American Handbook of Psychiatry

THE NEURAL ORGANIZATION OF LANGUAGE: APHASIA AND NEUROPSYCHIATRY

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Aphasia and Neuropsychiatry

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e-Book 2015 International Psychotherapy Institute

From *American Handbook of Psychiatry: Volume 4* edited by Silvano Arietti

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The Neural Organization Of Language: Aphasia And Neuropsychiatry[1]

> . . . the thought which only seemed naked was but pleading for the clothes it wore to become visible, while the words lurking afar were not empty shells as they seemed, but were only waiting for the thought they already concealed to set them aflame and in motion.
>
> —Vladimir Nabokov

The History of Aphasia

From Gall to Wernicke

Although there are references to speech loss from cerebral lesions dating as far back as the *Hippocratic Corpus* of 400 B.C., the modern era is usually taken to begin with the phrenology of Franz Joseph Gall in the early 19th century. His work had far-reaching implications, but for the still unborn field of aphasia research it signaled a shift in attention away from the holistic approach which was current at the time to the possibility of a cerebral localization of speech. Gall reasoned specifically from a single instance in which large eyes and a prodigious verbal memory happened to occur in the same individual, a childhood acquaintance, that speech was a function of the frontal lobes. The French neurologist Bouillaud was so impressed by this assertion that he offered an award of 500 Frs. to anyone who could disprove

it. Bouillaud also wrote an historically important paper in which he distinguished between the sign function of speech and its articulatory apparatus (i.e., between internal and external speech), and on the basis of a few cases argued that the "legislative organ of speech" resided in the anterior (frontal) lobes of the brain.

Paul Broca, a student under Bouillaud at Bicêtre Hospital, could not fail to be influenced by the exciting debate stimulated by these ideas. An opportunity to settle the issue finally occurred when a fifty-one-year old patient with excellent comprehension but almost complete loss of speech was admitted to the ward. The postmortem examination, from which date one can ascribe the beginnings of the science of aphasia, demonstrated, as predicted by Bouillaud, a large Sylvian lesion in the left hemisphere, the center of which was in the third, and partly the second, frontal convolution. Broca conceived the speech loss, aphemia, to be a kind of ataxia of those movements which served for the articulation of words. In subsequent papers he defined the "motor speech area" as consisting of the posterior part of the third or inferior frontal convolution (F3), and by 1865 sufficient data had been collected to suggest a possible relationship, in right handers, to the left hemisphere. It is of interest that the term "aphemia," chosen by Broca for this disorder, was criticized by Trousseau on the grounds that it connoted infamy (i.e., unspeakableness), rather than lack of speech. Gradually it has become customary to use the term "aphasia" for loss of speech *and* writing, and

"aphemia" for loss of speech alone.

Certainly it can be said that at that time the various approaches to the problem of aphasia had not yet hardened into the distinct schools of thought that so characterized later work in the field. While Broca is often represented as the earliest "localizer," an impartial reading of his papers gives a very different impression. For example, his treatment of "aphemia" as a return to a childhood stage in speech development foreshadows modem accounts of agrammatism and phonemic disintegration. Moreover, Broca stressed that aphemia was a type of motor speech disorder, and distinguished it, as had Bouillaud before him, from the true language disturbance of verbal amnesia.

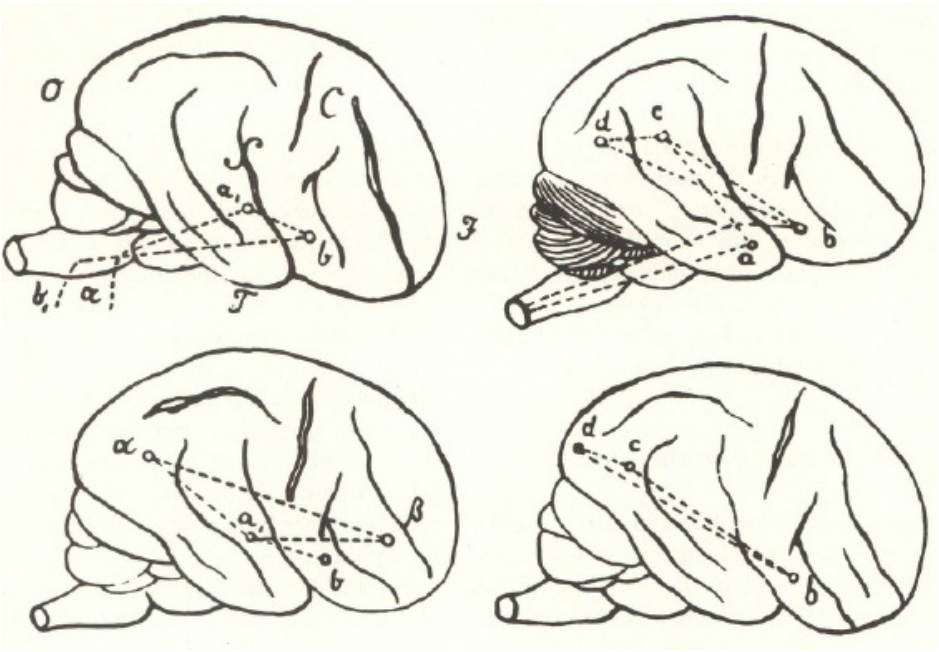
Hughlings Jackson was generally sympathetic to Broca's work, though he disapproved of the distinction between articulation and word memory, these being just different aspects of the capacity to produce words. Following J. G. F. Baillarger, Jackson stressed the common dissociation between voluntary and involuntary performances in motor aphasia, and suggested a special relation of the latter to minor hemisphere. In later writings Jackson emphasized that the aphasic, though speechless, was not wordless, and that aphasia consisted not of a loss of speech but a loss of the ability to "propositionize," defining a proposition as a relation of words such as to make one new meaning. Perhaps Jackson's chief contribution to aphasia theory, and particularly to what was later to become psychoanalytic theory, was his

evolutionary account of levels of function. According to this view, successively higher levels of functional organization were laid down in the course of encephalization, each new level suppressing and having a degree less automatization than that which came before. This conception had a clear impact on Freud's early thinking and without doubt figured prominently in the topographic theory and the account of repression.

The ontogenetic interpretation of Broca, and the phylogenetic account of Jackson, were destined to survive but a short time in neuropsychology. In 1874 Carl Wernicke, after six months on an aphasia service, published his monograph *Der aphasische Symptomenkomplex.* Following T. Meynert's demonstration of the central terminations of the auditory nerve, Wernicke argued that destruction of the sound images of words, laid down adjacent to the acoustic projection zone in the posterior part of the superior temporal convolution (T_1), should result in an inability to understand or repeat speech. Since patients with impaired speech comprehension appeared to recognize objects, and could express some needs by mimicry, the concepts corresponding to these sounds images were thought to be intact. Thus, three forms of aphasia could be distinguished: (1) motor or Broca's aphasia; (2) sensory aphasia (with destruction of the auditory sound images); and (3) verbal amnesia, due to involvement of the posterior concept field (Begriffsfeld). Moreover, Wernicke also commented that a lesion *between* the "sensory" and "motor" zones should produce a condition in which

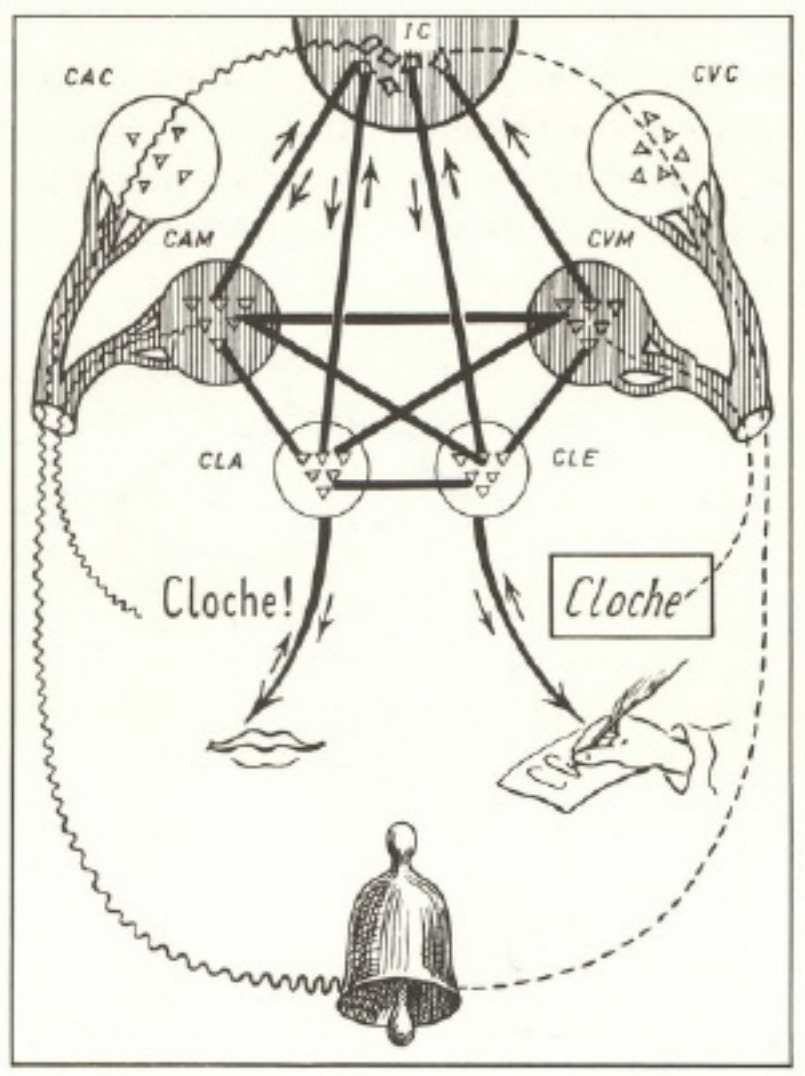
comprehension was preserved, speech was intelligible though paraphasic, and repetition was selectively impaired. This latter disorder, described on a theoretical basis only, was termed conduction aphasia, (Leitungsaphasie). The simple diagrams which Wernicke employed to illustrate these aphasic disorders (Figure 10-1) lead to the brain maps of L. Lichtheim and others, achieving in the latter part of the 19th century an almost baroque complexity, as in the ornate but wholly imaginary diagram of Charcot (Figure 10-2).

Figure 10-1.



Diagrams from Wernicke representing hypothetical sensori-motor centres and conducting pathways

Figure 10-2.



Charcot's illustration of mechanisms involved between hearing the sound of a bell, and producing the word "bell" in speech and writing. (Reprinted with

the permission of Butterworth & Company.)

The association theory of Wernicke, in providing a reductionistic alternative to the genetic accounts of Broca and Jackson, had an enormous appeal at the time and continued to dominate thinking until the critique of Pierre Marie in 1906. However, the Wemicke-Lichtheim model was challenged in one short but important monograph.

The Contribution of Freud to Aphasia

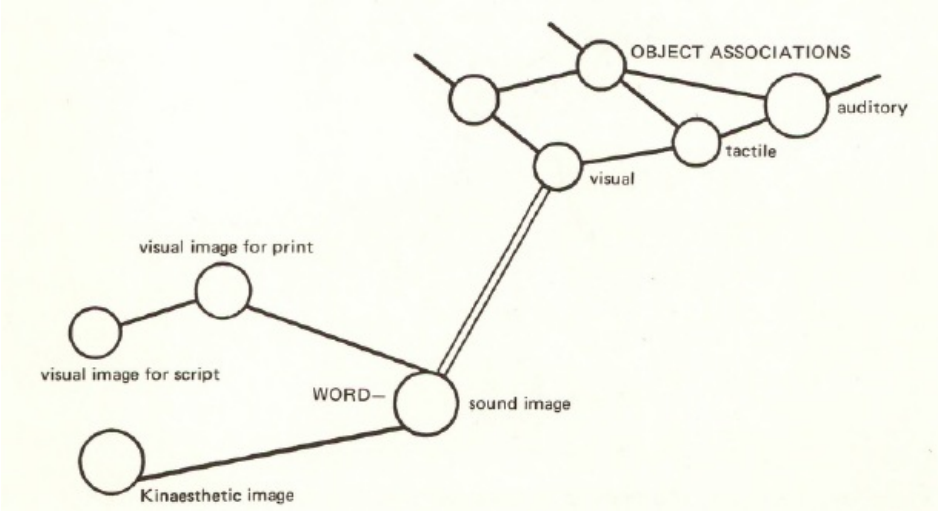
In 1891, when Freud's modest study of aphasia first appeared, the school of Wernicke was the most influential in Europe. It is only against this background that one can sense the daring—indeed, revolutionary—flavor of Freud's work.[2] The book is chiefly concerned with a refutation of the localizationist (centers and pathways) model in favor of a concept of a unitary cortical speech zone:

> Our concept of the organization of the central apparatus of speech is that of a continuous cortical region occupying the space between the terminations of the optic and acoustic nerves and of the areas of the cranial and certain peripheral motor nerves in the left hemisphere. . . . We have refused to localize the psychic elements of the speech process in specified areas within this region . . . (and) the speech centres are, in our view, parts of the cortex which may claim a pathological but no special physiological significance, [p. 67]

In relation to this speech zone, language was built up through a process of *psychological* association (Figure 10-3). Accordingly:

From the psychological point of view the "word" is the functional unit of speech; it is a complex concept constituted of auditory, visual and kin-aesthetic elements, [p. 73]

Figure 10-3.



Schema of the formation of a word concept, from Freud.

