Spring 1982

The Multi-Facets Of Aphasia: A General Orientation To The Disorder

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THE MULTI-FACETS OF APHASIA:
A GENERAL ORIENTATION TO THE DISORDER

Submitted in partial fulfillment of the requirements for graduation with honors to the Department of Communication Arts at Carroll College, Helena, Montana

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March 23, 1982
This thesis for honors recognition has been approved for the Department of Communication Arts.

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ACKNOWLEDGEMENTS

The undertaking of this thesis project was made very meaningful by the thoughtful suggestions, constructive criticisms, and helpful input provided by many individuals both at Carroll College and the University of Montana. I wish to express my deepest gratitude to Dr. Kellogg Lyndes of the University of Montana Communication Sciences and Disorders Department. He spent many hours explaining, refining, and directing me toward a clearer understanding and presentation of the disorder of aphasia. At the same time, I would like to express my thanks for the suggestions given by staff members at the University. Specifically: Dr. Charles Parker, and Fran Tucker. In addition, Linda Fifer-Prill, a speech therapist with the Missoula Community Rehabilitation Hospital, who spent an afternoon providing me with data concerning patients she has worked with in therapy. I would like to extend my thanks to my reader, Dr. Jean Smith, professor of Biology at Carroll, for the comments she made during her reading of the thesis. Likewise, I would like to recognize my thesis director and advisor, Mr. Harry Smith. His confidence and support over the last four years cannot adequately be expressed by simple words. His willingness to help increase my knowledge will never be forgotten.
Finally, I express my thanks to all my family and friends who shared in my frustrations, but mainly my joys, throughout the writing of this thesis.
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INTRODUCTION

The communication of our thoughts, viewpoints, and emotions are synonymous with being human. By interacting and identifying with other humans through a mutually symbolic language code, man expresses ideas about self and world. This is possible because humans possess a dynamic center, the human brain, to control, reason, and formulate their perceptions and thoughts. "By means of complex cerebral processes, people attach meaning to symbols, combine symbols to represent impressions, and understand other's speech, thinking, and ideas" (U. S. Department of Health, Education and Welfare, 1979, p. 2). These processes, carried out by the human brain, control man's higher mental, motor, and sensory functions as well as playing an important role in speech and language comprehension and production.

Within this communication, the transmission of information is enhanced by the means of gestures, facial expressions, and other non-verbal modalities. It is through these modalities that humans reveal their motives and character, in addition to the general information they desire to transmit. "What is needed to analytically learn from communication encounters is a system for listening beyond what is said, to how it is said, and what it does" (Larsen, 1976, p. 7). This
point becomes even more significant as humans engage in communication encounters with those individuals handicapped in the communication skills. It will be those adults, experiencing damage to the cerebral hemispheres from any number of etiologies, on which this paper is focused.

This language disturbance, termed aphasia, is an immensely complex condition usually caused by a lesion in the language dominant cerebral hemisphere. Authors formulating hypotheses about aphasia date back to the time of Hippocrates. He believed disturbances in speech and language functioning were associated with lesions in the brain (Sessler, 1981, p. 206). The effects of these lesions resulted in the disruption of communication ability; therefore, not only is language impaired, but understanding of the spoken word, reading, writing, and arithmetic abilities may be disrupted as well. In addition, depending on the location and the extent to which the cerebral damage involves the language dominant hemisphere, the degree of impairment varies across the aphasic population. Essentially, the reduction within each of these language skills is seen in the overall pattern of aphasic impairment demonstrated by each individual (Cohen, 1971, p. 2).

According to Jon Eisonson (1967), these aphasic impairments are disruptions of an individual's verbal habits as well as other patterns of communicative behavior. The most severely reduced are the processes which require a "transition from inner symbolization into a conventional, interpersonal
linguistic system" (U. S. Dept. of HEW, 1967, p. 36). It is the use of the conventional language formulation which is damaged and less often the aphasic individual's cognitive capabilities, knowledge, or intelligence.

Although there are copious definitions of aphasia, the writer considers the following statement by Schuell, Jenkins, and Jimenez-Pabon (1975) to incorporate the major characteristics encompassing aphasia. They define aphasia as a "reduction of available language that crosses all language modalities and may or may not be complicated by perceptual or sensorimotor involvement, by various forms of dysarthria, or by other sequelae of brain damage" (p. 3).

The purpose of this paper is to explore the previously established fundamental principles presented in the literature concerning aphasia. Hopefully, this will demonstrate the necessary background underlying this disorder as well as developing a better understanding of the aphasic individual. Certain assumptions are utilized with aphasic individuals; therefore, an understanding of the principles and behavioral changes surrounding aphasia allows a clearer path toward intervention and rehabilitation.

Although theories are formulated illustrating the skill and behavior changes manifested through aphasia's occurrence, we cannot neglect the fact that it is the individual with unique, personal characteristics and qualities upon which we base our concern. Each person, depending on his personality, experiences, and education brings an unlimited set of
circumstances that have been disfigured through brain injury into panorama. The focus of concern among individuals responsible for a patient's recovery develops from an appreciation of the extensive range of varying language and non-language deficits which affect each individual.

It is the writer's intent to address the nature and extent of aphasia in the following pages, hopefully remaining mindful of the uniquely human aspects of those who suffer from this disorder. In addition, the brevity of presentation from which these previously attained insights are derived must be acknowledged. As mentioned before, aphasia is an immensely complex area with the current state of the art of treatment still dependent on further research, not only with the disorder of aphasia, but extensive research in the area of neurology as well.
CHAPTER 1
CEREBRAL FUNCTIONING IN RELATION TO APHASIA

In publications devoted to the topic of aphasia, etiology is a well understood area encompassing this complex language problem. Ordinarily, aphasia is the repercussion of one of the following: hemorrhage, thrombosis, embolism, infection, tumor, traumatic injury, or concussion, to those regions of the brain responsible for speech and language functioning. The identity of these regions, indicated by the diagram (Fig. 1) from the findings of Penfield and Roberts (1959), receives confirmation from a myriad of anatomical and physiological evidence. In addition, this evidence demonstrates there remains an elaborate interconnection among all levels of the cortex and brain stem. Therefore, speech and language functions exist within a sensorimotor cerebral cortex. In more detail, as Penfield (1957) stimulated the postcentral and precentral gyri, the responses obtained were both sensory and motor in nature. Therefore, "these areas are a mixed sensorimotor area" (Schuell, 1974, p. 27). By correlating these findings to the disorder of aphasia, Darley (1978) refers to aphasia "as involving part of the so-called association areas of the brain which integrate the complex sensory and motor mechanisms" (p. 78).
Fig. 1. Speech Centers Identified by Penfield and Roberts (1959). Drawing summarizing the areas in language dominant cerebral hemisphere which are normally devoted to the ideational elaboration of speech. These areas illustrate where stimulation or excision produced language disturbance.
Many authors have conceptualized the relationship existing between aphasia and the brain mechanisms thought accountable for these language disabilities. From landmark investigations concerning brain organization, we are able to infer the possible outcomes from damage to these areas (Branch, Milner, and Rasmussen, 1964; Gazzaniga and Sperry, 1967; Penfield and Rasmussen, 1957; Penfield and Roberts, 1959). More specifically, the understanding and knowledge of brain organization allows discovery and exploration into the many facets of this complex language disorder. Unfortunately, as seen with nearly every clinical problem, explanations consist of a variety of viewpoints, biases, organizations, and nomenclature. Within the theoretical explanations of aphasia, the reader encounters the biases of the localizationists at one end of the continuum and those biases of the non-localizationists at the opposite end. In spite of these differences, the most constructive approach appears to be to incorporate portions of these two theoretical positions, fitting them to meet the particular needs of the aphasic individual. Understandably, the nature and scope of aphasia presents a unique challenge for students of communication disorders to undertake.

Localizationists Versus Non-Localizationists

This challenge begins with a general orientation concerning the essential characteristics encompassing the two extreme explanations regarding aphasia, those from the
localizationists and non-localizationists respectively. The discussion is initiated by the localizationist viewpoint stating that specific symptoms from cerebral damage can be localized as damage in specific areas of the brain. In essence, the aphasic's performance can be associated with the specific neurological mechanism thought to be accountable for a particular form of the disability. Through the accomplishments of Broca, Wernicke, and their contemporaries certain organic impairments which manifest themselves in speech and language function have been reliably assigned to lesions in specific regions of the brain (Goodglass and Kaplan, 1972, p. 1; Sessler, 1981, p. 205). Perkins (1976) summed up the localizationist viewpoint in this manner: "In comparing the brain to any other mechanisms, albeit an extraordinary complex organism, and therefore impairment of a particular part of that mechanism results in a particular disturbance" (p. 85). Therefore, with information regarding the site and extent of cerebral damage, certain assumptions can be formed concerning the loss of specific behaviors.

On the other hand, a rebuttal point from the non-localizationists has equally important considerations. Their underlying arguments are based on the belief that language disturbances cannot be totally correlated with the precise location of the cerebral insult that causes them. As Brookshire (1978) explains, "non-localizationists maintain that the brain functions as a single integrated unit and lesions in any given area will not result in disruptions
which are limited to specific abilities, but instead will be generalized behavioral deficits" (p. 21). This line of thought has been advanced in the monumental works of Hughlings Jackson (1915), Pierre Marie (1951), Henry Head (1926), and Kurt Goldstein (1942). Conversely, in review of the literature, some non-localizationists may postulate that clusters of behaviors result from damage to certain brain regions. It may be concluded that through observation of these behavioral clusters, certain hypotheses lead to reasonably accurate assumptions regarding the damaged area of the brain.

Reiterating, the frameworks presented above are the extreme positions concerning the relationships between cerebral insult and the resultant disabilities of language functioning. In like fashion, both positions present valid arguments worth considering. The writer tends to be persuaded toward a bias of Wepman's (1951) in which he does not overlook the views held by the localizationists; however, he states, "a more hopeful prognosis can be made for aphasic adults when recovery follows reintegrations of the remaining cortical tissue into a functioning whole" (p. 18). Although the evidence from adult aphasics suggests "brain dysfunction impairs language processing in a fairly specific manner" (Hubbell, 1981, p. 137), the reader is cautioned against relying completely on any proposed anatomical locations as being causal for behaviors that may be observed as they are manifested among aphasic patients.
Cortical Division of Speech and Language Processing

A second qualification which needs to be addressed concerns the proposed division of speech and language production between the two cerebral hemispheres. Despite the fact they appear structurally identical, the two cerebral hemispheres are not functionally similar. For the majority of individuals, the left cerebral hemisphere handles the symbolic system of learned rules, that is, our language capacities. For that reason, it has been identified as the language dominant hemisphere. This division of labor is readily apparent in split-brain subjects. When the corpus callosum is surgically disconnected, the right hemisphere becomes almost inarticulate and agraphic; although further examination finds it "superior in managing melodic information spatial, temporal relations" (Cotman and McGaugh, 1980, p. 386) which are equally important and integral parts of language production. This evidence from split-brain subjects estimates that in 90 percent of the population the left cerebral hemisphere governs the processing, storing, and retrieval of linguistic symbols (Perkins, 1978, p. 88). In Milner's terms, "the functions of the left hemisphere may be termed verbal, sequential, or analytic and those of the right as non-verbal, spatial, or synthetic" (Milner, 1974, p. 2).

In summary, although a majority of our linguistic abilities have been determined by many investigators to reside in the left cerebral hemisphere (Cotman and McGaugh,
1980; Geschwind, 1970; Milner, 1974; Sperry, 1974, 1977; and Penfield and Roberts, 1959), the right hemisphere is shown to be important in perception of phonological and intonation cues (Blumstein and Cooper, 1974; Blumstein and Goodglass, 1972). Thus, the dynamic interaction of both cerebral hemispheres cannot be segmented or ruled out. In order to be in agreement with current theory, it can be suggested that language production is bilateral. In essence, there is therapeutic value in stimulating both hemispheres in the recovery of language processing following damage to the language dominant cerebral hemisphere (Albert, Sparks, Helm, 1973).

Furthering the discussion of cerebral dominance logically leads to commenting on peripheral handedness which is considered to be the inverse of cerebral dominance for language. Research conducted at the Montreal Neurological Institute demonstrates language dominance to be distinct from handedness. Stated more strongly, when lesions occur in the left temporal and/or parietal regions of right handed individuals, an impairment in many areas of language processing may be expected (Gilbert, 1972).

