65 The Neural Architecture of Language Disorders

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ABSTRACT   Traditional models of language processing in the brain presume certain functions for Broca's area, Wernicke's area, and the fiber tracts that connect them. These descriptions have served a useful clinical purpose over the last century, but recent advances in the study of language and in structural and functional neuroimaging have found them somewhat insufficient. This chapter discusses findings from our work with aphasic patients examining the relationship between specific speech and language disorders and the lesions underlying these deficits. It is shown that traditional language areas may serve somewhat different functions than originally described, and that the identification of more specific deficits and their neuroanatomic correlates can lead to more informative mapping of language functions in the brain.

Much has been written about the behavioral deficits observed in the various types of aphasia, or language disorders, that arise from injury to the brain. Some describe the clinical manifestations of the disorders, others, the implications for theories of normal language processing. In this chapter, we discuss the brain areas that have been associated with these disorders and the current neurological model of language processing that was derived from these patient studies. We begin with a review of the clinical observations of Broca and Wernicke—observations that laid the foundation for how most clinicians and neuroscientists believe the brain processes language. We provide some basic information about the three aphasias that are most pertinent to this model and show how these descriptions have changed since they were first presented more than 100 years ago. Finally, we present our view of the organization of language in the brain by reviewing what we have learned from our patients with aphasia. We review the speech and language deficits we have studied and the correlations we have found with the brain areas that were destroyed by these patients' injuries. With these more recent data, we hope to provide some suggestions for expanding current models concerning the neural architecture of language disorders.

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The classical theory

In the late 1700s, the simple observation of a 14-year-old formed the basis of a theory concerning the neural mechanisms of language. At that time, Franz Joseph Gall noticed that some of his more articulate friends also had markedly protruding eyeballs. He reasoned that the areas of the brain behind both eyes must have grown larger to accommodate the superior language skills of his friends and were thus pushing the eyeballs forward. With this, Gall instigated the popular practice of phrenology in which bumps on the head were related to enhanced "faculties" of the mind. Even after the phrenology fervor had subsided, many scholarly societies still debated whether speech resided in the frontal lobes.

It was in this context that (Pierre) Paul Broca, a surgeon with an interest in anthropology, saw a patient with an infected leg and a right hemiparesis who also had lost the capability of speech. The patient, whose name was Leborgne, could only utter the single syllable, "tan," which he used each time he initiated speech. Broca thought the patient understood most of what was said to him and thus presented as the perfect case to prove Gall's theory of speech in the frontal lobes. As it happened, the patient died a few days later and Broca was able to view the brain at autopsy. Indeed, a lesion was found in the frontal lobe on the posterior surface of the third frontal gyrus on the left side (Broca, 1861), the area later referred to as "Broca's area" (figure 65.1). Broca also noted softening as far posterior as the parietal operculum; but since he was only looking for involvement of the frontal lobes, this observation was deemed less important. In order to preserve the brain for posterity, it was never cut, and the extent of the lesion medially or posteriorly was not known until a century later (Signoret et al., 1984). By that time, the notion that the frontal lobes were involved in speech was firmly set.

Broca believed that Leborgne's deficits affected only his articulation abilities, not his language; hence he termed the deficit "aphemia," not "aphasia." Later, he published another, very similar case, also with a right
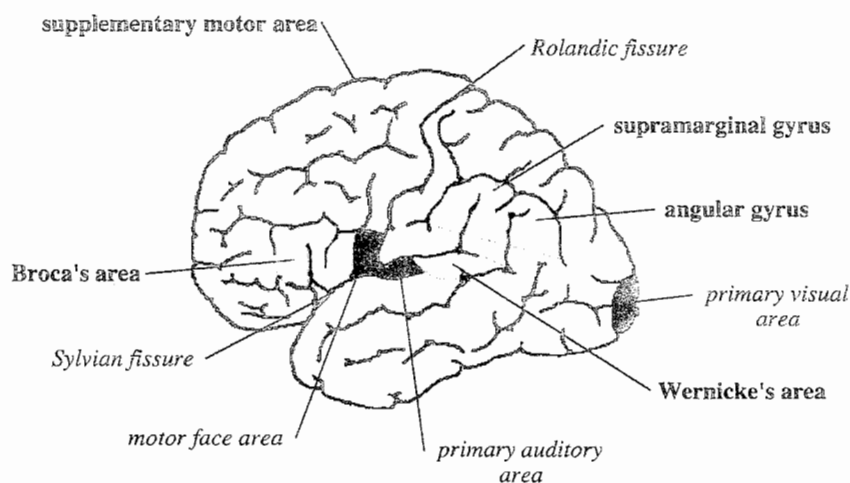
FIGURE 65.1   Lateral view of the left hemisphere with some of the brain areas implicated in language labeled in boldface.

hemiparesis and a lesion in the left frontal lobe. Two years later, he had accumulated six more cases, none of which came to autopsy, but all of which had a right hemiparesis. By 1865, Broca realized that the co-occurrence of the right hemiparesis and the speech deficit was beyond coincidental and concluded that it was the *left* frontal lobe, at least in right-handers, that controlled the ability for speech.

