Cerebral Glucose Metabolism: Differences in
Wernicke's, Broca's, and Conduction Aphasias

E. Jeffrey Metter and Daniel Kempler
Veteran's Administration Medical Center, Sepulveda, California and
School of Medicine, University of California, Los Angeles, California

Wayne R. Hanson
Veteran's Administration Medical Center, Sepulveda, California

Catherine Jackson, John C. Mazziotta and Michael E. Phelps
School of Medicine, University of California, Los Angeles, California

Anatomic classifications of aphasia propose that lesions to specific brain regions result in definable and distinct aphasic syndromes.   Broca's, Wernicke's and Conduction aphasias, the syndromes which have formed the core of traditional aphasia classification and modeling, have been of particular interest as they are all felt to result from perisylvian damage. A number of studies have shown differences in regional damage with specific syndromes (Kertesz, 1979; Kertesz et al., 1979; Mazzocchi et al., 1979). However, several problems exist with classification based on localization.   First, it is difficult to predict aphasic syndrome based on lesion localization (Benson, 1979).   Second, recent studies have found specific language dysfunctions in all aphasic subjects relatively independent of aphasic syndrome (e.g. the syntax deficit found even in Broca's aphasia (Zurif, 1984)).

Research with (F18)fluorodeoxyglucose PET has shown that structural lesions are associated with metabolic changes in brain regions beyond the site of structural damage. We have previously demonstrated that specific metabolic patterns exist but made no attempt to correlate metabolic and structural patterns with specific aphasic syndromes (Metter et al., 1985). Of particular interest has been metabolic changes in structurally undamaged pre-frontal cortex, caudate, thalamus and contralateral cerebellum (Metter et al. 1986).   With these issues in mind, we compared structural and metabolic distribution in Wernicke's, Broca's, and Conduction aphasias.

METHODS

Subjects.   Thirty-five patients were selected who had an aphasia attributed to a single vascular lesion, with no previous history of stroke, and no evidence of a second stroke on x-ray CT or neurological examination. Patients were studied greater than one month post onset of the stroke.   None were taking anticonvulsants, antidepressants or sedating medications.   Each subject was given the Western Aphasia Battery (WAB), which was used to classify the type of aphasia based on criteria presented by the test (Kertesz, 1981). Based on the WAB, eight subjects (23%) had Broca's aphasia, four (11%) Wernicke's aphasia, and five (14%) Conduction aphasia.   This distribution is similar to that found by other researchers (Benson, 1979); Goodglass and Kaplan, 1972; Kertesz, 1979).

PET.   Each patient was studied using FDG in a resting state with eyes and ears unoccluded.   Patients lay on the scanner bed in a darkened room, listening to ambient room noise and had FDG injected intravenously. After 40 minutes scanning was begun. All patients were studied on the NeuroECAT (CTI, Knoxville, TN) (Hoffman et al., 1983).

Scans were studied quantitatively by analyzing 16 regions of interest in each hemisphere outlined on a video monitor using an interactive program. Regional measures were reported as local cerebral metabolic rates for glucose (LCMRGlc) as determined by the model of Phelps et al. (1979). Because of marked individual variations in global metabolic rates of glucose and small sample size, regional LCMRGlc were expressed as a left/right hemisphere ratio for each region. Right hemisphere LCMRGlc for these 16 regions have been shown to differ from control subjects, with most of the variation being accounted for by reduced high frontal LCMRGlc and elevated head of the Caudate LCMRGlc. Thus, in general, the left/right ratio reflects left hemisphere hypometabolism.

X-ray CT. Each subject had an x-ray CT on a Picker 1200SX or GE 8800 scanner at approximately the time of FDG PET. Regions which corresponded to the regions measured by PET, as well as the insula, anterior internal capsule, posterior internal capsule, lenticular nuclei, and body of the caudate were analyzed by a neuroradiologist unfamiliar with the project, and by the first author. Each region was graded on a 5-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. Ninety percent overall agreement in ratings was obtained. The score for each region was the average of the two readings.