While there remains a relationship between cerebral dominance and handedness, the following figures will illustrate the incompleteness of this relationship: In Sessler's book, *Stroke*, she cites that in 97 percent of the population the language center resides in the left cerebral hemisphere with 89 percent of the people using their right hand.
Sessler (1981) estimates right hemisphere as the language center in 3 percent of the population (p. 205). Other literature (Anthony and Thibodeau, 1979; Cotman and McGaugh, 1980; and Gilbert, 1972) approximates 90-95 percent of the right handers to be left hemisphere dominant for speech and language. Also stating, 5 percent of the population are left handed with two-thirds of the 5 percent being left hemisphere dominant (Brookshire, 1978, p. 22).

Hence, it is valid and necessary to keep this imperfect relationship in mind when considering site and extent of lesions in speech and language disturbance. Since certain language functions, especially those for producing linguistically correct utterances or comprehending the linguistic messages transmitted by others, are located mainly with the left cerebral hemisphere, the conclusion of Pick (1973) can generally be accepted. He states, "Right handed individuals receiving lesions to speech and language related structures in the left hemisphere will acquire aphasic disorders, with minimal aphasia or the absence of aphasia when the lesion is on the right" (p. 38).

**Brain Mechanisms of Thought, Speech, and Language**

After recognition of the supporting evidence establishing the left cerebral hemisphere's dominant role in language behavior, certain questions typically arise: Is there localization of thought, language, and speech within the language dominant hemisphere; How are the various tasks of thought,
language, and speech performed; are the three interconnected in any way? Other questions arise concerning the essence of our cognitive strategies, the means by which humans process information. In order to obtain any intent from language after utterances have been recognized, they need to be actively processed in some sort of cognitive fashion (Hubbell, 1981, p. 37).

These questions have been studied by numerous researchers following again in the footsteps of Broca and Wernicke who formed the initial perceptions (Brookshire, 1978; Eisonson, 1973; Geschwind, 1970; and Lyons, 1981). In what may be termed "landmark discoveries," Penfield and Roberts (1959) and Penfield and Rasmussen (1968) demonstrated results that supported the findings of Broca and Wernicke. During their neurosurgical procedures, they noted that electrical stimulation of Broca's area, the motor speech area in the inferior frontal gyrus of the left cerebral hemisphere, interfered with the patient's attempts to nominate words in order to express ideas. Similar stimulation of Wernicke's area, described as the superior and posterior portion of the temporal lobe, caused destruction to the patient's understanding of oral language (Nicolosi, Harryman, Kresheck, 1978, p. 6). Finally, an area located anteriorly to the Rolandic motor foot area within the midsaggital fissure is referred to as the supplementary speech motor area. Penfield and Roberts (1959) conclude this area to be the most dispensable of the three areas investigated; however, prolonged
aphasia may result from damage to the supplementary speech motor area as well. Penfield's and Rasmussen's (1968) conclusions were substantial in that they demonstrated an extraordinary degree of specialization for speech within the language dominant hemisphere (Fig. 2). "Removal or injury of any one of the three (frontal, parietal, and temporal speech areas) will inevitably produce aphasia" (p. 200). Conversely, it was pointed out that removal of the sensorimotor cortex between the frontal and parietal speech areas did not result in aphasia. In a similar manner, aphasia was not produced during removal of the anterior and inferior parts of the dominant temporal lobe.

The following summary of the major defects which are produced by unilateral ablation of the cortical areas may be explained as follows (Fig. 3):

(1) paralysis and hypertonia of arm or leg--precentral gyrus;
(2) loss of tactile discrimination and position sense in arm and leg--postcentral gyrus;
(3) homonymous hemianopsia--banks of the calcarine fissure;
(4) defect in planned initiative--frontal lobe;
(5) aphasia--three specific areas of dominant hemisphere (Penfield and Rasmussen, 1968, p. 200).

The previous observations seem to bring into perspective the complex issue being addressed by the current chapter. At the present time, there seems to be a "void in our knowledge about how people actually store and process language" (Hubbell, 1981, p. 30). The questions outlined may never be completely understood due to the fact the intricate brain
Fig. 2. Areas of Vocalization and Aphasic Interference with Speech (Penfield and Rasmussen, 1968, p. 106).

Fig. 3. Summary of Major Deficits Produced by Unilateral Cortical Ablation and Destruction (Penfield and Rasmussen, 1968, p. 199).
mechanisms that are associated in the production and comprehension of language may never be more than hypotheses. Hopefully, further research will continue to produce new insights into the mechanisms of language processing and production.

Although inferences must be used, the implications for a descriptive analysis of the areas recognized as having a role in human speech and language ability seems mandatory. The previously mentioned significance of Broca's, Wernicke's, and the supplementary motor speech areas is necessary to this discussion, but not sufficient when making these inferences concerning other cortical areas and neural pathways crucial in speech and language functioning.

Within the five lobes of the cerebrum (Fig. 4) lie the primary sensory and motor cortical areas. Briefly, these areas and their surrounding regions are specialized for the elaboration of sounds (auditory), sight (occipital), bodily sensations (somesthetic), and movement (motor) in very important ways. More importantly, they are joined to each other by association fibers which provide for synthesis among all the cortical areas of varying function (Cotman and McGaugh, 1980, p. 21; Sessler, 1981, p. 205). In addition, the cortical areas related to speech and language processing are diagrammed with their respective Brodmann numbers (Fig. 5). Their identified functions are as follows:
Fig. 4. Cerebral Lobes Including Primary Areas of Information Input to Human Cortex (Penfield and Roberts, 1959).

Fig. 5. Diagram of Cerebral Cortex Illustrating Areas of Localization According to Brodman '09 (Gray's Anatomy, 1959).
Areas 1, 2, 3  Perception of touch, pressure, temperature, and movement of muscles
Area 4      Motor projection areas for voluntary movements
Area 6      Extrapyramidal area
Area 7A     Minor formulative and elaborative speech center
Area 7C     Integrative center for the vocal expression of emotion
Areas 8, 9, 10, 11  Frontal association (ideational) areas
Area 22     Auditory reception area
Areas 41, 42 Wernicke's auditory apperception area
Area 44     Broca's area (Schuell et al., 1975, p. 25).

Thus, with an appreciation for the intercommunication and synthesis among all cortical and the association fibers, certain disorders can be localized within specific areas due to the particular range of abilities in speech and language processing remaining available to the patient. It should be noted that this relationship is not a perfect relationship, but it is viable enough to assist in evaluations by both therapist and physicians.

Two sources, notably Geschwind (1964) and Brookshire (1978), have developed and presented a "neuroanatomical explanation of the aphasias, focusing on the region surrounding the Sylvian fissure in the language dominant hemisphere" (Brookshire, 1978, p. 25). Two of the important structures, Broca's and Wernicke's areas, have previously been described; therefore, the following structures need to be included: the primary auditory cortex which is associated with Areas 22, 41,
and 42. Areas 41 and 42 (Gyrus of Hescl) lie superior and within the Sylvian fissure; whereas Area 22 is the superior temporal gyrus and may best be considered the region accountable for the primary perception of auditory stimuli. The arcuate fasciculus is the association fiber tract connecting cortical areas in the mid-temporal lobe (Wernicke's area) to Broca's area. "The arcuate fasciculus is thought to be the major pathway by which the acoustic-phonemic patterns of words are transmitted forward to Broca's area for motor encoding" (Brookshire, 1978, p. 25).

Concluding, the four regions thought to have extensive control in speech and language processing, namely Broca's and Wernicke's areas, the primary auditory cortex, and the arcuate fasciculus, provide the final theoretical explanations involving brain mechanisms on which this chapter was built. In the ensuing chapters, the writer structures the discussion to encompass only the linguistic symbolic aspects of language, specifically the language comprehension and production disruptions seen in aphasia. There will be no mentioning of the associated deficits, apraxia and dysarthria, which may transpire in conjunction with aphasia as well. The reader must realize that apraxia, dysarthria, and aphasia can all occur concurrently. In essence, either apraxia or dysarthria is frequently associated with aphasia; however, there may be isolated incidences of each disorder as well.

Because of the complexity of the original topic, aphasia, the writer chooses to forego any further discussion relating
to apraxia and dysarthria beyond a brief description of each. The rationale for this decision is based on Darley's (1978) hypothesis that apraxia and dysarthria are essentially motor speech disorders. They do not represent linguistic processing disorders per se, but speech production disorders. In a similar manner, this topic remains a controversial issue and the following is simply a brief resume of each disorder.

**Apraxia**

Apraxia refers to a mechanical speech disorder characterized by an inability to voluntarily produce correct positions and movements of the muscles of the larynx, pharynx, tongue, lips, and cheeks to form speech sounds and sequence these sounds in the proper order to make desired words, phrases, or sentences. The apraxic patient uses this same musculature for the vegetative functions of blowing, chewing, swallowing, licking, and even some perseveration of automatic phrases is seen. Darley (1978) explains, "Apraxia is an inability to perform a skilled voluntary act in spite of the fact there is no impairment of muscular control" (p. 179).

A mild apraxic's speech can be intelligible within the context of an utterance; however, the contents contain inconsistent sound substitutions. For instance, the same patient articulates "pencil" at different times as "fensel," "pendel," or "penser." In the more severe cases, the myriad of sound substitutions cause speech to sound like jargon or the
patient will be only able to repeat a syllable or phrase over and over. For example, the observer may hear "kee kee kee," profanities, or four-letter words in every attempted verbalization (Broida, 1979, p. 87; Cohen, 1977, p. 14).

The following description by Darley, Aronson, and Brown (1978) explains the fluency problems of apraxia of speech to be related to an impairment of motor speech programming:

As aphasics speak, they struggle to position their articulators correctly. Their visible and audible struggle to produce correct articulatory postures and to accomplish the necessary postures in forming words. Their articulation is off-target. They often recognize that they are off-target and effortfully try to correct the error. Their errors recur, nonetheless, but they are not always the same; the errors on a series of trials are highly variable. As patients struggle to avoid articulatory errors by careful programming of muscle movements, they modify their rate, stress, and prosodic elements of speech. Thus, the expected acoustic pattern of their speech is altered as well as their articulation (p. 250).

**Dysarthria**

Unlike apraxia, dysarthria is an impairment of speech or voice resulting from weakness, paralysis or incoordination of one or more of the muscles used in these acts. "It is manifested by disorders of respiration, phonation, resonance, articulation, prosody, and generally includes some combinations of these disorders" (Cohen, 1977, p. 15). In the milder forms of dysarthria, the patient can be well understood; yet he sounds slightly different from the way he habitually talked.
In the more extensive cases, dysarthria varies in degree from poorer intelligibility to a total inability to speak a single word. Within the more severe forms, the resultant etiology stems from bilateral subcortical (midbrain or brain stem) traumatic injury or neurogenic disorders as: "cerebellar ataxia, pseudobulbar palsy, bulbar palsy, amyotrophic lateral sclerosis, myasthenia gravis, Parkinson's disease, dystonia, chorea, multiple sclerosis, and cerebral palsy" (Cohen, 1977, p. 15).

Again, the description by Darley (1978) seems to summarize the disorder in an appropriate manner:

Dysarthria is a generic term which embraces a large family of speech problems resulting from a lesion of some motor portion of the central or peripheral nervous systems. Because of the lesion, muscles innervated by the affected nerves function inefficiently, displaying a degree of weakness, slowness of movement, lack of coordination, alteration of muscle tone, or a combination of these. These alterations of function may be manifested in any or all of the basic processes involved in the execution of speech--respiration, phonation, resonance, articulation, and prosody (p. 493).

In conclusion, the intent of the foregoing chapter was to present a general overview of the brain mechanisms underlying speech and language processing and production. At least a cursory understanding of some of these neurological processes and entities in man is necessary in order for the reader to better appreciate the disorder of aphasia.
CHAPTER 2

PSYCHOLINGUISTIC DIMENSIONS OF APHASIA

In the previous section, references pertaining to the gross neurological substrait of speech, language, and communication were detailed without regard for their uniqueness. In order to more fully understand the relationship existing between these concepts, each separate entity deserves individual inspection. The previous statements seem to be clarified through a communication model (Fig. 6) which presents an underlying framework useful when discussing speech, language, and communication: "A source (S) encodes or invents a message (M) which is then conveyed, verbally or nonverbally, along some channel (C) to a receiver (R) who then decodes or reinterprets the message and tries to respond to it using some form of feedback" (Larsen, 1976, p. 7).

