4 Aphasia: classification of the syndromes

Introduction

The human brain is well protected by the skull. Yet there are a number of possible ways for the brain to become injured. During a collision, the brain can be smashed against the skull with enough force to create a "closed head injury." Something (a bullet, knife, piece of metal, etc.) might strike the skull with enough force to penetrate it. Or the problem could originate inside the skull, with infection, tumor, or broken blood vessels damaging brain tissue. No matter what the cause of the brain injury, it is unlikely that the entire brain will be equally affected. Some areas will be "harder hit" than others.

When the brain is injured, the problems of the patient will vary depending on the extent and location of the damage. A particular injury might cause only visual problems or problems only in moving certain sets of muscles. The injuries of particular interest to us in this chapter are those that cause problems with language. In our efforts to understand the brain representation for language, we will need to look carefully to see which locations in the brain will lead to language problems after injury and which locations will not. As noted earlier, language deficits acquired after brain injury are called "aphasia." We will see that not all "aphasics," that is, people with aphasia, have the same symptoms.

The most devastating kind of linguistic deficit is the total inability to communicate using language. The patient cannot speak more than a few words or syllables, and understands very little. When this type of deficit persists, it is referred to as "global aphasia" and is

usually the consequence of damage to large portions of the left hemisphere of the brain. However, not all patients who are completely unable to communicate immediately following a brain injury are true global aphasics. In some cases, the patient's condition resolves over weeks or months into partial deficits. In rare instances, there can also be complete recovery. In patients who do experience complete recovery of their linguistic abilities after being totally aphasic in the time immediately after their injury, we see evidence that linguistic competence – their inner knowledge of language – may be preserved even in cases of severe problems with "performance," that is, impairment in speaking or understanding language. The only alternative explanation would be that the patients somehow reconstructed their grammar in the relatively brief time between injury and recovery.

Patients with less extensive damage will, as a rule, have less extensive loss of linguistic abilities. To the extent that different sets of symptoms are associated with injuries in different brain areas, we can develop a more detailed map of language functions in the brain. In aphasia, we see language difficulties without cognitive impairment. Such patients may produce only sparse and disconnected words, but have no trouble on non-verbal tests of IQ, nor trouble cooking, or walking a complex route home. Other brain-damaged patients may, by contrast, exhibit cognitive impairment without linguistic difficulties. These patients produce and comprehend language well, but show problems on tests of memory for visually presented non-verbal material, puzzle completion, and other non-verbal IQ tests. Such problems affect daily tasks such as cooking, memory for common routes, and the like. In light of the linguists' concept of a mental grammar made up of various subcomponents (phonology, syntax etc.), we might expect language breakdown to occur along exactly these lines. One patient might have trouble with sentence formulation and another with word formation or pronunciation. We might also expect processing problems to be different from production problems. And in fact we believe that the differential sparing of phonological, morphological, syntactic, and semantic abilities in aphasic patients speaks grossly for the organization of linguistic components as described by linguists (see chapter 11). However, when we look at the actual

language produced by people with brain injuries and at their comprehension abilities, we begin to see that the correspondence between symptoms and site of injury, and between linguists' grammars and patients' deficits is not nearly as simple as it might be.

In the remaining pages of this chapter we will present some general facts about syndromes – common clusters of symptoms – seen in aphasia in monolingual adults, and the brain areas they are associated with (see summary Table 4.1). This chapter will provide the background necessary for an appreciation of the more detailed studies presented in the next chapter. In that chapter we will treat specific symptoms like agrammatism and look at some interesting controversies, where the language produced by a group of aphasics could be explained by more than one possible understanding of language organization.

Broca's aphasia

In his famous 1861 paper the French neurologist, Broca, presented data from a patient called "Tan" or "Tan Tan" in the literature, as "Tan" was the only syllable he could speak. Tan repeated "Tan" as necessary, with good intonation, as if to convey a message. His comprehension was relatively spared, and he appeared irritated he could not get his message across. A post-mortem examination of his brain showed the lesion – the area of brain damage – to be confined mostly to the lower areas of the left frontal lobe. This area became known as Broca's area (see Figure 1.1). Great difficulties with producing speech became known as Broca's aphasia, although more characteristic than the extremely severe aphasia of Tan Tan is a somewhat milder form. The classic Broca's aphasic in today's taxonomy is considered "non-fluent" in that his speech is slow, deliberate, and effortful, often with omission of grammatical markers (e.g. "Boy go store" instead of "The boy has gone to the store"). Yet comprehension is spared.

Consider the following speech sample from a Broca's aphasic. The patient, L.M., was a sixty-four-year-old man who had a stroke which damaged a large portion of his left hemisphere, leaving him paralyzed on his right side. He also experienced a number of language problems including some problems with naming and

Table 4.1. *Aphasia symptoms*

Syndrome	Speech	Comprehension	Repetition	Naming	Lesion site[a]
Broca's aphasia	poor, non-fluent	good	poor	poor	anterior
Wernicke's aphasia	fluent, empty	poor	poor	poor	posterior
Conduction aphasia	fluent	good	poor	poor	arcuate fasciculus
Anomic aphasia	fluent with circumlocutions	good	good	poor	anywhere
Global aphasia	virtually none	poor	poor	poor	large
Transcortical motor aphasia	little	good	good	not bad	outside in frontal lobe
Transcortical sensory aphasia	fluent	poor	good	poor	outside in parietal lobe

Note: [a]*Lesion site* refers to areas in or relative to the language zone delimited in Figure 1.1. Thus "anterior" means "the anterior part of the language area;" "anywhere" means "anywhere in the language area."

repetition. He was able to understand everyday conversations but did not do well on tests of comprehension of complex syntax. His most marked difficulty was in the production of speech. As part of his evaluation he was asked to describe the picture below which is known as the "cookie theft picture" from the Boston diagnostic aphasia examination (Goodglass and Kaplan, 1972). (See Figure 4.1) His description follows (the examiner's remarks are in square brackets):

> kid . . . kk . . can . . . candy . . . cookie . . . caandy . . . 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 [The examiner asks: What is she doing with the dishes?] discharge no . . . I forgot . . . dirtying clothes [?] dish [?] water . . . [The examiner probes: What about it?] slippery water . . . [?] scolded . . . slipped

In this brief excerpt of the patient's speech we can see some of the common features of the speech of non-fluent aphasics. His speech is effortful with pauses, false starts, and unclear words. He seems to have some word-finding difficulty ("discharge" for 'washing dishes') and he seems to be aware of his difficulties ("no . . . I forgot"). Only a few stock phrases are repeated smoothly ("easy does it" and "that's all," for example). Nouns are the most common words in this excerpt, however verbs also occur relatively frequently. Functors (that is, articles, prepositions and other free grammatical morphemes) as well as bound morphemes (that is inflectional and derivational affixes) are rare.

Wernicke's aphasia

In 1874 the German neurologist Carl Wernicke presented information on two patients whose speech was markedly different from that of Broca's patient. Their speech was relatively "fluent" – that is, the intonation and pace appeared normal – but it contained unusual semantic features. The patients would frequently use elaborate descriptions, called "circumlocutions," instead of

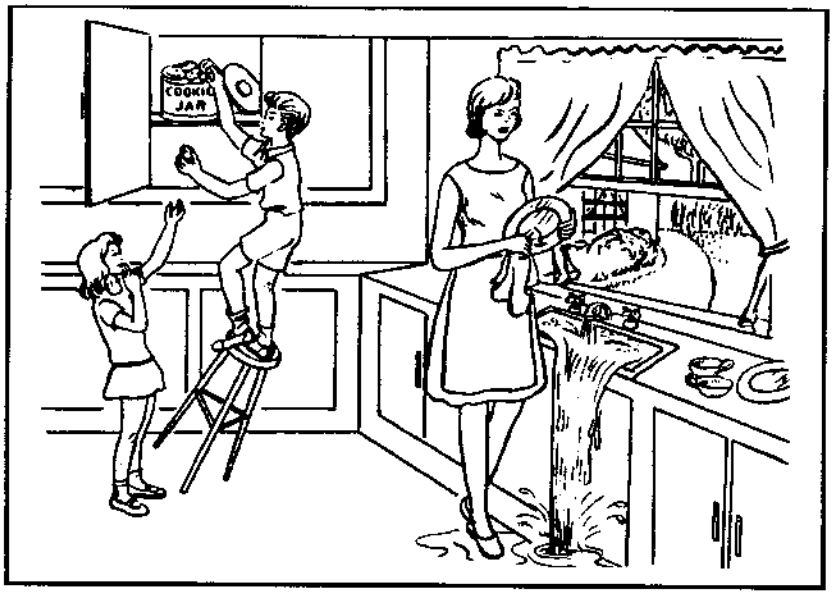

Figure 4.1 The Cookie Theft picture of the Boston Diagnostic Aphasia Examination. (Reprinted from Goodglass and Kaplan, 1983.)

simple words. Sometimes words would be only barely recognizable because of phonemic substitutions. At other times the patients would create new words altogether; these came to be called "neologisms." Unlike Broca's patient, Tan Tan, whose comprehension seemed unimpaired, the comprehension of these patients was severely impaired. Their lesions were posterior to the lesion Broca had identified in Tan Tan; in Wernicke's patients the damage was in the area at the back and top of the temporal lobe now known as Wernicke's area. The collection of symptoms he described is now known as Wernicke's aphasia, and it is characteristic of damage to Wernicke's area (see Figure 1.1).

The following sample from the speech of A.M., a seventy-five-year-old man with Wernicke's aphasia, presents a striking contrast to the speech of the Broca's aphasic, L.M. Although pauses and word-finding problems are also found, A.M.'s speech flows much more freely. Grammatical morphemes occur quite frequently, although overall sentence structure can be somewhat bizarre.

