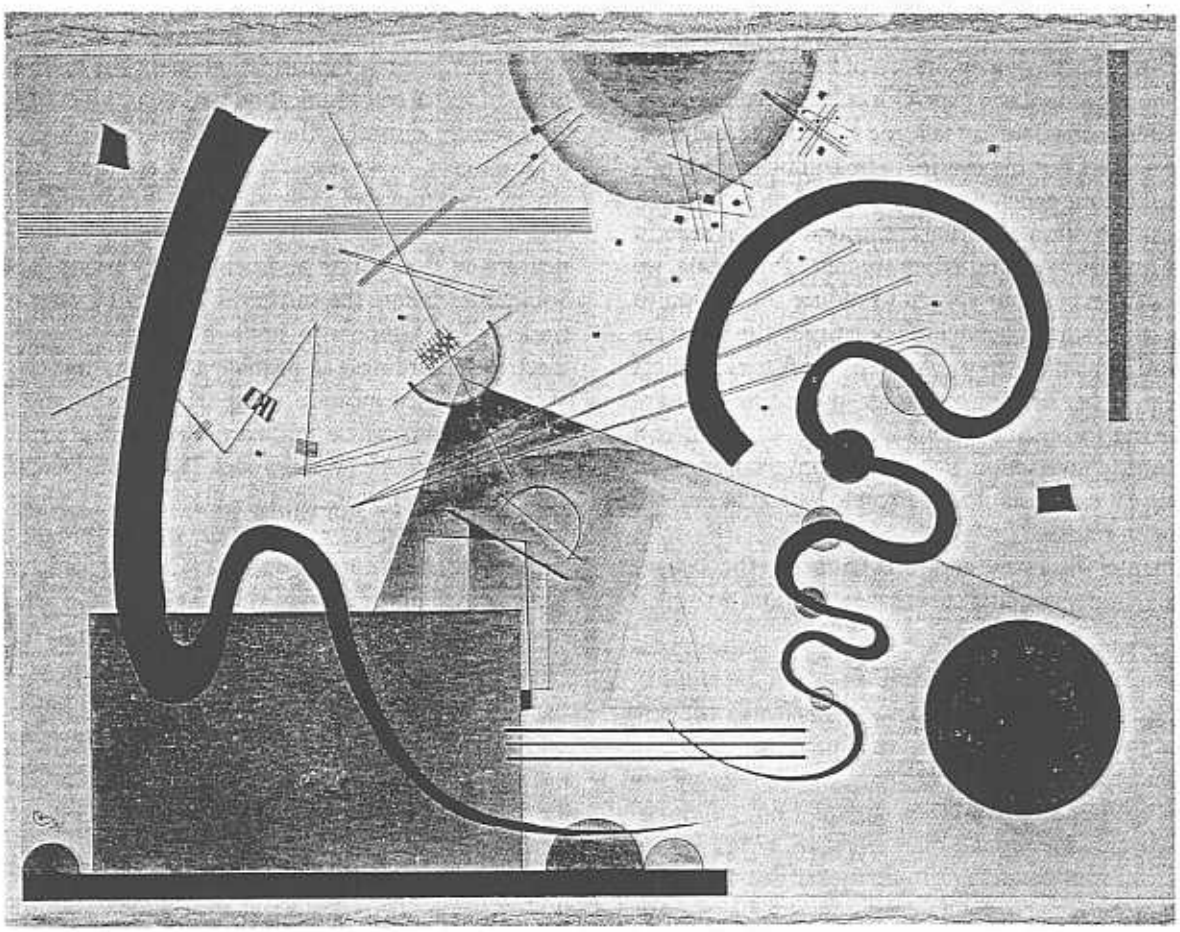
CHAPTER 16

Human Communication



Zwei Bewegungen by Wassily Kandinsky.

CHAPTER OUTLINE

Verbal behaviors constitute one of the most important classes of human social behavior. Our cultural evolution has been possible because we can talk and listen, write and read. Language enables our discoveries to be cumulative; knowledge gained by one generation can be passed on to the next.

The basic function of verbal communication is seen in its effects on other people. When we talk to someone, we almost always expect our speech to induce the person to engage in some sort of behavior. Sometimes, the behavior is of obvious advantage to us, as when we ask for an object or for help in performing a task. At other times we are simply asking for a social exchange: some attention and perhaps some conversation. Even "idle" conversation is not idle, because it causes another person to look at us and say something in return.

This chapter discusses the neural basis of verbal behavior: talking, understanding speech, reading, and writing.

Speech Production and Comprehension: Brain Mechanisms

Our knowledge of the physiology of language has been obtained primarily by observing the effects of brain lesions on people's verbal behavior. Although investigators have studied people who have undergone brain surgery or who have sustained head injuries, brain tumors, or infections, most of the observations have been made on people who have suffered strokes, or **cerebrovascular accidents**. The most common type of cerebrovascular accident is caused by obstruction of a blood vessel. The interruption in blood flow deprives a region of the brain of its blood supply, which causes cells in that region to die.

Another source of information about the brain mechanisms of verbal communication has been studies of patients with seizure disorders that are severe enough to require brain surgery. As we saw in Chapter 5, seizure surgery usually entails removal of a seizure focus—a region of the brain that includes scar tissue or other abnormalities that irritate neurons in the vicinity and periodically trigger a seizure. Sometimes, before the surgery is performed, a set of electrodes will be temporarily implanted in the patient's brain. Electrical recordings can be made through these electrodes to try to find the location of the seizure focus, and the patient's reactions can be studied while electrical stimulation is delivered through the electrodes. Then, if the patient is operated on, the surgeon can stimulate various regions of the brain and observe the effects of the stimulation on the patient's verbal behavior. (As we saw in Chapter 5, such surgery is performed under local anesthesia so that the patient can remain conscious.) Finally, if part of the brain is removed, the patient's behavior before the surgery can be compared with his or her behavior after removal of the brain tissue.

Although one might think that patients like these would be ideal subjects for studies of the brain mechanisms of language, we must remember that their brains contain abnormalities. If they did not, they would not be candidates for surgery. Many of these abnormalities occurred early in life—for example, as a consequence of obstetric difficulties. We know that when damage occurs in the immature brain, the course of development is altered. Thus, the brain of an adult with a long-standing seizure disorder is likely to be different from that of a person without such a disorder. In

cerebrovascular accident A "stroke"; brain damage caused by occlusion or rupture of a blood vessel in the brain.

fact, Devinsky et al. (1993) used cortical stimulation to map the location of speech areas of the temporal lobe of seizure patients and found a more widespread or atypical distribution of these areas in patients with a history of early onset of seizures. Thus, we must be careful in drawing conclusions about the location of brain regions that are involved in specific functions from patients undergoing seizure surgery.

A third source of information about the physiology of language comes from studies using functional imaging devices. In recent years, researchers have used PET and functional MRI to gather information about language processes from normal subjects. In general, these studies have confirmed or complemented what we have learned by studying patients with brain damage.

The most important category of speech disorders is aphasia, a primary disturbance in the comprehension or production of speech, caused by brain damage. Not all speech disturbances are aphasias; a patient must have difficulty comprehending, repeating, or producing meaningful speech, and this difficulty must not be caused by simple sensory or motor deficits or by lack of motivation. For example, inability to speak caused by deafness or paralysis of the speech muscles is not considered to be aphasia. In addition, the deficit must be relatively isolated; that is, the patient must appear to be aware of what is happening in his or her environment and to comprehend that others are attempting to communicate.

Lateralization

Verbal behavior is a *lateralized* function; most language disturbances occur after damage to the left side of the brain. The best way to determine which side of the brain is dominant for speech is to perform a *Wada test*, named after its inventor. (As we saw in Chapter 15, this test is also used to assess memory functions.) A patient who is about to undergo surgery that might encroach on a speech area receives a short-acting anesthetic in one carotid artery and then, when the effects have worn off, in the other. This procedure anesthetizes first one cerebral hemisphere and then the other; thus, in a few minutes the involvement of each hemisphere in speech functions can be assessed. In over 95 percent of right-handed people the left hemisphere is dominant for speech. That is, when the left hemisphere is anesthetized, the person loses the ability to speak. However, when the right hemisphere is anesthetized, the person can still talk and carry on a conversation. The figure is somewhat lower in left-handed people: approximately 70 percent. Therefore, unless I say otherwise, you can assume that the brain damage described in this chapter is located in the left (speech-dominant) hemisphere.

Why is one hemisphere specialized for speech? The perceptual functions of the left hemisphere are more specialized for the analysis of sequences of stimuli, occurring one after the other. The perceptual functions of the right hemisphere are more specialized for the analysis of space and geometrical shapes and forms, the elements of which are all present at the same time. Speech is certainly sequential; it consists of sequences of words, which are composed of sequences of sounds. Therefore, it makes sense for the left hemisphere to have become specialized at perceiving speech. In addition, as we saw in Chapter 8, the left hemisphere is involved in the control of sequences of voluntary movements. Perhaps this fact accounts for the localization of neural circuits involved in speech production, as well as speech perception, in the left hemisphere.

The brain is asymmetrical in structure as well as in function. For example, the size of speech areas in the frontal and temporal lobes is larger in the speech-dominant hemisphere, and there is even some evidence for differences in the size of some populations of neurons in these regions (Galaburda, Rosen, and Sherman, 1991; Hayes and Lewis, 1993; Foundas et al., 1996).

Although the circuits that are *primarily* involved in speech comprehension and production are located in the left hemisphere, it would be a mistake to conclude that the right hemisphere plays no role in speech. Speech is not simply a matter of talking—it is also having something to say. Similarly, listening is not simply hearing and recognizing words—it is understanding the meaning of what has been said. When we hear and understand words, and when we talk about or think about our own perceptions or memories, we are using neural circuits besides those directly involved in speech. Thus, these circuits, too, play a role in verbal behavior. For example, damage to the right hemisphere makes it difficult for a person to read maps, perceive spatial relations, and recognize complex geometrical forms. People with such damage also have trouble talking about things like maps and complex geometrical forms or understanding what other people have to say about them. The right hemisphere also appears to be involved in organizing a narrative—selecting and assembling the elements of what we want to say (Gardner et al., 1983). As we saw in Chapter 11, the right hemisphere is involved in the expression and recognition of emotion in the tone of voice. And as we shall see in this chapter, it is also involved in control of *prosody*—the normal rhythm and stress found in speech. Therefore, both hemispheres of the brain have a contribution to make to our language abilities.

aphasia Difficulty in producing or comprehending speech not produced by deafness or a simple motor deficit; caused by brain damage.

Speech Production

Being able to talk—that is, to produce meaningful speech—requires several abilities. First, the person must have something to talk about. Let us consider what this means. We can talk about something that is currently happening or something that happened in the past. In the first case we are talking about our perceptions: things we are seeing, hearing, feeling, smelling, and so on. In the second case we are talking about our memories of what happened in the past. Both perceptions of current events and memories of events that occurred in the past involve brain mechanisms in the posterior part of the cerebral hemispheres (the occipital, temporal, and parietal lobes). Thus, this region is largely responsible for our having something to say.

Of course, we can also talk about something that *did not* happen. That is, we can use our imagination to make up a story (or to tell a lie). We know very little about the neural mechanisms that are responsible for imagination, but it seems likely that they involve the mechanisms responsible for perceptions and memories; after all, when we make up a story, we must base it on knowledge that we originally acquired through perception and have retained in our memory.

Given that a person has something to say, actually doing so requires some additional brain functions. As we shall see in this section, the conversion of perceptions, memories, and thoughts into speech makes use of neural mechanisms located in the frontal lobes.

Damage to a region of the inferior left frontal lobe (Broca's area) disrupts the ability to speak: It causes **Broca's aphasia.** This disorder is characterized by slow, laborious, and nonfluent speech. When trying to talk with patients who have Broca's aphasia, most people find it hard to resist supplying the words the patients are obviously groping for. But although they often mispronounce words, the ones they manage to come out with are usually meaningful. The posterior part of the cerebral hemispheres has something to say, but the damage to the frontal lobe makes it difficult for the patients to express these thoughts.

People with Broca's aphasia find it easier to say some types of words than others. They have great difficulty saying the little words with grammatical meaning, such as *a, the, some, in,* or *about.* These words are called **function words,** because they have important grammatical functions. The words that they do manage to say are almost entirely **content words**—words that convey meaning, including nouns, verbs, adjectives, and adverbs, such as *apple, house, throw,* or *heavy.* Here is a sample of speech from a man with Broca's aphasia, who is trying to describe the scene shown in *Figure 16.1.* As you will see, his words are meaningful, but what he says is certainly not grammatical. The dots indicate long pauses.

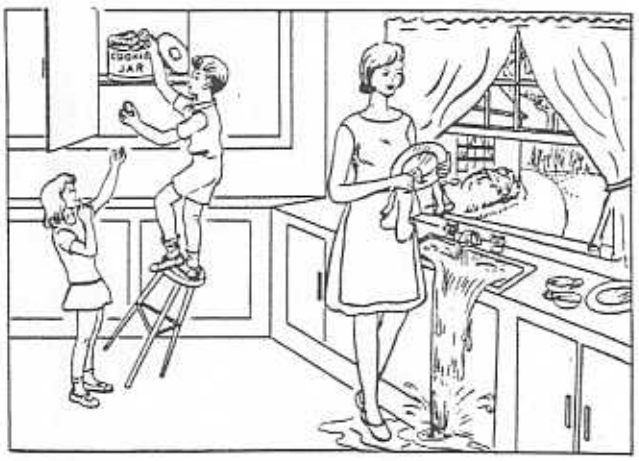


Figure 16.1

The drawing of the kitchen story, part of the Boston Diagnostic Aphasia Test.

(From Goodglass, H., and Kaplan, E. *The Assessment of Aphasia and Related Disorders,* 2nd ed. Philadelphia: Lea & Febiger, 1983. Reprinted with permission.)

> kid . . . kk . . . can . . . candy . . . cookie . . . candy . . . well I don't know but it's writ . . . easy does it . . . slam . . . early . . . fall . . . men . . . many no . . . girl. Dishes . . . soap . . . soap . . . water . . . water . . . falling pah that's all . . . dish . . . that's all.

> Cookies . . . can . . . candy . . . cookies cookies . . . he . . . down . . . That's all. Girl . . . slipping water . . . water . . . and it hurts . . . much to do . . . Her . . . clean up . . . Dishes . . . up there . . . I think that's doing it. (Obler and Gjerlow, 1999, p. 41)

People with Broca's aphasia can comprehend speech much better than they can produce it. In fact, some observers have said that their comprehension is unimpaired, but as we will see, this is not quite true. Broca (1861) suggested that this form of aphasia is produced by a lesion of the frontal association cortex, just anterior to the face region of the primary motor cortex. Subsequent research proved him to be essentially correct, and we now call the region **Broca's area.** (See *Figure 16.2.*)

Broca's aphasia A form of aphasia characterized by agrammatism, anomia, and extreme difficulty in speech articulation.

function word A preposition, article, or other word that conveys little of the meaning of a sentence but is important in specifying its grammatical structure.

content word A noun, verb, adjective, or adverb that conveys meaning.

Broca's area A region of frontal cortex, located just rostral to the base of the left primary motor cortex, that is necessary for normal speech production.

Lesions that produce Broca's aphasia are certainly centered in the vicinity of Broca's area. However, damage that is restricted to the cortex of Broca's area does not appear to produce Broca's aphasia; the damage must extend to surrounding regions of the frontal lobe and to the underlying subcortical white matter (H. Damasio, 1989; Naeser et al., 1989). In addition, there is evidence that lesions of the basal ganglia—especially the head of the caudate nucleus—can also produce a Broca-like aphasia (Damasio, Eslinger, and Adams, 1984). Figure 16.3 shows the averaged plot of PET scans of regional blood flow from a group of subjects who were reading words aloud (Leblanc et al., 1992). As you can see, the task activated subcortical regions under Broca's area (including the head of the caudate nucleus) as well as the neocortex. (See *Figure 16.3.*)

Recent studies using PET scanners have shown that a *functional* lesion can often be much more extensive than the area of primary tissue damage. For example, Metter (1991) notes that small lesions in the basal ganglia and in the adjacent subcortical white matter can cause decreased metabolism of a fairly large region of the frontal cortex—even when autopsy shows no loss of neurons in the cortex. In addition, lesions in the frontal lobe can often cause decreased metabolism in the temporal and parietal lobes, presumably because the lesions disrupt connections between these areas. Thus the full extent of a lesion will often be underestimated by examining CT or MRI scans alone.

What do the neural circuits in and around Broca's area do? Wernicke (1874) suggested that Broca's area contains motor memories—in particular, *memories of the sequences of muscular movements that are needed to articulate words.*

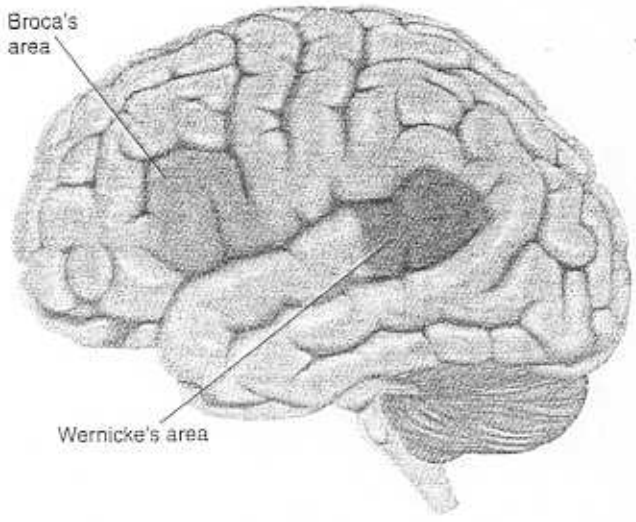


Figure 16.2

The location of the primary speech areas of the brain. (Wernicke's area will be described later.)

