Fig. 2. Scoring procedures vary. For the CT and PET analyses, the total number of subtests passed (correctly understood) in each component was the measure of comprehension for that component. For the linguistic analyses, the total percentage of correct responses for each component (e.g. closed class morphology or simple syntax) was used for the comparisons.

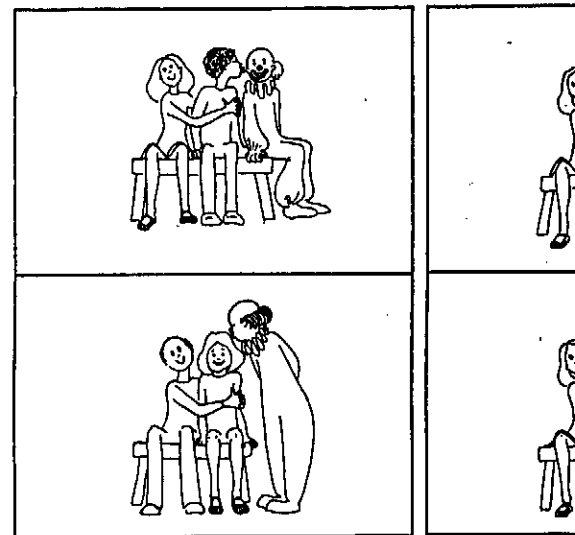


Figure 2. Sample Picture Response Array for Syntax
"The Girl is Hugging the Boy that the Clo

RESULTS

To assess the first question, *What is the relationship between structural damage and comprehension of morphology and syntax?* Correlation coefficients were calculated between CT region ratings and comprehension of morphology and syntax subtests. There were no significant correlations between structural damage and comprehension of morphology or syntax. Correlations between comprehension of complex syntax and structural damage which were a single temporal lobe measure (Wernicke's area, $p = 0.0191$) and one of the Broca's measures ($r = 0.333$) do not change significantly if the two morphology subtests are summed into two scores, one representing morphology (S

region) and one representing syntax (which remains significant for only the Wernicke region). These results are presented in Table 2 and Fig. 3.

TABLE 2
Correlations Between Grammatical Comprehension and Areas of Structural Damage

Region	Morphology		Syntax	
	Inflectional	Closed Class	Simple	Complex
Frontal 4	0.13258	0.17396	0.13001	0.17184
Frontal 3	0.13429	0.09260	-0.00437	-0.02843
Frontal 2	0.16253	0.23612	-0.01750	-0.05676
Frontal 1	0.11041	0.09016	0.02878	-0.02018
Broca's 2	0.04916	-0.07377	-0.22932	-0.26261
Broca's 1	0.07721	-0.16985	-0.22207	-0.33331*
High parietal	0.13957	-0.24723	-0.17918	-0.01002
Parietal	-0.00758	-0.22977	-0.15700	-0.20939
Wernicke's 2	-0.06162	-0.01690	-0.18236	-0.26723
Wernicke's 1	-0.31034	-0.21056	-0.29063	-0.37844*
Temporal 2	-0.06796	-0.23157	-0.14757	-0.28108
Temporal 1	-0.21539	-0.23200	-0.18830	-0.13536
Occipital	-0.10477	-0.16082	-0.15247	-0.13932
Caudate	-0.11597	-0.21291	-0.04250	-0.12640
Thalamus	0.36587	0.03987	-0.26442	-0.09843
Cerebellum	-0.05958	-0.06909	-0.11664	0.03958
Ant. int. cap.	0.10978	0.11903	0.07533	0.07289
Post. int. cap.	0.07683	0.11746	-0.10390	0.10243
Lenticular nuc.	0.15070	0.17987	0.09327	0.18513
Insula	0.04404	0.21771	-0.05146	0.07064

* $p < 0.05$.

