

Conduction Aphasia: Subgroups Based on
Behavior, Anatomy and Physiology

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Conduction aphasia has been called the "unwanted child" of our field (Green and Howes, 1977). Since its inception, it has been alternately embraced, discarded and renamed. Although only a small number of patients (about 10%) are diagnosed with conduction aphasia (Benson, 1979), the symptoms, the psychological models and the neuropathological causes of the syndrome are still the subject of active debate over a century after it was first identified. Creative and competing theories have been proposed to explain this aphasic profile, but thus far little agreement has been reached.

Briefly stated, the symptoms of conduction aphasia are (1) striking difficulties in repetition of spoken language alongside (2) relatively good comprehension, (3) fluent but often circumlocutory and/or paraphasic spontaneous speech, and (4) word-finding difficulties (Benson, 1979; Kertesz, 1979).

Since the time of Wernicke (who predicted such a disorder in 1874), both the neuroanatomical and the cognitive bases of the syndrome have been disputed (e.g., Green and Howes, 1977; Strub and Gardener, 1974; Benson et al., 1973). The cognitive issues currently hinge on determining whether the repetition deficit is due to an auditory-verbal short-term memory deficit (e.g., Warrington and Shallice, 1969) or a linguistic disturbance (Strub and Gardner, 1974).

The neuroanatomical debates involve discussion of the neurological mechanism responsible for such a syndrome, and presume various psychological models of language. Several distinct models have been presented. First, and perhaps most popular, is the disconnection theory, first proposed by Wernicke and later championed by Geschwind (1965). In this model, human language is subserved primarily by two independent psychological and cortical centers; an anterior, speech production center or Broca's area, and a posterior auditory perception and apprehension center, or Wernicke's area. These two centers are connected by a long fiber tract, which according to Geschwind (1965), "runs from the posterior superior temporal regions, arches around the posterior end of the Sylvian fissure and then runs forward in the lower parietal lobe, eventually to reach the frontal lobe, and in particular Broca's area." In conduction aphasia these connective fiber tracts are

purported to be damaged, disconnecting the anterior language production and the posterior language perception centers.

An alternative model of the disturbance in conduction aphasia (or "central" aphasia, to use his term) is attributed to Goldstein (1948). Goldstein proposed that at the heart of language ability was a single cognitive and cortical center. This center of word-concepts is responsible for "inner speech," a psychological phenomenon which is involved in both perception and production of speech. Although this "glossopsychic" field of word concepts (as this center was called) interacts with centers subserving auditory, motor speech and nonverbal thought, it is essentially independent from them all. In this model, the language disturbance associated with central aphasia is attributed to the damaged center of word concepts, not to a disconnection between two interacting language centers. Goldstein implicated the insula as well as adjacent regions of the temporal and parietal lobes as sites in which damage might cause this type of aphasia.

Data on the structural damage which causes conduction aphasia are relatively consistent in implicating the temporo-parietal area of the dominant hemisphere, and indicate that damage may involve the parietal operculum, the left auditory complex, the left insular region and the supra-marginal gyrus as well (Damasio and Damasio, 1980). Although CT data have allowed us to confirm and refute theories of localization with respect to other major aphasic syndromes (e.g., Hayward, Naeser and Zatz, 1977; Kertesz, Harlock and Coates, 1979; Basso, Lecours, Moraschini and Vanier, 1985), they are less helpful with conduction aphasia. The major neuroanatomical theories of conduction aphasia are consistent with structural damage in the temporo-parietal area of the dominant hemisphere and cannot be distinguished on this basis alone.

Are there data which may help us decide between competing theories of conduction aphasia? We think physiological data may yield precisely the information we need. Studies of brain function (e.g., cerebral blood flow and glucose metabolism using Positron Emission Tomography), allow us to see how areas of the brain in, around, and distant from the lesion site respond to structural damage (Hanson, Metter, Riege, Kuhl and Phelps, 1983; Metter, Hanson, Riege, Jackson, Mazziotta and Phelps, 1985). These procedures reveal systems within the brain by demonstrating that damage in one region creates functional deficits in other, related regions. In this way, areas of brain which normally work together are highlighted when damage occurs in one part of the functional network (Metter, Mazziotta, Itabashi, Mankovich, Phelps, and Kuhl, 1985). At times, we can infer functional disconnections from such patterns. For example, in about half of aphasic patients studied by Metter et al. (1986), left hemisphere lesions produced contralateral cerebellar abnormalities. These findings can be interpreted as a disconnection between motor tracts involving both the left cortical hemisphere and the right cerebellum.

