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Disorders of Language: The Aphasias

Language is particularly interesting from a neurobiological point of view because its specific and localized organization has given us the keenest insight into the functional architecture of the dominant hemisphere of the brain. The study of language also represents a striking example of how neurobiology, in collaboration with disciplines ranging from anthropology and linguistics to developmental and clinical neurology, might help us understand even the most complex of human behaviors.

Language Is Distinctive from Other Forms of Communication

Language is distinguished from other kinds of human communication by its creativity, form, content, and use.

Creativity. Just as vision is not simply an assembly of sensations but the outcome of a transformational or creative processing of physical stimuli by the brain, so is speech creative and transformational. We do not learn a language by repeating memorized stock sentences, but by understanding the rules for creating meaningful utterances. With every new thought we speak we create original sentences. Listening is also creative. We readily interpret the sentence spoken by others.

Form. Language is formed from arrangements of a limited set of sounds in predictable sequences that signal content. Each of the world's languages is based on a small fraction of the sounds humans are capable of making, and not all languages use the same set of sounds. The sounds that make up a language are called *phonemes*. These are the smallest differences in sound that distinguish different contents, for example, the difference between the sounds *d* and *t*.

Content. In natural language two further levels of structure can be distinguished: (1) the combination of phonemes to form words (morphology), and (2) the combination of words to form phrases and sentences (grammar). Unlike simple sign systems, in which meaning is tied to highly specific situations, language provides a means of shaping and communicating abstractions whose meanings are independent of the immediate situation. Language is rooted in the ability to give a single name to various appearances under different conditions. In addition, language has an emotional content that is supported by such extralinguistic means as gesture, tone of voice (flatness, whining, whispering, loudness), facial expression, and posture. Specific languages have different content structures.

Use. Language is fundamentally a means for social communication. Language is not merely a neutral medium of exchange of facts and observations about the world. Whenever we speak or write we have a social purpose. Through language we organize our sensory experience and express our thoughts, feelings, and expectations.

Certain diseases interfere more with one than with another of these features. Form can be affected by disease of the cerebellum, resulting in dysarthria (the inability to articulate words clearly), or by lesions of the cerebral cortex, resulting in Broca's aphasia. Content is disturbed in Wernicke's aphasia, in conduction aphasia, and in schizophrenia. Use is affected in the aprosodias, and in some psychiatric illnesses, such as schizophrenia.

In this chapter we shall consider the distinctive features of human language and examine why animal research has increased understanding of human language only modestly. In contrast, much has been learned about language from two sources: from the study of language acquisition in children and from neurological disorders of language. Therefore, we shall review the major findings regarding the development of language and then examine in some detail the clinical disorders of speech, reading, writing, and gesture. This family of disorders can now be understood with a model of language developed by Karl Wernicke in the nineteenth century and expanded first by Norman Geschwind, and more recently by Antonio Damasio, Michael Posner, and Marcus Raichle.

In 1984 Damasio and Geschwind summarized the progress in our understanding of the biological basis of language:

> As late as the mid 1960s, the standard view regarding cerebral dominance for language stated that [language] had no anatomical correlates, that it did not exist in other species, and that its evolution in humans could not be studied. . . . But the discoveries of the past 15 years have proven that each of these standard views was false and have opened up entirely new avenues of study.

It is these avenues that we shall pursue here.

Animal Models of Human Language Have Been Largely Unsatisfactory

Approaches to a neural analysis of cognitive and other behavioral functions have often depended on animal models. Considerable effort therefore has been expended in developing animal models of language. Animals as simple as crickets and bees have an elementary form of communication, a sort of natural language. The song of birds is even more elaborate (see Chapter 61). Nevertheless, these forms of communication cannot be considered interpersonal—they are at best inter*individual*—and their form, content, use, and creativity are highly stereotyped.

What about our closest relatives, the nonhuman primates? Do they have creative language? Can they be used to study human speech? In the past few decades opinion on this question has swung back and forth several times. In the 1930s it was generally thought that chimpanzees could learn to speak if they were raised in a human home as human children are. With this idea in mind, William and Lorna Kellogg raised a chimpanzee, Gua, with their own child. The chimpanzee adopted many human behaviors, understood a few spoken commands, and mastered a

few hand gestures, but never learned to speak. By the early 1960s chimpanzees were thought to lack the intellectual capacity for language. Noam Chomsky, a linguist, wrote in 1968: "Anyone concerned with the study of human nature and human capacity must somehow come to grips with the fact that all normal humans acquire spoken language whereas acquisition of even the barest rudiments is quite beyond the capacity of an otherwise intelligent ape."

Shortly thereafter it was discovered that the vocal apparatus of chimpanzees is unable to produce the full range of human sounds. The possibility remained, however, that chimpanzees might show a capacity for language if they did not have to produce speech sounds. Allen and Beatrice Gardner circumvented the need for sound production by training a female chimpanzee named Washoe to use signs borrowed from American Sign Language, the language of the American deaf. Within four years Washoe achieved a vocabulary of 160 words, including signs for objects (bird, hand), attributes (blue, green, different), and modifiers (more, less). Although these results demonstrate that chimpanzees can learn words and use symbols, the vocabulary they acquire is much smaller than that of a human infant. A child of four has a vocabulary of more than 3000 words, as compared with Washoe's 160.

To explore whether chimpanzees understand relationships, David Premack trained a chimpanzee, Sarah, to communicate with plastic chips that had different signs inscribed on them. In this training Premack tried to preserve many features that are universal in natural languages. He taught Sarah to interpret commands contained in an arrangement of the signs on different chips and to construct her own sentences. Sarah eventually learned the concepts of negation, similarity, and difference, the expression "is the name of," compound sentences, *if–then* statements, and how to ask questions. Most interesting were experiments in which Premack showed Sarah pairs of objects in which the second was a transformed version of the first (an apple and an apple cut into pieces; a dry towel and a wet towel). Sarah was then asked to select one of several other objects that would explain the correlation (for example, a knife and a bowl of water) and insert it between these pairs. She made the appropriate choice about 80% of the time. Sarah appeared to be able to express in symbols her understanding of the causal relationship between physical events.

