Panel: Aphasia With and Without Adjectives
Introduction

Jim Aten
Veterans Administration Medical Center, Long Beach, California

Three questions seem appropriate to consider for our discussion of the topic "Aphasia: With and Without Adjectives." These are: 1. What do test results tell us about whether patients are similar or different in reading, writing, speaking and listening performances and do they merit sub-classification? 2. What do studies of auditory deficits tell us about modifying or not modifying aphasic disturbances? 3. What kinds or types of oral expressive problems do we see? Reading disturbances appear to exist in isolation, so some aphasiologists, Darley (1982) being one, concede that the term dyslexic or alexic can be justified to describe that distinct problem. Time does not permit analyzing writing problems, the fourth component of the typical aphasic language-symptom complex.

Darley (1982) lucidly discusses the problems with our current aphasia tests. His critique merits our attention and action. The specific question here is, Do the results from testing large numbers of aphasic patients support a unified view of the disorder called aphasia? I propose that the very points of criticism of tests outlined by Darley offers an explanation as to why large numbers of aphasic patients have not been found to be significantly different or 'subtyped' within groups. Our tests are, for the most part, poorly normed, lack precision in establishing "can" and "can't do" behaviors, tend to obscure processes of decoding by demanding encoded responses via modalities that are also impaired, contain artificial ceilings under the rubric of being homogeneous or expedient, fail to account for sequential responding deficits or generic problems such as attention and memory or perceptual deficits, and do not satisfactorily rate severity in either a linguistic or a functional sense. These multitudinous problems operate to mask differences between patients even though severity may be controlled. Few have done more extensive testing of aphasic patients or held a stronger view than Hildred Schuell in arguing for aphasia to be classified as a unitary disorder. Yet Schuell, as we all know, had five subgroups with their descriptive, 'modified' or subclassified headings. These groups performed differently on testing and recovered at differing rates and to differing degrees. A thorough recent study by Hanson and colleagues (1982) factor analyzed Porch Index of Communicative Abilities (Porch, 1967) test results from a large number of aphasic patients and stated, "...differences in language impairment may exist between patients with the same level of deficit." (P. 367). They further stated, "A system of classification based on overall severity alone ignores the patterns of impairment that are differentiated by the presence or absence of specific deficits and may result in a considerable loss of information." (P. 367). This is not to say that their data support the "sparing" of some language dimensions, as speculated but not systematically measured by some in all too many of our current classification systems with loose use of modifiers. Hanson et al. (1982) reported that differences in degree of impairment of one or another language dimensions was clearly noted by their five predictive clusters or factors. One example cited was Category 3, involving poor speech but good gestures, and they comment that such a patient may well be a candidate for Amerind (and I would add Visual Action Therapy). So, let us develop tests that improve our data base, lest we exacerbate the condition called

'premature hardening of the categories' or lose valuable information that meaningfully describes differing performances requiring differing treatments.

Question Two concerns how patients listen. Darley (1982) states on page 43 "...studies have confirmed the fact that auditory comprehension is comparably impaired in all 'types' of aphasia." A major work by Orgass and Poeck (1966) was cited as partial proof. Orgass and Poeck analyzed Token Test results on a very small number of patients. They found no differences between groups as measured by the Token Test. A similar study, using more subjects (Poeck, Kerschensteiner, Hartje, 1972) administered the Token Test and found nonfluent aphasic persons not to be superior to fluent aphasic persons overall, or on any subtest. They did qualify their findings, however, in stating,

> "Obviously, the behavioral response called for by the Token Test is made up of a sequence of processes between the decoding of the instruction and the carrying out of the required action. It might well be that these processes are disturbed differentially by anterior and posterior lesions." (P. 303)

Shewan and Canter (1971) stated that Wernicke-type patients had much difficulty with syntactic complexity and in abstracting meaning from complex grammatic structures. They stated further that there was evidence in verbal output for both quantitative and qualitative differences, but that receptive language deficits seemed to differentiate groups only quantitatively. It is these quantitative differences that just may be of clinical significance to us despite the lack of qualitative differences. Baker and Goodglass (1979) reported that Wernicke patients needed over three times as long (650 msec compared to 200 msec) to recognize names of objects compared to Broca patients in a study of reaction times. Further, the Wernicke patients were the only group not to benefit from repetition of the stimuli. Do these differences in performances between groups of patients merit a label or modifier? Well, that's what we are discussing.