It follows that:

. . . all aphasias originate in interruption of associations, i.e., of conduction. Aphasia through destruction or lesion of a centre is to us no more and no less than aphasia through lesion of those association fibres which meet in that nodal point called a centre, [pp. 67-68]

On this basis Freud attempted a reclassification of the aphasias, an attempt far from successful, for even he had to confess that:

I am well aware that the considerations set out in this book must leave a

> feeling of dissatisfaction in the reader's mind. I have endeavored to demolish a convenient and attractive theory of the aphasias, and having succeeded in this, I have been able to put into its place something less obvious and less complete, [p. 104]

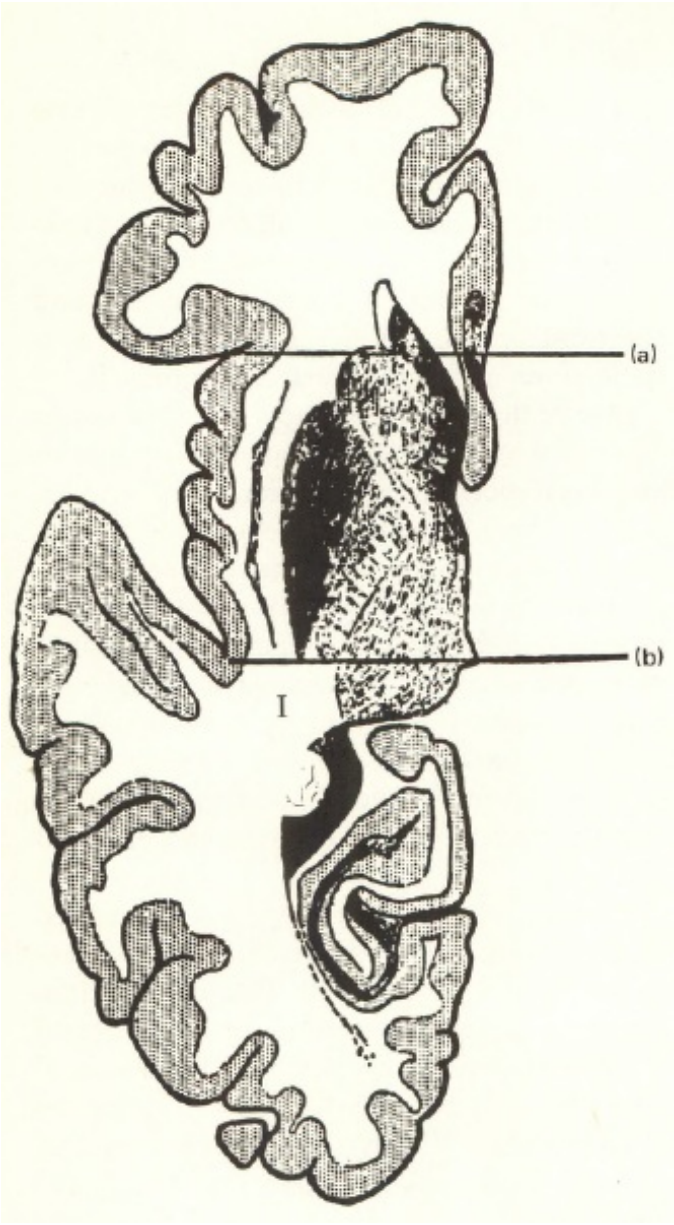
The reader does sense, however, that the work on aphasia served to liberate Freud's thinking from the anatomically bound dogmas of the time, and encouraged him to proceed into psychological speculation without the gnawing feeling that anatomy—at least the anatomy of the day—must always have the last word. There is, moreover, much in this monograph which presaged his later formulations. Specifically, one notes the application of the Jacksonian concept of dissolution to the pathology of learned associations; there is the importance given to the "word" as the final segment in thought production, signaling the prominent position later to be given to verbalization in the psychoanalytic method; there is a suggestion that the analysis of paraphasic errors may have played a part in his later concept of "slips-of-the-tongue;" and chiefly, to my mind, there is the central idea that if neuropsychological symptoms could result from a breakdown in learned associations, psychological symptomatology might result from the formation of pathological associations.

A Search for New Formulations

Whereas Freud's lucid and meticulous criticism of the classical school fell on deaf ears, Pierre Marie's aggressive paper of 1906 came like a

bombshell. The very subtitle of this paper “La troisieme circonvolution frontale gauche ne joue aucun role special dans la fonction du langage,” was an indication of Marie’s extreme dissatisfaction with the excessively localizationist approach to aphasic disorders. Marie held that the expressive defect in motor “aphasia” was actually an anarthria due to involvement of the zone of the lenticular nucleus (Figure 10-4). Wernicke’s, or true, aphasia was a kind of intellectual defect resulting from a posterior lesion. A combination of anarthria and the comprehension defect of true aphasia was responsible for “Broca’s aphasia.”

Figure 10-4.


(a)
(b)
I

A lesion of the quadrilateral space of Marie, lying between the anterior (a) and posterior (b) extent of the lenticular nucleus, produces an anarthria, while a lesion at I, involving the lenticular zone and also interrupting temporoparietal fibers, accounts for true aphasia.

At the same time that Marie was attempting to rid neurology of its aphasia brain maps, as naive as they were numerous, another and more constructive trend was under way. The point of view was beginning to emerge that language was not a piecemeal assembly of smaller units but rather a productive activity within a cognitive matrix. The influence of Humboldt was still present in the developing science of linguistics, and this, combined with the hierarchic theory of Jackson and the mental structuralism of the Wurzburg school, came together in Arnold Pick's new concept of the aphasias.

For Pick the aphasias were disruptions at sequential stages in the realization of speech out of thought. He described four stages in the transition of thought to speech: an early stage (1) in which thought is formulated with increasing clarity out of memory in such a way that its partial contents are combined to a type of schematic or structural whole; the second stage (2), that of structural thought, is prior to linguistic formulation; there is a preparation toward a predicative arrangement, and elements of tone, tempo, and grammar come into play; the next stage (3), that of the sentence pattern, develops under the influence of an emotional factor, and leads to the

automatic choice of words. There is a correspondence between Pick's account of intuitive (1) and structural (2) thought, and the *Bewusstseinslage* and *Bewusstheit* of the Wurzburg school, as well as with the (later) "sphere" and "concept" of Paul Schilder. Moreover, the possibility that language issued out of a prefigurative ideational stage embedded in a spatial attitude leads to the concept of spatial defects in the semantic aphasia of Head (see p. 257), as well as the more recent notion that the memory trace may be integrated in the space-coordinate system. Pick's work has been discussed in recent publications by Spreen and Brown.

In England, Head, who was familiar with Pick's writings, attempted to incorporate them with personal observations of aphasic patients. His classification of verbal, syntactic, nominal, and semantic aphasia represents an advance only in the postulation of the final of these forms, semantic aphasia. Even here, however, the effort to bring this disputed "deep-level" aphasia into relation with disorders of spatial-constructional thought tended to weaken the force of Head's argument.

The two other major figures of the time, Karl Kleist and Kurt Goldstein, were unable to resolve their dynamic psychological point of view with a localizationist mentality. Kleist, for example, attempted to translate Pick's classification into an extreme form of (myeloarchitectonic) cortical localization. While there is much of value in Kleist's work, a cursory glance at

his pathological specimens is enough to dissuade even the most sympathetic reader from too ready an acceptance of his anatomical theories. On the other hand, Goldstein did not even attempt to superimpose his view of the psychology of language on a pathological anatomy, but wisely elected to treat the psychological and pathological aspects separately. With regard to the former, his contribution has to be measured by the exhaustive scholarship which was brought to bear on every phase of his work, the Gestalt orientation, and emphasis on organismic factors. The cognitive basis of language was always in the foreground of his work. Perhaps the one concept for which he is best known is the distinction of "abstract" and "concrete" behavior. However, most workers now recognize that disorders which were attributed to alteration of the abstract attitude, e.g., anomia, occur without such alteration, while concrete thinking occurs in the absence of true anomia. For this reason, a classification of aphasia based on the concept of "abstraction" and "concreteness" does not have wide appeal. According to Goldstein, anomia (anomic or amnesic aphasia) was a disorder of thought (i.e., of abstraction), while Broca's aphasia was chiefly a defect of the final stages of word production. Central (conduction) aphasia was a disturbance *between* the two, at the transition of thought to speech, viz., a defect of "inner speech." To some extent this classification recalls the microgenetic account of Pick, though Goldstein's pathological descriptions, and his interpretations of the pathological anatomy, did not deviate greatly from the original views of

his teacher, Wernicke.

In addition to this line of study, which was fundamentally a continuation of certain trends in the early German school of aphasia, there were also during this time several other noteworthy contributions. Weisenberg and McBride introduced American readers to the historical debate surrounding various issues in the field, and provided a healthy—even if somewhat vacuous—alternative to the rigid classifications then available. Johannes Nielsen was for many years one of the principle authorities on aphasia in the United States. His work, like that of Kleist, was characterized by erudition and a dynamic point of view not readily apparent on superficial reading. Penfield and Roberts gave valuable descriptions on the effects of stimulation of speech cortex in waking subjects, and argued, chiefly from negative extirpations, that thalamo-cortical connections played a central role in the anatomical organization of language. Some of these traditions have been carried on in England, by Brain and Critchley among others. In Germany, the Gestalt approach has been furthered by the work of Bay and Conrad, and in France the best known authors are Alajouanine, Lhermitte and coworkers, and Hecaen.

Status of the Field

There are two major orientations in modern aphasia research, both of

which have grown out of the classical tradition: the argument from the psychological point of view, and the argument from the point of view of anatomy.

Psychological Accounts of Aphasia

A great number of distinct theories fall into this category. Of these, one of the more progressive is the current attempt to bring linguistic description into relation with aphasic symptomatology. Psycholinguists have shown increasing interest in aphasic language and the term neurolinguistics is often taken as a designation of this new synthetic approach.

One of the earliest attempts in this direction was Jakobson's study of aphasic breakdown and correspondences with language acquisition in the child. More recently, utilizing Luria's classification and the distinction implicit in this system of posterior spatial (simultaneous) and anterior temporal (successive) processes, two major categories of aphasic disturbance have been distinguished, a *similarity* disorder, characterized by an inability to select and identify, and a *contiguity* disorder, characterized by an inability to combine and integrate.

There have also been attempts to demonstrate correspondences between aphasic language and expectations of distinctive feature theory. Especially important in this regard are studies by Blumstein and Lecours and

Lhermitte. The transformational grammar of Chomsky has been tested against aphasic language in studies by Weigl and Bierwisch. In this respect, the reader is referred to studies by Goodglass, and Zurif on agrammatism; Green, Kreindler, and Kertesz on jargonaphasia; and Marshall and Newcombe, and Rinnert and Whitaker on semantic paraphasia. A review of work in psycholinguistics and aphasia was published in 1973.

The term "neurolinguistics" appears to have been introduced by Henri Hecaen, who has also developed a linguistic typology of the aphasias. Accordingly, three major aphasic groups, expressive, amnesic, and sensory, are distinguished. Within the expressive group, there are three forms: (1) an impairment of phonemic realization (motor aphasia); (2) an impairment of syntactic realization (agrammatism); and (3) an impairment of programming at the level of the phrase (conduction aphasia). Amnesic aphasia is a selectional disorder, often linked to other aphasic forms. Within the group of sensory aphasia, three elements can be isolated: word deafness, impaired verbal comprehension, and a disorganization of attention. These elements often occur together in varying degree, and determine the pattern of expressive language.

The classification of Luria is a departure from standard works chiefly in the functional approach toward each aphasic syndrome, and not in the description of the symptom complex per se. The following six forms are

distinguished: (1) *sensory* aphasia, in which the expressive pattern is attributed to impaired phonemic discrimination; (2) *acoustic-amnestic* aphasia, which differs from the above chiefly in the improved repetition; (3) *afferent* and (4) *efferent* motor aphasia, which incorporate distinct aspects of Broca's aphasia; (5) *semantic* aphasia, which seems to include amnestic aphasia, and is similar to Head's account; and (6) *dynamic* aphasia, with reduced spontaneity of speech, similar to a mild transcortical motor aphasia. However, objections can be raised against this classification on several counts. For example, the impairment of phonemic discrimination, central to the sensory forms, is tested chiefly through productive systems; phonemic discrimination is an extremely resistant ability in a wide range of aphasic patients with disturbed speech comprehension; evidence for the kin-aesthetic basis and postcentral localization of afferent aphasia is wanting; dynamic aphasia seems to merge with the reduced speech picture of dements and various types of partial mutism. Moreover, as suggested by the syndrome designations, there is assumed to be a specific functional impairment in each disorder, i.e., in verbal memory, acoustic sensation, the evidence for which is at best controversial. Finally, the pathological account of primary and secondary cortical "analysers" in relation to these disorders does not take us very far beyond classical speculations regarding a similar role for primary (projection) and secondary (association) cortex. Nonetheless, Luria's work is extremely valuable for the ingenious testing methods and careful clinical

observation, the thorough study of individual cases and the application of an experimental approach to traditional "bedside" technique. From the point of view of theory, the major contribution is the concept of aphasia as a disturbance in cognitive function. Thus, speaking of language organization, Luria has written that the system of semantic codes "possesses a complex hierarchical structure. It begins with the system of words, behind each of which there stands not only a unitary image, but a complex system of generalizations of those things which the word signifies." Similarly, perception is studied not as a simple receptive function but as an active process, comparable to speech and motility. Perception involves ". .. the recognition of the dominant signs of an object, the creation of a series of visual hypotheses or alternatives, the choice of the most probable of these hypotheses, and the final determination of the required image . . ." The reader will note that this sequence is identical to other descriptions of stages in the course of problem-solving behavior, i.e., thinking.

Eberhard Bay has also viewed aphasia as a disturbance in concept formation. However, Bay's model is incomplete and to a degree expedient, and exception can be taken to many interpretations, e.g., the account of agrammatism as an economy of effort or the explanation of paraphasia as secondary to lack of speech awareness and logorrhea.