Not long thereafter, Carl Wernicke, a 26-year-old physician, described another type of language problem. He had examined two patients with profound deficits in understanding spoken language. Their speech, though perfectly fluent, was incomprehensible and riddled with nonsense words that did not exist in German. Wernicke examined the brain of one of these patients and found a lesion in the posterior part of the superior temporal gyrus, posterior to primary auditory cortex (figure 65.1). The lesion of the other patient was assumed to be in the same place. He associated this region (later termed "Wernicke's area") with the storage of "the auditory memory for words." Wernicke believed that comprehension deficits were caused by the loss of these memories, while the distorted output was due to the inability to monitor self-spoken auditory images (Wernicke, 1874).

By documenting this second type of language disorder, Wernicke provided a formal distinction between two components of language, firmly departing from previous phrenological notions that language was a single entity, localizable in one brain region. With the addition of this new aphasia, differences were drawn between the "expressive" aphasia of Broca and the "receptive" aphasia described by Wernicke. Though these terms are now considered somewhat imprecise, the differentiation was an important one for its time and set aphasiologists to

thinking about what other components of the language system could be affected by brain injury.

Wernicke made another important contribution to the study of brain and language. He detailed an impressive theory establishing the notion of brain centers and the connections between them. He believed that auditory memories, first evoked in the posterior superior temporal gyrus, were then passed on to Broca's area where they were prepared for articulation. He thereby predicted another type of aphasia, "conduction" aphasia, caused by lesions to the pathway *between* these centers, resulting in an interruption of the transmission of linguistic information from Wernicke's area to anterior speech areas. The result would be a disruption in verbal production, since auditory word images evoked in Wernicke's area would be cut off from articulatory mechanisms in the frontal lobe.

This "connectionist" approach, as it came to be called (not to be confused with the "connectionism" of today's models), became extremely popular. Though it underwent a period of disfavor in later years, it returned to popularity with the work of Norman Geschwind. In his influential paper, "Disconnexion syndromes in animal and man" (Geschwind, 1965), he presented and explained several behavioral deficits in terms of the lesion in major centers and the disconnections between them. For the study of aphasia, he assimilated the most current information from behavioral neurology, psychology and linguistics and formulated a working model of the neural mechanisms of language (see Geschwind, 197... This model assumed the involvement of Wernicke's area in language comprehension and that of Broca's area in articulation and possibly in assisting in the grammatical organization of language. Geschwind also identified the arcuate fasciculus as the fiber pathway that should con-

BOX 65.1

Spontaneous speech examples from patients with Broca's and Wernicke's aphasia on a picture description task

Patient WR (Broca's aphasia)

> "O, yeah. Det's a boy an' a girl ... an' ... a ... car ... house ... light po' (pole). Dog an' a ... boat. 'N det's a ... mm ... a ... coffee, an' reading. Det's a ... mm ... a ... det's a boy ... fishin'." (Elapsed time: 1 min, 30 s)

Patient OB (Wernicke's aphasia)

> "Ah, yes, it's, ah ... several things. It's a girl ... uncurl ... on a boat. A dog ... 'S is another dog ... uh–oh ... long's ... on a boat. The lady, it's a young lady. An' a man. They were eatin'. 'S be place there. This ... a tree! A boat. No, this is a.... It's a house. Over in here ... a cake. An' it's, it's alot of water. Ah, all right. I think I mentioned about that boat. I noticed a boat being there. I did mention that before.... Several things down, different things down ... a bat ... a cake ... you have a...." (Elapsed time: 1 min, 20 s)

nect Wernicke's and Broca's areas as it passes through the parietal lobe. Lesions to it would presumably cause a disconnection of these two areas, resulting in the repetition deficit so characteristic of conduction aphasia, the other type of aphasia predicted by Wernicke more than a century ago.

Current descriptions of the aphasias

Current descriptions of aphasic deficits have modified Broca's and Wernicke's definitions considerably. Recall that Broca considered the deficit in his patients to be exclusively in articulation. He was confident that his patients understood everything said to them. Wernicke viewed his patients' problem as one in the storage of "the auditory memory for words." He believed that the loss of these memories resulted in the comprehension deficit, while an inability to monitor self-spoken auditory images resulted in his patients' distorted output. Today, we describe the main aphasias in a somewhat different way.

BROCA'S APHASIA The most striking characteristic of Broca's aphasia is the slow and effortful speech and the lack of grammatical markers in language production. Utterances are produced in a telegraphic or agrammatic style, with patients relying mostly on high-frequency content words and omitting the smaller function words that convey mostly grammatical information (see box 65.1.) Repetition is impaired in the same fashion as their spontaneous speech. Word-finding is also impaired and contributes to the difficulty in production.

Patients with a more severe form of Broca's aphasia can produce nothing more than recurring utterances— syllables, words, or phrases repeated again and again

(e.g., "/tono tono/," "yes, yes," "Sweet sweetie, I miss sweet sweetie")–like Leborgne's "tan." Patients with a milder Broca's aphasia tend to produce sentences and phrases that contain some grammatical organization. However, some aphasia batteries will not recognize a patient as a Broca's aphasic unless there is a complete absence of such structures in the patient's spontaneous speech.

