**APHASIC SYNDROMES**

**CLASSICAL APHASIC SYNDROMES**

The aphasic syndromes were first postulated in 1865 by Liebenheim, who built upon prior work, especially studies by Broca (1861) and Wernicke (1874). We begin with a discussion of these three studies.

Broca (1861) described a patient, Leborgne, with a severe speech output disturbance: Leborgne’s speech was limited to the monosyllable “tan.” In contrast, Broca described Leborgne’s ability to understand spoken language and to express himself through gestures and facial expressions, as well as his understanding of nonverbal communication, as normal. Broca claimed that Leborgne had lost “the faculty of articulate speech.” Broca related this impairment to damage of neural tissue: Leborgne’s brain contained a lesion whose center was in the posterior portion of the inferior frontal convolution of the left hemisphere. The lesion extended posteriorly into the parietal lobe. Broca related the most severe part of the lesion to the expressive language impairment. This area became known as “Broca’s area.” Broca argued that it was the neural site of the mechanism involved in speech production.

Over the ensuing years, many other cases of language impairments were described. In some, speech impairments were related to lesions in the left frontal lobe; other speech impairments were associated with more posterior lesions. In 1874, Wernicke published a paper that appeared to reconcile many of these different findings. He described a patient with a speech disturbance, but one that was very different from that seen in Leborgne. Wernicke’s patient was fluent; her speech, however, contained words with sound errors, other errors of word forms, and words that were semantically inappropriate. Also, unlike Leborgne, Wernicke’s patient did not understand spoken language. Wernicke argued the two impairments—that of speech production and that of comprehension—by arguing that the patient had sustained damage to “the storehouse of auditory word forms.” Under these conditions, speech would be expected to contain the types of errors that were seen in this case, and comprehension would be affected. Establishing the location of the lesion in this case was more problematic, however, as Wernicke did not have the opportunity to perform an autopsy on this patient. He did examine the brain of a second patient, however, whose language had been described by her physician in terms that made Wernicke think she had had a set of symptoms that were the same as those seen in his case. The lesion in this second patient occupied the posterior portion of the first temporal gyrus, also on the left. Wernicke suggested that this region, which came to be known as “Wernicke’s area,” was the locus of the “storehouse of auditory word forms.”

Wernicke’s paper was the first to describe an aphasic syndrome, in the sense of a constellation of symptoms. He had found two deficits—fluent paraphasic speech and poor auditory comprehension—and he related them both to a single functional abnormality: abnormal representations of the sound patterns of words. If he was right, these two symptoms ought to pattern together: they ought to improve naturally at similar rates, respond favorably to effective treatments, and worsen in response to the same aggravating conditions such as fatigue or distraction.

Wernicke went a step beyond relating the symptoms he saw in his patient to a single underlying deficit. He related them to the location of the lesion that he thought produced this deficit, in the area of the brain adjacent to the primary auditory cortex (Heschl’s gyrus). It made sense to Wernicke that a lesion in this location would affect the long-term storage of the sounds of words, because he thought that auditory stimuli were processed in a special way (e.g., as language) in the areas of cortex just adjacent to the primary auditory cortex. Wernicke also noted that Broca’s area, in which a lesion was thought to produce an impairment of motor speech, was adjacent to the motor cortex. He developed the germ of the general theory that receptive aspects of language processing are localized adjacent to primary sensory cortex and output language processes are adjacent to primary motor cortex. The permanent storage of the sounds of words in a receptive area of the brain was due to children first hearing, and only later producing, the words of their language.
Broca and Wernicke thus gave us the three fundamental principles that underlie the classical aphasic syndromes:

1. Language processors are localized (Broca, 1861).
2. Diverse language symptoms can be due to an underlying deficit in a single language processor (Wernicke, 1874).
3. Language processors are localized in brain regions because of the relationship of the processor to sensory or motor functions (Wernicke, 1874).

It remained for Lichtheim to apply these three principles to a wider range of symptoms. Lichtheim recognized seven syndromes (excluding those that affected reading and writing, which are not dealt with here):

1. Broca's aphasia: a severe expressive language disturbance reducing the fluency of speech in all tasks (repetition and reading as well as speaking) and affecting elements of language such as grammatical words and morphological endings, without an equally severe disturbance of auditory comprehension.
2. Wernicke's aphasia: the combination of fluent speech with erroneous choices of the sounds of words (phonemic paraphasias) and an auditory comprehension disturbance.
3. Pure motor speech disorders: anarthria, dysarthria, and apraxia of speech: output speech disorders due to motor disorders, in which speech is misarticulated but comprehension is preserved.
4. Pure word deafness: a disorder in which the patient does not recognize spoken words, but spontaneous speech is normal.
5. Transcortical motor aphasia: a disorder in which spontaneous speech is reduced but repetition is intact.
6. Transcortical sensory aphasia: a disorder in which a comprehension disturbance exists without a disturbance of repetition.
7. Conduction aphasia: a disturbance in spontaneous speech and repetition, consisting of fluent paraphasic speech, without a disturbance in auditory comprehension.