Klaus Conrad conceives aphasia as an arrest or interruption in the

microgenesis of cognition. An aphasia is a pregestalt (Vorgestalt) stage in the process of language formation. Conrad has distinguished four levels of pathophysiological change which, from the highest to the lowest are, respectively, *Strukturwandel, Gestaltwandel*, *Funktionswandel,* and *Formivandel.* Pathology induces a change in functional level, not a loss of function. The reduced level then determines the symptomatology. However appealing this approach, the discussion of aphasia is not altogether successful, for a general theory of regression does not account for the diversity of aphasic symptoms. Conrad has also helped to clarify the problem of "severity" in hierarchical systems. In Conrad's view, the lower (i.e., word close) the lesion, the more severe, but more restricted, the local effect, while higher (i.e., thought close) defects produce a slight impairment in more widespread functions, and involve more of the patient's native personality.

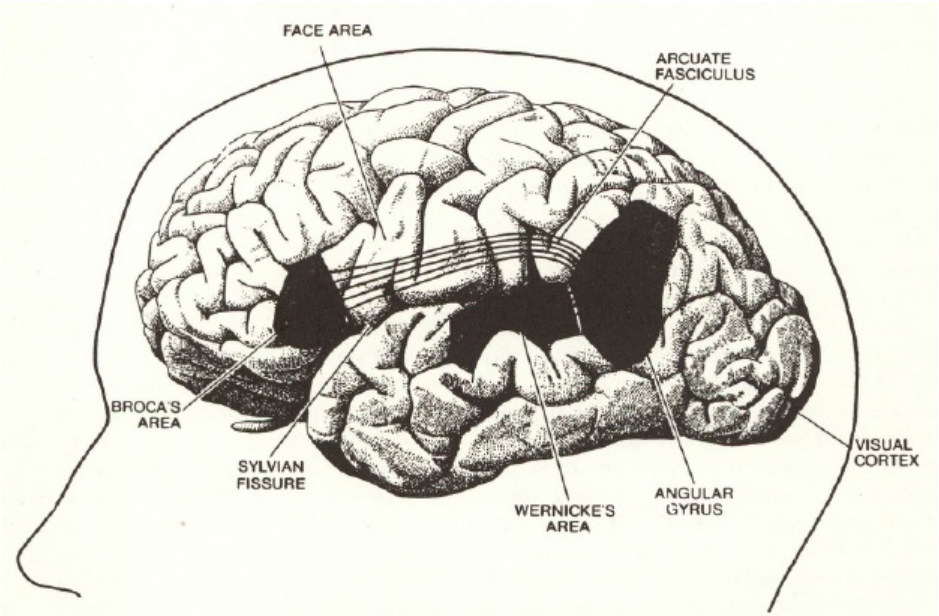
Anatomical Theories of Aphasia

Psychological studies of aphasia have not yet succeeded in the formulation of a unitary theory of these disorders, nor are such theories commonly attempted. However, caution has not been the most distinguishing characteristic of the anatomical school. Although there continue to be minor disputes over the specific role in language of one or another anatomical structure, the basic approach, on which there is, regrettably, essential agreement, has remained unchanged for a century after Wernicke's

monograph. The position has been summarized by Geschwind.

According to this view (Figure 10-5) speech is perceived by way of (left) Wernicke's area, and from there conveyed to "Parietal association" cortex for comprehension. Language is presumed to be formed in some way in the posterior part of the brain and passed forward to Broca's area for articulation. Repetition is accomplished through a cortical reflex circuit, comprising Wernicke's area, Broca's area, and the *fasciculus arcuatus* between, though this pathway is not usually specified as that underlying the postero-anterior flow (development) of spontaneous speech. The aphasias represent disruptions of these processes (actually, the processes are inferred from their pathology to be localized to these areas). Thus, a lesion of left posterior superior temporal gyrus is said to produce Wernicke's (sensory, receptive, jargon) aphasia, lesion of the posterior inferior frontal gyrus, Broca's (motor, expressive, anarthric) aphasia, and lesion of the *fasciculus arcuatus,* conduction (central, repetition) aphasia.

Figure 10-5.

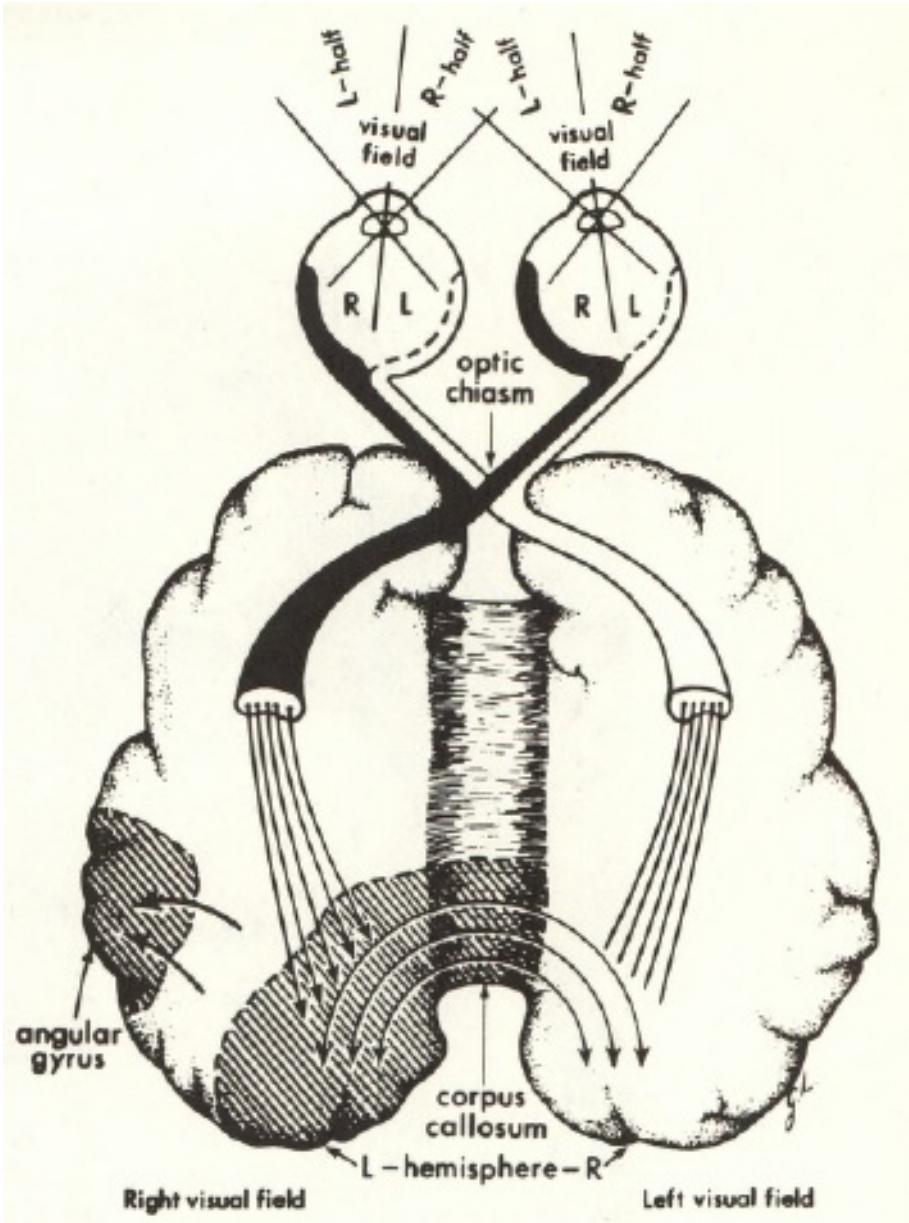


A contemporary diagram of speech cortex, illustrating structures which are presumed to be involved in the production of aphasia, i.e., Broca's area in motor aphasia, Wernicke's area in jargonaphasia, angular gyrus in anomia, and arcuate fasciculus in "conduction" aphasia. (From "Language and the Brain," by N. Geschwind. Copyright c 1972 by Scientific American, Inc. All rights reserved.)

Anomia (amnestic, nominal aphasia) is due to lesion of parietal cortex (angular gyrus), but does not have the strong localizing features of the other syndromes. The transcortical aphasias occur with selective preservation of the primary speech zone. Disorders of reading, writing, and praxis are aligned with this anatomical account through interpretations based on the effects of lesion of the *corpus callosum.* For example, the syndrome of "pure alexia" or word blindness is explained through the destruction of the left occipital

cortex and the splenium of *corpus callosum,* which produces a state in which the patient presumably can see written words in the intact left visual field but is unable to read because of interruption of callosal fibers conveying the perception of these words to “speech cortex” for language analysis (Figure 10-6).

Figure 10-6.


L-half
R-half
L-half
R-half
visual field
visual field
R
L
R
L
optic chiasm
angular gyrus
corpus callosum
L – hemisphere – R
Right visual field
Left visual field

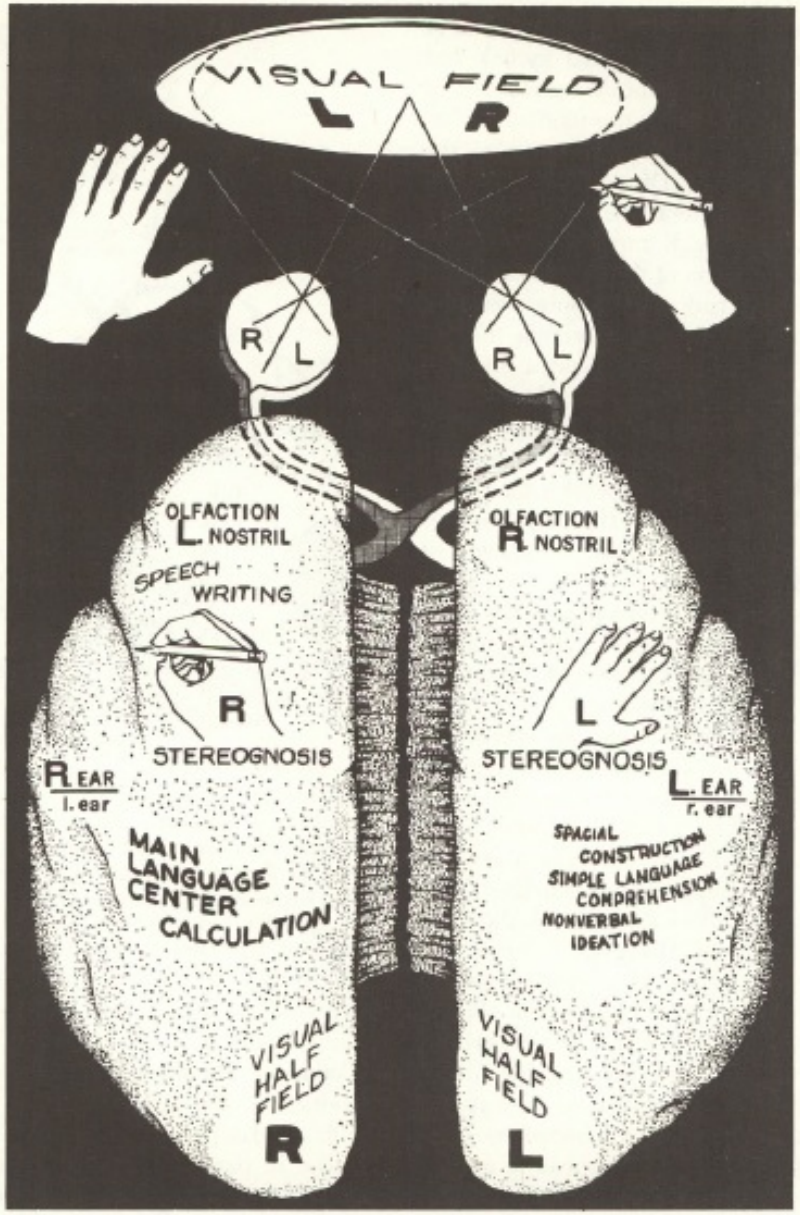
According to the classical account of pure alexia, a lesion of left occipital cortex and splenium of corpus callosum results in an interruption in the flow of visual information to left speech cortex, while lesion of angular gyrus leads to alexia with agraphia. This condition, however, can be explained on a perceptual basis through a reduced functional level in right occipital lobe.

This classical account of aphasia has been reinforced through findings in patients undergoing complete surgical section of the *corpus callosum* as a form of treatment for epileptic seizures. Two recent reviews by Dirnond and Gazzaniga are available. Following this operation, patients demonstrate a relative inability to name objects tactually with the left hand, or read material presented tachistoscopically to the left visual field, nor can they carry out to command skilled actions with the (distal) left extremities. However, patients are able to identify the tactual or visual object or word by selecting it (the appropriate object) from an assortment, if this is done nonverbally and with the left hand. This has led to the conclusion that right-hemispheric contents are isolated from dominant left-hemispheric, and that, to some extent, one can speak of a separate consciousness in each hemisphere. Evidence for a left-hemispheric priority in verbal tasks, and a right-hemispheric priority on visual-spatial performance, has given rise to speculations regarding different forms of thought in each hemisphere. Such considerations range from the improbable (Figure 10-7) to the absurd. The wide interest in studies of this type, and the readiness with which many students accept the simple interpretations offered, suggests that we are at the beginning of a wave of neophrenology that could lead to a long unproductive period in

neuropsychology.

Comment. There is no question but that the introduction of linguistic concepts and methods has had a profound effect on research in aphasia. In particular, interest in transformational grammar, and experimental studies stimulated by this model, have helped to bring about a considerably more dynamic approach to the problems of aphasia than has characterized the field in the past. There is also increasing dissatisfaction with previous theories of aphasia. This includes those on the one hand in which some common element is isolated from the symptomatology and then employed to explain all the other symptoms, e.g., as has occurred in regard to "abstract attitude," Gestalt formation, etc., as well as, at the other extreme, accounts in which a specific (disordered) function is proposed for each element of the symptom complex, e.g., as in stimulus response or association theories of aphasia.

Figure 10-7.


