CHAPTER 11

APHASIA: BEHAVIORAL ASPECTS*

J. P. Mohr and Murray Sidman

Introduction

THE SUBJECT MATTER of aphasia encompasses a spectrum ranging from the practical assessment of an acutely brain injured patient to the abstract theory of language. Since definitions of aphasia vary with the approach to the subject matter, descriptions adequate for one purpose are often inappropriate for another. This chapter is oriented toward the behavioral features of aphasia deficits.

Any understanding of aphasia requires consideration of the roles played by individual variables in the performance profile observed in a given patient at a given time. In roughly descending order of importance, these variables include the methods used to delineate the deficit, the site of the brain injury, the patient's age and handedness, the rapidity of onset, duration, causative agent, the size of the brain injury, and coexisting motor and sensory deficits. Singly, and in combination, they can account for many seemingly contradictory or only loosely comparable features of different cases of aphasia.

A theoretical structure is helpful, but not a prerequisite in approaching the subject of aphasia. The spectrum of traditional and current theories of aphasia can be accommodated within the following elementary summary. At the lowest level of complexity,[26,30,50] the basic instrumentalities subserving discrimination, replication, and production of verbal stimuli, the phonologic aspects of language,[52] are considered to require the proper functioning of the cortical surface and subcortical white matter structures grouped around the Sylvian fissure of the left cerebrum. Auditory inputs from the brain stem pass via white matter pathways to the primary auditory cortex (Heschl's transverse gyri) located in the superior temporal lobe at the posterior region of

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the Sylvian fissure. Vocal outputs are controlled by the primary motor cortex (Rolandic fissure), subserving movements of the oropharynx, larynx, and resipiratory apparatus. Control of the individual movements, transitions of movements, and melodic sequences[20] involved in speaking aloud is exerted via the adjacent premotor cortex in the inferior frontal region (Broca's area).[6] Fiber pathways in the arcuate fasciculus, deep to the insula, may link the auditory and vocal motor regions to permit repeating aloud from dictation.[14] At a level of greater complexity, organization and comprehension of conversational speech, especially its semantic and syntactic aspects,[52] are traditionally thought to reflect activity of the inferior parietal and posterior temporal regions adjacent to the auditory cortex, the combination usually referred to as Wernicke's area.[50] Combined lexic and graphic activity is also thought to involve inferior parietal activity,[10] especially those portions adjoining the more posteriorly situated occipital lobe, whose main function involves processing of visual inputs. The most complex, abstract, and theoretical levels of language activity are considered to involve preverbal thought, i.e., the formulation of the basic message to be conveyed;[7,37] posterior and deep temporal-lobe functions may underlie these processes, the documentation for which remains theoretical and introspective at best.

Two main variations of the foregoing underlie most writings in the field of aphasia, even though they are not always explicitly stated. In the first variation,[20,27,50] brain mechanisms underlying language behavior are seen to reflect the interaction of relatively autonomous cerebral regions, i.e., auditory, visual, and motor. Constellations of individual findings (syndromes), which constitute clinical aphasia, reflect focal brain injuries (lesions) of varying origin, involving the cortical surface "centers" or the white matter pathways, separately or in various combinations. In the second variation,[30] only one cerebral region, situated in the posterior portion of the Sylvian fissure, is crucial for language behavior. The cerebral regions serving sensory input and motor output are seen functionally as essentially centripetal or centrifugal, respectively, to this central zone. Syndrome analysis is directed toward discovering evidence of deficits thought to reflect damage to the central language mechanism, irrespective of the input or output channels involved in the behavior being tested. Such deficits are considered aphasic, while involvements reflecting only damage to the centrifugal or the centripetal functions are not.

The basic theoretical formulations outlined above have served as the foundation for most of the many different viewpoints toward aphasia. It is all the more unfortunate, in view of the enormous amount of study given the subject, that basic ambiguities still prevent a clear understanding of the subject.

Testing for Aphasia

The major aphasic syndromes were originally described from clinical observations. Although ingenious tests were often used in assessing the classic cases, the data now available are in most cases summary notes which reflect the investigator's interpretation of the behavior more than they do the actual behavior itself. The subsequent development of methods for analyzing behavior has resulted in a continual updating of the components of individual aphasic syndromes, with some divergence from previous interpretations.

Much of the controversy in the field of aphasia stems from clinical differences among patients with apparently similar lesions. Much of this variability reflects the selection of the aphasic population to be tested, the actual tests administered, and the methods of test administration.

Case Selection

A variety of approaches has been used in the selection of cases. Historically, the report of a single case or a few cases showing virtually identical findings, has set the precedent still favored by many investigators. Reports of one, or of a limited number of cases, generally include anatomic findings proved by autopsy, and involve intensive study for varying periods of time, and/or show singular or unique findings bearing on aphasia theory, all encompassed in a readably brief account. Taken together, fewer than one hundred such cases have contributed the majority of the data upon which the major current ideas on aphasia depend. Yet even these intensively studied single cases have undergone only a limited number of tests. It has been argued[18] that such cases are so rare and unusual that they are not representative of the field of aphasia in general. This contention has been countered[51] by the point that the combined factors of anatomy and pathologic processes in naturally occurring illness usually result in brain injuries whose location and extent encompass so many important regions simultaneously that most cases are too complex to permit a detailed analysis. The rare case of sharply specifiable deficits is of value as the exception that helps clarify the rules.

Another approach to case selection has been to study a large group sharing in common some major variables, such as site of lesion, etiology, age, etc. War injuries are a prototype of this approach.[40] These studies provide corroboration for the individual case reports. They suffer from the statistical summary approach in which details of individual cases can get lost in group averages.

In yet another approach, the purpose has been to screen an unselected population using a single test[4] or series of tests.[49] Separation of the case material in the groups is then based on the responses made by individuals. Such collections of cases seem to show the most general, and least specific, findings. Critics contend that the nonspecificity of the findings reflects the inclusion of cases differing widely in type, whose individual differences disappear when the data are averaged together. Supporters point to the need to establish an approach to deficit profile without dependence on these traditional criteria, so as to permit some validation of the traditional means of classifying cases.

Test Methods

The authors believe that the methodology used to approach a case of aphasia is basic to all other considerations, since it provides the data from which the theories should be derived. Accordingly, test methodology will be discussed before taking up the analysis of aphasia.

In his monograph on aphasia in 1874, Wernicke[50] noted a tendency for patients to seize upon any kind of cues available to them when they experienced difficulties with the tests designed to assess their language behavior. Although he recorded this observation—that patients may use any of a number of possible means to approach a task—in his early monograph on aphasia, deliberate specification of individual parameters in aphasia testing has received intensive attention only in recent years. As late as 1966[17] proposals could still be found calling for standardization of the stimulus, response, and other variables involved in aphasia testing.

At the present time, since major research centers tend to maintain and use their own methods, data from different centers are often not strictly comparable. Ambiguities and differences in the observation of aphasic behavior are a paramount source of disagreement, and a review of the major methods used to evaluate aphasia patients seems justified.