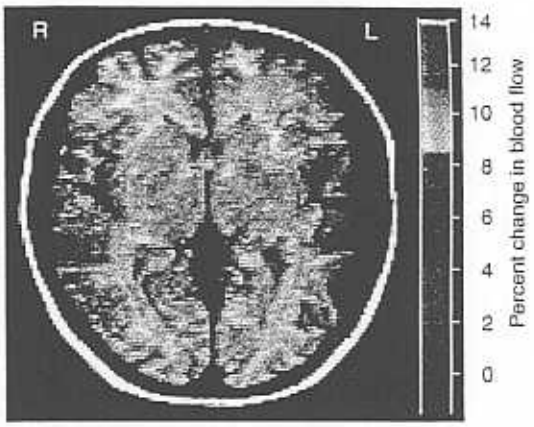


Figure 16.3

An averaged plot of PET scans of regional cerebral blood flow, superimposed on an MRI scan, taken while the subjects were reading words aloud. Note that the region of activation includes subcortical regions as well as the cerebral cortex of Broca's area. Also note that left and right are reversed.

(From Leblanc, R., Meyer, E., Bub, D., Zatorre, R. J., and Evans, A. C. *Neurosurgery,* 1992, *31,* 369–373. Reprinted with permission.)

Talking involves rapid movements of the tongue, lips, and jaw, and these movements must be coordinated with each other and with those of the vocal cords; thus talking requires some very sophisticated motor control mechanisms. Obviously, circuits of neurons somewhere in our brain will, when properly activated, cause these sequences of movements to be executed. Because damage to the inferior caudal left frontal lobe (including Broca's area) disrupts the ability to articulate words, this region is the most likely candidate for the location of these "programs." The fact that this region is directly connected to the part of the primary motor cortex that controls the muscles used for speech certainly supports this conclusion.

But the speech functions of the left frontal lobe include more than programming the movements that are used to speak. Broca's aphasia is much more than a deficit in pronouncing words. In general, three major speech deficits are produced by lesions in and around Broca's area: *agrammatism, anomia,* and *articulation difficulties.* Although most patients with Broca's aphasia will have all of these deficits to some degree, their severity can vary considerably from person to person—presumably, because their brain lesions differ.

Agrammatism refers to a patient's difficulty in using grammatical constructions. This disorder can appear all by

agrammatism One of the usual symptoms of Broca's aphasia; a difficulty in comprehending or properly employing grammatical devices, such as verb endings and word order.

itself, without any difficulty in pronouncing words (Nadeau, 1988). As we saw, people with Broca's aphasia rarely use function words. In addition, they rarely use grammatical markers such as *-ed* or auxiliaries such as *have* (as in *I have gone*). For some reason they *do* often use *-ing*, perhaps because this ending converts a verb into a noun. A study by Saffran, Schwartz, and Marin (1980) illustrates this difficulty. The following quotations are from agrammatic patients attempting to describe pictures:

Picture of a boy being hit in the head by a baseball:

The boy is catch . . . the boy is hitch . . . the boy is hit the ball. (Saffran, Schwartz, and Marin, 1980, p. 229)

Picture of a girl giving flowers to her teacher:

Girl . . . wants to . . . flowers . . . flowers and wants to . . . The woman . . . wants to . . . The girl wants to . . . the flowers and the woman. (Saffran, Schwartz, and Marin, 1980, p. 234)

So far, I have described Broca's aphasia as a disorder in speech *production*. In an ordinary conversation Broca's aphasics seem to understand everything that is said to them. They appear to be irritated and annoyed by their inability to express their thoughts well, and they often make gestures to supplement their scanty speech. The striking disparity between their speech and their comprehension often leads people to assume that their comprehension is normal. But it is not. Schwartz, Saffran, and Marin (1980) showed Broca's aphasics pairs of pictures in which agents and objects of the action were reversed: for example, a horse kicking a cow and a cow kicking a horse, a truck pulling a car and a car pulling a truck, and a dancer applauding a clown and a clown applauding a dancer. As they showed each pair of pictures, they read the subject a sentence, for example, *The horse kicks the cow.* The subjects' task was to point to the appropriate picture, indicating whether they understood the grammatical construction of the sentence. (See *Figure 16.4.*) They performed very poorly.

The correct picture in the study by Schwartz and her colleagues was specified by a particular aspect of grammar: word order. The agrammatism that accompanies Broca's aphasia appears to disrupt patients' ability to use grammatical information, including word order, to decode the meaning of a sentence. Thus, their deficit in comprehension parallels their deficit in production. If they heard a sentence such as *The man swats the mosquito,* they would understand that it concerns a man and a mosquito and the action of swatting. They would have no trouble figuring out who is doing what to whom. But a sentence such as *The horse kicks the cow* does not provide any extra cues; if the grammar is not understood, neither is the meaning of the sentence.

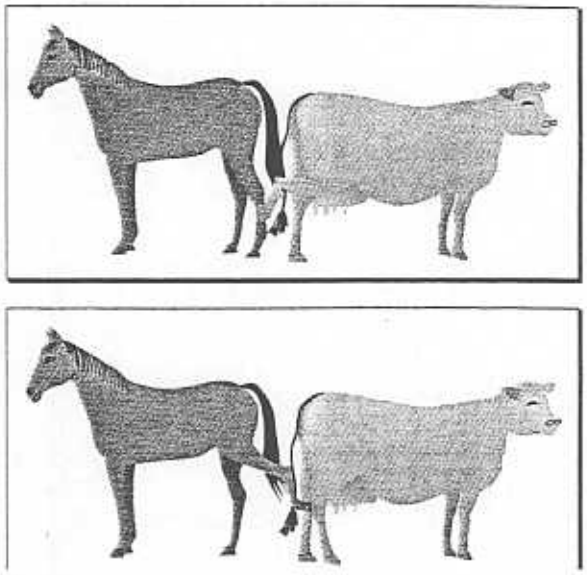


Figure 16.4

An example of the stimuli used in the experiment by Schwartz, Saffran, and Marin (1980).

The second major speech deficit seen in Broca's aphasia is **anomia** ("without name"). Anomia refers to a word-finding difficulty; and because all aphasics omit words or use inappropriate ones, anomia is actually a primary symptom of *all* forms of aphasia. However, because the speech of Broca's aphasics lacks fluency, their anomia is especially apparent; their facial expression and frequent use of sounds like "uh" make it obvious that they are groping for the correct words.

The third major characteristic of Broca's aphasia is *difficulty with articulation.* Patients mispronounce words, often altering the sequence of sounds. For example, *lipstick* might be pronounced "likstip." People with Broca's aphasia recognize that their pronunciation is erroneous, and they usually try to correct it.

These three deficits are seen in various combinations in different patients, depending on the exact location of the lesion and, to a certain extent, on their stage of recovery. We can think of these deficits as constituting a hierarchy. On the lowest, most elementary level is control of the sequence of movements of the muscles of speech; damage to this ability leads to articulation difficulties. The next higher level is selection of the particular "programs" for individual words; damage to this ability leads to anomia. Finally, the highest level is selection of grammatical structure, in-

anomia Difficulty in finding (remembering) the appropriate word to describe an object, action, or attribute; one of the symptoms of aphasia.

cluding word order, use of function words, and word endings; damage to this ability leads to agrammatism.

We might expect that the direct control of articulation would involve the face area of the primary motor cortex and portions of the basal ganglia, while the selection of words, word order, and grammatical markers would involve Broca's area and adjacent regions of the frontal association cortex. Some recent studies indicate that different categories of symptoms of Broca's aphasia do, indeed, involve different brain regions. Dronkers (1996) appears to have found a critical location for control of speech articulation: the left precentral gyrus of the insula. The insular cortex is located on the lateral wall of the cerebral hemisphere behind the anterior temporal lobe. Normally, this region is hidden and can be seen only when the temporal lobe is dissected away. (See *Figure 16.5.*) Dronkers discovered the apparent role of this region by plotting the lesions of patients with and without apraxia of speech who had strokes that damaged the same general area of the brain. (Apraxia of speech is an impairment in the ability to program movements of the tongue, lips, and throat that are required to produce the proper sequence of speech sounds.) Figure 16.6(a) shows the overlap of the lesions of twenty-five patients with apraxia of speech. As you can see, a region of 100 percent overlap, shown in yellow, falls on the left precentral gyrus of the insula. (See *Figure 16.6a.*) In contrast, *none* of the lesions of nineteen patients who did not show apraxia of speech included damage to this region. (See *Figure 16.6b.*)

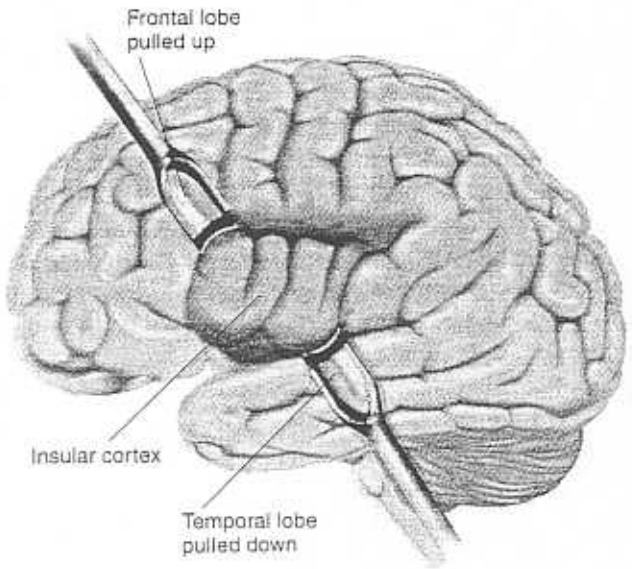


Figure 16.5

The insular cortex, normally hidden behind the rostral temporal lobe.

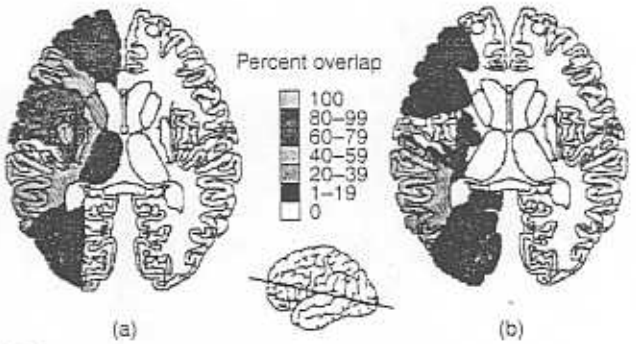


Figure 16.6

Evidence for involvement of the insular cortex in speech articulation. Percentage overlap in the lesions of twenty-five patients (a) with apraxia of speech and (b) without apraxia of speech. The only region common to all lesions that produced apraxia of speech was the precentral gyrus of the insular cortex.
(From Dronkers, N. F. *Nature*, 1996, *384*, 159–161.)

At least two functional imaging studies support Dronkers's conclusion. Kuriki, Mori, and Hirata (1999) and Wise et al. (1999) found that pronunciation of words caused activation of the left anterior insula.

Another region of the brain seems to be involved in speech production. Studies have found that damage to the periaqueductal gray matter (PAG) of the midbrain disrupts vocalization in a variety of species, including frogs, cats, dogs, monkeys, and apes. In addition, electrical or chemical stimulation of the PAG elicits vocalization, and the firing rate of single neurons in this region increases when an animal vocalizes (Jürgens, 1998). Lesions of the PAG also cause mutism in humans. For example, Esposito et al. (1999) described the case of a woman with a PAG lesion who could understand speech and respond to it nonverbally but could not make any speech sounds herself—even in response to pain. However, she could move her lips as if she were speaking. For example, when an examiner asked her if she were thirsty, she moved her lips as if to say "oui." (The patient spoke French.)

The agrammatism and anomia of Broca's aphasia are normally caused by subcortical damage or damage to the neocortex of the inferior frontal lobe. These findings are supported by a PET study by Stromswold et al. (1996). Subjects listened to syntactically complex sentences and had to decide whether they made sense. For example, "The dog that the cat scratched chased the mouse" makes sense, while "The mouse that the cat scratched chased the dog"

apraxia of speech Impairment in the ability to program movements of the tongue, lips, and throat required to produce the proper sequence of speech sounds.

does not. The process of listening to such sentences and judging their plausibility certainly exercises neural circuits involved in comprehension of grammar, and the investigators found that doing so increased the activity of Broca's area, especially the part closest to the lateral fissure. A functional MRI study with Japanese subjects obtained similar results (Inui et al., 1998).

Experiments have shown that people with Broca's aphasia have difficulty carrying out a sequence of commands such as "Pick up the red circle and touch the green square with it" (Boller and Dennis, 1979). This finding, along with the other symptoms I have described in this section, suggests that an important function of the left frontal lobe is sequencing—of movements of the muscles of speech (producing words) and of words (comprehending and producing grammatical speech).

One study has found that agrammatism can be caused by damage to the cerebellum. Traditionally, the cerebellum has been regarded as a computer involved in coordinating movements, especially those involving precise timing. Neurologists have long recognized the fact that damage to the cerebellum can disrupt motor control of speech, which is not surprising when we consider the role the cerebellum plays in all forms of rapid, skilled movements. The speech of patients who have cerebellar damage is slow and monotonous, syllables tend to be separated by brief pauses, and enunciation is imprecise (Adams and Victor, 1981). However, the advent of PET and functional MRI scanning has found that even when movement is controlled for, engaging in various types of verbal activity activates the cerebellum (Fiez, 1996). The cerebellum may even be involved in grammatical aspects of speech. Silveri, Leggio, and Molinari (1994) studied a patient with cerebellar damage with agrammatic speech, characterized by uninflected verb forms. The following quotation (a word-by-word translation from the original Italian) illustrates the agrammatic nature of this patient's speech:

> I was watching television. One moment after, immediately after, to feel one half not to go. To have an attack, to be unable to speak. Upstairs there was my wife sleeping because it was midnight. I suddenly to stand up suddenly to fall down. Not to do anything because there was the carpet. Not to speak then to vomit all night long. I to vomit. My son came, soon an injection he gives and the drips. I to wait that here at the Polyclinic is admitted that free the bed. To wait that here to be the bed free to give the bed free.

Speech Comprehension

Comprehension of speech obviously begins in the auditory system, which detects and analyzes sounds. But *recognizing* words is one thing; *comprehending* them—understanding their meaning—is another. Recognizing a spoken word is a complex perceptual task that relies on memories of sequences of sounds. This task appears to be accomplished by neural circuits in the middle and posterior portion of the superior temporal gyrus of the left hemisphere—a region that has come to be known as Wernicke's area. (Refer to *Figure 16.2.*)

Wernicke's Aphasia: Description

The primary characteristics of **Wernicke's aphasia** are poor speech comprehension and production of meaningless speech. Unlike Broca's aphasia, Wernicke's aphasia is fluent and unlabored; the person does not strain to articulate words and does not appear to be searching for them. The patient maintains a melodic line, with the voice rising and falling normally. When you listen to the speech of a person with Wernicke's aphasia, it appears to be grammatical. That is, the person uses function words such as *the* and *but* and employs complex verb tenses and subordinate clauses. However, the person uses few content words, and the words that he or she strings together just do not make sense. In the extreme, speech deteriorates into a meaningless jumble, illustrated by the following quotation:

Examiner: What kind of work did you do before you came into the hospital?

Patient: Never, now mista oyge I wanna tell you this happened when happened when he rent. His—his kell come down here and is—he got ren something. It happened. In thesse ropiers were with him for hi—is friend—like was. And it just happened so I don't know, he did not bring around anything. And he did not pay it. And he roden all o these arranjen from the pedis on from iss pescid. In these floors now and so. He hadn't had em round here. (Kertesz, 1981, p. 73)

Because of the speech deficit of people with Wernicke's aphasia, when we try to assess their ability to comprehend speech, we must ask them to use nonverbal responses. That is, we cannot assume that they do not understand what other people say to them just because they do not give the proper answer. A commonly used test of comprehension assesses their ability to understand questions by pointing to objects on a table in front of them. For example, they are asked to "Point to the one with ink." If they point to an object other than the pen, they have not understood the re-

Wernicke's area A region of auditory association cortex on the left temporal lobe of humans, which is important in the comprehension of words and the production of meaningful speech.

Wernicke's aphasia A form of aphasia characterized by poor speech comprehension and fluent but meaningless speech.

quest. When tested this way, people with severe Wernicke's aphasia do indeed show poor comprehension.

A remarkable fact about people with Wernicke's aphasia is that they often seem unaware of their deficit. That is, they do not appear to recognize that their speech is faulty, nor do they recognize that they cannot understand the speech of others. They do not look puzzled when someone tells them something, even though they obviously cannot understand what they hear. Perhaps their comprehension deficit prevents them from realizing that what they say and hear makes no sense. They still follow social conventions, taking turns in conversation with the examiner, even though they do not understand what the examiner says—and what they say in return makes little sense. They remain sensitive to the other person's facial expression and tone of voice and begin talking when he or she asks a question and pauses for an answer. One patient with Wernicke's aphasia made the following responses when asked to name ten common objects:

toothbrush → "stoktery"
cigarette → "cigarette"
pen → "tankt"
knife → "nike"
fork → "fahk"
quarter → "minkt"
pen → "spentee"
matches → "senktr"
key → "seek"
comb → "sahk"

He acted sure of himself and gave no indication that he recognized that most of his responses were meaningless. The responses he made were not simply new words that he had invented; he was asked several times to name the objects and gave different responses each time (except for *cigarette*, which he always named correctly).