These CT results contrast with the significant correlations found between regions of hypometabolism (PET) and (poor) performance on linguistic measures. Firstly, occipital lobe metabolism correlated with all linguistic measures, possibly reflecting the proximity of temporal lobe language zones to the occipital lobe, and the visual processing component of these language tasks. Secondly, posterior temporal lobe (not Wernicke's area) correlated significantly with comprehension of both syntax measures and closed class morphology. Thirdly, the parietal lobe measure correlated

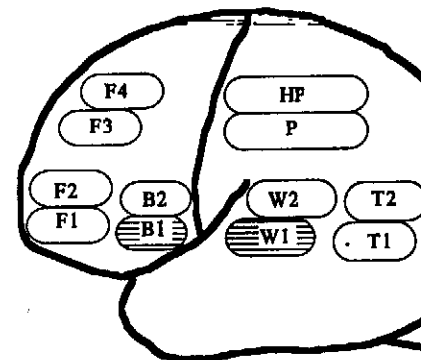


Figure 3. Regions of Structural Brain Damage Which Correlate with Grammatical Comprehension Deficits.

with both simple and complex syntax comprehension. If summed to yield a single morphology score and a single syntax score, the correlations in these regions remain essentially the same, with the exception that the high parietal region significantly correlate with total syntax comprehension ($r = 0.17$), but it did not correlate with either simple or complex syntax comprehension.

Notwithstanding the possibility that this many comparisons are statistically meaningless yet statistically significant correlations with behavioral symptoms, a conservative view of these findings is consistent with the hypothesis that metabolic damage as reflected in PET corresponds to the behavioral symptomatology of aphasia than structural damage. This is borne out in the greater number of significant correlations between PET and the higher level of significance reached in the CT. That is, for example, although only a weak correlation was found between structural damage in Wernicke's area and comprehension of syntax ($p < 0.05$), the relationship was more strongly represented in the PET. To reinforce the important point that any model of brain-behavior relationships must consider the interplay of structural damage and the metabolic state throughout the brain.

To address the third question of the study, *What is the relationship between structural damage and linguistic performance?*, we conducted two separate analyses. We looked at performance of each diagnostic category (Broca's aphasia, Wernicke's aphasia, Anomic) on morphology and syntax subtests. A two-factor analysis of variance revealed a significant effect of group [$F(3,39) = 7.848$, $p < 0.05$].

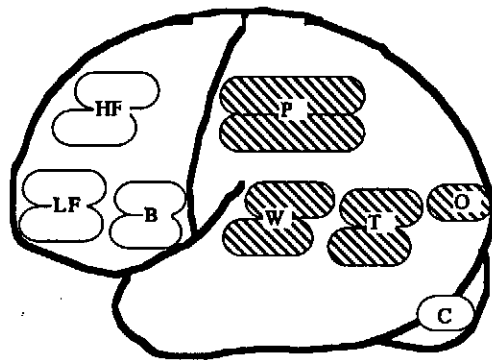


Figure 4. Regions of Glucose Hypometabolism which Correlate with Grammatical Comprehension Deficits.

sentence type [$F(1,39) = 20.846, p = 0.0001$], and a group-by-sentence type interaction [$F(1,3) = 3.861, p = 0.0164$]. Examining the data in more detail, we find that the effect of group reflects the different levels of severity of each diagnostic group with Anomics being least impaired and Wernicke's being most impaired. The effect of sentence type is shown in Fig. 5 by the consistently superior performance of all four groups on morphology compared with syntax. The group-by-sentence type interaction stems from the fact that the two less impaired groups (Anomic and Conduction) perform similarly on both morphology and syntax items, while the two more severely impaired patient groups (Broca's and Wernicke's) both perform significantly worse on syntax items ($t = 3.653, p = 0.0044$ and $t = 5.825, p = 0.0021$ respectively).

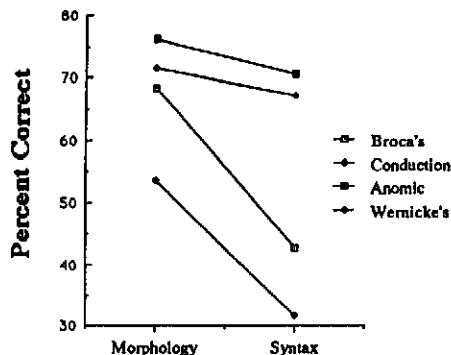


Figure 5. Comprehension of Morphology and Syntax for Four Groups of Aphasic Patients.