An excerpt from an interview with him follows. A.M. has been asked what brought him to the hospital. The examiner's interpretations are in square brackets:

> Is this some of the work that we work as we did before?... All right... From when wine [why] I'm here. What's wrong with me because I... was myself until the taenz took something about the time between me and my regular time in that time and they took the time in that time here and that's when the the time took around here and saw me around in it it's started with me no time and then I bekan [began] work of nothing else that's the way the doctor find me that way....

Two problems apparent in the speech of A.M., which we did not find in the speech of the non-fluent aphasic, are misselections of the sounds of words (called phonemic paraphasias – e.g. "wine" for "why") and a lack of meaningful content. Although one can find phonemic paraphasias in Broca's aphasics, they occur with far less frequency. The characteristic "off" syntax (e.g. "What's wrong with me because...") is called paragrammatism.

Conduction aphasia

The most widely discussed other aphasic syndrome is "conduction aphasia." The key symptom of conduction aphasia is an inability to repeat spoken language. It was originally conceived of as a disconnection of Broca's and Wernicke's areas due to damage to the structure that connects them known as the arcuate fasciculus (see Figure 1.1).

In conduction aphasics, a relatively spared Broca's area was thought to control the motor functions necessary for producing spontaneous speech, and a relatively spared Wernicke's area was thought to allow for good comprehension. Repetition, however, requires rapid communication between the two areas via the arcuate fasciculus according to this model, so the patients' ability to repeat was impaired. Since it is unusual to find a lesion restricted exactly to this area, patients might have some comprehension and/or production difficulty as well, but their repetition would be markedly more impaired. In the next chapter we discuss the more current understanding of conduction aphasia.

Anomic aphasia

All patients with aphasia of any type have anomia, that is, problems remembering the names of things, but one set of relatively mildly impaired patients are called "anomic aphasics" because their naming problem is their only language problem. Asked to name the picture of a pen, for example, an anomic patient might say "Oh, right, one of those things you use for writing – not, a pencil – I have one right here." The cognitive psychologist Ashcraft (1993) wrote about a temporary anomia he experienced as the result of an aneurysm – a ballooning of one of his brain's arteries that drew blood away from his language area. One day Ashcraft was sitting at his desk when his assistant came in to ask him what to do with a computer printout. He realized he was unable to name the experiment it referred to, although he knew exactly which one it was, nor could he say the words "printout," "experiment," or "data" despite the fact that he used these words quite frequently. When he turned to the computer to log-off, he was unable to remember the command "logoff." Although he was not particularly worried at his inability to locate these words, he realized something was wrong and tested his physical abilities by walking to the bathroom and back. He called home and, because he was speaking hesitantly, his wife asked if he was okay. He said, "I guess I'm confused," but was unable to explain how he could no longer remember words. After he started several sentences with non-substantive words, e.g. "well, we were," his wife insisted that he be driven to a hospital.

About 40 minutes after the beginning of the incident, just as he was about to leave his office with his assistant, he looked at the computer and now found he could log-off easily. Also, the words he had been looking for returned and he said them aloud to himself to assure himself that he could. In the hospital, it was determined that an arterio-venous malformation in the anterior left temporal lobe had diverted blood from nearby brain tissue, thus resulting in the transient anomia. In anomic aphasia, then, relatively small lesions anywhere within the language area are seen to result in difficulty finding specific substantive words. Syntax remains unimpaired, however, and comprehension is quite spared.

Other cortical syndromes

In addition to Broca's, Wernicke's, and conduction aphasia, other standard syndromes include "pure word deafness," "alexia," "transcortical motor aphasia" and "transcortical sensory aphasia. Pure word deafness is an inability to make sense of oral language in a person with normal hearing. Asked "What did you eat for breakfast?" the patient may respond "Breakfast. Breakfast? It sounds familiar but it doesn't speak to me." This syndrome results from an injury to Heschl's gyrus. Similarly, when visual linguistic stimuli are not processed due to injury to the angular gyrus, the resulting deficit is called "alexia." The pure alexic can speak and understand language well but can no longer read. (See chapter 9.)

Transcortical motor aphasia and transcortical sensory aphasia parallel Broca's and Wernicke's aphasias respectively. However, in the transcortical syndromes, repetition is entirely spared because the lesions are beyond ("trans") the language area. Patients with transcortical motor aphasia will initiate little language and what they say will be fragmentary although not agrammatic (e.g. Examiner: Can you tell me the story of what brought you to the hospital? Patient: A stroke). Their comprehension is relatively spared. Patients with transcortical sensory aphasia have poor comprehension and fluent but semantically empty speech, except when they are repeating, of course.

Subcortical aphasias

We have metaphorically "only touched the surface" in our discussion of language deficits after brain damage so far. We have presented each of the aphasias above in terms of damage to the cortex or "gray matter." However, as we saw in chapter 3, there are a number of subcortical ("white matter") areas thought to be involved in normal language. Characteristic of the subcortical aphasics is the clinicians' inability to decide if the patients are "fluent" or "non-fluent." Such patients do not produce large amounts of language uninterruptedly and with exuberance, as a Wernicke's aphasic will; their speech is sparse and slowed, although grammatically correct.

Alexander and Naeser (1988) describe a set of subcortical aphasias associated with damage to different subcortical structures. Patients with lesions in the insula/internal capsule area show a mild fluent aphasia, similar to conduction aphasia, with phonemic substitutions (e.g. *hand* pronounced as /hæld/) especially in repetition and oral reading. These authors describe symptoms similar to those of transcortical motor aphasia in patients with white matter damage farther forward in the brain. Such patients appear to have an intact grammar, but they have sparse output. They seem to have lost the "drive to speak." The authors show that auditory comprehension deficits may result from lesions in the temporal isthmus (see Figure 2.6). Finally, they point out that with enough subcortical damage a patient may even be globally aphasic. Alexander and Naeser note that many previously confusing findings with respect to symptoms and lesion site may become understandable with reference to subcortical damage. For example, a patient with agrammatic production and a lesion in Broca's area may show comprehension deficits more usually found in Wernicke's aphasia as a result of damage to the temporal isthmus, because that connects Wernicke's area to Broca's area.

Special patterns in aphasia

All of the questions of brain maturation and brain representation for language become more complex when we consider other populations. For example, speakers of a visual-gestural language must process and produce spatial information, often considered to be a right hemisphere function. Does this lead to more significant right hemisphere involvement in speech/language?

The weight of evidence in the literature would seem to support similar but not identical brain representation for signed and spoken languages. Corina et al. (1992) report on their study of a left-lesioned, native signer (WL). Although WL's post-stroke ability to pantomime and interpret *gestures* was essentially intact, he demonstrated marked aphasic symptoms in his signing. He had a Wernicke's-type aphasia with comprehension difficulties, neologisms and paraphasias parallel to those found in hearing patients.

Paul D., another aphasic signer reported on in Vaid and Corina (1989), had more Broca-like symptoms, with frequent, missing inflection. This patient also experienced intrusions by his left hand in signing, suggesting possible right-hemisphere influence.

J. Sarno et al. (1969) report the case of aphasia in a man deaf from birth who had acquired some speech through his five years of schooling in a school that promotes oral language for the deaf, as well as some American Sign Language and finger spelling. At the age of 69, he had a stroke that resulted in severe aphasia, apparently from anterior brain damage. Like a hearing individual with this sort of lesion, his comprehension in all modalities was better than his production. As a Broca's aphasic might point to his mouth in frustration that it does not say what he wants, this patient would point to his right hand. Indeed, like most bilingual aphasics (see chapter 10) his ability in his various modalities seemed to be proportionate to his abilities before the aphasia-producing stroke: he was best at signs, not so good at finger spelling, and particularly poor at lip-reading which had been his poorest modality before his stroke. As to his production of language, speaking was worst, writing and finger spelling were medial. Combining signs, finger spelling, and a bit of vocalization worked best for him.

Bilingual speakers are another population for whom the question of unusual brain organization has been raised. If a person grows up with two languages, do the two languages share "brain space"? Do the same left-hemisphere regions important for monolingual linguistic abilities support bilinguals' languages as well? Might the right hemisphere be more involved in language perception/production by bilinguals? Do people who begin learning a second language later than the first acquire similar brain representation? After brain damage do the two or more languages manifest the same sort of aphasia? We postpone a more full discussion of these issues until chapter 10.

Women aphasics constitute another "unusual" group, since so much of our knowledge about aphasia derives from war injuries and strokes, both more common in men than women. McGlone (1977) and Kimura (1983, 1993) have argued that incidence of aphasia is somewhat less in women than in men, even when the

lesser incidence of stroke among women is accounted for. Kimura and Harshman (1984) have also reported that the language area in women seems to be somewhat anterior to that of men. However, many studies find no differences between aphasia type or lesion size between the two genders (e.g. M. T. Sarno et al., 1985, Kertesz and Benke, 1989).

Hier et al. (1994) found small gender differences in aphasia following stroke, consistent with Kimura's notion. Also, Broca's aphasia was more frequent in men while the fluent aphasias (Wernicke's aphasia and anomic aphasia) *and* global aphasia were more frequent in women. Moreover the size of the brain damage required to result in aphasia was greater for men than for women, suggesting somewhat more diffuse organization of language in them (although this could relate to larger overall brain size – due to larger overall body size – for men as well); the authors note that the size of stroke damage on average was the same in men and women. In sum, the results are not in on subtle differences that may obtain in aphasia type and location between the genders.