Wernicke's Aphasia: Analysis

Because the superior temporal gyrus is a region of auditory association cortex, and because a comprehension deficit is so prominent in Wernicke's aphasia, this disorder has been characterized as a *receptive* aphasia. Wernicke suggested that the region that now bears his name is the location of *memories of the sequences of sounds that constitute words.* This hypothesis is reasonable; it suggests that the auditory association cortex of the superior temporal gyrus recognizes the sounds of words, just as the visual association cortex of the inferior temporal gyrus recognizes the sight of objects.

There seems little doubt that Wernicke's area is involved in learning. In fact, Jacobs, Schall, and Scheibel (1993) found that the average length of apical dendrites of pyramidal cells in Wernicke's area was positively related to a person's educational level.

But why should damage to an area that is responsible for the ability to recognize spoken words disrupt people's ability to speak? In fact, it does not; Wernicke's aphasia, like Broca's aphasia, actually appears to consist of several deficits. The abilities that are disrupted include *recognition of spoken words, comprehension of the meaning of words,* and the *ability to convert thoughts into words.* Let us consider each of these abilities in turn.

■ **Recognition: Pure Word Deafness** As I said in the introduction to this section, *recognizing* a word is not the same as *comprehending* it. If you hear a foreign word several times, you will learn to recognize it; but unless someone tells you what it means, you will not comprehend it. Recognition is a perceptual task; comprehension involves retrieval of additional information from memory.

Damage to the left temporal lobe can produce a disorder of auditory word recognition, uncontaminated by other problems. This syndrome is called **pure word deafness.** Although people with pure word deafness are not deaf, they cannot understand speech. As one patient put it, "I can hear you talking, I just can't understand what you're saying." Another said, "It's as if there were a bypass somewhere, and my ears were not connected to my voice" (Saffran, Marin, and Yeni-Komshian, 1976, p. 211). These patients can recognize nonspeech sounds such as the barking of a dog, the sound of a doorbell, and the chirping of a bird. Often, they can recognize the emotion expressed by the intonation of speech even though they cannot understand what is being said. More significantly, their own speech is excellent. They can often understand what other people are saying by reading their lips. They can also read and write, and sometimes, they ask people to communicate with them in writing. Clearly, pure word deafness is not an inability to comprehend the meaning of words; if it were, people with this disorder would not be able to read people's lips or read words written on paper.

Functional imaging studies confirm that perception of speech sounds activates neurons in the auditory association cortex of the superior temporal gyrus. Binder et al. (1994) found that the sounds of real words (such as *barn* and *box*) and pseudowords containing speech sounds (such as *narb* and *skob*) activated this region better than noise did. The stimuli activated the left and right hemispheres

pure word deafness The ability to hear, to speak, and (usually) to read and write without being able to comprehend the meaning of speech; caused by damage to Wernicke's area or disruption of auditory input to this region.

equally. Belin et al. (2000) found similar results and, in addition, found that as they presented more and more distorted speech, they saw parallel decreases in the subjects' ability to recognize words and the level of activation of the superior temporal gyrus.

What is involved in the analysis of speech sounds? Just what tasks does the auditory system have to accomplish? And what are the differences in the functions of the auditory association cortex of the left and right hemispheres? Most researchers believe that the left hemisphere is primarily involved in judging the timing of the components of rapidly changing complex sounds, whereas the right hemisphere is primarily involved in judging more slowly changing components, including melody. Evidence suggests that the most crucial aspect of speech sounds is timing, not pitch. We can recognize words whether they are conveyed by the low pitch of a man or the high pitch of a woman or child. In fact, Shannon et al. (1995) found that people had no difficulty understanding speech from which the experimenters had removed almost all the tonal information, leaving only some noise modulated by the rapid stops and starts that characterize human speech sounds. On the other hand, emphasis or the emotional state of the speaker is conveyed by the pitch and melody of speech and by much slower changes in rhythm. In other words, the sounds that convey the identity of words are very brief, whereas those that convey prosody (emphasis and emotion) are of longer duration. Perhaps the auditory system of the left hemisphere is simply specialized for the recognition of acoustical events of short duration.

In a review of the literature, Phillips and Farmer (1990) suggest precisely this hypothesis. They note that careful studies of patients with pure word deafness have shown that the patients can distinguish between different vowels but not between different consonants—especially between different stop consonants, such as /t/, /d/, /k/, or /p/. (Linguists represent speech sounds by putting letters or special phonetic symbols between pairs of slashes.) Patients with pure word deafness *can* generally recognize consonants with a long duration, such as /s/, /z/, or /f/. (Say these consonants to yourself, and you will see how different they sound from the first four examples.)

Phillips and Farmer note that the important acoustical events in speech sounds fall within a time range of a few milliseconds to a few tens of milliseconds. Speech sounds are made by rapidly moving lips, tongue, and soft palate, which produce acoustical events that can be distinguished only by a fine-grained analysis. In contrast, most environmental sounds do not contain such a fine temporal structure. The authors also note that "pure" word deafness is not absolutely pure. That is, when people with this disorder are tested carefully with recordings of a variety of environmental sounds, they have difficulty recognizing at least some of them. Although *most* environmental sounds do not contain a fine temporal structure, some do—and patients have difficulty recognizing them. For example, one patient with pure word deafness could no longer understand messages in Morse code but could still *send* messages that way.

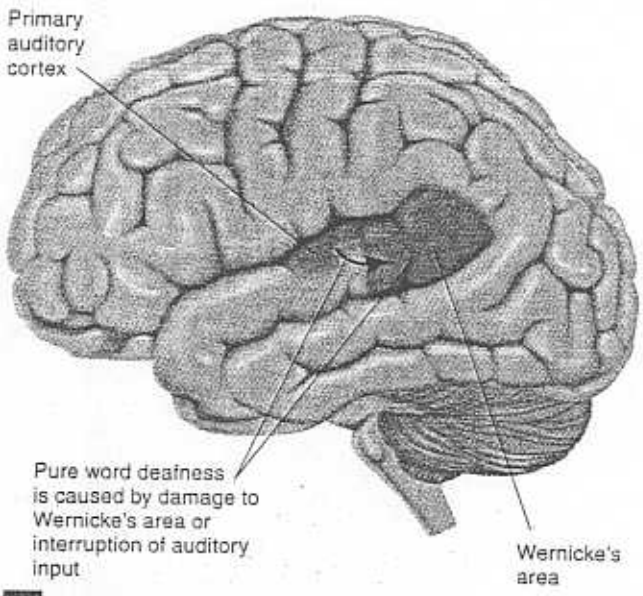


Figure 16.7

The brain damage that causes pure word deafness.

Apparently, two types of brain injury can cause pure word deafness: disruption of auditory input to Wernicke's area and damage to Wernicke's area itself. Disruption of auditory input can be produced by bilateral damage to the primary auditory cortex, or it can be caused by damage to the white matter in the left temporal lobes that cuts axons bringing auditory information from the primary auditory cortex to Wernicke's area (Digiovanni et al., 1992; Takahashi et al., 1992). Either type of damage—disruption of auditory input or damage to Wernicke's area—disturbs the analysis of the sounds of words and hence prevents people from recognizing other people's speech. (See *Figure 16.7.*)

■ **Comprehension: Transcortical Sensory Aphasia** The other symptoms of Wernicke's aphasia—failure to comprehend the meaning of words and inability to express thoughts in meaningful speech—appear to be produced by damage that extends beyond Wernicke's area into the region that surrounds the posterior part of the lateral fissure, near the junction of the temporal, occipital, and parietal lobes. For want of a better term, I will refer to this region as the *posterior language area.* (See *Figure 16.8.*) The posterior lan-

transcortical sensory aphasia A speech disorder in which a person has difficulty comprehending speech and producing meaningful spontaneous speech but can repeat speech; caused by damage to the region of the brain posterior to Wernicke's area.

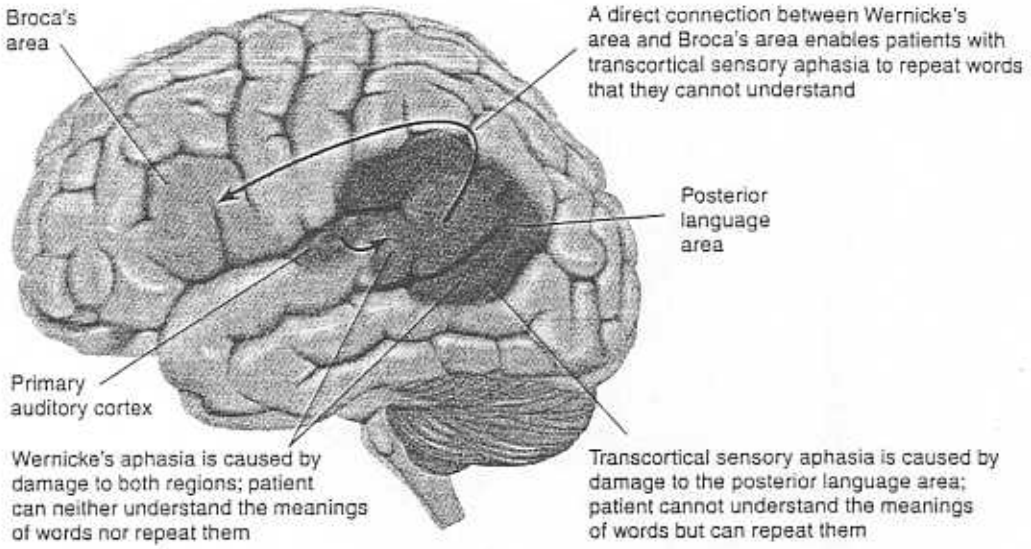


Figure 16.8

The location and interconnections of the posterior language area and an explanation of its role in transcortical sensory aphasia and Wernicke's aphasia.

guage area appears to serve as a place for interchanging information between the auditory representation of words and the meanings of these words, stored as memories in the rest of the sensory association cortex.

Damage to the posterior language area alone, which isolates Wernicke's area from the rest of the posterior language area, produces a disorder known as **transcortical sensory aphasia**. (See *Figure 16.9.*) The difference between transcortical sensory aphasia and Wernicke's aphasia is that patients with this disorder *can repeat what other people say to them;* therefore, they can recognize words. However, *they cannot comprehend the meaning of what they hear and repeat; nor can they produce meaningful speech of their own.* How can these people repeat what they hear? Because the posterior language area is damaged, repetition does not involve this part of the brain. Obviously, there must be a direct connection between Wernicke's area and Broca's area that bypasses the posterior language area. (See *Figure 16.8.*)

The fact that recognition and comprehension of speech require separate brain functions is illustrated dramatically by a case reported by Geschwind, Quadfasel, and Segarra (1968). The patient sustained extensive brain damage from carbon monoxide produced by a faulty water heater. (The damage included considerably more brain tissue than occurs in most cases of transcortical sensory aphasia, but it illustrates the distinction between the recognition and comprehension of speech.) The patient spent several years in the hospital before she died, without ever saying anything meaningful on her own. She did not follow verbal commands or otherwise give signs of understanding them. However, she often repeated what was said to her. The repetition was not parrotlike; she did

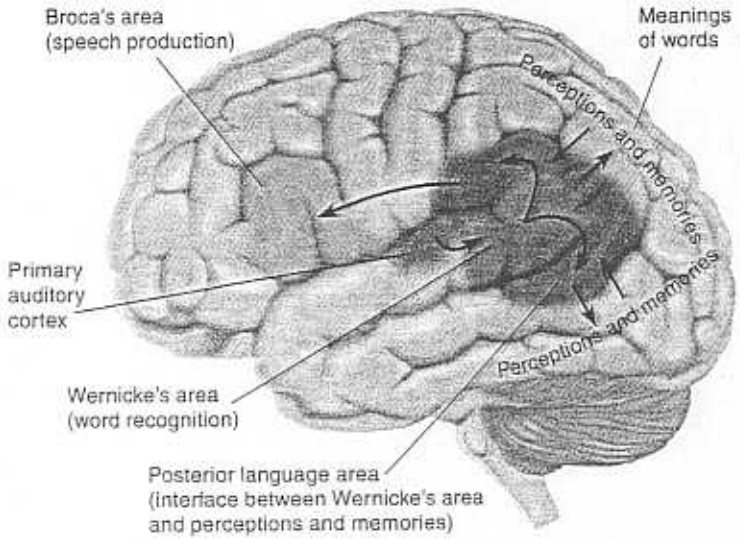


Figure 16.9

The "dictionary" in the brain. Wernicke's area contains the auditory entries of words; the meanings are contained as memories in the sensory association areas. Black arrows represent comprehension of words—the activation of memories that correspond to a word's meaning. Red arrows represent translation of thoughts or perceptions into words.

not imitate accents different from her own, and if someone made a grammatical error while saying something to her, she sometimes repeated correctly, without the error. She could also recite poems if someone started them. For example, when an examiner said "Roses are red, violets are blue," she continued with "Sugar is sweet and so are you." She could sing and would do so when someone started singing a song she knew. She even learned new songs from the radio while in the hospital. Remember, though, that she gave *no signs of understanding anything she heard or said.* This disorder, along with pure word deafness, clearly confirms the conclusion that *recognizing* spoken words and *comprehending* them involve different brain mechanisms.

In conclusion, transcortical sensory aphasia can be seen as Wernicke's aphasia without a repetition deficit. To put it another way, the symptoms of Wernicke's aphasia consist of those of pure word deafness plus those of transcortical sensory aphasia. (See *Figure 16.8.*)

■ **What Is Meaning?** As we have seen, Wernicke's area is involved in the analysis of speech sounds and thus in the recognition of words. Damage to the posterior language area does not disrupt people's ability to recognize words, but it does disrupt their ability to understand them or to produce meaningful speech of their own. But what, exactly, do we mean by the word *meaning?* And what types of brain mechanisms are involved?

Words refer to objects, actions, or relations in the world. Thus, the meaning of a word is defined by particular memories associated with it. For example, knowing the meaning of the word *tree* means being able to imagine the physical characteristics of trees: what they look like, what the wind sounds like blowing through their leaves, what the bark feels like, and so on. It also means knowing facts about trees: about their roots, buds, flowers, nuts, and wood and the chlorophyll in their leaves. These memories are stored not in the primary speech areas but in other parts of the brain, especially regions of the association cortex. Different categories of memories may be stored in particular regions of the brain, but they are somehow tied together, so that hearing the word *tree* activates all of them. (As we saw in Chapter 15, the hippocampal formation is involved in this process of tying related memories together.)

In thinking about the brain's verbal mechanisms involved in recognizing words and comprehending their meaning, I find that the concept of a dictionary serves as a useful analogy. Dictionaries contain entries (the words) and definitions (the meanings of the words). In the brain we have at least two types of entries: auditory and visual. That is, we can look up a word according to how it sounds or how it looks (in writing). Let us consider just one type of entry: the sound of a word. (I will discuss reading and writing later in this chapter.) We hear a familiar word and understand its meaning. How do we do so?

First, we must recognize the sequence of sounds that constitute the word—we find the auditory entry for the word in our "dictionary." As we saw, this entry appears in Wernicke's area. Next, the memories that constitute the meaning of the word must be activated. Presumably, Wernicke's area is connected—through the posterior language area—with the neural circuits that contain these memories. (See *Figure 16.9.*)

The process works in reverse when we describe our thoughts or perceptions in words. Suppose we want to tell someone about a tree that we just planted in our yard. Thoughts about the tree (for example, a visual image of it) occur in our association cortex—the visual association cortex, in this example. Information about the activity of these circuits is sent first to the posterior language area and then to Broca's area, which causes the words to be set into a grammatical sentence and pronounced. (See *Figure 16.9.*)

What evidence do we have that meanings of words are represented by neural circuits in various regions of the association cortex? The best evidence comes from the fact that damage to particular regions of the sensory association cortex can damage particular kinds of information and thus abolish particular kinds of meanings. For example, I met a patient who had recently had a stroke that damaged a part of her right parietal lobe that played a role in spatial perception. She was alert and intelligent and showed no signs of aphasia. However, she was confused about directions and other spatial relations. When asked to, she could point to the ceiling and the floor, but she could not say which was *over* the other. Her perception of other people appeared to be entirely normal, but she could not say whether a person's head was at the *top* or *bottom* of the body.

I wrote a set of multiple-choice questions to test her ability to use words denoting spatial relations. The results of the test indicated that she did not know the meaning of words such as *up, down,* and *under* when they referred to spatial relations, but she could use these words normally when they referred to nonspatial relations. For example, here are some of her incorrect responses when the words referred to spatial relations:

A tree's branches are *under* its roots.
The sky is *down.*
The ceiling is *under* the floor.

She made only ten correct responses on the sixteen-item test. In contrast, she got all eight items correct when the words referred to nonspatial relations such as the following:

After exchanging pleasantries, they got *down* to business.
He got sick and threw *up.*

Damage to part of the association cortex of the *left* parietal lobe can produce an inability to name the body parts. The disorder is called autotopagnosia, or "poor knowledge of one's own topography." (A better name would have been *autotopanomia*, "poor naming of one's own topography.") People who can otherwise converse normally cannot reliably point to their elbow, knee, or cheek when asked to do so and cannot name body parts when the examiner points to them. However, they have no difficulty understanding the meaning of other words.