These syndromes are listed in Table 2–1. Lichtheim argued that these syndromes followed lesions in regions of the brain as depicted in schematic form in Figure 2–1. Before discussing Lichtheim's schema for relating these syndromes to these lesions, however, we need to mention two more aspects of Lichtheim's model. The first is that Lichtheim made the assumption that the meaning of words resided in the superior portion of the parietal lobe, indicated as C, for Concepts, in Figure 2–1. The second is that he assumed, and then justified on the basis of cases he described, that in speech production word meanings activated both word sounds in Wernicke's area and the motor speech planning mechanism in Broca's area, hence the two arrows originating in the Concept Center (C) in Figure 2–1.

With this background, Lichtheim's linking of the seven syndromes to lesion sites is quite straightforward. Broca's aphasia, which affects expressive language alone, is due to lesions in Broca's area, the center for motor speech planning adjacent to the motor strip. Wernicke's aphasia follows lesions in Wernicke's area that disturb the representations of word sounds. Pure motor speech disorders arise from lesions interrupting the motor pathways from the cortex to the brainstem nuclei that control the articulatory system. These disorders differ from Broca's aphasia in that they are not linguistic; they affect articulation itself, not the planning of speech. Pure word deafness affects the transmission of sound input into Wernicke's area. It therefore disrupts word recognition but not speech, since words themselves are intact and accessible for speech production purposes. Transcortical motor aphasia results from the interruption of the pathway from the Concept Center to Broca's area. This affects speech, but not repetition or comprehension. Transcortical sensory aphasia follows lesions between Wernicke's area and the concept center; repetition of words is intact, but comprehension is affected. Finally, Conduction aphasia follows from a lesion between Wernicke's area and Broca's area. Repetition is affected, but comprehension is intact. Speech is also affected, in the same way as in Wernicke's aphasia, because the sound patterns of words, though activated, are not transmitted properly to Broca's area to plan speech.

These syndromes have had a checkered history. They have been criticized on neuropsychological grounds (Marie, 1906; Moutter, 1908), dismissed as simplifications of reality that are only of help to schoolboys (Head, 1928), and ignored in favor of different approaches to language (Jackson, 1876; Goldstein, 1946). Nonetheless they have endured. Benson and Geschwind (1971) reviewed the major approaches to aphasia as they saw them, and concluded that all researchers recognized the same basic patterns of aphasic impairments, despite using different nomenclature.
Lichtsheim's seven original syndromes have been amplified. Three more syndromes have been added by theorists such as Benson (1979) and Lichtsheim's model has been rounded out with specific hypotheses about the neuroanatomical bases for several functions that he could only guess at. The three additional syndromes described by Benson (1979) are anoma, global aphasia, and isolation of the speech area. The symptoms and related neural localizations are shown in Table 2-2. Additional neuroanatomical foundations were first suggested in a very influential paper by Geschwind (1965), who argued that the inferior parietal lobe was a tertiary association area that received projections from the association cortex immediately adjacent to the primary visual, auditory, and somesthetic cortices in the occipital, temporal, and parietal lobes. Because of these anatomical connections, the inferior parietal lobe served as a cross-modal association region, associating word sounds with the sensory qualities of objects. This underlay word meaning, in Geschwind's view. Damasio and Tranel (1993) extended this model to actions, arguing that associations between word sounds and memories of actions were created in the association cortex in the inferior frontal lobe. Geschwind (1965) and Damasio and Damasio (1980) also argued that the anatomical link between Wernicke's and Broca's areas (in which a lesion caused conduction aphasia) was the white matter tract known as the "arcuate fasciculus." These extensions and clarifications of Lichtsheim's model are shown in Figure 2-2.