Fig. 6. A Model of Communication.
"Human relationships develop and are maintained through communication. Communication may be operationally defined as a transfer of information by means of language, facial expression, posture, gesture, tone of voice or choice of clothing" (Hubbell, 1981, p. 83). From this definition, it may be proposed that communication occurs whenever information is exchanged verbally or nonverbally between humans. Hence, the idea that humans cannot not communicate emerges into discussions. The writer tends to agree that humans cannot not communicate, insofar as the topic concerns those individuals in which language or speech are not disordered. The writer would be more reluctant in agreeing should speech, language, and communication be disordered. Hypothesizing, if an individual's speech and language processes are affected, then to some extent their ability to communicate in a number of instances is disadvantaged and disordered as well. Referring to aphasia, the point being expressed is that many times the aphasic individual's language and feelings cannot be communicated accurately. Therefore, it is being argued that they are not communicating in ways beneficial for themselves, which deserves special consideration beyond the simple statement, humans cannot not communicate. Indeed, as receivers of the aphasic individual's communicative intents, particularly close monitoring and conscious attending is required in order to accurately perceive the messages they are attempting to send. In addition, the receiver's attitude becomes affected by the aphasic's communication difficulties.
"Because language is a means of communication, the use of language cannot be separated from participation in human relationships" (Hubbell, 1981, p. 83). Language becomes further developed by the formulations of Bloom and Lahey (1978). In their definition, they submit, "Language is a code whereby ideas about the world are represented through a conventional system of arbitrary signals for communication" (Bloom and Lahey, 1978, p. 4). Enhancing Bloom and Lahey's conceptualization, our language system embraces nothing simplistic; it is a changeable, rule-governed structure conveyed through a myriad of assorted channels. In essence, the language system may be spoken about as being very flexible, adaptable, and expandable (Lyons, 1981, p. 19).

Relating this definition of language to the disorder of aphasia, it may be said that the system of language, its processes, components, function, and modalities generate a multidimensional language system which underlies the disorder of aphasia. In discussing the multidimensionality of the language system, the question is raised as to whether or not language is a uni- or multidimensional system (Jones, Wepman, 1961; Schuell, Jenkins, 1962). In essence, the careful clinical assessment of patients by Smith (1977) fails to reveal any isolated linguistic deficits among aphasic patients (in Emerick, 1979, p. 266). Typically, aphasic individuals manifest a degree of disorder within all areas of language use. Consequently, it is advantageous to focus
our attention in the direction of these language dimensions and their possible display among the aphasic population.

Before doing so, one final distinction between speech, language, and communication deserves attention. Speech may be defined as the "expression of thoughts and emotions involving the production of meaningful combinations of distinctive speech sounds" (Funk and Wagnalls' Standard American Dictionary, 1977, p. 1287). According to traditional phonological theories, "these distinctive speech sounds are the minimal units, the morphemes, in the sound system of language which become the spoken medium for the transmission of language" (Crystal, 1980, pp. 265, 327).

Language Levels of Complexity

After designating language as a system of learned rules for the construction of innumerable utterances (Lyons, 1980), three distinctions between the levels of language complexity appear to require description:

1. **Automatic level.** This level requires minimal language skill in order to engage in utterances such as poems, prayers, songs, social gestures, swearing, and words like "yes," "no," "I don't know," "I can't," and "You see." Because these well-practiced, highly used utterances seem to assume the state of permanency, they are often able to endure the destruction resulting from cerebral insult. Phatic communications such as verbal greetings, salutations, and social amenities form good examples of these types of

2. Imitative level. At this level the linguistic skills performed are more arduous in comparison to the skills needed at the automatic level. Primarily, imitative language is utilized in order to imitate other individuals. Specifically, this level of language involves the decoding of a verbalized message by a receiver who must encode an identical message. Cognitive processing of imitative messages may or may not be a significant component at this level (Cohen, 1971).

3. Symbolic level. Arriving at the highest level of language complexity, symbolic language skill refers to the creation of non-automatic formulations and novel utterances. This level of language skill is used on a continual basis during the encounters of everyday life. Following cerebral damage, the symbolic language skills are frequently more severely impaired. As Emerick concludes (1979), "Aphasia is a disturbance in the very attribute that is so uniquely human, a person's ability to symbolize" (p. 266). Emerick's quote may require slight interpretation due to any confusion concerning the use of the term "symbolic." Most aphasic patients will not be able to symbolically externalize their language; however, they are quite capable of cognitively constructing ideas for processing. It should be noted, however, that their cognitive constructions may not be at a level commensurate with premorbid skills. They will be at
levels where there is a strong psychosocial need to express the results of their cognitive processes (Cohen, 1971; Emerick, 1979; Schuell et al., 1975).

Language Components: Semantic, Syntactic, and Phonological

As the discussion continues toward a more complete understanding of the psycholinguistic dimensions of aphasia, the topic at hand focuses on the semantic, syntactic and phonological components (domains) which are vital and necessary in order for language to be efficiently used. Each component is addressed separately in order to develop an understanding of the resultant problems likely to be manifested within each area. It also must be emphasized strongly that each component entails a comprehension as well as a production element. In the contents of an article by Cutting and Pisoni (1978), Lashley was quoted as saying, "The processes of comprehension and production have too much in common to depend on wholly different mechanisms" (p. 43).

Expanding on the preceding comment, a description of both elements used in testing may provide the necessary clarifications. For instance, if a desire existed to examine the expressive components of language, the means to determine the quality of this expressive language could possibly include the instrumental input channels of auditory, visual, or even tactile senses to elicit the sample of language to be examined. In this manner, the instrumental input channels are used to elicit the expressive behavior. In a similar
manner, the receptive components require designation of
the instrumental response modalities of speech, writing,
or gesture which the subject may use in order to demonstrate
comprehension (Brown, 1972, p. 14).

Semantic Component Problems

"A breakdown occurs in the relationships between
meaning and language" (Hubbell, 1981, p. 62). An aphasic
individual may demonstrate difficulties in his capacity to
retrieve the accurate words or concepts from the lexicon
previously agreed upon by the culture and utilized by the
individual. The contents of the lexicon still prevail;
evertheless, the aphasic's difficulty lies in the ability
to apply the word correctly. It seems as though the indi-
vidual's thoughts become inhibited by a language barrier;
thereby impairing the representation of ideas through language.
Many times, they will be cognizant that their production does
not coordinate with the intended message and attempts are
made to produce the correct response. It should be noted
that in other cases, they are not aware their response is
incorrect. This discrepancy stems from one of the strangest
characteristics of aphasia, that of inconsistency. The
patient may not have words available with any degree of
consistency from one communication encounter until the next
involvement. Imagine the impediment as the aphasic patient
knows he wants to say "newspaper" and he hears himself utter
"water" or "flabinsop" (Hayes and Greenberg, 1976, p. 7).
In addition, if a listener is not alert, the utterances of an aphasic may appear inappropriate and confused. The fact still remains that the aphasic's confused utterances are not related to mental dysfunction. They are simply unable to recall words in accordance with those that non-aphasics have in their lexicon (Darley, 1978, p. 93). The preceding sentence may appear contradictory due to the fact that the retrieval of the correct words is part of normal mental function. This is true; however, psychological dysfunction or retardation is not part of aphasia in its original form. The transfer of thought processes to their encoded symbolic form seems to be the area of dysfunction.

Syntactic Component Problems

"Syntax may be defined as the rules for the construction of expressions" (Hubbell, 1981, p. 62). Therefore, the aphasic patient's ability to use these syntactical rules may be disordered resulting in statements which are incomplete, improperly patterned, or reversed. They are unable to sequence their language while using the rules of grammar in order to correctly combine the important sentence elements. In the process of maturation, the normal human develops the ability to apply syntactical rules quickly and efficiently usually within conscious effort. It would seem that as language reaches its mature form, the application of syntactical rules becomes an almost automatic function.
The rules for ordering the words of an utterance escape the aphasic. Without these rules available, the aphasic will struggle and gesture and begin and hesitate before they attempt to utter a message such as "I now have to go to the store." The utterance may sound something like, "I...uh...store...uh...go...no." It may be added that the aphasic may continue with a particular word (or demonstrate some form of physical mannerism) after it is no longer appropriate to do so. This type of performance of the same action over and over when it is no longer appropriate is termed perseveration. Pick (1973) illustrates its relationship to temporal lobe lesions explaining, "Perseveration is the multiple repetition of the same word distortion" (p. 64).

Some perseverations of a particular word may last for a day, weeks, months, or even years. In other cases, perseverative language production may not present itself at all or may be evident in only the early stages of the disorder. Some aphasics come up with a repeated word during one period of the day and later a different word emerges as a perseverative manifestation. The example which follows develops from the word "coffee" that the patient seemed to perseverate on early in the day: "I want shave...Where is coffee, I mean, uh-shave, uh-coffee, no--coffee, uh ra or, shave my coffee" (Broida, 1979, p. 47).

The amount of patience required by the clinician is immense as they listen to the patient painfully work his way through these perseverations. If the listener can supply
the word needed, the perseveration may cease or it may develop into a new perseveration or simply continue in its original form.

Phonological Component Problems

Phonology is described "as the science of speech sounds and sound patterns" by Sloat, Taylor, and Hoard (1978, p. 1). According to these authors, each language has a discrete set of sound patterns for use when forming words into an acceptable arrangement. In addition, there are various processes by which sounds are added, deleted, or changed. There are relationships among sound segments, the articulation of speech, and other linguistic elements which are rule bound for the specific language being spoken. These rules then dictate many of the constraints under which words are constructed (Bernthal and Bankson, 1981, p. 48).

Some aphasic patients seem to no longer recognize these rules as an integral part of the production of an utterance. The aphasic may pronounce the word "trabiz" for "table" indicating the trouble involves the incorrect application of phonological rules; yet the word appears within the context of the situation to be an approximation of the semantic content. In other circumstances, the aphasic may speak quite fluently, using long involved sentences consisting entirely of phonologically inaccurate words (nonsense words). These repetitive utterances, completely devoid of content, contain various combinations of nonsense words with little or no meaning being apparent (Hayes and Greenberg, 1976, p. 9).
To varying degrees, with differences being demonstrated by each patient, the semantic, syntactic, and phonological components of language are found to be deficient within all areas of language use. In addition, the literature would appear to support the idea that as the level of language increases, the disruption becomes more apparent. Furthermore, the degree of the cerebral damage as well as the deficits in language production and comprehension will not be in one component area, but they will exist in varying degrees within each area (Schuell, 1966, p. 9).

Subsequently, the preceding section characterized aphasia as a complication in linguistic functioning or within the techniques for utilizing language rules. In many instances, thoughts seem to approach a language barrier where they remain incapable of being transferred into the appropriate language structures. It must be emphasized that these difficulties can be recognized within the area of comprehension as well.

Language-Dependent Behaviors

Basically, language provides a symbol system whereby individuals use the components of a language to express or interpret the messages received. Concurrently, language is hypothesized to be a vehicle for the expression and formulation of thoughts and perceptions. Beyond these, there are certain behaviors which are not generally considered integral parts of the language system. They are activities which utilize this system and may be referred to as language-dependent
behaviors. They are composed of the following: "reading, problem solving, speech, understanding non-verbal signals, creative thinking, gesture, writing, spelling, and language facilitated concept development" (Emerick, 1979, p. 93). Each of these is disordered in varying degrees throughout the aphasic population. In the subsequent pages, a more detailed examination of the particular symptoms within these areas provides a more specific outline of the psycholinguistic dimensions likely to be presented within the aphasic population. A careful consideration of the language-dependent behaviors can provide clinically practical information. Important information is gained from this type of description and becomes more valuable to those who associate with an aphasic than simply discussing the subtypes. In essence, the use of an "adequately descriptive outline furnishes a map of the territory that any reader can decipher" (Darley, 1978, p. 195). In addition, the information provided by these descriptors will establish the length and complexity of the messages an aphasic individual can handle. More specifically, the exact description within the modalities of speaking, reading, writing, listening, and gesture are supplemented through an evaluation of the following: the aphasic individual's need for repetition, cueing, as well as their spontaneous speech, unrecognized errors, and the nature of any self-correction (Brookshire, 1978, p. 26; Darley, 1978, p. 195; Hayes and Greenberg, 1976, p. 10).
In discussing the language of the aphasic individual, certain clarifications need to be expressed. Regardless of etiology, there is an extremely broad range of deviations among aphasic individuals with respect to the extent, location, and degree of cerebral damage (U. S. Dept. of HEW, 1967, p. 51). Furthermore, there is a general consensus within the field regarding the concept of a period of spontaneous recovery; although depending on the author, there are varying time frames in which this improvement takes place. During the post-traumatic period, the brain continues through a healing process which is estimated to last from three to six months. After this period, referred to as the period of spontaneous recovery (Porch, 1973), the language deficits will no longer progress as a result of spontaneous recovery. This notion of a period of spontaneous recovery for language functions was confirmed in a study done by Culton (1969). In addition, he demonstrated the most rapid recovery occurred during the first month following the onset of aphasia. Broida (1979) presents an encouraging statement by saying, "Aphasics tend to reach a plateau and with language stimulation many continue to progress for months and even years after spontaneous recovery has been completed" (pp. 18-19).

