

Wrinkled Feet

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Do you remember the manticore? The beast with a man's head, a lion's body, and a dragon's tail? Probably he had some difficulty deciding whether to reason, roar, or spit fire. Clinical aphasiologists have had to be clinical manticores. Except for a short period after World War II when Wepman, Eisenson, and the others were treating brain-damaged soldiers in relative anonymity, clinical aphasiology has had to create and evaluate programs for aphasic patients, establish clinics, educate professional associates and the public, and act against indifference and even hostile attack, skills best learned by one with both earthly and mythical powers. We've needed creativity, courage (with all due respects to Rensberger who reminds us, in his The Cult of the Wild (1978) that the lion is not what human myth would have him be) and the ability to fling stinging darts with a flick of the tail.

Group studies by Wertz and his colleagues (Wertz, Collins, Weiss, Brookshire, Friden, Kurtzke, and Pierce, 1978) in the Veterans Administration Medical Center System, by Vignolo and his colleagues in Italy, (Basso, Capitani, and Vignolo, 1979) and by a host of others have demonstrated that we do good. More importantly, these researchers have advertised clinical aphasiology's willingness and competence to evaluate treatment in ways that satisfy the rigors of the scientific method. And while physicians and others remain who have not gotten the word or who have it but don't believe it, I think it is nonetheless possible for the first time to molt our dragons' tails and busy our heads with more crucial clinical business. It is my view of that business which fills the rest of this discussion.

On Theories in Clinical Aphasiology

Seron and his colleagues (1978) say about research and rehabilitation in neuropsychology that there is

"well-developed fundamental research with its schools, concerns, and methodological principles. On the other hand, there are therapeutic practices which, with the notable exception of Soviet works, have not given rise to any general theoretical considerations" (P. 76).

I think they would want to include clinical aphasiology in neuropsychology; they certainly had American as well as European management of the aphasic patient in mind.

Most of us would object to the extravagance of the charge, if not with its spirit. For example, Wepman in 1953 provided us with stimulation, facilitation, and motivation, and with the order to select the retained input-output pathway and develop that pathway until the patient can use it to let us know his mind. Schuell (Schuell, Carroll, and Street, 1955) two years later, told us "auditory stimulation may well be considered the foundation of all aphasia therapy" (P. 43). In 1974 Martin defined aphasia as a

"reduction in efficiency of the action and interaction of those cognitive processes which support language" and aphasia treatment as

"an attempt to manipulate or excite the activity of certain cognitive processes, not in the hope of curing aphasia, but of enabling the individual organism to achieve its maximum potential" (P. 80).

As clinical aphasiology has continued to mature, it has inherited, borrowed, and created a number of principles and "theoretical considerations" about what aphasia is and what its proper treatment should be. Holland (1979) has reviewed these as have Brookshire (1978), Eisenson (1973), and others.

The problem, as I see it, is not that we lack principles but that most of them have escaped rigorous testing. A companion problem is that aphasic patients' responses to treatment have been inadequately assayed for what they might reveal about both aphasic and normal communication. Four treatment principles in need of testing and the assumptions about aphasic and normal speech and language functions underlying them will be analyzed. The paper will not end in a sharp point capable of tearing the membranes of particular schools or points of view. Instead I hope to encourage us to test the fascia of each point of view regardless of how little or much we may like it.

Principle One: Bombard the Auditory Modality

Schuell and her colleagues (Schuell, Carroll and Street, 1955) nurtured and probably parented this one.

"Clinical findings indicate that impairment of auditory retention and recall are reversible to a considerable degree; and that improvement of articulation, word-finding, reading, and writing often results from the use of a single therapeutic principle: strong, controlled, intensive auditory stimulation" (P. 43).

Underlying this principle are several assumptions: 1) the core of aphasia is an auditory deficit, or a deficit in reauditorization; 2) stimuli in other modalities are converted to auditory equivalents, and it is these auditory equivalents which treatment must strengthen; and 3) a child's comprehension precedes his production, an ordering to be preserved in treatment activities for the aphasic adult.