Table 2-2. Additional Classical Aphasic Syndromes

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Clinical Manifestations</th>
<th>Hypothetical Deficit</th>
<th>Classical Lesion, Location</th>
</tr>
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<tbody>
<tr>
<td>Anomic aphasia</td>
<td>Disturbance in the production of single words, most marked for common nouns; intact comprehension and repetition</td>
<td>Disruptions of concepts and/or the sound patterns of words</td>
<td>Inferior parietal lobe or connections between parietal lobe and temporal lobe, can follow many lesions</td>
</tr>
<tr>
<td>Global aphasia</td>
<td>Major disturbance in all language functions</td>
<td>Disruption of all language-processing components</td>
<td>Large portion of the perisylvian association cortex</td>
</tr>
<tr>
<td>Isolation of the language zone</td>
<td>Disturbance of both spontaneous speech (spare, halting speech) and comprehension, with some preservation of repetition; echolalia is common</td>
<td>Disconnection between concepts and both representations of word sounds and speech production mechanism</td>
<td>Cortex just outside the perisylvian association cortex</td>
</tr>
</tbody>
</table>

In a famous paper, Hughlings Jackson described a patient, a carpenter, who was mute but who mustered up the capacity to say "Master's in response to his son's question about where his tools were. Jackson's poignant comments convey his emphasis on the conditions that provoke speech, rather than on the form of the speech itself.

The father had left work; would never return to it. He was away from home; his son was on a visit, and the question was directly put to the patient. Anyone who saw the abject poverty the poor man's family lived in would admit that these tools were of immense value to them. Hence we have to consider as regards this and other occasional utterances the strength of the accompanying emotional state. (Jackson, 1875, p. 181)

Goldstein (1948) was concerned about whether patients, both those with and those without aphasia, were capable of abstracting away from the immediacy of a situation to consider longer-term goals—a capacity he called the ability to assume "the abstract attitude."

Both Jackson and Goldstein sought a description of language use as a function of motivational and intellectual states, and tried to describe aphasic disturbances of language in relation to the factors that drive language production and make for depth of comprehension. This is surely a vital aspect of understanding language impairments. In many ways it is more humanly relevant than a description of language impairments that focuses on which phonemes are produced in spontaneous speech or repetition. Unfortunately, Jackson and Goldstein's approach is a very intractable goal, both in terms of psychological descriptions and in terms of relating these specific motivational states to the brain. Wernicke, Lichtsheim, Geschwind, and the researchers who conceived and developed the framework of the classical syndromes focused aphasiology on the description of the linguistic representations and psycholinguistic operations that are responsible for everyday language use. The three and a half decades since publication of Geschwind's paper have brought new evidence for these syndromes and their relationships to brain lesions. Aphasic syndromes have been related to the brain using a series of neuroimaging techniques—first fMRI scanning.
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then computed tomography (CT), magnetic resonance (MR), and positron emission tomography (PET). All have confirmed the relationship of the major syndromes to lesion locations. Broca's aphasia is associated with anterior lesions; Wernicke's aphasia is associated with posterior lesions, centered in the temporal–parietal juncture (Hayward et al., 1977; Naeser and Hayward, 1978; Kertesz, 1979; Naeser, 1989). Pure motor deficits of speech are associated with subcortical lesions (Collett et al., 1983; Alexander et al., 1987; Naeser et al., 1989). Pure word deafness is associated with lesions in the auditory association areas and surrounding white matter tracts, often bilaterally (Denes and Semenza, 1975; Auerbach et al., 1982; Collett et al., 1984; Metz-Lutz and Dahn, 1984). Transcortical motor aphasia and transcortical sensory aphasia are associated with watershed infarcts between the anterior and middle cerebral arteries (transcortical motor aphasia; Freedman et al., 1984) and between the middle and posterior cerebral arteries (transcortical sensory aphasia; Kertesz et al., 1989). Conduction aphasia is associated with smaller lesions that often appear to affect the arcuate fasciculus (Damasio and Damasio, 1990). When the lesioned brain is imaged by PET, which is sensitive to metabolic, as opposed to its structural abnormalities, damage following stroke is seen to be more widespread and the relationships between syndromes and lesion are less obvious, but even then these relationships are discernable (Metter et al., 1983, 1984, 1986, 1988, 1989, 1990, 1995; Kempler et al., 1985, 1991). Concepts of how the brain is organized have changed considerably since 1965, let alone since 1855. Neuroscientists no longer use the term "center." Instead, they talk about "systems neuroscience" in which there are "distributed large-scale neocortical nets" with "functional specializations" (Mesulam, 1980). There are new models to explain how the elements in the brain accomplish computations (Rumelhart and MacClelland, 1986). There are new classifications of types of cortex (Mesulam, 1996). But despite these changes in both terminology and in our ability to model language processing, the central idea that underlies the classical syndromes—that the brain supports complex psychological functions through a set of connected areas, each related to a set of cognitive operations—is ubiquitous in cognitive neuroscience. Although the term "center" is anachronism, the essence of the concept is alive and well and constantly applied to other terms, such as aphasia. Recent reviews of aphasia in leading medical journals (e.g., Damasio, 1992), while acknowledging the changes in neuroscience, retain the aphasic syndromes and their relationships to the brain. Besides their clinical value, the syndromes also have prognostic significance. Kertesz and McCabe (1977) have shown that Wernicke's aphasia tends to resolve toward either conduction aphasia or anomia, and global aphasia toward Broca's aphasia. The relation between lesion site and recovery in specific syndromes has been charted (Kertesz et al., 1979, 1985). If we return to the original Webster definition ("a number of symptoms occurring together and characterizing a particular disease") and the requirement that a syndrome be a useful category, and ask if the classical syndromes are a success, the answer is clearly "yes." Nonetheless, these classical aphasic syndromes are under attack. The dissatisfaction many researchers feel toward these syndromes comes from several quarters. One problem lies with the level of description of language and language functioning that they present. A related problem is that they do not cover all patients' symptoms well. A third problem is that there are many exceptions to the patterns of localization they hypothesize. A fourth is related to the utility of these syndromes in developing approaches to therapy. We shall review these issues in turn.