In a review of the literature, certain references concerning language-dependent behaviors seem to indicate that speaking and writing are motor abilities and listening and reading are considered sensory abilities. This tendency, according to Schuell (1974), disregards the idea that
perception of visual or auditory images is not an all-or-none sensory event. These learned discriminations involve both sensory and motor operations which can no longer be considered to have a single one-to-one relationship with stimulus directions. "There are space effects, time effects and intraserial effects as well as other problems that must be taken into account" (p. 81).

If we evaluate the functional and integrative structures of the nervous system, it may be inferred that the cerebral cortex and thalamus are given the highest level responsible for the analysis of sensory events. "The cortex and thalamus consult on almost every decision--sensory, motor, or otherwise" (Cotman and McGaugh, 1980, p. 41). Hence, before signals reach the cortex a large amount of analysis and programming occurs throughout the central nervous system. Basically, our behavior is the ultimate result of integration throughout an economical, purposeful, flexible, and reliable nervous system. Therefore, during the assessment of the symptoms of aphasia, we are directed to remember the receptive, integrative, and expressive deficiencies coexist; yet they will not be uniform in degree of impairment. This would add further credence to Schuell et al.'s (1975) hypothesis that the human brain functions as a whole for purposes of processing language.
Vocabulary reduction. The lexicon from which an individual chooses his words becomes restricted following cerebral insult. Not only is the vocabulary reduced, but the aphasic requires an increased amount of time in order to nominate the words desired in their utterances as well. Regarding the inconsistency seen in asphasia, it should be noted that the patient may have a word available one moment and not the next. The reduction within the aphasic individual's vocabulary can generally be expressed as a function of the word's frequency of use. Generally speaking, those words used more regularly appear to have more potential to remain as part of their lexicon or if lost, they then too return more rapidly than those words which are not high use words (Sessler, 1981, p. 209).

A phenomenon viewed among normal individuals illustrates that their "ability to read a word on short exposure or hear it correctly in the midst of noise is a function of the frequency of the word's usage" (Schuell et al., 1975, p. 101). This evidence obtained from non-aphasics demonstrates that the aphasic individual has similar problems only at a more severe level. Because their word selection and nomination problem occur with increased frequency, the cognitive system is operating less efficiently. This would support the idea that language-supported cognitive processes may also suffer impairment, but offers no evidence for other cognitive activities to be reduced.
The reduction in vocabulary is frequently evidenced by word substitutions used by the aphasic individual. They substitute words having no relationship to the intended target word or their word substitutions may correspond directly in meaning to the desired word (Cotman, 1971, p. 4). The descriptive term for this type of behavior is referred to as circumlocution. Primarily, the aphasic will "talk around" the subject in order to possibly retrieve the desired lexical item. The patient may be assisted in this task if they are asked for other words which may be used to convey the intended message. For many aphasics, nouns seem to be the most difficult parts of speech to recall; therefore, if they are asked, "What would the item be used for?", the question may provide the necessary information the aphasic requires in order to produce the desired word.

In summary, examples of vocabulary reduction are difficult to illustrate. Vocabulary reduction was originally referred to as anomia or as the absence of nouns. Subsequent literature supports the concept that the nouns and prepositions are probably the more frequently affected. Conversely, any grammatical element may be affected or the word loss may in fact be random as well. Thus, a patient may fail to generate any word or group of words that he might wish to produce. Likewise, vocabulary retrieval varies from day to day as well as the failure to retrieve the word. It becomes apparent that the patient's need for frequent word substitutions can greatly impair and diminish the quality of the language productions.
Verbal retention span. It is apparent that the aphasic individual's ability to retain relevant information becomes limited as restraints are placed on their storage capacity for verbal material. The reductions frequently result in a loss of specific detail rather than in the general gestalt of the message. Primarily, they demonstrate an inability to accurately process the syntactically relevant information being presented within an utterance due to the fact their memory for grammatical markers, function words, or other sentence components is impaired (Goodglass and Kaplan, 1972, p.7). As established by Clark (1977) these elements play a vital role in language processing models. "They are the basis for most of the proposed syntactic strategies involved in phrasing and assigning a syntactical structure to sentences" (p. 79). Without the availability of the function words, grammatical markers, and other sentence components, the patient's language processing strategies are impoverished. In addition, the loss of verbal fluency interrupts the sequence of cues provided by our sequential verbal habits, thereby diminishing grammatical intactness (Caramazza, Zurif, and Gardner, 1978, p. 667).

In essence, verbal retention span may be illustrated through a categorization presented by Arthur J. La Pointe (1970):

1. Noise Buildup—the aphasic patient is capable of retaining the information presented up to a point. Then, the environmental noise becomes the overriding factor which hampers any further retention of a message;
2. Retention Span Limit--the patient is able to retain a certain amount of information and after that limit is reached, they are unable to retain any further material;

3. Intermittent Reception Problem--the aphasic retains certain segments of information; however, other portions are not retained. Generally, this category, explaining an aphasic's verbal retention span, is most often used. It has been submitted that the complexity of the message and the extent of neural dysfunction represent the underlying factors of this kind of verbal retention problem.

**Auditory comprehension.** Throughout the aphasic population, in varying forms and degrees of impairment, there is a disability within the area of auditory comprehension.

Schuell et al. (1975) points out that "our language is dependent upon discrimination, recognition, and recall of learned auditory patterns and feedback processes" (p. 5). Generally, the impairment which transpires is not complete. This can provide for occasional recognition of a few words, although the aphasic individual will be unable to comprehend the entire verbal message. In addition, a problem exists in that those people associating with aphasic patients are unable to determine the extent to which they are able to decode the auditory elements. We must be cognizant of the fact that the patient may understand much of what is being expressed, no matter how reduced their comprehension abilities are perceived. If we unintentionally make references relating to the patient, these expressions may be potentially damaging to the patient.
The pervasive character of this aphasic symptom is continually the source of limitation and frustration. The reason is that numerous directions, digit series, listings, and lengthy messages rely heavily on complete auditory comprehension (Goodglass and Kaplan, 1972, p. 9). Noteworthy, even though the language symbols cannot be understood by the patient, they may reliably draw meaning from gestures, facial expressions, and the overall emotion of the speaker's approach. It is worthwhile to remember that these non-verbal cues may represent the primary avenue in which meaning comes across to the aphasic patient. Concluding, "Any impairment in auditory comprehension is unrelated to hearing loss or lack of intelligence" (U. S. Dept. of HEW, 1979, p. 8). Conversely, the degree and pattern of perceptual impairment may be highly correlated with the degree of language comprehension impairment.

It would seem necessary to emphasize once again that no two people are alike in either their language abilities or disabilities. This statement became quite apparent as the variety of symptomological designation developed to describe aphasia were reviewed. In order to have a better understanding of aphasia, further observations and exploration of language processing deficits beyond the background presented in the next few pages is necessary. Because certain symptoms seem to occur with greater regularity, it will be these symptoms upon which the remainder of the chapter is focused.
Paraphasia. Paraphasia represents one of the more prominent, unique, and theoretically challenging changes which develops among aphasic individuals. It is characteristically found in the patient who speaks fluently and may be described in the following production by an individual when asked to identify a picture of a rose: "It's a roolin, rooner, no...now let's see (automatic utterance), it's a raze...runner?" From another example in reference to identifying a book, the reply went something like, "Boos...boat, bug, boot, but, bot...I don't know" (Burns and Canter, 1977).

A number of authors divide paraphasia into three categories (Buckingham and Rekart, 1979; Goodglass and Bloomstein, 1974; Goodglass and Kaplan, 1972), which are as follows:

1. Phonemic paraphasia. "An aphasic's spoken transformation, in which phonemes are the transformed units, that is to say, the addition, deletion, replacement, or displacement of phonemes, are the key factors" (Burns and Canter, 1977, p. 492). The phonemic confusion is present during good agility of production as illustrated in these examples where the patient was asked to respond to a stimulus picture (when the stimulus was a bag): "Bushel, no...bas, no, not a basket...It's a funny word, isn't it?...Bas?" (When the stimulus picture was a vat) "The vat thinks, oh, that was a blowy...the vat thinks, leaks." (When the stimulus picture was a lime) "Lions are sobbers, subbers, sours, sums...limon are...no, limes are sours."
2. Semantic paraphasia. "The unintended word is inadvertently used in place of another word usually within the connotative sphere of the intended word" (Goodglass and Kaplan, 1972, p. 8). Further analysis of this type of paraphasia shows an interesting substitution. At times, the substituted word has some relationship to the word produced in error. The patient may say "chair" for "table" or "bread" for "butter." Then, the words may have an opposite meaning, such as when the aphasic says, "yes" for "no" or "fast" for "slow" (Goodglass and Blumstein, 1973, p. 62). The following sample illustrates a patient's semantic organization which has broken down in certain semantic fields. These are taken from a sample obtained by Buckingham, Jr. and Rehart (1979). The utterance samples are from spontaneous speech with the target word indicated in parentheses.

I. Parts of the Body

A. Eyes/Ears

(1) "I talked with the lady that made the ear side (hearing aid)...for my eyes (ears)."

(2) "One thing I would like to do...is to get a different--uh-- of my eye (ear) on this side." (Patient points to her left ear.)

(3) "I would be able to hear you better with that than the eye (ear) . . . ." (p. 200)

3. Random paraphasia. This type of syllable substitution appears capricious in that sentence segments are unrelated; they lead nowhere; the speech is rambling and
circumlocutory (Goodglass and Blumstein, 1973, p. 7). Hopefully, the example to follow will successfully differentiate this category of aphasia:

A gentleman was talking with his therapist and he looked at her emerald saying, "That's a nice shiny shan, I mean a shiny thing, a nice garment, a nice emeret...no, that's a damn dumb thing and I don't know why you have to have it anyhow."

From these examples regarding the kinds of paraphasic errors, it should be noted that "all three categories frequently exist in a single patient (Goodglass and Blumstein, 1973, p. 8). The patient is generally unaware of the meaningless utterances produced and frequently becomes disturbed because listeners do not respond appropriately to what he feels was an accurate utterance. Basically, the aphasic individual is confronted with a word-finding lapse, which is resolved when the patient as they audibly search for the word from among various words associated in meaning, sound configuration, or both.

In summation, the patient's speech may consist of an inappropriate selection of words in sentences that are otherwise well formed, the words selected may be disfigured sound configurations, or there can be capriciously made syllable substitutions as well. Undoubtedly, whatever the form, there is likely to be a serious breakdown in communication between the speaker and receiver.
Jargon. "In neurology, jargon is defined as utterances that have no meaning" (Broida, 1979, p. 92). The definition extends further to include the fact that the patient speaks without halts, hesitations, or corrections; their speech is slightly more rapid than usual with good rhythm and intonation; and they generally feel their communicative intents are satisfactory. Placing this in the context of our communication model, the aphasic behaves as if neither he nor the receiver has any difficulty encoding or decoding the message. Unfortunately, for the receiver the decoding is nearly impossible.

Jargon, commonly seen in most aphasics, is characterized by varied degrees of impairment, although the absence of meaning within all verbalizations is due mainly to the patient's inability to use words in conventional ways. Even though the verbalizations are for the most part meaningless, the rhythm and prosody of the speaker's native language remains intact. This important feature of jargon is thought to be a product of the non-language dominant hemisphere. Unfortunately, correct use of rhythm and prosody becomes troublesome for the listening audience. The reason is that when the utterance is first heard it appears to resemble correctly spoken language; however, through more careful listening, it is evident that the utterance lacks linguistic content.
The severest form consists of sounds which may be recognizable as representative of a speaker's language; however, there are no recognizable words or meaning. "Others retain enough formatives such as articles, pronouns, and conjunctions to suggest a syntactical pattern, but in place of the nouns, verbs and modifiers are paraphasias" (Goodglass and Blumstein, 1973, p. 302). Finally, the patient's speech consists of entirely recognizable words although the relationship among the words breaks the conventional rules of normal language. A few examples should conclude the discussion of this symptom of aphasia (from Goodglass and Blumstein, 1973):

1. "Then two o'clock is it already three goes along."
2. "I might make be seeing but make me say the wrong thing."
3. "I went to this school too in in at fear high school."
4. "They had a big long eighty eighty spin." (p. 306)
5. "What a buffalong."