Data demonstrating that comprehension improves with training and that auditory programs can improve performance in other modalities are neither abundant nor compelling. To be ignored for the moment are studies demonstrating that comprehension differs for different kinds of stimuli. These studies, usually descriptive and normally based upon only one or a few sessions with each subject, tell clinicians that aphasic patients can understand some things and not others (Darley, 1976); they are mute about whether systematic exposure to stimuli the patient can understand is therapeutic, about whether a patient is better able to understand something that is more difficult as a result of having understood something that was easier. One of the most impressive treatment studies where a clinician spent several sessions trying to improve aphasic understanding is by West (1973). She demonstrated that the comprehension of aphasic patients could be improved on Token Test-like items, and that the training generalized to

untreated items and to performance in other modalities. Holland and Sonderman (1974) using a similar, but not identical, program improved patients on Token Test-like training items but noted little generalization, even to similar items. In another study, Liebergott and Holland (1971) managed to improve comprehension, and they discovered that comprehension training generalized to production if they were careful about how production was scored. They also admit that such generalization

"while statistically significant, is not as great as we would have liked, possibly because all of the subjects in the study had such severe dysarthria and/or apraxia, as to cause many of their responses to be unintelligible" (P. 121).

Maybe we are asking too much of comprehension training; maybe also some of the assumptions underlying the auditory bombardment notion are in need of revision. For example, comprehension deficits are usual but not inevitable in aphasia. Stroke usually leads to a language deficit across all modalities; tumor does so less frequently. With a tumor, the language deficit may be confined to one or two modalities, and if the temporal area is preserved, comprehension may be relatively intact. This would be a trivial observation therapeutically if it were not linked with the second assumption, that stimuli in other modalities are converted to auditory equivalents. Some version of this idea no doubt motivated Schuell's intensive auditory programs and the expectation of generalization she held out for these programs. But as Smith (1975) and others have reported, the correspondence of graphemes, for example, and phonemes is not all that strong. Nor does all language function, regardless of modality, converge on the temporal lobe of the major hemisphere (Hier and Mohr, 1977).

Even more to the point, it may be, as Lesser (1978) says, that reception does not inevitably precede production, not in children and not in adults; and, in fact, it may be that "there are separate competencies for speech and for comprehension" (P. 48). Gardner and Winner (1978) seem to believe it, for they postulated, after studying the imitative skills of aphasic patients, that "production and comprehension capacities are organized differently in the nervous system" (P. 176-177). Prins, Snow, and Wagenaar (1978) may believe Lesser also because their study of physiological recovery in spontaneous speech production and in sentence comprehension for 74 aphasic patients leads them to say that,

"aphasic deficits in spontaneous speech production and in sentence comprehension are to some extent distinct, with their own recovery histories" (P. 206).

To say that two systems have separate competencies or that they are organized differently is not to imply a lack of interactions. Speech and comprehension obviously do interact in both normal and aphasic speakers. Nor is it to say that we need to abandon what, for lack of a better term, we might call auditory stimulation treatments. It is to say, however, that the idea of two systems (comprehension and production) rather than one (an auditory-vocal one) provides us with a number of additional interesting hypotheses for clinical testing. It seems likely, at least to me, that comprehension and speaking can be dissociated in aphasia, in which case auditory stimulation, even if protracted, would be feckless in improving the patient's speech. Unlike Kushner and Winitz's (1977) patient who

apparently began to name as a result of a purely auditory and pointing program, the patient with a comprehension-speaking dissociation would not. The Winitz and Reeds (1975) program, adopted for use with an aphasic patient by Kushner and Winitz, is but one method which would allow us to test the dissociation hypothesis clinically.

Also, it may well be as Prins, Snow, and Wagenaar (1978) have hypothesized, that the right hemisphere is better able to help the left with certain kinds of functions such as comprehension than with others, for example, production. They were anticipated in this by Pick (1973). If Pick and his beneficiaries are correct, certain therapy methods presently in vogue, such as heightening action imagery (West, 1977), may be appropriate not to language training in general, but to performance only in certain modalities.

We have hypotheses to test before we can conclude that the ear will always drag the tongue along, and it seems premature to accept uncritically that comprehension training must always be first or most important in aphasia treatment.

Principle Two: Use More Nearly Intact Modalities To Improve Performance in Less Intact Ones

This principle is so common that its origins are (or were for me) impossible to trace. Perhaps the most embellished statement of the principle is contained in the deblocking method of Weigl (1970) and Weigl and Bierwisch (1973) which Von Stockert (1978) calls "one of the most advanced techniques of systematic and programmed language therapy" (P. 100). Of the deblocking of semantic fields, Weigl (1970) says,

"the effect of deblocking is based on the prestimulation of certain semantic fields, using for this purpose an intact, 'non-blocked' channel as well as the existing connexion between the prestimulated and the blocked word (sentence)" (P. 288).