Conclusion

The different aphasia syndromes are linked to damage in different areas of the central left hemisphere. Problems in coming up with specific lexical items arise with mild damage anywhere within the "language area" around the Sylvian fissure. Problems with producing the sounds of language correctly and in generating syntactic strings of words are associated with predominantly anterior lesions including Broca's area. Problems with comprehension and "empty" speech are associated with damage to posterior regions around Wernicke's area. Problems with repetition can arise with damage to either of these areas, but problems exclusively or predominantly with repetition arise when the pathways between the two areas are damaged. Damage to subcortical structures that underlie the language areas can also result in aphasia by cutting links crucial for producing language.

5 Aphasia: what underlies the syndromes

The previous chapter gave a general overview of the types of symptoms seen in aphasia. We discussed some early ideas about what these symptoms meant for theories of brain representation for language. More modern research has allowed us a closer look at injured brains in live patients and a more developed, theoretical basis for creating language tests for aphasic patients. In this chapter we consider the explanations that have been suggested for agrammatism, Wernicke's aphasia, the diagnostic dichotomy *fluent*: *non-fluent*, and conduction aphasia.

Agrammatism

A subset of patients with Broca's aphasia fit the criteria for agrammatism that is, speech which is essentially devoid of appropriately used closed class or function words. The speech of these patients is generally slow and effortful. Some may also have phonetic difficulties. Early research on the nature of the deficit in agrammatism referred only to these production problems. More recent studies have turned up subtle comprehension deficits as well. Not all patients experience the same problems to the same extent. For this reason, there is some disagreement about the status of agrammatism. Some researchers say it is a collection of unrelated symptoms each of which might be more profitably studied separately. Others consider the production deficits definitional and are unconcerned with the other aspects of agrammatics' linguistic abilities. Still others attempt explanations which would account for all of the observed symptoms.

Consider the questions that arise from speech samples such as that in chapter 4 from L.M., the patient with Broca's aphasia. Recall that he makes frequent pauses and shows some transient difficulty with articulation ("can . . . candy"). Might all of his difficulties be attributed to an inability to reliably activate the neurons needed to move the speech muscles? He also shows some lexical disturbances, however. In fact he says the word "candy" when he wants to say "cookie." When the examiner asks him about what the woman was doing with the dishes, and the answer is "washing them" he says "discharge my . . I forgot . . dirtying clothes." It is possible that he is simply having trouble remembering the words he wants. Alternatively, perhaps the representations for many words in his lexicon are now incomplete. In that case, a lack of subcategorization frames – information about what sorts of nouns verbs must take – might account for L.M.'s inability to produce well-formed sentences.

Different parts of speech seem differentially affected. While he has some whole phrases, they are formulae, e.g. "well, I don't know." When he is actually trying to describe the picture, he is much more likely to use verbs and nouns, rather than modifiers or functor words. Does this mean that these word categories were formerly stored in different brain areas? Or does their different rate of occurrence in L.M.'s speech result from a difference in the roles of, say, nouns and adjectives or prepositions and nouns in some post-lexical stage of speech production?

Finally, we noted that L.M. did not experience difficulty in comprehending everyday conversations but performed poorly on tests of understanding complex syntax. For example, if he were asked "The lion was killed by the tiger; who died?" he is more likely to pick the tiger than the lion. To what extent do his comprehension difficulties parallel his production problems? Do phonological features of words play a role in his comprehension problems? Can he understand words he is unable to produce in confrontation naming tasks? Are there syntactic constructions not found in his free speech yet comprehensible to him? A brief discussion of some specific approaches to agrammatism should illustrate the progress made in understanding the effects of lesions resulting in Broca's aphasia, as well as the extent of disagreement

as to the proper interpretation of the linguistic disturbances in non-fluent aphasia.

Analysis of the sound patterns in the speech of Broca's aphasics is perhaps the simplest place to begin. It is generally agreed that most of the sound errors in Broca's aphasia result from difficulties in the end stage(s) of speech production. There may be some distortion in the quality of speech sounds. This is referred to as "dysarthria." There may also be some blurring of important distinctions in the patient's language. For example, Blumstein et al. (1977) investigated the voice onset time (VOT, the time when the vocal cords start vibrating relative to the release of the stop) for voiced (e.g. [b]) and voiceless (e.g. [p]) stop sounds in patients classified as Broca's or Wernicke's aphasics. They found that whereas normal speakers and Wernicke's aphasics showed no overlap of onset times across series of voiced and voiceless stops, Broca's aphasics did not have this "buffer zone." Broca's aphasics were inconsistent; for some of their voiceless targets, they actually started their vocal cords vibrating earlier than for some of their voiced sounds.

One interesting attempt at locating all of the symptoms of Broca's aphasia in a single linguistic component was Kean's 1977 theory that agrammatism resulted from a phonological deficit. Kean analyzed the language produced by Broca's aphasics and their comprehension difficulties. She found that the words that created the most difficulties for these patients were those which the phonological component of the grammar did not treat as full-fledged words. Although prepositions, articles and other so-called "function words" do not form a coherent morphological or syntactic class, they are demonstrably different from "content words" phonologically. They generally do not attract stress in a sentence and they undergo some of the same low-level phonetic processes (e.g. vowel reduction) in English. If a Broca's aphasic's linguistic system were only able to deal with full-fledged phonological words, then the patient would omit grammatical markers (whether bound or free morphologically) in production and ignore them in processing. Although this theory was an important contribution, it was abandoned as evidence accumulated that few languages (English and the other languages considered by Kean in her analy-

sis) had this parallel dichotomy of phonological words/non-words and function/content words.

One of the directions taken by researchers in morphological and/or syntactic aspects of agrammatism is to look for dissociations of the various symptoms of the syndrome. Linebarger, Schwartz, and Saffran (1983) studied the ability of agrammatic patients with deficits in comprehension and production and found them surprisingly unimpaired on a grammaticality judgment task. The authors interpreted these results as evidence for agrammatics' preserved syntactic competence; instead, they suggested, agrammatics have a disruption in the assignment of thematic roles to sentence constituents.

Grammaticality judgment tasks generally involve decisions as to the well-formedness of a string after the subject has read or listened to the string. This is different from the usual process of sentence comprehension which must happen as the sentence is being produced. A number of researchers have designed tasks which evaluate the morphological and syntactic "on-line" processing of sentences by aphasic patients.

A 1987 paper by Tyler and Cobb presents the results of a linguistic experiment conducted with their agrammatic patient DE. In this experiment they asked DE to listen for words in sentences and timed how long it took him to respond. Immediately before the word DE was to listen for, there occurred one of three word-types: a complex word consisting of a root plus contextually appropriate suffix (e.g. "waste*ful*" cook), a complex word consisting of a root plus contextually inappropriate suffix (e.g. "was*tage*" cook) or a root-suffix combination which resulted in a non-word (e.g. "wastely"). The suffixes were of two types: derivational – changing the part of speech, e.g. writ*er*, and inflectional – contributing syntactic information, e.g. writ*es*.

When unimpaired subjects did this task, they were slower to find the words when they came after inappropriate suffixes, presumably because they spent some time trying to integrate the peculiar suffix into the sentence context. Since unimpaired subjects have access to both the syntactic information which determines the presence of inflectional endings and morphological information which determines the presence of derivational end-

ings, it is not surprising that this difference was apparent for both derivational and inflectional suffixes.

When DE attempted these word-finding tasks, his responses were fast and accurate, well within normal limits. But his pattern of delays was unlike that of the normal subjects. For derivational suffixes, DE took significantly more time to report having found the target word when it came after inappropriately suffixed words than after appropriately suffixed words. However, after inflectional suffixes, DE showed no difference between appropriate and inappropriate suffixes. If it is true that words with derivational suffixes are listed separately in the mental dictionary whereas inflectional suffixes are listed apart from any roots they might attach to and are simply added on to roots because of the syntactic context in which the root appears, then it would seem that the damage caused by DE's accident has selectively disturbed the lexical representation for inflectional suffixes.[2]

In much the same way that Tyler used an on-line task to test DE's sensitivity to morphological facts about English, Baum (1989) tested the sensitivity of a group of Broca's aphasics to syntactic properties of English. Baum compared the ability of normals and aphasics to listen for target words in long and short sentences. Some of the long sentences and some of the short sentences involved local dependencies (i.e. relationships between constituents in the same clause). The rest of the long and short sentences contained long-distance (across clause boundary) dependencies. Normals showed grammaticality effects in both local and long-distance types. That is, they took longer to respond to words that occurred in ungrammatical contexts than they did to respond to words that occurred in grammatical contexts. The syntactic structure they were building facilitated their word-monitoring. The seven Broca's aphasics in this experiment showed grammaticality effects only in sentences where the crucial context was a local dependency. This could be due to the fact that local violations were not exclusively syntactic; they were also morphological or lexical. It could be because the long-distance dependencies always involved syntactic gaps. Grodzinsky (1984) had posited that, for agrammatics who had syntactic problems with comprehension, these arose from the inability to process syntactic

traces of items that have been referred to earlier in a sentence. Either way, the fact that these aphasics show on-line sensitivity to material which is problematic in their production and/or comprehension is indicative of a processing deficit rather than a structural deficit.