VISUAL FIELD
L R
R L
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OLFACTION
L. NOSTRIL
OLFACTION
R. NOSTRIL
SPEECH
WRITING
R
L
STEREOGNOSIS
STEREOGNOSIS
R. EAR
l. ear
L. EAR
r. ear
MAIN
LANGUAGE
CENTER
CALCULATION
SPACIAL
CONSTRUCTION
SIMPLE LANGUAGE
COMPREHENSION
NONVERBAL
IDEATION
VISUAL
HALF
FIELD
R
VISUAL
HALF
FIELD
L

A more extreme representation of left and right hemispheric functional asymmetry. (From "Perception in the Absence of the Neocortical Commissures," in D. A. Hamburg, K. Pribram, and A. Stunkard, eds. Perception and Its Disorders. New York: Williams & Wilkins. Reprinted with the permission of the publisher and the Association for Research in Nervous and Mental Disease.)

Psychological models of aphasia must, it would seem, conform to the constraints imposed by pathological correlations of aphasic syndromes. The pathology of aphasia is neither obvious nor random but is a subtle clue to the anatomy and organization of normal language. Both language production and the anatomical structure by which it is supported develop in an orderly way. In pathology, the change in language and the change in structure are inseparable and equally lawful. Structure is not a rigid skeleton on which function is superimposed, but is an organic form created by the continuous flow of process. Seen in this light, the *combined* study of aphasic language and of its correlated brain pathology appears to be the most trustworthy guide to an understanding of the structure of real language.

Typology of Aphasia

Introduction

Language develops through a formative or microgenetic process as a component of cognition. There are several more or less arbitrary stages in this process, though normally we are aware of only the final product. In

various states, for example during sleep or hypnagogy, one may see these earlier, otherwise concealed (i.e., traversed), levels appearing as pathological speech forms. Generally this is a transient phenomenon. However, with structural brain lesion the "earlier" stage, the aphasic syndrome, may become the final speech product and this product may persist indefinitely as a relatively stable form. Each type of aphasia, therefore, can be conceived as a preliminary level in normal language which pathology has brought to the fore. Moreover, at each of these levels, the "pathological" language form, the aphasia, also points to a corresponding level in cognitive development. Thus we may study an aphasia both from the point of view of language, as a manifestation of a prefigurative stage in the normal process, and from the point of view of cognition, as exhibiting features characteristic of whatever cognitive stage happens to be realized in the momentary language level.

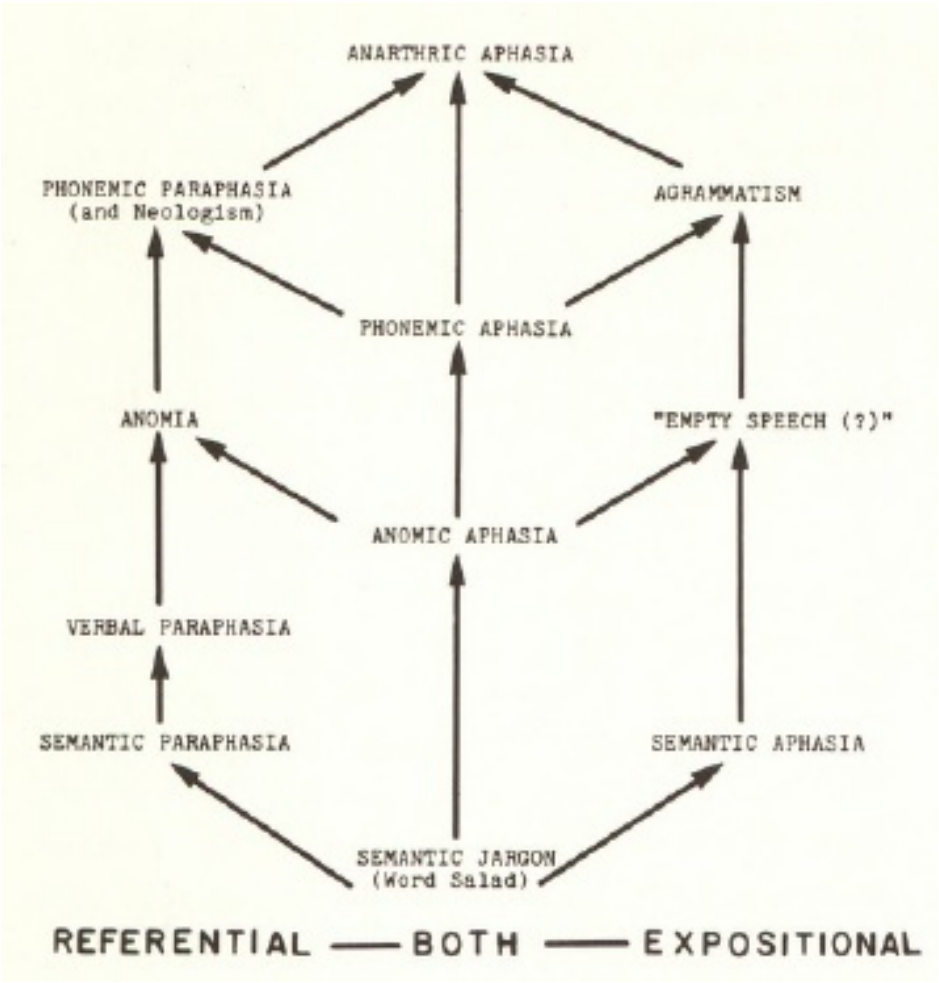
When we look at aphasia from this standpoint, questions arise concerning some of the most basic aspects of brain study. For example, the view that an aphasic syndrome is the result of a combination of two or more discrete defects must be treated with great caution. Wernicke's aphasia is not word deafness *plus* verbal paraphasia *plus* anosognosia *plus* euphoria, but rather is a defect in cognition at some level where processes underlying these disorders (rather, achievements) are coextensive. The aphasic syndrome represents a molar level to which the patient has been reduced and is not a compilation of disorganized functions.

This, in turn, has implications for our understanding of *severity.* Within the posterior or fluent aphasias, for example, it would be misleading to speak of a severe jargon or a mild paraphasia. This ignores the change in the *qualitative* aspects of the jargon, or the paraphasia. When semantic jargon deteriorates it may become neologistic; when verbal paraphasia deteriorates it may approach semantic jargon. The central point is that an alteration of one element in the disorder, say in comprehension or in repetition, will always be accompanied by a change in other elements as well. If there is sufficient change the result is a new syndrome and not just a more severe manifestation of the original condition. We may say that severity in a microgenetic system always entails a difference of kind as well as degree.

In the classification that follows, the aphasias are arranged in such a way as to reflect the sequence of stages in normal language production (Figure 10-8). This sequence unfolds on an axis between a semantic or selectional process and a stage of phonemic encoding. The precursors of the words, the forms or clusters of the utterance-to-be, emerge through a semantic operation by means of which the developing utterance is shaped in the direction of the final performance. At this stage, there is a "noun priority" in the entry of lexical items into the forming sentence pattern. A transition then occurs from the ordered abstract-sentence frame to the phonemic representatives of the constituent words in preparation for articulation. At this stage, the small (function) words are introduced. In the course of this

process both a referential (i.e., nominative) and an expositional (i.e., discursive) orientation can be discerned, a discovery which has helped to clarify some of the complex interrelationships between these forms.

Figure 10-8.



The aphasias can be aligned in a transitional series corresponding to the sequence of stages in normal language production. These relationships are especially evident in the course of recovery or deterioration. In pathological states the arrows are to be considered bidirectional. A distinction is made as to whether there is preferential involvement in referential or expositional speech (partial aphasic forms) or both (major syndromes). Neologistic jargon is not depicted but represents involvement of both the semantic and

phonemic levels.

Apart from its linguistic character, each of the aphasias incorporates aspects of a corresponding level in cognition. A change in awareness or in affect, the presence or absence of delusional or hallucinatory phenomena, these are not additions to the clinical picture but have an inner bond with the aphasic form. These alterations in cognition will be briefly noted in the description of each syndrome and more fully discussed in the final section. Reference will be made to the pathological "locus" of each type of aphasia, reserving a more thoroughgoing discussion for the following section.

Description of the Aphasias

Semantic Disorders

Semantic Jargon. This is basically a disorder of word meaning that involves both naming and conversational speech in the presence of moderate loss of oral comprehension. The disorder is associated with a lesion of the posterior-middle and superior-temporal gyrus (posterior T_2 and T_1), often bilaterally. In older patients the lesion is more commonly unilateral and on the left side. Semantic jargon is one form of Wernicke's (receptive, sensory) aphasia. Such patients produce good words and sentences, but with defective meaning. An example from Alajouanine et al. is a patient who described a *fork* as "... a need for a schedule" or another who defined a *spoon* as ". . . how many

schemes on your throat." Another patient, asked about his poor vision, said "My wires don't hire right." A case of Kreindler et al. replied to a question about his health with: "I felt worse because I can no longer keep in mind from the mind of the minds to keep me from mind and up to the ear which can be to find among ourselves." A patient of Heilbronner responded to a similar question with "Yes, I think that I am now so safe than now much with others to some extent directly." Occasionally, neologisms are present which may lead to strikingly bizarre utterances. Thus an aphasic physician, asked if he was a doctor, said "Me? Yes sir. I'm a male demaploze on my own. I still know my tubaboys what for I have that's gone hell and some of them go."

Speech production is fluent, there is no word search, in spite of incorrect choices, and vocabulary use is fairly good, at times even pretentious. There is semantic or verbal paraphasia on tests of naming and repetition. This refers to a substitution of one word for another, e.g., "table" for "chair." However, in semantic jargon the link between the substitution and the target word is often not so clear as in the "in-class" substitution of this example. Rather, a patient might call a chair an "engine," or an "Argentina." The term *semantic paraphasia* can be used for this latter type of substitution, and *verbal paraphasia* for categorical substitution.

Comprehension is moderately impaired, though ordinarily some understanding is possible, while reading aloud and writing show alterations

parallel with speech. About 20 percent of such patients are hemiparetic, the rest often ambulatory and with few or no "hard" neurological findings. In such patients, a distinction from psychotic language or thought disorder is frequently difficult. This is particularly so in view of the fact that there is commonly a euphoric, even manic, mood elevation or aggressivity, and auditory hallucinations may occur during the course. A paranoid state is not uncommon, and may make speech therapy difficult or impossible. Patients tend to be logorrheic, and show partial or complete absence of awareness of their defective speech. However, they usually reject jargon spoken by the examiner, and resist efforts at correction of their own speech. The awareness of speech content, as with all other elements of the syndrome, may change from moment to moment. Awareness appears to be inversely related to the semantic "distance" of the utterance from its presumed goal.

This stage of unintelligible semantic jargon may resolve in one of two directions, to involvement of expositional speech with intact naming, or involvement of referential speech (naming) with preserved conversation. The former is termed "semantic aphasia," the latter (pure) "semantic paraphasia." Both of these disorders occur with bilateral temporal-lobe pathology.

Semantic Aphasia. This disorder was first described by Head as an interruption at a prelinguistic phase in the thought-speech transition. Patients demonstrated a want of recognition of the full significance of words

and phrases apart from their verbal meaning. There was a failure to comprehend the final aim or goal of an action and an inability to clearly formulate a general conception of what was heard, read or seen in a picture, although many of the details were enumerated. Memory and intelligence were relatively intact, counting was possible, but calculations were impaired and there was a failure to understand jokes, games, and puzzles. In Head's descriptions the recorded statements and short letters of his cases do not always convey to the reader the full flavor of the defect as emphasized in the commentary. Nor did his spatial tests clearly illustrate the specific nature of the disorder. However, most of his cases demonstrated some grammatical disturbance. Thus one patient wrote: "Just a few lines to let you know that I am getting on all right and I shall will be home again. I must tell you that Uncle George and Aunt Ann cane (came) and see me yesterday and more so Bob Higgins so I am very Lucky for getting friends." On another occasion, this patient remarked, "I was worked for . . ." Another patient wrote: ". . . one could spend one's time in a more profitably . . ." and another said: "If I pay too much attention I get wrong with what I've got to do." Another patient said: "My son is just home from Ireland. He is a flying man. Takes the ship about to carry the police to give information, to carry the letters of the police."

More recent studies show that in semantic aphasia there is a disturbance of contextual meaning, through which utterances of *skewed meaning* are produced. The disorder is especially prominent in proverb, story,

or picture interpretation. Consider this example of a patient's written description of his speech (patient's capitals and punctuation).

> Speech that could be found as a Type of speed I believe. I possible mood of my own maybe because of misunderstanding. Possibly because of my own thought In a certain way. a friend of mine told myself. I had a "cast Iron Fact" especially during a conversation, [p. 45]

The disorder is apparent in speech and writing. Speech is fluent and somewhat logorrheic, and may have a confabulatory flavor (see below). Comprehension may be quite good, while naming, reading aloud, and repetition are intact. Spatial-constructional difficulty may or may not be present, and the neurological examination can be normal except for the aphasia. Patients tend to be euphoric with partial insight into their disability. Paranoia and hallucination are not prominent features, but too few cases have been described to be more precise on this point.

Semantic Paraphasia. In this disorder which has been (incorrectly) termed "nonaphasic misnaming," conversational speech is fairly well preserved but errors occur on tests of object naming. These take the form of "associative" responses, e.g., a pipe is called a *smoker,* glasses a *telescope.* The pretentious and facetious quality of the paraphasia appears when a doctor is called a *butcher* or a syringe a *hydrometer to measure fluids.* The paraphasia affects about 10-15 percent of names produced, depending upon test item. Although the object-naming difficulty may follow a word-frequency

distribution, this not true for the paraphasic response.

The disorder usually occurs in the context of diffuse disease, drowsiness, or confusion. Speech is fluent, at times logorrheic, but not clearly aphasic. Comprehension is good and repetition is preserved. Patients show euphoria, reduced speech awareness, and/or denial. There is a similarity with certain Korsakoff patients who may also show semantic paraphasia restricted to naming tasks, as in the Korsakoff patient who referred to the examiner as "Herman Joseph Prince Macaroni."