BEHAVIORAL

Although all aphasia tests are behavioral, few investigators explicitly and systematically use the principles and techniques that stem from objective behavioral science. We have, therefore, used the term "behavioral" to characterize our own approach.[44]

With some oversimplification, we can specify three major classes of behavioral variables which may interact with physiological processes to govern a person's interaction with his environment. First, all behavior, including that exhibited in aphasia tests, is governed by its consequences. Rather than depend solely on a patient's presumed motivation to do well in our tests, we provide explicit positive rein-

forcement, i.e., to encourage correct responses. Behavioral deficits, aphasic or other, may result from the breakdown of the controlling relation between behavior and its consequences, and the terms, "motivational" or "reinforcement deficit," are often applied. Little is known about such clinical deficits in humans; it need only be said here that a patient is likely to exhibit no consistent behavior if presumed reinforcers in the test situation are ineffective, and any conclusions about aphasia will be untenable in such patients.

A second class of variables is subsumed in the term, "stimulus control." Appropriate behavior occurs in response to stimuli which set the occasion for reinforcement, as determined by a person's behavioral history. When we observe that a particular stimulus occasions a response, and that its absence fails to do so, we have a controlling relation between stimulus and response. An example of the complexity involved in stimulus control is the relation between traffic lights and a driver's behavior. We have achieved considerable initial support for the notion that many aphasic deficits represent breakdowns of stimulus control; for example the controlling relation between printed words and oral naming (speech deficit), between pictures and written naming (writing deficit), or the nonverbal selection of appropriate pictures in response to printed words (reading comprehension).

The third class of variables may be termed "instructional." These include the constant stimuli of the test environment, the test procedures themselves, and the specific instructions given to the patient about what he is expected to do. Clearly, a patient who is not sensitive to instructional factors will exhibit test behavior that is unrelated to the purposes of the tests. Like motivational deficits, instructional deficits invalidate any conclusions specific to aphasia. Since aphasia, by its very nature, represents a communication disorder, instructional deficit is often difficult to circumvent in aphasia evaluation. The problem can be overcome by appropriate use of effective reinforcers, which function nonverbally to inform the patient when he is performing as requested.

Controls for reinforcement and instructional deficits are built into the procedures of the tests, which are, themselves, oriented toward the analysis of stimulus-control deficits characteristic of aphasia. The sequence of tests, furthermore, has been designed to reveal intact forms of stimulus control, thereby reducing the number of factors that must be considered to play a role in the patient's deficit. The tests themselves simply required the patient to name orally, write, or match (select from a number of alternatives) visual, auditory, or palpated test stimuli, such as single letters, three-letter picturable nouns and their pictures, color names and their colors, digit names and their digits, and manipulable objects. These tests demonstrate the control exerted by each stimulus (visual, auditory, or palpated) over each type of response (oral, naming, writing, and matching). The test battery yields a stimulus-control matrix in which stimulus (input) channels, response (output) channels, and controlling stimulus-response or stimulus-stimulus relations can be evaluated.

Such systematic behavioral evaluation has revealed six large groups of patients, five of which have not yet been extensively studied. The first group includes patients whose deficit is so mild as to escape detection by simple tests. These cases are frequently considered normal on initial brief bedside examinations. It remains to be seen whether more complex materials, at the sentence, paragraph, and syntactical level, will reveal deficit constellations similar to those shown in other patients tested with the simpler materials.

The second two groups are at the other end of the scale, and completely new test procedures will be required to study them effectively. The most severe deficits are those in which reinforcement is inadequate to maintain behavior, thereby precluding the delineation of a deficit profile. The few such patients we have tested have been those with medially placed frontal lesions exhibiting symptoms of hydrocephalus, clinical states of delirium, and dementia. This is a potentially fruitful area for the application of Pavlovian conditioning techniques. Also untestable by the present methods are patients with deficient instructional control, for their test behavior is completely unrelated to our test materials and procedures. These patients include a number of cases exhibiting the bedside syndrome of central, or Wernicke's, aphasia. Instructional deficits can be differentiated from reinforcement deficits only if reinforcement can be shown to be effective in some other kind of test, such as a less demanding visual or auditory discrimination, in which the need for instructional control is minimal.

The fourth and fifth groups are those who show deficient input (stimulus)[33] or output (response) channels.[36,37] These two groups include the vast majority of cases labelled in a brief bedside examination as showing "agnosia," "pure" word blindness, deafness, mutism, etc. Input deficit reveals itself when a particular type of stimulus fails consistently to control *any* type of response. Output deficit reveals itself when a particular type of response consistently fails to occur in the presence of *any* stimulus. The functions of the input and output channels are assessed by identity tests. These involve a response which is physically identical to the test stimulus. For example, repeating dictated words aloud, copying printed words, and choosing from among a visually presented set of words one which is typed and spelled exactly like the test stimulus, are all examples of responses which are physically identical to the test stimulus. These identity tests require no previous experience with the stimuli and serve principally to test the adequacy of stimulus discrimination and response production in the input and output channels used for testing.

Once these identity tests have shown the adequacy of the input and output channels, those channels and stimuli found adequate can then be used to explore the specificity of stimulus control in "nonidentity" tasks. In these tests, the response required is not physically identical to the test stimulus. Examples include spoken responses to visual stimuli, written response to dictated stimuli, selection of choices (matching) in which, for example, the test stimuli are pictures, and the comparison stimuli are words.

The sixth group of patients, with intact input and output channels, display differential relational deficits between otherwise normally functioning stimulus and response systems. This group, which includes the vast majority of patients whose conventional clinical bedside evaluation reveals clear evidence of aphasic disorder, has revealed a number of deficit profiles. Some include classical syndromes, some appear to be previously undescribed, and some are mainly of methodological and interpretive interest.*

Other investigators have independently devised methods similar in principle to our behavioral model. The principle of using common manipulable object stimuli presented separately in visual, auditory, and palpated form for separate spoken and written naming responses began with Head's six objects.[17] It was popularized in the United States,[8] was increased to twenty objects,[48] is found in modified form as a basis for a currently popular aphasia test battery,[38] and, in reduced form, is present as a subtest in many other aphasia test batteries. Extensive use has been made of the matching-to-sample paradigm as a means of "facilitating" correct responses on verbal tests requiring spoken or written responses where errors appeared.[21,22,48]

Similar procedures have seen extensive use in production examinations of inter- and intramodality performances in cases of surgical sections of the corpus callosum.[13,14]

Traditional Test Batteries

Another major approach to delineation of aphasic deficits involves the presentation of a wide variety of individual tests, each designed to assess a given aspect of behavior, without deliberate continuity of stimulus material, input and response channels, or reinforcement across the spectrum of tests. Each test in the subgroups is constructed to stand individually and have its own validity. The performance profile that results for a given patient is compared with that obtained in normals and in other aphasic patients.

The corpus of tests included in these traditional batteries appears to have arisen from the large variety of individual tests created by

* See references 23, 24, 33, 34, 43, and 44.

General Properties of Aphasia

Cases of aphasia share many general features of behavior with normal subjects, especially when the latter are tired or tested under difficult conditions. Reinforcement that is inadequate to maintain behavior in the face of frequent errors commonly leads to breakdown of the control exerted by the test procedures. This state of affairs is revealed in a number of ways. The patient may simply stop responding. He may perseverate previously correct responses, even though these responses are complex, i.e., writing whole words. At times, long delays occur before he responds. He complains of being tired or uncomfortable; lame excuses of poor vision, inadequate education, unfamiliarity with the tests, etc., are common. Occasionally, outbursts of anger occur, with the patient scattering the test stimuli around, rising and leaving the test site, turning away, or even assaulting the examiner. Control over the patient's behavior can usually be reestablished by changing to a task he can easily accomplish, increasing the reinforcement, slowing the rate of testing, and similar devices. The patient's ability to return to the task, and perform reliably over a long test session, suggests that "fatigue," traditionally considered a major variable in aphasia, is a reflection of the test procedures. Signs of fatigue are mostly evident when the patient is having difficulty with the test.