The models of conduction aphasia presented above yield empirical predictions for brain function. If conduction aphasia is truly a disconnection syndrome, we should be able to observe distant effects of local structural damage. Specifically, post-Rolandic structural damage should create metabolic abnormalities in Broca's area. On the other hand, a Goldsteinian model does not necessarily predict any effect outside the lesion site. In this paper, we present data from studies of brain function as measured by glucose metabolism which test these models of conduction aphasia.

METHODS

As part of a larger study on brain function in aphasia, unselected patients with radiological evidence of single (left hemisphere) events and aphasia were studied with behavioral and brain imaging techniques. All patients were right handed. All subjects were given the Western Aphasia Battery (Kertesz, 1982), The Porch Index of Communicative Ability (Porch, 1971), non-Contrast X-ray CT scan and resting state F-18 FDG PET scan.

Subjects. Six patients diagnosed as presenting symptoms consistent with conduction aphasia were studied individually. Subject information is presented in Table 1.

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 Table 1. Subject information including (1) sex, (2) age at time of testing, (3) months post onset (MPO) at time of testing, (4) etiology, (5) fluency, (6) repetition, and (7) comprehension ratings (out of 10) from the Western Aphasia Battery, (8) Western Aphasia Battery Aphasia Quotient, and (9) PICA overall percentile.

	SEX	AGE	MPO	ETIOL	FLU	REP	COMP	AQ	PICAX
1.	M	62	2	CVA	7	6.6	8.4	62.2	33%ile
2.	F	67	10	CVA	8	6.7	9.2	84.5	65%ile
3.	F	37	79	hem	6	6.7	8.4	76.1	66%ile
4.	M	55	35	CVA	9	6.6	9.4	84.5	65%ile
5.	M	60	6	CVA	8	6.1	7.5	74.1	61%ile
6.	M	70	7	CVA	8	5.9	7.1	58.3	48%ile
Means:		58.5	23.2		7.6	6.4	8.3	73.3	56%ile

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Imaging Procedures and Analysis

X-ray CT. Twenty-one cerebral regions of interest were mapped on non-contrast CT scans and were rated using a 5-point scale (0 = normal, 1 = atrophy, 2 = structural damage with no tissue loss, 3 = structural damage with partial tissue loss, and 4 = structural damage with complete tissue loss) by a neuroradiologist who was naive to the project and by a neurologist working on the project (EJM). Regional scores showed a 90% agreement, and the two ratings were averaged.

PET. Patients were studied on the NeuroECAT 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 [¹⁸F]fluorodeoxyglucose (FDG) injected intravenously. After 40 minutes, scanning was initiated. For the present analysis, each of 32 cerebral regions was outlined on a video monitor using an interactive program, and regional metabolic rates for glucose (LCMRGlc) were derived. Due to the small sample size and individual variations, left/right ratios over all brain regions were calculated. Ratios of less than .90 were considered abnormal, when compared with 22 normal controls.

Language Analysis

All patients received standard administrations of the Western Aphasia Battery (Kertesz, 1982) and the Porch Index of Communicative Ability (Porch, 1971). Aphasia diagnoses were made on basis of the Western Aphasia Battery results.

RESULTS

Structural damage. Our results are consistent with other reports in the literature which indicate damage in the temporo-parietal areas (see Figure 1). Considering those regions with an average rating of 1.5 or greater (on the 5-point scale) as indicating structural damage, the lesions were confined to the temporal and parietal lobes, with three subjects having insular damage as well. Notably, there is no damage observed in the frontal lobes, confirming a post-Rolandic structural locus for conduction aphasia.

Metabolic effects. As a group, the patients showed left/right metabolic asymmetry in (1) the same regions where there was evidence of structural damage and (2) in Broca's area (divided in our regional analyses into two measures: B1 and B2). The greatest asymmetry was in temporo-parietal regions, consistent with the site of structural damage. As a group, we saw mild distant but localized effects in Broca's area (see Figure 1).