Patients with Broca's aphasia were long thought to have intact language comprehension. This is clearly not the case. Although these patients appear to follow conversations with little difficulty, careful testing reveals that complex grammatical structures requiring the manipulation of grammatical information or the processing of grammatical rules elude them. Thus, the sentence "The boy kissed the girl" may be understood perfectly, while the more complex sentence "The girl was kissed by the boy" may not, though their meanings are identical. There is much debate as to the nature of this phenomenon—whether it is due to a central syntactic processing deficit or to an attentional or working memory disorder, and whether this deficit is associated with Broca's area. No doubt this debate will continue for years to come, as syntax is a critical part of human language, conceivably comprising the fundamental difference between our communication system and those of other species.

Speech deficits such as *dysarthria* and *apraxia of speech* frequently accompany Broca's aphasia. Dysarthria is the inability to control the muscles of articulation. Speech sounds are systematically distorted such that all of the patient's utterances sound similarly weak and flaccid or spastic, depending on the type of dysarthria. Apraxia of speech is an articulatory programming disorder that produces errors that, while inconsistent, approximate the target word (e.g., saying /yawyer/ for

"lawyer" or /chookun/ for "cushion"). These disorders also contribute to the fluency deficits of patients with Broca's aphasia and must be distinguished from the aphasia itself.

WERNICKE'S APHASIA   Patients with Wernicke's aphasia have a deep disruption in their ability to use language to express their ideas. Whereas Broca's aphasics can participate in a conversation and get most of its meaning, Wernicke's aphasics can understand very little of what is said and contribute less in return. Again, there is considerable range in these characteristics, with some patients communicating some linguistic information and others producing only jargon, or meaningless sounds.

Patients with Wernicke's aphasia score poorly on tests of auditory and reading comprehension. In severe cases, their ability to understand even single words is compromised. Comprehension of sentences and phrases is even more impaired. The spontaneous speech of Wernicke's aphasic patients is very fluid, quite the opposite of the halting and telegraphic style of Broca's aphasia (see box 65.1). Sentences appear to be well-formed but are often riddled with *paraphasias*, words that are substituted for the intended word. If one did not speak the patient's language, one would be unable to detect anything particularly wrong, the speech output being so fluent. Word retrieval on confrontation naming tasks is severely impaired. Patients with Wernicke's aphasia not only have difficulty in finding the correct word but often cannot recognize the correct name even when it is offered to them.

CONDUCTION APHASIA   The hallmark of conduction aphasia is the repetition deficit in contrast to relatively preserved comprehension. Patients with conduction aphasia can repeat single words and short high-frequency sentences (e.g., "He is not coming back") fairly easily. Longer sentences, or those that might occur less frequently in normal language (e.g., "The pastry cook was elated"), are far more difficult for conduction aphasics. In contrast, their auditory comprehension is relatively intact, even demonstrating their understanding of a sentence they were just unable to repeat.

Other aphasias include *global aphasia* (in which language is very severely impaired, yielding something of a combination of Broca's and Wernicke's aphasia), *anomic aphasia* (in which a word-finding problem is the most significant impairment), and the *transcortical aphasias* (in which repetition is spared). Clinically, these aphasias are seen very often, but are less relevant to our discussion and will be treated cursorily. More detailed descriptions can be found in clinical reference books such as Benson

and Ardila (1996), Goodglass (1993), and Goodglass and Kaplan (1972).

Does the classical theory explain these disorders?

Since Broca's and Wernicke's observations, converging evidence from several other sources has emerged to support some of their claims. For example, Broca's important distinction between left and right hemisphere contributions to speech continued to find support through numerous lateralization studies, including those with split brain patients, and those using dichotic listening and visual half field testing. In addition, observations of aphasic patients confirmed that the aphasia of 90–98% of right-handed patients was caused by left hemisphere lesions. The work of Rasmussen and Milner (1977) also demonstrated that 95% of right-handed patients undergoing Wada testing for epilepsy surgery became temporarily aphasic after injection of the anesthetic into the left internal carotid artery feeding the left cerebral hemisphere. Furthermore, Penfield and Roberts (1959) found that electrical stimulation to the left hemisphere of the brain caused aphasia-like symptoms more often than right hemisphere stimulation.

But evidence about the localization of language and speech *within* the left hemisphere has been less well substantiated. For the most part, the classical model is successful at describing patients' disorders and in predicting the brain areas that are compromised. However, there are numerous occasions on which the model fails. Some examples from our own clinic may help to illustrate this point.

Patient JC has a large frontal lobe lesion encompassing Broca's area and areas anterior and superior to it, extending deep into underlying white matter (figure 65.2). Traditional theory would predict that this patient should have a Broca's aphasia, producing short telegraphic sentences consisting mostly of high-frequency content words and few function words (prepositions, conjunctions, etc.), with impaired comprehension for complex grammatical constructions (embedded clauses, passive voice, etc.). Though JC's speech is slow and effortful, the sentences he produces are perfectly grammatical and contain numerous examples of complex constructions. In addition, his language comprehension is virtually unaffected. Another patient, JH, does have a persisting Broca's aphasia with the symptoms just described, but his lesion completely spares Broca's area.