Agrammatics have also been shown to be sensitive to certain syntactic properties of the very "closed-class" elements which are so often missing or misused in their production. Shankweiler et al. (1989) provided evidence of such sensitivity in an on-line task. They asked aphasics and normal controls to judge the grammaticality of sentences and determine the location of any ungrammaticality. The violations were of two types. One involved the substitution of an inappropriate, closed-class word of the same syntactic category (e.g. The good-natured baker put *at* a white hat). In the other type an inappropriate closed-class word of a different syntactic category was substituted (The good-natured baker put *is* a silk scarf). Overall, the aphasics had lower accuracy and longer response times than the controls. This could reflect either a loss of the relevant structures or a deficit in linguistic processing. However, the aphasic and control groups showed similar patterns of response times and accuracy to the two violation types. Both groups were sensitive to this difference. Again, this argues for preserved syntactic knowledge in the agrammatic patients.

Similarly, Blumstein et al. (1991) show that the Broca's aphasics in their experiment were building syntactic structure as they attempted to process sentences in a lexical decision task. The patients' decisions about whether a particular set of letters constituted an English word were significantly slower when the target was found in an ungrammatical setting.

If agrammatic aphasics are attempting to build syntactic structure in processing sentences, what explanations might there be for the impoverished structure of their speech? Zingeser and Berndt (1990) – see also Berndt et al., 1997a and 1997b – suggest that agrammatics may have particular difficulty with verb retrieval in production and thus more trouble with sentence structure since they are not getting subcategorization information. They compare agrammatics to anomics who have a specific difficulty in retriev-

ing nouns. Since nouns do not carry information about particular complements necessary for creating grammatical sentences, the production of patients with anomia remains fairly fluent.

Nouns and verbs do, however, differ in other ways as well. Joanette and Goulet (1991) suggested that the difficulty experienced by agrammatic aphasics in producing verbs may be more appropriately considered a text-level difficulty at the point where sentences are constructed about things (noun phrases: NPs) and what they do (verb phrases: VPs). Verbs carry a greater part of the information in a sentence. They determine which NPs may serve as their subjects (selection restrictions) and which complements are required (subcategorization). Agrammatics may find the more propositional verbs more difficult than the relatively non-propositional nouns.

Berndt and Zingeser (1991) reply that the text-level model that Joanette and Goulet proposed is a discourse-processing model, not a model of sentence production. Since any non-noun might have a propositional function in some discourse unit, it is difficult to see how such a text-level analysis might explain word-class differences in retrieval. The conflicting frameworks within which the two sets of authors work do not allow for a direct comparison, but it is certainly conceivable that both syntactic category and discourse function play significant roles in agrammatic production.

Each of the papers discussed in this section presents a particular dissociation of abilities in agrammatic patients. Specifically, dissociations have been found between comprehension and production difficulties, between a combined comprehension/production difficulty and preserved knowledge of grammaticality, and between various aspects of on-line processing. Taken together, this body of work (along with many other studies not discussed here) points toward a preserved syntactic competence in agrammatism with difficulties arising from damage to only some aspects of processing or production mechanisms.

Not all researchers would agree with this conclusion. Some believe that the evidence suggests that "agrammatism" should not be considered a syndrome. Instead, each of the symptoms, since they are dissociable, should be considered separately.

A particularly interesting set of dissociations is reported by

Caramazza and Miceli (1991) in the comprehension and production of a *fluent* aphasic whose initials are E.B. When asked to point to which of two pictures represents a spoken or written sentence, E.B. had severely impaired ability to assign thematic role – to decide which noun in a sentence is the doer, and which the done-to – in active and, especially, passive sentences. This is similar to the analysis offered for the non-fluent patients in Linebarger et al. (1985). However, phonologically and morphologically, E.B.'s production is essentially normal. The patient's performance on a well-formedness judgment task is also normal. Hence there is a double dissociation of asyntactic comprehension and difficulty in processing closed-class elements. The authors reject the explanation of a simple role-assigning heuristic (e.g. "Make the first noun the agent"). They point out that this would lead to incorrect assignments in what are called non-reversible passives, that is, sentences like "The ice-cream was eaten by the boy," where it is not possible that the boy was eaten by the ice-cream, as well as in the reversible passives (e.g. "The man was pushed by the woman") where errors are actually found. The authors suggest that the actual *representation* of (some) verbs is disturbed in this patient and presumably in other patients with these types of comprehension problems, even though they have frontal lesions and the non-fluent production and relatively spared comprehension usually associated with Broca's aphasia.

Arguments against agrammatism as an aphasia syndrome are also made without appeal to fluent aphasics. Some researchers who have looked at differences in the production and comprehension of non-fluent patients who are speakers of languages other than English have concluded that the differences from language to language are greater than the differences among aphasia types within a single language. For example, in languages where inflectional endings carry more semantic weight than they do in English, non-fluent aphasics omit them less often. (Miceli et al. 1989, Bates et al. 1987, Bates, Wulfeck and MacWhinney 1991.)

One strong proponent of agrammatism as a theoretically coherent category has been David Caplan (1991). He does not deny the variability in the difficulties experienced by agrammatic patients (c.f. Miceli et al. 1989) but rather he claims that this variability is a

result of the broadness of the category "agrammatism." His claim is that there is evidence to suggest that although function words and inflectional morphemes, which are both frequently omitted in the speech of non-fluent aphasics, do not form a syntactic class, there are production mechanisms – yet to be fully described – that treat them similarly.

Grodzinsky (1991) also defends researchers' attempts to make generalizations about agrammatic patients' data. He points out that both clinical and theoretical definitions of agrammatism refer to types of omissions and substitutions, NOT to quantities of omissions and substitutions. As to the suggestion that the relationship between comprehension and production deficits in agrammatism is unpredictable enough to weaken the hypothesis of a single, underlying deficit, Grodzinsky replies that when only those patients who have both non-fluent speech and a paucity of morphosyntactic markers in their speech are considered, the relationship becomes more predictable.

Although Grodzinsky argues for the coherence of agrammatism as a syndrome, he does not agree that syntactic competence is spared with impaired production mechanisms causing the typical difficulties. He asserts that the central deficit in agrammatism is what is called the deletion of traces. Consider the sentence "The girl was pushed by the boy." It can be diagrammed thus if we assume **t** stands for the trace left behind when a word has been moved from its position in an underlying sentence:

$$[\text{The girl}]_i \text{ was pushed } \mathbf{t}_i \text{ by } [\text{the boy}].$$

The sentence derives from "The boy pushed the girl." We understand that the NP "the girl" is the object of the verb "push," in this theory, because it left behind a trace, here labelled "**t**" when it moved from object position. If that trace were deleted in agrammatism, the noun phrase "the girl" would be considered the "doer" rather than "done to" as the first noun in a sentence usually is the "agent."

Clearly the phenomenon of agrammatism forms the crux of several important arguments in neurolinguistics today: (1) whether it even exists as a unified phenomenon, (2) if it does, whether it reflects actual grammatical breakdown, (3) if so, what

grammatical principles and/or structures are affected. Because the field has many researchers debating these questions currently, it is hard to tell when or how these questions will be resolved.

Wernicke's aphasia

In reviewing some of the approaches to the analysis of agrammatic production and comprehension, we see that no one theory is perfectly able to account for all of the data presented. Descriptively, however, it remains clear that there are some robust differences between the speech of Broca's aphasics with more anterior lesions and that of the Wernicke's aphasics with more posterior lesions. Even in cross-language studies aimed at finding similarities between anterior and posterior aphasics, such as Bates, Wulfeck and MacWhinney (1991), "subtle processing differences" between Broca's and Wernicke's aphasics are found.

The linguistic deficits in Wernicke's aphasia tend to be more lexical-semantic. In a study of the sorts of substitution errors made by aphasic patients, Ardila and Rosselli (1993) find that substitutions of an individual phoneme within a word occur only in patients with lesions close to the Sylvian fissure, that is Broca's aphasics (who make a lot of them), conduction aphasics, and Wernicke's aphasics. (These aphasics, as it happens, were speakers of Spanish, although there is no reason to believe that this finding would not hold equally for speakers of other languages.) Patients with anomic aphasias tended to have smaller posterior lesions than Wernicke's aphasics. They made virtually no phonemic paraphasias (substitutions of individual phonemes) and primarily semantic, verbal paraphasias. The Wernicke's aphasics, by contrast, with sizable posterior lesions, made both phonemic paraphasias and neologisms, that is non-words where the target word is unrecognizable. The lesions of the Wernicke's patients also extended higher than those of the anomics, on average. Patients with lesions outside what is traditionally considered the "language area," that is transcortical motor aphasics with lesions in the frontal lobe, made extremely few substitutions. Such an analysis confirms our notion that the language area around the Sylvian fissure is crucial for production of lexical items. Posterior regions

within this area are required for generating target words themselves; the entire path must be available for speaking the entire string of phonemes in the appropriate order.

Recall the sample of speech from A.M. in the previous chapter. His discourse is called "empty," in part, because it is repetitious, but also, more importantly, because it contains "words" – really phoneme strings – that are not words in English, like *taenz*. This phenomenon, associated with posterior aphasics, is called *neologistic jargon*. In their book on the topic, Buckingham and Kertesz cite a severely impaired patient, B.F., who answered the examiner's question "Who is running the store now?" with the following:

> "I don't know. Yes, the bick, uh, yes I would say that the mick daysis nosis or chpickters. Course, I have also missed on the carfter teck. Do you know what that is? I've, uh, token to ingish. They have been toast sosilly. They'd have been put to myafa and made palis and, uh, myadakal senda you. That is me alordisdus. That makes anacronous senda." (Buckingham and Kertesz, 1976: 21.)

At a few points in such a paragraph we think we know what the target is (e.g. when the patient says *ingish* we suspect he may be commenting on his problems with English). At other points, it is unclear whether he is even responding to the question that was asked. Some aphasiologists believe that patients indeed have targets in mind, but are entirely unable to reach them in production, and due to the severe comprehension deficits are unable to monitor their own output, that is, to realize it does not make sense and needs to be corrected. Butterworth (1979), by contrast, suggests that there is a "random phoneme generator" operating that kicks in when target words cannot be found, yet the speaker feels the need to speak.