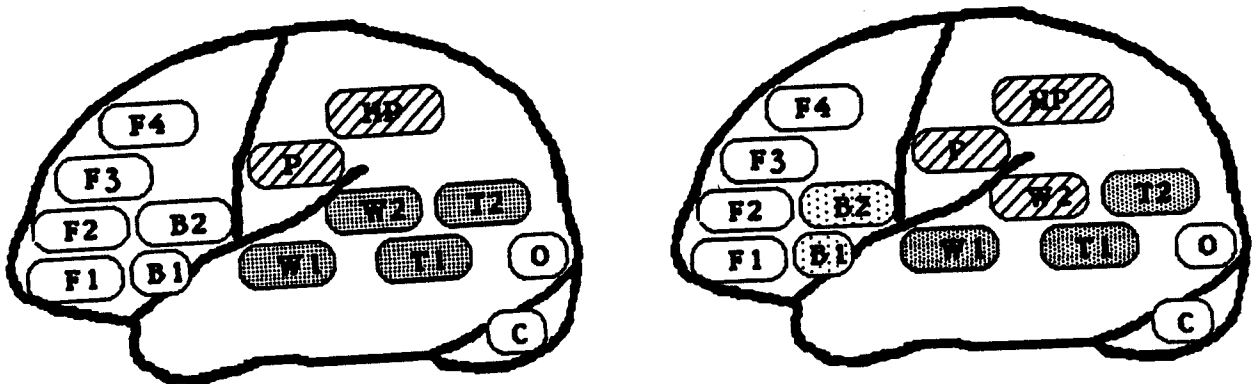


Figure 1. Cortical regions which demonstrated structural damage on X-ray CT (left) and left/right metabolic asymmetries (right) for six patients with conduction aphasia. Severe structural damage (rating > 2) was observed in Wernicke's and Temporal regions, while milder damage (rating 1.5 - 2) was noted in the Parietal measures. Severe metabolic asymmetry ($< .60$) was observed in W1, T1, T2; moderate asymmetry (.61 - .79) was seen in P, HP and W2; mild asymmetry (.80 - .90) was observed in B1 and B2.

Individual variability in CT and PET. It is notable, however, that this group of patients is not homogeneous. Their CT and PET scores showed striking individual differences. Structurally, the lesions are all similar in their post-Rolandic location. However, three patients' lesions extend into the insula. Metabolically, distinct subgroups also emerge. In particular, only a subset of the subjects contribute to the observed changes in Broca's region. Three patients demonstrated metabolic abnormalities in both Broca regions, three did not.

DISCUSSION

We can assume that (1) remote metabolic effects in Broca's area would support a disconnection hypothesis and (2) the absence of remote frontal effects would support a non-disconnection or "central" deficit notion. Neither hypothesis is clearly supported by the metabolic data presented here, although the data can be interpreted to support either. First, there is a common element of structural and metabolic abnormality in the temporal-parietal regions. This is consistent with a central deficit assuming that the relevant central processor is localized to those regions. With this interpretation, the metabolic effects observed in Broca's area in a subset of the patients could be attributed to additional damage independent of the clinical syndrome. Second, the data can be used to support both hypotheses: some patients with conduction aphasia present evidence of disconnection (those with metabolic asymmetry in Broca's area) and others present evidence of a central deficit (those with no frontal changes in metabolism). Subgroups within conduction aphasia have been proposed by other researchers (e.g., Kertesz et al., 1977), and would not be altogether surprising.

Preliminary analysis of the metabolic data does support the notion of (at least two) subgroups in this population. If these two subgroups are truly distinct, we should be able to correlate X-ray CT and behavioral differences with observed metabolic differences. Investigation of individual patient data reveals the following two subgroups.

One group contained three patients who were relatively nonfluent with moderate to severe word-finding deficits and few or no paraphasias as demonstrated in the following excerpt from a narrative description of the Cookie Theft picture (Goodglass and Kaplan, 1972):

First, there's a cookie jar.
A boy takes the, the cookie.
On top of st- - - air, stool, fall.
A girl but, um, by....a woman, dry, dish.
Water come over, um, the cup....tree.
Um, window.

In severity, this group is moderate (AQ 76-85; PICA overall percentiles between 65 and 72) and their spontaneous speech was most similar to mild anterior (Broca's) aphasic patients. This group presented significant insular damage on X-ray CT, and showed substantial metabolic asymmetry in Broca's area on FDG PET. We refer to this subgroup as "nonfluent" (see Figure 2) and appears to be similar to what Kertesz (1979) calls "efferent conduction aphasia."