RESULTS

Table 1 shows the average test scores on the WAB for the three aphasic groups. The Wernicke's aphasic patients showed the most severe aphasia as estimated by the aphasia quotient (AQ), and were associated with the most severe comprehension and naming dysfunction of the three groups. The Broca's aphasic patients had the most severe problems with spontaneous speech, while the conduction aphasia patients had near normal comprehension with more severe repetition difficulties.

Figure 1 shows the distribution of glucose metabolism and the associated structural damage as measured by x-ray CT for Broca's, Wernicke's and Conduction aphasic subjects. The following observations can be made from the group comparisons. (1) Broca's aphasic subjects had the largest structural damage with the lesion extending farther frontally and involving more deep brain structures, including insula and basal ganglia, similarly to cases described by Mohr et al. (1978). (2) All three syndromes had similar structural involvement of the superior temporal lobe (Wernicke's area), and all involved the parietal lobe, though Broca's aphasia tended to show more parietal and less posterior temporal structural damage than the other two syndromes. (3) Conduction aphasia differed from Wernicke's aphasia, with greater damage to the posterior temporal regions and less involvement in the insula and lenticular nuclei. (4) Metabolically, the three aphasias showed a similar degree of left < right metabolic asymmetry in the temporal lobe. (5) The three aphasias differed in the extent of prefrontal lobe, Broca's region, and, to a lesser extent, parietal lobe metabolic asymmetry. All Broca's aphasic patients showed severe left/right asymmetry in these regions, Wernicke's showed mild-to-moderate aysmmetry and Conduction aphasia normal in the prefrontal cortex.

===

Table 1. **Western Aphasia Battery** scores for each aphasic syndrome.

	Spontaneous Speech (20)	Comprehension (20)	Repetition (10)	Naming (10)	AQ (100)
Conduction	17.2+1.5	17.5+1.8	6.6+.5	7.8+1.9	81+6
Wernicke's	8.8+1.3	10.0+3.5	3.0+2.7	1.2+1.0	36+11
Broca's	6.9+3.8	14.7+3.1	3.3+2.1	3.3+2.8	43+19

===

Each score is the mean +/- standard deviation
Number in parenthesis is the total possible score for the test

DISCUSSION

The three aphasic syndromes differed primarily in the extent of deep (insular, lenticular, and internal capsule) structural damage and the metabolic changes in prefrontal cortex. Previous studies have not demonstrated physiologic changes in the prefrontal cortex in these aphasic syndromes.

Structural lesions in the prefrontal area have been associated with transcortical motor aphasia, which has been described as an adynamic state by Luria (1970). Freedman et al. (1984) proposed that transcortical motor aphasia represents a disconnection of the supplementary motor area from Broca's region, with the supplementary motor area being the initiator of expressive activity. Fuster (1980) believes that the frontal lobes are responsible for temporal planning, which is disrupted in transcortical motor aphasia. These hypotheses suggest that the frontal lobes are involved with the initiation and maintenance of behavior.

We have found a strong correlation between metabolic measures for prefrontal regions with those for Broca's area, parietal, caudate, thalamus and cerebellum in aphasic subjects (Metter et al. 1986). These regions formed a strong component, accounting for 61% of the variance in a principal components analysis. The measures correlated with functional motor loss of the arms and legs, spontaneous speech (WAB) and writing (WAB). In normal subjects a strong correlation was found between prefrontal cortex and decision bias determined by signal detection theory (Riege et al., 1985). Together the observations suggest a role in planning and execution of behavior, as previously hypothesized.