We also analyzed the data looking at the morphology (bound vs closed class morphology; simple vs complex syntax) syndrome. Comparison of the four aphasic groups and four controls (two factor ANOVA with repeated measures) revealed a significant effect of group [$F(3,39) = 5.632, p = 0.0026$] and a significant effect of sentence type [$F(3,39) = 1.958, p = 0.0503$]. Figure 6 shows that the significant

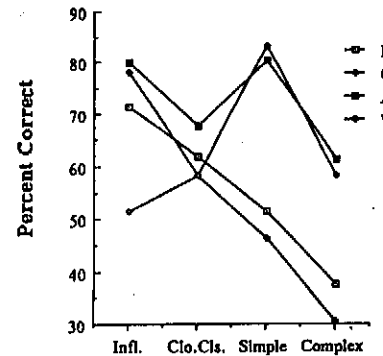


Figure 6. Comprehension of Four Aspects of Grammar, Closed Class Morphology, Simple Syntax, and Complex Syntax for Four Groups of Aphasic Patients.

TABLE 3
Correlations Between Grammatical Comprehension and Cerebral Metabolism

Region	Morphology	
	Inflectional	Closed class
High frontal	0.10266	-0.03453
Low frontal	0.03590	0.08604
Broca's	0.0089	0.00827
Parietal	0.11496	0.26147
Wernicke's	0.21304	0.08727
Temporal	0.30933	0.34974*
Occipital	0.36549*	0.39183*
Caudate	0.00313	-0.05520
Thalamus	0.03120	-0.10246

* $p < 0.05$.
† $p < 0.001$.
‡ $p < 0.01$.

the fact that the anomics and conduction aphasics were generally less impaired (particularly on syntax items) than the Broca's and Wernicke's patients. *Post-hoc* Scheffe *F*-tests again revealed significant differences between the mildly impaired patients (Anomic and Conduction) and the severely impaired patients (Broca's and Wernicke's) on comprehension of simple syntax, and a significant difference between Wernicke's and all other groups on comprehension of inflectional morphology. There were no significant differences between the aphasic groups on comprehension of closed class morphology or complex syntax.

SUMMARY AND DISCUSSION

This investigation of comprehension, brain structure and brain metabolism in a population of aphasic patients has confirmed and perhaps expanded what we know about the relationship between the brain and behavior. Firstly, our comparison of comprehension performance with ratings of structural lesions demonstrated that relatively small and well-localized structural lesions in the frontal and temporal lobe cause significant syntactic comprehension deficits. This, of course, is not surprising, and is consistent with findings from the time of Wernicke to the present. Perhaps more interesting, none of the morphological comprehension scores reached significance with any particular region of structural damage, suggesting that these two aspects of language (morphology and syntax) may be differentially disrupted by structural lesions in the left hemisphere. This could be seen as a confirmation of linguistically motivated distinctions between the structural properties of morphological and syntactic components of the grammar. Our subsequent comparison of comprehension performance with metabolic lesions revealed a much less familiar picture of brain-behavior relations. Specifically, poor comprehension of both syntax and morphology seem to correlate significantly with metabolic lesions in the posterior temporal, parietal and occipital lobes, yielding an impression of a much wider focus of comprehension within the left hemisphere than was given by the CT data. While it is probable that some of this correlation may reflect perhaps extraneous aspects of the task (e.g. using visual response arrays will involve the functions of the occipital lobe which are not necessarily part of language performance), it would be imprudent to disregard all of these findings as artifactual. It is interesting to observe that the correlations between comprehension and metabolic abnormalities not only cover a wider region of the left hemisphere than structural correlations, but are in fact higher correlations and include both syntax and morphology. It is likely that what we are observing in these results is the fact that Broca's and Wernicke's areas, while structurally important for auditory comprehension, do not operate in isolation, but rather in tight integration with surrounding temporal, parietal and occipital regions. Taken together, the CT and PET data confirm what we already knew from previous studies: small structural lesions

create large metabolic lesions. This investigation has gone beyond the traditional behavioral data into the larger picture. Brain-behavior correlations in grammatical comprehension, appear to indicate stronger correlations between impairment and the larger metabolic lesions than between impairment and the smaller structural lesions. These findings suggest that our hard-won maps of localization of function within the brain are somewhat less localized to small regions. Minimally, the choice of methodology (e.g. CT vs PET) will create a degree to which cortical function is focally represented.