The other group contained three patients who had paraphasic, often neologistic, fluent speech as seen in the following excerpt from a Cookie Theft narrative:

....Well, the lady, the lady, she is playing' the w-wash, washer, wash window, wash, not wash window, chap, pray, parade, play plate plate plate plate. He's takin' a plate. And uh, she's washin' a balus, baluser, how, uh, socks, ho- army, not army, but pair, cow, koos, cows, cap, uh, she using a a a string up a string and uh a opter, and uh d-drum, ice, dee grice, the on the- the water is in the option, in the closed, she's in the water, poster, poster, embryo....

These patients presented a more severe aphasia than the nonfluent group (AQ < 75; PICA overall percentile under 61) and their spontaneous speech was similar to patients with Wernicke's aphasia. They demonstrated marked posterior structural damage on X-ray CT and only mild metabolic asymmetry in one of the Broca regions. We refer to this subgroup as "fluent" (see Figure 3) and they appear to be similar to Kertesz's (1979) classification of "afferent conduction aphasia."

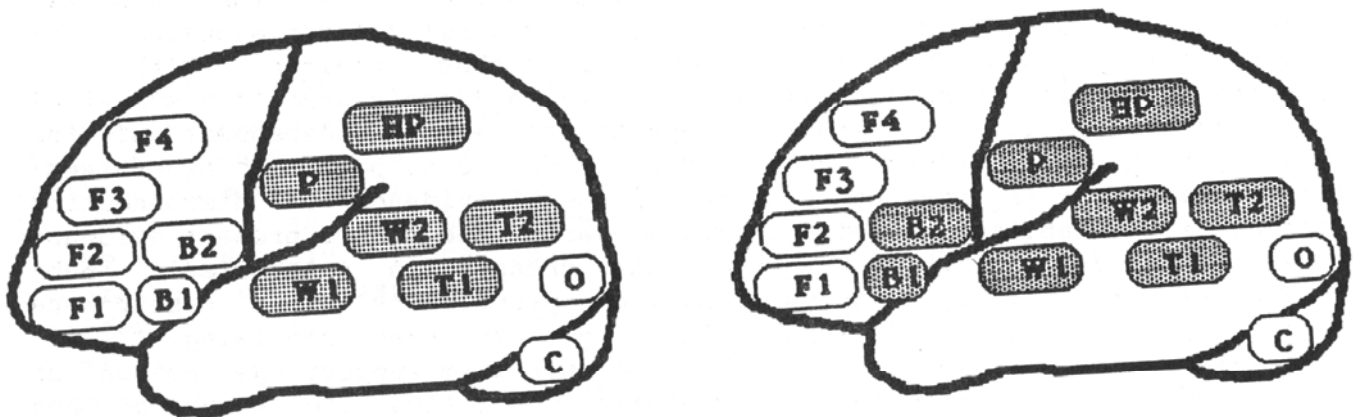


Figure 2. Cortical regions which demonstrated structural damage on X-ray CT (left) and left/right metabolic asymmetry on FDG PET (right) for nonfluent patients with conduction aphasia (N=3). Ratings of structural damage for regions shown were > 2; measures of metabolic asymmetry for regions shown were between .30 and .79