The errors occurring when the test situation maintains adequate control over the patient's behavior take three main forms,[24] which are also common with normal subjects. Repetition of a previous response or portion thereof (perseveration) is common. In many instances, a correct response given previously is repeated on a subsequent trial when the patient is having trouble with the test. At times, the source of this repetitious response (perseveration) is less clear. Many nonperseverative errors, such as literal or verbal errors, also show evidence of control exerted by the test situation. Literal errors approximate the desired response along some physical parameter, and take the form of similar sounds ("tog" for "dog") or shapes ("d" for "b"), etc. The response may bear so little physical resemblance to the one desired as to be characterized as neologism or jargon. Verbal errors share some functional class with the desired response; "cow" for "dog," "green" for "orange," and occasionally, "grass" for "green." At levels more complex than words, errors may appear in word sequence or sentence structure (semantic errors); grammatical construction may become simplified (agrammatism); the patient may accept as correct familiar sequences of words into which the examiner has deliberately substituted unexpected words or even neologisms;[52] other forms of errors may occur which become more and more difficult to separate from performances which characterize normal people deficient in education.

As patients and normal cases are retested over extended periods of time, general improvements in performance occur.* In oral and written naming, verbal paraphasic errors continue but are increasingly represented by names within the test set and decreasingly by names not in the test set. Even the patients' spontaneous responses gradually become restricted to words that are involved in the test itself. Presentation of the first letter or two letters of short words frequently used in the tests are sufficient for the experienced patient to respond correctly; introduction of novel stimulus materials prompts a dramatic reduction in the rate of performance and an increase in errors. Repeated testing with stimuli previously found difficult is associated with considerable evidence of patient dissatisfaction as soon as the first trial occurs, demonstrating his learned familiarity with the components of the test. In addition to gradual learning, some of the improvements are sudden, even after long periods of poor performance on a given test, and appear to represent newly discovered abilities, whose origins remain obscure.[35] In most instances, however, the performance improves in a slow but steady fashion.

A dichotomy in performance between identity and nonidentity tests[44] also characterizes aphasic and normal cases. Scores on tests for which identity responses are available equal or exceed those tests for which these responses are not available (nonidentity). Identity tests (see the section on test methods, p. 281) must be subdivided into first- and second-order identities for this rule to hold. In first-order identity tests, the patient need only indicate the physical identity of the same stimulus presented twice in the same modality. For example, a patient points to the blue color identical to the blue test stimulus, palpates a skeleton key exactly as he palpated the same key just before, nods when he hears the same word heard earlier as the test stimulus, etc. In second-order identity tests, the patient is required to cross a modality or to produce a response which takes a physical form identical to the test stimulus. Examples include repeating from dictation, copying on paper from sight or touch, and matching palpated manipulable objects to visual manipulable objects. Such tests, although they do not involve actual physical identities, can nevertheless be done correctly by normal subjects even if they have had no previous experience with the stimuli. No exception occurs to the rule that first-order identity performances equal or exceed nonidentity performances on equivalent tests, but an occasional deficit in performance of second-order identity tests may occur in aphasic patients when the equivalent nonidentity test is intact. For example, when presented with a series of dictated letters spelling a word, the patient may succeed in pronouncing the word at a time when he experiences difficulty repeating the sequence of individual letters. In general, however, both first- and second-order identity tests are accomplished successfully at times when the nonidentity forms of the test are not.

When identity tests are done poorly, input or output deficits must be suspected. When identity tests are done well, poor performances on nonidentity tests reveal relational disorders, i.e., responses are deficient only in relation to certain stimuli, or stimulus control is deficient only when certain responses are called for. Relational disorders, i.e., impaired performance on tests in which the correctly spoken, written, or matching-to-sample response requires previous experience with the test stimulus, prove to be critical components of syndromes that have classically emphasized input or output deficits, and may be taken to define the most interesting aspects, at least, of aphasia.

* See references 17, 23, 24, 33, 35, and 44.

Syndromes with Greatest Emphasis on Output Channel Deficits

VOCAL OUTPUT CHANNEL AND GENERAL RELATIONAL DISORDER

Behavioral studies of cases which initially appear to typify the clinical bedside syndrome of total aphasia, and later are consistent with Broca's aphasia,[6] have corroborated traditional features, but, in addition, have revealed a number of findings hitherto undescribed in these syndromes.[35] These new findings prompt a reconsideration of the anatomical mechanisms and explanations.

The deficit profile has four main components.[35] A double deficit is found in oral naming; first, the patient is mute and produces no vocal responses on either identity or nonidentity tests. Later, the mutism clears away, as indicated by satisfactory oral naming in identity tests of repeating from dictation. From that point on, the second disorder, a relational deficit, is revealed: impaired performance in nonidentity oral naming tests. In contrast with oral naming, the performance on identity tests of written naming and matching-to-sample are intact from the beginning. Later, when oral-naming identity performance becomes adequate, so that anarthria can no longer account for poor scores on nonidentity oral naming tests, nonidentity written and oral naming can be compared in response to the same stimuli. At this point, the third deficit component appears, i.e., superiority of nonidentity written naming over nonidentity oral naming. The fourth component is demonstrated in all response forms and stimulus materials in nonidentity tests, namely, performance on tests involving the sounds of words exceeds performance on tests involving the sounds of single letters. This component is demonstrated by better scores in matching and writing of

dictated words than of single letters, and better scores in the oral naming of visually presented words than of single letters. By contrast, most wholly visual tests are performed satisfactorily for both materials: The patient can match dissimilarly shaped upper- with lower-case letters having a name in common (i.e., E—e), and even can match scrambled words with pictures. Interestingly, one test ostensibly involving wholly visual functions, matching visual letters with homonymous visual words that do not contain the letter (c—sea, q—cue, i—eye), is done poorly. The time required for the delineation of each of the main features of the syndrome varies from a few weeks to several years in individual cases.

The initial mutism is severe. Only a few noises are made in forced exhalation. With time, vocalization emerges to testable levels. It shows elements of dyspraxia, revealed by improper setting of the oropharynx, and impaired coordination of respiration with vocalization, resulting in lack of smooth speech melody i.e., dysprosody. Despite traditional emphasis on the attributes of the vocal response,[1,15,27] performance on the identity tasks in repeating from dictation follows the expected patterns of exceeding that of the nonidentity tasks of producing the same names in response to appropriate visual, palpated, or even nonverbal sound stimuli.