As for Wernicke's area, patient MC has a lesion encompassing Wernicke's area and should have impaired comprehension and a Wernicke's aphasia. Instead, this patient has intact auditory comprehension and the repetition deficit typical of conduction aphasia. Patient OB
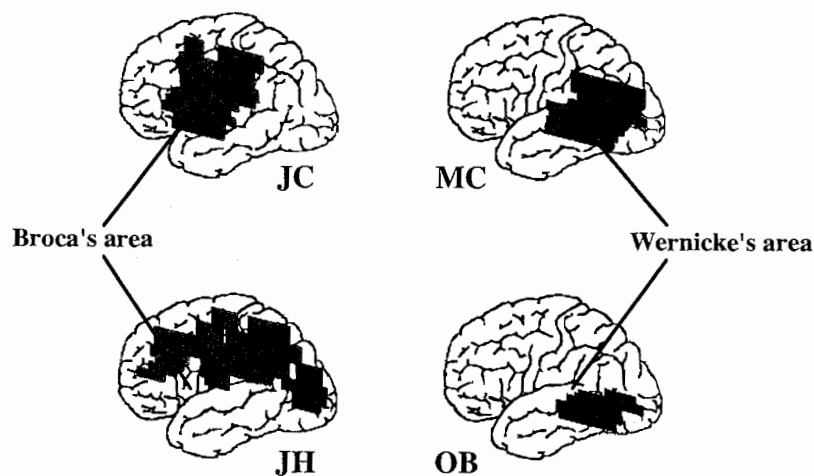
FIGURE 65.2   Top: Computer-reconstructed lesions of two patients, one with a lesion involving Broca's area (patient JC) and one with a lesion in Wernicke's area (patient MC). Neither of these patients was classified with the Broca's and Wernicke's aphasia predicted by traditional theory. Bottom: Two patients who do classify with a Broca's aphasia (patient JH) and a Wernicke's aphasia (patient OB). Neither of these patients has a lesion in Broca's or Wernicke's areas, respectively.

has a dense Wernicke's aphasia with severely impaired comprehension and conversation littered with paraphasic errors. His lesion should include Wernicke's area. But it doesn't. In fact, his lesion spares Wernicke's area and instead involves the middle temporal gyrus and underlying white matter.

None of these patients is left-handed or multilingual; nor does any have an early neurological or medical problem that might have caused a reorganization of language functions in their brains. Moreover, these patients are not isolated examples. Even after Broca's historic paper, numerous cases were presented that refuted Broca's claim (e.g., Charcot cited in Finger, 1994; Bateman, 1870; Brown-Sequard, 1877; Marie, 1906; Moutier, 1908). Several found patients with lesions to Broca's area who had no Broca's aphasia at all or patients with Broca's aphasia and no lesion in Broca's area. Even the renowned neurosurgeon Wilder Penfield was reported to have completely removed Broca's area with no persisting speech or language impairments. Mohr (1976) did an extensive review of the literature on Broca's area and Broca's aphasia and found that it takes a much larger lesion than Broca described to produce a persisting Broca's aphasia. Other studies have looked at the relationship between Broca's area and Broca's aphasia and Wernicke's area and Wernicke's aphasia, with most finding a far-from-perfect correlation (e.g., Basso et al., 1985; Bogen and Bogen, 1976; Dronkers, Redfern, and Ludy, 1995; Mazzocchi and Vignolo, 1979; Murdoch et al., 1986). Why does the model fail so often?

One reason the model is often inadequate is that it oversimplifies the richness and complexity of language. Language is more than just speaking and listening; it consists of complex rules that govern the way we combine sounds and signs into words and sentences to express thoughts to other human beings who share the same set of rules. At the very least, it involves retrieving words that label concepts, retrieving and applying the grammatical constraints that convey the relationships between the words, preparing the words for articulation, attaching the appropriate intonational patterns and social rules particular to the situation, and then producing the utterance through speech or signed gestures. The field of linguistics is entirely dedicated to studying the intricacies of this complex system, to an extent that could never have been appreciated in Broca's or Wernicke's time.

Second, many studies have attempted to localize behaviors that were not stable. It is now known that in patients with acute Broca's aphasia in the first few weeks after injury, the condition often evolves into a milder form of language impairment, where the deficit is largely one of word-finding. In patients with acute Wernicke's aphasia, the condition almost always evolves into a milder conduction aphasia or anomic aphasia. These early deficits are most likely influenced by the effects of the lesion on neighboring or connected brain regions and do not accurately reflect the deficits caused by lesions to these areas. In our opinion, this lesion–deficit correlation must wait until the behavior has stabilized.