Weigl adds that the deblocking must be automatic or unconscious, which is to say, that the word to be deblocked is not identified nor is the deblocking stimulus. While deblocking can work, according to the author, for performance of any type in any of the modalities, this discussion of the method will be limited to the deblocking of words (names) because naming, besides being the most fascinating and difficult of linguistic performances (Maruszewski, 1975), is also the most thoroughly studied.

The main assumption underlying the principle of deblocking is that aphasia is a performance rather than a competence deficit. The matter is well put by Whitehouse, Caramazza, and Zurif (1978) in their introduction to a study of naming in Broca's and anomic aphasia:

"Thus, as one possibility, although aphasic patients may control conceptual elements at a nonlinguistic level, it is nonetheless conceivable that these elements are insufficiently structured for adequate lexical organization. Alternatively, it is possible that brain damage spares the conceptual structures underlying lexical organization but dissociates and disrupts mechanisms responsible for addressing and/or retrieving information from these structures" (P. 65).

The first statement defines a competence deficit, the second, one of performance. For a response to be deblocked, competence needs to be preserved.¹

A second assumption, and one that has theoretical and data support, is that words are represented in the brain by clusters of associations. Goodglass and Baker (1976) define the semantic field operationally as recognition of seven classes of association with a target word: actual noun, superordinate term, usual environmental location, action performed, another member of the same category, descriptive adjective; and a phonologically similar term. Weigl (1970) identifies somewhat different components, including "Ontogenetically developed connexions between 'meanings,' i.e., 'object-meanings' as well as 'word-meanings'" (P. 287). The different kinds of cuing to deblock the target and in particular the presence of "object meanings" which can be tapped by seeing real or pictured objects, and "word-meanings," which can be tapped by auditory stimuli, provide the neuropsychological basis for Weigl's method of presenting a real object before speaking its name in the effort to elicit the name of that object. A third assumption, although not one inextricably linked with the semantic field idea, is that anomia in aphasia is a unitary symptom; all aphasic patients have difficulty with naming, only the deficit's severity distinguishes one patient from another.

Weigl's own deblocking experiments seem to confirm the performance rather than competence deficit in aphasia as do other, more extensive naming experiments like that of Wiegand-Crump and Koenigsnecht (1973). They taught words to four anomic patients and found that not only did the patients learn, but that the learning generalized to untreated names, leading them to the conclusion that the anomic's competence is preserved. Mills, Know, Juola, and Salmon (1979) used error inconsistency in a naming task to support the same conclusion. They say "the inconsistency of the errors also lends support to the notion that the aphasic subject who demonstrates a word retrieval problem suffers from an impairment of processing rather than a deficiency in the contents of the lexicon" (P. 86). Goodglass and Blumstein (1973) are not so sure. In their introduction to Weigl and Bierwisch they say

"an alternate interpretation (to that of Weigl and Bierwisch) is that gaps in the aphasic's imperfectly retained competence may be temporarily restored by cuing or 'deblocking,' with the result that adequate performance becomes possible" (P. 11).

Studies by Pease and Goodglass (1978), Goodglass and Baker (1976), Tsvetkova (1975), and by Whitehouse, Caramazza, and Zurif (1978) all challenge the concept of aphasia as a performance deficit. Pease and Goodglass, after studying the effects of six cuing conditions on Broca's, Wernicke's, and anomic patients, conclude that retrieval deficits are compounded by gaps in the semantic field or lexical storage, gaps which they consider to be impaired competence. Goodglass and Baker also conclude that aphasic patients know less about objects than they previously did, despite being able to name them. Based on the drawings of aphasic adults,

¹ Actually the situation is more complex than this. Even the globally aphasic patient whose semantic fields can hardly be considered intact can occasionally produce a word with strong provocation.

Tsvetkova (1975) suggests that the anomic aphasic has a deficit in the 'standard images' of words, a deficit which seems more akin to competence than to performance. Whitehouse, Caramazza, and Zurif (1978) studied the effects of form and function on naming by Broca's and anomic aphasic patients. They concluded, because the Broca's patients so clearly resembled normal speakers and the anomics differed so radically, that the anterior patients had "relatively intact underlying lexical structures with normal abilities to integrate perceptual and functional information and to deal with fuzzy conceptual boundaries" (P. 71). The anomic patients, on the other hand, had "an impairment in the underlying conceptual organization of the lexicon rather than retrieval difficulties" (P. 63). Besides, this study, by supporting the hypothesis that competence may be impaired in some but not all patients, broadens the issue by also flying in the face of the assumption that naming deficits are of a single kind.