Thus, chimpanzees (and probably gorillas as well) are able to communicate through symbols in a rudimentary fashion. It is not certain, however, if they can go beyond that. For example, there is no evidence as yet that chimpanzees can understand syntax, the rules that organize words into sentences, so that they can creatively recombine words and express different ideas with them. Thus, Washoe can use the words *Washoe*, *me*, and *banana*, but most students of language think that she cannot distinguish "me give Washoe banana" from "Washoe give me banana." Indeed, most linguists are struck by the *noncreative*, imitative, and mechanical nature of the language acquired by chimpanzees.

Although the analogy between the use of human language forms by chimpanzees and the fluent and creative language of humans seems weak, this work does show that apes (and even much simpler animals, as we now have good reason to believe) share with humans certain cognitive capabilities such as knowledge of causality. Whether these capabilities are crucial to linguistic competence, however, remains unclear. It is hard to know, for example, whether animals do not express propositions because their communication abilities are insufficient or because they do not think in this way.

Because animal models have proven to be of limited usefulness in the study of human language, students of language rely primarily on anthropological, developmental, and clinical studies.

What Is the Origin of Human Language?

Although it is difficult to pinpoint the time or way in which language evolved, some cerebral structures that are prerequisite for language appear to have arisen early in human evolution. This conclusion has come from the work of Marjorie LeMay, who examined endocranial casts of human fossils. In most individuals the left hemisphere is dominant for language and the cortical speech area of the temporal lobe (the planum temporale) is larger in the left than in the right hemisphere. Since important gyri and sulci often leave an impression upon the skull, LeMay searched the fossil record for the morphological asymmetries associated with speech in modern humans and found them in Neanderthal man (dating back 30,000 to 50,000 years) and in Peking man (dating back 300,000 to 500,000 years). The left hemisphere is also dominant for the recognition of species-specific cries in Japanese macaque monkeys, and asymmetries similar to those of humans are present in brains of modern-day great apes, such as the chimpanzee. Whether these anatomical and functional asymmetries originally evolved for language, for other forms of communication, or for an entirely different function, is not known.

Although the anatomical structures that are prerequisites for language may have arisen early (perhaps as many as 500,000 years ago), many linguists believe that language *per se* emerged rather late in the prehistoric period of human existence (about 100,000 years ago) and that perhaps it arose only *once*. According to this view, all human languages are thought to have arisen together with primate evolution from a single language first spoken in Africa.

Did human language evolve from ape-like communication? Since human evolution is itself not understood, and since apes, as we have seen, have only rudimentary language capabilities, these questions are speculative. Two hypotheses about the origin of language have been advanced: gestural and vocal.

Gestural theories propose that language evolved from a system of gestures that emerged when certain apes assumed an erect posture, freeing the hands for social com-

munication. Subsequently, vocal communication may have arisen to free the hands for purposes other than communication. *Vocal theories* contend that language evolved from an extensive group of instinctive calls that were expressive of emotional states, such as distress, elation, and sexual arousal. About 100,000 years ago changes in the structure of the mouth, jaw, and vocal tract made it possible to control the production of different sounds reliably and consciously. As a result, sounds could at least in principle be used creatively in different combinations. When these ancestors of modern humans dispersed into separate colonies, geographical isolation allowed for the development of different sound systems. The possibility that language emerged *once* in history might explain why all human languages have so many features in common.

Alternatively, language may have emerged from the coevolution of gesture and vocalization. This possibility might account for the still inexplicable correlation of verbal language and hand dominance (gesture), both localized to the left hemisphere.

Is the Capability for Language an Innate Skill or Learned?

Although the acquisition of language clearly involves learning, studies of the anatomical localization of language and of language development in children suggest that a large part of the process is innate. First, as we saw in Chapter 53, both natural and sign language functions are localized; language is predominantly represented in the left hemisphere.

Second, the localization of language in the left hemisphere seems to be related to anatomical differences between the two hemispheres. For example, the planum temporale, the area of the temporal lobe specialized for speech, is larger in the left hemisphere in most right-handed people (Chapter 53). Third, this anatomical asymmetry in the planum temporale is present early in development (by the thirty-first week of gestation), suggesting that this asymmetry does not develop in response to experience but is innate.

Fourth, infants at birth are sensitive to distinctions in a broad range of sounds, an ability that is crucial for the comprehension of any human language. Indeed, some of this sensitivity is lost later, when a specific language is acquired. For example, most adult Japanese cannot perceive the difference between the sounds of *r* and *l*. Japanese infants can distinguish these sounds, however, and only lose this ability when they mature. Peter Eimas has suggested that the neural basis of this decline in perceptual discrimination is similar to that underlying the loss of visual acuity in kittens raised in a restricted visual environment (see Chapter 60).

Finally, there are universal regularities in the acquisition of language. Children progress from babbling to one-word speech, to two-word speech with syntax, to complex speech (Table 54–1). Some children progress through these stages faster than others, but the average age for each stage is the same in all cultures. Moreover, language capacity (as measured by the ability to acquire a new language) is reduced dramatically after puberty. These several findings suggest that there is a critical period during development when language, whether verbal or signed, is acquired effortlessly. Presumably, this period of development corresponds to the maturation of the human brain, although studies have not yet attempted to correlate language acquisition with maturation of specific areas related to language. During this period children learn the rules of language by simply listening to the speech around them. These rules, the grammar of the language, are clearly understood by the time the child begins to form sentences. Although a specific language must be learned through experience, Noam Chomsky argues that humans have some innate program that prepares them to learn language in general. According to Chomsky, an infant learns a language by testing the specifics of the language heard daily against a genetically determined system of rules or

TABLE 54–1. Stages of Development in the Acquisition of Language

Average age	Language ability
6 months	Beginning of distinct babbling.
1 year	Beginning of language understanding; one-word utterances.
1½ years	Words used singly; child uses 30–50 words (simple nouns, adjectives, and action words) one at a time but cannot link them to make phrases; does not use functors (the, and, can, be) necessary for syntax.
2 years	Two-word (telegraphic) speaker; 50 to several hundred words in the vocabulary; much use of two-word phrases that are ordered according to syntactic rules; child understands propositional rules.
2½ years	Three or more words in many combinations; functors begin to appear; many grammatical errors and idiosyncratic expressions; good understanding of language.
3 years	Full sentences; few errors; vocabulary of around 1000 words.
4 years	Close to adult speech competence.