Other investigators have reported verbal deficits that include disruption of particular categories of meaning. McCarthy and Warrington (1988) reported the case of a man with left temporal lobe damage (patient T.B.) who was unable to explain the meaning of words that denoted living things. For example, when he was asked to define the word *rhinoceros*, he said, "Animal, can't give you any functions." However, when he was shown a *picture* of a rhinoceros, he said, "Enormous, weighs over one ton, lives in Africa." Similarly, when asked what a *dolphin* was, he said, "a fish or a bird"; but he responded to a *picture* of a dolphin by saying, "Dolphin lives in water . . . they are trained to jump up and come out . . . In America during the war years they started to get this particular animal to go through to look into ships." Clearly, patient T.B. has lost not his knowledge of specific animals but only the ability to name them. Presumably, the damage to his brain disconnected circuits involved in the recognition of words from those involved in his memories of animals. When T.B. was asked to define the meanings of words that denoted inanimate objects (such as *lighthouse* or *wheelbarrow*), he had no trouble at all.

Functional imaging studies of people without brain damage confirm these findings. Several experiments have found that perception of words and concepts from different categories activate different parts of the brain. For example, Spitzer et al. (1995) had people name pictures of items that belonged to four different categories: animals, furniture, fruit, and tools. Functional MRI scans revealed some category-specific sites of activation in the frontal and temporal lobes.

More widespread damage to the temporal and parietal lobes can cause a general loss in comprehension—and not simply naming—presumably because of damage to regions of the brain that contain specific memories. For example, Damasio and Tranel (1990) studied a patient who had sustained severe damage to the temporal lobes. Besides becoming amnesic (his hippocampal formation was destroyed bilaterally), he had lost a considerable amount of specific information. For example, he recognized that a raccoon was an animal but had no idea of where it lived, what it ate, or what its name was. Hodges et al. (1992) reported several similar cases, caused by progressive degeneration of the temporal lobes. One patient was asked, "Have you been to America?" She replied, "What's America?" When she was asked, "What is your favorite food?" she said, "Food, food, I wish I knew what that was" (p. 1786). Another patient was frightened when he found a snail in his garden and thought that a goat was a strange creature. Hodges and his colleagues suggest the term *semantic aphasia* to refer to this syndrome.

So far, most of the studies I have described have dealt with comprehension of simple concepts: spatial direction and orientation, body parts, animals, and other concrete objects. But speech also conveys abstract concepts, some of them quite subtle. What parts of the brain are responsible for comprehending the meaning behind proverbs such as "People who live in glass houses shouldn't throw stones" or the moral of stories such as the one about the race between the tortoise and the hare?

Studies of brain-damaged patients suggest that comprehension of the more subtle, figurative aspects of speech involves the right hemisphere in particular (Brownell et al., 1983, 1990). Functional imaging studies confirm these observations. Bottini et al. (1994) had people listen to sentences and judge their plausibility. Some sentences were straightforward and factual. For example, "The old man has a branch as a walking stick" is plausible, whereas "The lady has a bucket as a walking stick" is not. Other sentences presented metaphors, the comprehension of which goes beyond the literal meaning of the words. For example, "The old man had a head full of dead leaves" is plausible, whereas "The old man had a head full of barn doors" is not. The investigators found that judging the metaphors activated parts of the right hemisphere, while judging factual sentences did not. Nichelli et al. (1995) found that judging the moral of Aesop's fables (as opposed to judging more superficial aspects of the stories) also activated additional regions of the right hemisphere.

■ **Repetition: Conduction Aphasia** As we saw earlier in this section, the fact that people with transcortical sensory aphasia can repeat what they hear suggests that there is a direct connection between Wernicke's area and Broca's area—and there is: the arcuate fasciculus ("arch-shaped bundle"). This bundle of axons appears to convey information about the *sounds* of words but not their *meanings*.

autotopagnosia Inability to name body parts or to identify body parts that another person names.

arcuate fasciculus A bundle of axons that connects Wernicke's area with Broca's area; damage causes conduction aphasia.

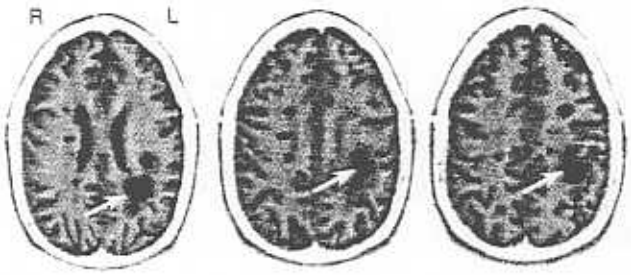


Figure 16.10

MRI scans showing subcortical damage responsible for a case of conduction aphasia. This lesion damaged the arcuate fasciculus, a fiber bundle connecting Wernicke's area and Broca's area. (From Arnett, P. A., Rao, S. M., Hussain, M., Swanson, S. J., and Hammeke, T. A. *Neurology, 1996, 47,* 576–578.)

The best evidence for this conclusion comes from a syndrome known as conduction aphasia, which is produced by damage to the inferior parietal lobe that extends into the subcortical white matter and damages the arcuate fasciculus (Damasio and Damasio, 1980). (See *Figure 16.10.*)

Conduction aphasia is characterized by meaningful, fluent speech; relatively good comprehension; but very poor repetition. For example, the spontaneous speech of patient L.B. (observed by Margolin and Walker, 1981) was excellent; he made very few errors and had no difficulty naming objects. But let us see how patient L.B. performed when he was asked to repeat words:

Examiner: bicycle
Patient: bicycle
Examiner: hippopotamus
Patient: hippopotamus
Examiner: blaynge
Patient: I didn't get it.
Examiner: Okay, some of these won't be real words, they'll just be sounds. Blaynge.
Patient: I'm not . . .
Examiner: blanch
Patient: blanch
Examiner: north
Patient: north
Examiner: rilld
Patient: Nope, I can't say.

You will notice that the patient can repeat individual words (all nouns, in this case) but utterly fails to repeat nonwords. People with conduction aphasia can repeat speech sounds that they hear *only if these sounds have meaning.*

Sometimes, when a person with conduction aphasia is asked to repeat a word, he or she says a word with the same meaning—or at least, one that is related. For example, if the examiner says *house,* the patient may say *home.* If the examiner says *chair,* the patient may say *sit.* One patient made the following response when asked to repeat an entire sentence:

Examiner: The auto's leaking gas tank soiled the roadway.
Patient: The car's tank leaked and made a mess on the street.

The symptoms that are seen in transcortical sensory aphasia and conduction aphasia lead to the conclusion that there are pathways connecting the speech mechanisms of the temporal lobe with those of the frontal lobe. The direct pathway through the arcuate fasciculus simply conveys speech sounds to the frontal lobes. We use this pathway to repeat unfamiliar words—for example, when we are learning a foreign language or a new word in our own language or when we are trying to repeat a nonword such as *blaynge.* The second pathway is indirect and is based on the *meaning* of words, not the sounds they make. When patients with conduction aphasia hear a word or a sentence, the meaning of what they hear evokes some sort of image related to that meaning. (The patient in the second example presumably imagined the sight of an automobile leaking fuel onto the pavement.) They are then able to describe that image, just as they would put their own thoughts into words. Of course, the words they choose may not be the same as the ones used by the person who spoke to them. (See *Figure 16.11.*)

The symptoms of conduction aphasia indicate that the connection between Wernicke's area and Broca's area appears to play an important role in short-term memory of words and speech sounds that have just been heard. Presumably, rehearsal of such information can be accomplished by "talking to ourselves" inside our head without actually having to say anything aloud. Imagining ourselves saying the word activates the region of Broca's area, while imagining that we are hearing it activates the auditory association area of the temporal lobe. These two regions, connected by means of the arcuate fasciculus (which contains axons traveling in *both* directions), circulate information back and forth, keeping the short-term memory alive. Baddeley (1992) refers to this circuit as the *phonological loop.*

Functional imaging studies support this hypothesis. For example, Paulesu, Frith, and Frackowiak (1993) observed activation of Broca's area and a region within the posterior language area while subjects were remembering sets of six consonants. (See *Figure 16.12.*) Fiez et al. (1996) obtained similar results in a task that required subjects to remember pronounceable pseudowords. They found that the subjects

conduction aphasia An aphasia characterized by inability to repeat words that are heard but normal speech and the ability to comprehend the speech of others.

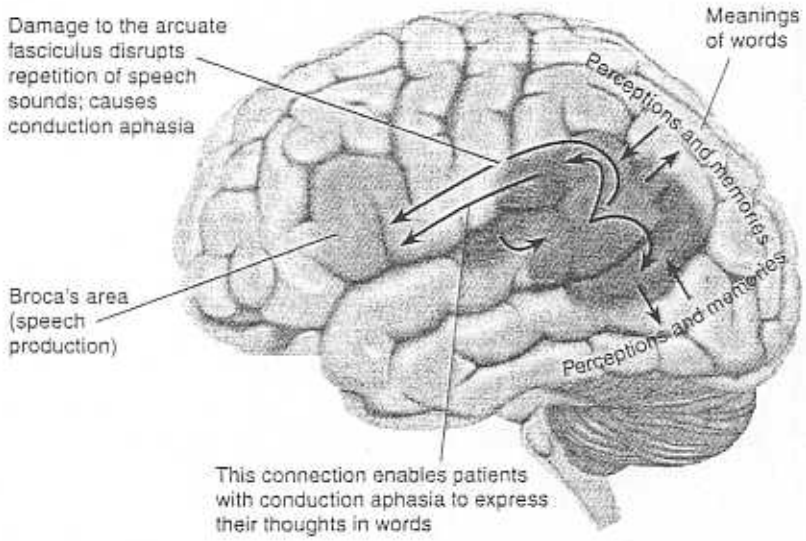


Figure 16.11

A hypothetical explanation of conduction aphasia. A lesion that damages the arcuate fasciculus disrupts transmission of auditory information, but not information related to meaning, to the frontal lobe.

who performed best at this task showed the greatest activation of Broca's area, while subjects who did poorly showed greater activation of the occipital lobe. The subjects read the pseudowords on a screen before the PET scan and then remembered them during the 40 seconds when the machine was performing a scan. Perhaps, reasoned Fiez and her colleagues, the subjects who did poorly were trying to remember what the pseudowords looked like rather than how they sounded, a less effective strategy in such a task.

Memory of Words: Anomic Aphasia

As I already noted, anomia, in one form or other, is a hallmark of aphasia. However, one category of aphasia consists of almost pure anomia, the other symptoms being inconsequential. Speech of patients with anomic aphasia is fluent and grammatical, and their comprehension is excellent, but they have difficulty finding the appropriate words. They often employ **circumlocutions** (literally, "speaking in a roundabout way") to get around missing words. Anomic aphasia is different from Wernicke's aphasia. People with anomic aphasia can understand what other people say, and what they say makes perfect sense, even if they often choose roundabout ways to say it.

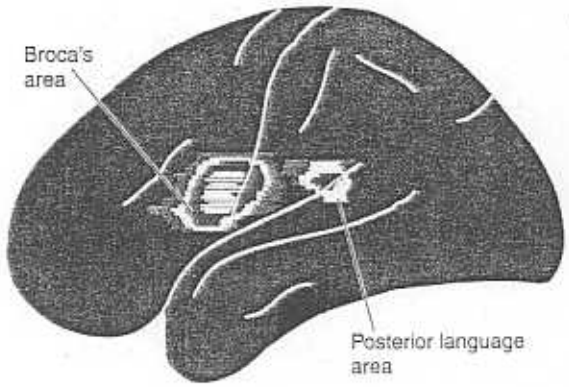


Figure 16.12

The phonological loop. A PET scan showing activation of Broca's area and the auditory association cortex during rehearsal of six consonants.

(Adapted from Baddeley, A. D. *Current Biology*, 1993, *3*, 563–565; after data from Paulesu, E., Frith, C. D., and Frackowiak, R. S. J. *Nature*, 1993, *362*, 342–344.)

The following quotation is from a patient that some colleagues and I studied (Margolin, Marcel, and Carlson, 1985). We asked her to describe the kitchen picture shown earlier, in *Figure 16.1*. Her pauses, which are marked with three dots, indicate word-finding difficulties. In some cases, when she could not find a word, she supplied a definition instead (a form of circumlocution) or went off on a new track. I have added the words in brackets that I think she intended to use.

Examiner: Tell us about that picture.

Patient: It's a woman who has two children, a son and a daughter, and her son is to get into the . . . cupboard in the kitchen to get out [*take*] some . . . cookies out of the [*cookie jar*] . . . that she possibly had made, and consequently he's slipping [*falling*] . . . the wrong direction [*backward*] . . . on the . . . what he's standing on [*stool*], heading to the . . . the cupboard [*floor*] and if he falls backwards he could have some problems [*get hurt*], because that [*the stool*] is off balance.

circumlocution A strategy by which people with anomia find alternative ways to say something when they are unable to think of the most appropriate word.

Anomia has been described as a partial amnesia for words. It can be produced by lesions in either the anterior or posterior regions of the brain, but only posterior lesions produce a *fluent* anomia. The most likely location of lesions that produce anomia without the other symptoms of aphasia, such as comprehension deficits, agrammatism, or difficulties in articulation, is the left temporal or parietal lobe, usually sparing Wernicke's area. In the case of the woman described above, the damage included the middle and inferior temporal gyri, which include an important region of the visual association cortex. Wernicke's area was not damaged.

When my colleagues and I were studying the anomic patient, I was struck by the fact that she seemed to have more difficulty finding nouns than other types of words. I informally tested her ability to name actions by asking her what people shown in a series of pictures were doing. She made almost no errors finding verbs. For example, although she could not say what a boy was holding in his hand, she had no trouble saying that he was *throwing* it. Similarly, she knew that a girl was *climbing* something but could not tell me the name of what she was climbing (a fence). In addition, she had no trouble finding nonvisual adjectives; for example, she could say that lemons tasted *sour*, that ice was *cold*, and that a cat's fur felt *soft*.

For several years I thought that our patient was unique. But more recently, similar patterns of deficits have been reported in the literature. For example, Manning and Campbell (1992) described a patient who had difficulty naming objects but not actions. Some patients have even more specific deficits; Semenza and Zettin (1989) described a patient who had great difficulty with proper nouns (names of people and places). Damasio et al. (1991) studied several patients with similar deficits and concluded that anomia for proper nouns is caused by damage to the temporal pole, whereas anomia for common nouns is caused by damage to the inferior temporal cortex. Damage to both regions causes anomia for both types of nouns. They suggest that the important distinction between the two types of words is that proper nouns are specific to particular individuals (people or places), whereas common nouns apply to *categories*. Presumably, the cortex of the temporal pole is specifically involved with recognition of individuals.

What about the ability to name actions? As we saw, the anomic patient my colleagues and I studied had no trouble with verbs. Neither did the anomic patients studied by Semenza and Zettin (1989), Manning and Campbell (1992), or Damasio et al. (1992). Several studies have found that anomia for verbs (more correctly called *averbia*) is caused by damage to the frontal cortex, in and around Broca's area (Damasio and Tranel, 1993; Daniele et al., 1994). If you think about it, that makes sense. The frontal lobes are devoted to planning, organizing, and executing actions, so it should not surprise us that they are involved in the task of remembering the names of actions.

Several functional imaging studies have confirmed the importance of Broca's area and the region surrounding it in production of verbs (Petersen et al., 1988; Wise et al., 1991; McCarthy et al., 1993; Fiez et al., 1996). In these studies, subjects either read or heard nouns and then had to say (or think to themselves) verbs describing actions appropriate to these nouns. For example, on reading or hearing the noun *hammer*, they might think of the verb *pound*. Figure 16.13 shows a PET scan from people who generated verbs in response to written nouns. The activity produced by simply reading nouns aloud has been subtracted out, leaving only the activity associated with the verb generation process. Presumably, the activity in the temporal lobe represents neural processes involved with comprehension of the nouns, while the activity in the frontal lobe represents the neural processes directly involved with thinking of appropriate actions and the associated verbs. (See *Figure 16.13*.)

A PET study by Martin et al. (1996) investigated the brain regions activated by naming pictures of animals and tools. They found that naming both categories activated the inferior temporal cortex (the ventral stream of visual

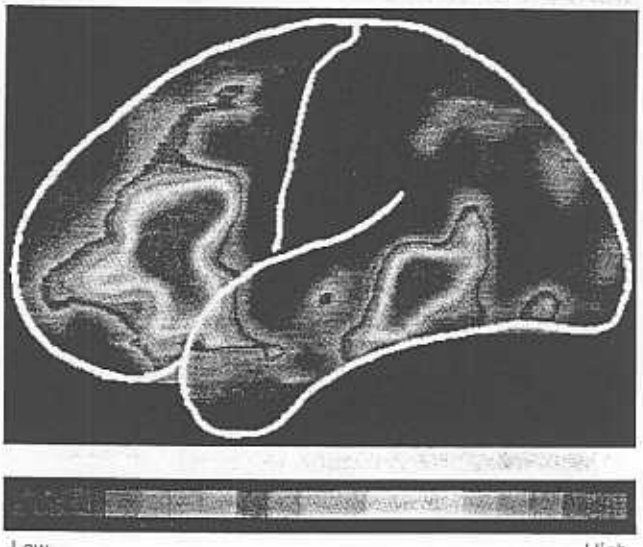


Figure 16.13

A PET scan showing the regions of activation while people thought of verbs that depicted actions appropriate to nouns supplied by the experimenters.