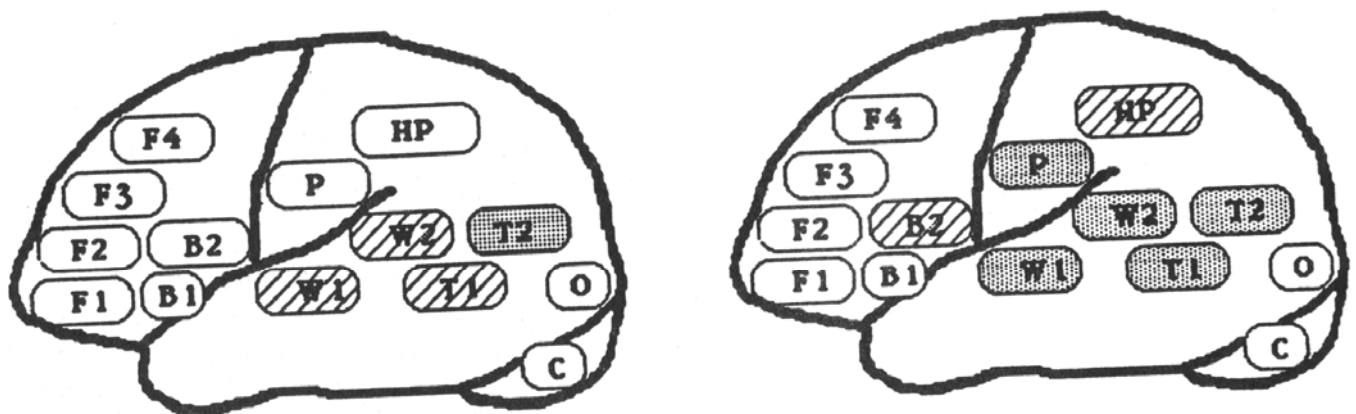


Figure 3. Cortical regions which demonstrated structural damage on X-ray CT (left) and left/right metabolic asymmetry on FDG PET (right) for fluent patients with conduction aphasia (N=3). Structural damage rated > 2 (severe) is dotted; damage rated 1.5 - 2 (mild) is striped. Metabolic asymmetry under .80 is dotted; milder asymmetry (between .80 and .90) is striped.

On the one hand, the simplest explanation for the different subgroups may be to relegate the two observed patterns to subsyndromes: Conduction C - a variety caused by a central disorder and Conduction D - a variety caused by disconnection. Although we have shown that patient subgroups can be identified on X-ray CT (with and without insular damage), FDG PET (with and without substantial asymmetry in Broca's area), and behavior (severity, fluency, paraphasias), it is not necessarily the case that two subgroups correspond to the two traditional explanations of conduction aphasia. While

the both-theories-are-right solution to the variety observed within conduction aphasia may be appealing, it has its drawbacks as well.

Although assigning the individual patients with conduction aphasia to subsyndromes has the benefit of capturing the similarity between them (in so far as they all have disproportionate difficulty with repetition and they all have "conduction" in their diagnosis), there are two difficulties. First, this classification system or typology attributes similar symptoms (i.e., disproportionate difficulty with repetition) to two distinct neurological mechanisms (disconnection and central deficit). This is not parsimonious. Second, patients with conduction aphasia have behavioral similarities with patients exhibiting other syndromes but this similarity is ignored by the "Conduction C & D" classification system. Recall that one subgroup is relatively less fluent (some individuals actually agrammatic) and on X-ray CT have slightly more anterior and insular damage. These characteristics are similar to patients with Broca's aphasia. The other group has fluent paraphasic speech output and on X-ray CT demonstrates posterior temporal damage and no evidence of insular damage. These patients resemble patients with Wernicke's aphasia. In short, then, the subgroup approach to conduction aphasia proposes distinct neurological mechanisms for patients within a single syndrome, and glosses over behavioral and neurological similarities between this and other aphasic syndromes.

An explanation of conduction aphasia suggested by Levine and Calvanio (1982) may offer us an appealing alternative. Their formulation assumes that language function is mediated by auditory and sensorimotor centers, but that these centers are tightly integrated, and in fact are functionally overlapping. The neurological aspect of this model, likewise, assumes two partially overlapping cortical centers within the left hemisphere. The poles of this system correspond to classical auditory and motor centers, with the intermediate cortex subserving both auditory and motor functions. This "overlap" cortex is neurologically more variegated than the classical centers dedicated to auditory perception and motor speech. It includes neurons which are tightly correlated with speech production, neurons which are tightly correlated with auditory perception, neurons which are equally associated with perception and production, etc. This area is particularly important for behavior (such as repetition) which integrates perception and production. Within this model, conduction aphasia results from damage to the intermediate perisylvian cortex, distinct from that associated strictly with perception (Wernicke's area) or production (Broca's area) but which is correlated with both perception and production. This theory contrasts with the Wernicke-Geschwind approach in that the cortex involved is not merely connective fiber tracts between the two centers. This approach also contrasts with Goldstein's model since the relevant cortex is not a third independent center, but consists of features common to both auditory perception and speech production centers. Such a theory, sketched only in outline form here, has several benefits. First, it is consistent with X-ray CT data which suggests that conduction aphasia arises from structural lesions which often lie between Wernicke's and Broca's areas (Kertesz, 1979). Second, it predicts variation in behavior within conduction aphasia, such that some lesions may compromise motor output and produce Broca-like symptoms while other lesions may involve perceptual abilities and produce Wernicke-like symptoms. This theory can also account for the differences observed in glucose metabolism--certain lesions are likely to interfere with cortex which is more intimately linked to Broca's area, while some lesions will not.