(Based on E. H. Lenneberg, 1967.)

generative grammar. These rules reflect innately determined neural mechanisms that limit the possible characteristics of a natural language. That is, children have an innate ability to recognize the *universals* that characterize a natural language in the environment. When exposed to a language with these universals, a child learns it avidly. Chomsky argues that a language that violated these universals would be unlearnable.

In summary, linguists and psychologists now believe that the mechanisms for the universal aspects of language acquisition are determined by the structure of the human brain. According to this view, the human brain is prepared as a result of development to learn and use speech. The particular language spoken and the dialect and accent are determined by the social environment.

The question now being debated by linguists is whether linguistic universals derive from the neural structures specifically related to language acquisition or from cognitive universals that are more general. Chomsky argues that there are neural constraints specific to language acquisition, but many psychologists disagree. Children are able to understand abstract rules before they learn to speak. They can, for example, distinguish between causative and noncausative actions.

The challenge for the neurobiological approach to cognition and language is to address these problems. One avenue of investigation has come from the study of aphasia. Researchers working with aphasic patients are asking two sorts of questions. First, are disorders of language isolated cognitive disorders, or are they related to more general disturbances of cognitive processes? Second, what are the neural structures that underlie the innate universal rules of grammar?

Aphasias Are Disorders of Language that Also Interfere with Other Cognitive Functions

The aphasias are disturbances of language caused by insult (vascular damage, trauma, or tumor) to specific regions of the brain—usually, but not invariably, to regions of the cerebral cortex. Damage of the cerebral cortex does not result in an overall reduction in language ability; rather, lesions in different parts of the cerebral cortex cause selective disturbances. Furthermore, these disorders involve more than a breakdown in the production and comprehension of spoken language: The damage to the brain often affects other cognitive and intellectual skills to some degree. For example, as we shall see later, some aphasic patients have difficulty comprehending both speech and writing (Wernicke's aphasia). Others have difficulty expressing thoughts in either written or spoken language (Broca's aphasia).

Such selective disruptions of cortical function afford unusual insight into how the brain is organized for language. One of the most impressive insights has been provided by Ursula Bellugi and her colleagues in their study of sign language. Unlike speech, signing is expressed by hand gestures rather than by sounds, and is perceived by visual rather than auditory pathways. Nonetheless, signing, which has the same structural complexities characteristic of spoken languages, is also localized to the left hemisphere. Thus, following lesions in the left hemisphere, deaf individuals become aphasic for sign language. Lesions in the right hemisphere do not produce these defects in signing. Moreover, defects in signing following left hemisphere damage can be quite specific, involving either sign comprehension and grammar or signing fluency.

This illustrates three points. First, the left hemisphere contains the cognitive capability for language and this capacity is independent of the sensory or motor modalities used for processing language. Second, speech and hearing are not a prerequisite for the emergence of language capabilities in the left hemisphere. Third, spoken language represents only one of a family of cognitive skills mediated by the left hemisphere.

The aphasias are distinguished from other disorders of speech, such as *dysarthria*, a disturbance in articulation, and *dysphonia*, a disturbance in vocalization. These disorders result from weakness or incoordination of the muscles controlling the vocal apparatus and are simply disorders of the mechanical process of speech. They do not basically affect language comprehension or the central processes of expression. Patients with cerebellar disorders who are dysarthric, or those with Parkinson's disease who are dysphonic, retain their language ability despite severe speech impairment. In contrast, the hallmark of aphasia is a disturbance in language ability, either in comprehension or production, or both, that is not attributable to a mechanical impediment.

The most common cause of aphasia is head trauma, which produces 200,000 cases in the United States each year. The next most frequent cause is stroke: 40% of major vascular events in the cerebral hemispheres produce language disorders. In the United States stroke leads to 100,000 cases of aphasia each year. Studies of patients with discrete vascular lesions have increased our understanding of aphasia because these lesions do not progress and the anatomy of the damaged region often directly relates to the distribution of critical blood vessels.

The Wernicke–Geschwind Model for Language Is a Useful Clinical Model for Distinguishing Damage to the Two Major Language Regions of the Brain

There is no universally accepted classification for the aphasias. A useful classification was developed by Geschwind and Damasio as an elaboration of the Wernicke–Geschwind model of language and gesture, and we shall use that scheme here (Figure 54–1).

This model for language can best be illustrated by considering the simple task of repeating a word that has been heard. According to the original Wernicke–Geschwind

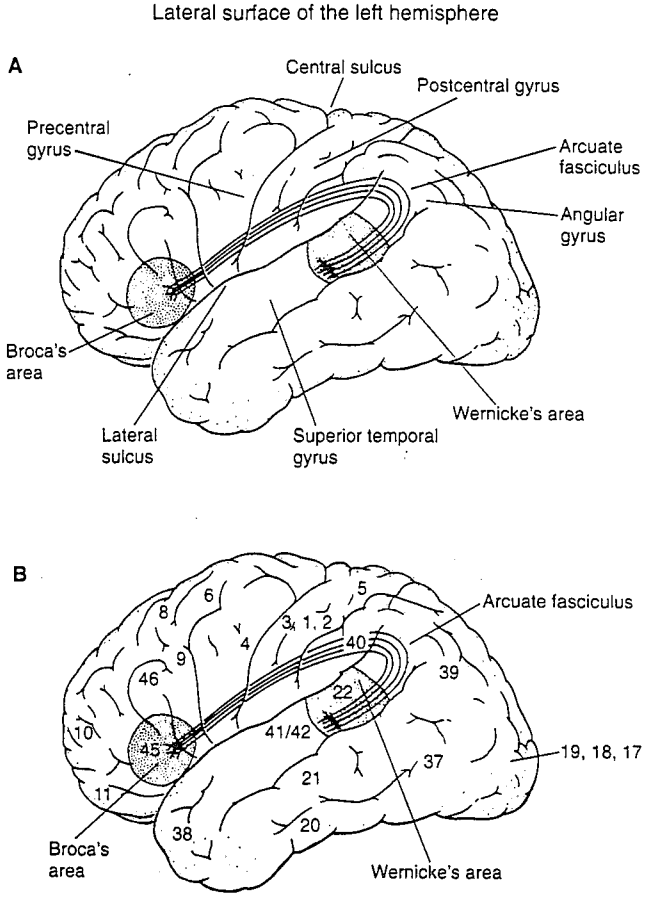

FIGURE 54–1
Primary language areas of the brain.