Reading. Since a majority of aphasic individuals demonstrate impairments within their verbal attention span and vocabulary, a part of their reading problem may be related to these deficits. Many aphasics show some reduction within the modality of reading for other reasons as well. Depending on their individual pattern of impairment, the aphasic patient tends to make errors similar to those made during speaking or listening. In addition, the frequency with which these errors occur can be a function of the patient's degree of familiarity with a sequence of words.
(Schuell, 1974, p. 93). Generally, reading rate decreases as a result of the aphasic individual's difficulty in retaining and integrating what they read. Further difficulties stem from the concomitant problems associated with spatial disorientation or visual processing involvement. For instance, the patient may not be able to keep their eyes fixed on the line or page they are reading or they may find it equally difficult to keep their place. The more severe aphasic may be completely unable to visually discriminate between letters or words. Of those who are less impaired, "They appear to confuse letters and words having similar configurations (i.e., in lower case print, they confuse /w/ and /m/; /p/, /b/ and /d/; and /p/ and /q/" (Broida, 1979, p. 60). Finally, the patient may not be able to translate from cursive to printed material. This important concept should be observed carefully by the clinician before any presentation includes stimulus materials employing differently formed words.

Subsequently, the variety of impairments which may or may not be observed with some aphasics might be characterized as follows:

1. The aphasic may comprehend single words, but not complete sentences or they seem to be able to graph the meaning within single sentences, yet not paragraphs;

2. Frequently, the aphasic individual understands the main issue comprising a newspaper or journal article, although they appear to lose the more specific features
and underlying points contained within the article;

3. Many aphasics may omit, add, or substitute words into the passages they are reading, thereby creating a distortion to the meaning of the passage;

4. Some patients will be capable of reading aloud; however, their silent reading is impaired. In the same manner, certain aphasics retain the ability to read silently, yet not aloud;

5. Finally, the more severely impaired patient may be unable to recognize letters which in themselves have lost any meaning (Cohen, 1971; Schuell, 1974).

Writing. The ability to write is among the highest levels of symbolic processing; therefore, within the aphasic population, it can be the most profound language dependent behavioral disability. Generally, writing skill is more severely impaired during the early period following onset of aphasia. Primarily, the patient writes in the same manner as he talks, although the writing errors frequently occur to a greater degree. Patients who demonstrate improvements in their verbal skills may not show equal gains in their writing skills. For some patients, there are continued writing difficulties (Broida, 1979, p. 69).

A main issue in writing stems from the fact that the aphasic's vocabulary is reduced, "thus decreasing the recall of their lexical items and disadvantaging their writing ability" (Cohen, 1971, p. 12). Since a patient is unable to think of an appropriate lexical choice, they usually
are not able to write this item either. At the same time, in certain types of aphasic, the patient may be able to write in a limited capacity even though the language produced is unintelligible.

Another facet of writing deficiency concerns the decline seen in the aphasic individual's spelling skill and accuracy. In their writing attempts, some aphasics may find difficulties in the formation of individual words' symbols and they tend to add, substitute, or omit various portions of words and sentences. Primarily, "when a patient is unable to retrieve the appropriate symbol, a random scribbling is observed" (Hayes and Greenberg, 1976, p. 43). Then too, in some patients and not others, their writing is further impoverished by visual and spatial processing deficiencies.

Subsequently, the impairments recapped below illustrate the difficulties an aphasic patient may have to contend with while attempting to produce a finished product:

1. Writing problems are frequently profound;

2. In attempting to write sentences, the difficulty arises in finding a lexical item, spelling it, and producing the desired end product using integrated grammar and syntax (i.e., the aphasic patient may be attempting to write, "I want to see a movie," The results: "He seesh mofe" or "alfus arg wunch."

3. Their attempts resulting in a jumbled collection of meaningless symbols may appear quite accurate as far as the aphasic's perception is concerned;

4. Some aphasics will not attempt to write;

5. Many are unable to write the correct symbols;

6. Others are able to copy from a printed or written example;
7. Paralysis of the upper limb is frequently a concurrent problem adding to the existing linguistic deficits or forcing use of the non-favored hand, thereby further complicating the problem (Broida, 1979, p. 68; Schuell, 1974, p. 97).

Numbers, arithmetic, and time. The last area regarding language dependent behaviors presents some interesting concepts. The reason becomes apparent when the reader considers that arithmetic is a form of language consisting of highly symbolic numbers. Numbers represent concepts; therefore, it is reasonable to assume there is a myriad of variations expressed in the aphasic individual's use of numbers, arithmetic, and time. Hence, these difficulties are best described in the statements below:

1. The patient may not have a concept of what counting means; however, he will be able to count by rote. If the patient were asked to count a group of items, they would be unable to deal with that concept;

2. In attempting to count a series of items, the patient may be able to count the items necessary; but not quantify the final amount reached. Hence, the total quantity could not be named;

3. There are some patients who are able to name numbers correctly when reading and they retain number concepts; yet they are unable to perform calculations;

4. Generally, patients are inconsistent and consequently unreliable in their adequacy with numbers. The following example illustrates this point: a patient may write a check for $300.00 instead of $30.00; they may order 4000 items instead of 400; patients use two cups of flour when the recipe calls for three; they may make an appointment for 1:30 when they mean 3:30; and they expect you home in two hours when you said five hours (Broida, 1979, p. 78; Cohen, 1971, p. 13).
In conclusion, hopefully the preceding section regarding the psycholinguistic dimension of aphasia has established a basic orientation concerning the language deficits which may be observed with the aphasic individual. The challenge is to use these descriptions only as a starting point from which to explore and investigate the varying language deficiencies presented by a particular aphasic individual.
CHAPTER 3

BEHAVIORAL, PSYCHOLOGICAL AND PHYSICAL ABERRATIONS

The treasure of communication, often only slightly considered and taken for granted, takes on added meaning and regard when suddenly it is reduced or even lost as a result of cerebral damage. Humans generally have a better opportunity to remain at ease in their association with other individuals when speech and language functions remain intact. Upon sudden loss of the ability to communicate, as in the case of the aphasic patient, the psychological and behavioral reactions to follow are natural consequences which correlate with the amount of reduction and loss each person experiences. In one respect, the aphasic will grieve over his loss. "Grief is a complex progression involving many emotions and attempts to adjust and cope with the loss" (Tanner, 1980, p.20). Therefore, an understanding of the grieving process has application for the psychological and behavioral aberrations seen in the aphasic population. Undoubtedly, the aphasic patient's reaction to their reduction or loss of communicative skills, physical abilities, and a comfortable routing becomes a major contributor of the psychological and behavioral reactions demonstrated by the patient.
In the same manner, it will be these reactions which determine the course and objectives on which to establish a more profitable rehabilitation program.

Perception of the Loss

The understanding of the loss, especially the human response to it, presents major implications to the clinician assigned an aphasic client. Schneider (1974) contributes to this knowledge by proposing that loss is characterized into five dimensions. The scope of this paper does not warrant extensive discussions of each dimension; however, acknowledging them seems appropriate. Hence, the five dimensions as set down by Schneider are: 1) loss of significant person, 2) loss of some aspect of self, 3) loss of external objects, 4) developmental loss, and 5) loss of predictability of usual reinforcements and rewards (from Tanner, 1980, p. 917).

Focusing for a moment on the fifth dimension, Tanner (1980) submitted a ponderous statement in reference to aphasia. He suggests, "Aphasia is perhaps the most significant disorder seen by the speech clinician which results from loss in this dimension." His remark brings to mind the devastation an aphasic patient experiences. "In aphasia, loss is not limited to verbal expression; aphasics also have difficulty in reasoning, memory, and comprehension" (p. 919).

With these points in mind, it becomes a challenge for developing the sensitivity necessary to deal with the aphasic patient's psychological and behavioral reactions.
numerous changes, varying from day to day, that the patient must confront. The recovery process is a constant struggle, consisting of acceptance of seemingly unyielding deficits and frustration in accomplishing improvements over these impairments. In every case, the recovery process is overshadowed and threatened by a myriad of patient reactions to daily situations which confront them. It will be the speech clinician, who must be a sensitive receiving instrument and respond in a purposeful and meaningful way in order to promote the patient's acceptance of loss of predictability.

Other authors have developed differing views concerning the psychological stages undergone by the patient as a result of the occurrence of brain injury. In making reference to the patient's acceptance of the loss, a description of the various stages contained in the process according to Kubler-Ross (1969) seems to adequately categorize the process. A denial stage emerges in order for some aphasics to accept and adapt to their new circumstances. Kubler-Ross states, "Denial functions as a buffer after unexpected, shocking news, allows the patient to collect himself and given time, mobilize the less radical defenses of anger, bargaining, and depression" (p. 39). It is important to recognize these defense mechanisms as well as others: suppression, rationalization, displacement, projection, and repression. These appear in various cases and should they extend beyond a person's initial adjustment period may call for experienced psychological intervention.
Aphasic Emotional Reactions

It is a difficult task to describe the "typical" reactions of aphasic patients to their deficits and disabilities. Put another way, there may be as many aphasic reactions as there are aphasics. Each person's premorbid mental attitudes and desires will contribute immensely to the success and failure experienced during the postmorbid period (U. S. Dept. of HEW, 1967, p. 3). Ordinarily, personalities are portrayed by the descriptive adjectives: introvert, extrovert with either a pessimistic or optimistic attitude. So, the outgoing optimistic individual has a strong basis from which to manage the devastating changes and loss received from a cerebral insult. The premorbid personality hinders or promotes the patient's chances for a more complete rehabilitation (Sessler, 1981, p. 221). As Moss (1972) reiterates, "The certain type of personality traits existing before the cerebral damage may well influence the patient's attitudes" (p. 37). Therefore, the classical textbook definitions and the adequate description of reactions will vary with each aphasic patient.

The sudden and dramatic onset of aphasia effectively modifies all facets of a normal functioning individual's life, leaving some with residual impairments in the areas of physical well-being, vocational opportunities, home management, and communication (U. S. Dept. of HEW, 1967, p. 1). Initially, the postmorbid period is marked by episodes which instill frustration and fear in both the aphasic
and those with whom a close association exists. The impairment may diminish the individual's awareness of the happenings within the immediate environment. "Brain damage knocks the patient's awareness awry" (U. S. Dept. of HEW, 1967, p. 37), thereby causing the pervasive feeling of fear to emerge. The aphasic frequently becomes chronically apprehensive, developing an exaggerated concern for environmental expectations. Many of these qualities were observed by Eric Hodgins (1967), an aphasic individual himself. He maintains, "Aphasics are concerned about any responses they will need to construct; whereas in the time prior to the cerebral damage there was significantly less reason for agonizing about environmental expectations and responses" (p. 37). Generally, the aphasic cannot appreciate fully what they encounter which leaves them fearful in the face of their future encounters with other humans.

The aphasic reaction might often be referred to as catastrophic. This term grew out of the monograph by Goldstein (1942) where he described a catastrophic reaction as "a dazed look, agitation, anxiousness, fumbling, irregular pulse, sullen, evasive, and a temper" (p. 70). The problem which arises concerns the use of the label catastrophic, in reference to all emotional outbursts seen in aphasic patients. Schuell (1972) reports there have been relatively few catastrophic reactions during the many years she has worked with aphasics (p. 278). Because every human can react in a catastrophic manner when they are confronted with a frustrating
situation, there is a need to exercise care in the use of this term when describing the emotional behavior of aphasic individuals. The label develops a negative attitude in which biases and opinions concerning aphasic individuals can hinder their interactions and relationships. It is true that many aphasics behave in ways "totally incomprehensible to the non-aphasic" (Weinhouse, 1981, p. 34); however, by simply being aware of the signs of tension and incipient stress often helps the clinician or family member alleviate the changes for any emotional outbursts characteristic of the catastrophic reaction described by Goldstein.

**Etiological Constituents**

The characteristics found in human emotions are what guide, restrain, and protect each individual. For the aphasic, a marked loss in emotional control arises out of two etiological constituents: the cerebral damage and the aphasic's endeavors to regain "ego integrity while struggling with the inadequacy of a non-integrated cerebral structure" (Wepman, 1951, p. 32).

"Modifications of personality and intellect may be attributed to cerebral insult. They may be considered aspects of the syndrome of organicity" (U. S. Dept. of HEW, 1967, p. 38). Studies have demonstrated the frontal cortex to be the vital controller in personality. The frontal lobe's structural features, its great size and colossal amount of direct connections with other central nervous system parts,
are essential for mankind's unique attributes of personality, emotions, and stable purposeful behavior. Its largest portion appears involved in "regulating emotional tone, assigning priorities to bodily and environmental demands and stabilizing programs for meeting short term and long range goals" (Cotman and McGaugh, 1980, p. 781).

The landmark investigations providing valuable insights concerning the controls exercised by this region were conducted by numerous individuals including Lashley, Freeman and Watts, and Penfield. The behavioral aberrations noted, comparable in all cases, were nearly all non-language. Major deficits noted by Freeman and Watts (1942) eloquently attest to the importance of this cerebral region. In prefrontal lobectomy patients, their behavior was portrayed as: "A progression toward a disoriented apathetic state where there is little initiative or concern about self and world, the individual is indifferent to society, has a poor attention span, lacks interest in recovery, with delays in concrete thinking, judgment, and planning ability" (p. 208).