SUMMARY AND CONCLUSIONS

In conclusion, conduction aphasia represents a syndrome which is unified by a picture of relatively poor repetition of spoken language alongside relatively good comprehension and fluent speech. The common structural (X-ray CT) and functional (FDG PET) features of these patients appears to be temporo-parietal involvement. This common structural and metabolic abnormality may account for the symptoms shared by all six subjects. However, a closer investigation of behavioral, anatomical and physiological profiles indicates heterogeneity within this group of patients. In particular two subgroups of patients emerge: one subgroup is relatively nonfluent and demonstrates metabolic asymmetry in Broca's area; another subgroup is characterized by fluent but paraphasic speech and shows little metabolic abnormality in Broca's area. Rather than proposing two distinct subgroups of conduction aphasia (one due to a disconnection syndrome and one not), we support a model in which conduction aphasia is the result of damage to perisylvian cortex and is not primarily a disconnection syndrome. This area of perisylvian cortex contains neurons strongly linked to both perceptual and motor areas of cortex. Variation of function within this cortex and variation of lesion location helps to explain the variety of phenomena observed in conduction aphasia.

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DISCUSSION

- Q: What linguistic bases can you enlist to account for fluency or non-fluency?
- A: I actually don't think of fluency and nonfluency as linguistic. I think of nonfluency as coming from a difficulty in initiation, which I don't think of as a linguistic issue.
- Q: Does it have anything to do with the way these people process language? Does fluency have anything to do with the output of language, either as the selection of the unit of language, or is it better accounted for by motor constructs? If the latter is true, then I don't know why we're using it to describe aphasia.
- A: I hope that it involves both linguistic and motor, and that there are natural categories that we label "fluency" and "nonfluency," and that with each label a lot of things follow. It's not clear to me exactly what those things are. For instance, nonfluent aphasic people--are they

agrammatic or aren't they? Equally, with preserved fluency, it's not clear that you have preserved linguistic processing. I hope that we'll arrive at a classification where "fluency" and "nonfluency" will carry with them a whole lot of other attributes, both motor and linguistic.

Q: Did any subject sometimes repeat a sentence and sometimes not repeat that same sentence so well?

A: We did not do the same sentences twice on them. So I don't know.

Q: The question is then, how can a real disconnection account for that variability within a subject, if it's truly a disconnection syndrome?

A: I think the very fact that there is that kind of variability means that it is NOT a disconnection syndrome. With a disconnection syndrome you wouldn't expect qualitative differences in fluency. I hope I didn't lead you to believe that I was proposing that the disconnection theory was right.

Q: How can you relate behavior back to a resting state?

A: We presume that the resting state has something to do with brain function. The next step, of course, is to find out what those areas which are metabolically abnormal during a resting state, do, say, when the patient is repeating. My presumption at this point is that those areas which are metabolically abnormal in a resting state will mean something for the patients when they are actually using language. That the resting state means something (in relation to behavior) is an assumption that we'll have to prove.

Q: Have you seen any non conduction aphasic people with the patterns of metabolic disturbance that you've discussed for these conduction aphasic people?

A: Similar. What Metter showed really was that there are a lot of patients with posterior damage that show Broca and prefrontal hypometabolism. Now, that could be interpreted as a disconnection syndrome between posterior and frontal areas. In which case, most of our patients demonstrate a "disconnection" syndrome of some sort.

C: If you have seen ANY non conduction aphasic person with a similar pattern of metabolic disturbance, then it becomes hard to argue that the metabolic disturbance explains the conduction aphasia.

A: I disagree with that. Let's look at the structural data. We can see the same lesion in different people causing all sorts of different kinds of aphasia. What the metabolic data are doing is adding information as to what kinds of effects that focal structural damage have elsewhere. Whenever we look at structural damage and say, "Ah!, this is associated with such-and-such a behavior," we're looking at a correlation. What we're trying to do is take that correlation a step further by looking at what kinds of effects that structural damage has elsewhere in the brain. We're not saying that the patterns that we're seeing are specific to a specific kind of aphasia. We don't have that knowledge right now. We've seen the same patterns of metabolic changes that we see in conduction aphasia in other kinds of aphasia. We've seen them in some with anomic aphasia.