Finally, aphasiologists have never agreed on what we are trying to localize. Broca thought it was the faculty of articulation that resided in Broca's area; later in history, it was syntax. In fact, Broca's aphasia is a *syndrome*, a cluster of aphasia deficits that collectively form a pattern. Patients with Broca's aphasia have numerous
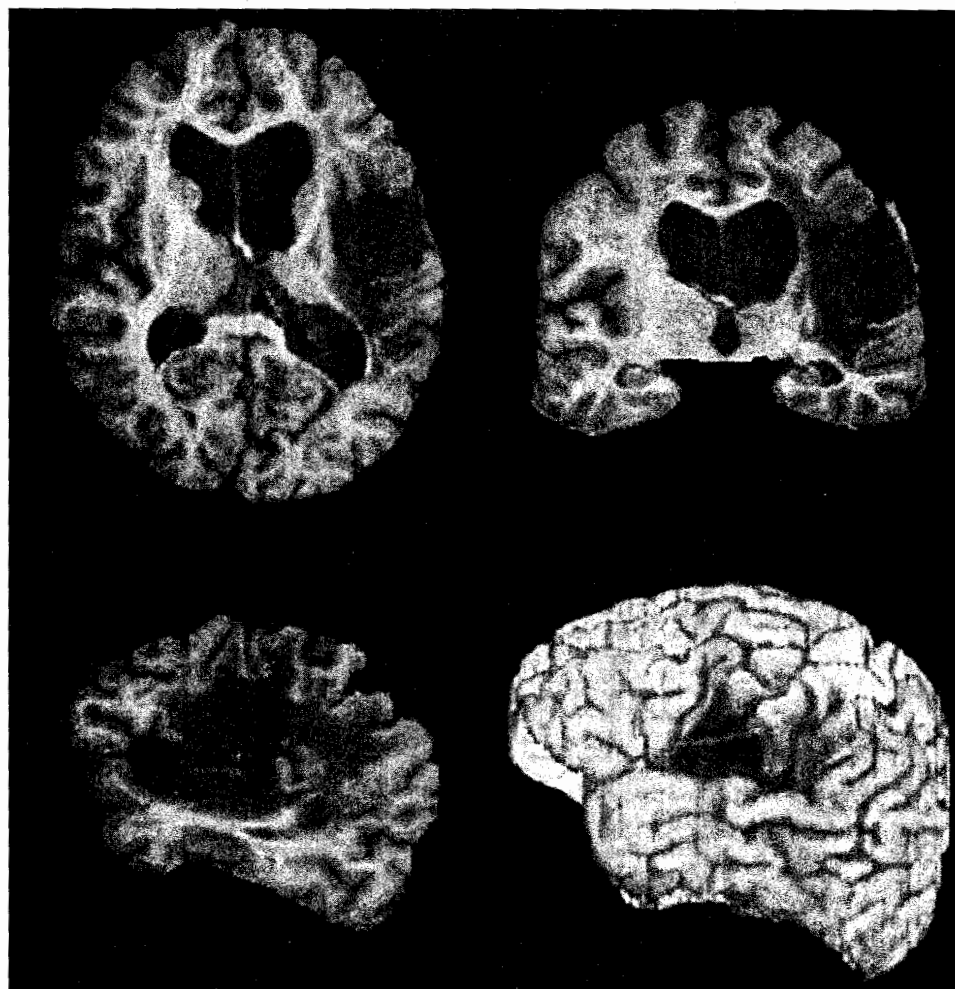
FIGURE 65.3   This three-dimensional MRI reconstruction depicts the lesion in a patient with anomic aphasia and an apraxia of speech. The upper left image is a horizontal slice showing the left hemisphere lesion, while the upper right depicts a coronal section. (The images conform to radiologic convention, with the left hemisphere shown on the right side.) In the lower left is a sagittal section through the left hemisphere. The lower right image shows the 3D reconstruction of the brain with part of the lesion visible on the lateral surface of the left hemisphere.

problems: They are impaired in articulatory agility, word-finding, repetition, and comprehension for complex grammatical structures. Realistically, it makes little sense to suppose that all of these behaviors would be located in one area of the brain. Instead, we find that looking for brain areas associated with more specific components of the speech and language process results in far more reliable correlations. We illustrate this point in the following section.

Recent contributions

Over the last several years, we have looked closely at the specific deficits in our aphasic patients and evaluated these in parallel with a careful analysis of lesion sites. Owing to the large number of aphasic patients at our fa-

cility, we have been able to impose several controls on our studies. First, all of the patients we study have suffered a single cerebral infarction (stroke) with no previous neurologic or psychiatric history that might influence their results. All are right-handed, native English-speaking, and have normal or corrected-to-normal vision and hearing. All speech and language testing is performed at least one year after the stroke to ensure that the deficits are stable and persisting. Each patient has undergone CT or MR imaging at least 3 weeks postonset so that the boundaries of the lesion can be clearly discerned and reliably reconstructed. Most cases are imaged close to the time of testing, and on some patients, we have also obtained 3D MRIs (figure 65.3; see also color plate 43). Patients' lesions are reconstructed onto templates and entered into a microcomputer running