Grober et al. (1980) found that posterior-lesioned aphasic patients had disruption of underlying semantic structure (concepts) whereas anterior-lesioned patients had difficulties with more peripheral retrieval mechanisms. Perhaps this finding explains in part why some patients talk differently than others and might respond to different cueing or facilitating strategies in treatment. So we must ask, Would a label help us achieve that distinction and that end? LaFranchi, Aten, and Brick (1982) found semantic and phonological differences between anterior- and posterior-lesioned patients when semantic differences were controlled. The posterior-lesioned patients were significantly different in perceiving phonemically sequenced words, words that they could identify more accurately when phonemic discrimination and retention was eliminated from the task. These patients also produced different patterns of expressive deficits and consequently were classified by the Boston Test (Goodglass and Kaplan, 1972) as revealing distinctly different profiles. This latter finding of listening differences leads to the final question for consideration, namely do aphasic patients speak in ways that are meaningfully different?

The fluency/nonfluency dimension needs little confirmation, and Kerschensteiner, Poeck, and Brunner's (1972) detailed mathematical analyses confirmed two distinct groups, corresponding to the clinical syndromes of fluent and nonfluent.

Let us turn to a type of patient recognized by some as having a problem existing either with aphasia or as distinct from the central language impairment--the patient with speech behavior labeled apraxia of speech (excluding dysarthria, but subsuming a myriad of other articulatory deficits). One of the more stimulating discussions of the topic of apraxia is found in Kelso and Tuller (1981). A quote may set the tenor of the comments that follow:

> "It is an interesting but perhaps distressing feature of science that different areas of study, each bearing a strong potential relationship to the other, can function independently, each in its own oblivion...such a situation appears to exist between those who would seek to understand the motor functions of the central nervous system via investigations of clinical disorders and those who seek to understand the underlying behavioral process involved in the acquisition of skill and the control of movement in normal human populations." (Pp. 224-225)

Kelso and Tuller discuss the need for viewing heterarchial styles of organization constrained by context as opposed to traditional hierarchical views and models of the CNS and its function. They suggest that apraxia of speech may be characterized by a disruption of the normally invariant timing relations among articulators. Some proof for this may be found in the work of Itoh, Sasanuma, and Ushijima (1977), Freeman, Sands, and Harris (1978), and Kent and Rosenbek (1982). These attempts to objectively define articulation errors that comprise apraxia of speech are noteworthy, and, in my opinion, should be applied to those phonetic/phonemic errors that occur in posterior-lesioned patients who have more quantitative difficulty in comprehension, speak fluently and without struggle, usually fail to monitor their errors and to self-correct, and, according to Burns and Canter (1977), Trost and Canter (1974), and Deutsch (1983) make more errors in word-final positions and on polysyllabic sequences when contrasted with apraxia of speech patients. I refer, of course, to patients who have been labeled Wernicke types and who produce speech labeled as literal paraphasic. Darley (1982) states that the motor programmer may be separately disturbed (and I believe him), but I do not agree that the motor programmer is responsible for all articulation errors in the aphasic patient. Phonetic, phonemic, morphologic, and semantic units must be selected for inclusion in the program and all of this must occur before execution or production takes place. If studies such as the classic and frequently quoted Johns and Darley (1970) work had more carefully selected and described patients with only anterior lesions, who more consistently self-monitored and self-corrected and who had homogeneous ability to comprehend (Aten, Johns, Darley, 1971), the distribution of errors in words would probably have shown the same predilection for occurrence at the onset of the syllable as did the studies cited above. In other words, I propose that when patients with differing lesion sites and differing listening abilities are grouped together, the differences between two differing types of articulatory problems are obscured. Is it not possible that lumping together patients with a variety of articulatory behaviors is attributable, in part, to our failure to consider labels and modifiers?

In conclusion, given the present "state-of-the-art" of our test development with inadequate data bases, it is little wonder that Goodglass admits to being able to classify less than half of the aphasic patients tested. Test results do reflect lesion size, site, and etiological factors to cite only a few of the myriad of variables. The larger and the more central the dominant hemisphere lesion, the more likely we are to have a patient before us whose

test scores show uniform impairment and whose aphasia defies adjectives or subclassification. The smaller the lesion, be it anterior with apraxia of speech, nonfluency and less decoding impairment, or posterior, with inordinately greater fluency and qualitatively different articulation errors (literal paraphasias), the more likely we are to see nonuniform impairments. The more peripheral the lesion, the more likely the patient is to demonstrate specific language disturbances attributable to an etiology other than thromboembolic and require a very specific, focal treatment.

