Language deficits due to brain damage

With all these caveats in place, let me turn to a brief survey of what *is* known about language and the brain, and what sorts of conclusions might be drawn.

The study of language deficits due to brain damage has a long history. In fact, the localization of language in the brain was one of the earliest examples of localization to be discovered, and it served as a major impetus to other brain research.

In 1864, the French surgeon Paul Broca showed that patients with a particular set of linguistic deficits proved upon autopsy to have damage to a particular area in the left frontal lobe. He also pointed out that corresponding right-hemisphere damage showed little effect on speech. The area in question has come to be known as *Broca's area*, and the set of symptoms as *Broca's aphasia*. Broca's aphasics seem to understand what is said to them, but their speech is slow, effortful, and poorly articulated. They have evident difficulty in finding words. Here are two representative quotes from Broca's aphasics (which in the transcripts I have available do not record the problems in pronunciation):

(1) *a* Me . . . build–ing . . . chairs, no, no cab–in–nets. One, saw . . . then, cutting wood . . . working . . .
b Cookie jar . . . fall over . . . chair . . . water . . . empty . . . ov . . . ov . . . [Examiner: "overflow"] Yeah.

Notice that these quotes are not just slowed-down sentences. They are missing a great deal of the grammatical tissue that holds normal speech together—things like articles, auxiliary verbs, and tenses. For this reason Broca's aphasia is also called *agrammatism*. (I should add that sometimes Broca's aphasics can still swear fluently, and they can often sing.)

A quite different set of symptoms was identified by Karl Wernicke in 1874. In people with *Wernicke's aphasia*, Broca's area is intact, but there is damage in the left temporal lobe, in an area now called *Wernicke's area*. The speech of Wernicke's aphasics is altogether fluent—if anything, it tends to come out in a big rush. Taken a few words at a time it often makes a little sense, but the larger parts don't fit at all, and there are often insertions of nonsense words:

(2) *a* [Examiner: "What kind of work have you done?] We,

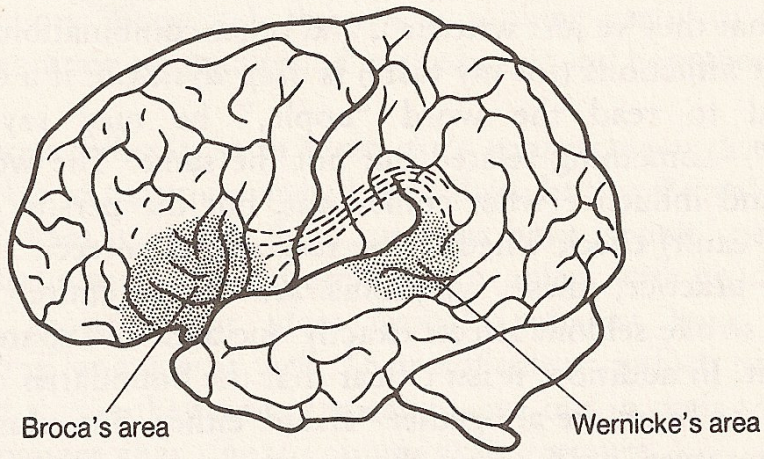

Figure 11.1 *The areas of the left hemisphere relevant to language*

the kids, all of us, and I, we were working for a long time in the . . . you know . . . it's the kind of space, I mean place rear to the spedwan . . . [Examiner: "Excuse me, but I wanted to know what work you have been doing."] If you had said that, we had said that, poomer, near the fortunate, forpunate, tamppoo, all around the fourth of martz. Oh, I get all confused.

b Well, this is . . . mother is away here working out o'here to get her better, but when she's working, the two boys looking in the other part. One their small tile into her time here. She's working another time because she's getting, too.

Not only don't Wernicke's aphasics make much sense, they don't seem to understand much of what you say to them. For instance, for the most part they don't follow instructions well. By contrast with Broca's aphasics, who are painfully aware of their deficits, Wernicke's aphasics often seem unaware that they aren't making sense, and they become irritated at people who can't understand them.

There are many other sorts of language deficits due to brain damage. *Anomic aphasics* have word-finding deficits, more extensive versions of the experiences we all have occasionally in not being able to think of a word or a name. *Conduction aphasics* have relatively fluent meaningful speech, but with many errors of pronunciation; and, oddly, they have great difficulty repeating sentences spoken to them. A whole class of deficits involves reading: there are patients

who can speak but not read, read but not write, write but not read (even what they've just written!), and other combinations. One of the strangest afflictions (for my taste) is *deep dyslexia*: if a deep dyslexic is asked to read the word "apple," he may say "fruit" or "flower"—something related but not the same. The word seems to get in and influence what comes out, but the person doesn't (and possibly can't) check whether the response is correct.