Mechanism of the Semantic Disorders. Three disorders of semantic origin have been described: (1) semantic aphasia, when context (expositional speech) is primarily affected; (2) semantic paraphasia, with disturbance in referential speech; and (3) semantic jargon, when both reference and context are involved. The fact that the first and second forms occur independently indicates that neither is a partial expression of the other, though semantic jargon may be taken as a combination of the two. The mechanism which accounts for the disorder is similar in both the expositional and referential forms. In semantic aphasia the speaker is unable to use the verb or predicate of the forming utterance as a free unit to which the subject and object only partially relate. A combination of any two of these elements (e.g., subject and verb, or verb and object) tends to determine the third. The direction of this pressure is not invariably subject → verb → object, but is often the reverse. All

lexical items may be affected, and it may be difficult to determine which element of the phrase is defective (if content words, paraphasia; if function words, paragrammatism). In the above example, "speech that could be found . .." acceptable bondings occur between individual words (speech that, that could, could be found) but not between the initial and latter segments of the phrase. The disorder has a close relation to schizophrenic speech. Consider an example from the study of paralogic by von Domarus, quoted by Arieti:

Certain Indians (A) / are / swift (x)
Stags (B) / are / swift (x)
∴ Certain Indians (A) are stags (B)
Here, A ≅ x becomes A = x
B ≅ x becomes B = x
A ≅ x ≅B becomes A = x = B

This is quite similar to what occurs in semantic aphasia. In the following example from a Cloze test an aphasic patient was required to insert words deleted from a test phrase. The patient's solution is in brackets. Test phrase is "The baby—something that he had—done before." [p. 49]

A.	x.	B.
The baby	[was]	something
that he had	[been]	done before

Here A ≅ x and x ≅ B becomes A = B

The inserted word agrees with those in its immediate surround (e.g., baby *was, was* something; had *been, been* done) and a partial fit is accepted as

satisfactory. Responses to proverb tests show identical errors, the patient generally interpreting one component of the proverb partially and then attempting to consolidate it obliquely to the other components.

In semantic aphasia the noun phrase tends to become stabilized at the expense of its predicative relationships. Context is adapted to subject. One might say that the noun phrase conditions the predicate rather than being contained within, or defined by, it. This has a determining effect upon utterances in which topics are developed within understood contexts. In semantic paraphasia (see below), misnamings show the influence of implicit contexts derived from the examiner's knowledge of the object to be named. However, predicative or contextual function is otherwise adequate and acts to normalize noun production in conversational speech.

In semantic paraphasia there is an identification of two otherwise disparate subjects (e.g., "doctor" and "butcher") on the basis of one or two shared attributes (e.g., white coat, cutting, etc.). Consider the following example:

Task	*Presented object A*	*Shared predicate C*	*Paraphasic response B*
Naming	bedpan	stool, sitting, etc.	"piano stool"

Mechanism

$A \cong B$

$B \cong C$

$\therefore A = C$

Related Disorders. Similar language disturbances have been noted in schizophrenic patients. For example, the utterance "A boy threw a stone at me to make an understanding between myself and the purpose of wrongdoing." is similar in structure to that of semantic aphasia, while Arieti's example of word salad, "The house burnt the cow horrendendously always," is very close to semantic jargon. The disorder of semantic paraphasia is recalled in the "associative" misnamings of schizophrenic patients, as in "le song" for bird, "le kiss" for mouth. Similarities between schizophrenic and aphasic speech have also been discussed by Schilder, Critchley, and Alajouanine. Arieti has given a full and lucid discussion of the problem of schizophrenic language, and has demonstrated the central position of paralogical thinking.

Kleist commented that paralogia was a confabulation within the verbal sphere. This concept is probably identical with the "confabulation d'origine verbale" of catatonic patients. If paralogia is a kind of "verbal" confabulation, it may be asked to what extent this relates to the confabulation of Korsakoffs syndrome and related confusional states. Language of this type has been described in Korsakoff's syndrome, as in the response of a patient to the proverb *Safety First,* "It's rather a lateral term which means it could apply to a host of things. A road for one thing." Victor has commented that aphasic errors are common during the confusional prelude of the amnestic syndrome.

It is likely that an inner bond exists between the semantic aphasic complex and confabulation. Confabulation is no more the "filling in of a gap in memory" than is paraphasia a compensation for a memory loss. In confabulation there is substitution *of* a semantic field, in semantic paraphasia, there is substitution *within* the semantic field. The two speech forms reflect the microgenetic *level* of disruption and are not unrelated psychological deficits. In this respect it is of interest that patients with semantic jargon have often been described as having features of the Korsakoff syndrome. Moreover, the possibility that an inner relationship exists between amnestic confabulation and schizophrenic paramnesia has not received sufficient attention.

Nominal Disorders

The developing linguistic form, having more or less successfully traversed the semantic or selectional stage, proceeds toward the "abstract representation" of the (correct) lexical item. Disorders at this level are, therefore, characterized by improved control of word-meaning but inability to evoke the intended word. As with the preceding stage, anomia is not a single entity but is rather a series of (pathological) speech forms which point to one or another segment or phase of the process of language production. A disturbance at this stage may occur to some extent independently in referential speech (as in anomia proper, i.e., word-finding difficulty) and in

expositional speech (so-called *empty speech* of anomia, circumlocution). Verbal paraphasia occurs as well, and is to be distinguished from semantic paraphasia, with which it has generally been equated, on the basis of the "in-class" substitutions ("shaver" for razor, "green" for red). Verbal paraphasia is to be conceived as an intermediate stage between semantic paraphasia and anomia proper.

Background. The concept of verbal amnesia as a defect in the mental evocation of words was an early development in aphasia study. As a distinction was drawn between internal and external speech, verbal amnesia, as a disturbance of the internal phase of language, came to be set against motor aphasia, which was a disturbance of the external phase. This early view gave way to a division of anomia into specific visual, auditory, tactile, and motoric forms, and for a time the concept of a pure anomia regardless of sensory modality was abandoned (see Pitres, for a review of the historical period). The modern conception of anomia dates from the papers of Kurt Goldstein.

According to Goldstein, the difficulty in naming objects derived from an inability to assume an "abstract attitude" with regard to the item being tested. Words which could not be produced as names, or which could be produced but not brought into relation with the object designated, appeared spontaneously in conversation. This indicated that word memory was

preserved. Thus it must be the conditions under which the word *is evoked* that are altered, viz., a loss of the ability to apply words as symbols for objects, i.e., as word concepts. This difficulty became even more apparent if the patient was asked to sort objects according to various attributes such as color, size, or shape. The inability to give the name of a single object reflected a disturbance of the word concept of that object, and this disturbance was exaggerated by the requirement that diverse objects be categorized according to shared attributes.

Goldstein's description of amnesic aphasia (anomia) achieved wider acceptance than his psychological account. It was pointed out that abstraction was frequently impaired in the absence of anomia, and that anomia occurred with categorical behavior that was not strikingly abnormal, or if so, no different from that seen in other aphasic syndromes. Also to be included in this period are works by Heilbronner and Lotmar, particularly as concerns verbal paraphasia. Lotmar especially discussed the spheric nature of word substitution, and attempted to show how apparently random substitutions occurred through intermediate links.

Recent studies have shown that word frequency is an important factor in the anomic defect. It has been shown in normal subjects, in dysphasics, and in patients with organic dementia, that word-finding difficulty relates to the vocabulary frequency of the target item, i.e., the object or action to be named.

In a study deriving from this work, A. Wingfield, cited by Oldfield, demonstrated that perceptual identification does not show the same frequency dependency as does object naming. This led Oldfield to propose a two-stage model of naming, an initial stage of perceptual identification and a second stage of word finding, only the latter of which is dependent on word frequency. There is some evidence that the specific anomias (e.g., "visual" or "tactile" anomia), and true or aphasic anomia relate to involvement at each of these respective stages.

Verbal Paraphasia. This disorder refers to a stage where the lexical item, the word, has realized (been selected to the point of) a categorical approximation, e.g., "shaver" for razor, "green" for red. There is some ability to self-correct, i.e., some awareness of speech error, but this may differ from one moment to the next, depending on the nature of the substitution. Although the difficulty in naming may have a relationship to the vocabulary frequency of the target word, i.e., patients having more difficulty with rare than common words, the paraphasic errors do not appear to show this effect. Thus, patients may say "spectacles" for glasses, or "fuchsia" for red. While this form of language is often admixed with other anomic features (see below), the absence of verbal paraphasia in anomia proper should not be interpreted as a reluctance to speak or a more careful search for words. Verbal paraphasia is not a reflection of personality type; rather it reflects a cognitive level around which the "personality" is organized. Features of this cognitive

level include some degree of euphoria, a more active, though not logorrheic, speech flow than in anomia, and partial awareness of the disorder.

Anomic Aphasia (anomia, amnesic or nominal aphasia). Patients of this type have difficulty in word finding which affects nouns preferentially. Typically, such patients can point to the correct object when it is named, can repeat the object name, and can select the correct name from a group, although they are unable to name the object directly. This is true for "visual naming," as well as naming through other perceptual modes, e.g., touching the object, hearing the sound of the object, etc. Patients are also unable to name from a description or definition of the object, e.g., "what do you use to sweep the floor?" The word-finding difficulty may be akin to the common phenomenon of word lapse or the forgetting of a name or place in the speech flow. Not uncommon is the incipient "tip-of-the-tongue" nature of the needed word. Patients may be able to give the initial letter of the target word or the number of syllables, and can use the test object appropriately. These features suggest that word meaning is relatively well preserved and that some "skeleton" or abstract frame of the intended word is available. The disorder may be limited to referential speech, or may appear in conversation with circumlocution and emptiness of speech. The true anomic who does not produce verbal paraphasias has a more acute awareness of his difficulty and may show frustration and catastrophic reactions.

The difficulty in word finding tends to occur in the following direction: nouns → verbs → grammatical (function) words. Abstract nouns may be more difficult than concrete nouns. When the disorder involves both referential and expositional speech, a "nonfluent" state can result. Such patients have greatly reduced speech with only a starter phrase or a stereotypy available, such as "Well I . . ." or "It's a . . ." Speech tends to be limited to small grammatical words and simple verbs. This condition can be distinguished from anterior nonfluency (i.e., Broca's aphasia) by the reciprocal order of word loss. In the anomic, the small verbs and function words are the last, not the first, to disappear.

Word-finding difficulty occurs in various organic and nonorganic states. Anomia and circumlocution have been described in schizophrenia. Chapman has emphasized that schizophrenic patients "have a true difficulty in word finding, although it tends to be episodic in occurrence and very similar to the paroxysmal dysphasia which occurs in temporal lobe epilepsy." Anomic errors are also common in fatigue and distraction, and in sleep and transitional utterance.

Anomia tends to be associated with either unilateral or diffuse lesions. In anomia and in verbal paraphasia, lesions may occur outside the classical speech areas. The more severe "nonfluent" anomia occurs with unilateral (left) temporo-parietal lesion. Lesions of the posterior middle-temporal gyrus

(T_2) and its continuation to angular gyrus appear to be highly correlated with this form. The more fluent the anomia, the more likely is diffuse pathology or lesion outside the speech area.

Anomia occurs in dementia, increased intracranial pressure, postanaesthetic or confusional states, as well as with subcortical or thalamic lesion, where it is most likely due to a referred effect on cortex.

Comment on the Semantic and Anomic Disorders. The various disorders which have thus far been discussed can be aligned in a series which retraces the microgenetic development of normal language. The sequence of semantic jargon, through associative and then categorical substitution to true anomia, corresponds to stages in the normal productive process. Within the semantic "segment," the progression is through systems or fields of word meaning of wide "psychological distance." These lead to more narrow "associative" responses which represent an intermediate stage between semantic jargon and correct word selection. Anomia points to a stage where the correct word has been all but selected but cannot yet be fully realized in speech. The anomic stage corresponds to the emergence of the correct lexical item preparatory to phonemic encoding.

In addition to this linguistic change, there is an evolution of other aspects of cognition. Thus, in semantic jargon there is euphoria, at times

mania, often with a paranoid trend. There is logorrhea and a lack of awareness of speech error. This picture gives way in semantic aphasia and semantic paraphasia, to a mitigation of logorrhea and euphoria, with patchy but still incomplete awareness of difficulty. This continues into verbal paraphasia where incorrect words (e.g., "table" for chair) can often be rejected. There is a transition from active, but not logorrheic, speech to hesitancy, and finally to an inability to speak at all. The transition from one state to another occurs *pari passu* with increasing awareness of speech errors, improved self-correction, and step-by-step transformation from one affective and behavioral form to another.

Phonemic Disorders

These disorders point to a stage in the production of language where the intended word, having been properly selected, does not achieve correct phonemic realization. According to whether the defect is expressed primarily in referential or expositional speech, we can distinguish, respectively, phonemic paraphasia and phonemic aphasia. Ordinarily these are included together in the syndrome of central or conduction aphasia.

Background. The phonemic disorders were originally defined on an anatomic basis by Wernicke without regard to the qualitative aspects of the speech of such patients. The disturbed function of repetition was gradually

singled out as central to the syndrome and attributed to damage to a pathway between the posterior and anterior speech areas (see Brown for further discussion). Kurt Goldstein argued against an interruption of a conducting pathway in favor of a more dynamic interpretation. Goldstein believed the condition represented an impairment at the thought-speech transition, and termed it "central" aphasia, placing emphasis on the paraphasia as reflecting a disturbance of inner speech. Goldstein's comments regarding a possible relationship between anomic aphasia and central (phonemic) aphasia are worth quoting in full:

> A combination of amnesic aphasia with symptoms of central aphasia is frequent. There arises the question of whether we are dealing with an accidental combination due to similar locality of the underlying lesion, or whether there is an inner relationship between both defects. As little as we are able to say now, the latter possibility is worth pondering in respect to the closeness of the phenomenon of inner speech to the nonspeech mental process, [pp. 277-278]

Phonemic Paraphasia. In this disorder, the disturbance chiefly affects nouns and is apparent on tests of object naming. Spontaneous speech is often quite good with few or rare paraphasias. Patients make errors of the type: "cable" for table, or "predident" for president. Repetition may be involved in a similar manner. Comprehension may be quite good. Such patients are usually classified as mild "conduction" aphasics or resolving "motor" aphasics.