percent overlap

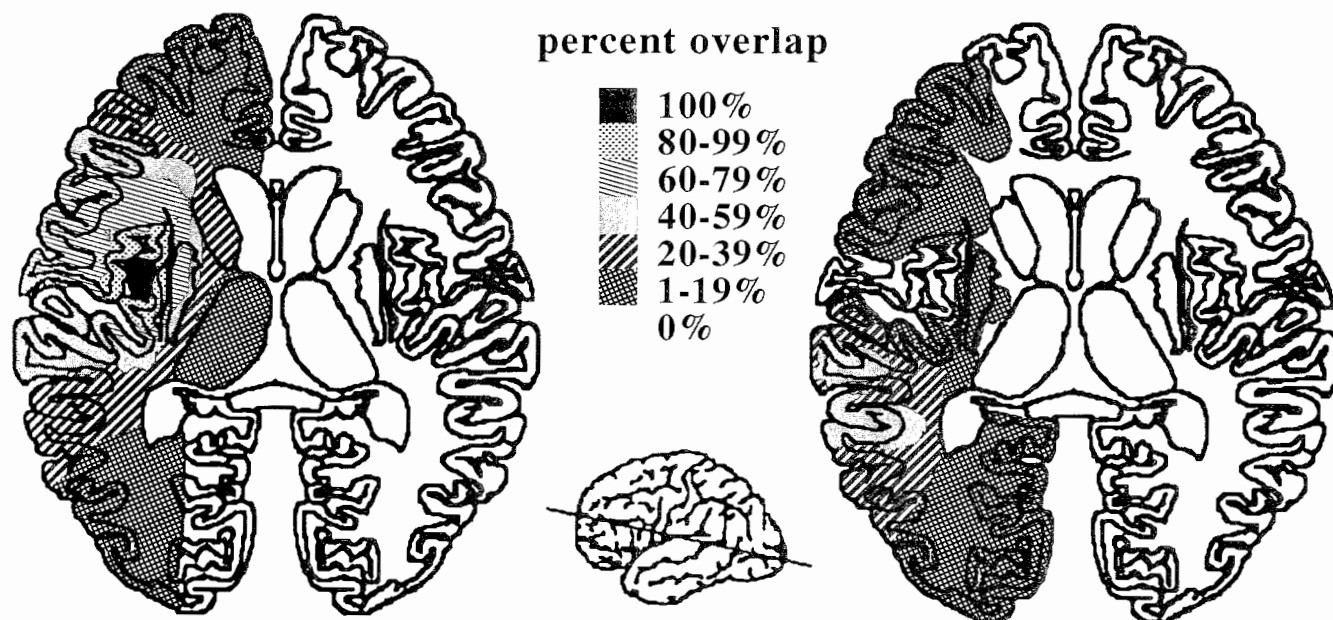|	
■	100%
	80-99%
	60-79%
	40-59%
	20-39%
	1-19%
	0%

FIGURE 65.4   Lesion overlapping in 25 patients with apraxia of speech (left) and 19 patients without this disorder (right). All of the 25 patients with apraxia of speech have a lesion encompassing a small section of the insula, as shown in yellow, while the lesions of the 19 patients without apraxia of speech completely spare the same area.

software developed at our facility (Frey et al., 1987). Patients who exhibit similar deficits are grouped together and their lesions overlapped by the computer to reveal any common areas of infarction. In this way, lesion locations can be determined that are shared by all patients exhibiting the same disorder.

We have accumulated substantial behavioral and imaging data on more than 100 patients, each of whom met all of these criteria. In our first test of this analysis, we examined 12 right-handed chronic patients with a persisting Broca's aphasia and overlapped their lesions to see if Broca's area was involved in all cases, as predicted by traditional theory (Dronkers et al., 1992). It was not. Two cases had lesions that completely spared Broca's area, even though their aphasia was classified as Broca's with a standardized aphasia battery. In addition, we found ten other patients with lesions in Broca's area who had no Broca's aphasia at all, confirming Mohr's finding. Most of these were mildly anomic, and one was a conduction aphasic with a large posterior lesion extension. Thus, we received our first confirmation that the classical model would not be able to explain all of our patients' language deficits.

Yet the lesion overlapping of the Broca's aphasics did yield a consistent relationship. All of the patients with Broca's aphasia, even those without lesions to Broca's area, had a lesion that encompassed part of the insula, the island of cortex deep within the cerebral hemispheres. This led us to explore whether this area might

be the critical area for Broca's aphasia, much as Marie (1906) had once suggested. In fact, we found numerous patients with lesions in this area of the insula who did not have a Broca's aphasia, but did have an apraxia of speech in common. This disorder is believed to be a deficit in planning the movements necessary for speech, with patients producing inconsistent errors that are phonemically similar to the target word. We found that when we overlapped the lesions of 25 patients who were diagnosed with this disorder, all had involvement of a discrete area of the superior tip of the precentral gyrus of the insula (Dronkers, 1996; see figure 65.4). To be certain that the finding was not artifact, we also overlapped the lesions of 19 patients without apraxia of speech and found that their lesions covered nearly as large an area within the left cerebral hemisphere, but completely spared the part of the precentral gyrus that was lesioned in all of the patients with the disorder (figure 65.4; see also color plate 44). This clear dissociation led us to link this new functional area with this articulatory planning deficit.