In practice, these symptoms are rarely entirely "clean": a random stroke seldom affects exactly one area and spares everything around it. In addition, it isn't clear that the boundaries of any of the areas in the brain are altogether "clean" either. But, along with most of the literature, we'll accept the conventional idealizations for now.

What has gone wrong in these aphasias? One widespread approach seeks to view aphasia as a disorder of general conceptual thinking. There are indeed afflictions of *dementia* that result in generalized diminutions of cognitive functions, usually as a result of widespread brain deterioration due for instance to Alzheimer's disease. These may simultaneously affect naming, ability to pantomime, purposeful movement, memory, and reasoning. But aphasias are not like this: they often leave the nonlinguistic abilities intact. And, conversely, the ability to produce grammatical sentences is often spared in the face of other substantial cognitive loss due to brain damage. This points to language ability being quite distinct from general-purpose cognitive functioning.*

Wernicke himself proposed a different account of aphasia. He observed that Wernicke's area is near the area of the brain involved in hearing, and that Broca's area is adjacent to the area that controls motor movements of the vocal tract. He suggested, therefore, that Wernicke's area stores the auditory memories of words, and Broca's area stores the memories for how to pronounce them. This nicely explains the fact that Wernicke's aphasics can articulate language but can't understand it (even their own), and that the reverse is true of Broca's aphasics.

However, while Wernicke's account acknowledges the specialization of language ability, it is still inadequate. Language doesn't just consist of knowledge of the sounds of words and how to pronounce them. Rather, auditory and motor abilities are only the most superficial part of language. Most of the interest is in the abstract

* Notice that this evidence for the specialization of the language capacity parallels the evidence drawn from the different varieties of genetic brain impairment discussed in Chapter 9.

phonological and syntactic patterns that organize both perception and production. Wernicke's theory has nothing useful to say about how these abilities are manifested in the brain, and how they could be disrupted.*

A serious difficulty with Wernicke's approach was discovered in the 1970s. Up to that time, it had been thought that Broca's aphasics understand what is said to them, and just have trouble speaking. So Broca's aphasia was viewed purely as a problem with production. It turns out, though, that most Broca's aphasics have problems understanding language as well. Their difficulties, in fact, turn on just the sorts of things they most typically leave out in their speech: the parts of language that signal grammatical structure.

Here's one kind of experiment that reveals this comprehension deficit. Suppose we show Broca's aphasics pictures of a boy hitting a girl and of a girl hitting a boy. Then we ask them: "Which of these pictures goes with the sentence I'm now going to say to you?" If we say (3a), they get the right answer, but if we say (3b), they choose randomly.

(3) *a* The boy hit the girl.
b The boy was hit by the girl.

Apparently, the "was" and "by" of the passive sentence (3b), both functional words, are causing them difficulty.

Similarly, Broca's aphasics have trouble telling the difference between sentences (4a) and (4b). The reason, evidently, is that they cannot take account of the functional word "the," so both sentences sound to them like (4c).

(4) *a* He showed her baby the pictures. (Who saw the pictures? The baby did)
b He showed her the baby pictures. (She did)
c He showed her baby pictures. (ambiguous)

(Incidentally, notice that Broca's aphasics are not simply *ignoring* functional words; if they did, they would always guess that (3b) means the same as (3a). So there is some more subtle story here.)

Since Broca's aphasia usually affects both comprehension and

* You can't blame Wernicke; hardly anything was known about the psychology of language in 1874. In the light of what is now known about language, though, it is hard today to condone explaining aphasia by means of Wernicke's hypothesis, as was done in a recent public television series on the brain.

production, it looks as though it is a deficit in a part of the mental grammar that is shared between the two—in the processing of phonological or syntactic structure. By contrast, Wernicke's aphasia appears to be a disruption of the linkage between language and thought—also in both comprehension and production. Returning to our functional diagram of the crude organization of language, we can place these aphasias approximately in the regions designated in Figure 11.2.

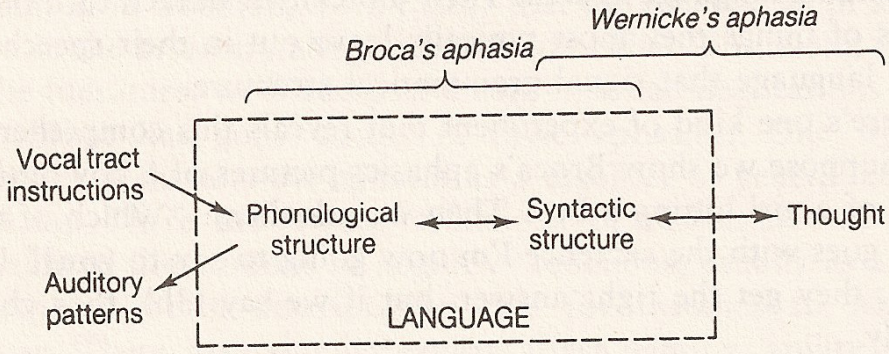

Figure 11.2 *The areas of linguistic information affected by Broca's and Wernicke's aphasias*