Phonemic Aphasia (central, conduction aphasia). When conversational

speech shows a picture of fluent phonemic paraphasia with phonemic errors on naming and repetition tasks and good comprehension, the diagnosis of phonemic aphasia is in order. There is a close resemblance to phonemic paraphasia, the distinction resting on the improved speech and defective naming and repetition of the former, and the more impaired spontaneous speech of the latter, where naming may be relatively well preserved and repetition is involved at the phrase, rather than single-word, level. This disorder may be present at the start and may appear in the course of a deteriorating anomia and as a stage in the recovery of a neologistic jargon (see below). An example of such speech is that of a patient who, when asked where she lived, said: "I have been spa staying with a friend of mine but I do hate to imp impose on her. I want to pay my own way. Do they have some sort of chart where you can take this tee tee . . ." When phonemic aphasia develops out of a neologistic jargon (q.v.), speech is more active with some neologism and comprehension is less well preserved. Such a patient described his speech difficulty as: "Well it's very hard to because I don't know what it would my pi why what's wrong with it, but I can't food, it's food and rood to read the way I used to do all right off . "

The disturbance is equally present in naming and repetition and in a manner generally comparable to conversational speech. This is particularly evident when phonemic aphasia appears in the deterioration of an anomia. Thus, if an anomic patient is asked to name an ashtray, the word is not

produced but can be repeated. In the regression of the anomia, the patient will first fail to cue with the initial sound of the word, i.e., when the examiner says “ash . . . ,” but will still repeat the word “ashtray.” At a later stage failure will occur in spite of a strong phonemic cue, e.g., “ashtr . . . ,” in which all but the final syllable of the word is given, but the word “ashtray” can still be repeated. Ultimately a stage is reached where the patient can neither cue nor repeat. At this point the patient is a phonemic (conduction) aphasic. In this example we can see that the *disorder of repetition is only a failure to name given the whole word as a cue.* The transition from the *anomic,* who repeats the word but fails to name with a cue up to the penultimate syllable, and the *phonemic aphasic* who fails given a cue including the final syllable (i.e., on repetition) establishes a functional continuity between these two disorders. There is a different speech form in these patients since the phonemic aphasic has achieved a linguistic level beyond that of the anomic. There is also a heightened awareness of speech content. Circumlocution has given way to deficient production, frustration to selfcorrection.

With regard to anatomical correlation, the evidence suggests that dominant posterior-superior temporal gyrus and its “parietal continuation” as supramarginal gyrus are chiefly involved. Cases with a lesion of angular gyrus have been reported, as well as instances in younger patients with a lesion limited to the left Wernicke’s area.

Phonemic aphasia is uncommon in non-organic states, but phonemic errors may occur in speech during fatigue or distraction. An example of such errors in normal sleep utterance is the following: "David, I day (?say) David . . . that's you that day dated day dravid Dave dravid about 25 or 30 noked naked day dreams." The "clang association" is more prominent than is generally seen in phonemic aphasia, although clang errors are prominent in neologist jargon (see below).

The Problem of Neologism

Aphasic jargon with neologism is a disturbance altogether different from semantic jargon, although both disorders are often treated as different manifestations of Wernicke's aphasia. As in the semantic, nominal, and phonemic disorders, there may be two expressions of the defect, in referential speech, as neologistic paraphasia, and in both referential and expositional speech, as neologistic jargon.

Neologistic Paraphasia. In this disorder, speech is generally comprehensible with occasional neologism, often in the context of fluent phonemic paraphasias. The neologism appears especially when a highly specific response is demanded, e.g., on proverb interpretation, and under the conditions of naming. An example is the following, from a patient who was questioned about his work: . . it was my job as a convince, a confoser, not

confoler but almost the same as a man who was commersed." Another patient described her accident in this way, "So when I passed drive I told him let me drive. I had go so he let me go, so I went, wen in and went in on the semidore." The neologism primarily affects content words with relative sparing of the small grammatical words. The disorder is probably closely allied to phonemic aphasia and paraphasia, the neologism at times appearing as a phonemic error severe enough to render the word unintelligible.

Neologistic Jargon. This disorder refers to speech so pervaded by neologism that it is no longer intelligible. The neologisms may range from wordlike products to a series of clang contaminations. Thus, one patient responded to the idiom "swell-headed" with the interpretation, "She is selfice on purpiten," while at another time, asked about her speech problem, she said: "Because no one gotta scotta gowan thwa thirst gell gerst derund gystrol that's all." A progression may be seen from fluent, logorrheic neologistic speech with few clang associations, to reiteration of certain neologisms and perseverations on the basis of sound similarity to clang association so intense that it seems to determine the jargon output, e.g., "Then he graf, so I'll graf, I'm giving ink, no, gefergen, in pane, I can't grasp, I haven't grob the grabben, I'm going to the glimmeril let me go."

In such patients, comprehension is severely impaired. Naming and repetition are characterized by neologistic responses, e.g., "galeefs" for comb,

"errendear" for yellow. There is a lack of awareness of speech errors, and patients will gesture actively, seemingly convinced that they are communicating something to the examiner. There is heightened affectivity, often with euphoria and exaggerated expression. It is of interest that patients will appear to accept their own jargon if it is recorded and played back to them, but will reject the same (transcribed) jargon if it is spoken to them by an examiner.

The pathological location of the lesion is in the dominant posterior superior temporal region. There is evidence that the lesion incorporates both Wernicke's area proper and supramarginal gyrus.

In schizophrenia, neologisms are more often of the "portmanteau" type, either as fusions of separate words, e.g., "mondteufel," "cage-weather juice," "snowhousehold," or assimilations of otherwise recognizable components of separate words, e.g., "enduration" for *endure* plus *concentration.* These forms can perhaps be explained along the lines suggested for semantic paraphasia. Occasionally, unintelligible utterances may occur, e.g., "I have seen you but your words alworthen" (Question: What does alworthen mean?) "Ashers guiding the circumfrax." (see Bleuler for other examples). In schizophasic jargon, one may encounter utterances of the type: "Ulrass Asia peru arull pelhuss Pisa anuell pelli." Similar types of jargon may be seen in transitional states, e.g., "amarande es tifiercia," and sleep speech, e.g., "she shad hero sher

sher sheril shaw takes part . . . " A form of aphasic jargon referred to as undifferentiated or phonemic jargon may resemble such utterances, e.g., "Eh oh malaty, eh favllity, abelabla tay kare abelabla tay to po sta here, aberdar yesteday (?yesterday)

Interpretation of Neologistic Jargon. Although the place of neologistic jargon in the aphasias is uncertain, there is evidence that, at least in the most florid cases, it may represent a combination of semantic jargon and phonemic aphasia. In such cases, semantic paraphasias would be produced which would not achieve correct phonemic realization, the result being a phonemic distortion superimposed on a semantic paraphasia. This is consistent with the fact that neologistic jargon tends to improve to either semantic jargon or phonemic aphasia. Thus, if the semantic disorder clears, the patient is left with a phonemic defect, while clearing of the phonemic disorder would reveal the underlying semantic disturbance. In other (milder) cases, however, the neologism probably consists of a normal underlying word frame which is distorted to the point of unintelligibility by phonemic paraphasia. In addition, there are certainly many instances, as illustrated above, where the neologism is a result of clang associations and/or word fusions.

Anarthric Aphasia

Included in this group are disorders at the final stage in speech

production, disturbances affecting expositional speech primarily—as in agrammatism—and disturbances affecting both referential and expositional speech, anarthric or Broca's aphasia. While these disorders are considered *as if* they were impairments at a stage in advance of that involved in phonemic aphasia, viz., at the terminal grammatization and articulation, it may well be that they represent alterations in a motoric or action system organized, not in sequence, but in parallel with posterior linguistic structures.

Background. The historic period (discussed on pp. 243-244) during which the symptomatology and pathological correlative features of Broca's aphasia were worked out, gave way to a series of analytic studies which began with investigations of agrammatism. Isserlin and Pick noted a gradation in Broca's aphasia from mild hesitation and stammering in speech, through agrammatism to a stage of near muteness. The agrammatic stage, characterized by a predominance of nouns and verbs (especially infinitives), lack of prefixes and suffixes, and pronoun confusion closely resembled an early stage of childhood speech.

More recently, Alajouanine has made important contributions to our understanding of stereotypies and speech awareness in the Broca's aphasic. Following the approach and classification of Hughlings Jackson, Alajouanine has emphasized the automatic nature of the stereotypy and the lack of awareness which accompanies it, and he has distinguished four stages

through which the stereotypy resolves. There is an initial stage of modification in which, through intonational adjustments, the stereotypy comes to express a wide variety of emotional states; then a stage of checking the stereotypy which signals the patient's first awareness of the utterance, followed by a transitional period in which other expressions, automatic or not, come to accompany the original, but now impersistent stereotypy; finally, there is abolition of the stereotypy with gradual return of speech into an agrammatic phase.

Sabouraud et al. have characterized the fundamental defect in Broca's aphasia as an inability at different levels to pass from one complete utterance to another. This results from a loss of contrasting features in the expression; i.e. those oppositions which provide for lexical definition are conserved while contextual contrast is lost. Luria has distinguished two forms of frontal aphasia, a kinetic or efferent motor aphasia, and a kinesthetic or afferent motor aphasia. He argues that these two independent conditions constitute what is usually called Broca's aphasia.

Agrammatism (telegrammatism). This disorder is characterized by relatively good use of nouns or substantives and a loss of the small function or grammatical words. This is especially prominent in conversational speech, but is generally present in repetition, reading aloud and writing as well. The disturbance may be present from the start, as in the so-called one-word or

holophrastic sentence, e.g., the patient saying "water" or "glass, water" instead of "May I have a glass of water?" This may improve to more typical agrammatic speech:

> My uh mother died uh, me uh, fifteen uh, oh I guess six months my mother pass away . . . my brother in uh Baltimore an stay all night an 'en I lef' for Florida, Mammi Beach, an uh, an uh, anen uh, Mammi Beach an stay all night and back again. Hitch hike.

With continued improvement this leads to a stage of relatively good speech with loss of inflections, restriction of verbs to the infinitive or present tense and an absence of unstressed grammatical words. Agrammatism has been considered a kind of speech economy, an articulatory defect primarily affecting grammatical words, and a true grammatical deficit. In our view the problem may be considered a deficiency in phonemic realization affecting grammatical words primarily. Since there is a graded entry of nouns, verbs and function words into the forming sentence, i.e., leading from an initial noun priority to the final grammatization, there is preservation of these content words which have already achieved a stage of phonemic encoding. For this reason it is the still developing function words, and particularly the late-added inflections, which are preferentially involved. This helps to explain why the order of word loss in agrammatism (grammatical words → verbs → nouns) is reciprocal to that of anomia (nouns → verbs → grammatical words). In the latter, the nouns are the first to appear and are therefore the first to be lost, whereas in agrammatism the nouns have completed their development

and are therefore the most resistant.

Agrammatism is the commonest form of aphasia in dextrals with right hemispheric lesions and is probably more common in aphasic lefthanders regardless of side of lesion. In such patients, language organization is similar in some respects to that of children in whom, next to muteness, agrammatism is the most common aphasic form. It is also of interest that agrammatism has been described in catatonic schizophrenia, though it is by no means common in this disorder. The pathological localization of agrammatism is presumably the same as for anarthric aphasia.

Anarthric Aphasia (Broca's, motor, expressive aphasia). In this form the usual picture is one of nearly total speech loss, often with no verbalization apart from a stereotypy or automatism. Comprehension may be well preserved but other speech performances are about equally impaired. At times, naming and repetition may be slightly better than conversational speech Such patients may improve to phonemic paraphasia or to agrammatism, depending on whether the content words or the terminal grammatization is chiefly affected. Less commonly there is recovery to dysarthria with abolition of the stereotypy and the gradual return of labored but nonaphasic speech.

In addition, the majority of patients are hemiplegic, and most have facial

and leftsided apraxia. Writing is impaired to the same extent as speech. In cases where writing is markedly superior to speech, a diagnosis of "pure" motor aphasia may be considered, although the existence of this form is now held in some doubt. The term "nonfluency" is often used in relation to such patients. This concept includes a number of disturbances, however, such as dysprosody, dysarthria, agrammatism, and short "phrase length," so that unless the precise characteristics of the nonfluent condition are specified, the concept itself is of little value. Patients with anarthric aphasia tend also to be somewhat apathetic and passive in their behavior. Some writers have commented on the loss of volition or will (*Willenlosigkeit*), an attitude which is, in fact, more common than the frustration or despair often identified with this disorder. At times, one may see apathy give way to euphoric elation or labile crying during the stereotypic utterance. Awareness of the difficulty may change from moment to moment in relation to the dominant speech form, i.e., volitional or automatic speech.