The duration of the mutism is variable. In a few right-handed cases, the deficit ameliorates in a dramatically brief period—days to one or a few weeks.[32] Such rapid amelioration in a right-handed patient with left inferior frontal infarction has been considered a sign of superficial involvement of a cortical surface.[16] The intact intrahemispherical pathways (arcuate fasciculus) through which the central language zone (Wernicke's) is considered to relate to the ipsilateral inferior frontal region (Broca's area), and thence transcallosally to the nondominant inferior frontal region, have traditionally[16] been presumed sufficient to permit the nondominant inferior frontal region to mediate the vocal responses and permit the "recovery." Recently,[32] righthanded cases have been followed through this period of dramatically rapid amelioration of vocal speech deficit. Detailed autopsy evidence showed major damage to the dominant inferior frontal region, including the pathways considered necessary to mediate "recovery." Traditional formulations do not explain these cases, and alternative pathways, as yet undelineated, must be considered. The findings suggest the need for revision of current notions of cerebral "dominance" for speech, and indicate that the degree to which the inferior frontal regions share the mediation of vocal speech is only poorly understood.

The superiority of written over oral naming, when identity responses for both are intact, calls into question some notions of how writing behavior is mediated. Most classic[26] and many modern[25] accounts indicate that the deficit in written naming is a reflection of that in oral naming, and is at least as severe, usually more so. Accounts of aphasic deficits consider that writing reflects two components. In the first component, the morphology of the individual letters and digits is believed to depend on a direct pathway from visual to motor regions which guide hand movements.[26] Until recently, no theory has challenged the classic notion that the second component, the verbal content of the writing, depends upon pathways which pass through Broca's area, and presumably utilize it as a way station: "one speaks as one writes."[25] The only quantitative study[35] of this important subject, revealing a superiority of nonidentity written naming over nonidentity oral naming when both were adequate on identity tests, challenges this classical interpretation. The independence of written and oral naming suggests a new view,[35] which does not assume an obligatory relation between written and oral naming based on a unitary brain mechanism. Instead, the coexistence of superficially similar deficits in written and oral naming may merely reflect anatomical proximity of the two regions subserving these separate motor responses, favoring their common involvement by a single pathological lesion. Such anatomic proximity implies no functional interdependence between the two areas.

The more severe deficit with letter rather than with word sounds, common to written and oral naming, appears also in matching-to-sample behavior. The emphasis in traditional formulations,[9,26,27,50] which envisioned the two naming deficits as reflecting correlated output disorders, can be properly shifted to include all forms of behavior. As a result, the deficit can be considered central to the input and output channels, per se. It must be pointed out, however, in anticipation of the following section on central aphasia, that the deficit profile in which nonidentity tasks show better scores with words than letters is opposite to that commonly found in cases conforming to traditional criteria for central aphasia.[44] Instead, this disproportionate deficit in nonidentity tasks involving the sounds of letters appears unique to this syndrome.[35]

Explanation of the data requires still further revision of accounts of both Broca's and total aphasia. Classical writings have explained the syndrome of total aphasia as a combination of Broca's and Wernicke's (central) aphasia.[27] The syndrome outlined above, although it conforms to classical clinical bedside criteria for total aphasia, is not explainable as a simple combination of Broca's and central aphasia. In addition, the complexity of the satisfactory responses in many nonidentity tasks suggests that the term, "total aphasia," is misleading. The deficit appears highly specific to certain verbal tasks, with disproportionately better performances on others of seemingly similar or greater difficulty.

Definitions of Broca's aphasia have given greatest attention to the disorder in oral speech,[2] with emphasis on the dyspraxic, dysprosodic, dysgrammatic components; on the issue of coexisting dyspraxias for nonvocal movements involving the same oropharyngeal musculature; on the coexistence of facial, lingual, and palatal paresis; on the issue of cerebral dominance; and on the exact location and depth of the lesion. Scanty information exists on the writing deficit, which is usually explained on the basis of the presumed dependence of verbal content on vocal speech, implicit or explicit. Broca's two cases[6] appear to have had principally disorders of vocalization. Unsettling reference,[5,27,49] however, has always been made to mild or moderate impairments in "comprehension," which occur in tests of silent reading and in performance of multistep dictated or printed commands. Ingenious tests with normals,[29] in which the tongue has been restrained, have shown impairments in reading, implicating vocal speech deficit as a partial explanation for the otherwise unaccountable deficits in comprehension in Broca's aphasia. Such explanations, however, do not account for the deficiencies in response to auditory dictated commands. Another approach has been anatomical,[30] suggesting that clinically unsuspected posterior extension of the lesion has occurred along the postcentral and parietal operculum, accounting for the minor central aphasia impairments. As emphasized above, however, the behavioral deficit in response to dictated stimuli in this syndrome is not typical of central aphasia. Finally, little or no qualitative differences separate the vocal and graphic behavior in total and Broca's aphasia.

The ambiguities surrounding the definition of Broca's aphasia have not been clarified over the years. Considering the great similarity between later cases of the traditional bedside syndrome of total aphasia, the uncertain status of "comprehension" in cases of Broca's aphasia, the anatomic problems surrounding the extent of the lesion in autopsied cases, and the wide variation in the course of the deficit, one might ask whether actual deficit features or mere historical precedent substantiate the syndrome of Broca's aphasia. The present authors suspect that the understandable desire to honor Broca's efforts at anatomicopathologic correlation serve as the chief basis for continued recognition of a separate syndrome referred to as Broca's aphasia. Further analysis of the syndrome of which the classical Broca's and total aphasia appear to be elements may be expected to modify views concerning the function of the anterior Sylvian operculum and the cerebral organization of language.

DISPROPORTIONATE LITERAL PARAPHASIA

In this syndrome,[29] errors appear in both identity and nonidentity oral naming tasks, but not in equivalent tasks involving matching-

to-sample. Although this syndrome is classified as both an identity and nonidentity output disorder of oral naming, the patient shows none of the mutism characteristically observed in the syndrome described above. Instead, vocalizations occur readily, but are equally erroneous on identity and nonidentity tasks. For example, repeating aloud, reading from text, and oral naming of visual, auditory, or palpated stimuli show similar scores with similar errors. In contrast to the deficit in oral naming, tasks not involving a spoken response, such as matching-to-sample, are done extremely well, and written naming is often quite satisfactory. The patient's exasperation and efforts at self-correction of his oral naming errors attest to his ready awareness of the deficit. The patient's errors include a disproportionate number of literal paraphasias, involving close anatomic approximations of the oropharyngeal positions required to produce the correct responses in each of the articulatory classes from lip to pharynx position. Errors increase with the rate of speech and with the proximity of the oropharyngeal settings required to produce the sequences of syllables.

In Wernicke's original scheme,[50] the term "conduction aphasia" was proposed for the syndrome, which could be considered to reflect interruption of the pathways from the "sensory" (Wernicke's) speech region to the "motor" (Broca's) regions. As originally constructed, the syndrome contained three elements. First, comprehension would be intact, since Wernicke's region was preserved. Second, the motor elements of speech (articulation, prosody) would be intact, reflecting the spared motor-speech regions. Third, content of speech would be paraphasic, as tested by spontaneous speech, reading aloud, and repeating from dictation. This third feature, the only real deficit to be found, was the expected result of the pathologic interruption of pathways linking Wernicke's region to the motor (Broca's) speech region. It is important to stress that the deficit was to take the form of paraphasic oral speech. Only the motor elements—articulation and speech melody—were considered to be normal, indicating that the deficit in speech does not merely reflect involvement of the inferior frontal (Broca's) region.