(From Fiez, J. A., Raichle, M. E., Balota, D. A., Tallal, P., and Petersen, S. E. *Cerebral Cortex*, 1996, 6, 1–10.)

processing) and Broca's area. However, animal naming selectively activated the visual association cortex of the medial occipital lobe. Naming tools selectively activated the left middle temporal gyrus and the left premotor cortex—the same region that is activated when people imagine they are making hand movements. (See *Figure 16.14.*)

The picture I have drawn so far suggests that comprehension of speech includes a flow of information from Wernicke's area to the posterior language area to various regions of sensory and motor association cortex, which contain memories that provide meanings to words. Production of spontaneous speech involves the flow of information concerning perceptions and memories from the sensory and motor association cortex to the posterior language area to Broca's area. This model is certainly an oversimplification, but it is a useful starting point in conceptualizing basic mental processes. For example, thinking in words probably involves two-way communication between the speech areas and surrounding association cortex (and subcortical regions such as the hippocampus, of course).

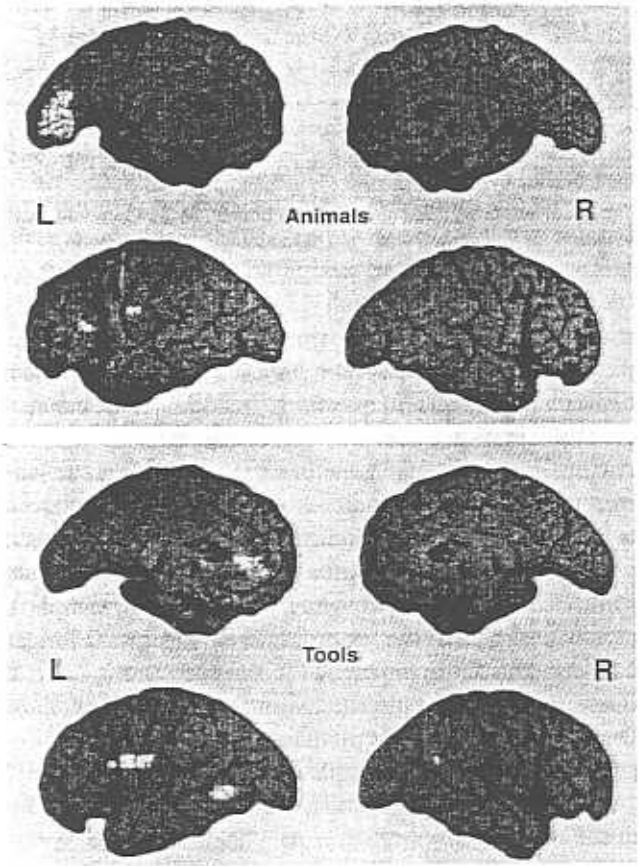


Figure 16.14

PET scans showing the regions of activation when people named pictures of animals (top) or tools (bottom).

(From Martin, A., Wiggs, C. L., Ungerleider, L. G., and Haxby, J. V. *Nature*, 1996, *379*, 649–652.)

Aphasia in Deaf People

So far, I have restricted my discussion to brain mechanisms of spoken and written language. But communication among members of the Deaf community involves another medium: sign language. Sign language is expressed manually, by movements of the hands. Sign language is *not* English; nor is it French, or Spanish, or Chinese. The most common sign language in North America is ASL—American Sign Language. ASL is a full-fledged language, having signs for nouns, verbs, adjectives, adverbs, and all the other parts of speech contained in oral languages. People can converse rapidly and efficiently by means of sign language, can tell jokes, and can even make puns based on the similarity between signs. They can also use their language ability to think in words.

Some researchers believe that in the history of our species, sign language preceded spoken language—that our ancestors began using gestures to communicate before they switched to speech. You may recall (from Chapter 6) that Rizzolatti and his colleagues (Gallese et al., 1996; Rizzolatti et al., 1996) found an area of the rostral part of the ventral premotor cortex in monkeys that became active whenever the monkeys either *saw* or *performed* various grasping, holding, or manipulating movements. Presumably, these *mirror neurons* would play an important role in learning to mimic another animal's hand movements. Indeed, they might have been involved in the development of hand gestures used for communication, and they undoubtedly are used by deaf people when they communicate by sign language.

The grammar of ASL is based on its visual, spatial nature. For example, if a person makes the sign for *John* in one place and later makes the sign for *Mary* in another place, she can place her hand in the *John* location and move it toward the *Mary* location while making the sign for *love*. As you undoubtedly figured out for yourself, she is saying, "John loves Mary." Signers can also modify the meaning of signs through facial expressions or the speed and vigor with which they make a sign. Thus, many of the prepositions, adjectives, and adverbs found in spoken languages do not require specific words in ASL. The fact that signed languages are based on three-dimensional hand and arm movements accompanied by facial expressions means that their grammars are very different from those of spoken languages. Therefore, a word-for-word translation from a spoken language to a signed language (or vice versa) is impossible.

The fact that the grammar of ASL is spatial suggests that aphasic disorders in deaf people who use sign language

might be caused by lesions of the right hemisphere, which is primarily involved in spatial perception and memory. However, all the cases of deaf people with aphasia for signs reported in the literature so far have involved lesions of the left hemisphere (Hickok, Bellugi, and Klima, 1996). Therefore, sign language, like auditory and written language, appears to rely primarily on the left hemisphere for comprehension and expression.

Lipreading

When deaf people communicate with hearing people who do not know sign language, they must do so in writing or by reading the hearing people's lips. What most people do not realize is that even hearing people sometimes use visual information about movements of a speaker's mouth to assist their comprehension of speech. Many studies have shown that in noisy environments, speech comprehension is much improved when the listener can watch the speaker's lips. People are not usually aware of this phenomenon, although they become acutely aware of a desynchronization between lip movements and speech sounds, such as that produced by a movie with a faulty sound track. Two recent studies have investigated the neural basis of lipreading. Campbell, De Gelder, and De Haan (1996) found that although the right hemisphere is more important in judging the identity of faces, the left hemisphere is more important in judging the positions that the lips take when a person is producing different vowel sounds. (For example, the lips describe a circle when we say /oo/, but the corners of the lips are drawn apart when we say /ee/.)

Calvert et al. (1997) obtained functional MRI scans from people who were watching a silent videotape of speakers slowly saying a list of numbers. The subjects had to repeat silently to themselves the numbers they saw being mouthed. The regions of the brain that were activated by performance of this lipreading task included parts of the visual association cortex (including area V5, which is devoted to the analysis of movement), the angular gyrus of the parietal lobe, and the primary auditory cortex and auditory association cortex of the temporal lobe—especially in the left hemisphere. (See *Figure 16.15.*) A similar pattern of activation was seen when subjects watched videotapes of people saying pronounceable pseudowords but not when they watched people making movements of their jaws and lower face with the lips closed.

Prosody: Rhythm, Tone, and Emphasis in Speech

When we speak, we do not merely utter words. Our speech has a regular rhythm and cadence; we give some words

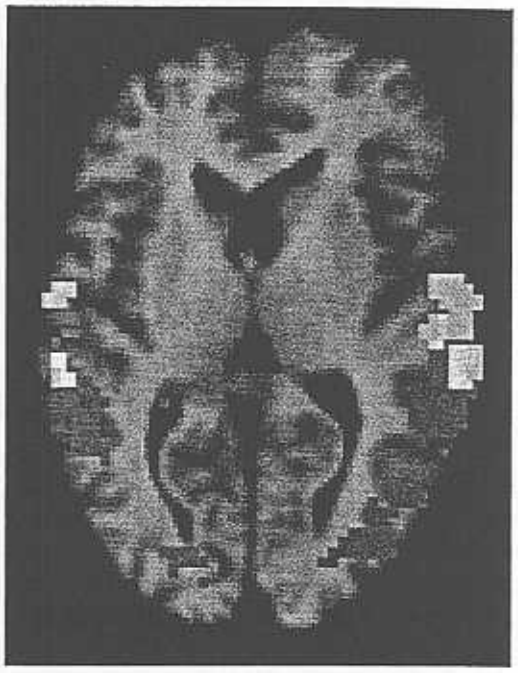


Figure 16.15

A PET scan showing regions of activation that accompanied auditory speech perception (blue), silent lipreading (magenta), and their overlap (yellow).

(From Calvert, G. A., Bullmore, E. T., Brammer, M. J., Campbell, R., Williams, S. C. R., McGuire, P. K., Woodruff, P. W. R., Iversen, S. D., and David, A. S. *Science*, 1997, *276*, 593–596.)

stress (that is, we pronounce them louder), and we vary the pitch of our voice to indicate phrasing and to distinguish between assertions and questions. In addition, we can impart information about our emotional state through the rhythm, emphasis, and tone of our speech. These rhythmic, emphatic, and melodic aspects of speech are referred to as prosody. The importance of these aspects of speech is illustrated by our use of punctuation symbols to indicate some elements of prosody when we write. For example, a comma indicates a short pause; a period indicates a longer one with an accompanying fall in the pitch of the voice; a question mark indicates a pause and a rise in the pitch of the voice; an exclamation mark indicates that the words are articulated with special emphasis; and so on.

The prosody of people with fluent aphasias, caused by posterior lesions, sounds normal. Their speech is rhythmical, pausing after phrases and sentences, and has a

prosody The use of changes in intonation and emphasis to convey meaning in speech besides that specified by the particular words; an important means of communication of emotion.

melodic line. Even when the speech of a person with severe Wernicke's aphasia makes no sense, the prosody sounds normal. As Goodglass and Kaplan (1972) note, a person with Wernicke's aphasia may "sound like a normal speaker at a distance, because of his fluency and normal melodic contour of his speech." (Up close, of course, we hear the speech clearly enough to realize that it is meaningless.) In contrast, just as the lesions that produce Broca's aphasia destroy grammar, they also severely disrupt prosody. In patients with Broca's aphasia, articulation is so labored and words are uttered so slowly that there is little opportunity for the patient to demonstrate any rhythmic elements; and because of the relative lack of function words, there is little variation in stress or pitch of voice.

Evidence from studies of normal people and patients with brain lesions suggests that prosody is a special function of the right hemisphere. This function is undoubtedly related to the more general role of this hemisphere in musical skills and the expression and recognition of emotions: Production of prosody is rather like singing, and prosody often serves as a vehicle for conveying emotion.

Weintraub, Mesulam, and Kramer (1981) tested the ability of patients with right-hemisphere damage to recognize and express prosodic elements of speech. In one experiment they showed their subjects two pictures, named one of the pictures, and asked the subjects to point to the appropriate one. For example, they showed the subjects a picture of a greenhouse and a house that was painted green. In speech we distinguish between *greenhouse* and *green house* by stress: *GREEN house* means the former, and *GREEN HOUSE* (syllables equally stressed) means the latter. In a second experiment, Weintraub and her colleagues tested the subjects' ability simply to detect differences in prosody. They presented pairs of sentences and asked the subjects whether they were the same or different. The pairs of sentences either were identical or differed in terms of intonation (for example, *Margo plays the piano?* and *Margo plays the piano*) or location of stress (for example, *STEVE drives the car* and *Steve drives the CAR*). The patients with right-hemisphere lesions (but not control subjects) performed poorly on both of these tasks. Thus, they showed a deficit in prosodic comprehension.

To test production, the investigators presented two written sentences and asked a question about them. For example, they presented the following pair:

> The man walked to the grocery store.
> The woman rode to the shoe store.

The subjects were instructed to answer questions by reading one of the sentences. Try this one yourself. Read the question below and then read aloud the sentence (above) that answers it.

> Who walked to the grocery store, the man or the woman?

The question asserts that someone walked to the grocery store but asks who that person was. When answering a question like this, people normally stress the requested item of information—in this case they say, "The *man* walked to the grocery store." However, Weintraub and her colleagues found that although patients with right-hemisphere brain damage chose the correct sentence, they either failed to stress a word or stressed the wrong one. Thus, the right hemisphere plays a role in production as well as perception of prosody.

Two regions of the brain are especially important in understanding and producing speech. Broca's area, in the frontal lobe just rostral to the region of the primary motor cortex that controls the muscles of speech, is involved with speech production. This region contains memories of the sequences of muscular movements that produce words, each of which is connected with its auditory counterpart in the posterior part of the brain. Broca's aphasia—which is caused by damage to Broca's area, adjacent regions of the frontal cortex, and underlying white matter—consists of varying degrees of agrammatism, anomia, and articulation difficulties.

Wernicke's area, in the posterior superior temporal lobe, is involved with speech perception. The region just adjacent to Wernicke's area, which I have called the posterior language area, is necessary for speech comprehension and the translation of thoughts into words. Presumably, Wernicke's area contains memories of the sounds of words, each of which is connected through the posterior language area with memories about the properties of the things the words denote. Damage restricted to Wernicke's area causes pure word deafness—loss of the ability to understand speech but intact speech production, reading, and writing. Wernicke's aphasia, caused by damage to Wernicke's area and the posterior language area, consists of poor speech comprehension, poor repetition, and production of fluent, meaningless speech. Transcortical sensory aphasia, caused by damage to the posterior speech area, consists of poor speech comprehension and production, but the patients can repeat what they hear. Thus, the symptoms of Wernicke's area consist of those of transcortical sensory aphasia plus those of pure word deafness. The fact that people with transcortical sensory aphasia can repeat words they cannot understand suggests that there is a direct connection between Wernicke's area and

Broca's area. Indeed, there is: the arcuate fasciculus. Damage to this bundle of axons produces conduction aphasia: disruption of the ability to repeat exactly what was heard without disruption of the ability to comprehend speech.

The meanings of words are our memories of objects, actions, and other concepts associated with them. These meanings are memories and are stored in the association cortex, not in the speech areas themselves. Pure anomia, caused by damage to the temporal or parietal lobes, consists of difficulty in word finding—particularly in naming objects. Some patients have a specific difficulty with proper nouns, while others have difficulty with common nouns; most patients have little difficulty with verbs. Damage to Broca's area and surrounding regions disrupts the ability to name actions—to think of appropriate verbs. Brain damage can also disrupt the "definitions" as well as the "entries" in the mental dictionary; damage to specific regions of the association cortex effectively erases some categories of the *meanings* of words.

Prosody includes changes in intonation, rhythm, and stress that add meaning, especially emotional meaning, to the sentences that we speak. The neural mechanisms that control the prosodic elements of speech appear to be in the right hemisphere.

Because so many terms and symptoms were described in this section, I have provided a table that summarizes them. (See *Table 16.1.*)

Disorders of Reading and Writing

Reading and writing are closely related to listening and talking; thus, oral and written language abilities have many brain mechanisms in common. This section discusses the neural basis of reading and writing disorders. As you will see, the study of these disorders has provided us with some useful and interesting information.

Relation to Aphasia

The reading and writing skills of people with aphasia almost always resemble their speaking and comprehending abilities. For example, patients with Wernicke's aphasia have as much difficulty reading and writing as they do speaking and understanding speech. Patients with Broca's aphasia comprehend what they read about as well as they can understand speech, but their reading aloud is poor, of course. If their speech is agrammatical, so is their writing; and to the extent that they fail to comprehend grammar when listening to speech, they fail to do so when reading. Patients with conduction aphasia generally have some difficulty reading; and when they read aloud, they often make semantic paraphasias (saying synonyms for some of the

Table 16.1
Aphasic Syndromes Produced by Brain Damage

Disorder	Areas of lesion	Spontaneous speech	Comprehension	Repetition	Naming
Wernicke's aphasia	Posterior portion of superior temporal gyrus (Wernicke's area) and posterior language area	Fluent	Poor	Poor	Poor
Pure word deafness	Wernicke's area or its connection with primary auditory cortex	Fluent	Poor	Poor	Good
Broca's aphasia	Frontal cortex rostral to base of primary motor cortex (Broca's area)	Nonfluent	Good	Poor[a]	Poor
Conduction aphasia	White matter beneath parietal lobe superior to lateral fissure (arcuate fasciculus)	Fluent	Good	Poor	Good
Anomic aphasia	Various parts of parietal and temporal lobes	Fluent	Good	Good	Poor
Transcortical sensory aphasia	Posterior language area	Fluent	Poor	Good	Poor

[a]May be better than spontaneous speech.

words they read), just as they do when attempting to repeat what they hear. Depending on the location of the lesion, some patients with transcortical sensory aphasia may read aloud accurately but fail to comprehend what they read.

There are a few exceptions to this general rule. For example, Semenza, Cipolotti, and Denes (1992) studied a patient with a severe fluent aphasia. Although she could not understand the speech of others, she could read. She clearly understood what she was reading, because she could follow written instructions. And although her spontaneous speech was meaningless and she could not say the names of objects, she could write their names, and she could read aloud. Clearly, her comprehension and production of oral language were very different from her comprehension and production of written language. Although cases like this one are rare, they do indicate that our verbal abilities make use of a large number of individual neural modules. Reading and writing undoubtedly share many modules with oral comprehension and production, but some modules are devoted to particular methods of communication.

Pure Alexia

Dejerine (1892) described a remarkable syndrome, which we now call pure alexia, or sometimes *pure word blindness* or *alexia without agraphia*. His patient had a lesion in the visual cortex of the left occipital lobe and the posterior end of the corpus callosum. The patient could still write, although he had lost the ability to read. In fact, if he was shown some of his own writing, he could not read it.

Several years ago, some colleagues and I studied a man with pure alexia who discovered his ability to write in an interesting way. A few months after he sustained a head injury that caused his brain damage, he and his wife were watching a service person repair their washing machine. The patient wanted to say something privately to his wife, so he picked up a pad of paper and jotted a note. As he was handing it to her, they suddenly realized with amazement that although he could not read, he was able to write. His wife brought the note to their neurologist, who asked the patient to read it. Although he remembered the gist of the message, he could not read the words. (See *Figure 16.16* for another example.)

