

Language Disorder Associated with Alzheimer's Dementia, Left Hemisphere Stroke, and Progressive Illness of Uncertain Etiology

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The prevalence and character of the language disorder in dementia of the Alzheimer type (DAT) is not completely understood. Cummings *et al.* (1985) reported that aphasia is "...consistent manifestation of DAT..." (p.396). In contrast, Mayeux *et al.* (1984) found that only 5% of a series of patients with DAT showed language changes as a first symptom. Other recent literature suggests that a slowly progressing language disorder may exist in isolation, with intellectual, behavioral, and affective signs of dementia appearing later (Mesulam, 1982; Morris *et al.*, 1984; Wechsler, 1977).

STATEMENT OF THE PROBLEM

These differing reports bring to mind two questions raised by Wertz (1982).

First, are the language deficits seen in demented patients described best as aphasia? And, second, if they are not, can one differentiate the language deficits seen in dementia from those present in aphasia? (p.350)

The term "aphasia" for diverse neurologic entities, Wertz suggests, may not be very useful because prognostic and management decisions are likely to be different. His second question, however, raises a greater concern for me -- diagnostic certainty. Can (or how can) speech and language pathologists tell the difference among these language disorders?

The purpose of this paper is to compare the language performance of three types of patients: presumed dementia of the Alzheimer type (DAT), left hemisphere stroke (LHS), and progressive language disorder (PLD) in the absence of definitive neurologic diagnosis. Specific questions were: 1) Do these three groups differ in terms of the aphasia quotient (AQ), reading quotient (RQ), or writing quotient (WQ) or any subtests comprising these summary scores obtained from the Western Aphasia Battery? 2) Do these three groups differ in terms of speech fluency, phonologic integrity, or type of aphasia, as determined using the WAB criteria for classification? 3) Will language performance discriminate among the three diagnostic groups?

METHOD

Subject Characteristics. Subject characteristics are summarized in Table 1. Group 1 was comprised of 10 patients with dementia of the Alzheimer type. Each diagnosis was made by a neurologist using commonly accepted stringent criteria, both inclusionary and exclusionary. A prominent feature of all patients was recent memory deficit affecting activities of daily living. Group 2 was comprised of 10 patients selected from our files who had sustained a single left hemisphere stroke and had no history of speech, language, neurologic, or psychiatric problems. LHS Ss were matched to DAT Ss by age and sex. Group 3 was comprised of five patients who presented to the neurologist with progressive speech and language difficulty as the chief complaint. Some measurable memory impairment was present in all patients, but neither as the first nor the prominent feature. DAT was being considered by the neurologist as a possible diagnosis but the atypical presentation precluded a definitive diagnosis pending further studies and longterm followup.

Table 1. Characteristics of three subject groups: Dementia of the Alzheimer type (DAT), Left hemisphere stroke (LHS), and Progressive language disorder (PLD).

Group	Sex	Handedness	Age	Education	Months post onset
DAT (N = 10)	4 f 6 m	all right	61.5 (S.D. 6.7)	13.5 (S.D. 2.9)	34.3 (S.D. 17.8)
LHS (N = 10)	4 f 6 m	all right	63.7 (S.D. 8.6)	12.5 (S.D. 2.8)	8.6 (S.D. 14.3)
PLD (N = 5)	2 f 3 m	4, right; 1, ambi- dextrous	62.8 (S.D. 8.1)	13.2 (S.D. 2.9)	28.4 (S.D. 13.4)

Note. N.S. differences except MPO: LHS significantly lower MPO than DAT & PLD.

Evaluation. Physical findings and head CT scan information are summarized in Table 2. DAT and PLD Ss are clearly distinguished from the LHS Ss by the absence of physical deficits. CT scans were normal or diffusely and bilaterally abnormal in DAT Ss. CT scans on LHS Ss, when obtained, confirmed ischemic or hemorrhagic focal lesions. Three of five Ss had normal scans, and two of five had focal atrophy in the left perisylvian region posteriorly. All Ss were evaluated with the Western Aphasia Battery (WAB) by an experienced speech and language pathologist.

Table 2. Signs of physical weakness and head CT findings in three groups of subjects.