The frontal cortex is generally involved in aphasics to the extent of seriously interfering with their ability to "respond appropriately in emotional and social spheres" (Cotman and McGaugh, 1980, p. 721). For the uninformed, the emotions once held in "abeyance which flare in behavior seldom seen before the cerebral damage" (U. S. Dept. of HEW, 1979, p. 8) may appear unreasonable. It is through the knowledge of the frontal cortex that any misunderstandings are
held to a minimum. When intellectually functioning individuals become lewd, profane, apathetic, and untidy, our understanding of the etiology, cerebral damage, brings into perspective behavioral aberrations likely to transpire. An aphasic's demeanor may change drastically from day to day, even from moment to moment. If we disregard the behaviors demonstrated by mentally ill patients, there is almost "no other disability where as much inconsistency of behavior is to be noted" (Wepman, 1951, p. 32). In many instances, an aphasic's actions will seem to contradict the present situation.

**Emotional Lability**

Emotional lability is a reaction which predominates as one of the more devastating alterations encountered usually following cerebral damage. With the aphasic's threshold of emotional reaction lowered, crying and laughing outbursts frequently prevail, usually without any relationship to environmental events. In fact, some patients may laugh and cry simultaneously. In this context, the emotional lability is organic in etiology. Crying, the more frequently seen behavior, is quite indicative of emotional lability. Generally before complete reduction of edema, the return of homeostatis to the brain, and the regaining of the natural state of metabolism, the excessive crying is caused by the cerebral damage and indirectly connected to the depression experienced by the patient. In the case of organic emotional
lability, the emotional behavior may be interrupted by diverting the patient's attention through the presentation of new questions or by calling out their name. On the other hand, if the patient is truly depressed, these tactics will not generally interrupt the tears. "There is much to be said for interruption of organic emotional lability" (American Heart Association, 1981, p. 28), since the emotional expression is due to a cerebral injury problem of which the patient has little control. The interruption can prevent embarrassing and fatiguing displays by the aphasic patient.

Besides the organic reason for the emotional lability, another contributor stems from the communication impairment. Communication serves as a "release valve" for emotions arising in relation to something the aphasic has deep feelings or acute anxiety about. Van Riper (1963) describes the numerous facets of communication to include those which humans rely on for the expression of their troubles and feelings (p. 5). Ultimately, emotions normally liberated through verbal behavior multiply, presenting themselves during insignificant events. Therefore, as physiological and psychological conditions improve, the emotional lability is likely to diminish as well (Schuell et al., 1975; Sessler, 1981).
Anger and Aggression

In addition to the emotional lability of crying and laughing, anger and aggression are frequently displaced and randomly projected into the environment by the patient to those individuals providing therapy, care, and support. "Anger and aggression are the consequences of the loss experienced by the aphasic" (Tanner, 1981, p. 921). Their actions and words won't represent a cause and effect relationship. The emotional difficulties they are battling are attributable to the primary etiological factor, brain damage. Because they are communicatively handicapped, the aphasic's expression of anger and aggression is often seen in their lack of cooperation, bizarre behavior, or in a physical display of emotional feelings.

Depression

Finally, depression, a natural reaction to the calamity, occurs throughout the aphasic population. "Depression is a dynamic phenomenon. The patient is not happy or sad, the patient is depressed with associated psychological reactions" (Tanner, 1981, p. 921). As long as this state does not continue for extended periods of time, it is not considered pathological, unhealthy, or abnormal. Many consider anger and aggression healthy reactions; however, such indicators as difficulty maintaining attention, reduced motivation, and lack of response need to be monitored closely (Sessler, 1981; pp. 205-220; Hayes and Greenberg, 1976, p. 18; American
Heart Association, 1979, pamphlet).

Is It Mental Deterioration?

Subsequently, aphasics display signs and symptoms along similar lines of individuals suffering from a progressive mental deterioration. They may sound and behave peculiarly; therefore, people tend to speculate the aphasic may have "lost his mind" (Hayes and Greenberg, 1976, p. 2; Licht, 1975, pp. 95-96; U. S. Dept. of HEW, 1967, pp. 36-39). There are some aphasic symptoms which seem to parallel schizophrenia or psychosis; however, they are not actually representing these problems. Cognitive functioning relies on an integration of somatic, visceral, auditory, visual, and olfactory stimuli, along with correlation among past experiences and sensory impressions (Licht, 1975, p. 94). Although sometimes difficult to determine, a distinction between mental deterioration and aphasia exists. The aphasic impairment, orderly, systematic and limited to the components within central language processes is also "disproportionate to their overall level of cognitive functioning." Conversely, "demented individuals have comparable difficulty within all cognitive functions" (Darley, 1978, p. 93).

Summarizing, aphasics appropriately interact with others and their behavioral patterns distinguish them from patients whose illogical responses are due to disorganized thinking associated with diffuse brain disease. In the same manner, the behavioral and psychological aberrations mentioned by no
means incorporate the spectrum of reactions potentially seen in the aphasic patient. It cannot be stated strongly enough that there are as many aphasic reactions as there are aphasics.

Physical Aberrations

The discussion thus far presents a general orientation encompassing the major behavioral and psychological aberrations generally observed among aphasic individuals. Besides these aberrations, certain physical problems often accompany aphasia in varying degrees. Therefore, following cerebral insult, "the aphasic patient will have a multidimensional problem with which to contend" (Hayes and Greenberg, 1976, p. 15).

A high percentage of aphasic patients experience hemiplegia (unilateral paralysis) or hemiparesis (unilateral muscle weakness) involving the extremities on the contralateral side of the body to the cerebral damage. The main contributing factor to this unilateral type damage stems from the fact that muscles on one side of the body are controlled by the opposite cerebral hemisphere. During cerebral vascular accidents, primarily only one hemisphere's blood supply becomes interrupted. In this manner, damage to the left cerebral hemisphere may result in either paralysis or muscle weakness to the arm, leg, or to both extremities. For many patients, the improvement outlook is favorable, especially due to the administration of physical therapy.
It would be misleading to assume total rehabilitation of the involved extremities. The truth to the situation is that numerous patients regain functional ambulation of the leg. Conversely, the rehabilitation of the paralyzed or weakened arm is less often accomplished, thereby leaving it with more involvement. In general, there is only a minority of the cases where no resolution develops permitting functional ambulation and gross use of the involved arm and hand (Fowler and Fordyce, 1974; American Heart Association, 1969).

Besides the involvement of the extremities on one side of the body, muscle groups responsible for the control of the right side of the face, mouth, and tongue may be affected as well. This paralysis or weakness results in drooling, slurring of speech, and the involved side of the face will droop. If the cerebral damage results from a traumatic injury causing more widespread damage within both hemispheres, then the paralysis may be bilaterally affecting arms, legs and face.

In addition to any hemiplegia or hemiparesis, the aphasic individual can experience severe headaches and a susceptibility to convulsions exists as well. In the majority of individuals who experience convulsions, anti-convulsant medication seems to control the effects quite well. In a similar manner, the use of medications may have side effects which can interfere with therapy and communication encounters in general.
Finally, visual impairment may be most frequently described as a reduction or loss in the patient's peripheral vision. Although the visual capacities of the eye are not completely reduced, the patient has difficulties in seeing objects located to their left or right. If this visual field impairment, termed hemianopsia, occurs deep within the optic pathway of the left cerebral hemisphere, the impairment impedes the impulse transmission of the left half of the retina, resulting in the loss of right field vision. As physical stabilization returns, this condition may still remain, thereby requiring compensatory head and eye movement in order to overcome the visual field limitation (American Heart Association, 1979; U. S. Dept. of HEW, 1967).

Concluding with a statement by Boone (1967), he expresses, "that the locus of a lesion will have a more obvious relationship to the amount and type of physical problems; whereas the site and extent of cerebral lesion may not have a direct relationship to the type of severity of aphasia" (p. 18).

In summary, although the aim of the preceding chapter was to establish the various components encompassing the aphasic's behavioral, psychological, and physical aberrations, there is an underlying message which needs to be stressed. We are reminded of the immense amount of individuality expressed among aphasic patients. Reiterating again, there are as many forms of aphasia as there are aphasics. Briefly,
the environmental forces, patient and family personalities, as well as their premorbid state will all play vital roles in the aphasic individual's progress and rehabilitation. Each contributing factor deserves attention and consideration as plans are established for the aphasic patient's therapy.
CHAPTER 4

A REVIEW OF TWO APHASIC INDIVIDUALS

As this general orientation regarding the nature and extent of aphasia nears completion, the writer intends to demonstrate various aspects presented in the preceding chapters by reviewing the cases of two aphasic individuals. The material demonstrated through the first case stems from an interview conducted by the writer with a patient who has demonstrated an exceptionally good recovery. The gentleman, L.G., discusses the various aspects surrounding his cerebral vascular accident, his rehabilitation, feelings and the state of his condition in the fifth year since the accident occurred. The main reason for reviewing L.G.'s situation stems from the fact that his rehabilitation illustrates a remarkably successful comeback, one which many people felt would be impossible for him to accomplish. In addition, the quality degree of educational value is established through this interview as well.

Finally, the writer emphasizes the valuable experience obtained during the direct contact with L.G. It was through this interview that many of the aphasic symptomatology became more highly appreciated and apparent. Likewise, a statement by L.G. seems to reflect the devastation felt
by a person when they experience aphasia. L.G. said, "I
would never wish the condition of aphasia on my worst
enemy." That statement expresses quite adequately the very
essence of the anxiety suffered by the aphasic patient.

In the review compiled on R.L., the second patient,
an interesting aspect underlying aphasic rehabilitation
seems to surface. After examining the case history on R.L.,
one overriding factor arises. This factor stems from the
debilitating aspects that occur as a result of a very stress-
filled home environment and how this environment seems to
affect the aphasic's speech. The stress which developed
through her particular circumstances was one of the under-
lying factors contributing to R.L.'s speech processing
difficulties. This factor is discussed in an early session
with her speech clinician. The session's contents are
duplicated in order that the types of aphasic symptomatology
may be seen as well as to illustrate the clinician's pro-
cedures aimed at helping R.L. become more relaxed, thereby
hopefully establishing a more optimum condition for therapy
to occur.
Interview With LG

Much of the case history concerning LG is discussed during the interview; however, the following background information is necessary in order to accurately discuss LG's situation and struggle to overcome aphasia. In the writer's contact with one of LG's speech clinicians, the clinician illustrated LG's main speech and language problems to include: severe word-finding difficulties (anomic aphasia), jargon and paraphasia were present in his speech during the early stages, LG's spelling was poor, a hand transfer was made in writing because the use of his right arm failed to return (unfortunately, the functional use of the arm will never return), and in general the damage resulting from the cerebral vascular accident was to a greater extent in LG's frontal lobe with some damage occurring in the posterior aspects of the left cerebral hemisphere.

The speech clinician attributed LG's exceptional recovery to be due to four factors. Considering the fact that LG had two separate incidences of stroke with resulting severe neurological damage, the patient has demonstrated remarkable improvements. Secondly, LG is a highly motivated individual and he did not accept a preceding evaluation which stated he would never be more than a vegetable. In addition, LG received extensive therapy twice daily from two speech clinicians. He was required to continually practice the lessons established by the clinicians in various kinds of
homework as well as using a special audio recording device. Finally, one last remark by the clinician seems to demonstrate how LG's personal friends contributed to the success he experienced. Many of LG's personal acquaintances inquired how they might be able to help LG. This beneficial factor establishes the necessity of the patient's total environment and how every person the patient comes in contact with is a significant component of the rehabilitation process.

The interview, conducted on February 20, 1982 in LG's office, is transcribed on the following pages. It would appear from direct transcription that considerable problem remains with the patient's language skills. It is unfortunate, however, that the reader will be unable to fully appreciate the effective communication skills LG is currently demonstrating. His use of the phatic components of communication are used with great skill as well as the prosodic features of language. The disruptions apparent in the transcript of the interview are not as detrimental to communication as they would appear in written form.
MM: Would you discuss your activities prior to your stroke?

LG: Well, before I had my stroke, I was employed here of course, uh and uh and it's much the same as after the stroke; but, uh of course uh, I used to hunt a great deal and uh and uh boat a lot and so on. Uh, now my activities have gone down somewhat because of the failure of my right arm to get better. Uh, but uh, I think uh that I uh well, let me put it this way, I drove my trailer to Vancouver, British Columbia and back uh 3 years ago. You see, this stroke happened 5 years ago. Well, it's almost 5 years ago March 21, that's when it happened and uh, I uh used to get along on 5 or 6 hours a night and now I have to have 8 or 9 (laughing). You know what I mean and so on. Uh, I, I, I think I was going on, oh, nervous energy too much.