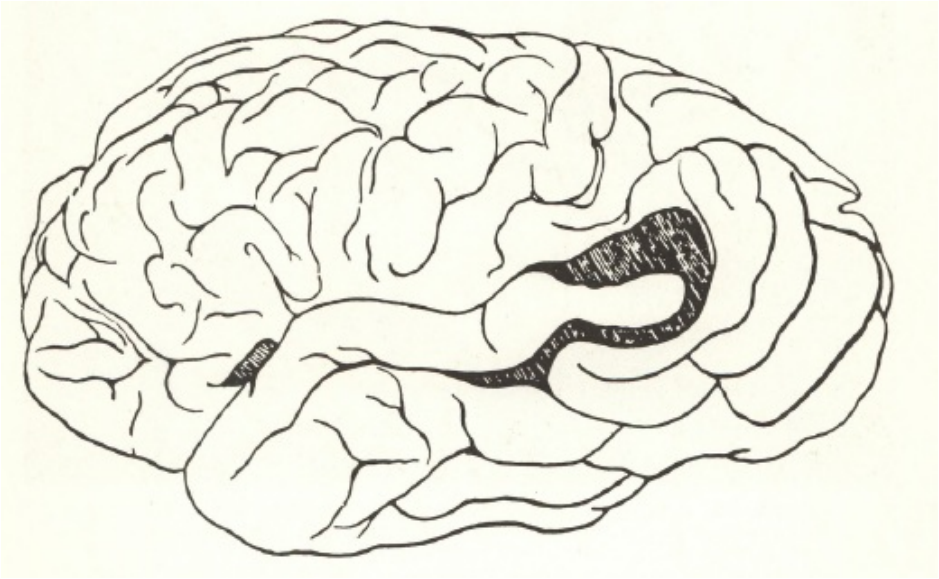
Although there has been much controversy over the exact borders of Broca's area, there is general agreement on the central importance of the posterior part of the inferior or third frontal convolution (F3). Goldstein cited evidence for a more extended speech zone, involving the precentral operculum and mechanisms in this latter area for movement of the mouth, tongue and larynx.

Comment. In the preceding discussion, the major aphasic disorders have been reviewed from the point of view of a model of normal language production. Accordingly, the aphasias represent disruptions of (actually, a coming-to-the-fore of) earlier or prefigurative stages in the formative process. It now remains to bring the transcortical aphasias and the so-called isolation syndrome into relation with this model.

"Transcortical" Aphasia

Background. This group of disorders comprises three major forms, transcortical motor aphasia (TMA), transcortical sensory aphasia (TSA), and combined transcortical aphasia (CTA) or "isolation" syndrome. Common to all forms is the occurrence of good (echolalic) repetition. In respect of the above forms, this occurs in the context of impaired speech, impaired comprehension, or impairment of both speech and comprehension. Historically the concept of a speech area separated from other portions of the cortex was first suggested by Huebner in 1889, on the basis of a single case with loss of speech and comprehension, but relatively good writing, both spontaneous and to dictation, reading aloud and repetition. The brain showed two principal lesions (Figure 10-9), softening around the posterior part of T_1, presumably interrupting connections between Wernicke's area and the parietal-concept field, and a small area of softening in F3 considered (in my view, incorrectly) to be of no importance.

Figure 10-9.



Heubner's drawing of the major lesions in a case of echolalia with markedly reduced speech and comprehension (combined transcortical aphasia, "isolation syndrome").

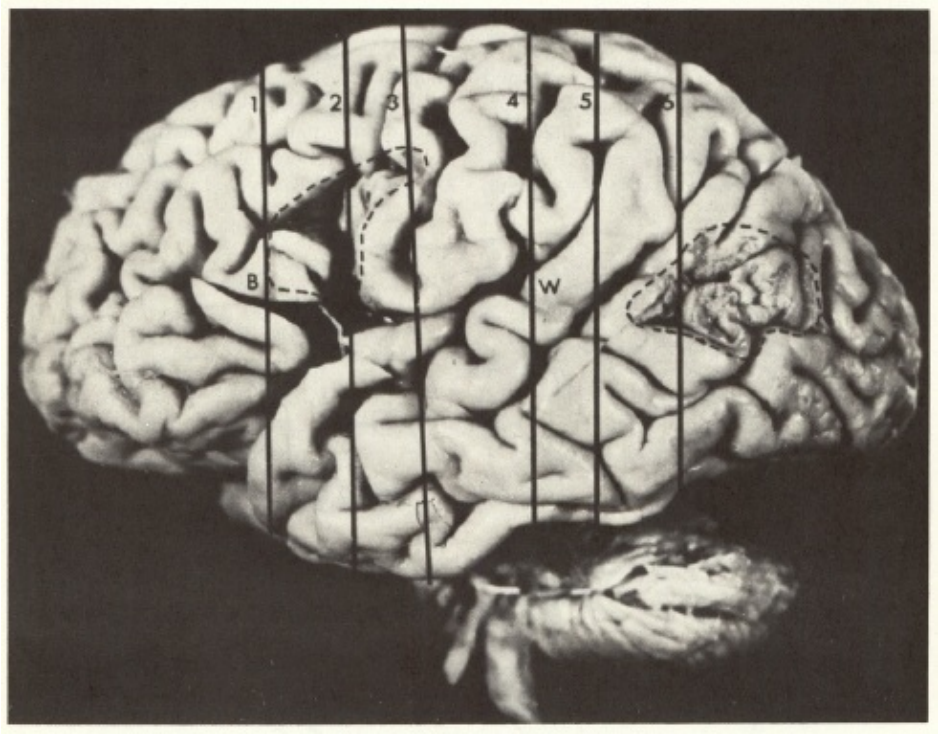
Subsequently, cases of echolalia with temporal-lobe atrophy were described by Pick and Liepmann. The chief clinical feature of these and all subsequent cases is echolalia. This is characterized not simply by the ability to repeat but by compulsive and automatic repetition.

The echo response is a brief, precise and often explosive utterance which differs from the approximations of childhood imitation. Echolalia is not a parrotlike reflex function. There is often "personalization" of the content,

e.g., the patient asked “How are you?” echoes “How am I?” Moreover, it invariably has a social character, the response occurring only when the patient is addressed. There is also a completion effect," patients finishing incomplete rhymes or phrases, e.g., “ham and . . . (eggs).” Patients may also correct in the echo an incorrect grammatical form in the presentation.

In dementia, echolalia occurs with widespread but predominantly temporal-lobe atrophy. In aphasic states, there may be partial lesion of either anterior (TMA) or posterior (TSA) speech areas, or both (CTA) (see Figure 10-10). Echolalia may result from a predominantly posterior lesion assumed to interrupt parietal associations when there is diffuse atrophy or a smaller anterior lesion; or there may be a large infarct in the center of the dominant Sylvian speech zone." Geschwind et al. have described a demented echolalic with diffuse pathology sparing the Sylvian speech area. These authors argued that the *intact* portion of cortex and intervening arcuate fasciculus mediated the echolalic repetition, speech initiation and comprehension having been lost as a result of destruction of the remainder of the cortex. Echolalia is a symptom in a variety of late-stage dementias. It occurs in schizophrenia and mental deficiency. In the latter, it may represent the furthermost stage of language acquisition.

Figure 10-10 .



A personal case of combined transcortical aphasia (CTA). There is a large cystic infarct in the left posterior inferior frontal region, destroying much of Broca's area and extending subcortically to involve the region of traversal of the arcuate fasciculus. There is another area of superficial softening in posterior middle temporal gyrus. The pathology of CTA is a partial lesion of the anterior and posterior speech zone, bringing about a reduced functional level in performances supported or mediated by these areas.

Interpretation of Echolalia. In aphasic states it is not uncommon to have echolalia at the level of single words or very short phrases. This may occur in phonemic and in jargon aphasia, and is a partial expression of the more pronounced echo response seen in the so-called transcortical aphasia.

In the *motor* form of transcortical aphasia, echolalia stands out against a background of reduced spontaneous speech. The pathology of this disorder is incompletely understood, but often there is a partial involvement of Broca's area. In transcortical *sensory* aphasia, there is a more automatic echo response appearing in the context of reduced comprehension. In this disorder the pathology appears to be the subtotal involvement of Wernicke's area. The isolation syndrome may correctly be conceived as a *combined* (motor and sensory) transcortical aphasia.

The anatomical limits of Broca's and Wernicke's areas are defined on an arbitrary basis so that it is unclear how a pathological lesion can be said to "surround" or lie on the periphery of these areas.

It is more likely that there is a *partial* lesion of either the anterior or the posterior speech zone, or both, and that this pathology brings about a deterioration or regression of function within those damaged areas. There is evidence for such partial lesions in all cases described, not only in the two focal cases but in a variety of diffuse atrophies as well.

The need for a more dynamic account of this disorder is emphasized by cases such as that of Stengel (and one of my own cases) where CTA occurs with destruction of the entire (left) Sylvian area. To say that the echo response derives from the opposite hemisphere is not to solve the problem

but only to transfer it to the other side, for it is impossible to say whether the echo response reflects the degree to which the left hemisphere has been reduced or the highest level of which the right hemisphere is capable. Both arguments, in fact, amount to the same thing, since echolalia, like every other aphasic syndrome, is determined by the *combined performance of both residual left and intact right hemispheric capacity.* This concept of a linguistic regression induced by partial (or complete) damage to both of the (left) cortical speech zones, and of the resultant symptom, echolalia, as an *achievement* of the combined action of both hemispheres, helps to bring this disorder into relation with other conditions, e.g., dementia or mental retardation, where echolalia occurs, respectively, as a final stage in deterioration or as an endpoint in development.

The Neural Organization of Language

In historical writings on aphasia, it was generally maintained that the localization of a specific function could be inferred from an impairment of that function with focal pathology, that a lesion of a specific area gave rise to a symptom through disruption of the normal mechanism localized in, or mediated by, the area in question. Gradually, however, it has become clear that the anatomical structure which mediates language and cognition is as dynamic as the psychological systems which it supports. The "centers" of traditional aphasiology may rather be considered as *levels* by means of which

language is carried one stage further. Similarly, the conducting pathways of the classical theory are not to be conceived as channels for the association of ideas, to link up perceptions to movements, or written words to spoken sounds, but are more likely concerned with temporal interrelationships between various levels in the cognitive structure. The nature of the anatomical organization underlying language production can best be understood through a consideration of the process of cerebral dominance or lateralization.

Dominance

Although estimates differ, it is generally assumed that about 85 percent of the population is right-handed, and of these nearly all have left-hemispheric dominance for language. Among left-handers, there is a slightly greater tendency for left-hemispheric language dominance than right. In a large group of unselected patients there is about an 80 percent chance of developing some degree of aphasia with a left-hemispheric lesion regardless of handedness, and conversely, if one looks at an unselected population of right and left handed aphasics, about 95 percent have a left-hemispheric lesion. Furthermore, studies by Brown and Wilson, suggest that hemispheric dominance for speech may be independent to an extent from hemispheric dominance for praxis, and that degree of speech lateralization may be inversely related to the priority of the opposite (usually right) hemisphere in

spatial performance. Among the procedures currently being used to study hemispheric dominance are selective intracarotid amytal injection, dichotic listening and unilateral ECT (electro-convulsive therapy).

Lateralization and the Formation of the Speech Area

Hemispheric dominance or lateralization for language is not a state which is achieved at a certain time, say by age five, ten, or twenty, but is rather a process which, in a normal brain, may continue throughout life. Moreover, there is fundamentally no difference between lateralization and "localization." Rather they are different aspects or phases of a unitary process. The initial phase, *interhemispheric specification* (lateralization), leads to a diffuse language organization in the left hemisphere. This is followed by a second phase of *intrahemispheric specification* ("localization") in which progressive differentiation occurs within the wider speech zone of that (the dominant) hemisphere.

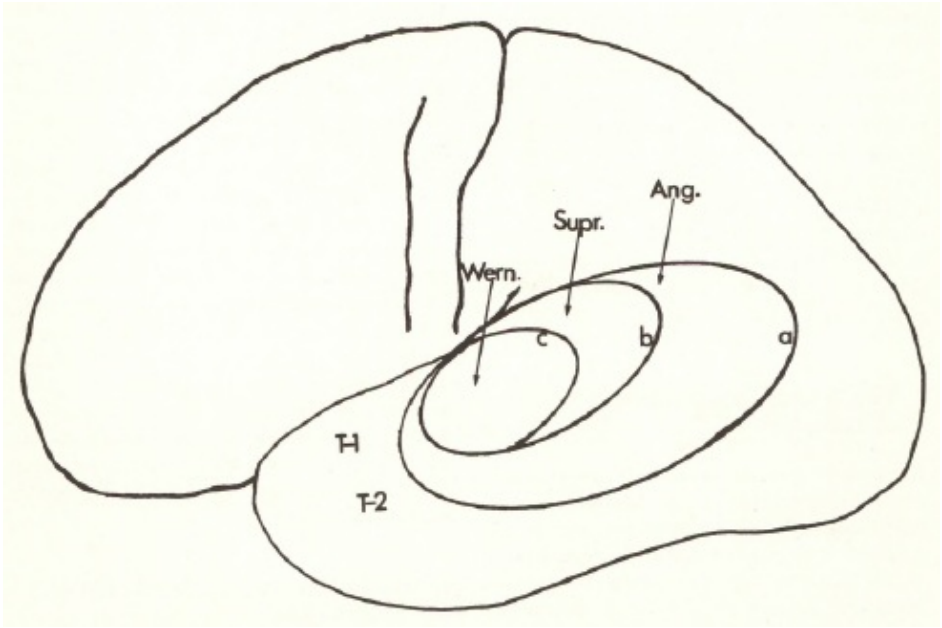
If we examine the effects of a lesion of left Wernicke's area (posterior T_1), we discover that the form of aphasia produced by this lesion differs according to the age of the patient. Such a lesion in a five-year-old child produces a "motor" type of aphasia, with mutism or agrammatism. In a ten-year-old child, one sees an anomic aphasia, while at that same age and on into middle life, a phonemic (conduction or central) aphasia may result. Finally, in

late life, this lesion produces a jargonaphasia. Thus, four different types of aphasia can occur with the same lesion, depending on the age of the patient. At the very least this is persuasive evidence against a naive function localization.