Group	PHYSICAL WEAKNESS		Missing	CT FINDINGS		
	No indication	Indication		Normal	Abnormal Diffuse	Abnormal Focal
DAT (N=10)	10	0	0	4	6	0
LHS (N=10)	1	9	4	0	0	6 ^a
PLD (N=5)	5	0	0	3	0	2 ^b

^aLeft hemisphere ischemia or hemorrhage

^bLeft perisylvian dilation suggesting lobar atrophy

RESULTS

The first question was: Do these three groups differ in terms of the aphasia quotient, reading quotient or writing quotient or any subtests comprising these summary scores?

The first procedure involved Pearson Product Moment Correlations for all linear variables. (See Table 3 for a list of the WAB tests.) For all three groups, language summary scores (AQ, RQ, WQ) were positively correlated at $p < .05$, as were all the scores comprising them. Correlations of language scores with nonlanguage variables (age, education, and MPO) were not significant. (Lack of apparent linear relationship precluded use of any non-language variable as a covariate.)

The second procedure involved an analysis of variance. This analysis revealed no significant differences among the three groups for the three language summary scores. Subsequent one-way ANOVA's among all the subtest scores also showed no significant differences. Table 4 shows that the AQ was least impaired and the WQ most impaired in all groups.

Table 3. Western Aphasia Battery subtests (Kertesz, 1982).

Aphasia Quotient	Reading Quotient	Writing Quotient
Speech content	Paragraphs	Name & address
Speech fluency	Sentences	Spontaneous writing
Comprehension	Oral	Dictated sentence
Yes-no question	Comprehension	Dictated words
Auditory word	Words	Alphabet
Sequential commands	Letters	Numbers
Repetition	Recognize orally	Dictated letters
Naming	spelled words	Dictated numbers
Objects	Spell words orally	Copied Sentence
Word fluency		
Sentence completion		
Responsive speech		

Table 4. Western Aphasia Battery summary scores for three groups of patients.^a

Group	Aphasia Quotient		Reading Quotient		Writing Quotient	
	Mean	(S.D.)	Mean	(S.D.)	Mean	(S.D.)
DAT (N=10)	75.0	(23.1)	62.9	(32.3)	58.1	(35.9)
LHS (N=10)	67.5	(20.7)	57.9	(32.7)	46.1	(32.5)
PLD (N=5)	56.9	(21.4)	43.1	(29.7)	38.5	(32.3)

^a3 x 3 ANOVA revealed N.S. differences

The second question was: Do these three groups differ in terms of speech fluency, presence of speech sound selection or sequencing errors, or types of aphasia as defined by the WAB?

Table 5 summarizes the speech fluency characteristics of the Ss using a 3-point scale, which is described in the legend. This summary table shows that most DAT Ss were fluent or showed reduced speech fluency presumed to be related to anomic hesitations and pauses; one S (the most severe) was nonfluent. Four LHS Ss were fluent, four showed reduced speech fluency, and two were nonfluent. Three of five PLS Ss showed reduced speech fluency related to anomic

hesitations, one (the mildest overall) had preserved fluency, and one (the most severe) was nonfluent.

Table 5. Speech fluency characteristics of groups of subjects.

Group	Fluent ^a	Reduced Fluency ^b	Nonfluent ^c
DAT (N=10)	6	3	1
LHS (N=10)	4	4	2
PLD (N=5)	1	3	1

^aFluent: normal; near normal; may have subtle hesitations related to anomia; or may be hyperfluent.

^bNoticeably reduced speech fluency primarily related to anomic hesitations, with melody, articulatory agility, grammatical repertoire intact.

^cNonfluent speech characterized by reduced phrase length, altered prosody and articulatory agility; often grammatical problems.

Table 6 summarizes the presence of phonologic errors occurring in spontaneous speech or on repetition tasks. The 2-point scale described in the legend does not account for the frequency or severity of such errors, but only the presence of such errors. Five of the 10 DAT Ss showed no phonemic errors; five Ss did. Most of the LHS Ss had phonologic errors (the least severe in this group did not), and the PLD Ss were roughly equally divided.

Table 6. Presence of phonologic errors in three groups of patients.

Group	Absent ^a	Present ^b
DAT (N=10)	5	5
LHS (N=10)	1	9
PLD (N=5)	2	3

^aNormal; no speech sound selection and/or sequencing errors.

^bSpeech sound selection and/or sequencing errors are present; such errors may be frequent or infrequent.