Although patients with pure alexia cannot read, they can recognize words that are spelled aloud to them; therefore, they have not lost their memories of the spellings of words. Pure alexia is obviously a perceptual disorder; it is similar to pure word deafness, except that the patient has difficulty with visual input, not auditory input. The disorder is caused by lesions that prevent visual information from reaching the extrastriate cortex of the left hemisphere

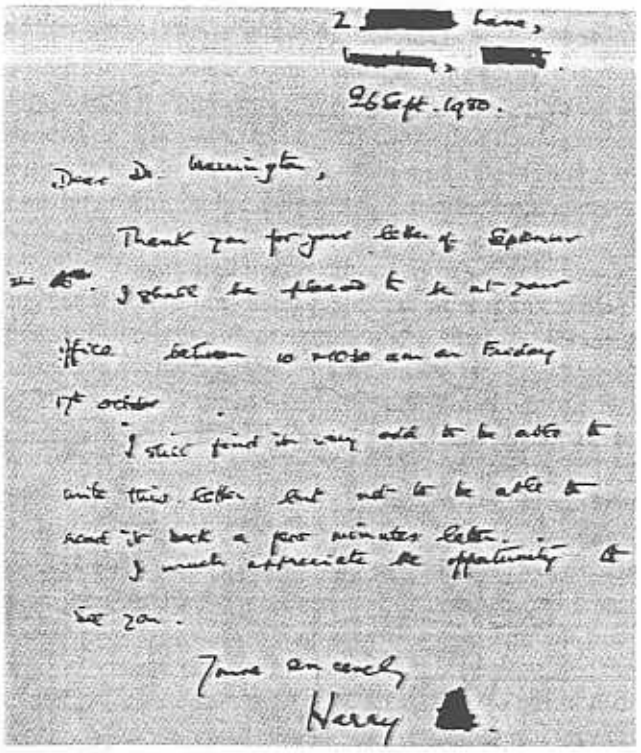


Figure 16.16

A letter written to Dr. Elizabeth Warrington by a patient with pure alexia. The letter reads as follows: "Dear Dr. Warrington, Thank you for your letter of September 16th. I shall be pleased to be at your office between 10 & 10:30 am on Friday 17th october. I still find it very odd to be able to write this letter but not to be able to read it back a few minutes later. I much appreciate the opportunity to see you. Yours sincerely, Harry X.

(From McCarthy, R. A., and Warrington, E. K. *Cognitive Neuropsychology: A Clinical Introduction*. San Diego: Academic Press, 1990. Reprinted with permission.)

(Damasio and Damasio, 1983, 1986). Figure 16.17 explains why Dejerine's original patient could not read. The first diagram shows the pathway that visual information would take if a person had damage *only to the left primary visual cortex*. In this case the person's right visual field would be blind; he or she would see nothing to the right of the fixation point. But people with this disorder can read. Their only problem is that they must look to the right of each word so that they can see all of it, which means that they read somewhat more slowly than someone with full vision.

Let us trace the flow of visual information for a person with this brain damage. Information from the left side of the visual field is transmitted to the right striate cortex

pure alexia Loss of the ability to read without loss of the ability to write; produced by brain damage.

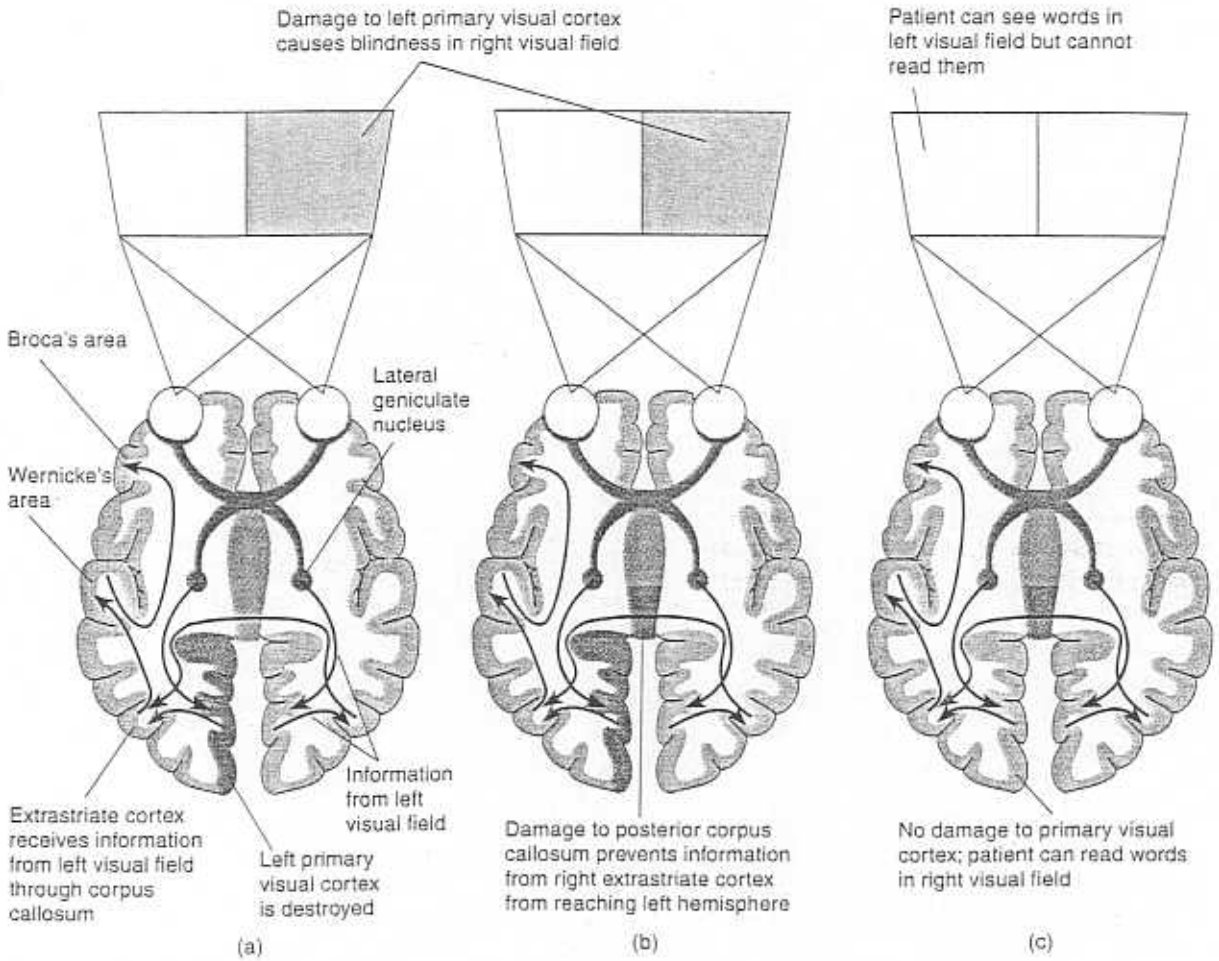


Figure 16.17

Pure alexia. Red arrows indicate the flow of information that has been interrupted by brain damage. (a) The route followed by information as a person with damage to the left primary visual cortex reads aloud. (b) Additional damage to the posterior corpus callosum interrupts the flow of information and produces pure alexia. (c) Damage to the posterior corpus callosum alone produces left hemialexia: an inability to read words presented in the left visual field.

(primary visual cortex) and then to the lingual and fusiform gyri—a region of extrastriate cortex involved in the recognition of written text. From there the information crosses the posterior corpus callosum and is transmitted to the left extrastriate cortex and then to speech mechanisms located in the left frontal lobe. Thus, the person can read the words aloud. (See *Figure 16.17a.*)

The second diagram shows Dejerine's patient. Notice how the additional lesion of the corpus callosum prevents visual information concerning written text from reaching the posterior left hemisphere. Without this information the patient cannot read. (See *Figure 16.17b.*)

If the model presented in Figure 16.17 is correct, we would predict that a lesion that is restricted to the posterior corpus callosum should cause a left *hemialexia*—an inability to read words presented entirely in the left visual field. In fact, Binder et al. (1992) studied a patient who had precisely that lesion and precisely that deficit. Their patient, a thirty-year-old woman, was operated on to remove a vascular malformation in her brain. While removing the malformation, the surgeons were obliged to damage the posterior end of the corpus callosum. When Binder and his colleagues tested the woman later, they found that she often made errors in reading that involved the left side of words—the part that would fall into her "word blind" visual field. For example, she read *car* as *ear* and *seat* as *heat*. In addition, she could not read simple three-letter words presented to her left visual field. (See *Figure 16.17c.*)

I must note that the diagrams shown in Figure 16.17 are as simple and schematic as possible. They illustrate only the

pathway involved in seeing a word and pronouncing it, and they ignore neural structures that would be involved in understanding its meaning. As we will see later in this chapter, evidence from patients with brain lesions indicates that seeing and pronouncing words can take place independently of understanding them. Thus, although the diagrams are simplified, they are not unreasonable, given what we know about the neural components of the reading process.

Presumably, some parts of the visual association cortex are involved in perceiving written words. The fact that damage to the posterior end of the corpus callosum disrupts the exchange of information concerning the shape of words suggests that the extrastriate cortex may be responsible for this analysis. Petersen et al. (1990) obtained results that support this suggestion. The investigators used a PET scanner to measure regional cerebral blood flow while presenting subjects with four types of visual stimuli: unfamiliar letterlike forms, strings of consonants, pronounceable nonwords, and real words. They found that one region of the medial extrastriate cortex was activated only by pronounceable nonwords or real words. Their finding suggests that this region, which includes the fusiform and lingual gyri, plays a role in recognition of familiar combinations of letters. (See *Figure 16.18.*)

You will recall that writing is not the only form of visible language; deaf people can communicate by means of sign language just as well as hearing people can communicate by means of spoken language. Hickok et al. (1995) reported on a case of "sign blindness" caused by damage similar to that which causes pure alexia. The patient, a right-handed deaf woman, sustained a stroke that damaged her left occipital lobe and the posterior corpus callosum. The lesion did not impair her ability to sign in coherent sentences, so she did not have a Wernicke-like aphasia. However, she could no longer understand other people's sign language, and she lost her ability to read. She had some ability to comprehend single signs (corresponding to single words), but she could not comprehend signed sentences.

You will recall from Chapter 6 that visual agnosia is a perceptual deficit in which people with bilateral damage to the visual association cortex cannot recognize objects by sight. Patients with pure alexia do *not* have visual agnosia; they can recognize objects and supply their names. Similarly, people with visual agnosia can still read. Thus, the perceptual analysis of objects and words requires different brain mechanisms. This fact is both interesting and puzzling. Certainly, the ability to read cannot have shaped the evolution of the human brain, because the invention of writing is only a few thousand years old, and until very recently, the vast majority of the world's population was illiterate. Thus, reading and object recognition use brain mechanisms that undoubtedly existed even before the invention of writing. As Patterson and Ralph (1999) conclude, natural selection has provided us with brain mechanisms for visual perception, speaking, and comprehending spoken language. Our ability to recognize words and understand them undoubtedly utilizes these mechanisms.

Presumably, once we perceive and identify a written word, we can understand its meaning (and the meaning of sentences composed of groups of words) by using the same brain mechanisms used to understand speech. The question is, what is the nature of the perceptual mechanisms used to identify written words? Why can a person with damage to one part of the brain recognize objects but not words, while a person with damage to another part of the brain can recognize words but not objects? Behrmann, Nelson, and Sekuler (1998) suggest that recognition of real objects involves several different types of cues: depth, color, luminance, shadow, surface texture, and so on. On the other hand, recognition of written words involves detection of a few specific features—primarily edges, line lengths, and angles, which are analyzed by the extrastriate cortex. In other words, there is more redundancy in the perception of objects, and loss of some of the information will still permit a person to recognize them reasonably well.

Careful study of the perceptual abilities of people with pure alexia has found evidence for other perceptual deficits

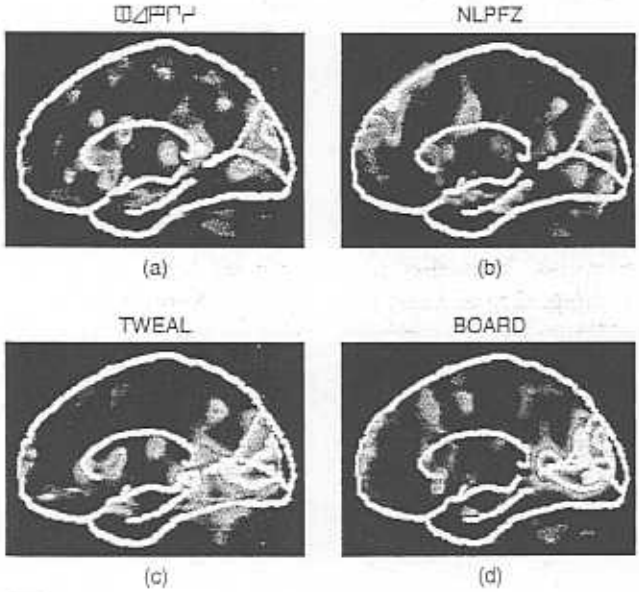


Figure 16.18

PET scans of the medial surface of the brains of subjects who read (a) letterlike forms, (b) strings of consonants, (c) pronounceable nonwords, or (d) real words.

(From Petersen, S. E., Fox, P. T., Snyder, A. Z., and Raichle, M. E. *Science*, 1990, *249*, 1041–1044. Reprinted with permission.)

besides word blindness. For example, two studies found that musicians with pure alexia also lost the ability to read music (Horikoshi et al., 1997; Beversdorf and Heilman, 1998). The patient studied by Horikoshi and his colleagues was also unable to recognize visual symbols such as road signs. Visual recognition of words, musical notes, and other symbols presumably involves similar types of perceptual analysis.

Toward an Understanding of Reading

Most investigators believe that reading involves at least two different processes: direct recognition of the word as a whole and sounding it out letter by letter. When we see a familiar word, we normally recognize it by its shape and pronounce it—a process known as **whole-word reading**. (With very long words we might instead perceive segments of several letters each.) The second method, which we use for unfamiliar words, requires recognition of individual letters and knowledge of the sounds they make. This process is known as **phonetic reading**.

Evidence for our ability to sound out words is easy to obtain. In fact, you can prove to yourself that phonetic reading exists by trying to read the following words:

glab trisk chint

Well, as you could see, they are not really words, but I doubt that you had trouble pronouncing them. Obviously, you did not *recognize* them, because you probably never saw them before. Therefore, you had to use what you know about the sounds that are represented by particular letters (or groups of letters, such as *ch*) to figure out how to pronounce the words.

The best evidence that proves that people can read words without sounding them out, using the whole-word method, comes from studies of patients with acquired dyslexias. *Dyslexia* means "faulty reading." *Acquired* dyslexias are those caused by damage to the brains of people who already know how to read. In contrast, *developmental* dyslexias refer to reading difficulties that become apparent when children are learning to read. Developmental dyslexias, which may involve anomalies in brain circuitry, are discussed in a later section.

Figure 16.19 illustrates some elements of the reading processes. The diagram is an oversimplification of a very complex process, but it helps to organize some of the facts that investigators have obtained. It considers only reading and pronouncing single words, not understanding the meaning of text. When we see a familiar word, we normally recognize it as a whole and pronounce it. If we see an unfamiliar word or a pronounceable nonword, we must try to read it phonetically. (See *Figure 16.19*.)

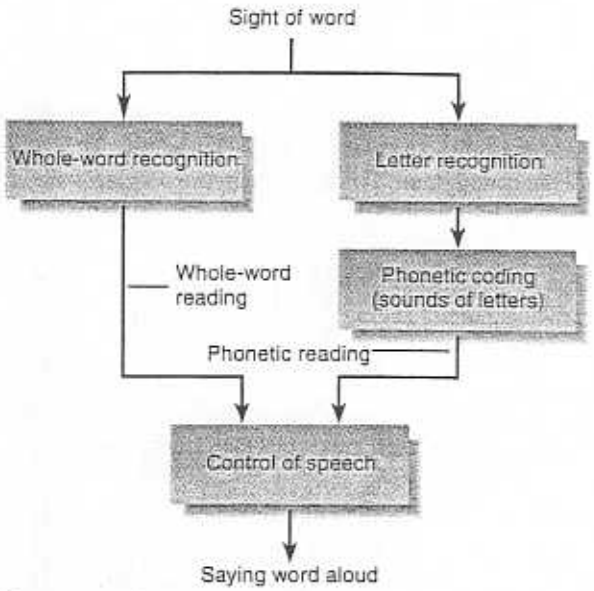


Figure 16.19

A simplified model of the reading process, showing whole-word and phonetic reading. Whole-word reading is used for most familiar words; phonetic reading is used for unfamiliar words and for nonwords such as *glab, trisk,* or *chint.*

Although investigators have reported several types of acquired dyslexias, I will mention five of them here. **Surface dyslexia** is a deficit in whole-word reading, usually caused by a lesion of the left lateral temporal lobe (Marshall and Newcombe, 1973; McCarthy and Warrington, 1990). The term *surface* reflects the fact that people with this disorder make errors related to the visual appearance of the words and to pronunciation rules, not to the meaning of the words, which is metaphorically "deeper" than the appearance. Because patients with surface dyslexia have difficulty recognizing words as a whole, they are obliged to sound them out. Thus, they can easily read words with regular spelling, such as *hand, table,* or *chin*. However, they have difficulty reading words with irregular spelling, such as *sew, pint,* and *yacht*. In fact, they may read these words as *sue,*

whole-word reading Reading by recognizing a word as a whole; "sight reading."

phonetic reading Reading by decoding the phonetic significance of letter strings; "sound reading."

surface dyslexia A reading disorder in which a person can read words phonetically but has difficulty reading irregularly spelled words by the whole-word method.

phonological dyslexia A reading disorder in which a person can read familiar words but has difficulty reading unfamiliar words or pronounceable nonwords.