There have been lots of fascinating experiments that attempt to pin this down further—to develop theories about *exactly* what part of mental grammar is disrupted in Broca's aphasia. None of these theories has yet proven entirely satisfactory, though we have learned a lot about Broca's aphasia from them. It would take us too far afield here to explain the various theories and how they differ. However, given that Broca's aphasics have both phonological and syntactic problems—and that more specific deficits in either phonology or syntax are not so frequently attested—it may not make sense to seek a single grammatical problem in Broca's aphasia. Rather, some researchers are coming to the conclusion that Broca's aphasia is a general deficit in the ability to process fine details of linguistic structure. In addition, it may be that phonological and syntactic abilities are closely interspersed in Broca's area (in adjacent layers of cortical tissue, say), so that a lesion to one almost inevitably affects the other as well.

In any event, even for the intensively studied Broca's aphasia, it's still hard to draw a clear relation between the deficit we observe, the affected brain area, and the functional organization of grammar.

So much the worse for the other aphasias, which have received considerably less attention from neurolinguists.

Aphasia in ASL

In one of the most spectacular findings of the last ten years, it has turned out that precise analogues of Broca's and Wernicke's aphasias occur in brain-damaged speakers of ASL—and parallel brain areas are affected! An ASL Broca's aphasic signs slowly and leaves out all the grammatical inflections of location and style of movement; an ASL Wernicke's aphasic signs fluently but confusingly, and shows obvious comprehension problems. Not only that: sign language aphasics can produce and comprehend pantomime despite their language deficits, and they're fine at using their hands for purposes other than language.

And not only *that*. There exist *right*-hemisphere deficits that impair one's understanding of space, typically producing a so-called *left neglect*. People with these deficits fail to see things in the left half of their visual field, fail to draw the left-hand side of pictures, and fail to dress the left-hand side of their bodies. But if they happen to be ASL speakers, they still use the left side of the space in front of them *just for the purpose of signing ASL syntax*. Another kind of right-hemisphere damage can lead to loss of the ability to produce facial expressions. But despite such damage, ASL speakers can produce the aspects of facial expression that are relevant to ASL grammar, using the very same muscles.

In each of these cases, the differential character of the deficit shows that it is not a case of motor paralysis: the muscles can still be activated. What is damaged is the part of the brain that organizes the use of the muscles into coherent actions. And it turns out that the part of the brain organizing *ASL* action is not the same as the part that organizes ordinary action.

You can see why these results are so exciting. They confirm just about all the main points we made about sign language on the basis of the functionalist approach, but now adding evidence in terms of brain damage.

1. ASL is a language, not a collection of pantomimes and facial expressions. And it is localized in the language areas of the brain, in a different place from pantomimes and facial expressions.

2. The use of space in ASL is grammatical, not just a collection of pointing gestures. And the use of space for pointing at things out in the world is localized in a different place in the brain from the grammatical use of space for ASL.
3. The grammar of ASL is, with only a few exceptions, entirely parallel to the grammar for spoken languages. And brain damage to English and ASL speakers produces parallel deficits.
4. The fact that signed and spoken grammars have similar organization shows that mental grammar is abstract—it does not have directly to do with auditory and vocal tract function. The existence of sign language aphasias shows vividly that Wernicke was wrong about the function of the language areas—they have to do with abstract *linguistic* function, not especially with the auditory–vocal channel.

All the research on aphasia, both spoken and signed, is remarkable—I can't stress enough how hard it is to work with brain-damaged subjects and to obtain reliable experimental results. Yet this research suffers from some of the same limitations as the rest of neuroscience: even if we can identify a particular deficit in function as due to damage in a particular brain area, this doesn't tell us how that brain area works. We don't know how the neurons are wired up to do what they do. Ultimately, that's what we have to understand in order to explain how language (or anything else) works in the brain.

Brain variation and plasticity

What I've said so far about the brain localization of language applies most reliably to right-handed adults who have no left-handers in their families. With other groups—children, left-handers, and right-handers with left-handed relatives—the probable severity of aphasia from damage to the "language" areas in the left hemisphere is lower, and the probable recovery from such aphasias is better.*

What's the difference? There are three possibilities: In children, etc., either (1) language is encoded more diffusely in the left hemisphere; or (2) language is encoded to some degree in the right

* Some researchers also include women among the "exceptions" to the generalizations about brain localization of language; but the evidence is less conclusive.