The data on diagnostic groups and grammatical comprehension at similarities and differences between the aphasic syndromes are relevant to auditory comprehension. Our findings were consistent with previous reports. Firstly, contrary to recent reports indicating that morphology is particularly vulnerable to brain injury in aphasia (e.g. Bates and Warrington 1982), morphological comprehension appeared to be better preserved than syntactic comprehension. In fact, all of our aphasic groups performed better on morphology than on syntax (although the performance difference was not significant for the more severely impaired Broca's and Wernicke's groups). These findings are relevant to the centuries-old debate regarding the relationship between comprehension impairment with aphasic syndromes. It is not clear if there has been a general consensus on the status of comprehension impairment. On the one hand, the standard lore in aphasiology was that Broca's and Wernicke's (Wernicke's) aphasics suffer from specific auditory comprehension impairment, while in other aphasic syndromes (e.g. Goodglass and Kaplan 1972) it is production. Recent investigations with Broca's aphasics have emphasized the relationship between comprehension and production (Caramazza and Zurif 1979; Wulfeck 1988). Our findings do not clearly support either view. Although there were significant differences in overall severity, we did not find correlations between the grammatical comprehension performance of Broca's and Wernicke's aphasics. Although it is unquestionable that Broca's and Wernicke's aphasics exhibit characteristic symptoms (e.g. fluency), our data suggest that a traditional model of aphasia may not map neatly or meaningfully on to the data. Rather, grammatical comprehension appears quite different in Broca's and Wernicke's syndromes.

The picture of aphasic impairments that we are building up is quite different to a small degree to traditional views established by analysis of brain structure and performance on standardized aphasia batteries. In keeping with previous work we find that aphasic patients have language comprehension impairment associated with temporal lobe lesions. However, the details of both the findings are somewhat different than the traditional model.

We find that the locus of damage which contributes most to the comprehension deficit is not focal, but rather involves the entire posterior peri-sylvian region, including widespread temporal, parietal and occipital areas. Secondly, we find little to distinguish traditional aphasic syndromes in terms of grammatical comprehension as measured by the CYCLE. In general, we believe this suggests a more unified view of aphasia and a more complex view of the mapping of language onto the brain. Under this view, some aphasic symptoms reflect a neurological commonality among aphasic patients, and therefore serve to de-emphasize syndrome-specific differences. Our findings may be interpreted to suggest, therefore, that language function, as an example of cognitive functions more generally, may not be as focally represented in the brain as suggested on the basis of structural data alone.

NOTES

1. It should be noted that we have not studied transcortical aphasics with damage in the supplementary motor area. These more purely frontal lobe aphasias may behave differently than the more posteriorly based aphasias.

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Appendix

Examples of Grammatical Comprehension Stimuli

MORPHOLOGY

Inflectional morphology

Possessive 's

Point to the baby's bear.

Verb singular

The sheep stands.

Noun plurals

Point to the picture of the boxes.

Tense/aspect -ed

The mother dressed the baby.

Closed Class morphology

Tense/aspect "will"

The girl will open the present.

Case marking preposition "with"

The boy is carrying the suitcase with the man.

SYNTAX

Simple sentences

Active voice

The dog is chasing the cat.

Passive voice

The boy is being pushed by the girl.

Wh- Object questions

Who is the girl pushing?

Complex sentences

Subject relatives

The boy who is smiling is pulling the girl.

Object relatives

The boy is pushing the girl who is happy.

Subject relatives ending in noun-verb

The girl who is pulling the boy is smiling.

Object relatives with relativized object

The boy is kissing the girl that the clown is hugging.

Double embedding

The girl that is chasing the clown that is big is little

Double function relatives

The boy that the girl is pulling is pulling the clown.