Note that functor words and inflectional affixes are produced in abundance in Wernicke's aphasia. However, the two instances of "paragrammatism" that can be seen in the passage in the previous chapter ("work of nothing else" and "the doctor find me") do indicate that A.M.'s syntax is at least "slightly off."

Wernicke's aphasics' comprehension is more impaired than their production. They would have trouble, for example, answering a simple question from The Boston Diagnostic Aphasia Exam

such as "Does a good pair of rubber boots keep water out?" Of course, to say that a patient's comprehension is relatively more impaired than that same patient's production is not to say anything at all about the severity of the comprehension deficit. Comprehension can be measured in a number of semantic tasks. Naming of pictures of objects and/or actions may be impaired. Patients may be unable to point to a written representation of orally presented words.

Zurif and Caramazza (1976) established that their Wernicke's patients' ability to make judgments about the relatedness of words was severely impaired. Their task consisted of groups of three words, only two of which would normally be considered semantically related (e.g. husband-turtle-wife). Their patients' choices about which two of the three words went best together was essentially random. However, just as was the case with Broca's aphasics, tasks that involve on-line access of semantic information demonstrate that, although perhaps not available for conscious reflection, patients do retain much knowledge of semantic category.

Tyler (1988) reports that fluent aphasics with impaired comprehension show evidence of the ability to build syntactic structure in on-line tasks. She created a task in which subjects listened for target words in three different types of "sentences." Some were normal sentences. Others were semantically anomalous but syntactically well formed, like Chomsky's famous example "Colorless green ideas sleep furiously." The third type was a scrambled string with no semantic or syntactic structure. We know that normals find words in such a task faster when the words come later in a sentence; the syntactic structure they are building makes the task easier. This is called the "word-position effect." Tyler's fluent aphasic subjects showed word-position effects for the normal sentences and the anomalous ones but not for the scrambled ones. This suggests that they, like the normals, were aided in the word-finding task by the syntactic structure they were building. The only sentences for which they did not show a word-position effect were precisely those which had no syntactic structure (i.e. the scrambled sentences).

If there is spared ability in Wernicke's aphasics to construct

syntactic structures, why then do we see the strange structures of paragrammatism? Deeper aspects of structure may be spared while surface aspects may be impaired. The severe difficulty Wernicke's aphasics have in choosing meaningful, lexical items in their speech renders it hard for us to be sure they intend to produce interpretable propositions, however.

An alternative distinction between non-fluent and fluent aphasics

Jakobson (1941 and 1968) pointed out an alternate way to contrast non-fluent Broca's aphasics with Wernicke's fluent aphasics. His idea derives from the notions that de Saussure developed, distinguishing paradigmatic from syntagmatic aspects of language. Recall that words that are syntagmatically related can occur right next to each other, as in "President Clinton" or "Queen Elizabeth" or "John walked." Words that are paradigmatically related to each other can substitute for each other; words like "president" and "queen" and "officer" are all paradigmatically related; as are words like "he," "it," and "they." As Luria described the problem in 1973, appropriate choice of phonemes and words is a paradigmatic activity because one can select any of a number of possible candidates, while the constructing of these words into sentences is a syntagmatic activity. Thus patients who make literal paraphasias, substituting one or two phonemes into a word, or patients who misselect words, tend to be the patients with posterior brain damage, while patients with problems constructing sentences will be patients with anterior brain damage and non-fluent aphasia.

Luria is quite explicit in linking these "Two Basic Kinds of Aphasic Disorders" (the article's title) to lesion localization; disorders of paradigmatic systems are associated with posterior regions in the language area. Disturbances in the left temporal lobe cortex bring about breakdown in what he calls the *phonematic code*; disturbance in the parietal lobe results in problems with organization of articulatory processes. Disturbance at the temporo-parieto-occipital junction in the left hemisphere results in sparing of the paradigmatic systems for phonemes and articula-

tion, but problems in what Luria terms the semantic system, that is the system responsible for lexical selection. As a consequence, the patient substitutes incorrect words for the target words. All these types of lesions do not result in any problems in the syntagmatic system; those problems are the result of more anterior lesions. With brain damage in anterior speech areas, by contrast, paradigmatic abilities such as phoneme selection and lexical selection are relatively spared, while the ability to construct words into sentences is impaired.

Conduction aphasia

In the previous chapter, we discussed a particular kind of aphasic syndrome in which the principal difficulty is neither production nor comprehension but rather repetition. This syndrome, conduction aphasia, was, we mentioned, previously attributed to a damaged arcuate fasciculus, the structure thought to be responsible for "conducting" information from the comprehension area (i.e. Wernicke's area) to the production area (i.e. Broca's area). Our modern imaging techniques have shown us that not all patients who have a particular difficulty with repetition have damage to the arcuate fasciculus.

Most striking, in addition to the inability to repeat, is conduction aphasics' phonemic paraphasias, that is, their substitution of phonemes within their target word. Characteristically, the conduction aphasic will approach the target word in successive attempts: e.g., Goodglass gives the following example of a patient trying to name a whistle "tris . . . chi . . . twissle" (Goodglass, 1993, page 142). It would appear that the patient has information about the target lexicon in mind, but is unable to assemble it in production. Indeed, Kohn (1984 and 1992) has suggested that the problem lies in the stage of "programming" the motor articulation of the planned word. Conduction aphasics, unlike Wernicke's aphasics, are distressed in their awareness that they have not succeeded in producing the correct word.

Pate, Saffran, and Morton (1987) examined the errors made by one conduction aphasic, NU, in great detail. In tasks like oral reading, NU produced many phonemic paraphasias (e.g.

[tɛvəlɪʃə] for television). In addition, he often left out unstressed syllables in longer words. However, in metalinguistic tasks, NU was able to tell words from non-words even when the non-words were created in ways that mimicked his errors in repetition. He was within normal, adult range on a syllable-counting test. Also, NU often correctly pronounced his target word after several attempts. These facts indicate that the phonological representations of words in NU's lexicon were probably intact. Interestingly, the great majority of NU's errors occurred inside words. In fact, he was more likely to make errors on a single four-syllable word than on a multi-word unit of eight or more syllables! Clearly the word is an important unit in NU's phonological planning with errors occurring as he attempts to put phonemes into the appropriate positions.

The current perspective on comprehension and production as complex phenomena has encouraged researchers to consider the errors made by conduction aphasics as evidence for a disruption at a specific level of language production. The most frequent level suggested is probably the "positional level" of a production model such as that described in Garrett (1980), that is, the level at which phonemes are placed into position in a word. In his model of sentence structure, much referred to in neurolinguistic work, this level follows the functional level at which basic substantives necessary to express a sentence's meaning have been selected. At the positional level, a sentence frame, including functors, is generated and the substantives are actually composed.

Conclusion

The phenomena dealt with in this chapter – agrammatism associated with Broca's aphasia, lexical substitutions associated with Wernicke's aphasia, and phonemic substitutions associated with conduction aphasia – can each be linked to a particular brain area that, when damaged, results in the deficit. These areas are, of course, respectively, the areas in and around Broca's area, Wernicke's area, and the arcuate fasciculus. By a standard neuropsychological line of reasoning, we assume that if a specific language behavior stands out as particularly impaired when others

are spared, this dissociation means the area in question is crucial for performance of that language behavior in normals. Thus neurolinguists conclude that Broca's area is crucial for production of syntactically fleshed-out sentences, Wernicke's area is crucial for producing meaningful speech (as well as for comprehension), and the arcuate fasciculus (or, in Luria's theory, the parietal lobe) is necessary for stringing phonemes into the words they compose.

6 Childhood aphasia and other language disorders

Many linguists believe that the ability to acquire language is innate. These linguists point out that there are universal principles of how human language is structured (e.g., all languages will have adjectives as well as nouns) and, in addition, there are language-specific factors or parameters (such as the fact that adjectives precede the nouns they modify in English but follow them in Spanish). Infants' brains are, presumably, structured so that they will easily learn exactly how the universal elements are expressed in the language(s) they are exposed to, and pick up the language-specific features as well.

But how is the brain involved? By the time these infants become adults their left hemisphere will be primarily responsible for language organization and processing. A number of electrophysiological techniques have been used to demonstrate that the left hemisphere is dominant for language in early infancy before language is learned (e.g. Mills, Coffey-Corina, and Neville, 1993). We might then ask ourselves whether the left hemisphere controls language even in the very young. The data from childhood aphasia provide a partial answer to this question. First we must distinguish two sorts of language disturbance in childhood, language disturbance that results from sudden brain damage, as in the case of a car accident, and developmental dysphasia, that is, the inability to acquire language or aspects of language due to some brain damage before or around birth.

Aphasia in childhood

In some ways the aphasias of childhood are similar to those of adulthood. One sees an immediate interruption in the language abilities of whatever stage of language development the child has achieved at the time of the accident. In the child, however, unlike the adult, substantial recovery takes place following brain injury. Interestingly, the patterns of aphasia seen in childhood are not exactly like those of adulthood. Most strikingly, there are virtually no reports of the "fluent" aphasias in children. Rather, even when the damage is to an area that in an adult would be associated with a Wernicke's aphasia, that is, a posterior lesion, the child will produce slow effortful speech with reduced syntactic complexity if not outright agrammatism.