The next step involved an analysis of aphasia type using the WAB criteria for classification (Kertesz, 1982). The percentage of patients from each group fitting into an aphasia profile is shown in Table 7. The distribution of "fluent aphasia" versus "nonfluent aphasia" is roughly similar across the diagnostic groups. However, the majority of DAT Ss fell into the "anomic aphasia" classification and none into the "Broca's" profile. The PLD group, in contrast, showed a distribution across aphasia types comparable to the LHS group. Due to the small numbers involved in each sample, these distributions must be viewed with caution.

The third question was: Will language performance on the WAB discriminate among the three diagnostic groups?

We answered this question by a discriminant function analysis using the three language summary scores (AQ, RQ, WQ). The purpose of this analysis is to assign individuals to groups on the basis of their three scores, and to see if the predicted classification is similar to the true diagnosis. Table 8 shows the results of this analysis. Only 7 of 10 LHS Ss were correctly classified; 6 of 10 DAT Ss; and, 2 of 5 PLD Ss.

Table 7. Classification of three groups of subjects using the WAB.

	DAT (N=10)	LHS (N=10)	PLD (N=5)
Global	10%	0%	0%
Broca's	0%	20%	20%
Isolation	0%	0%	0%
Transcortical motor	0%	0%	0%
Wernicke's	10%	10%	40%
Transcortical sensory	0%	0%	0%
Conduction	20%	30%	20%
Anomic	60%	40%	20%
Nonfluent	10%	20%	20%
Fluent	90%	80%	80%

Table 8. Discriminant function analysis with three WAB summary scores.

		DAT	PREDICTED LHS	PLD
	DAT (N=10)	6	3	1
TRUE	LHS (N=10)	3	7	0
	PLD (N=5)	2	1	2

The results of the ANOVA statistic and the discriminant function analysis suggest that our 25 Ss are -- quantitatively at least -- very much alike in their language abilities.

DISCUSSION

In 1982, Appell, *et al.*, used the Western Aphasia Battery to study the language of 25 chronic Alzheimers dementia patients. They reported that auditory-verbal language was less impaired than reading or writing. Most of their subjects had fluent speech with impaired comprehension. Broca's and transcortical motor aphasias were absent. On these points, the present data agree. Our subjects differ from Appell *et al.*'s in that half of our DAT Ss showed some phonemic paraphasias, while none of Appell's did. Most of our DAT Ss showed anomic aphasia; their subjects showed transcortical sensory and Wernicke's aphasia. Morris *et al.* (1984) suggest that aphasia in DAT progresses from anomic to Wernicke's to transcortical sensory. Differences in stage of disease between Appell *et al.*'s subjects and those in this study may account for differences in aphasia type.

We agree with Bayles (1982) who found that the semantic system is more affected than either the syntactic or phonologic systems in DAT patients. Bayles *et al.* (1982), and Bayles *et al.* (1985) also highlight the disruption of pragmatic ability, as reflected in spoken discourse, as a unique feature in dementia. Though a refined analysis of spoken discourse is not included in the Western Aphasia Battery scoring system, we observed several features described by Bayles *et al.* (1985). These deviant features include repetition of ideas and use of stereotyped phrases. When reduced speech fluency is present, it appears to be related to anomia, false starts, sentence revisions, and intrusion of tangential, egocentric statements (Bayles *et al.*, 1982).

I would like to highlight two features that seem to distinguish DAT from LHS, which objectively derived scores on the WAB may tend to mask. 1) Most DAT subjects showed anomia as a prominent feature, with relatively less impairment of phonologic and syntactic systems. The LHS Ss, in contrast, showed problems across the board. 2) DAT Ss showed specific deficits in spoken discourse, while LHS Ss showed relatively preserved discourse.

The PLD subjects show a less clear pattern of language difficulty. Anomia is present, but this is not a differentiating feature. Both fluent and non-fluent aphasia types are represented in our small sample, including Broca's (1), Wernicke's (2), conduction (1), and anomic (1). The fluent PLD subjects looked quite a bit like our DAT subjects. The nonfluent PLD subjects looked quite a bit like our LHS subjects. However, none were complacent about their language disorders, and all showed relatively preserved discourse.