One explanation for the frontal differences and its association with aphasia is that the degree of frontal hypometabolism is determined by lesion size and is mainly associated with aphasia severity. However, Wernicke's aphasic subjects as a group were more severely aphasic than Broca's and Conduction aphasic subjects (based on WAB AQ), yet their lesions were similar in size to the Conduction patients and less extensive than Broca's patients. Alternatively, the prefrontal differences could relate to differences in the
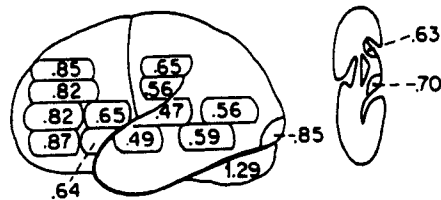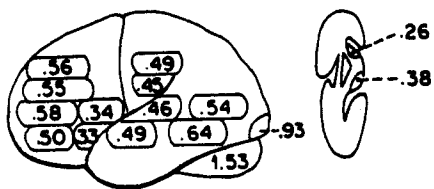
-99-
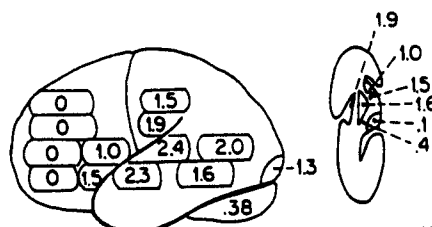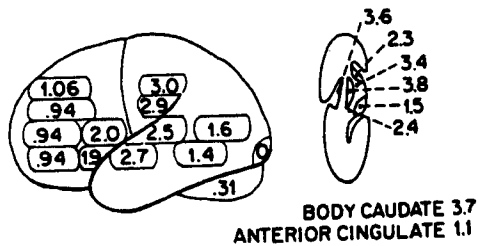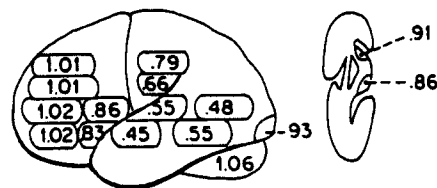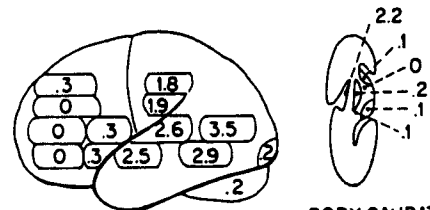
Figure 1. Metabolic and structural distributions for Wernicke's, Broca's, and Conduction Aphasia. The figure presents either the left/right metabolic ratio, and the mean structural score from x-ray CT for each region examined.

extent of damage to deep brain structures.   However, the depth of the lesion could not explain differences in severity,   as Wernicke's   aphasic patients were as severely aphasic as were Broca's patients.

The metabolic and structural dysfunction in the three aphasic syndromes suggest an account for the classic debate between those who view aphasia as a single syndrome and those viewing it as a collection of multiple and distinct syndromes.   A striking observation in our subjects was the similarity of structural and metabolic dysfunction in the measured parts of the temporal lobe.   The presence of such dysfunction in Broca's aphasia may well explain the presence of language comprehension abnormalities found in this group as well as with fluent aphasias (Zurif, 1984).   A common language deficit in all groups may reflect the commonality of regional hypometabolism observed in the temporal and parietal regions.   This would be consistent with the theoretical position that there is only one aphasia,   resulting from temporoparietal dysfunction,   differences between syndromes being essentially epiphenomena (Poeck, 1983).

Conduction aphasia was of particular interest because in the classical model it is attributed to disconnection of the frontal from posterior language areas by damage to the arcuate fasciculus (Geschwind,   1965;   Benson et al.,   1973), although other explanations have been offered (Warrington and Shallice,   1969;   Shallice and Warrington,   1977; Levine and Calvanio, 1982). The mild degree of Broca's regional asymmetry and absence of frontal asymmetry in conduction compared with Wernicke's and Broca's aphasia argues against a disconnection explanation.   The observations are consistent with a performance model,   where performance differences between the three types of aphasia are seen as resulting from functional frontal changes.   The milder degree of communication problems found in conduction aphasia is associated with a normal appearing frontal region, while the severe struggle in communication in Broca's aphasia corresponds to severe frontal asymmetry.   Such differences argue for breakdown in functional brain systems which must interplay for productive communication.

ACKNOWLEDGMENT

REFERENCES

Benson,   D.F.,   Sheremata,   W.A.,   Bouchard,   R.,   Segarra,   J.,   Price,   D. and Geschwind,   N.   Conduction aphasia-a clinicopathological study.   Archives of Neurology, 28:339-346, 1973.

Benson,   D.F.   Aphasia, Alexia, Agraphia.   New York: Churchill/Livingston, 1979.