A. The classical nomenclature of gyri and sulci are indicated in this lateral view of the exterior surface of the left hemisphere. Broca's area, the motor-speech area, is adjacent to the region of the motor cortex (precentral gyrus) that controls the movements of facial expression, articulation, and phonation. Wernicke's area lies in the posterior superior temporal lobe near the primary auditory cortex (superior temporal gyrus) and includes the auditory comprehension center. Wernicke's and Broca's areas are joined by a fiber tract called the *arcuate fasciculus*. In the figure Broca's and Wernicke's areas are referred to as *regions* to indicate their status as part of complex networks rather than independent language *centers*.

B. The cytoarchitectonic areas (Brodmann's classification) are illustrated in this lateral view of the left hemisphere. Area 4 is the primary motor cortex; area 41 is the primary auditory cortex; area 22 is Wernicke's region; and area 45 is Broca's region.

model, this task involves transfer of information from the basilar membrane of the auditory apparatus to the auditory nerve and medial geniculate nucleus. The information then flows first to the primary auditory cortex (Brodmann's area 41), then to the higher-order auditory cortex (Brodmann's area 42), before it is conveyed to a specific region of the parietal–temporal–occipital association cortex, the *angular gyrus* (Brodmann's area 39), which is thought to be concerned with the association of incoming auditory, visual, and tactile information. From here the information is projected to Wernicke's area and then, by means of the arcuate fasciculus, to Broca's area, where the perception of language is translated into the grammatical structure of a phrase and where the memory for word articulation is stored. This information about the sound pattern of the phrase is then conveyed to the facial area of the motor cortex that controls articulation so that the word can be spoken.

A similar pathway was thought by Wernicke and Geschwind to be involved in naming an object that has been visually recognized (Figure 54–2). According to their model visual information is transferred from the retina to the lateral geniculate nucleus, and from there to the primary visual cortex (Brodmann's area 17). The information then travels to a higher-order center (area 18), where it is conveyed first to the angular gyrus of the parietal–temporal–occipital association cortex, and then to Wernicke's area, where the visual information is transformed into a phonetic (auditory) representation of the word. The phonetic pattern is formed and then conveyed to Broca's area by means of the arcuate fasciculus.

The original Wernicke–Geschwind model made several interesting predictions that are useful clinically. First, it predicted the outcome of a lesion in Wernicke's area. Words reaching the auditory cortex fail to activate Wernicke's area and thus fail to be comprehended. If the lesion extends posteriorly and inferiorly beyond Wernicke's area, it will also affect the pathway concerned with the processing of visual input to language. As a result, the patient will be incapable of understanding either the spoken or the written word. Second, the model correctly predicts that a lesion in Broca's area will not affect the comprehension of written and spoken language, but will cause a major disruption of speech and verbal production because the pattern for sounds and for the structure of language are not passed on to the motor cortex. Third, the model predicts that a lesion in the arcuate fasciculus, by disconnecting Wernicke's area from Broca's, will disrupt verbal production because the auditory input is not conveyed to the part of the brain involved with production of language.

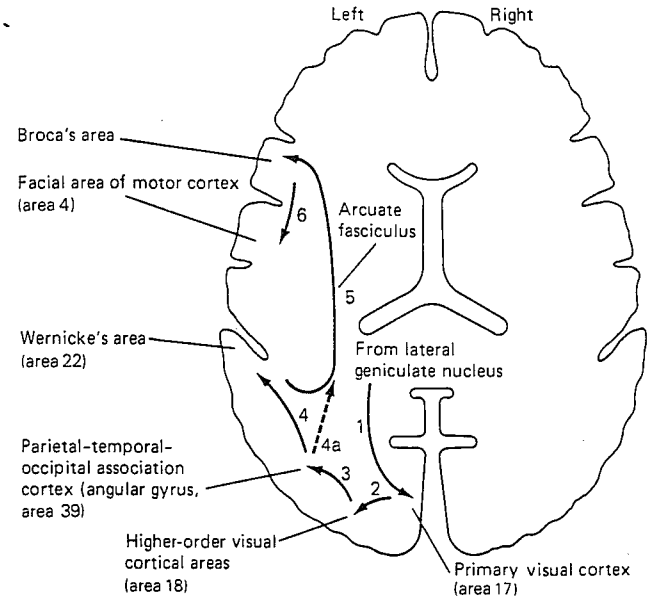

FIGURE 54–2
The neural pathways involved in naming a visual object according to the Wernicke–Geschwind model of cortical processing. The diagram here shows a schematic drawing of a horizontal section of the human brain at the level of the corpus callosum. The naming begins with input from the retina through the optic nerve. Recent evidence suggests that the actual flow of information is almost identical to the sequence shown here, except that, following step 3, a component of the arcuate fasciculus (4a) conveys information directly from the association cortex to Broca's area, bypassing Wernicke's area. (Adapted from Patton, Sundsten, Crill, and Swanson, 1976.)