In 1977, Wechsler described a 67-year-old male who showed a slowly progressive aphasia as the first sign of dementia. Aphasia was characterized by semantic and phonemic paraphasias, paragrammatism, comprehension difficulty, alexia and agraphia. CT scan showed dilation of the left posterior Sylvian fissure, indicating focal atrophy. In 1982, Mesulam described six cases of slowly progressive aphasia without generalized dementia, five of whom presented as "anomic aphasic" in the early stage. Aphasia progressed five years or more before other signs of mental deterioration presented. In the five cases, CT scans showed enlarged perisylvian fissures on the left, suggesting lobar atrophy. In the cases reported by Wechsler (1977) and Mesulam (1982), the language disorder presented early and in virtual isolation from deficits of memory, behavior, or affect, so that these cases did not fit well into established diagnostic entities such as Alzheimer's disease (memory deficit early) or Pick's disease (behavior, personality changes early). The five cases of PLD described here presented a similar diagnostic dilemma. Aphasia was an early and prominent sign. The disorder was slowly progressive.

Gordon and Selnes (1984) also reported progressive aphasia in six patients. Through complete psychometric evaluations, they found that deficits, though subtle, were widespread. They caution us that these "atypical" focal cases presenting as progressively aphasic may in fact be indistinguishable from other dementias and may ultimately progress to a similar global involvement. Until followup evaluation or autopsies are available on these "atypical" cases, the exact nature of this dementing illness will remain unclear. It may be that there are several types of DAT. One type may be heralded by aphasia as an early and prominent feature (Morris *et al.*, 1984).

I would like now to return to the original questions. First, can we distinguish the language of DAT from the language following focal abrupt onset lesions? Within the limitations of the battery used and the small numbers of subjects in this study, the data suggest that the speech and language pathologist may have difficulty differentiating the language deficits seen in DAT patients from those seen in left hemisphere stroke patients. The language

disorders in our DAT subjects and our PLD subjects strongly resemble the language disorder in our LHS subjects.

Second, is the term "aphasia" an appropriate label for the language disorders seen in dementing illness? Cummings et al. (1984) believe that language alteration "...is a consistent manifestation of DAT and inclusion of aphasia as a diagnostic criterion will improve diagnostic accuracy in the clinical identification of DAT" (p. 396). Obler and Albert (1981) state: "...language disturbance is ALWAYS present in a more or less severe form" (p. 389).

One may argue that aphasia is aphasia only if the etiology is known (i.e., stroke and not dementia) as Darley (1982) suggests, or that the lesion is focal and not diffuse, and is abrupt in onset and not progressive, as Bayles et al. (1982) suggest. Such restrictions on the definition of "aphasia" are, I think, difficult to defend. I agree with Wilson et al. (1981) who said:

The reluctance to term (language disorder in dementia) (a)phasic may reflect confusion between psychological and anatomical concepts. This uncertainty should have no effect on how these disturbances are characterized from a psycholinguistic standpoint, however. (p. 10)

They further say:

Although the pattern of language deterioration in dementia differs from that seen in the classic aphasic syndromes, many of the specific abnormalities are indistinguishable... (p. 10)

In summary, the language of three types of patients was studied. Ten DAT, 10 LHS, and 5 PLD Ss of uncertain etiology (some or all of whom may have Alzheimers disease) were tsted with the WAB. The severity of language disorder and the relative impairment of aphasia, reading and writing scores were not significantly different. Subjects from each group showed reduced speech fluency, phonologic errors, and represented a variety of "aphasia types." Finally, language data alone were insufficient to correctly classify many subjects into their true diagnostic groups.

In conclusion, the language disorders of DAT, LHS, and PLD are difficult to distinguish by means of strictly quantitative analyses. Yet the experienced clinician will perceive qualitative differences, and these should not be ignored. In order for us to make accurate differential diagnoses based on language data alone, I believe it will be necessary for us to follow the lead of researchers such as Bayles and her colleagues, Obler and Albert (1981), and Shekim and LaPointe (1984), who have described unique features of spoken discourse in dementia patients. At the same time, because we do not want to disregard the similarities among these disorders, I believe that we would be wise to continue to use standard aphasia batteries in conjunction with supplementary approaches until we are sure we know what it is we are looking for.