MM: The time before the stroke?

LG: Yeah, and uh uh but I had had high blood pressure uh before my stroke, but, but it was under control, I thought.

MM: Did you take medication for your high blood pressure?

LG: Yeah, mm, hum, uh but I wasn't on it at the time of the stroke, you see. In other words, I uh contrary to what I know now, I'm on high blood pressure for the rest of my life, you see.

MM: And at that time they didn't feel you had to take it every single day?

LG: Yeah, yeah. I don't know what happened. All I can remember of course, we skied 14 miles the day before and uh.

MM: Did you do that often, ski that far?

LG: Oh yeah, we were at Essex uh the day before it happened. That's between West Glacier and East Glacier. Up on the hill, you see and uh, we were part of a group and we skied from the very summit of the pass, uh down along the highway to the road. Uh, maybe it was not 14 miles maybe it was several miles. I would guess maybe that's it. Anyway, it was a long way and uh then, uh we came home about 10:30 uh at night and put the skis away and so on and uh and got into bed. About 3:30 in the morning I woke up and I couldn't talk and my right side was paralyzed all the way down including my leg and so on.
(It should be of interest to the reader to know that one of those times when stroke occurs most frequently is after a patient has been engaged in heavy activity and then goes into a resting state.)

MM: When you tried to talk simply nothing came out?

LG: I couldn't say a word.

MM: Then how did you wake your wife?

LG: Yeah, I shook her.

MM: What did she do?

LG: Well, of course, she called the doctor right away. She said, it looked like a stroke and uh then, uh, the doctor was uh across the park from me, just two blocks away. He got right over there and I think maybe he saved my life, you see.

MM: Because he got there so quickly?

LG: He was so close and I went to the hospital and uh spent two days in intensive care, uh never saying a word of course. Then, when I got out on Thursday morning, I had another stroke and that did it as far as paralyzing my arm and so on, you see what I mean?

MM: So, if you wouldn't have had the second stroke, the damage maybe wouldn't have been so extensive and you would have regained partial use of your arm?

LG: Yeah, right.

MM: In reference to people talking to you, could you understand what they were saying?

LG: Every word.

MM: How did they communicate with you? How did they treat you?

LG: Oh fine. They could uh, they uh well, they know by my reactions that I was understanding them, you see what I mean?

MM: By your eyes, facial movements, and other non-verbal signs?

LG: Yeah.

MM: The reason for my question is that I was curious if they appeared to treat you as though maybe because you couldn't talk that you would not be able to understand them either.
LG: I know, but aphasia doesn't work that way. You understand everything uh is going around you, you know, but can't respond.

MM: Then, how did that make you respond? What did you do to try to make yourself talk?

LG: Well (laughing together), I suppose that I could say "yes" and "no" about after uh a week's time or something like that. The therapist out at the hospital, she said I had oh about 13 words after a week's time. She counted them.

MM: Do you remember what any of these other words were?

LG: Not really, you know, maybe door, window, and things like that.

MM: Words that were very common to you?

LG: Yeah, and then of course, I went for therapy twice daily.

MM: Starting immediately?

LG: Well actually--it's a little foggy now--but I think after the first week or something like that. I uh took a wheel chair down of course and sat in it and uh then she held up pictures. You know, window, door, and so on and I was to identify these things. I couldn't for a time and then I gradually got better, you see, as the swelling went down, you know, uh, I suppose I had a uh a rupture in the uh right uh, what do they call it? (pauses for a short time) cartoid artery or something like that, I don't know for sure. And they didn't operate you see, cuz it was too deep. I never had surgery.

MM: Will you ever need surgery?

LG: No, I don't think so. But uh, uh maybe, I don't know. The hand uh still bothers me, but I have gotten used to it, you see.

MM: You said you were completely paralyzed in your arm and leg. How about your face? Did it droop?

LG: Oh yes, yes. I couldn't move this side (referring to the right side).

MM: How long before you regained functional use of your leg?
LG: It's hard to tell. My leg, being bigger muscles came back first, you see, and my face uh, it is still improving. In other words, you see, if I go like this (smiled) you can see the dimple is not apparent in my right cheek, you see what I mean? But look at my left one.

MM: Oh yeah, you can tell. I am sure that since the time of your stroke, it has gotten somewhat better. What are some of the improvements that you notice from day to day, if there are any?

LG: Well, not necessary like that. Of course, at first, it was like that, you know. The first three months was the key to my uh recovering the way I did, you see what I mean? In other words, if I hadn't had therapy and people who cared about me and so on and maybe just pulled a quilt over me and left me in a sunny corner of the room, you see I could be still there today, I think.

MM: Do you feel your personality and drive were factors in your recovery?

LG: Well of course, I think so uh because uh uh I didn't take no for an answer, you see. In other words, I was going to get better and I was going to be back to work in the Fall. I knew that.

MM: Were you able to return to work in the Fall?

LG: Yeah, uh uh after six months. I knew what I had to do, if I could just force myself to do it, you see.

MM: Tell me some of the things you did to improve.

LG: Well, I talked all the time, you see what I mean? Whether it made sense or not.

MM: Do you remember how much of your speech didn't make sense?

LG: Some.

MM: Did your words sound like jargon?

LG: No, no. First of all, I should say my spelling was affected of course. I had to learn to do that all over again and my foreign languages was affected. I couldn't pronounce Italian, French, or German, uh or Latin, let alone English, you see what I mean? They came rather quickly. I didn't, I won't say that I learned them back necessarily, but I became aware of certain things you see and this triggered off a new something in my brain and then it was all right again, you see what I mean? It was like learning them back but not quite, you see.
LG: (continued) In other words, uh I said now for French, the vowel /a/ is /ah/, you see and uh I marked that down in my brain and uh certain of those things, but I, I didn't, uh, it wasn't easy.

MM: Your therapy was quite extensive, I understand. Something like five hours a day?

LG: Four hours for five months.

MM: Would you describe the various activities you did during this time?

LG: They each took a certain segment. I had un three counselors. Dr. Lyndes was one and another one and another one. Depending on uh on uh the uh--what am I trying to say? Well, one would have to work two hours a day, you see what I mean? Oh, I don't know. It is kinda hard to recall what they did. (Stopped to show me samples of his writing.) Well, I got something here. This is after about five months, you see. They had me doing plain outline some stuff, you see what I mean?

MM: Looking through these examples an improvement in writing can be seen. You had to relearn to write using your non-preferred hand, isn't that right?

LG: Oh, yeah, and we worked on that in therapy too.

MM: What type of difficulties, if any, did you have with numbers, arithmetic and time?

LG: I had to be careful uh because I couldn't add or substract time, but the clock didn't bother me.

(It becomes apparent to the reader that the number of speech interruptions which occur are considerably reduced at this point in the interview. It was suggested by my advisor for this paper at the University of Montana that this was at least in part a product of LG growing more comfortable with our conversation. This appears to be typical of these patients even years after the acquisition of intra-cranial injury.)

MM: Do you have certain problems with your memory of things? Especially names?

LG: Mainly names. It is getting better slowly.

MM: Is there anything you do to help you remember?

LG: I write things down.
MM: That's probably one of the best ways to keep track and at the same time you are working on your writing. What about right after your stroke, did you remember things that happened in the past?

LG: I remembered things that uh happened far back better than close up, you see what I mean?

MM: So, you didn't remember what happened during your stroke or the day before?

LG: Well, I think I could remember all that, but I'm not sure. I think I didn't realize how bad I was off you see and I figure that's a good thing. Because I never thought that I wouldn't get better. This is the main thing you see, uh, toward therapy and things like that, because uh you know as well as I do, if a character comes in to you and you uh says I uh am going to make you better or something like that and he said uh no, you're not and that's two strikes against you, you see what I mean? I never thought about that. Of course, I'm an educated man, I think, and there's a lot of spinoffs from that direction because I knew what I had to do to get better.

MM: How did you act around other people if you got frustrated and depressed?

LG: Well, I suppose I got frustrated, but I have the knowledge that it doesn't help, so I didn't, you see what I mean?

MM: That had to be a definite plus in your favor.

LG: I think so. When they rolled me in to the therapy the first time, I wanted to help in the worse way, you know. I think the therapist thought generally good of me, but I don't know for sure. (We both chuckled about that sentence.)

MM: I was thinking about a statement you made regarding the fact that you wouldn't wish aphasia on your worst enemy. That really made me think just how awful aphasia must be.

LG: Well, it is terrible. You can understand everything that's going on around you, but uh, I had the good nurses and doctors, and there was nothing that uh went on that wasn't above board and so on. I've heard of people who have had aphasia and the nurses thought he could not understand them and they were speaking uh a little raunchy, you know, about his case and that is inexcusable. Well, to answer your question, I don't have any worse enemies to begin with so maybe that's good.
MM: Since you had troubles with names, what about with your family's names?

LG: My daughter, whose name is Caroline, I called her Twila, which is my sister's name, for the first year. I think for the first week uh or so I forgot my wife's name, but then I remembered.

MM: How about now? What are the difficulties, if any, with remembering close friends' names, etc?

LG: No, I think that I have that down now.

MM: Did you have troubles with concepts like up and down, here and there, and he and she?

LG: Uh, yeah. In other words, uh if I meant left, uh I would say right, you see what I mean? Up and down, I would get them changed around for an instance and then I would recover.

MM: So you were able to do a lot of self correcting if you heard yourself say something the wrong way?

LG: Yes, that's right.

MM: What was the extent of your spelling problems? Did you know what the letters of the alphabet were?

LG: Not originally. I think I would look uh at a /g/ and uh call it uh, uh a /b/ or something like that. I think I had to relearn the whole thing. This only lasted uh for the first uh month or so. Then uh the hardest thing for me, I think was to uh get me to read for pleasure. Because you see, it's work and uh, uh it still is, kinda. I can read, but I uh, out loud I make terrible mistakes yet.

MM: Would you give me an example of what your mistake would be?

LG: Well, uh I am uh hesitant about reading aloud, uh I can read aloud, but if I read silently, I can read twice as fast. In other words, the aphasia still bothers me uh reading out loud. A little bit.

MM: In the early stages of recovery, could you read a whole sentence?

LG: I think I could read silently, but giving the uh out of my voice, it uh added another dimension, you see, which I could not handle, you see what I mean?
(Began talking about his busy day.)

LG: I like it and so far it likes me. I get my 8½ hours of sleep. I uh I never dream since my stroke, that's another thing, or I never remember my dreams. I don't know whether other stroke victims have the uh same troubles or not. And I don't miss my dreaming that much because uh uh I dreamed the craziest things, so I don't miss it at all.

MM: Did you have troubles with colors?

LG: I have always uh had trouble with colors. I am color-blind!

MM: Tell me a little bit about your physical therapy.

LG: I had it uh for about two and a half years. I had recovered uh most of uh my walking on my own just by getting out of uh the wheelchair and doing that. But my arm, but they uh worked with it for two and a half years and since that time it has, I can uh say maybe gotten stiffer since it hasn't had therapy now, it did not help one bit. So I had to make the best I could with what I had.

MM: Did you have problems talking on the telephone?

LG: Oh yes, yes. That was uh the biggest problem I had. And uh my daughter and uh son would call up, but I could listen and uh occasionally I could speak up, but the problem was uh I would get all set to say something and they were on a different subject. They talked so fast. Thank the Lord, it has been only the last year and a half that I can hold my own on a phone conversation.

MM: Then when people were talking to you, was their rate too fast for you?

LG: Uh, it didn't matter I guess, uh I understood everything they said, you see, but I couldn't respond, that's all.

MM: Since I have asked all the questions that I wanted to, is there anything that you would like to add that I could relay that you think is important for people to know?

LG: Yes, check your blood pressure every six months or so whether you need it or not because I think my blood pressure was up and I I think that's what caused the stroke. Prevent it.
Review of RL

Again, the background information on the patient, RL, was provided by her speech clinician. RL received a depressed skull fracture in an auto accident in California. From a brain scan, areas of uptake were indicated, thus indicating the presence of subdural bleeding and therefore hematoma. Primary damage was in the mid-posterior portion of the left temporal lobe (Wernicke's area); however, some damage was present up into the tip of the temporal lobe as well. She had numerous broken bones, there was no hemiplegia, and RL was aphasic when she woke. Surgery was performed to evacuate the subdural hematomas.