Recent Cognitive and Imaging Studies of Normal Subjects and Aphasic Patients Have Clarified the Interconnections of the Two Language Regions

Even though the modified Wernicke–Geschwind model continues to be useful clinically, recent cognitive and imaging studies comparing the uses of language by normal and aphasic patients by Damasio, Raichle, Posner, and their colleagues indicate that the Wernicke–Geschwind model may be oversimplified in several ways. First, the emphasis in the Wernicke–Geschwind model on the importance of Broca's and Wernicke's areas for expression and reception was based on lesions that actually affected much larger regions. When lesions are restricted to the areas originally identified by Broca and Wernicke, they usually do not give rise to the full symptoms characteristic of Wernicke's or Broca's aphasia. The typical symptoms are usually the result of damage to the surrounding regions as well.

Second, the Wernicke–Geschwind model emphasizes the importance of cortical regions (and interconnecting pathways running through subcortical white matter). There now is evidence that subcortical structures, specifically the left thalamus, the left caudate nucleus, and adjacent white matter, also are important for language. For example, lesions in the left caudate lead to a defect in auditory comprehension presumably by interrupting the auditory–motor integration required for linguistic processing.

Third, as we saw in Chapter 1, an auditory input—a spoken word—is indeed projected from the auditory cortex to the angular gyrus and then to Wernicke's area before being conveyed to Broca's area (Figure 54–3). However, visual information, such as a written word, is not conveyed to Wernicke's areas, but goes from the visual association cortex directly to Broca's area. Words that are read are therefore *not* transformed into an auditory representation. Rather, visual and auditory perceptions of a word are processed independently by modality-specific pathways that have independent access to Broca's area and to the higher-order regions concerned with the meaning and expression of language.

Finally, cognitive studies of language disagree with the Wernicke–Geschwind model on more than the pathway for processing auditory information. For example, there is good evidence that not all auditory input is processed in the same way. Nonsense sounds—words without meaning—are processed independently from conventional, meaningful words. Thus, there are thought to be separate pathways for *sounds*, the *phonological* aspects of language, and for *meaning*, the semantic aspects of language. Similarly, although Broca's area is the common output for both spoken and written words that have meaning, there may be an independent output for nonsense words. Finally, a number of studies by psycholinguists indicate that patients with both Broca's and Wernicke's aphasia not only have language deficits but also have deficits in one or another aspect of cognitive processing. These difficulties muddy the simple distinction between impairment of reception or expression. In actuality, therefore, language deficits are never as pure as the Wernicke–Geschwind model would predict.

These and related findings indicate that language involves a larger number of areas and a more complex set of interconnections than just the serial interconnection of Wernicke's area to Broca's area. A more realistic scheme illustrating the neural processing of language is shown in Figure 54–3.

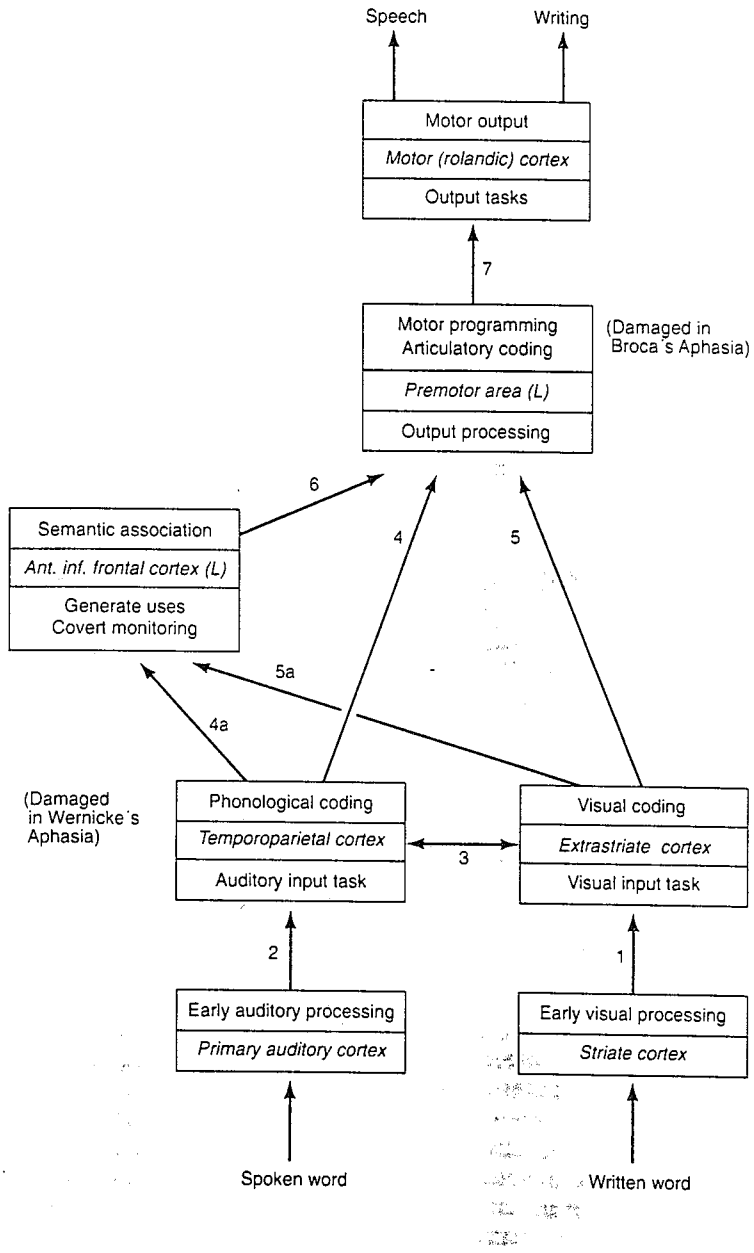

FIGURE 54–3
Recent models of the neural processing of language are more complex than the Wernicke-Geschwind model but nonetheless are built on its basic ideas. The particular model illustrated in this figure represents a fairly simple circuit and shows the relationship between various anatomical structures and functional components of language. Other networks are plausible. At each point in the circuit the anatomical structure is indicated in italics. The function of the structure is shown below the structure name and the specific language skill is shown above the name. Both visual and auditory inputs as well as spoken and written expression are illustrated. (From Petersen et al., 1988.)