Cases frequently appear clinically which exhibit paraphasic, normally articulated, and normally melodic speech, with superficially intact comprehension, and are considered to satisfy the criteria for conduction aphasia. In most such cases, however, deficits in comprehension can readily be brought out by testing silent reading or matching-to-sample, which do not involve oral speech. These cases are more frequently better reclassified as examples of mild central (Wernicke's) aphasia.

The search for cases defined by the more stringent criterion of no demonstrable deficit in comprehension, has yielded few cases of conduction aphasia.[16,20,28,36] Awareness of this interesting syndrome has increased only in the 1960s,[14] but most reports are in the early literature.[16,20,28,36] Presumably, their rarity reflects the greater likelihood that pathologic injuries to the fiber pathways connecting the Wernicke and Broca regions would not be as discrete as required. Instead, the injury is more likely to involve larger areas, and result in more traditional syndromes of central, motor, or total aphasia.

Even fewer cases satisfying the clinical criteria have provided autopsy data. Meager though these data are[20,28,36] they pose a problem in interpretation by classic theory, which predicts that the main lesion should lie in the pathways linking the auditory with the motor-speech regions. Attempts[14] to identify these pathways have focused on the arcuate fasciculus, a white matter bundle which appears to pass between the posterior superior temporal plane (Wernicke's region) and the inferior frontal region (Broca's region), and satisfies the gross anatomic requirements. Autopsy cases[20,28,36] of "conduction" aphasia, however, have shown cortical surface infarction, apparently of embolic origin, without necessary involvement of the more deeply situated arcuate fasciculus. To date, no cases have been reported that show pure involvement of the arcuate fasciculus. The clinical setting for such a lesion occurs occasionally in putamenal hemorrhage, in which the hemorrhagic mass is limited to the posterior lateral putamen and

the immediate surrounding area, which includes the arcuate fasciculus. In the one such case that has come to light,[31] the clinical syndrome was more of a central than a conduction aphasia.

Luria[10] has described a syndrome of *afferent motor aphasia.* In contrast with the usual form of motor aphasia, which he has referred to as "efferent," literal paraphasic errors in oral speech are attributable to anatomic settings of the oral apparatus that are imprecise but closely approximating those required. The lesion is presumed to lie in the postcentral region, interfering with sensory kinesthetic feedback from the oral cavity. The clinical findings agree with those delineated by behavioral methodology, adhering closely to classically defined conduction aphasia, but pointing clearly to mechanisms different in principle from those proposed classically.

The extent to which literal and verbal paraphasias occur independently of one another, as well as the basic deficit(s) reflected by literal paraphasia, remain important unclarified issues. Literal paraphasias that prove principally to reflect oropharyngeal anatomic approximations point to sensory and/or motor Rolandic deficits.[20] Traditionally,[10] by contrast, literal paraphasias are considered to take the form of homonyms of the desired response, and to reflect auditory input deficits. Verbal paraphasias, by contrast, are traditionally thought of as synonyms. However, few studies specify the relative frequency of each type. Furthermore, literal and verbal paraphasias are considered to occur together with such regularity as to suggest some mechanism in common, yet even fewer studies document the frequency with which they occur in the same case, especially a case with autopsy material. As a result, the theories on either form of paraphasia are largely speculative.

General Relational Disorders

A surprising proportion of cases tested by behavioral methods show deficits only on nonidentity tasks. No deficits are found for a given test stimulus on identity tests of repeating the stimulus from dictation, copying at sight, or matching the stimulus to its exact duplicate in the same modality. These intact performances permit the assertion that sensory discrimination and response production are adequate for these stimulus materials, and preclude an explanation of the impairments that is based on deficient input and output channels.

Although deficit profiles observed on nonidentity tests across the various stimulus materials take several forms, one in particular typifies that predicted by traditional formulations of central, or true, aphasia.[43,44] This profile shows a similar deficit in response to each of the classes of test stimuli. For example, in response to the same stimuli, whether they are single letters, words, pictures, color names, colors, digit names, digits, or manipulable objects, scores on nonidentity tasks of matching-to-sample exceed those for oral naming, which exceed those for written naming. Improvement occurs gradually with time and more or less equally with all types of test stimuli. At any point in time, errors may occur in response to any individual stimulus, but no individual stimulus reliably sets the stage for an error each time it is presented.

The traditional formulation[46,50] of the true or central deficit in aphasia involves disruption of a supramodal function whose normal role is to relate physically dissimilar stimuli which are verbally equivalent. This function was held to be accomplished by the "concept center."[26,50,51] Wernicke,[50] among others, considered this function actually to be performed by the portion of the brain outside those pathways subserving the instrumentalities of language.

Wernicke[50,51] argued that the initial acquisition of language is probably an auditory experience. Learning to speak aloud would involve auditory modulation of vocal efforts. Reading aloud would involve acquisition of an auditory-visual link between sounds and graphic stimuli, establishing pathways which would then permit instructions to the vocal apparatus for reading aloud utilizing the auditory region as an intermediate. A similar link would modulate graphic motor behavior. Lesions of the auditory region and connections would be expected to disrupt these relations.

The added assumption was that these separate behaviors permanently depend upon the auditory region. This dependence would account for the overall deficit in the utilization of the instrumentalities of language in lesions affecting the auditory region and related pathways.

Wernicke was careful to separate the essentially servile performances utilizing the instrumentality of language from the more abstract and poorly understood aspects of brain function involving "concepts." Diagrammatically,[50,51] his scheme showed pathways from the ear to the superior temporal lobe serving auditory speech discrimination; pathways from the superior temporal lobe to the inferior frontal region serving to convey the instructions for vocalization to the motor region; pathways from the inferior frontal region to the brain stem serving to innervate the bulbar apparatus to produce speech sounds; pathways from the superior temporal lobe to the occipital region linking auditory with visual functions to subserve reading. None of these pathways necessarily serves "understanding" or "central language function." Instead, pathways from the superior temporal lobe to the remainder of the brain were considered to permit the auditory experiences, and those visual and palpated sensory experiences translated into auditory equivalences, to arouse associations in the remainder of the brain which provide "meaning" to the stimuli. Similarly, pathways outside the main speech zone were considered to converge upon the motor speech regions (Broca's area) to permit "meaning" to be given to vocal utterances. Without challenging the notions in principle, Dejerine[9] added the angular gyrus as a word center, whose supramodal function was to relate auditory and visual lexical stimuli as verbal equivalents, and to guide the motor regions for graphic responses. Recent arguments[14] have modernized the proposal of the angular gyrus as exerting a supramodal function relating physically dissimilar but verbally equivalent stimuli. Others[11,29] have proposed essentially similar translatory functions for the inferior parietal regions, of which the angular gyrus is a component. These views argue that integration, or morphosynthesis,[11] is the basic function to be expected of the inferior parietal region, since its anatomic position lies between the main primary sensory receiving areas in the cerebral cortex.