With regard to Broca's aphasia, apraxia of speech is a disorder that nearly always occurs in patients with this type of aphasia. Thus, it is not surprising to find that the same area of the precentral gyrus of the insula that was lesioned in all of the patients with apraxia of speech was also lesioned in the patients with Broca's aphasia. Even if we overlap the lesions of patients with a milder, but still Broca-like aphasia, we find that these patients also

have lesions in the same area of the insula, and all have an apraxia of speech. Are we saying that this spot on the precentral gyrus of the insula is the new Broca's area? No. We are merely saying that apraxia of speech is one of the central deficits of patients with Broca's aphasia, and this particular behavior is likely caused by lesions to this specific area of the insula.

Another problem of Broca's aphasic patients is in sentence comprehension, particularly for complex grammatical constructions. We have found that patients with lesions to the anterior portion of the superior temporal gyrus have difficulty in processing sentences, particularly those with complex grammatical constructions (Dronkers et al., 1994). This area has rich connections to the hippocampus and may be involved in recruiting memory mechanisms that contribute to the processing of longer and more complex sentences. Other studies have also implicated this area in sentence processing with PET and fMRI technology (Bevalier et al., 1997; Mazoyer et al., 1993). Not surprisingly, this is also an area lesioned in many patients with persisting Broca's aphasia. This is not to say that this area is involved exclusively in sentence comprehension. Language processing at the sentence level is extremely complex and undoubtedly involves numerous brain regions and coprocesses.

A feature of more severe Broca's aphasia is that of "recurring utterances," those phrases that are involuntarily produced each time the patient attempts to speak. Here again, we find that Broca's aphasic patients with this characteristic share a common lesion (Dronkers, Redfern, and Shapiro, 1993). Specifically, all had lesions that severed the arcuate fasciculus in a region of the brain where the fibers of this important tract bundle together and ascend out of the temporal lobe to pass over the ventricles. This fiber bundle is susceptible to injuries in the deep parietal region, as even small strokes can completely bisect it, disrupting the transmission of information generated in posterior language zones to more anterior motor speech areas. Classical theory predicts that lesions to this fiber tract would result in repetition deficits. Instead, patients with this lesion cannot speak at all, much less repeat. Aphasic patients without this lesion are not impaired in this way.

Thus we see that trying to associate Broca's aphasia to any one area is futile since this aphasia type is, in reality, a syndrome complex consisting of many different individual deficits. Instead, each specific problem may be related to particular brain areas that subserve specialized functions. The fact that it takes such a large lesion to produce a persisting Broca's aphasia tells us that this lesion must encompass several different brain areas in order to capture all the structures involved in processing the different aspects of language that are disrupted in this syndrome.

With regard to Broca's area, we feel that a more conservative conclusion should be drawn concerning its function. Given that neurosurgical resection and other injuries to Broca's area result in only a passing motor speech deficit, it may be more reasonable to attribute a limited role specifically in articulation to this region, much as Broca himself had originally done. The location of this area, neighboring on motor face cortex in the primary motor strip, makes it a perfect candidate as a motor association area dedicated to the motor control of the speech musculature. It is likely that in our quest to associate deficits in higher linguistic functions to Broca's aphasia, we may have inadvertently assigned too great a responsibility to Broca's area. Another possibility is that it (or neighboring regions) may play a role in working memory for linguistic material (Stromswold et al., 1996) or function as part of an articulatory loop (Paulesu, Frith, and Frackowiak, 1993), as some recent PET studies have suggested.

This is also true of Wernicke's aphasia and the role we attribute to Wernicke's area. We have found that of seven patients with a persisting Wernicke's aphasia in our group of more than 100 chronic left hemisphere aphasic stroke patients, only five have lesions in Wernicke's area (Dronkers et al., 1995). Seven additional patients have lesions in Wernicke's area with no persisting Wernicke's aphasia. Others have found such discrepancies, as well (Basso et al., 1985). In fact, there is quite a bit of disagreement as to where Wernicke's area is (Bogen and Bogen, 1976), since Wernicke himself had no autopsy data on his first patient and the second patient was reportedly demented, with numerous other neuropathological findings.

We have found that, as in Broca's aphasia, it takes a larger lesion encompassing areas outside of Wernicke's area to produce a persisting Wernicke's aphasia. Most of our chronic Wernicke's aphasics have large temporal lobe lesions with total destruction of the posterior half of the middle temporal gyrus, and all have significant involvement of the underlying white matter. Patients with smaller temporal lobe lesions tend to have transient Wernicke's aphasia which resolves to a milder aphasia type within the first year of recovery. Interestingly, these smaller lesions can be anywhere within the posterior half of the temporal lobe and still produce a Wernicke-like aphasia for the first few months. Thereafter, the network of semantic information that resides in the posterior temporal lobe is apparently able to compensate for the hole in its web, with the patient recovering to a milder residual deficit.