PROBLEMS WITH THE CLASSICAL APHASIC SYNDROMES

A major limitation of the classical syndromes is that they stay at arm's length from the linguistic details of language impairments. The classical aphasic syndromes basically reflect the relative ability of patients to perform entire language tasks (speaking, comprehension, etc.), not the integrity of specific operations within the language processing system. This is not to say that there are no linguistic or qualitative descriptions of language in the characterizations of the classical aphasic syndromes, but to point out that they are incomplete and unsystematic. For instance, the speech production problem seen in Broca's aphasia can consist of one or more of a large number of impairments. Benson and Geschwind (1971) describe Broca's aphasia as follows:

The language output of Broca's aphasia can be described as nonfluent. It is sparse, dysprosodic, and poorly articulated; it is made up of very short phrases and it is produced with effort, particularly in initiation of speech. The speech consists primarily of substantive words, i.e., nouns, action verbs, or significant modifiers. The pattern of short phrases lacking prepositions is often termed "telegraphic speech." . . . Comprehension of spoken language is much better than speech but varies, being completely normal in some cases and moderately disturbed in others. (p. 7)

The abnormalities that determine that a patient is a Broca's aphasic are only related to each other at a very general level of description of the language-processing system—that of speech production. The definition of the syndrome ignores finer distinctions. For instance, the symptoms of "dysprosodic" speech, "poorly articulated" speech, and "short phrases lacking prepositions" are likely to reflect disturbances in the assignment of prosody, specifying the articulatory gestures for phonemes and construction of syntactic forms, respectively. If all we know about a patient is that he or she is a Broca's aphasic, we cannot tell which of these problems might exist.

The heterogeneity of deficits across patients with the same syndrome is not limited to Broca's aphasia. Patients with Wernicke's aphasia can have deficits affecting either the sounds of words or their meanings or both, as well as many of other language-processing deficits (Luria, 1987). There are at least two major deficits that underlie conduction aphasia, one affecting word production and one affecting verbal short-term memory (Shalloe and Warrington, 1977). Schwartz (1984) pointed out that the actual application of the criteria used to classify aphasic patients into the classical syndromes has led to a grouping together of many patients with no symptoms in common. At the same time as patients with the same syndrome can have different deficits, identical deficits occur in different syndromes. For instance, certain types of naming problems can occur in any aphasic syndrome (Benson, 1979).

Because of these problems, the classical syndromes do not do a very good job of classifying many aphasic patients. In practice, most applications of the clinical taxonomy result in widespread disagreements over a patient's classification (Holland et al., 1986) and/or in a large number of "mixed" or "unclassifiable" cases (Lecours et al., 1983). The criteria for induction in a syndrome are often somewhat arbitrary: how bad must a patient's comprehension be for the patient to be considered Wernicke's aphasic instead of conduction aphasic, or a global aphasic instead of a Broca's aphasic? There have been many efforts to answer this question (see, e.g., Goodglass and Kaplan, 1972, 1982; Kertesz, 1970), but none is satisfactory. As an example consider the patient whose scores on the Boston Diagnostic Aphasia Examination are shown in Figure 2–3. The patient does not fit the formal criteria set out by Goodglass and Kaplan for any syndrome.

The third problem that the classical aphasic syndromes face is that they are not as well correlated with lesion sites as the theory claims they should be. Several studies above found general correlations between lesion site and syndrome, but a closer look at those studies and at others reveals many discrepancies. First, virtually all studies exclude many types of lesions, such as various sorts of tumors and degenerative diseases, and others. The rule of thumb seems to be that the classical syndromes are only related to lesion sites in cases of rapidly developing lesions, such as stroke. Even with these types of lesions, the syndromes are never applied to acute and subacute phases of the illness. But the situation is worse than that. In the chronic phases of diseases such as stroke, at least 15% of patients have lesions that are not predictable from their syndromes (Basso et al., 1985), and some researchers think this number is much higher—as high as 40% or more, depending on what counts as an exception to the rule (de Bleser, 1988). Also, we now know
that the relationship between lesion location and syndrome is more complex than we had previously thought, even in cases where the classical localization captures part of the picture. Broca's aphasia, for instance, does not usually occur in the chronic state after lesions restricted to Broca's area, but requires much larger lesions (Mohr et al., 1978).