Lenneberg (1967) studied children with unilateral brain injury to analyze its effects on language, language development, and lateralization. His results are summarized in Table 6.1. Since infants were able to sustain significant brain damage and still acquire language normally, Lenneberg concluded that the two hemispheres are initially equally able to control language. This is known as the "equipotentiality" hypothesis. He also noted that the age at which persistent aphasic symptoms resulted from left-hemisphere injury was approximately the same age, around puberty, at which "foreign accents" became likely in second language acquisition. He proposed that the brain had a certain interval when its plasticity allowed for the flawless acquisition of language. During this time, new brain areas could assume the functions of injured areas. This is known as the "critical period hypothesis." Since Lenneberg proposed this hypothesis in 1967, numerous researchers have tested it to find out when the critical period ends. A particularly convincing study is that by Johnson and Newport (1989) that tested grammaticality judgment in a large group of subjects who had immigrated to the United States at different ages. When tested around a decade after their arrival, a clear decline in abilities was seen starting in people who arrived as early as age five, for certain syntactic phenomena.

Moreover more recent studies suggest that the right hemisphere is not entirely able to take over language functions, even in

childhood. There is neuroanatomical evidence to explain why this is. Maureen Dennis and her colleagues, for example, studied the language of people aged eight to twenty-eight who had had their right or left hemisphere removed six or more years previously. On the surface, the language of children who had had left-brain damage in early childhood looked normal as they participated in daily conversation or school. However grammatical tests such as choosing the correct picture out of two for reversible passives revealed below-normal performance (Dennis and Kohn, 1975).

They may, for example, avoid the passive construction in production. On tests of comprehension of complex constructions, they may tend to interpret the first noun phrase in a sentence as the agent or doer of the action, even in passives and other constructions where this is not the correct interpretation. These children are able to correctly interpret sentences with unusual word order when the roles of the sentential subject and object are pragmatically clear, such as in:

John ate the sandwich. *John* correctly given *agent* role.
The sandwich was eaten by John. *John* still the agent.

However, in so-called reversible passives, where the only cue about roles comes from the grammatical markers, problems of interpretation occur:

Dana kissed Val. vs. Dana was kissed by Val.
Dana was assigned the agent role in both cases.

As to the critical age hypothesis, based on a carefully selected series of brain-damaged children, speech-language pathologist Dorothy Aram (1988) challenges earlier work that showed differences between brain injury in the time around birth as compared to later in early childhood. She asserts that when proper patient selection criteria are used, the only important differences in language outcome years after the injury stem from the particular hemisphere injured and perhaps from the particular lesion site within the hemisphere. She analyzed the spontaneous speech of left- and right-hemisphere-damaged children and that of normal controls matched for such factors as age, sex, and socio-economic status as well as certain non-neurological health factors. She

Table 6.1. *Summary of linguistic and neurolinguistic development (adapted with permission from Eric H. Lenneberg's* Biological Foundations of Language*)*

Age	Usual language development	Effect on language of left lateral lesions	Other remarks
0–3 months	– Emergence of cooing	– No effect in 50% of cases; 50% with delayed onset (but normal development)	– No lateralization of function
4–20 months	– from babbling to words		
21–36 months	– Acquisition of language structure	– All language accomplishments disappear; language is reacquired with repetition of all stages	– Hand preference emerges – Left hemisphere begins to assume sole responsibility for language – Language appears to involve whole brain
3–10 years	– Grammatical refinement and expansion of vocabulary	– Aphasic symptoms; tendency for full recovery (except in reading and/or writing)	– Evidence for both hemispheres still active in language; right/left lateral-lesion disrupts language – Possible to re-establish language in right hemisphere if left is damaged

11–14 years	– Foreign accents in 2nd language learning	– Some aphasic symptoms are not reversible, particularly in traumatic lesions	– Lateralization is formally established – usually irreversibly – Language-free parts of brain cannot take over except where lateralization is incomplete (due to childhood pathology)
Mid-teens-senium	– Acquisition of 2nd language is increasingly difficult	– Aphasic symptoms may persist; symptoms present for more than 3–5 months are irreversible	– Language definitely lateralized in left hemisphere for 97% of population

found the speech of right-hemisphere-damaged children to be very similar to that of the non-brain-damaged controls. Left-hemisphere-damaged children had more difficulty with simple and complex sentences than did the normal controls. Both left- and right-hemisphere-damaged children showed some persistent difficulty in naming objects. Children with left-sided injury answered questions more slowly but more accurately than children with right-sided injury. Aram found no effect of age at the time of brain injury in any of her analyses. These data clearly argue against the idea of an initial state of hemispheric equipotentiality.

Anatomical studies (e.g. Galaburda and Kemper, 1979) have documented differences in the actual cell-level structure of the left and right hemispheres. The two hemispheres are not *identical* even pre-natally. Most interestingly, the left hemisphere in most people has a larger planum temporale, that is, more development on the left side of the brain in that core area of what will become the "language center." However, these structural differences do not necessarily preclude the possibility of equal potential for each hemisphere to assume language function.

On the basis of a review of the literature, Satz, Strauss, and Whitaker (1990) agree that current knowledge of neuroanatomy speaks against a complete interchangeability of the two hemispheres at birth. They suggest that Lenneberg was, however, partially correct. Their improved version of the equipotentiality hypothesis refers to the potential of left-hemisphere regions around the classical language area and right-hemisphere regions analogous to the left-hemisphere language areas to assume language functions in the event of damage to the normal left-hemisphere language areas.

Post-pubertal language acquisition

Further evidence on the critical period hypothesis comes from the studies of children acquiring language after puberty. Recall that Lenneberg predicted that it was hard to learn a second language after puberty due to crucial brain maturation being complete. By implication, individuals who were forced to acquire a first language after puberty should be equally unable to. One

opportunity to study such a child was afforded scientists by the case of Genie, a child whose abusive father had Genie isolated and physically restrained day and night in a small bedroom with little light and virtually no stimulation from the age of twenty months. This abusive father so threatened Genie's mother, who was herself becoming blind, that the mother did not report the father's neglect and abuse of Genie until Genie was thirteen and a half. After this, Genie was hospitalized and treated for malnutrition, and her opportunities to socialize and learn language began.

Genie had been beaten for making any noise in the period of her tragic isolation, so she was virtually unable to vocalize when she was found. Over the next four years a linguist, Susan Curtiss (1977), was able to observe the development of her language and test how it related to her brain activity. In many ways, Genie's language development was different from that of normal children, although she certainly acquired a substantial number of linguistic rules. The phonological sounds in her early words were more varied than those of normal children and her early two syllable words were not "reduplicated," that is, consisting of a single syllable spoken twice, as are those of normal children. Also, unlike normal children, she had no early intonation patterns. While normal children learn primarily nouns at first, Genie learned verbs and adjectives as well as nouns, but delayed combining them into two-word phrases much longer than normal children. Question production remained particularly difficult for Genie over the four years Curtiss studied her, as did appropriate use of pronouns. Her acquisition of negative sentences did not follow the standard pattern; for three years she used only one structure, e.g. "no more take wax" (p. 190). In her production of language, Genie followed a fixed word-order pattern, and in her comprehension she was not able to appreciate the word-order differences that distinguish active from passive sentences. Finally, while it may be said that Genie follows rules, she treats them as much more optional than normal children do. For example, while normal children go through a period of including only full forms, and only later learn ellipsis, from the start Genie would delete subjects, verbs, or objects from sentences, whether or not the listener could appropriately infer what they were supposed to mean, e.g. "Elevator hurt silly goose."

Several different tests suggested that Genie was using primarily her right hemisphere to learn language. Dichotic tests of language showed markedly greater left-ear performance than right-ear performance. A tachistoscopic test where Genie heard rhymes and had to point to pictures she saw of a word that rhymed with the word she heard tended to show a left visual field effect as well. Also, a pilot study using evoked potentials indicated right-hemisphere differences for processing nouns and verbs.

Not only language was being processed by the right hemisphere; most of these tests suggested that non-language abilities, such as processing environmental sounds, were also being handled by Genie's right hemisphere. Curtiss points out that many aspects of language that we will see (Chapter 7) have been associated with the right hemisphere, such as formulaic speech, are not among the aspects that Genie is particularly good at. She attributes this to Genie's extreme inability to socialize, and the fact that many aspects of right-hemisphere language are pragmatic. However, Curtiss attributes Genie's difficulties with acquiring appropriate syntactic and morphosyntactic rules and her problems using them when necessary to a general right-hemisphere "holistic" thinking style that Genie evidences. She compares this to a "sequential, analytic thinking" style that we associate with normal, left-hemisphere abilities.

Of course, Genie's case is not an ideal one for testing what happens with late language acquisition because there is some question as to whether she was mentally retarded from birth and thus her father was responding so cruelly to her abnormal development. The extreme deprivation that she suffered also may have had biological influences on her brain, so it is unlikely that we see in her simply an example of "normal" delayed language acquisition. Nevertheless the case has been a valuable one for provoking us to think about the issues of how language would develop after the critical period.

Developmental dysphasia

Difficulties with language in children that are not related to one-time brain accidents are of great import for the field of speech

language pathology. Among children with learning disorders, there are children with what is called specific language impairment (SLI). In these children other cognitive areas are normal or even better than normal, but language in particular is delayed abnormally. In such children no actual brain damage can be seen via any of the brain-imaging techniques, but unusual clusterings of cells have been found in some language areas of children who have died of unrelated causes.

There has been much debate in the field of childhood language disorders concerning whether there are specific subsyndromes of SLI that are primarily in production of speech or primarily in comprehension of speech. It seems that currently terms like "specific language impairment" apply to children with predominantly production problems, while children whose primary difficulties lie in making sense of language are said to have central auditory processing difficulties. A recent study employing evoked potential techniques confirms that the underlying behavioral causes for SLI differ among children. Some have difficulty with simple auditory processing while others have difficulty with simple visual processing (Neville et al., 1993).