With regard to Wernicke's area itself, we find that lesions there lead to the repetition deficits characteristic of chronic conduction aphasia (Dronkers et al., 1998) and not to a persisting Wernicke's aphasia. These patients have difficulty in holding the sentence in echoic memory and repeating these sentences verbatim, but have little difficulty in understanding the meaning of the sentence. They also have trouble deciding whether two words rhyme, particularly when orthographic or semantic cues cannot be used to make the decision (e.g., when the ends of the words do not share the same spelling or when the words are nonsense words with no meaning). Apparently, the echoic trace is lost, preventing patients from hanging onto the sounds of these words, making rhyme judgments difficult and verbatim repetitions of low-frequency phrases nearly impossible. Other work also points to the involvement of the superior temporal gyrus in the perception and immediate store of auditory information (Damasio and Damasio, 1980), while more complicated semantic processing involves larger temporal regions.

Conclusion

In sum, the earlier aphasiologists identified several key areas in the brain that they thought related to speech and language. Our analysis, with more modern tools and assessments, suggests a somewhat different role for these key areas than has been assumed over the years. Our findings indicate that Broca's area is not related to "speechlessness" as Broca claimed, nor necessarily to grammatical processing, as later psycholinguists believed. Its precise role remains to be seen, and current functional neuroimaging may help to cast some light on this issue (see Brown, Hagoort, and Kutas, this volume). Wernicke's area seems to be related more to echoic rehearsal than to "language comprehension," and the arcuate fasciculus to carrying all types of utterances forward to motor speech areas, not just those that need repeating.

Still, the observations of Broca and Wernicke have pointed us in the right direction toward understanding language and the brain. After all, Broca said the posterior inferior frontal gyrus was related to "the faculty of articulate language" and it is clear that frontal areas do contribute to speech output. Wernicke thought the posterior superior temporal gyrus responsible for language comprehension. Though there is some controversy regarding the role of that particular gyrus, it is generally accepted that the temporal lobe is critical for the storage and retrieval of words and their meanings.

In our view, temporal association cortex, in particular, the middle temporal gyrus, is the region of the brain that is most involved with the core components of language.

Lesions to this general area cause the most profound language deficits, as can be seen in our severe Wernicke's aphasic patients. These individuals demonstrate how destruction of large amounts of temporal cortex with extensive white matter damage can lead to permanent loss of important language functions and the effective use of language in both production and comprehension. Small lesions lead to temporary loss, as though the network is able to reorganize itself if the damage is not too extensive. Localization of specific functions within this temporal lobe network will most likely never be possible since it is built on individual experiences that differ from person to person.

On the other hand, there are speech and language mechanisms that are highly localizable, as our work with articulatory planning and echoic memory has indicated. It must be kept in mind that such functions represent input, output, or support mechanisms for the language system and thus would not be expected to vary greatly across individuals. The function of the arcuate fasciculus in transferring information between language areas should therefore also be consistent. The role of the anterior superior temporal gyrus in sentence comprehension also seems to be consistent across patients, but again, is most likely an area that provides support to a very complex process. Brain regions that contribute to such cognitive functions as attention, memory, working memory, and executive control certainly also play their roles in supporting the processes of language.

Finally, in evaluating the validity of the classical model, we must keep in mind how it was derived. Recall that Broca was very interested in whether Gall's idea of language in the frontal lobe could still be supported, even after other phrenological relationships were discarded. When he examined the brain of Leborgne, he was specifically looking for a lesion in the frontal lobes. When he found one, he took it as immediate confirmation of Gall's theory, even though he never cut the brain to see how extensive the damage was. In addition, Broca's patient had apparently suffered numerous strokes and it was never clear which one had led to his "speechlessness." Ironically, such a patient would never be included in today's localization studies. Broca's second patient also had a deeper frontal lobe lesion, though Broca considered only the posterior inferior frontal gyrus to be of importance. His subsequent cases were rarely autopsied, only assumed to have the same lesion.

Wernicke's cases are no more convincing by today's standards. One was demented, with widespread neuropathological changes, while the other patient recovered within weeks and was never autopsied. While many subsequent cases provided support for Broca's and

Wernicke's claims, many were also found to refute them. In fact, the Wernicke model fell out of favor for most of the century and was replaced with numerous other theories. It was not until Geschwind revived the model in the 1960s that it was again brought into vogue.

The last century has seen a blossoming of interest in the neural mechanisms of language and great changes in how these functions are investigated. New technologies and a better understanding of the behaviors we are trying to localize have merged to offer us some necessary modifications to the classical theory. The classical language areas may indeed contribute to language processing, but may do so in slightly different ways. It is also clear that other brain areas besides Broca's area, Wernicke's area, and the arcuate fasciculus can contribute to language processing. Some of these areas determined by lesion analysis include the insula, the anterior superior temporal gyrus, and a larger swath of temporal cortex than previously thought. These modifications do not belittle the older model in any way; they merely offer us the opportunity to improve upon it and to come to a better understanding of the areas involved in speech and language.

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