Freedman,   M.,   Alexander,   M.P.   and Naeser,   M.A.   Anatomic basis of transcortical motor aphasia.   Neurology, 34:409-417, 1984.

Fuster,   J.   The Prefrontal Cortex: Anatomy, Physiology, and Neuropsychology of the Frontal Lobe.   New York: Raven, 1980.

Geschwind,   N.   Disconnection syndromes in animals and man.   Brain, 88:237-294, and 585-644, 1965.

Goodglass,   H.   and Kaplan,   E.   The Assessment of Aphasic Disorders. Philadelphia: Lea & Febiger,   1972.

Hoffman, E.J., Phelps, M.. and Huang, S.C. Performance evaluation of a positron tomograph designed for brain imaging. Journal of Nuclear Medicine, 24:245-257, 1983.

Kertesz, A. Aphasia and Associated Disorders: Taxonomy, Localization, and Recovery. Orlando, Florida: Grune & Stratton, 1979.

Kertesz, A. Western Aphasia Battery. New York: Grune & Stratton, 1981.

Kertesz, A., Harlock, W. and Coates, R. Computer tomographic localization, lesion size and prognosis in aphasia and nonverbal impairment. Brain and Language, 8:34-50, 1979.

Levine, D.N. and Calvanio, R. Conduction aphasia. In H.S. Kirshner and F.R. Freeman (Eds.), The Neurology of Aphasia. Lisse: Swets and Zeitlinger B.V., 79, 1982.

Luria, A.R. Traumatic Aphasia. The Hague: Mouton, 1970.

Mazzocchi, R. and Vignolo, L.A. Localization of lesions in aphasia: Clinical-CT scan correlation in stroke patients. Cortex, 15:627-653, 1979.

Metter, E.J., Dempler, D., Jackson, C.A., Hanson, W.R., Riege, W.H., Camras, L., Mazziotta, J.C. and Phelps, M.E. Crossed cerebellar glucose metabolism in chronic aphasic patients. Neurology 38(Suppl 1):316, 1986.

Metter, E.J., Riege, W.H., Hanson, W.R., Jackson, C.A., Mazziotta, J.C. and Phelps, M.E. Remote metabolic effects in aphasic stroke patients. In R.H. Brookshire (Ed.), Clinical Aphasiology, Volume 15, 1985. Minneapolis, MN: BRK Publishers, 1985.

Mohr, J.P., Pessin, M.S. , Finkelstein, S. , Funkenstein, H.H., Duncan, G.W. and Davis, K.R. Broca aphasia: Pathological and clinical. Neurology 28:311-324, 1978.

Phelps, M.E., Huang, S.C., Hoffman, E.J., Selin, C.S., Sokoloff, L. and Kuhl, D.E. Tomographic measurement of local cerebral metabolic rate in humans with (F-189 2-fluoro-2-deoxyglucose: validation of method. Annals of Neurology 6:371-388, 1979.

Poeck, K. What do we mean by "Aphasic Syndromes?" A neurologist's view. Brain and Language 20:79-89, 1983.

Riege, W.H., Metter, E.J., Kuhl, D.E. and Phelps, M.E. Brain glucose metabolism and memory functions: Age decrease in factor scores. Journal of Gerontology 40:459-467, 1985.

Shallice, T. and Warrington, E.K. Auditory-verbal short-term memory impairment and conduction aphasia. Brain and Language 4:479-491, 1977.

Warrington, E.K. and Shallice, T. The selective impairment of auditory-verbal short-term memory. Brain 92:885-896, 1969.

Zurif, E. Psycholinguistic interpretations of the aphasias. In D. Caplan, A.R. LeCours and A. Smith, Biological Perspective on Language. Cambridge: MIT Press, 158, 1984.

DISCUSSION

Q: Will you tell me what you mean by language?

A: I don't have a good definition of language. It seems to me that languages encompasses much more than the discussions we had this morning and yesterday on the issues of linguistics. I'd much rather talk about communication, to be honest. I have real problems with people who say that this individual who can't communicate doesn't have a language problem. I don't have a good definition, maybe somebody else has one.