- Q: Have you addressed the reliability issue? Both of your physiological CT/PET measures, and of your behavioral observations. Have you obtained reliability on those observations?
- A: Yes and no. Certainly on the CT data, we have several people rate the CT scans. At this point Metter is the only one who has quantified the metabolic data. As for fluency, Cathy Jackson and I always do that together and we discuss it. This is NOT blind reliability certainly, but then neither of us pretend to know the answer by ourselves.
- C: You started with a patient group who were identified because they failed to repeat, and you wound up characterizing them in terms of their ability to comprehend and their fluency, and then describing the lesion sites as a function of whether they could comprehend and whether they were fluent. So the lesion-deficit correlations that you presented at the end no longer had anything to do with the repetition.
- A: I don't see it that way. They certainly differed on comprehension and fluency, but what they all had in common was a disproportionate difficulty with repetition.
- C: Then this variation in PET locations is irrelevant to the question of repetition. And repetition you are taking to be related to that central area of abnormality.
- A: Right.
- C: If that's the analysis then you do have to explain why there are patients with lesions in that area who do not have a repetition deficit, as I understand your data show. If the claim is that that area is critically involved in repetition, then the answer, it seems to me, is there are many reasons why people have difficulty repeating. That is, some will have difficulty repeating because they have what Shallice and Warrington call "short term memory deficit." Others will have difficulty repeating because of the sort of classical disconnection Kinsbourne described. Classically, a conduction aphasic is someone who recognizes a word, but can't pass the sound of that word on to the motor programming system. We don't know whether these people recognize words as words. A conduction aphasic in the classical sense is someone who can do an auditory lexical decision task and who has more trouble repeating single words than nonwords compared with normals. There may be two dozen other reasons why a patient might not repeat, all of which would be interesting. It seems to me one should start from those kinds of things if what one is interested in is the neural basis for individual functions, rather than the neuropathological correlates of symptoms.
- A: I agree.

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C: If you have seen ANY non conduction aphasic person with a similar pattern of metabolic disturbance, then it becomes hard to argue that the metabolic disturbance explains the conduction aphasia.

A: I disagree with that. Let's look at the structural data. We can see the same lesion in different people causing all sorts of different kinds of aphasia. What the metabolic data are doing is adding information as to what kinds of effects that focal structural damage have elsewhere. Whenever we look at structural damage and say, "Ah!, this is associated with such-and-such a behavior," we're looking at a correlation. What we're trying to do is take that correlation a step further by looking at what kinds of effects that structural damage has elsewhere in the brain. We're not saying that the patterns that we're seeing are specific to a specific kind of aphasia. We don't have that knowledge right now. We've seen the same patterns of metabolic changes that we see in conduction aphasia in other kinds of aphasia. We've seen them in some with anomic aphasia.

- Q: Have you addressed the reliability issue? Both of your physiological CT/PET measures, and of your behavioral observations. Have you obtained reliability on those observations?
- A: Yes and no. Certainly on the CT data, we have several people rate the CT scans. At this point Metter is the only one who has quantified the metabolic data. As for fluency, Cathy Jackson and I always do that together and we discuss it. This is NOT blind reliability certainly, but then neither of us pretend to know the answer by ourselves.
- C: You started with a patient group who were identified because they failed to repeat, and you wound up characterizing them in terms of their ability to comprehend and their fluency, and then describing the lesion sites as a function of whether they could comprehend and whether they were fluent. So the lesion-deficit correlations that you presented at the end no longer had anything to do with the repetition.
- A: I don't see it that way. They certainly differed on comprehension and fluency, but what they all had in common was a disproportionate difficulty with repetition.
- C: Then this variation in PET locations is irrelevant to the question of repetition. And repetition you are taking to be related to that central area of abnormality.
- A: Right.
- C: If that's the analysis then you do have to explain why there are patients with lesions in that area who do not have a repetition deficit, as I understand your data show. If the claim is that that area is critically involved in repetition, then the answer, it seems to me, is there are many reasons why people have difficulty repeating. That is, some will have difficulty repeating because they have what Shallice and Warrington call "short term memory deficit." Others will have difficulty repeating because of the sort of classical disconnection Kinsbourne described. Classically, a conduction aphasic is someone who recognizes a word, but can't pass the sound of that word on to the motor programming system. We don't know whether these people recognize words as words. A conduction aphasic in the classical sense is someone who can do an auditory lexical decision task and who has more trouble repeating single words than nonwords compared with normals. There may be two dozen other reasons why a patient might not repeat, all of which would be interesting. It seems to me one should start from those kinds of things if what one is interested in is the neural basis for individual functions, rather than the neuropathological correlates of symptoms.
- A: I agree.