Emphasis on this region as central to language function helps encompass many aspects of behavior in such cases. The patients exhibit a remarkable unawareness of the extent, the time, even the existence, of their deficit. Both literal and verbal errors (especially verbal) occur in all forms of language usage, in tasks involving comprehension, and in language formulation, with scarcely a pause for correction. Oral speech tends to contain far more words than expected or required for efficient communication. The term "logorrhea,"[7] also referred to as augmentation and press of speech,[16] denotes the tremendous barrage of vocalizations that frequently characterizes these cases of central aphasia. In addition, efforts to instruct the patient to modify his response for different tests frequently are unsuccessful;[16] they are often met with perseveration of previous responses or principles of response, even though the tests have changed. Particularly frustrating to the examiner is the frequent tendency of patients to respond to commands only by acknowledging that a command was given; efforts to vary the command by adding, "please," "I would like you to . . . ," etc., are frequently met by a reply like "O.K., I will," but with no actual performance. Even more suggestive of a unitary deficit is the all pervasive nature of the deficit in language usage, which appears in tests involving spoken, written, and matching-to-sample responses.

Despite the many indices favoring these all-encompassing views of language function, a series of findings, both anatomic and behavioral, remain unaccounted for. Anatomically, an occasional case whose deficit profile suggests the traditional syndrome of total aphasia is shown at autopsy to have a lesion wholly confined to the dominant temporal lobe.[16] The temporal-lobe mutism in these cases contrasts sharply with the logorrhea usually characterizing such lesions. While temporal-lobe mutism suggests that the posterior Sylvian regions exert the major controlling function over the output of the inferior frontal region, such findings pose the difficult problem of explaining opposite observations by the same anatomic lesion. Suggestions that the more commonly observed logorrhea represents a release effect in which the inferior frontal region "runs on unchecked," seem less tenable in view of the existence of temporal-lobe mutism. Another suggestion is that logorrhea may represent a functional sign of decreased awareness by the patient of the extent of his deficit.

Another major anatomic question remains on how limited a lesion may produce the syndrome.[45] Autopsies commonly show infarction which varies considerably from case to case, spreading over variable distances from the superior temporal plane to the parietal, occipital, and temporal regions. There are only a few well-studied cases of focal lesions confined to the superior temporal plane. As a consequence of the wide differences in the neuropathologic basis for the clinical syndrome, there is considerable variation in what different authors accept as the anatomical boundaries of Wernicke's area. For some, the area is considered to be confined strictly to the superior temporal plane just posterior to Heschl's transverse auditory gyri, and ending before or at the inferior parietal lobules posteriorly and the second temporal convolution inferiorly. Other authors consider that the region is simply the large posterior Sylvian territory, encompassing all the previously mentioned areas and extending as far back as the anterior occipital region. This lack of universal agreement as to the extent of Wernicke's area has led to considerable ambiguity in the components of the individual syndrome.

Behavioral findings provide yet another series of problems for unitary views of language function, as well as the opportunity to test a number of predictions implicit in traditional theses. As alluded to above under Vocal Output Channel, demonstration of opposite relational deficits in test scores with words and single letters between cases clinically classified as total aphasia or as central aphasia, respectively, leads to the realization that the relational deficit in total aphasia is not identical to that in central aphasia, and forces the abandonment of the assumption that a common deficit profile encompasses all relational performances in cases of aphasia. However, the coexistence of the severe output channel deficit in oral naming in total aphasia dilutes the significance of the findings somewhat, since other large differences separate the two types of cases.

The demonstration of differential deficits among patients who show only relational deficits further dispels notions of a unitary hierarchical deficit profile in aphasia. For example, some cases perform better in nonidentity tasks involving matching than in oral naming, and better in oral than in written naming,[43] while others show a superiority of nonidentity oral naming over both matching-to-sample and written naming for a given class of stimulus materials.[44] With different classes of stimulus materials, exceptions have been documented in which scores in nonidentity tasks with one material exceed those in another with one patient, while the opposite hierarchy of scores with these materials is seen in another patient.[44]

Evidence of still greater complexity in relational deficit profiles is provided by examples of different deficits with different materials in the *same* patient. One patient,[44] for example, experienced more difficulty in naming (reading) visual picture names than in naming the pictures; with colors and color names, however, the opposite was true—he had more trouble naming colors than visual color names.

Evolution of the deficit profiles across time also reveals a number of surprising changes. A smooth evolution sometimes occurs,[35] all scores rising uniformly and gradually to approximate satisfactory levels. In a number of cases, however, improvements occur gradually in one or more test stimulus materials, input, or response channels, leaving others essentially unchanged or improving at a much slower rate.[44] As a consequence of these unequal changes, the later profile is quite different from that predicted by the initial assessments. Autopsied cases[33] present anatomic findings for which a decision has to be made regarding the behavioral correlation. Failure of investigators to follow these evolutions has probably contributed significantly to interpretive

problems in retrospective reviews of clinical anatomical studies.

One byproduct of the systematic behavioral approach is the opportunity to assess predictions of deficit profiles based on traditional syndrome formulation. The behavior presumed to be involved in spelling, in particular, proved of interest.[34,43] The steps involved in pronouncing words in response to dictated spelled words, or conversely, in spelling aloud in response to dictated words, have been held to require, first, the "mental" transfer of auditory to visual images, and then the "reading" aloud of these mental images as words or sequences of single letters.[14] These views are the basis for explaining the impaired performance on spelling tasks by patients with the syndrome of dyslexia and dysgraphia. Destruction of the angular gyrus, held responsible for the mental transformations, would be expected to result in spelling deficits. By transforming the presumed mental operations into observable behavior, it was possible to test these predictions, and to find them unsupported by data. Patients who could pronounce dictated spelled words, and spell dictated pronounced words were, nevertheless, deficient in writing the dictated spelled words, that is to say, in explicitly demonstrating transformation of the auditory stimuli to their visual graphic equivalents. Nor could they read visually presented words aloud, the second presumed component of the mental task. Thus, explicit behavioral analysis revealed patients who could perform both spelling tasks, yet were unable to perform the tasks whose "mental" accomplishment was supposed to make spelling possible. Verifiable behavioral alternatives to such mentalistic mechanisms appear warranted if we are to avoid the postulation of plausible-sounding anatomic correlations to explain nonexistent behavioral processes, or vice versa.

The problems posed above for unitary notions of aphasia remain unsolved; the behavioral data are not as yet sufficient in scope to supplant traditional formulations in their entirety. Perhaps the major value of the behavioral observations at present is to call attention to the usefulness of the methodology. By delineating individual components of the deficit profile, some understanding of the hierarchies of relevant variables can be achieved. Behavioral studies also suggest that one should approach aphasia by emphasizing techniques which are most likely to reveal behavior that is still available to the patient, rather than design tests to promote errors. It may even become feasible to measure the deficits in aphasia by the lengths the examiner must go to provide a setting for the patient to accomplish the desired behavior. By placing the burden on the examiner to find the patient's capacities, deficits reflecting artifacts of the test situation would be reduced, and emphasis would shift to the delineation of variables which permit the patient to acquire new behavior, and perhaps mitigate his aphasia.

¶ Approach to a Clinical Case of Aphasia

The concern of the clinician approaching a case of aphasia is to clarify the syndrome presented sufficiently to make judgments on the likely anatomic regions affected and on the etiology of the brain injury.