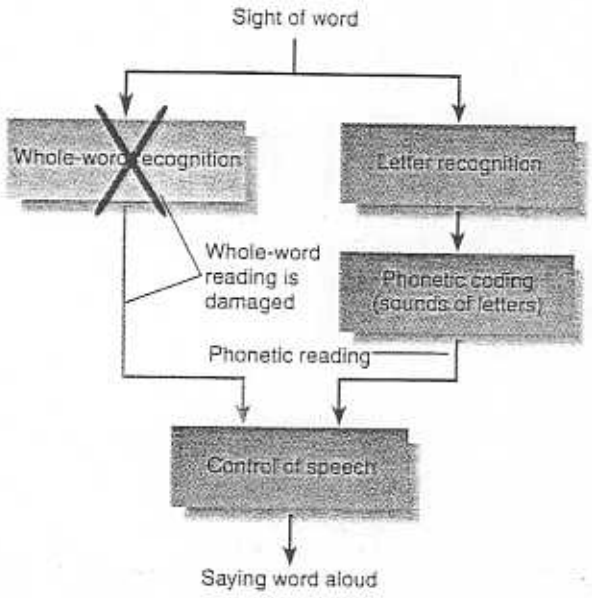


Figure 16.20

A hypothetical explanation of surface dyslexia. Whole-word reading is damaged; only phonetic reading remains.

pinnt, and *yatchet*. They have no difficulty reading pronounceable nonwords, such as *glab*, *trisk*, and *chint*. Because people with surface dyslexia cannot recognize whole words by their appearance, they must, in effect, listen to their own pronunciation to understand what they are reading. If they read the word *pint* and pronounce it *pinnt*, they will say that it is not an English word (which it is not, pronounced that way). If the word is one member of a homophone, it will be impossible to understand it unless it is read in the context of a sentence. For example, if you hear the single word "pair," without additional information, you cannot know whether the speaker is referring to *pair*, *pear*, or *pare*. Thus, a patient with surface dyslexia who reads the word *pair* might say, " . . . it could be two of a kind, apples and . . . or what you do with your fingernails" (Gurd and Marshall, 1993, p. 594). (See *Figure 16.20.*)

Patients with **phonological dyslexia** have the opposite problem; they can read by the whole-word method but cannot sound words out. Thus, they can read words that they are already familiar with but have great difficulty figuring out how to read unfamiliar words or pronounceable nonwords (Beauvois and Dérouesné, 1979; Dérouesné and Beauvois, 1979). (In this context, *phonology*—loosely translated as "laws of sound"—refers to the relation between letters and the sounds they represent.) People with phonological dyslexia may be excellent readers if they had already acquired a good reading vocabulary before their brain damage occurred.

Phonological dyslexia provides further evidence that whole-word reading and phonological reading involve different brain mechanisms. Phonological reading, which is the only way we can read nonwords or words we have not yet learned, entails some sort of letter-to-sound decoding. Obviously, phonological reading of English requires more than decoding of the sounds produced by single letters, because, for example, some sounds are transcribed as two-letter sequences (such as *th* or *sh*) and the addition of the letter *e* to the end of a word lengthens an internal vowel (*can* becomes *cane*). (See *Figure 16.21.*)

Phonological dyslexia is usually caused by damage to the left frontal lobe (Price, 1998; Fiez and Petersen, 1998). A PET study by Fiez et al. (1999) found that phonological reading activated Broca's area and the left insular region. They suggest that "phonological" reading may actually involve articulation—that we sound out words not so much by "hearing" them in our heads as by feeling ourselves pronounce them silently to ourselves. Of course, *both* processes could be taking place simultaneously.

The Japanese language provides a particularly interesting distinction between phonetic and whole-word reading. The Japanese language makes use of two kinds of written symbols. *Kanji* symbols are pictographs, adopted from the Chinese language (although they are pronounced as Japanese words). Thus, they represent concepts by means of visual symbols but do not provide a guide to their pronunciation. Reading words expressed in kanji symbols is

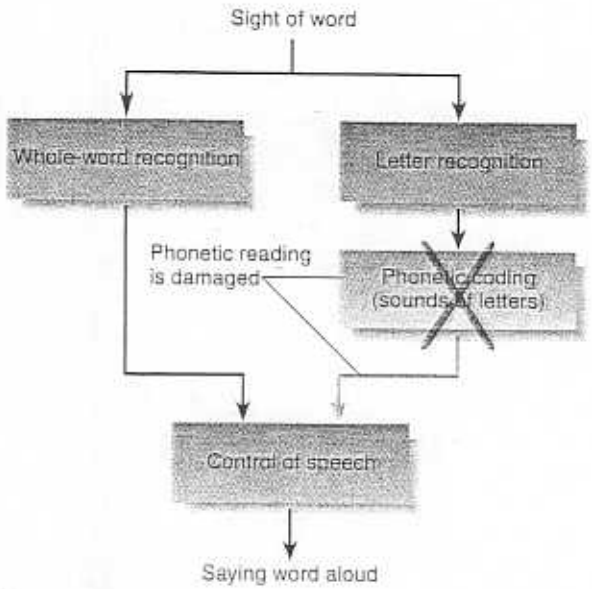


Figure 16.21

A hypothetical explanation of phonological dyslexia. Phonetic reading is damaged; only whole-word reading remains.

analogous, then, to whole-word reading. *Kana* symbols are phonetic representations of syllables; thus, they encode acoustical information. These symbols are used primarily to represent foreign words or Japanese words that the average reader would be unlikely to recognize if they were represented by their kanji symbols. Reading words expressed in kana symbols is obviously phonetic.

Studies of Japanese people with localized brain damage have shown that the reading of kana and kanji symbols involves different brain mechanisms (Iwata, 1984; Sakurai et al., 1994). Difficulty reading kanji symbols is analogous to surface dyslexia, whereas difficulty reading kana symbols is analogous to phonological dyslexia.

What would happen if individuals sustained brain damage that did not make them blind but destroyed their ability to read words by either the whole-word or phonetic methods? Would they be *completely* unable to read? The answer is no—not quite. They would have a disorder known as **word-form dyslexia** or **spelling dyslexia** (Warrington and Shallice, 1980). Although patients with word-form dyslexia cannot either recognize words as a whole or sound them out phonetically, they can still recognize individual letters and can read the words if they are permitted to name the letters, one at a time. Thus, they read very slowly, taking more time with longer words. As you might expect, patients with word-form dyslexia can identify words that someone else spells aloud, just as they can recognize their own oral spelling. Sometimes, the deficit is so severe that patients have difficulty identifying individual letters, in which case they make mistakes in spelling that prevent them from reading test words. For example, a patient studied by Patterson and Kay (1980) was shown the word *men* and said, "h, e, n, hen." (See *Figure 16.22.*)

As we saw earlier in this chapter, recognizing a spoken word is different from understanding it. For example, patients with transcortical sensory aphasia can repeat what is said to them even though they show no signs of understanding what they hear or say. The same is true for reading. Direct dyslexia resembles transcortical sensory aphasia, except that the words in question are written, not spoken (Schwartz, Marin, and Saffran, 1979; Lytton and Brust, 1989). Patients with direct dyslexia are able to read aloud *even though they cannot understand the words they are saying.* After sustaining a stroke that damaged his left frontal and temporal lobes, Lytton and Brust's patient lost the ability to communicate verbally; his speech was meaningless, and he was unable to comprehend what other people said to him. However, he could read words with which he was already familiar. He could *not* read pronounceable nonwords; therefore, he had lost the ability to read phonetically. His comprehension deficit seemed complete; when the investigators presented him with a word and several pictures, one of which corresponded to the word, he read the word correctly but had no idea what picture went with it.

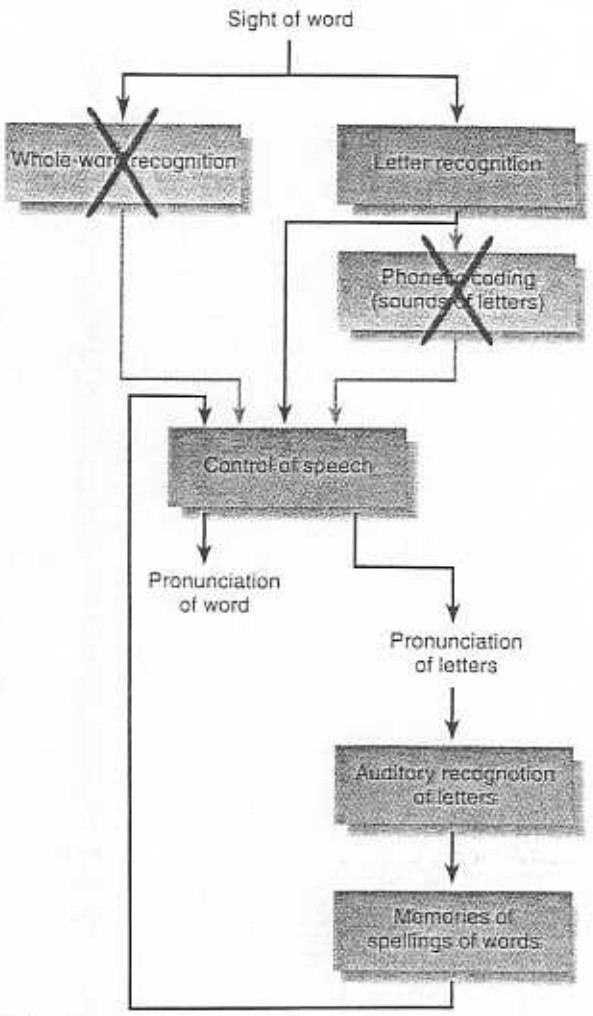


Figure 16.22
A hypothetical explanation of spelling dyslexia. The patient pronounces the letters, recognizes the words, and then says them.

Several investigators have reported a deficit opposite to that of direct dyslexia. People with this unnamed disorder (we could call it *comprehension without reading*) show some comprehension of words that they cannot read (Margolin, Marcel, and Carlson, 1985). Our patient, R.F., sustained a head injury in an automobile accident that destroyed much of her left temporal lobe and part of the an-

word-form dyslexia A disorder in which a person can read a word only after spelling out the individual letters.

spelling dyslexia An alternative name for word-form dyslexia.

direct dyslexia A language disorder caused by brain damage in which the person can read words aloud without understanding them.

terior occipital lobe. She had a classic case of anomic aphasia—in fact, I quoted her in the section on that topic earlier in this chapter. Although her speech was fluent and she could repeat whatever we said to her, she could not name most common objects, nor could she read most words. Nevertheless, she could match pictures of *objects she could not name* with *words she could not read*. For example, when we showed her the picture and words that appear in Figure 16.23, she immediately pointed to the correct word, *flag*, even though she could not name the object or read any of the words. (See *Figure 16.23.*)

Patient R.F. was utterly unable to read words phonetically. However, the fact that she could match words with pictures indicates that she could still *perceive* them by the whole-word method. This fact was made especially apparent one day when she was trying (without success) to read some words that I had typed. Suddenly, she said, "Hey! You spelled this one wrong." I looked at the word and realized that she was right; I had. But even though she saw that the word was misspelled, she still could not say what it was, even when she tried very hard to sound it out. That evening I made up a list of eighty pairs of words, one spelled correctly and the other incorrectly. The next day she was able to go through the list quickly and easily, correctly identifying 95 percent of the misspelled words. She was able to *read* only five of them.

Toward an Understanding of Writing

Writing depends on knowledge of the words that are to be used, along with the proper grammatical structure of the sentences they are to form. Thus, if a patient is unable to express himself or herself by speech, we should not be surprised to see a writing disturbance as well.

One type of writing disorder involves difficulties in motor control—in directing the movements of a pen or pencil to form letters and words. Investigators have reported surprisingly specific types of writing disorders that fall under this category. For example, some patients can write numbers but not letters, some can write uppercase letters but not lowercase letters, some can write consonants but not vowels, some can write cursively but not print uppercase letters, and others can write letters normally but have difficulty placing them in an orderly fashion on the page (Cubelli, 1991; Alexander et al., 1992; Margolin and Goodman-Schulman, 1992; Silveri, 1996).

The second type of writing disorder involves problems in the ability to spell words, as opposed to problems with making accurate movements of the fingers. I will devote the rest of this section to this type of disorder. Like reading, writing (or more specifically, spelling) involves more than one method. The first is related to audition. When children acquire language skills, they first learn the sounds of words, then learn to say them, then learn to read, and then learn to write. Undoubtedly, reading and writing depend heavily on the skills that are learned earlier. For example, to write most words, we must be able to "sound them out in our heads," that is, to hear them and to articulate them subvocally. If you want to demonstrate this to yourself, try to write a long word such as *antidisestablishmentarianism* from memory and see whether you can do it without saying the word to yourself. If you recite a poem or sing a song to yourself under your breath at the same time, you will see that the writing comes to a halt.

A second way of writing involves transcribing an image of what a particular word looks like—copying a visual mental image. Have you ever looked off into the distance to picture a word so that you can remember how to spell

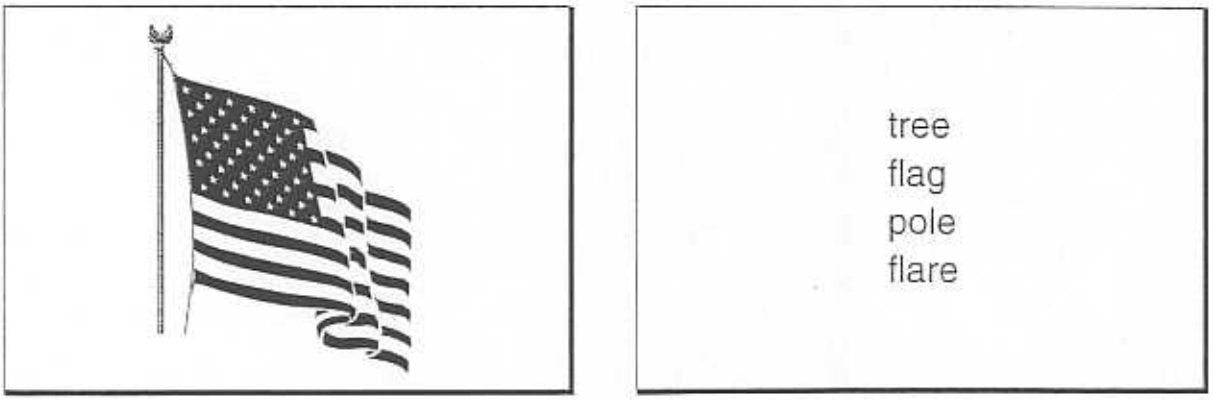


Figure 16.23

An item from a task given to patient R.F. Although she could not read, she could choose the word that went with the picture.

it? Some people are not very good at phonological spelling and have to write some words down to see whether they look correct. This method obviously involves *visual* memories, not acoustical ones.

A third way of writing involves memorization of letter sequences. We learn these sequences the way we learn poems or the lyrics to a song. For example, many Americans learned to spell *Mississippi* with a singsong chant that goes like this: M-i-s-s-i-s-s-i-p-p-i, emphasizing the boldfaced letters. (Similarly, most speakers of English say the alphabet with the rhythm of a nursery song that is commonly used to teach it.) This method involves memorizing sequences of letter names, not translating sounds into the corresponding letters. As you will recognize, it is exactly this method that permits people with word-form dyslexia to recognize words as they spell out their letters, one by one.

Finally, the fourth way of writing involves motor memories. We undoubtedly memorize motor sequences for very familiar words, such as our own names. Most of us need not sound out our names to ourselves when we write our signature, nor need we say the sequence of letters to ourselves, nor need we imagine what our signature looks like.

Neurological evidence supports at least the first three of these speculations. Brain damage can impair the first of these methods: phonetic writing. This deficit is called **phonological dysgraphia** (Shallice, 1981). (*Dysgraphia* refers to a writing deficit, just as *dyslexia* refers to a reading deficit.) People with this disorder are unable to sound out words and write them phonetically. Thus, they cannot write unfamiliar words or pronounceable nonwords, such as the ones I presented in the section on reading. They can, however, visually imagine familiar words and then write them. **Orthographic dysgraphia** is just the opposite: a disorder of visually based writing. People with orthographic dysgraphia can *only* sound words out; thus, they can spell regular words such as *care* or *tree*, and they can write pronounceable nonsense words. However, they have difficulty spelling irregular words such as *half* or *busy* (Beauvois and Dérouesné, 1981); they may write *haff* or *bizzy*. According to Benson and Geschwind (1985), phonological dysgraphia (impaired phonological writing) is caused by damage to the superior temporal lobe, whereas orthographic dysgraphia (impaired visual, whole-word writing) is usually caused by damage to the inferior parietal lobe.

The third method of spelling depends on a person's having memorized sequences of letters that spell particular words. Cipolotti and Warrington (1996) reported the case of a patient who lacked this ability. The patient sustained a left hemisphere stroke that severely disrupted his ability to spell words orally and impaired his ability to recognize words that the examiners would spell aloud. Presumably, his ability to spell written words depended on the first two methods of writing: auditory and visual. The examiners noted that when they spelled out words to him, he would make writing movements with his hand on top of his knee. When they asked him to clasp his hands together so that he could not make these writing movements, his ability to recognize four-letter words being spelled aloud dropped from 66 percent to 14 percent. It appears that he was using feedback from hand movements to recognize the words he was "writing" on his knee.