Our knowledge that the process of *inter*- and intrahemispheric language specification takes place during the life span helps to account for this phenomenon. In the young child, an initial diffuse left-hemispheric language organization accounts for the fact that a lesion of frontal, parietal, or temporal lobe (including Wernicke's area) produces a "motor" form of aphasia. Subsequently, within this wider area a new region will emerge (Figure 10-11(a)), a lesion of which (incorporating Wernicke's area) produces an anomic aphasia. Gradually into middle life, a still smaller region is differentiated within the previous zone, a lesion of which (again, including Wernicke's area) produces phonemic paraphasia and phonemic aphasia (Figure 10-11(b)). Finally in late life there is gradual differentiation of a still smaller zone (Wernicke's area proper), lesion of which produces jargonaphasia (Figure 10-11 (c)). Consistent with this is the fact that jargonaphasia is unusual in young adults where it generally requires bilateral lesions, possibly of limbic structures. The central point is that a two-phase developmental sequence, lateralization followed by intrahemispheric specification, creates a dynamic emergent structure which then supports the process of language production. At each stage in this process, involvement of the residual area of each

preceding stratum produces the form of aphasia identified with that stratum when it represented the dominant level in ontogenesis.

Figure 10—11.



An illustration of zones of core differentiation in the posterior language area. The angular (Ang.) and supramarginal (Supr.) gyri, and Wernicke's area (Wern.) represent ontogenetic levels in an evolving structural form, and are not the discrete anatomical loci of traditional cortical morphology.

Moreover, these strata are not to be conceived solely as neocortical differentiations, but as representatives of more widely distributed levels in cerebral phylogenesis. Further, the levels correspond to the three major sequential stages of language production which have been revealed in the

study of the posterior aphasias:

Phonemic	(specialized neocortical)	asymmetric, localized
Nominal	(neocortical)	unilateral but poorly localized
Semantic	(limbic)	bilateral

The semantic disorders are associated either with bilateral temporal lesions or unilateral (left) temporal lesion in the presence of mild generalized involvement. There is evidence in such disorders of bilateral limbic system lesion or involvement of the cortical representatives of limbic structures. The nominal disorders (anomia and verbal paraphasia) occur with either diffuse or focal involvement of neocortex; however, focal lesions cannot be accurately localized, and anomia can also result from subcortical lesion, probably through a referred effect on generalized neocortex. The phonemic disorders are strictly associated with asymmetric focal lesions. Neologistic jargon is also asymmetric and focal, as it concerns, either directly or in combination, a disruption at the phonemic level. This morphogenetic progression from a limbic, through a generalized-neocortical to a specialized (asymmetric) neocortical level provides a dynamic, emergent structure mediating corresponding stages in the microgenesis of language and cognition.

There is a close relationship between this ontogenetic process which builds up the speech area, and the process of encephalization. In fact, the asymmetric structure of the speech area is a continuation of a similar trend in

phylogeny. In this respect, the old theory of encephalization as a series of levels of progressively higher functional organization has been reexamined by Sanides, who has demonstrated that brain expansion occurs through a process of "core differentiation." According to Sanides, in the phylogeny of neocortex ". . . ever new waves of growth and differentiation evolved, and each time a new cortex developed as a core, displacing the previous core to a ringlike structure." There is a striking correspondence between this account and the description of differentiation within the posterior, and by implication, the anterior speech areas in the course of maturation. Sanides has also demonstrated that the "association" or "integration" cortex in man is not, as traditionally believed, the "highest" region of the brain, but *precedes* the "primary" or "projection" cortex in evolution. This is consistent with the idea that lesions of "integration" cortex produce, as in anomia, a disorder micro-genetically prior to disorders produced by lesion of "primary" cortex and immediate surround, e.g. phonemic aphasia and neologistic jargon. It is likely, therefore, that asymmetric neocortical organization, i.e., hemispheric specification for language, represents an ontogenetic solution to a phylogenetic problem, that of size limitations imposed upon an expanding brain in the course of evolution.

There have also been important recent findings with regard to cortico-cortical connections. There is evidence (in subhuman primates) that the frontal "integration" cortex receives projections from the various "sensory"

cortices and that it is in relation to limbic system by way of cingulate gyrus and lateral temporal lobe, and to thalamus via nu. dorsomedialis. Similarly, the temporo-parietal "integration" cortex receives short fiber connections from the various "sensory" cortices, and is also in relation to the limbic system via lateral temporal cortex and cingulate gyrus, and to the thalamus by way of lateralis posterior and pulvinar. Moreover, there are connections between parietal lobe and frontal granular cortex. These facts suggest that in primates, anterior and posterior "integration" cortices, i.e., regions homologous to the corresponding speech zones in man, are organized in a similar if not parallel fashion. Both areas are in relation to the parasensory cortices, both connect to medial and lateral limbic structures and have comparable thalamic representations.

These findings are of significance with regard to the problem of the relationship between the posterior and anterior speech areas. While it is generally believed that language is formed posteriorly and somehow conveyed, by way of the thalamus, insula or association pathways, to Broca's area for motor speech, there is, in fact, little evidence for this view. Rather, it may well be that a simultaneous realization occurs out of a common deep structure into the final linguistic and motoric components of the language act.

Comment. The neural organization of language is characterized by an evolving structural form which is built up during the course of life by a

continuous two-stage process, that of *inter-* and intrahemispheric specification. The process through which the language structure develops, moreover, is only a prolongation into ontogeny of an identical trend in phylogenesis. This genetic approach to the problem of language organization can recapture the dynamic element which is ignored by older static concepts of "centers" and conducting pathways.

The term *microgenesis* has been proposed for the continuous formative activity which underlies cognition. It is implicit that the process of microgenesis recapitulates the sequence of phylo- and ontogenetic forms. The described series of evolutionary and developmental levels supports this process of cognitive formation. Language, that is, the series of levels through which language develops, may be thought of as a final ontogenetic "sensorimotor" differentiation within the neocortical ground supporting cognition up to a prelinguistic phase.

General Aspects of Aphasia

Denial

Denial or lack of awareness of disease is a common manifestation in both organic and functional disorders. The first description was by von Monakow in 1885 in respect to two cases of cortical blindness, while the term

anosognosia, often applied to this phenomenon, was coined by Babinski for lack of awareness of hemiparesis. Lack of awareness is also characteristic of several aphasic forms, e.g., jargon, stereotypy, and echo responses. In general, three types of denial are recognized: (1) partial or complete unawareness of a deficit; (2) explicit denial of the deficit, or, in the case of hemiplegic denial, of the very existence of the hemiparetic limbs; and (3) denial associated with distortions, hallucinations, or other illusory phenomena referrable to the impaired body zone (e.g., phantom or reduplicated limbs, visual hallucination). The view that denial is a reaction of the personality as a whole to the disorder is contradicted by the selective nature of the symptom. Thus, patients with left hemiplegia may deny weakness in the arm but admit to weakness in the leg. This occurs when there is a return of threshold sensory or motor function in the lower extremity while the arm remains fully paralyzed. Similarly, there may be catastrophic depression over subtotal cortical blindness with persistent denial of a hemiplegia. One patient with a left hemiplegia and previous amputation of the first two fingers of the left hand was able to correctly explain why he could not move his amputated fingers, but when asked to move the other (paralyzed) fingers of the same hand, he refused to admit the paralysis. In a case of cortical blindness, there was denial for the totally blind right visual field and awareness of visual loss on the left side where only minimal vision remained (motion and light perception). Thus, denial may spare a less recent disorder, may involve one of

two (usually the more severely involved) hemiparetic limbs, and may spare deficient performances referrable to the same body zone, depending on the reason for the deficiency.

In patients with denial there is commonly some degree of disorientation, recent memory loss, and a confabulatory trend. There appears to be a relationship between the severity of the perceptual deficit and the confabulation. Cases of denial with fair visual or somaesthetic perception have a marked Korsakoff syndrome, whereas the more severe the perceptual impairment, the less prominent the Korsakoff and confusional state. This inner bond between the severity of the perceptual disturbance and the occurrence of confabulation is an important clue toward an understanding of the mechanism of denial.

Those forms of language accompanied by deficient insight, the stereotypy, the echo response, and certain types of jargon are not isolated problems but are part of a continuous series across the spectrum of linguistic change. A transition has been demonstrated between the stereotypic and the volitional utterance (see p. 265). Patients who recover from a Broca's aphasia do not recall the stereotypic content, but may painfully recollect their initial attempts to produce their own name. In echolalia, there is commonly an inverse relation between the fidelity of the repetition and the degree to which it is understood. Such patients may show echolalia for nonsense words or a

foreign language, and paraphasic repetition for their mother tongue. In some patients, a transition occurs between the echo and normal repetition. This takes place over four stages: (1) initial brief latency, explosive echo responses accompanied by euphoria or labile emotionality, and lack of awareness for the echoed content; (2) echolike responses with surprise or uncertainty (partial awareness) of the performance; (3) repetitions with paraphasia, especially phonemic paraphasia, with moderate awareness, and efforts at self-correction; and (4) complete failure of an anomic type, with acute self-awareness, frustration, and at times catastrophic reactions. These forms of repetition may coexist and alternate in a single patient, just as the Broca's aphasic may have concurrent stereotypy and volitional speech. In each instance, awareness can only be described in terms of a momentary state, as a part of a general attitude bound up with an utterance in the process of formation.

Affective Changes in Aphasia

It has long been recognized that aphasic patients tend to show different affective features according to the nature of their language disorder. Schilder wondered whether the apathy and/or depression of Broca's aphasia and the euphoria of Wernicke's aphasia were intrinsic to the speech disturbance rather than a secondary reaction. A study of linguistic change in aphasia suggests that the affective picture is indeed an inner component, not

something “added on” to the language disturbance. Moreover, the affective state is not specific to the “syndrome” but to the cognitive level, or speech content manifesting that level, which is in the foreground at the precise moment during which the affect is displayed. This helps to account for the fluctuation in affective state that occurs in aphasic patients, since this is correlated with a similar fluctuation in the linguistic-cognitive level.

In general, the semantic disorders are characterized by varying degrees of euphoria and excitement. In anomia, there is an improved awareness of the speech disorder, frustration, and some degree of self-correction and censorship. In phonemic aphasia, there is a more acute insight into the speech content, with improved self-correction. Marked alterations in affect are usually not apparent. In Broca’s aphasia, there is apathy, dullness, and some depression. Frustration and catastrophic reaction are not as common in this group as in anomia. Patients with neologistic jargon may be euphoric and excited. At times there is a definite paranoid trend, though systematized delusions are unusual. Paranoia may also occur in semantic jargon but is less common. In the rare disorder of word deafness, a condition characterized by impaired speech perception despite good hearing and nonaphasic speech, the presentation is almost invariably characterized by an acute psychosis with marked paranoid ideation and auditory hallucination. Many of these patients are initially hospitalized on a psychiatric service until diagnosis becomes clear. The etiology of paranoia in aphasia is uncertain, though there are at

least three possible explanations: (1) it might reflect a lesion of temporal lobe independent of the aphasia; (2) it might relate to impaired speech perception and in that respect would be comparable to the "paranoia of the deaf"; or (3) it might have an inner relationship with the language form of the aphasia.

Hallucination

Hallucination does occur in aphasia, chiefly, if not exclusively, with the temporal-lobe disorders, word deafness and (less commonly) jargon. In the former, auditory hallucination tends to appear at the onset, while in jargon it may intervene after several days. These hallucinations may consist of noises, single words, or sentences, e.g. the patient of Ziegler who heard such phrases as "Carl, we're going this way" and "It will be all right." As in other perceptual spheres, e.g., vision or somaesthesis, there is a relation between the density of the perceptual deficit and the likelihood of hallucination. In organic disease, hallucination often points to a lesion of the cortical projection zone of the hallucinated modality.

In schizophrenic patients, there is impaired comprehension during a bout of auditory hallucination. This has been attributed to inattention or distraction. Yet a similar phenomenon occurs in organic cases, where the hallucination seems to "fill the void" created by the loss of the perceptual channel. In general, there is a striking similarity between functional and

organic hallucination. The fact that in the former there may be a higher incidence of auditory than visual content, or that organic hallucination is less systematized, may reflect, respectively, only fortuitous anatomical factors (e.g., the rarity of a focal lesion restricted to auditory cortex) and duration, i.e. that systematization requires a more prolonged hallucinatory state than is generally seen in organic conditions. In fact, in some instances where hallucination may persist for several years, e.g., in "peduncular hallucinosis," patients may develop a hallucinatory psychosis with marked organization and systematization of the hallucinatory ideation. One difference between organic and functional cases in this respect may be the fact that the former do not show the same degree of fear or panic at the onset of the hallucinatory state; in fact, some organic patients appear to be entertained by their hallucinations. Certainly, this seems to be true for some cases of organic *visual* hallucination, but probably does not hold for auditory hallucination.

Toward a Unitary Model of Organic and Functional Disorders

Every symptom in an aphasic disorder points to a level in cognition. The change in affect or in awareness, the occurrence of delusional or hallucinatory states, the appearance of concrete or paralogical thinking, these are not, so to say, outside the aphasia but are a reflection of that cognitive level of which the aphasic speech form is one element.

A study of the aphasias suggests that the pattern of symptom formation is identical in organic and functional disorders. An aphasia represents a disruption in, i.e., a coming-to-the-fore of, higher levels in the linguistic component of cognition. Agnosia and apraxia are parallel disorders in the spheres of perception and action. The asymmetric organization of these "higher" levels accounts for their special relationship to organic, i.e., unilateral, pathology. In functional states, the picture also corresponds to a "coming-to-the-fore," but of lower levels in cognition. Symptoms may appear preferentially in the affective, motoric, perceptual, or linguistic components of cognition. Similar symptoms can be produced by organic lesions, if bilateral and precisely localized.

An approach to the aphasias, as well as to all psychopathological disorders, from the point of view of cognition can reveal this inner bond between organic and functional change.

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Notes

1 Supported in part through a grant from the Foundations' Fund for Research in Psychiatry.

2 Years later Freud was to write to Binswanger that Wernicke was ". . . an interesting example of the poverty of scientific thought. He was a brain anatomist and could not help dissecting the soul as he had the brain."