The clinical situations where assessment of aphasia is needed generally fall into four large groups. (1) The patient appears intact and the question arises whether there is any deficit in interpersonal communication at all. Examples include patients who have suffered traumatic head injury, are recovering from suspected encephalitis, or are in the early stages of suspected brain tumor or degenerative brain disease; (2) The patient is grossly aphasic. The approach in such a case involves the attempt to establish what positive behavior, of any kind, is available to the patient, so as to assess what regions of the brain can be inferred to have survived. Examples include patients suffering massive traumatic head injury, devastating strokes, serious encephalitis, and the like; (3) Aphasia may form an important part of the clinical picture and analysis of the positive and negative features of the aphasic deficit may provide diagnostic considerations not available by other means; and (4) There is a heterogenous group of aphasic syndromes which frequently pass unnoticed in the general physical and sometimes even in the neurologic examination. The alert consultant can find a fair percentage of such cases by constant readiness to pursue the required tests.

When the patient appears intact, he has to be presented with the most difficult of aphasic tests. The purpose is not to analyze errors, but to anticipate satisfactory performance. If the patient performs well, such tests should put questions of aphasia to rest. If he does poorly, little or no information regarding the nature of the aphasia has been provided. In such an instance, the examiner has learned merely that tests which do permit analysis of errors will be necessary. An example of a complex test is Marie's three-paper test.[4] Others include a complex picture of incongruous situations used in standard IQ tests, dictated or printed familiar metaphors (a rolling stone gathers no moss, etc.) and word problems from many of the standard IQ tests; the patient is required to describe or write his explanation or solution. In special situations, when the patient's deficits preclude lengthy written or spoken responses, difficult tests involving several steps can be created to permit a minimal motor response to reflect a great deal of complex unobservable behavior. For example, when a patient is asked to hold up the number of fingers that correspond to the position in the alphabet occupied by that letter in the alphabet sequence that comes immediately after the first letter in the name Boston. If he immediately puts up three fingers to correspond to the letter "C," a great deal of behavior has been assessed and the question of aphasia is largely settled. Clearly, these complex tests are of value only in saving examination time in the intact case.

Cases presenting a gross severe aphasia pose almost the opposite problem. In this situation, one attempts to determine what behavior, if any, is available to the patient. The patient should be roused to a state of full alertness, if necessary, before concluding that the patient is untestable. Then, initial attempts should be made to use the simplest and most direct commands, with simultaneous demonstrations of the desired movements. Should some response be forthcoming, it must be determined whether the patient is mimicking the movements or is responding to the content of the command. For spoken responses the examiner can dictate short sounds (ah) and encourage repetition. For graphic responses, simple shapes (circle), etc.; for motor responses, simple movements (wave) may serve to establish some behavior. Any identity tests performed satisfactorily serve to indicate that the input and response channels function per se.

Cases not coming under any form of identity test control can still be profitably examined by using aversive stimuli. Inferences regarding right hemisphere function can be gained in the patient for whom simple avoidance behavior can be conditioned by preceding a noxious stimulus delivered to the left side with a visual, auditory, or somesthetic stimulus. Some assessment of memory can also be made by repeating these tests at regular intervals without retraining.

If the simple identity tests can be performed, then simple nonidentity forms of the same tasks can be done. Advantage should be taken of any incidental movement by the patient, since such occurrence is proof of their availability as behavior per se. Examples include coughing, smiling, turning over in bed, etc. The words involved in commands for these movements should be used for the tests of repeating from dictation and copying from sight. Then these words can be used as dictated commands to try to elicit written responses, and as visual commands for praxic motor or spoken response. Should this much behavior be accessible, the patient can then be further analyzed as outlined in the next section.

Whatever data are obtained provide a baseline for observation of later changes. Declines in the behavioral state may prompt a change in the therapy, or improvement may demonstrate the effectiveness of treatment.

Should the tests described above demonstrate some nonidentity behavior, further

analysis of the case is justified. The case may be one for whom analysis of the aphasic syndrome will help clarify the diagnosis. Such efforts can be expected to take time. It will be necessary to use a variety of stimulus materials, to attempt to establish some form of behavioral control with reinforcement techniques (using spoken words, such as good, money, food, etc.), and the identity, then nonidentity, behavior with the various input, and response modalities.

A gratifying by-product of such an analysis is a surprising number of instances in which some differential performance profile emerges that permits a diagnosis of one of the less severe aphasia syndromes. Most frequently observed is a case whose deficit was initially interpreted as motor aphasia or even total aphasia, and for whom analysis permits classification as pure word mutism. Similarly, the rarer cases of pure word deafness usually are considered initially to reflect central, or Wernicke's, aphasia. In the more severe syndromes, the main purpose of such analysis is to establish a baseline for further changes. For example, a hypertensive hemorrhage frequently evolves from a syndrome of minimal central aphasia to fully developed total aphasia, as may temporal-lobe abscess and deep-seated primary or metastatic brain tumor. By contrast, embolic involvement of the cerebrum rather frequently begins as total aphasia only to change to motor aphasia or central aphasia, and finally to a syndrome of amnestic aphasia. Evolution toward or away from more serious deficits is frequently of great value in establishing the etiologic diagnosis in an individual case.

The last group of patients are those for whom the diagnosis of a specific syndrome may be overlooked in more routine clinical medical or neurologic examination. These syndromes require the use of special techniques for their delineation, but depend chiefly upon the awareness of the examiner that these syndromes can exist in a patient whose conversational behavior appears essentially normal. The syndromes include those of the pure alexias with or without agraphia, amnestic aphasia, and the syndromes of nondominant hemisphere ideomotor apraxia (not discussed in this chapter). More exotic behavioral syndromes include "simultanagnosia"[53] and Balint's syndrome.[3] The failure of spontaneous speech with preserved repeating from dictation which can transiently characterize involvement of the anterior cerebral artery territory in the dominant hemisphere,[42] and the syndromes of grossly inappropriate factual content of conversation which may occur in states of increased intracranial pressure and/or unilateral or bilateral frontal disease,[29] are all uncommon, and are beyond the scope of this chapter.