From a psychological evaluation of her condition, it was reported that RL suffered from organic psychosis, paranoid idation, and severe aphasia. This psychological evaluation, describing her verbal behaviors as a result of psychosis, was later proven incorrect. RL was functioning well in society before the accident, she performed well at her job, and her psychological state was not as extensive as previously evaluated.

Following RL's release from the hospital, she could not care for herself; therefore, she was placed in a rehabilitation center. Later, RL was removed from this center and taken to Montana by one of her daughters. This situation proved to be very stressful for her. An interesting point to be made concerning this situation is that RL would refer to the daughter
she was living with as her mother. Notably, her reasons for doing so stemmed from RL's negative feelings for the daughter, who had removed her without the rest of the family's knowledge. At this time, she was referred to a speech clinician, Dr. Lyndes (DKL) from the University of Montana Speech and Hearing Clinic.

DKL worked with her once every other week because of the distance necessary to travel in order to have therapy. All other sessions were handled by a communication aid. RL would not confide in the communication aid, yet she seemed to trust DKL. The therapy program was continued for approximately four months. Briefly, DKL described the major components encompassing RL's therapy program. There was work done with words she would frequently come in contact with, words relating to body parts so RL could describe any pains she might be having, and words relating to household objects were worked on as well. These words and phrases are necessary for functioning in her daily environment.

A major problem for RL was in the area of reception of speech; therefore, DKL made his rate of speech slower than normal without losing the necessary qualitative aspects of rhythm and prosody. In some of the examples of RL's speech illustrated, it will be apparent that it is filled with perseverations, automatic utterances, jargon, and paraphasias. It should be noted that RL considers her productions quite understandable; however, a review of her utterances will demonstrate this is not the case. In many of her sentences,
correct rhythm and prosody are present so the listener can infer that each perseveration is intended to represent different words. Although jargon and paraphasia cloud the meaning RL is trying to portray, sometimes the listener correctly interprets the utterance by using the context of situation in which it was spoken.

At one point during an early therapy session with RL, DKL had her repeat a grouping of two, three, and four digits. The reason was that he wanted to determine the number of stimuli which RL could successfully handle. Digits are used because they contain less linguistic value, although they do contain some linguistic content. From RL's performance it was apparent that she could adequately handle three stimuli. At this point, a transfer from digits to syllables was then made. DKL determined that RL worked quite satisfactorily at the symbolic level and he proceeded to focus the therapy program on this data.

Because RL confided in DKL, he was able to learn that RL had another daughter living in Hawaii. Contact was made with this daughter in hopes of alleviating some of the environmental problems that were a part of RL's life. Fortunately, the daughter had RL moved to Hawaii, thereby removing her from a potentially destructive and harmful environment. DKL reports that he received one personal letter from RL (which was syntactically correct) and it was evident that she was doing much better in her new environment.
The following transcript is taken from an early therapy session between Dr. Lyndes and RL. It should be pointed out that RL would use facial expressions and other phatic communication skills during her speech; however, she was far less capable in their use or in her oral language skills than the first patient, LG.

In regard to the translation of DKL's verbalizations, they were spoken at a moderate rate with care taken to include normal inflection. Some of the longer utterances were carefully broken by short pauses (indicated by three periods . . .) to allow the patient to assimilate what was being said without becoming overloaded auditorily.

RL: It's a bunch of bullshit (referred to as BS in remainder of examples).

DKL: The accident?

RL: No, the people that involve me.

DKL: Oh, I was going to say . . . that three years ago"

RL: And my ministers, my revelies are also for the birds.

DKL: Three years ago . . . that happened to my wife.

RL: Oh, I don't wanta care about it.

DKL: Our car was totally demolished.

RL: Sure, I know, I got a loud speech and that's the only thing I'm interested in.

DKL: You've got what? Tell me again.

RL: I have a lot of uh lee--vies and that's why I'm sick to death of it and that's why I want feather-buzit. I want out of it, I want rid of it, get it cured, or whatever it is that I have to do with it.

DKL: OK.
RL: And you tell me, I'm not got to tell you, you tell me what I have to do.

DKL: All right. One of the things... I can tell you right now... that's going to help you a great deal is that, in just the few short minutes... that you and I have been talking, I notice that you are understandably very tense about what's happened. The more tense you become, the harder it will be... for speech to come.

RL: More howder for it?

DKL: No, no, the harder it will be to get your speech going.

RL: In other words, if I cream the, the boy, uh the creek, uh, and the crease and stuff, then it goes easy. Is that what you say?

DKL: If you just relax.

RL: There's no, no such a thing as all that in my. I've been sitting and waiting, and waiting, and waiting, and waiting, until I'm sick to death of it.

DKL: I can understand that very well.

RL: And I want action to it. I don't have all that kind of BS to spend around and wait for somebody to, to, to.

DKL: Well, there's no magic formula.

RL: Well, I think there's more so than what they give a lot of time.

DKL: So, part of it... is consistently working on it.

RL: Some days I feel like a good ol' ta si sore tite in the wreck would just eliminate all this BS, you know that?

DKL: Yes, but it's something we are going to have to deal with... and they way we are going to deal with it best... is by letting go, being as relaxed as possible.

RL: Well, I'll try.

DKL: That a gal. That's what I like to hear.

RL: And that's not easy because I work with a dub edgers all the time. And I'm aware of it, I'm very much aware of it and I'm sick to death of these people that
(continued) give me a bunch of stuff that some some woman is knows all about you, you know, or knows all about me, or all that sort of thing. I'm interested in what I've got and how I can get rid of it and the first best trip now and that's my.

DKL: That a girl.

RL: I know, but it you're you're telling me that I don't have all of the knack, right?

DKL: No, no, I'm not. I'm telling you . . . that the more tense you become about it . . . the harder it's going to be. So, that's something . . . that's very easy for me to say and very difficult to do and I know that, but, it's a big important part.

RL: Well, we'll see.

DKL: Tell me what you can recall about your accident.

RL: Why do I have to?

DKL: Because I'm interested in . . .

RL: I don't know, I don't remember too much about it because I prefer to forget it. I'll tell you if I have to, but I just as soon. I want your know for me, not mine for you.

DKL: But I need knowledge of you . . . to design and work with your aid . . . in order to develop a therapeutic program.

RL: Well, I don't know how that's going to be good for but--

DKL: It's a very very basic part of . . . you work for the Air Force, is that correct?

RL: Well, certainly but that has nothing to do with the BS that is, is that, uh is cave my BS now. Certainly, I, I worked for the jeb. I had the job sure and that has nothing to do with I have now as far as I'm concerned.

DKL: But our talking back and forth . . . gives me some information.

RL: Well, you're picking me today because I'm in a vec tu verb nature and I shouldn't even mention a word today because that circus that I just got out of has ruined me for today. And I'm, I'm, I shouldn't even be talkin about the same thing. I'm tryin, I tryin,
RL: (continued) but I don't want to have to go back into a bunch of led knezzie stuff that's not, uh permanent, permanent unless I absolutely have to. I don't want to pie all this BS from here what's been alldin. I want what's got it now for me to get it and get it taken care of. You understand what I'm saying?

DKL: I understand very well.

RL: And I could care less about, about sue, who what character substituted me on a beast star, something like that you know. In other words, I don't want this, the less five stall unless I have to mess with it.
CHAPTER 5
DISCUSSION

To reiterate the purpose of the four preceding sections, this paper explores the previously established fundamental principles presented in the literature concerning the disorder of aphasia. Aphasia has been defined as an immensely complex disturbance in language and the understanding of the spoken word as well as in reading, writing, and arithmetic abilities resulting from lesions to the language dominant cerebral hemispheres. Generally, the overall aphasic impairment demonstrated by each individual includes reduction within each of the language skills mentioned. In reference to the degree of manifestation of aphasia observed within each individual, the point stressed is that our concern is for the individual with unique personal characteristics, experiences, and qualities. Basically, the interests of the patient are best served if we remember there are as many kinds of aphasia as there are aphasics. Classification is at best a highly general thing.

Reviewing the relationship existing between cerebral insult and aphasia symptomatology, arguments from both the localizationists and non-localizationists seem to present
valid positions worth considering. There is evidence that language processing may be specifically impaired according to the anatomical location of the cerebral damage. Conversely, we must not rely completely on the localizationist's viewpoint due to the degree of integration existing with the cerebrum.

After according the left cerebral hemisphere to be responsible in the majority of individuals for language processing, storing, and retrieval, the importance of the right cerebral hemisphere was demonstrated as well. Because the right hemisphere frequently handles our perception of phonological and intonation cues, its value in language production cannot be neglected.

Through the "landmark investigations" of Penfield and Roberts (1959) and Penfield and Rasmussen (1968), the significance of various cortical areas was illustrated. Their neurosurgical procedures indicated that certain disorders can be localized within specific areas of the left cerebral hemisphere.

Inferences may be made concerning the cerebral areas damaged depending on the range of speech and language abilities remaining available to the patient. The intercommunication and synthesis among all cortical areas must be emphasized as well as the perception that the intricate brain mechanisms associated with the production and comprehension of language require further research before a complete and
accurate understanding of these brain mechanisms is established.

While there are numerous facets of aphasia, the psycholinguistic dimensions represent a major area of concern underlying the disorder. Language provides a symbol system whereby individuals use its components to express or interpret messages received as well as for the formulation and production of thoughts and perceptions. It is this multidimensional system of language which is disadvantaged throughout the aphasic population in varying degrees.

Regarding the general aphasic involvement within the three levels of language complexity, the symbolic level appears the most substantially affected. Frequently, aphasics are unable to symbolically externalize their language, although the capacity remains for cognitively constructing ideas for processing.

After examining the semantic, syntactic, and phonological component areas of language, a further stigma surrounding aphasia becomes apparent. Aphasia is a complication in linguistic functioning or within the techniques for utilizing language rules. The deficits generally exist to varying degrees within each component area and each aphasic individual. It would appear that though the patient's thoughts and perceptions approach a language barrier where they remain incapable of being transferred into the appropriate language productions.
The particular changes observed within the areas of speaking, reading, writing, listening, and gesture provide a more specific description of the psycholinguistic dimensions existing among individuals with aphasia. Many aphasics demonstrate a reduction in their vocabulary, they require additional time in order to produce desired lexical items, and frequently these lexical choices are word substitutions. In a similar manner, problems arise in the patient's ability to retain relevant information. The reduction is generally most evident in a loss of specific detail rather than in the general gestalt of the message.

Besides reductions in vocabulary and verbal retention span, aphasic individuals demonstrate an impairment in auditory comprehension. This symptom, unrelated to hearing loss, is a continual source of frustration and limitation due to the fact numerous messages rely heavily on complete auditory comprehension.

Subsequently, a final summarization of the more regularly occurring aphasic symptomatology includes mention of paraphasia, jargon, perseverations, and use of automatic utterance. The symptoms are by no means an extensive representation; however, they do provide a general framework with which to view the disorder of aphasia.

Developing an appreciation for the varying communication and language deficits aphasic patients must confront can be only part of any discussion relating to the disorder. In a high percentage of patients, hemiplegia or hemiparesis
involving the extremities and face on one side of the body are major physical disabilities confronting the patient. In addition, the patient will often show difficulty with spatial-perceptual tasks, reductions are seen in visual field thus modifying vision, and they may have seizures as well.

With these numerous deficits in mind, the aphasic patient's reaction to their reduction or loss becomes a major contributor of the behavioral and psychological reactions demonstrated by the patient. Their reactions vary from day to day due to the fact that the recovery process is a constant struggle, consisting of almost unyielding deficits and frustrations in accomplishing improvements over their level of function.

Recapping the following reactions which may exist should establish for the reader a generalized sample of the various aberrations they may observe among aphasic individuals. Since the aphasic's threshold of emotional reaction is lowered, crying and laughing outbursts frequently prevail, usually without any relationship to environmental events. In a similar manner, anger and aggression are frequently displaced and randomly projected by the patient to those individuals providing therapy, support and care. In addition, the common occurrences of depression, fear, and frustration are other associated reactions generally observed.
In summary, although aphasics display various behavioral and psychological reactions, these patterns distinguish them from patients whose illogical responses are due to disorganized thinking associated with diffuse brain injury. Basically, the only definite concept is that there will be varying degrees of aphasic symptomatology presented with each patient. The challenge for individuals interested in rehabilitation is to remember the aphasic is a unique individual, with personal characteristics and attributes differentially affected through cerebral damage.

One final remark in regard to the writer's review of two aphasic patients is necessary in order to obtain closure. With the exceptions of the notations already added, no further attention has been directed to their specific cases or problems because the text of what transpired would seem sufficiently self-explanatory. By the inclusion of the texts produced by the two aphasic individuals, it is hoped that better amplification of the varying parameters of aphasia will be demonstrated for the reader.
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