C: What I am referring to in terms of the temporoparietal issue, is that if you structurally damage these regions, people will have major problems with communication, with understanding--they have problems putting together a correct output.

Q: Have you studied patients with anterior cerebral artery lesions, posterior cerebral artery lesions, and Parkinson's disease, and would any of those groups be useful controls as you try to develop hypotheses about functional deficits in more traditional patients?

A: Yes. We have studied two patients with anterior cerebral artery infarction. One of them was reported here in 1983. The other patient is included in Wayne Hanson's study. What we saw metabolically in one patient was that the metabolic defect extended far posteriorly, and in the other individual it extended into the parietal region, but spared the temporal lobe. As far as Parkinson's disease, we have studied a number of patients with this disorder. They show mild to moderate declines in global and cortical metabolism. To analyze the data in a different way, we examined cortical-cortical interrelations and found that in patients with Parkinson's or Huntington's disease, there was a major loss of cortical to cortical strong metabolic correlations. We have used this observation to argue that part of the function of the basal ganglia is to help the cortex communicate between regions. In fact, if you look at the metabolic changes that we see in aphasic individuals what you find is that the caudate is frequently metabolically involved. In another study, we found it to be strongly correlated with what happens in the frontal and parietal lobes.

C: Using the WAB taxonomic classification, a Broca's aphasic and a Wernicke's aphasic could be exactly the same except that the Broca's score could be one score lower on fluency. Similarly, a conduction aphasic patient could score exactly the same as a different Broca's aphasic patient, except for a one point difference in fluency. I think that your paper may not say anything about the reality of traditional aphasic classification, but may simply say that the greater the involvement of the prefrontal area, the more likely you are to have a loss in fluency, or the more loss in fluency you may have.

A: I agree with you. In a larger study, where we were interested in looking at the cerebellum, we found very strong correlations between contralateral cerebellar metabolism with frontal, parietal, caudate and thalamic metabolism. The behaviors found to correlate with these regional measures were hemiplegia, spontaneous speech and writing. The spontaneous speech measure was fluency. I think that fluency plays an important role. Despite the limitation of the WAB, we found that these groups defined with the WAB study showed very distinctively different patterns.

C: Because of the way it scores patients, I don't think that the WAB tells us anything about classification. For example, a conduction aphasic and a Wernicke's aphasic patient may be exactly the same on the WAB except for a one-point difference in auditory comprehension. Your Wernicke's patients had somewhat poorer auditory comprehension, or somewhat greater changes in the temporal parietal region than your conduction aphasic patients, so all that says is the more changes in the temporoparietal cue the more comprehension loss. I don't think that tells us anything about

classification. I think it only tells us that there are parts of the brain that are related to fluency, and there are parts of the brain that are related to auditory comprehension. You don't need to classify patients to come up with that.

A: The issue of the paper was not to support or not support this classificaiton, but rather to examine metabolic and structural differences. The conduction aphasic patients were very mild, while the Wernicke aphasic patients were quite severe, so that we are not talking about a single point difference on the WAB.

C: There may be some subjects that are quite similar and differ by only one point.

A: The point of the paper and the point of the approach was to look at what effect brain damage has on our ability to communicate. What traditional models tell us is that structural lesions in and around the perisylvian area are important for language. What the study I have presented tries to show is that if you go beyond structural damage and ask what is happening functionally within the brain, we find very prominent functional changes elsewhere. Now in this talk I haven't focused on the right hemisphere, where other changes may be important. From the way I have analyzed the data, it is very clear that there are prominent frontal changes that need to be accounted for.

C: I think that is the important part of your paper, and I think that the classification part of it muddies the waters.

A: The classification is the traditional way that people have analyzed aphasia clinically. I don't want to get into a debate about the value of the classification. I have chosen to use it as a tool to investigate our metabolic data.

Q: You stated that there are always motoric components to language tasks. You have also implied that your definition of language is more encompassing that communication per se. I wonder whether you have looked at gestural or constructional ability of these patients given the distant effects that you are discussing.

A: We haven't done it with these patients. We have in the past, with different groups of patients. We haven't done it in any extensive and consistent way.