We said at the beginning of this chapter that a good syndrome can account for variability. The variability in lesion–deficit correlations is a challenge to the classical aphasic syndromes. One approach to explaining this variability without abandoning the syndromes is to look for individual differences in the brain regions that can take over language after a lesion occurs. Some of the factors that affect this process are partially understood. For instance, handedness affects lateralization of language, and it is likely that there is more ability of the right hemisphere to take over language functions after a lesion occurs in patients who are not as strongly right-handed. Age affects recovery from lesions, with much better recovery seen in children than in adults and possibly some effect of age occurring within the adult life span. However, these factors do not explain all the observed variability. Lesions of similar size in similar locations can be associated with very different types and levels of impairments in patients with similar degrees of right-handedness, of similar ages, of the same sex, and with similar educational and socioeconomic backgrounds. The classical syndromes, and the model of language–brain relationships on which they are based, are missing something.

Some theories have gone farther in their criticisms of the classical syndromes in the area of syndrome–lesion correlations. They have argued that the relationships between the classical syndromes and the locations of lesions are not due to the effects of brain lesions on language but to the effects of lesions in different
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Table 2–3. Basic Levels of Language

<table>
<thead>
<tr>
<th>Level</th>
<th>Form</th>
<th>Associated Semantic Values</th>
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<tbody>
<tr>
<td>Lexical</td>
<td>Words consisting of sound units (phonemes) organized into higher-order structures (tense, syllables). Syllabic prominence is masked by stress or tone. Words have syntactic categories. Open-class, content words (noun, verb, adjective) accept new members. Closed class, function words (article, auxiliary, pronoun, etc.) are a fixed set.</td>
<td>Content words describe items, actions, or features. Function words make logical connections. Words are primarily denotative and related to categories.</td>
</tr>
<tr>
<td>Morphological</td>
<td>Words formed from other words by prefixes, suffixes, and infixes. Morphology can be inflectional or derivational.</td>
<td>Inflectional morphology indicates connections between words (e.g., agreement). Derivational morphology changes syntactic category (e.g., changes adjectives to nouns: happy → happiness). Declension, aspect, and temporal markings, and other phenomena are also morphological in nature.</td>
</tr>
<tr>
<td>Sental</td>
<td>Syntactic structures: hierarchically arranged sets of syntactic categories over which relations are defined (e.g., subject, object, co-command)</td>
<td>Semantic relations between words, such as thematic roles (agent, theme, beneficiary, etc.), attribution of modification, scope of quantification, etc. These &quot;propositional&quot; aspects of meaning describe events and states of affairs. Propositions can have truth values and be used to update semantic memory, to reason, and for other functions.</td>
</tr>
<tr>
<td>Discourse</td>
<td>Position in syntactic structures (e.g., first words play important roles in discourse). Intonational contrastive items is used to indicate some semantic value.</td>
<td></td>
</tr>
</tbody>
</table>

PSYCHOLINGUISTIC APPROACH TO APHASIC SYNDROMES

Many of the problems with the classical syndromes outlined above would be addressed, and possibly solved, if we described aphasic language impairments in terms of impairments of psycholinguistic operations rather than in terms of tasks. Researchers have included such descriptions in their accounts of syndromes since the beginning of the study of aphasia, but a true revision of the classical syndromes requires a thorough enumeration of all the psycholinguistic operations that are defective in a patient and a taxonomy of aphasic deficits in terms of such impairments. This approach can be seen as a natural extension of the approach that led to the classical syndromes, rather than as an entirely new direction. It adds to the classical approach a greater concern for the details of psycholinguistic processing, while essentially adopting the classical framework for the field as being the description of disorders in the tasks of speaking, auditory comprehension, reading, and writing.

Through the language code particular types of representations are connected to particular aspects of meaning. People use words to connect phonological units with items, actions, properties, and logical connections. In sentences words are related to each other in hierarchical syntactic structures to determine semantic relationships between them such as who is accomplishing or receiving an action. Table 2–3 attempts to systematically describe the levels of linguistic representations that are basic to language. Tables 2–4 and 2–5 and Figures 2–4 and 2–5 present models of how these forms are processed. Although Tables 2–3 through 2–5 and Figures 2–4 and 2–5 are highly simplified, they present the basic elements of language and the language-processing system.