Seven Types of Aphasia Can Be Distinguished and Related to Different Anatomical Systems

We now turn to the major clinical syndromes of aphasia. In practice, the symptoms of a patient may not always fall simply into one category or another because lesions producing cortical damage are not always coextensive with a functional site (Table 54–2).

Wernicke's Aphasia

Wernicke's aphasia is characterized by a prominent deficit in comprehension. The lesion primarily affects Wernicke's area—the left posterior portion of the temporal lobe, or Brodmann's area 22—although it often extends to the superior portions of the temporal lobe (areas 40 and 39) and inferiorly to area 37. When the lesion is extensive, comprehension of both visual and auditory language input is severely impaired. In contrast, speech is fluent. Language is normal in rate, rhythm, and melody, although patients may use the wrong word or combination of words (*paraphasia*). These patients tend to add additional syllables to words and additional words to phrases. They may make up new words, called *neologisms*. The neologistic or paraphasic distortions most frequently involve key lexical items (nouns, verbs, adjectives, adverbs), especially nouns.

Language may be excessive (*logorrhea*); this phenomenon has been called *press of speech*. Because of the abundance of words, their speech often conveys little meaning,

TABLE 54–2. Clinical Characteristics of Cortical Aphasias

Type	Verbal output	Repetition	Comprehension	Naming	Associated signs	Lesions
Broca's	Nonfluent	Impaired	Normal	Marginally impaired	RHP and RHH apraxia of the left limbs and face	Left posterior inferior frontal
Wernicke's	Fluent	Impaired	Impaired	Impaired		Left posterior superior temporal
Conduction	Fluent	Impaired	Normal	Impaired (paraphasic)	± RHS, apraxia of all limbs and face	Left parietal
Global	Nonfluent	Impaired	Impaired	Impaired	RHP, RHS, RHH	Left frontal temporal parietal
Anomic	Fluent	Normal (anomic)	Normal	Impaired	None	Left posterior inferior temporal, or temporal-occipital region
Transcortical motor	Nonfluent	Normal	Normal	Impaired	RHP	Left medial frontal or anterior border zone
Sensory	Fluent	Normal	Impaired	Impaired	± RHH	Left medial parietal or posterior border zone
Mixed	Nonfluent	Normal	Impaired	Impaired	RHP, RHS	Left medial frontal parietal or complete border zone

RHP, right hemiparesis; RHH, right homonomous hemianopsis; RHS, right hemisensory defect.

however. For example, when asked where he lived, a patient with Wernicke's aphasia replied, "I came there before here and returned there." Patients with Wernicke's aphasia fail to convey the ideas they have in mind, an impairment called *empty speech*. They generally are unaware of this failure, probably because language comprehension is impaired. The ability to repeat words and phrases is also impaired because comprehension is severely disturbed. In addition, patients with Wernicke's aphasia have severe reading and writing disabilities. Except for these symptoms of aphasia, other neurological signs may be absent, but occasionally a right visual field defect is encountered.

Broca's Aphasia

In Broca's aphasia comprehension is usually preserved, at least in part, but language production is not fluent. Patients have damage to the motor association cortex in the frontal lobe, usually extending to the posterior portion of the third frontal gyrus (Brodmann's areas 44 and 45) which forms part of the frontal operculum (Broca's area). In severe cases there is also damage to the surrounding premotor and prefrontal regions (areas 6, 8, 9, 10, and 46). The deficit in language production ranges from almost complete muteness to a slowed, deliberate speech constructed from very simple grammatical structures. Patients with Broca's aphasia use only key words. They usually express nouns in the singular, verbs in the infinitive or participle, and often eliminate articles, adjectives, and adverbs altogether. For example, instead of saying "the large gray cat," a patient with Broca's aphasia may say "gray cat."

These omissions are even more dramatic in more complex sentences. Here we can see the second characteristic of this defect: a breakdown in the construction and coordination of several constituent phrases within a sentence. Consider the sentence: "The ladies and gentlemen are now all invited into the dining room." A patient with Broca's aphasia may only be able to say "Ladies, men, room." When asked his occupation, a mailman with Broca's aphasia said "Mail . . . Mail . . . M . . . ." In addition to such telegraphic or nongrammatical speech, repetition is always impaired, and naming ability may be slightly to moderately impaired. Unlike Wernicke's aphasia, patients with Broca's aphasia are generally aware of these errors.

Although production of language is severely disturbed, comprehension of both spoken and written language is less disturbed, because Wernicke's area is not damaged. However, patients with Broca's aphasia have difficulty reading aloud, and writing (like speech) is abnormal. Work by Rita Berndt and Alfonso Caramazza suggests that Broca's aphasics may also have some difficulty comprehending those aspects of syntax that they have difficulty producing.

Because Broca's area is located near the motor cortex and the underlying internal capsule, a right hemiparesis and homonymous hemianopsia (loss of vision) is almost always present in this type of aphasia.

Conduction Aphasia

As pointed out in Chapter 1, conduction aphasia was predicted by Wernicke. He proposed that an area in the temporal lobe, concerned with the comprehension of language, projected to Broca's area by means of a pathway that connected the two regions. He therefore inferred that a lesion could leave both Broca's and Wernicke's areas intact but disconnect the two. Clinical studies verified

this prediction. Lesions in the arcuate fasciculus, which runs in the white matter and connects Wernicke's and Broca's areas, lead to a conduction aphasia. Damage to the fasciculus occurs with injury of the supramarginal gyrus of the parietal lobe, or, less frequently, injury of the posterior and superior aspect of the left temporal lobe (Figure 54–1). Thus, the lesion is not restricted to white matter but also involves the cortex.

Like patients with Wernicke's aphasia, patients with conduction aphasia are fluent but have many paraphasic errors, errors in which incorrect words or sounds are substituted for correct ones. The degree of fluency may be somewhat less than that seen in Wernicke's aphasia, but comprehension is good. However, damage to the pathways from Wernicke's area to Broca's area greatly impairs the ability to repeat. Other characteristics of conduction aphasia are also consistent with a functional separation of Broca's and Wernicke's areas. Naming is severely impaired. Reading aloud is abnormal, but patients can read silently with good comprehension. Writing may also be disturbed; spelling is poor, with omissions, reversals, and even substitutions of letters.