Japanese patients show writing deficits similar to those of patients whose languages use the Roman alphabet; some patients have difficulty writing kana symbols, whereas others have difficulty with kanji symbols (Iwata, 1984; Yokota et al., 1990). Kawamura, Hirayama, and Yamamoto (1989) reported a particularly interesting case of a man with damage to the middle part of the corpus callosum who could write kana symbols with both hands and could write kanji symbols with the right hand but not the left. He could *copy* kanji symbols with his left hand; he just could not write them down when the investigators dictated them to him. (See ***Figure 16.24.***) Another patient, reported by Tei, Soma, and Maruyama (1994), had the opposite symptoms: very bad kana writing with the nondominant hand but better kanji writing.

These results have interesting implications. Writing appears to be organized in the speech-dominant hemisphere (normally, the left hemisphere). That is, the information needed to specify the shape of the symbols is provided by circuits in this hemisphere. When a person uses his or her left hand to write these symbols, the information must be sent across the corpus callosum to the motor cortex of the right hemisphere, which controls the left hand. Apparently, information about the two forms of Japanese symbols is transmitted through different parts of the corpus callosum; the brain damage of the patient studied by Kawamura and his colleagues disrupted one of these pathways but not the other. (See ***Figure 16.25.***)

As we saw in the section on reading, some patients (those with direct dyslexia) can read aloud without being able to understand what they are reading. Similarly, some patients can write words that are dictated to them even though they cannot understand these words (Roeltgen, Rothi, and Heilman, 1986; Lesser, 1989). Of course, they cannot communicate by means of writing, because they cannot translate their thoughts into words. (In fact, because most of these patients have sustained extensive brain

phonological dysgraphia A writing disorder in which the person cannot sound out words and write them phonetically.

orthographic dysgraphia A writing disorder in which the person can spell regularly spelled words but not irregularly spelled ones.

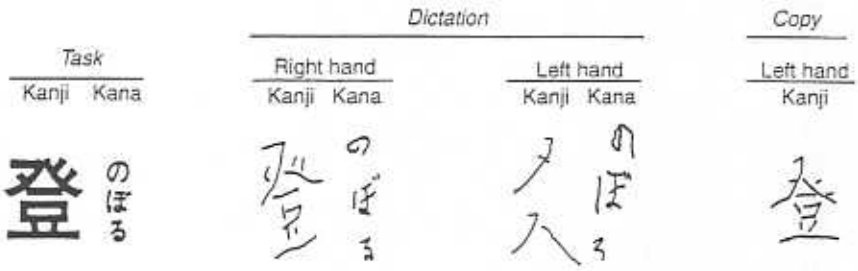


Figure 16.24

The writing of a Japanese patient with damage to the middle part of the corpus callosum. He could write both kanji and kana characters with his right hand, but he could not write kanji characters with his left hand (blue). He could, however, copy kanji characters with his left hand if he was given a model to look at. (From Kawamura, M., Hirayama, K., and Yamamoto, H. *Brain*, 1989, *112*, 1011–1018. Reprinted by permission of Oxford University Press.)

damage, their thought processes themselves are severely disturbed.) Some of these patients can even spell pronounceable nonwords, which indicates that their ability to spell phonetically is intact. Roeltgen et al. (1986) referred to this disorder as *semantic agraphia*, but perhaps the term *direct dysgraphia* would be more appropriate, because of the parallel with direct dyslexia.

Developmental Dyslexias

Some children have great difficulty learning to read and never become fluent readers, even though they are otherwise intelligent. Specific language learning disorders, called **developmental dyslexias**, tend to occur in families, a finding that suggests a genetic (and hence biological) component (Pennington et al., 1991; Wolff and Melngailis, 1994). Linkage studies suggest that the chromosomes 6 and 15 may contain genes responsible for different components of this disorder (Grigorenko et al., 1997; Fisher et al., 1999; Gayán et al., 1999). A study of fifty-six dyslexic boys in Sydney, Australia, found that two-thirds of them showed impairments in both phonological and word-form reading. Among the other third, 64 percent had difficulty only with phonological reading, and 46 percent had difficulty only with word-form reading (Castles and Coltheart, 1993). (You will recall that phonological difficulty is the primary symptom of phonological dyslexia, while word-form difficulty is the primary symptom of surface dyslexia.)

As we saw earlier, the fact that written language is a recent invention means that natural selection could not have given us brain mechanisms whose only role is to interpret written language. Therefore, we should not expect that developmental dyslexia involves only deficits in reading. Indeed, researchers have found a variety of language deficits that do *not* involve reading. One common deficit is deficient phonological awareness. That is, people with developmental dyslexia have difficulty blending or rearranging the sounds of words that they hear (Eden and Zeffiro, 1998). For example, they have difficulty recognizing that if we remove the first sound from "cat," we are left with the word "at." Difficulties such as this one might be expected to impair the ability to read phonetically.

Several studies have suggested that abnormal development of specific regions of the brain may be responsible for developmental dyslexia. However, in a review of the literature, Filipek (1995) concluded that imaging studies have failed to find a marker for developmental dyslexia—that is, a reliable abnormality that is universally found in a particular location in the brains of dyslexics. Over the years, investigators have reported finding differences in the size or shape of the corpus callosum or various parts of the region around the posterior part of the lateral fissure, but follow-up studies have failed to confirm the earlier ones.

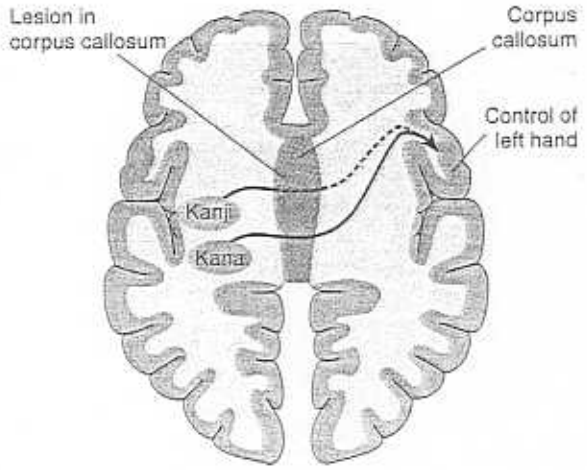


Figure 16.25

The role of the corpus callosum in Japanese writing. Information about kana and kanji characters apparently crosses different parts of the corpus callosum.

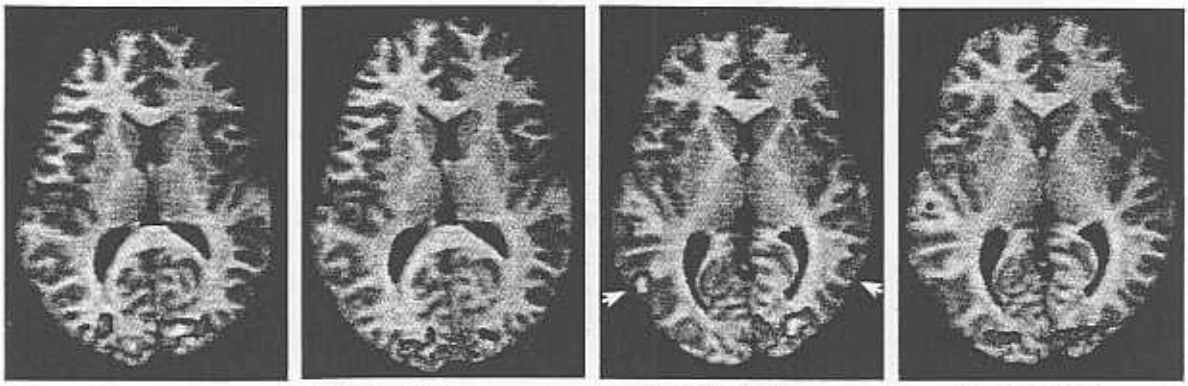
developmental dyslexia A reading difficulty in a person of normal intelligence and perceptual ability; of genetic origin or caused by prenatal or perinatal factors.

One finding has been receiving a considerable amount of attention in recent years. Galaburda and Livingstone (1993) found evidence for a deficit in the magnocellular layers of the lateral geniculate nucleus. As we saw in Chapter 6, the visual system has two major components, named after two types of layers in the lateral geniculate nucleus, which relays information from the retina to the visual cortex. The *magnocellular system* is more ancient. It consists of two layers of neurons with large cell bodies that transmit information about movement, depth, and small differences in contrast. The *parvocellular system*, which evolved more recently, consists of four layers of neurons that transmit information about color and fine details. Galaburda and Livingstone studied the brains of deceased patients who had had developmental dyslexia and discovered that the magnocellular layers of these people were disorganized. The cell bodies in these layers were 27 percent smaller, and they were more variable in their size and shape. The parvocellular layers were normal.

Why should abnormalities in the magnocellular system impair people's ability to read? Stein and Walsh (1997) note that the primary target of the magnocellular system is the posterior parietal lobe, the endpoint of the dorsal stream of the visual system. As we saw in Chapter 6, this system is concerned with the "where" of vision, while the ventral stream, which terminates in the inferior temporal lobe, is concerned with the "what" of vision. But why should the "where" system be so important to reading? Stein and Walsh point out that dyslexics often have trouble with spatial perception and of movements in space. They often transpose letters (for example, reading *saw* as *was*), they are often clumsy and have difficulties with balance, their handwriting tends to be very poor, they learn to walk later than most other children and have trouble learning to ride a bike, they are slower to learn to tell time or learn the days of the week and months of the year, they have difficulty reading maps and distinguishing between left and right, and they tend not to establish strong handedness. Such problems are often associated with damage or developmental abnormalities in the posterior parietal lobe. Perhaps, Stein and Walsh suggest, abnormal input to the parietal lobe caused by an abnormal magnocellular system impairs the development of this region and causes a variety of symptoms, including dyslexia.

In support of their hypothesis, Stein and Walsh note that many dyslexics complain that when they try to read, letters move around, merge, and become blurry. Their gaze is often unsteady, and their ability to read is sometimes improved by providing them with text with larger type (Cornelissen et al., 1991). They also note that monkeys with lesions of the posterior parietal lobes have no trouble discriminating between different shapes but have trouble distinguishing between reversals of the same shape, such as < versus > or b versus d (Walsh and Butler, 1996).

Eden et al. (1996) performed a functional MRI study that provides further support for Stein and Walsh's hypothesis. They found that although no differences were seen in the activity of the primary visual cortex to stationary visual stimuli, there were differences in activation of visual area V5 by moving stimuli. Area V5 receives input from the magnocellular system and is involved in the perception of motion. As Figure 16.26 shows, looking at a pattern of black dots moving against a gray background activated area V5 in control subjects (white arrows), but such activation was not seen in the brains of subjects with developmental dyslexia. (See *Figure 16.26.*)



Controls/Stationary pattern | Dyslexics/Stationary pattern | Controls/Motion | Dyslexics/Motion

Figure 16.26

Functional MRI scans from dyslexics and control subjects looking at a stationary pattern (a plus sign against a gray background) or at a moving pattern (an array of black dots moving against a gray background). Note that viewing the moving pattern activated area V5 of the visual association cortex in control subjects (white arrows) but not in dyslexics.
(From Eden, G. F., VanMeter, J. W., Rumsey, J. M., Maisog, J. M., Woods, R. P., and Zeffiro, T. A. *Nature*, 1996, *382*, 66–69.)

Lewis and Frick (1999) discovered a perceptual deficit that appears to be related to developmental dyslexia: row blindness. One of the basic principles of visual perception is that elements that resemble each other tend to be seen as part of a group. The investigators accidentally discovered that a dyslexic subject did not see Figure 16.27a as a set of four rows. However, when the array was rotated 90 degrees (Figure 16.27b), the subject saw four vertical columns of Os and Xs. (See *Figure 16.27.*) The investigators tested more people, and found that over half of the college-age dyslexics they tested showed row blindness but that few good readers did so. They suggested that row blindness may make it difficult for people to easily and quickly recognize lines of text and thus may contribute to difficulty in reading.

Geschwind and Behan (1984) noted that investigators have long recognized that a disproportionate number of people with developmental dyslexias are also left-handed. Furthermore, clinical observations suggested a relationship between left-handedness and various immune disorders. Therefore, Geschwind and Behan studied a group of left-handed and right-handed people to determine whether the relations were statistically significant. They found that they were: The left-handed subjects were ten times more likely to have specific learning disorders (10 percent versus 1 percent) and two and one-half times more likely to have immune disorders (8 percent versus 3 percent). The immune disorders included various thyroid and bowel diseases, diabetes, and rheumatoid arthritis. Of course, although the relation was statistically significant, it was not perfect. After all, most left-handed people are healthy and are good readers.

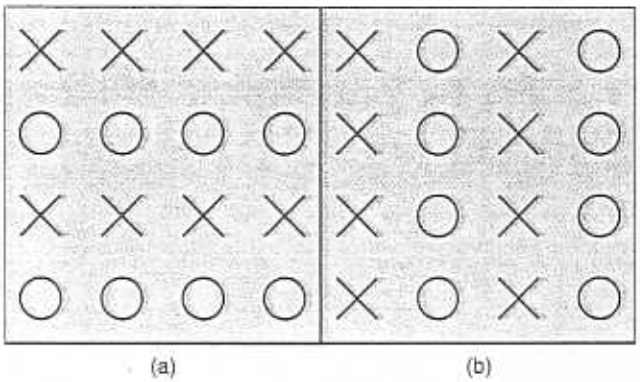


Figure 16.27

Row blindness. People with row blindness do not see four horizontal rows of Xs or Os (a), but they can see four vertical columns when the array is rotated 90 degrees (b).

(Adapted from Lewis, J. P., and Frick, R. W. *Neuropsychologia*, 1999, 37, 385–393.)

Interim Summary

Brain damage can produce reading and writing disorders. With few exceptions aphasias are accompanied by writing deficits that parallel the speech production deficits and by reading deficits that parallel the speech comprehension deficits. Pure alexia is caused by lesions that produce blindness in the right visual field and that destroy fibers of the posterior corpus callosum. Damage that is restricted to the posterior corpus callosum produces alexia only in the left visual field.

Research in the past few decades has discovered that acquired reading disorders (dyslexias) can fall into one of several categories, and the study of these disorders has provided neuropsychologists and cognitive psychologists with thought-provoking information that has helped them to understand how normal people read. Surface dyslexia, usually caused by damage to the left lateral temporal lobe, is a loss of whole-word reading ability. Phonological dyslexia, usually caused by damage to the left frontal lobe, is loss of the ability to read phonetically. Word-form (spelling) dyslexia is caused by a deficit in both phonetic and whole-word reading; patients can still recognize individual letters and can read slowly by pronouncing each letter. Direct dyslexia is analogous to transcortical sensory aphasia; the patients can read words aloud but cannot understand what they are reading. Some dyslexia patients can at least partially comprehend written words without being able to pronounce them; they can match corresponding words and pictures and recognize misspelled words that they cannot read.

Brain damage can disrupt writing ability by impairing people's ability to form letters—or even specific types of letters, such as uppercase or lowercase letters or vowels. Other deficits involve the ability to spell words. We normally use at least four different strategies to spell words: phonetic (sounding the word out), visual (remembering how it looks on paper), sequential (recalling memorized sequences of letters), and motor (recalling memorized hand movements in writing very familiar words). Two types of dysgraphia—phonological and orthographic—represent difficulties implementing phonetic and visual strategies, respectively. The existence of these two disorders indicates that several different brain mechanisms are involved in the process of writing. In addition, some patients have a deficit parallel to direct dyslexia; they can write words that they cannot understand.

Developmental dyslexia is a hereditary condition that may involve abnormal development of parts of the brain that play a role in language. Most developmental dyslexics have difficulty with phonological processing—of spoken words as well as written ones. Recent evidence suggests

that abnormal development of the magnocellular system of the lateral geniculate nucleus, seen in people with developmental dyslexia, may impair normal development of the posterior parietal lobe. A better understanding of the components of reading and writing may help us to develop effective teaching methods that will permit people with dyslexia to take advantage of the abilities that they do have.

Table 16.2 summarizes the disorders described in this section.

Table 16.2
Reading and Writing Disorders Produced by Brain Damage

Reading Disorders	Whole-word reading	Phonetic reading	Remarks
Pure alexia	Poor	Poor	Can write
Surface dyslexia	Poor	Good	
Phonological dyslexia	Good	Poor	
Spelling dyslexia	Poor	Poor	Can read words letter-by-letter
Direct dyslexia	Good	Good	Cannot comprehend words
Comprehension without reading	Poor	Poor	Show some comprehension of words
Writing Disorders	**Whole-word writing**	**Phonetic writing**	
Phonological dysgraphia	Good	Poor	
Orthographic dysgraphia	Poor	Good	

suggestedreadings

Kolb, B., and Whishaw, I. Q. *Fundamentals of Human Neuropsychology,* 4th ed. New York: W. H. Freeman, 1996.

Obler, L. K., and Gjerlow, K. *Language and the Brain.* Cambridge, England: Cambridge University Press, 1999

Parkin, A. J. *Explorations in Cognitive Neuropsychology.* Oxford, England: Blackwell Publishers, 1996.

Posner, M. I., and Raichle, M. E. *Images of Mind.* New York: Scientific American Library, 1994.

suggestedreadings

Sleep and Language

http://thalamus.wustl.edu/course/sleep.html

This Web site provides an overview of sleep phenomena and of language.

Dyslexia Web Resources

http://www.krgraphics.co.uk/texthelp/d_web.htm

The focus of the collection of Web links is on the topic of dyslexia.

Aphasia: Treatment, Prevention, and Cure

http://www.healthlinkusa.com/A.html

This page contain a series of links that will take students to a page devoted to the topic of aphasia.

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