Bibliography

1. ADAMS, R. D. and J. P. MOHR. "Affections of Speech," in M. M. Wintrobe et al., eds., *Harrison's Principles of Internal Medicine*, 7th ed., pp. 137–148. New York: McGraw-Hill, 1974.
2. ALAJOUANINE, T., A. OMBREDAME, and M. DURAND. *Le Syndrome de Désintégration Phonétique dans l'Aphasie*. Paris: Masson, 1939.
3. BALINT, R. "Seelenlähmung des 'Schauens,' Optische Ataxie, Räumliche Störung der Aufmerksamkeit," *Monatsschr. Psychiatr. Neurol.*, 25 (1909), 51.
4. BOLLER, F. and L. A. VIGNOLO. "Latent Sensory Aphasia in Hemisphere-Damaged Patients: An Experimental Study with The Token Test," *Brain*, 89 (1966), 815.
5. BRAIN, R. *Speech Disorders*. London: Butterworths, 1965.
6. BROCA, P. "Remarques sur le siège de la faculté du langage articulé; suivies d'une observation d'aphémie," *Bull. Soc. Anat.*, 6 (1861), 330.
7. BROWN, J. W. *Aphasia, Apraxia, and Agnosia*. Springfield, Ill.: Charles C. Thomas, 1972.
8. CHESTER, E. D. *"Aphasia," Bull. Neurol. Inst.*, 6 (1937), 134–144.
9. DEJERINE, J. and C. MIRALLIE. *L'Aphasie Sensoriélle*. Paris: Steinheil, 1896.
10. DEJERINE, J. and N. VIALET. "La Localisation anatomique de la cécité verbale," *C. R. Soc. Biol. (Paris)*, 4 (1891), 61.
11. DENNY-BROWN, D. and R. A. CHAMBERS. "The Parietal Lobe and Behavior," *Res. Publ. Ass. Res. Nerv. Ment. Dis.*, 36 (1958), 36.
12. EISENSON, J. *Examining for Aphasia*. New York: The Psychological Corporation, 1954.
13. GAZZANIGA, M. S., J. E. BOGEN, and R. W. SPERRY. "Observations on Visual Perception after Disconnection of the Cerebral Hemispheres in Man," *Brain*, 88 (1965), 221.
14. GESCHWIND, N. "Disconnection Syndromes in Animals and Man," *Brain*, 88 (1965), 237, 585.
15. ———. "Focal Disturbances of Higher Nervous Activity," in P. B. Beeson and W. McDermott, eds., *Cecil-Loeb Textbook of Medicine*, 13th ed., pp. 99–102. Philadelphia: Saunders, 1971.
16. GOLDSTEIN, K. *Language and Language Disturbances*. New York: Grune & Stratton, 1948.
17. HEAD, H. *Aphasia and Kindred Disorders of Speech*. New York; Macmillan, 1926.
18. HOWES, D. "Application of the Word-frequency Concept to Aphasia," in A. V. S. de Reuck and M. O'Connor, eds., *Disorders of Language*, pp. 47–75. Boston: Little, Brown, 1964.
19. JAKOBSON, R. "Towards a Linguistic Topology of Aphasic Impairments," in A. V. S. de Reuck and M. O'Connor, eds., *Disorders of Language*, pp. 2–42. Boston: Little, Brown, 1964.
20. KLEIST, K. *Gehirnpathologie*. Leipzig: Barth, 1934.
21. KREINDLER, A. and A FRADIS. *Performances in Aphasia*: Paris: Gauthier-Villars, 1968.
22. KREINDLER, A. and V. IONASESCU. "A Case of 'Pure' Word Blindness," *J. Neurol. Neurosurg. Psychiatry*, 24 (1961), 257.
23. LEICESTER, J., M. SIDMAN, L. T. STODDARD et al. "Some Determinants of Visual Neglect," *J. Neurol. Neurosurg. Psychiatry*, 32 (1969), 580.
24. ———. "The Nature of Aphasic Responses," *Neuropsychol.*, 9 (1971), 141.
25. LHERMITTE, F. and J. C. GAUTIER. "Aphasia," in R. J. Vinken and G. W. Bruyn, eds., *Handbook of Clinical Neurology*, Vol. 4, pp. 84–104. Amsterdam: North-Holland, 1969.
26. LICHTHEIM, L. "On Aphasia," *Brain*, 7 (1887), 433.
27. LIEPMANN, H. "Diseases of the Brain," in C. W. Burr, ed., *Curschmann's Textbook on Nervous Diseases*, Vol. 1, pp. 467–80, 518–51. Philadelphia: Blakiston, 1915.
28. LIEPMANN, H. and M. PAPPENHEIM. "Über einen Fall von Sogenannter Leitungsaphasie mit Anatomischem Befund," *Z. Neurol. Psychiatr.*, 27 (1914), 1.
29. LURIA, A. *Higher Cortical Functions in Man*. New York: Basic Books, 1966.
30. MARIE, P. "Révision de le quéstion de l'aphasie," *Sem Méd.*, 26 (1906), 241, 493, 565.
31. MASSACHUSETTS GENERAL HOSPITAL. *Case Records*, Autopsy No. 31772. Boston: 1968.
32. MOHR, J. P. "Rapid Amelioration of Motor Aphasia," *Arch. Neurol.*, 28 (1973), 77.
33. MOHR, J. P., J. LEICESTER, L. T. STODDARD et al. "Right Hemianopia with Memory and Color Deficits in Circumscribed Left Posterior Cerebral Artery Territory Infarction," *Neurology*, 21 (1971), 1104.
34. MOHR, J. P. and T. R. PRICE. "An Unusual Case of Dyslexia with Dysgraphia," *Neurology*, 21 (1971), 430.
35. MOHR, J. P., M. SIDMAN, L. T. STODDARD et al. "Evolution of the Deficit in Total Aphasia," *Neurology*, 23 (1973), 1302.
36. PERSHING, H. "A Case of Wernicke's Conduction Aphasia with Autopsy," *J. Nerv. Ment. Dis.*, 27 (1900), 369.
37. PICK, A. *Die Agrammatischen Störungen*. Berlin: Springer, 1913.
38. PORCH, B. *Porch Index of Communicative Abilities*. Palo Alto: Consulting Psychologist Press, 1970.
39. REITAN, R. "The Significance of Dysphasia for Intelligence and Adaptive Abilities," *J. Psychol.*, 50 (1960), 355.
40. RUSSELL, W. R. and M. L. E. ESPIR. *Traumatic Aphasia*. London: Oxford, 1961.
41. SCHUELL, H., J. J. JENKINS, and E. JIMENEZ-PABON. *Aphasia in Adults*. New York: Hoeber, 1964.
42. SCHWAB, O. "Über Vorübergehende Aphasische Störungen nach Rindenexcision aus dem Linken Stirnhirn bei Epileptikern," *Dtsch. Z. Nervenkeilk.*, 94 (1926), 177.
43. SIDMAN, M. "The Behavioral Analysis of Aphasia," *J. Psychiatr. Res.*, 8 (1971), 413.
44. SIDMAN, M., L. T. STODDARD, J. P. MOHR et al. "Behavioral Studies of Aphasia: Methods of Investigations and Analysis," *Neuropsychol.*, 9 (1971), 119.
45. STARR, M. A. "The Pathology of Sensory Aphasia with an Analysis of Fifty Cases in

Which Broca's Centre was not Diseased," *Brain*, 12 (1889), 82.

46. TEUBER, H. L. "Lacunae and Research Approaches to Them," in C. H. Millikan and F. L. Darley, eds., *Brain Mechanisms Underlying Speech and Language*, pp. 204–216. New York: Grune & Stratton, 1967.
47. WEIGL, E. "On the Construction of Standard Psychological Tests in Cases of Brain Damage," *J. Neurol. Sci.*, 3 (1966), 123.
48. WEIGL, E. and A. FRADIS. "Semiologische Untersuchungen der Alexie," *Zh. Nevropatol. Psikhiatr.*, 59 (1959), 1425.
49. WEISENBURG, T. and K. E. MCBRIDE. *Aphasia*. New York: Hafner, 1964.
50. WERNICKE, C. *Der Aphasische Symptomencomplex*. Breslau: Cohn & Weigert, 1874.
51. ———. "The Symptomcomplex of Aphasia," in A. Church ed., *Modern Clinical Medicine*, pp. 265–324. New York: Appleton, 1908.
52. WHITAKER, H. A. "Neurolinguistics," in W. O. Dingwall, ed., *A Survey of Linguistic Science*, pp. 136–252. College Park: University of Maryland Press, 1971.
53. WOLPERT, I. "Die Simultanagnosie—Störung der Gesamtauffassung," *Z. Neurol. Psychiatr.*, 93 (1924), 397.