We have performed several deblocking experiments; some of them were successful (Rosenbek, Green, Flynn, Wertz, and Collins, 1977), but the more provocative ones (at least for this discussion) were not. For example, we used a reading and writing program to try to deblock confrontation naming in a 55-year-old fluent aphasic patient. He could learn to write names after practicing them for only a few seconds each, but he could not read these nor did written naming transfer to verbal naming. Weigl and Bierwisch (1973) would say his problem is one of dissociation and that such a pattern "cannot be ascribed to different aspects of competence" (P. 19). Perhaps, however, our patient's drawings of objects were also distorted, he often failed to point to objects by name and function, and he sometimes failed to write names correctly. We might, therefore, erect a competitor for the Weigl and Bierwisch point of view, in the form of the hypothesis that words or semantic fields are not supramodality clusters and that competency can be modality specific. Related to this hypothesis is the Hier and Mohr (1977) position that Wernicke's aphasia is not a multimodality deficit unless the lesion involves temporal, parietal, and occipital regions. With lesions confined to the temporal lobe, listening and speaking may be impaired with "relative preservation of lexicographic language functions" (P. 124). Equally intriguing is their companion statement that "The traditionally postulated supra-modal unity of the syndrome (of Wernicke's aphasia) may well yield to more modality-based sets of deficit profiles" (P. 125).

Lesser (1978) suggests that 'deblocking' studies can tell us more about semantic fields. Such studies can also tell us more about modalities, cortical function, aphasia, and ultimately more about treatment. Schuell's idea that aphasia is a unitary disorder has comforted us because of its simplicity. We no longer need the comfort. We need instead the anguish that arrives with changing our minds or (at very least) with asking the questions and collecting the data that may force us to change.

Principle Three: Work with Task Continua within A Modality

This principle, like the odor of pinon on a summer's night, seems to emanate from all directions at once. Brookshire (1978) and LaPointe (1978) have described the principle and the assumptions underlying it, and Bollinger and Stout (1976) among many others, have developed a framework, "Response-Contingent Small-Step Treatment," that gives the principle life to sustain it in practice. Popular work books (Keith, 1972; Stryker, 1975;

Kilpatrick and Jones, 1977; Brubaker, 1978) perpetuate the principle as do general treatment articles (Linebaugh and Lehner, 1977; von Stockert, 1978). Typical of the variety in specific treatment regimes based on it are those by Holland and Sonderman (1974) using Token Test performance as a guide about where to start and where to go and by Linebaugh and Lehner (1977) using a hierarchy of ten cues to elicit names.

This is an especially alluring principle because it looks and sounds so right. The assumptions underlying it, like the principle itself, are sometimes embraced uncritically. The first assumption is that patients should have more successes than failures in treatment. Brookshire (1978) says errors should constitute no more than 20% of all responses, depending on the conditions, and success is more likely if easier tasks precede difficult ones. He has shown (1972), for example, that aphasic patients name "hard" words more successfully if such words are preceded by "easy" ones, both hard and easy being determined by the patient's responses on pretesting. He also confirmed a common clinical impression that "easy" words can be made more difficult by preceding them with "hard" words. Errors breed errors, a principle he has also demonstrated for auditory comprehension tasks (Brookshire, 1976). It is of interest that the facilitating effect of easy before hard was not demonstrated in sentence comprehension.

The second assumption is that tasks within a loop (input-integration-output) vary in difficulty in ways that clinical aphasiologists have learned to identify and that movement through the tasks is both possible and therapeutic. Holland and Sonderman's (1974) treatment program for treating comprehension with Token Test-like stimuli is based, whether consciously or not I cannot tell, on this assumption, but unfortunately is not a test of it. At least I cannot tell from their data whether performance at one level of Token Test difficulty was improved by having reached criterion at a previous level. Their observation about limited generalization to untreated items even of similar difficulty makes me wonder. One wonders even more perhaps about programs that repeatedly elicit the same response; for example, the names of ten items with a hierarchy of cues. Do we know that a name is more likely in confrontation if it has been elicited in cloze or in imitation; or if it is more likely in spontaneous speech if it has been correct once or even one hundred times in a confrontation naming drill? At issue is not whether aphasic patients learn; Carson, Carson, and Tikofsky (1968) among others (Pizzamiglio and Roberts, 1967) have shown us that they do. The issue is transfer or generalization. Aphasic patients, as Carson, Carson, and Tikofsky have also observed, may treat similar tasks as new tasks.