There are many levels of detail at which one can describe language and language-processing. Depending on the level of detail at which language impairments are described, there may be hundreds of primary language-processing impairments. For instance, we may recognize a disturbance of converting the sound wave-form into linguistically relevant units of sound—acoustic-to-phonemic conversion—or we may recognize disturbances affecting the ability to recognize subsets of phonemes, such as vowels, consonants, stop consonants, fricatives, and nasals (Saffran et al., 1976). For many clinical purposes, an adequate way to approach a psycholinguistic taxonomy of aphasic impairments is at the level of detail that identifies language-processing impairments in terms of sets of related operations responsible for activating the major forms of the language code and their associated meanings in the usual language tasks.
of speaking, comprehending auditorily presented language, writing, and reading. These levels of the language code are listed in Table 2–3. It needs to be recognized, however, that for many research purposes and for some clinical purposes, a more detailed taxonomy of linguistic representations and operations is needed.

The psycholinguistic approach to aphasia differs from the classical syndrome approach in recognizing more levels of language. Most (though certainly not all) of the researchers and clinicians who developed the classical syndromes concentrated on single words. In the psycholinguistic approach there is equal concern for disorders of the processes through which words are formed from one another (morphology and compounding) and those through which sentences, syntactic structures, intonation contours, and aspects of discourse structure and meaning are formed or used. For instance, a great deal of attention has focused on deficits in the ability to use syntactic structure to determine aspects of sentence meaning (Caramazza and Zurif, 1976; Caplan et al., 1985; 1986; Caplan and Hildebrandt, 1988; Grodzinsky, 1996; Berndt et al., 1996), an area of aphasiology that was never discussed in the classical syndromes. Thus progress has been made toward solving the first problem that confronts the classical syndromes—the need for more detail about the psycholinguistic operations affected in a patient.

A second way in which the psycholinguistic approach to aphasia differs from the classical syndromic approach is related to the first difference. In the classical approach to syndromes, each patient has to be classified into one and only one syndrome; a patient cannot be a conduction aphasic and a Wernicke's aphasic at the same time. In the psycholinguistic approach, a patient may have—indeed, is likely to have—more than one deficit. A patient can have an output disorder affecting one aspect of processing and a completely unrelated disturbance of reading. Thus the second major problem with the classical syndromes, the difficulty in fitting many patients into a single syndrome. This problem essentially evaporates.

We may pose the question of what constitutes a "syndrome" in this approach, if by "syndrome" we mean "a number of symptoms occurring together and characterizing a particular disease." The answer is that disturbances of some components of the language processing system have a number of functional consequences. For instance, most models of word sound production postulate a "phonological output lexicon" in which the sounds of words are represented, and a "phonological output
buffer" in which these sounds are used to plan articulatory gestures (see Fig. 2-4). Both these components are needed because the way in which a word is pronounced varies as a function of context while the permanent representation of the sound of a word presumably does not. The phonological output lexicon only deals with words, whereas the phonological output buffer is used for all speech production, including repeating and reading nonwords. A disturbance of the phonological output buffer would result in phonemic errors in all speech production tasks—spontaneous speech, object naming, repetition, and reading—and would affect both words and nonwords. This "syndrome" of the phonological output buffer has been described in many patients (see, e.g., Caplan and Waters, 1980). Disorders of the phonological output lexicon would only affect the activation of words from their meanings as in spontaneous speech, naming objects, and providing words from definitions, because both words and nonwords can gain access to the phonological output buffer through a word-lexical route in repetition and reading, as shown in Figure 2-4.

The psycholinguistic approach requires detailed study of individual patients to identify their deficits. This has led researchers to use new tasks, some of which are not part of the usual use of language, such as repetition and reading aloud of nonwords. For example, the model in Figure 2-4 would predict that a patient with a disturbance of the phonological output buffer would be able to activate the sounds of words from their meanings, even if the patient could not produce them correctly. Therefore, such a patient should be able to say whether the words for two pictures were the same, a task called "picture homophone matching." This is a very useful task for analyzing the speech production disorders seen in aphasia.
that emerges from the psycholinguistic lab, not from a study of everyday language use. There are also many psycholinguistic tasks that use reaction times as dependent variables (lexical priming, word recognition) and judgments of grammaticality or plausibility that have been helpful in studying aphasic patients. These tasks are particularly useful in describing patients' "on-line" language processing of language—the unconscious processes that go on when subjects speak and comprehend of speech. The use of these techniques has shown that some patients retain abilities that would not be suspected through consideration of their performances on standard tasks. For instance, use of priming tasks, in which subjects respond to a word faster if it has been preceded by a semantically related word, has shown that some Wernicke's aphasics retain at least some knowledge of the meanings of words, despite not being able to demonstrate such knowledge on word-picture matching tasks (Milberg and Blumstein, 1981; Blumstein et al., 1982). These findings provide new information that radically changes our ideas about what is wrong with such patients. The importance of this additional information extends beyond characterizing a patient's deficit and therefore being able to fit that patient into a category of aphasia. It is relevant to the issues of localization and therapy as well.