Many patients with conduction aphasia have some degree of impairment of voluntary movement.

Anomic Aphasia

In anomic aphasia the only disturbance is a difficulty in finding the correct words. This is an unusual form of aphasia that typically follows lesions in the posterior aspect of the left inferior temporal lobe, near the temporal–occipital border. Occasionally, patients with anomic aphasia also have a defect in the right superior quadrant visual field.

Global Aphasia

Patients with global aphasia are unable to speak or comprehend language; they cannot read, write, repeat, or name objects. Lesions that cause global or total aphasia usually include the entire perisylvian region, thereby compromising both Broca's and Wernicke's areas and the arcuate fasciculus. Symptoms also include a complete right hemiplegia, right hemisensory defect, and usually a right homonymous hemianopsia.

Transcortical Aphasias

Transcortical aphasias have two important characteristics: (1) the patients have the ability to repeat spoken language, and (2) their lesion lies outside the perisylvian language centers. These aphasias most often result from vascular damage at the junction between the middle, anterior, and posterior cerebral arteries, a region known as the *border zone* or *watershed area*. This border zone includes association areas that are important for memory of the meaning of words and the supplementary motor cortex, which is important for skilled motor acts.

Transcortical motor aphasia results from a lesion that disconnects Broca's area from the supplementary motor cortex. The lesion is usually in the frontal lobe anterior to Broca's area. The lesion gives rise to a nonfluent aphasia in which the patient cannot produce creative speech. The patient will attempt conversation but can utter only a few syllables. In striking contrast, these patients are able to repeat words and phrases well. Comprehension of language is less disturbed, as is reading (both silently and aloud), but writing may be impaired seriously.

Transcortical sensory aphasia follows disconnection of Wernicke's area from the posterior parietal temporal association area. This gives rise to a fluent aphasia with defective comprehension, to a defect in thinking about or remembering the meaning of signs or words. The patient cannot read or write and has marked difficulty in finding words, but is able to repeat spoken language easily and fluently. This type of aphasia usually results from a lesion in the parietal–temporal–occipital junction.

A combination of transcortical motor and transcortical sensory aphasias produces *mixed transcortical aphasia* or *isolation of the speech area*. This is an extremely rare disorder. The patient is unable to speak unless spoken to, and responses are usually a direct echo of the examiner's words, a behavior called *echolalia*. The patient is not competent in any other language function.

Subcortical Aphasia

We have so far considered some aphasia due to *cortical* damage. Lesions that do not affect the cerebral cortex, typically vascular lesions in the basal ganglia and thalamus, can also result in aphasia.

Lesions in the left caudate nucleus or putamen cause a fluent aphasia with neologistic language. The language deficit is characteristically transient, however. Lesions in the thalamus can produce an aphasia that is often similar to that observed in the transcortical aphasias. The most frequent signs are a combination of paraphasia, poor comprehension of spoken language, and an intact ability to repeat. These disorders are typically transient; many patients fully recover.

Hypometabolism in the corresponding left temporoparietal area has been observed in patients with impaired comprehension following a subcortical aphasia. This also supports the concept that normal language is dependent not only on cortical–cortical but also on subcortical connections.

Certain Affective Components of Language Are Affected by Damage to the Right Hemisphere

We have so far considered only the cognitive components of language. Human language, and more generally human communication, has important affective components as well. These components include musical intonation (*prosody*) and emotional gesturing.

Elliott Ross found that certain affective components of language rely on specialized processes of the right hemi-

sphere. Disturbances in affective components of language associated with damage to the right hemisphere are called *aprosodia*. The organization for prosody in the right hemisphere seems to mirror the anatomical organization for the cognitive aspects of language in the left hemisphere. Thus, patients with lesions in the anterior portion of the right hemisphere have a flat tone of voice whether they are happy or sad. Patients with posterior lesions do not comprehend the affective content of other people's language.

Some Disorders of Reading and Writing Can Be Localized

Reading disorders are either congenital (called the *dyslexias*) or acquired (called *acquired dyslexias* or *alexias*). We shall first focus on the alexias because they are particularly instructive for understanding language and illustrate interesting extensions of the Wernicke–Geschwind model of language.

Alexias and Agraphias Are Acquired Disorders of Reading and Writing

Alexia (disruption of the ability to read) and agraphia (disruption of the ability to write) are quite remarkable because they demonstrate that small lesions of the brain in an adult can selectively destroy the ability to read or write, or both, without interfering with speech or other cognitive functions. This discovery was made by the French neurologist Jules Dejerine, who described word blindness in two papers published in 1891 and in 1892. In the first, Dejerine described a patient with a disorder of both reading and writing (alexia with agraphia). The second patient had a pure word blindness (alexia without agraphia).

Word Blindness Accompanied by Writing Impairment (Alexia with Agraphia). The first patient described by Dejerine could speak and understand spoken language, but had ceased to be able to read or write. Autopsy of this and later cases revealed that alexia with agraphia is usually associated with lesions of the angular or supramarginal gyrus of the parietal–temporal–occipital association cortex. As we saw in Chapter 53, this association cortex is concerned with the integration of visual, auditory, and tactile information. Once integrated, the information is conveyed to the speech areas of the temporal lobe and then to those of the frontal lobe. When the association cortex of the angular or the supramarginal gyrus is damaged, patients cannot read or write because they cannot connect visual symbols (letters) with the sounds they represent. Similarly, these patients cannot recognize words spelled out loud, nor can they spell. They also are unable to recognize embossed letters by feeling the letters because the angular and supramarginal gyri mediate the transfer of cutaneous sensory information into language areas.

Pure Word Blindness: Alexia without Agraphia. Dejerine's second patient could speak. An intelligent and highly articulate man, he suddenly observed that he could not read. The patient was able, however, to derive meaning from words spelled aloud and was able to spell correctly. Even though he could not comprehend written words, he could copy them correctly and could recognize and understand them after writing the individual letters.