Assumption two, therefore, is in need of scrutiny. We have already mentioned Carson, Carson, and Tikofsky's observation that aphasic patients fail to use old information to deal with new tasks. To this we would add the possibility that activities operating within the same loop (or appearing to operate within the same loop) in normal speakers can be dissociated in aphasia. Imitation, which some treatment programs place in the auditory-vocal loop, and with which treatments to improve speech often begin, is a good example. It has been suggested that ability to imitate may be independent of some or all other language performances, especially comprehension and especially in fluent aphasic patients by Saffran and Marin (1975) and by Ludlow (1977) after her review of selected literature. In addition, the performance of conduction, transcortical motor, and

transcortical sensory aphasic patients strongly implies that imitation ability is independent not only of comprehension but of spontaneous speech competence (unless the aphasic patient has a coexisting motor speech disorder).

Perhaps we are lugging our task continua around in the wrong direction. Instead of preparing them the night before and bringing them to our sessions or to our research, we ought to arrive at treatment empty handed (but not empty headed) and leave the session with an orderly set of tasks that have helped the patient move his communication toward greater volitional-purposive controls. I think some of those things that seem so orderly to our common sense are not at all orderly to our patients.

Principle Four: Do Not Strip Language of its Context

Both Holland (1977, 1978, 1979) and Davis and Wilcox (1979) have written perspicacious reviews of the concepts that have lead clinical aphasiologists toward language pragmatics—toward the context of language rather than its form. These authors argue that the journey has serious and happy implications for aphasia treatment, because pragmatics encourages the clinician to value what the patient can do rather than what he cannot, and to work for "conveying a message" rather than for "linguistic adequacy." Holland (1977) for example, urges aphasic patients and aphasia clinicians to "concentrate on relevant communicating and relevant language" (P. 175).

And how is pragmatic treatment different from traditional treatment? Holland (1977) says traditional treatment "is disproportionately centered on the propositionality of an utterance, not on its communicative value" (P. 170). She says also that

"Language pathology is a prescriptive profession, basically attuned to fixing up what is 'wrong' with a relatively inflexible, if fuzzy, definition of what is 'right,' and bustling about to change wrong to right" (P. 173).

And how do we look as we bustle about? Davis and Wilcox (1979) say

"Emphasis is placed on selection of the stimulus or antecedent event to elicit an expected response. Objectives directed toward improving impaired verbal modalities are added to this formula, so that a traditional treatment task involves several stimulus-response trials focusing on the patient's use of either receptive or expressive language processes" (P. 16).

The emphasis, to use terminology popularized by Chapey (1977), is on convergent tasks or tasks which "require subjects to converge upon one correct, previously agreed upon answer" (P. 257). Such emphasis, Chapey would argue, is misplaced, especially if it dominates every treatment session. In the pragmatic view, the last time a strict naming drill was appropriate was when Adam was invited to name the animals.

Recognizing that pragmatic treatment can so easily degenerate into what Taylor (1964) has called unspecific stimulation, Davis and Wilcox (1979) have built an elaborate and controlled treatment called PACE (Promoting Aphasics' Communicative Effectiveness). This treatment, which intends to help a patient "communicate as independently as possible," is based on four principles; the exchange of new information, allowing the patient to choose the modalities to communicate with, equal participation

by patient and clinician, and clinician feedback. The core of the program are sets of object pictures which the patient and the clinician alternately send and receive information about. The patient is free to gesture, speak, write, or combine these modes so long as he gets the message to the listener.

This principle assumes much that has already been reviewed, foremost among the assumptions being that aphasia is a performance rather than a competence deficit. This competence, goes the next assumption, allows them to be good communicators even if they are not good talkers. A third assumption is that a patient is more likely to communicate an idea the next time if he has done it successfully one time, and that successful communication of one idea will generalize, so that other untreated ideas become easier to express as well.