Some children with SLI have primarily phonological problems. Their speech remains unintelligible much longer than that of normally developing children. Such children invariably make systematic errors, for example, "deleting" (i.e. of not producing) final consonants, or producing velar consonants (e.g. /k/) at a more forward point in the mouth (e.g. as /t/). Different children will consistently evidence different systematic deviations from the norm, although there are some processes that are frequent among children with phonological disorders (e.g. consonant cluster reduction).

The brain-based causes of phonological problems remain unknown. One theory is that frequent bouts of ear infections (otitis media) result in enough poor hearing at crucial developmental points to interfere with the child's appreciation of what a set of sounds in a given environment should sound like. Some children with no history of otitis media also have abnormal phonological systems, however. Moreover, they tend to distinguish minimal pairs of words in their production of them, albeit by unconventional

means (e.g. using vowel shortening where a final consonant should occur: e.g. /pa/ for "pot" but /pa:/ for "Pa"). Such problems tend to run in families, suggesting a biological basis for the problem, even if actual brain lesions cannot be demonstrated. For such children the problem would seem to lie somewhere in the motor-planning system that converts phonological representations to spoken words.

Another form of SLI is reflected in problems in morphosyntax. In a recent set of studies, Gopnik and her colleagues focus on the hereditary component in this form of specific language impairment. The particular difficulty with morphology reported in this three-generation family is rare, but Gopnik was able to document its existence in a grandmother, in four out of five of her children (all three daughters and one of the two sons), and in eleven of the 24 grandchildren. In these individuals both phonology and morphosyntax are impaired. Indeed, the children are regularly unintelligible up until the age of seven despite normal hearing and intelligence.

In their first set of studies, they noted that dysphasic individuals in this family had particular difficulty with comprehending plurals (e.g. "Show me the books" as compared to "Show me the book") and difficulty making grammaticality judgments on sentences containing errors of number ("the boy eats three cookie"), person ("the boy kiss a pretty girl"), tense ("yesterday the girl pet a dog") and aspect ("the little girl is play with her dog") (Gopnik and Crago, 1991). Argument-structure errors in which, for example, a verb that should take a direct object did not (e.g. "the nice girl gives") were relatively well corrected, by contrast. Production of tense forms was impaired, as was production of plurals for nonsense words. In writing there was a discrepancy between regular and irregular verbs; family members had learned the forms for irregular verbs, but consistently erred on regular verbs, often giving the unmarked form! Problems also were seen with comprehending negative passives, and with derivational morphology.

Similarly with respect to pluralization, the dysphasic subjects had difficulty making plurals on nonsense words like *wug* and *zoop*. One subject whispered "add an s"; another turned *sas* to /*sæsIz*/ and then added the syllabic plural to all the remaining items (e.g.

/zuplz/). In later analyses, Goad and Rebellati (1994) conducted phonetic analyses of the plural forms that dysphasics in this family produced. In fact the subjects do not assimilate for voicing, that is, they do not mark plurals with a /z/ sound for words that end in voiced consonants, and with a /s/ sound for words that end in unvoiced consonants. This suggests that their pluralization is performed by compounding rather than by a normal rule of affixing.

Particularly striking was comparing the way the dysphasics and their normal siblings took these tests. For the dysphasics the tasks were difficult and time-consuming "as if they were taking a test in a language they did not know particularly well"; for the normals the tasks were quite simple and self-evident.

In a series of papers published in the early 1990s (Matthews, 1994, Gopnik and Crago, 1991; Dalalakis, 1994a and 1994b; Fukuda and Fukuda, 1994; Goad and Gopnik, 1994; Gopnik and Crago, 1994; Gopnik, 1994a, b and c; Kehayia, 1994) Gopnik and her team evaluate more specifically the problems the specific language-impaired members of this family have with pluralization, tense and adjectival inflections in English, and related phenomena in Japanese, Greek, and Inuktitut. They analyze spontaneous discourse, grammaticality judgment tasks, and nonsense-word production tasks. In each instance they are able to ascertain that the language-impaired members of a family, like the unimpaired members, appreciate the meaning that inflectional categories must bear (one vs. more than one, currently or in the past) but are unable to automatically apply the rules. On virtually all these tests of inflectional morphology, the language-impaired subjects performed markedly worse than the unimpaired members of their family, despite normal cognitive abilities generally. On a task to test patients' abilities with grammatical number, for example, they asked subjects to tell whether sentences such as "I drove past several truck on the way home" sound natural or unnatural, or whether subjects were unsure. Rather than automatically applying rules, the subjects have learned rules that they can articulate. However they do not apply them consistently (e.g. when asked how the past tense is produced, one subject said "if it's today, it's *ing*, like *swimming*. 'I went swimming today' and 'yesterday I swamt' ") (Matthews, 1994: 133).

While it has been generally understood that specific language impairment tends to run in families, the studies by Gopnik and her colleagues are the first to give such clear-cut indication of genetic predilection for a very specific disorder. Currently there is no information about the brains of the subjects, although apparently they have no history of birth disorders or frank brain damage. However, the specificity of the tasks and analyses Gopnik and her team have employed raises the distinct possibility that biology determines the specific ways that cellular arrangements and connections in the brain can facilitate morphosyntactic aspects of language processing and production.

Numerous explanatory hypotheses have been generated to explain the language disorders of specific language impairment. Some have argued that the problems with inflectional morphology are a secondary by-product of perceptual problems, either in terms of articulation or phoneme perception. Gopnik and her colleagues are able to demonstrate that their subjects perform like normals on the phoneme perception task and generate markedly more errors in speech production on inflectional affixes than on the same structures when they do not function as inflectional affixes. They maintain, rather, that because the language learning of SLI children is delayed, their brains' abilities to acquire morphosyntactic rules for automatic production are dysfunctional. Thus only explicit knowledge of the rules can be applied, resulting in subtle, and sometimes not so subtle, errors (Paradis and Gopnik, 1994).

Conclusion

Study of children with developmental language disorders is of great importance to help those whose brain damage requires remediation. Such cases are also useful for determining the psychological reality of the various aspects of language that can be impaired in such children while others are spared, such as phonological processing. However, they are hard to evaluate in terms of brain regions involved because the nature of the brain malfunction is rarely, if ever, clear. By contrast, the study of childhood aphasia contributes indications of the specific, especially syntactic,

abilities that the left hemisphere is particularly good at. At the same time the similarity of all aphasias in children suggests that language abilities are more diffusely organized, at least within the language area, in children than in adults.

7 Right-brain damage

In this chapter, we will consider possible contributions of the right hemisphere to language perception, production and use. Data presented in the aphasia chapters have suggested that the left hemisphere is solely responsible for language. Only in the chapter on language disorders in children was the possibility raised that the right hemisphere *can* participate in language acquisition, in cases of early left-brain damage.

Right-hemisphere lesions do not typically result in any of the classic patterns of language loss. In fact the scarcity of patients with linguistic deficits following right-hemisphere injury was one of the pieces of evidence for lateralization of language in the left hemisphere for most of the population. The right hemisphere has been thought to have little or no language ability except in the case of extensive early left-hemisphere damage. Rather, the right hemisphere has been connected with processing emotions, visuo-spatial materials, music, and the like.

One logical possibility is that the right hemisphere has nearly the same potential for language acquisition as the left but, for efficiency, yields to the left hemisphere. If it were simply more efficient to have one hemisphere take responsibility for language, however, we would expect a random distribution of language in the hemispheres. That is, about half of humans would have the right hemisphere dominant for language, and half would have the left hemisphere dominant. Indeed, this seems to be the case for other animals like mice (if we look at pawedness, of course, not language, as the measure of lateral dominance). It could also be the case that the right hemisphere does acquire some linguistic

ability which is then suppressed by the dominant left hemisphere. More likely, it is currently thought, the right hemisphere and the left hemisphere have different patterns of cellular interconnection. These patterns differ so that the standard, left-hemisphere patterns are most appropriate for the analytic abilities needed for language, and perhaps for their later automatization as well, but the right-hemisphere systems, particularly good for holistic sorts of knowledge, can, in a pinch, take on language processing abilities as well. Evidence bearing on this question comes from a number of different populations which we will consider in turn.

Hemispherectomy

In the previous chapter we mentioned the small number of children with a neurological disorder known as Sterge-Weber-Dmitri syndrome, which includes problems with the blood supply to the cortex. The best possible results for these children are achieved by early removal of the entire affected hemisphere. Recall that the remaining hemisphere is able to assume much of the function of the removed hemisphere. Whether the left or the right hemisphere is removed, the children do acquire language. Children who have had their right hemisphere removed are linguistically indistinguishable from children who have intact brains. However, children who have had their left hemisphere removed evidence subtle linguistic difficulties.

Studies of these children with early hemispherectomy, we conclude, cast doubt on *total* equipotentiality of the hemispheres. They also establish considerable right-hemisphere ability to assume language function.

Linguistic abilities in right-brain-damaged adults

The right-brain-damaged patient

Unlike left-brain-damaged patients, who appear in their person to be very similar to who they were before the brain damage, right-brain-damaged patients are perceived as being somehow different. They no longer care for themselves or their dress as carefully as

they may have before the brain damage. Their face and their prosody convey what is called "flat affect," as if they were not engaged in a conversation, and their behavior is often quite inappropriate. They may joke about some of the topics we do not generally discuss in our society (such as sex) inappropriately, or interrupt meetings or serious conversations with unrelated comments on things that have distracted them. When they themselves are talking, they may stray off on unimportant tangents and forget to return to the topic at hand. Thus, while their linguistic abilities per se are not markedly impaired, as they are so noticeably in aphasic patients, there are a number of language and communication deficits that they display, which, in turn, yield information about the way the right hemisphere participates in language and communication.