The patient was blind in the right visual field (indicating damage to the left visual cortex) but otherwise had normal visual acuity. Postmortem examination of this and other patients revealed damage to the left occipital (visual) cortex and the splenium (the posterior portion of the corpus callosum), which carries visual information between the two hemispheres by interconnecting area 18 of the occipital cortex of one hemisphere with that of the other. Although the visual information from the left visual field could still be processed by the right hemisphere, damage to the splenium prevented its transfer to the angular gyrus and to language areas of the left hemisphere.

As might be predicted from the location of the lesion, many patients have selective deficits in visual perception due to damage in the visual portion of Brodmann's area 18 (see Chapter 29). For example, 50% of patients with pure alexia have either a *color agnosia* (they are capable of matching colors but cannot name them) or an *achromatopsia* (they cannot perceive color and therefore see objects only as shades of gray).

John Trescher and Frank Ford extended Dejerine's findings by noticing that surgical disruption of the splenium (the posterior portion of the corpus callosum) results in the loss of reading ability in the left but not the right visual field. In contrast, section of the anterior portion of the corpus callosum (which does not transmit visual information) does not interfere with reading. However, patients in whom the anterior portion of the corpus callosum has been transected cannot write with their left hands (controlled by the right hemisphere), because the right hemisphere no longer has access to the left hemisphere language centers. The patients also cannot name objects held in the left hand because the somatic sensory information does not reach the language areas in the left hemisphere.

Phonetic Symbols and Ideographs Are Localized to Different Regions of the Cerebral Cortex. An interesting disturbance in reading and writing occurs among the Japanese. There are two distinct systems of writing Japanese. One, *kata kana*, is phonetic: words are represented by a series of phonetic symbols (graphemes). There are 71 graphemes in the *kana* system. The other writing system, *kanji*, is in good part ideographic: root words are represented by one or more ideograms derived from Chinese. There are over 40,000 *kanji* ideograms to which are added affixes for phonemic reference. *Kana* words are comprehended syllable by syllable and, unlike Western words, are not easily identified at a glance. In contrast, the *kanji* system represents both sound and meaning; it has both phonetic and morphemic reference.

Because these two writing systems rely on phonemic processing to differing degrees, one might expect that certain focal lesions might affect reading or writing in one system but not the other. This is in fact the case. Both systems rely on language centers in the left hemisphere but each is processed by a different intrahemispheric mechanism. Lesions of the angular gyrus of the parietal–temporal–occipital association cortex severely disrupt reading of *kana* (syllabic) writing, but leave comprehension of *kanji* (ideographic) writing largely intact. Such lesions can disrupt reading of *kanji* to some degree, but the disruption entails primarily phonemic processing; patients may be unable to read the *kanji* word aloud but can accurately explain its meaning. In contrast, these patients are unable to understand the same idea expressed in *kana*.

These observations support the conclusion from brain imaging studies that the angular gyrus of the left hemisphere, concerned with auditory representation, is not involved with the processing of the visual representation of words. Other dissociations between the processing of *kana* and *kanji* scripts also occur and have provided further insight into the mechanisms of information processing in the production and comprehension of language.

Dyslexia and Hyperlexia Are Developmental Disorders of Reading

Dyslexia is an inability to read effortlessly or with understanding. Except for the reading impairment, the cognitive and intellectual capacities of these children are often normal and may even be superior. Children with dyslexia seem particularly impaired in phonemic processing—the ability to associate letters with the sounds they represent. However, they can usually understand other signs or symbols of communication, such as traffic signs or words that have a unique visual appearance (such as the Coca-Cola trademark). Indeed, Paul Rozin and his colleagues have found that American dyslexic children can easily learn to read English when entire words are represented by single characters rather than a sequence of characters. The specificity of this disorder and the parallels to alexic disorders caused by strokes have led to the suggestion that dyslexia might result from abnormalities in connections between visual and language areas.

Some dyslexic children also exhibit a strong tendency to read a word from right to left (confusing words like "was" and "saw") and have particular difficulty distinguishing between letters that have the same configuration but in different orientations (for example, p and q, or b and d). These mistakes occur in both reading and writing. These errors and the disproportionate percentage of left-handers among dyslexics led Samuel Orton to suggest that dyslexia might involve a deficit in the development of dominance by the left hemisphere. Albert Galaburda and Thomas Kemper have provided evidence supporting this hypothesis. They found that the normal hemispheric discrepancy in the size of the planum temporale was much reduced in dyslexic males. In addition, the left planum temporale exhibited striking cytoarchitectonic abnormalities, including an incomplete segregation of cell layers. In contrast, the right hemisphere appeared normal. These observations suggest that normal migration of neurons to the left cortex during development is slowed in dyslexic patients.

An Overall View

Language is a uniquely human ability. In both its written and spoken forms it represents meaningful interpersonal interaction, not just in the present but also across time. The study of language therefore presents problems of common interest to biology and the humanities. Given this special opportunity we may well ask, What can neurobiologists say to psychologists and humanists that would shed light on the biological process of human cognition?

The first and most important insight is that language abilities can be localized to one of the two cerebral hemispheres. The hemispheric asymmetry that ultimately gave rise to language emerged early in human evolution, perhaps as early as 300,000 years ago. The capability for language seems to be present at birth, and universal features of language are thought to derive in part from the structure of the cortical regions concerned with language in the left hemisphere.

From a biological standpoint, language is not a single capability but a family of capabilities, two of which, comprehension and expression, can be separated by distinctive functional sites in the brain. As first suggested by Wernicke, profound aphasia can result from simply disconnecting these two sites. Success in correlating major components of language with different anatomical regions led to the development of a simple model of language, the Wernicke–Geschwind model, which can account for a family of language-related disorders. This model, although clinically helpful, is overly simple and incorrect in detail.

Despite some notable insights, the neurobiological understanding of language is very rudimentary. The Wernicke–Geschwind model, although modified since its introduction, is only a beginning in the localization of cognitive functioning. It has, however, provided an important bridge between the analysis of language and its disorders by psycholinguists and the neuroanatomical localization of language function by neural scientists.

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