The majority of aphasic patients may not merit 'subtyping' from large groups. As a clinician, however, I am concerned not with group results and trends, but rather with the individual areas of strengths and the deficits of the patient before me. If taxonomy aids in matching type of impairment to type of language impairment and to selective treatment methods, I choose to accept "adjectives with aphasia" in carefully measured doses. Rosenbek's (1979) comments are cogent still, that not all treatment should be geared towards priming the ear to wag the tongue. Nonfluent aphasic patients need to "GO" with expanded grammar and semantic flow, fluent aphasic patients need to do what one of our patients said so well, "I'd better shut up so I can talk." I'll do the same.

REFERENCES

Aten, J.L., Johns, D.F. and Darley, F.L. Auditory perception of sequenced words in apraxia of speech. Journal of Speech and Hearing Research, 14, 131-143, 1971.

Baker, E. and Goodglass, H. Time for auditory processing of object names by aphasics. Brain and Language, 8, 355-366, 1979.

Burns, M.S. and Canter, G.J. Phonemic behavior of aphasic patients with posterior cerebral lesions. Brain and Language, 4, 492-507, 1974.

Darley, F.L. Aphasia. Philadelphia: W.B. Saunders Co., 1982.

Deutsch, S.E. Prediction of site of lesion from speech apraxic error patterns. In J. Rosenbek (Ed.), Apraxia of Speech: Physiology, Acoustics, Linguistics, and Management. San Diego: College-Hill Press, 1983.

Freeman, F., Sands, E., and Harris, K. Temporal coordination of phonation and articulation time in a case of verbal apraxia: A voice onset time study. Brain and Language, 6, 106-111, 1978.

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

Grober, E., Perecman, E., Kellar, L. and Brown, J. Lexical knowledge in anterior and posterior aphasics. Brain and Language, 10, 318-330, 1980.

Hanson, W., Riege, W., Metter, J., and Inman, V. Factor derived categories of chronic aphasia. Brain and Language, 15, 369-380, 1982.

Itoh, M., Sasanuma, S., and Ushijima, T. Velar movements during speech in a patient with apraxia of speech. Brain and Language, 7, 227-239, 1979.

Johns, D.F. and Darley, F.L. Phonemic variability in apraxia of speech. Journal of Speech and Hearing Research, 13, 556-583, 1970.

Kelso, J. and Tuller, B. Toward a theory of apractic syndrome. Brain and Language, 12, 224-245, 1981.

Kent, R.D. and Rosenbek, J.C. Prosodic disturbance and neurologic lesion. Brain and Language, 15, 259-291, 1982.

Kerschensteiner, M., Poeck, K., and Brunner, E. The fluency-nonfluency dimension in the classification of aphasic speech. Cortex, 8, 233-247, 1972.

LaFranchi, B., Aten, J.L., and Brick, N. Differentiating phonologic and semantic factors influencing word recognition in aphasic patients. Paper presented at Academy of Aphasia, Lake Mohonk, New York, October, 1982.

Orgass, B. and Poeck, K. Clinical validation of a new test for aphasia: An experimental study of the Token Test. Cortex, 2, 222-243, 1966.

Poeck, K., Kerschensteiner, M., and Hartje, W. A qualitative study on language understanding in fluent and nonfluent aphasia. Cortex, 8, 299-304, 1972.

Porch, B.E. Porch Index of Communicative Ability. Palo Alto, CA: Consulting Psychologists Press, 1967.

Shewan, C. and Canter, G. Effects of vocabulary, syntax, and sentence length on auditory comprehension in aphasic patients. Cortex, 7, 209-226, 1971.

Trost, J.E. and Canter, G.J. Apraxia of speech in patients with Broca's aphasia: A study of phoneme production accuracy and error patterns. Brain and Language, 1, 63-79, 1974.

Rosenbek, J.C. Wrinkled feet. In R.H. Brookshire (Ed.), Clinical Aphasiology: Conference Proceedings, 1979. Minneapolis, MN: BRK Publishers, 1979.