In the psycholinguistic approach to aphasia, the problem of localization of language is addressed by attempting to determine the location of the various language processors. In this enterprise, information from deficit–lesion correlation is used: the deficits are defined in terms of language processing components, not syndromes. It also relies on changes in hemodynamic responses to language processing as measured by PET and functional magnetic resonance imaging (fMRI), electro- and magnetoencephalographic correlates of language processing (evoked response potentials [ERPs] and magnetoencephalographic [MEG] studies), direct activation and recording from cortex, and transcortical cervical stimulation. One major advantage of approaching aphasia in terms of deficits in language-processing operations is that it allows the study of aphasia to make contact with this increasingly rich literature in a way that studying the classical syndromes cannot.

By identifying the neural localization of language processing operations, researchers could explain the variability in the relationship of lesion sites to the classical syndromes. If each language processor is localized in a different region of the brain, variability in the lesion sites associated with the classical syndromes may be related to the variability of deficits found across patients within the same syndrome, which we discussed above. However, there is very good evidence that the picture is far more complex. First, many modern studies have identified areas of the brain outside the traditional language area in the perisylvian association cortex as being important to language (e.g., the inferior temporal lobe; see Damasio et al., 1996). This requires some revision of the classical models, though perhaps not revisions that are beyond the spirit of those models. More important is the fact that many studies point to some degree of individual variability in the localization of specific language processing components (see Caplan, 1987; Howard, 1995, for discussion). A psycholinguistically based theory cannot explain such variability any better than the theory on which the classical syndromes are based can, but it differs from the classical theory in that it doesn't have to.

The classical model ties the relationship of language-processing components to sensory and motor areas of the brain, via the functional relationship of language functions to sensory and motor processes. There is no obvious reason that these relationships should vary across individuals. In contrast, in the psycholinguistic approach, language is seen as an abstract code, and language processing as a correspondingly abstract set of operations, and is not placed in the Procrustean bed in which the marriage between language processors and sensory–motor functions is consummated in the classical theory. Variability in localization of language processors, if it exists, though still a challenge for any theory, is thus easier to accommodate in a psycholinguistic approach than in the classical framework.

The psycholinguistic approach also addresses the last problem we discussed for the syndromes—use of the description of aphasia that is generated by a diagnostic taxonomy for planning therapy. When aphasic disturbances are characterized as disorders of particular linguistic representations in the tasks of speaking and comprehension, the therapist has something to work on. This is only one step toward developing effective therapy programs. Many patients have multiple impairments, and the therapist must decide which of these are important to address in any individual patient. To plan therapy rationally, diagnosis must be coupled with knowledge about the natural history of impairments and their responses to therapy, as it is very likely that some impairments improve on their own more than others or are particularly resistant to therapy, at least in certain cases. The clinician must decide when to abandon a direct effort to ameliorate a disorder in favor of trying to bypass it with other functions, such as in the use of augmentative communication systems. There are a variety of ways to understand and modify aspects of most aphasia syndromes; the psychological terms does, however, start the process of planning therapeutic interventions.

The newly developing psycholinguistic approach is far from perfect. It does not attempt to deal with the issues that were raised by Jackson, Head, Goldstein, and others regarding the mechanisms that regulate language use. It has not paid much attention to many aspects of aphasia that are not immediately related to psycholinguistic processing, such as a patient's fluency. For instance, most patients with agrammatism, a speech output disorder that affects function words and morphology, speak slowly, while patients whose disturbance affects the choice and ordering of phonemes in content words speak at normal rates. Why does one deficit slow down the speech planning process and the other does not? Is this because of the relationship of the lesions to these disorders to the motor system, or because there is a true functional relationship between the speed of language planning and the level of the language code being planned? Do difficulties in planning function words, morphology, and syntactic structures slow down the speech-planning process, while difficulties in planning the phonemic content words do not? The psycholinguistic approach has not addressed questions such as this.

The psycholinguistic approach has also not fulfilled its promise to add clear information to our knowledge of the relationship of language processors to the brain through deficit–lesion correlations. For instance, there is a raging controversy over the localization of the language processors involved in syntactic comprehension (Grodzinsky, 2000). Progress has been slow in this area, in part because of the difficulty in obtaining adequately detailed measurements of both language and lesions in a large enough set of patients, and in part because of different results in activation studies that may reflect differences in tasks, populations, imaging techniques and methods of analysis across studies.