Pragmatic programs are appealing. Clinicians become communicators rather than coaches or instructors, and presumably the patient is allowed to use his strengths and to be rewarded for them. And what of the assumptions? Iconoclasts might observe that not all patients show the competence for questioning, warning, advising, ordering and the rest that popular anecdotes and some literature (Boller and Green, 1972) ascribe to them. Stereotyped, undifferentiated and, therefore, noncommunicating expressions and gestures are reasonably common, especially from more severe patients, as are expletives uttered in restaurants much to the shame of the spouse, and failure to quit talking when the clinician's phone rings or a colleague knocks on the door, and as are failures to move in a wheelchair so that another patient can get by. Perhaps the profane patient's competence is too weak to combat his damaged brain's perseverative drive. Perhaps fatigue, field defects, and despair explains episodes of inappropriate activity. Perhaps. Perhaps, too, the lesion that batters performance also disfigures competence. Competence may be a range, wider in some aphasic patients than in others.

I think, too, that we must worry about how much a patient is changed each time he gets his point across. Unless a program builds on systematic repetition and feedback as PACE does, success may not father success. Unique responses are not necessarily therapeutic. Conditions often conspire to somehow jerk a response from even a globally aphasic patient. How many of us have had to call the family aside to caution them that a glimmer of recognition or the sparks from a "No" are too weak to signal recovery, in fact are so weak that they may not be seen again.

To the degree that the response is an automatic reactive one (automaticity is probably a range as well), is the patient the better for its performance? We might hypothesize that the neural substrates for such responses are different from those of volitional-purposive production and to elicit such responses 10 or a 100 times will have no permanent effect. Finally we have the problem of definition. When are naming or the much maligned "point to treatments" pragmatic exercises and when are they barren, traditional drills? It may be that Holland (1977), Tikofsky (1979), Davis and Wilcox (1979), and the others are only warning clinicians about mindlessness. When our minds are on establishing the conditions so that the patient can have his say about something important, the treatment will be appropriate, regardless of what we call it. The thing to be done is to join Davis and Wilcox in demonstrating that such exercises are not merely relevant, but that they are also therapeutic and that they help more than other approaches.

Conclusion

Clinical aphasiologists, probably like all the other "ologists," are swept along in a stream of change. We flow from one new idea, or method, or luminary to another. The scenery and the passions change, and the variety invigorates us. One drawback, however, or so it seems to me, is that we never get to know enough about where we have been. We are swept toward a new idea before we have had a chance to know and test the old. It seems to me that the clinicians in this room ought to decide to place themselves firmly in the stream of change at whatever location and depth each prefers, and that we ought not to move until we have tested and gotten to know what is true and what is false about some little area of treatment. That testing may well leave us fighting the current for a long time. But perhaps we as a conference can reward that steadfastness. We can do so by admitting next year only people with wrinkled feet.

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Discussion

Note: Discussants did not ask questions; they did react to the content of both Martin and Rosenbek's papers. These reactions are summarized in order. The one response by Martin and the one by Rosenbek will be identified so that the reader will have an easier time interpreting what went on.

- 1: Speaker described a patient whose responses to treatment seemed to confirm the dissociation between speaking and comprehension that plagues some aphasic patients.
- 2: Speaker described two patients whose responses confirmed the same thing.
- 3: Speaker reminded the audience that when production is being drilled, comprehension is also, but that the reverse is not true.
- 4: Martin's paper reflected a search for Truth; Rosenbek's, a search for Good. The search for truth is a search for models or for the general explanation of things. The search for good is a search for those things to help us with the next patient through the door. Both searches have accompanying dangers. The first may insulate us from realities; the second may blind us to the responsibility for making sense of daily observations.
- 5: Martin objected to any implication that the search for truth was not clinically viable. He said that his content was compatible with Rosenbek's although he might disagree with certain of Rosenbek's techniques and interpretations of data.
- 6: Speaker thought there should be no dichotomy between the two searches. With right hemisphere patients, especially, the researcher needs both to develop models and collect descriptive information.
- 7: Speaker reminded the audience that clinicians need to identify forces in the patient's environment that influence his speech. Also reminded us that an aphasic patient is more vulnerable than a nonaphasic one to environmental influences.

8: My grandfather used to tell me that truth was good.

9: Speaker reminded us that much of traditional practice is data based and successful and that clinicians should not throw that work away. We should enjoy the old work and be challenged by the new.

10: Rosenbek replied that traditional methods are responsible for many successful treatments, and traditional views of what aphasia is help to explain those successes. His paper was an attempt to explain the failures and how some of those might be changed to successes if clinicians would attempt to translate recent research findings into clinical hypothesis.

11. Speaker said that one of the groups in the Veterans Administration Cooperative Study received the kind of therapy Rosenbek was describing, one group received the kind Martin was describing. Both groups improved.