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DISCUSSION

- Q: I don't think just because someone has a language disorder necessarily means that someone has aphasia. Otherwise we would be tempted to say that somebody who is mentally retarded, has schizophrenia, has autism or any other number of language deviations has aphasia. If you sit and talk to people who have dementia and if you sit and talk with people who have aphasia, do you sense that they both have the same disorder?
- A: I recognize the arguments on both sides, and I'm not particularly concerned as to whether we use the term aphasia. The point I am trying to make is that many of the positive features such as phonemic paraphasia, semantic paraphasia are very similar. Some of the descriptive work I have done in the past highlights what I think I know about the qualitative differences and there are many, but we don't have a standardized battery to measure those and distinguish those. This study sets up goals for the future. The standard aphasia battery that we have is not sensitive to the differences and we need to develop a test to help us objectify the differences. I like the term aphasia until I have something better to describe the ways in which the groups are similar.

- Q: If you use the Western Aphasia Battery as a vehicle, you are to some extent pouring a patient's behavior into that test format and into the numbers of that test. And then you come up with what the test sees. I think you are wise to say that we need some other investigative tool. I think that the science of linguistics, for example, provides a mechanism that makes no prior judgment as to the nature of the language one is looking at. The Western Aphasia Battery makes a prior assumption that we are looking for aphasia. If you are looking for aphasia, you will find aphasia. But I do think it's important what we use the term aphasia for and what we call aphasia if it's going to be a diagnostically meaningful term leading to a certain rehabilitative regimen. I think we should keep the term aphasia separate from the idea of the language behavior and language deviation.
- A: I think we should not ignore the ways in which disorders of the groups are similar. I interact with neurologists. We communicate about language disorders with the term aphasia. In my reports, I try to point out the ways in which I think this patient is different from the focal aphasias, but the neurologists in my center as well as those in the literature are using this term.
- Q: Would you want to regroup any of your progressive language disturbance patients into your Alzheimer's group?
- A: No. The fluents do tend to look more like the Alzheimer's patients, but the Alzheimer's are a pretty diverse group. Ultimately Alzheimers disease is a neurologic diagnosis and not solely a linguistic one.
- Q: Was Pick's Disease ever considered for any of your progressive language disturbances?
- A: Yes. Both Alzheimer's and Pick's were considerations. The behavioral personality changes that typically accompany Pick's Disease were not present, so in that respect they were atypical of Pick's. And the significant recent memory problem was not present, which made them atypical of Alzheimer's. But, indeed, both Alzheimer's and Pick's are considerations. Morris et al. (1984) talk about both of these and the difficulties in the diagnosis. The nonfluent subjects looked like focal aphasia, and they were not complacent about their disorder. I would say that two of the five patients in the PLD group are very questionable as to diagnosis.
- Q: How severe were your Alzheimer's patients?
- A: They were moderately advanced, as described by the neurologist. I do have IQ data but they were not included in this study.
- Q: Were they living by themselves? Did they require nursing care?
- A: No. None of the patients were in nursing home facilities at the time.
- Q: How about your progressive language disordered patients?
- A: None of them were either.
- Q: Were they functionally as severely impaired as the Alzheimer's?
- A: In terms of activities of daily living, no. They required some supervision, but they could carry through tasks, and grooming was fine in all cases.
- Q: I wonder if you share my concern about the way numbers get tossed around on the Western. I look at the mean score for the demented subjects--75

- with a standard deviation of 23.1. But then I see that all of them are classified into an aphasic group and none exceeded the cutoff.
- A: I don't observe the cutoff on the Western: I don't think that just because a patient gets 93.9 that they are not aphasic. All the patients had aphasia, though they may have exceeded the cutoff.
- Q: Kertesz says that 93.8 is not aphasic. But you classify these folks into the types in Table 9.
- Q: Do you have any feelings about whether the nonlanguage tests on the Western Aphasia Battery would differentiate these groups?
- A: No. I typically don't do the whole cortical quotient. Most of these patients do have full psychometric evaluations, so I don't replicate that. I think we could learn a lot by doing supplementary nonlanguage tasks.
- Q: In evaluation, is it true that the history is everything and that testing tends to make dissimilar conditions appear similar?
- A: I consciously did not emphasize the history in this study. We always take a history and the family interview is very, very important. My intent was to be as objective as possible in looking at the language data alone to see if any differences would be present. As you see, in Table 2 particularly, you just look at the physical signs and you see automatically some distinctions there. However, this does not tell me what the language is going to look like. So, by no means am I suggesting that we ignore the history--particularly if we are the entry point for that patient. We must take all the data into consideration.