Finally, the psycholinguistic approach has not yet spawned many diagnostic tests utilized by a large number of clinicians, at least not in the U.S. But major progress is being made in research studies of language impairments. But some bench-to-bedside transfer of concepts and methods has occurred, and more of this is likely to take place in the coming years.

**CONCLUSION**

Syndromes are part and parcel of medicine, and it is only to be expected that the concept of a syndrome would be applied to the study of aphasia by its founders, all of whom were medical men. It is also natural to assume that the neural tissue responsible for speech planning and comprehension would be close to that involved in motor planning and auditory perception, respectively. Finally, it is understandable that an approach to language disorders would have developed that centered on the usual tasks to which language is put and that incorporated descriptions of language features that are obvious "to the naked eye," so to speak—words, their meanings, their sounds, grammatical words and endings, and sentences. Syndromes that incorporated these features have helped clinicians and researchers
to describe and classify aphasia for over a century. Was this effort successful? Let us go back to what we said a successful syndrome does: It relates symptoms functionally, explains individual variability in symptom occurrence, excludes symptoms that co-occur with those in the syndrome that are not functionally related to those in the syndrome, and predicts natural history.

The classical syndromes had partial success in the first and last of these areas. They relate some syndromes functionally and have some predictive value. However, the approach fails in the areas of explaining individual variability and excluding co-occurring symptoms. Even in the areas in which it has been successful, its success is at best partial: it only deals with some aspects of language, and then quite generally. As researchers with backgrounds in linguistics and psychology entered the field of aphasia in the 1960s, concepts about language structure and processing have had greater impact on the way in which we think about language disorders, the relationship of language to the brain, and the approaches we might take to therapy. This approach has yielded a fair literature, but it is still truly in its infancy. It can be seen as a challenge to the classical syndromes, or as an extension of them. Does it do better than the classical approach to syndromes according to our criteria? Certainly, in the areas of relating symptoms functionally and excluding co-occurring symptoms that are not part of a syndrome, the psycholinguistic approach has proven more successful. It offers promise in the areas of dealing with individual variability and predicting natural history in more detail than the classical approach. But work on the aphasia is far from over.

The current scene is a vibrant one, in which hypotheses about language itself, its processing, its neural basis, and its disorders are all being investigated and debated vigorously. Unlike 20 or so years ago, very little is agreed upon today by experts in most of these areas. The truly encouraging feature of current research is that the combination of the conceptual framework within which aphasia is approached with the behavioral and imaging investigative techniques now available offers unparalleled opportunities to make progress in this area.

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REFERENCES
Phonologic Aspects of Language Disorders

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Phonology is the subfield of linguistics concerned with the structure and systematic patterning of sounds in language (Altmann et al., 1984). This chapter will focus on studies of acquired language disorders and on slip-of-the-tongue data (spontaneous) in normal subjects, with reference to the experimental psychology literature where relevant.

The chapter begins with a review of disorders of phonological processing. Following this, a theoretical model employing the conceptual framework of parallel distributed processing (PDP) is introduced as a means of both explaining empirical observations in a cogent fashion and of potentially relating behavior to neural microstructure. The chapter concludes with a consideration of the anatomy of phonological processing. See Nadeau (2001) for more detailed review.

DISORDERS OF PHONOLOGICAL PROCESSING

PHONOLOGICAL SELECTION ERRORS

Phonological selection errors result in the production of incorrect phonemic sequences, easily recognizable when they constitute neologisms. Such errors provide the best evidence that the brain does implicitly recognize phonemes as operational units, and that this sublexical knowledge is accessible from the neural representation of concepts and meaning. Several major types of single phoneme errors may be observed (Blumstein, 1975a).

Non-environmental:
- Substitution: /tita/"teams"  /tita/
- Simplification: /plit/"pretty"  /plit/
- Addition: /papapa/"papa"  /papapa/

Environmental:
- Asimulation within a word: "Crete"  /trit/
- Asimulation across word boundaries: "roast beef"  /rod guft/

Metathesis (exchange): "degrees"  /gedreits/

Environmental (sequential, contextual) errors may account for over 70% of errors in slip-of-the-tongue corpora and for 50%–70% of phonological errors in the language of patients with jargon aphasia (Lecours and Lhermitte, 1969; Schwartz et al., 1984). Substitution errors are more common than other non-environmental errors, and they tend to be relatively more common in aphasic language than in slip-of-the-tongue corpora (Poncet et al., 1972; Halpern et al., 1976; Niemi and Koivuselkä-