Tone and prosody

The possibility of right-hemisphere contributions to normal language processing and production needs to be considered at a number of different levels. We will start with phonology – the sound system – and then turn to levels of lexical representation, syntax, and text production or pragmatic levels. The phonology of all natural languages contains some prosodic component. For example, all languages use prosody to distinguish questions from assertions. In addition, some languages, like Chinese and Vietnamese, use tone as a suprasegmental distinctive feature (that is, it can distinguish between two words that otherwise sound exactly alike). So, for example, in Toisanese Chinese, the word /hau/ spoken with a high flat pitch means "mouth" while the same string of sounds spoken with a falling pitch started at a mid-level point means the relative location "behind." Since the right hemisphere is dominant for the perception of musical tone, might it also be essential in the production/perception of linguistic tone and/or prosody?

Some researchers have thought that the tone in tone languages might be processed predominantly by the right hemisphere. However, Van Lancker & Fromkin's 1973 dichotic-listening study indicated this was *not* true for normal subjects. Apparently be-

cause tone systems serve linguistic functions, discriminating among words that are otherwise pronounced the same, left-hemisphere dominance was shown among Thai speakers. A recent set of studies of aphasic Thai and Chinese speakers provides converging evidence that the left hemisphere is responsible for tone, as tone production (Gandour et al., 1992; Eng Huie, 1994) and perception (Eng Huie, 1994) are impaired in left-hemisphere-damaged aphasics.

However, even in non-tone languages such as English, stress and intonation play a role in the grammar of the language. Standard aspects of linguistic intonation that serve syntactic function (say, to distinguish questions from assertions in many languages) appear to be dominated by the right hemisphere, researchers such as Blumstein and her colleagues (e.g. Blumstein and Cooper, 1974) have demonstrated. Similarly, the perception and production of emotional non-grammatical prosody (say, to convey anger or enthusiasm) is certainly essential for normal participation in everyday discourse. In dichotic-listening tasks, there is a left-ear advantage, suggesting right-hemisphere dominance, for affectively intoned material as well. Subjects are more accurate in judging whether two emotionally intoned sentences are the same or different when the sentences are presented to the left ear. They are also quicker and more accurate in naming the affect presented in the stimulus.

Lexicon

It is generally understood that the words of our internal lexicons are represented primarily in the left hemisphere. However, work with split-brain patients has suggested that there is some lexical knowledge in the right hemisphere of the split-brain patient as well. If a written word is directed to the right hemisphere of such a patient, by presenting it in the left visual field (see Figure 2.8) and preventing the subjects from turning their heads so that it can be seen via the right visual field, the patient will not be able to say the word aloud. However, particularly with concrete and imageable words, such patients will be able to point (with the left hand, of course, but not the right) to a picture of what the word

represents, even when they may actually speak saying they have no idea!

Joanette (1994) reports on a comparison of the lexico-semantic production of right-brain-damaged patients and normal controls. In a test of verbal fluency, subjects were asked to name as many words as possible within a two minute time limit. There were four conditions for this production task. In two of the conditions, each of the words had to begin with the same letter. This tested the patients' ability to access vocabulary based on the *form* of the word. In the other conditions, the words needed to be associated with the same *semantic* category (e.g. animals, furniture). The right-brain-damaged subjects performed very similarly to the normal subjects in the conditions based on form. They were also quite similar for the first thirty seconds of the semantic condition. However after the first thirty seconds, the right-brain-damaged subjects produced significantly fewer responses than the normals. This impairment implies that there is some right-hemisphere contribution to the lexical access process for meaning but not for form.

A study by Bloom et al. (1990) demonstrated that right-brain-damaged patients had some difficulty with lexical selection in story-telling contexts, consistently choosing words of less emotional intensity than did their left- brain-damaged or normal control counterparts. They were, in general, less successful in using words to convey emotion.

Syntax

Right-brain-damaged patients also seem to have less flexibility than normals in assigning structural properties to sentences (Schneiderman and Saddy, 1988). For example, in order to appreciate the ambiguity of a sentence such as *The boy hit the man with the cane*, it is necessary to see both the possibilities: "with the cane" can be an adjective phrase describing the man or an instrumental phrase modifying the way in which the boy hit the man. The patients tested by Schneiderman and Saddy could not identify both possibilities.

Emotion in discourse

At the level of text production and language use, the most compelling evidence for right-hemisphere involvement is in the area of emotional aspects of discourse. As we mentioned above, the right hemisphere is dominant for processing emotional prosody. Facial expression of emotion is also controlled predominantly by the right hemisphere, as is appreciation of emotional, facial expressions. Yet when Blonder, Bowers, and Heilman (1991) asked their left-brain-damaged, right-brain-damaged, and normal control subjects to perform a number of tasks designed to distinguish among prosodic, facial and lexical indications of emotion, their right-brain-damaged subjects were able to infer the emotional content of sentences describing emotional situations. This was true for sentences in which the emotional content was signalled directly (e.g. "You were delighted by the bonus") as well as for more difficult "connotative" (e.g. "It was the third anniversary of the death of your child") or "interpretive" (e.g. "After you drink the water, you see the sign") sentences. This speaks against a general disruption in the ability to appreciate emotional situations in patients with right-brain damage.

The Blonder et al. study confirmed previous research on right-brain-damaged patients' difficulty in distinguishing faces and facial expressions. However, their right-brain-damaged subjects also had significantly more difficulty than either the normal control or left-brain-damaged subjects in interpreting *verbal* descriptions of emotional expressions such as "He scowled" or emotional prosody (e.g. "He spoke quickly and breathlessly"). This suggests an actual disruption in the representation of non-verbal (gestural and prosodic) emotional expressions.

Discourse appropriateness

In addition to lexical access and the representation of non-verbal communicative gestures as well as the verbal expression of emotion, a role for the right hemisphere in the organization of discourse has been suggested. To demonstrate the difference between well-formedness at the sentence level and at the discourse

level, it is only necessary to examine the difference in status between two strings depending on the context. As sentences, it is clear that *Butter is yellow.* is well-formed and **From work.* is ill-formed. However, in the context of a discourse such as the following, clearly the judgments are reversed:

> Speaker A: I'm surprised to see you at this bus stop. Where are you coming from?
> Speaker B: From work./*Butter is yellow.

Speakers of any language have certain expectations with respect to the organization of both texts (written or oral) and conversations. Learning to produce well-formed discourse is an unconscious process, just as native speakers of a language are not consciously aware of the grammar of their language and learn grammatical rules without explicit instruction. Although the criteria for well-formed discourse may vary slightly from culture to culture, there are a number of pragmatic features of discourse that tend to be observed universally. These expectations were described by the philosopher Paul Grice (1975) as a basic principle of cooperation covering four major conversational "maxims":

(1) *Maxim of Quantity* In a communicative exchange, participants should not provide too much or too little information.
Where are you coming from?
*Somewhere./*I'm coming from West 34th street and Broadway. I walked east for two blocks, crossed the street after waiting for the light and then walked one block north.

(2) *Maxim of Relevance* Participants should maintain a topic from one utterance to the next, with topic shifts allowed only at certain junctures. This is the maxim violated in the "Butter is yellow" example above.

(3) *Maxim of Manner* Information should be presented in an organized and clear manner.

> *My hometown has five shopping malls. It is the county seat. My father and mother were both born there. My hometown is a midwestern town of 105,000 inhabitants situated at the center of the Corn Belt. I was brought up there until I was thirteen years old. (Example taken from Finegan & Besnier, 1989)

(4) *Maxim of Quality* In general, people should say and write only things they believe to be true.

Although the fourth maxim clearly has more to do with the content of discourse and other areas of cognition, the first three have an impact on the structure of discourse. Other authors have expanded on Grice's original conversational maxims to capture further generalizations about our unconscious expectations for language use. For example, participants in a discourse must choose and maintain an appropriate level of formality depending on the context. This makes "lexical selection" an important feature of discourse construction; words must be selected to fit the text (*Bye-bye Your Highness; see you!).

Similarly, appropriate phonological and syntactic features must be used. For example, one common feature of casual spoken English is the pronunciation [n] for the ending -ing as in:

I love to go swimmin' and fishin'.

However, in the context of a formal sentence, such casual pronunciations are inappropriate:

*I've been hypothesizin' about nuclear fusion.

Conversely, the use of formal features can be inappropriate in certain contexts. For example, the use of the passive construction would not be appropriate for highly marked, caretaker speech:

*Come on sweetie, all your nice soupy-woupy must be eaten now!

Studies of the discourse produced by right-brain-damaged patients reveal subtle deficits in some of these verbal pragmatic aspects. Bloom et al. (1992) examined the performance of right-brain-damaged patients, left-brain-damaged patients and normal controls on a story-telling task using pictures designed to elicit emotional or non-emotional discourse. The subjects were shown cartoon drawings created for this study, some of which involved an emotional event (such as a girl losing her dog and seeing him run over), and others of which involved a non-emotional event (such as how to fry an egg). In this study, the focus was on the

amount of information given. Both left-brain-damaged and right-brain-damaged patients showed some difficulty in producing appropriate amounts of information in their discourse. This is to be expected for the aphasic left-brain-damaged patients. However, the right-brain-damaged group, while producing similar amounts of discourse to that of the normal controls, still failed to produce appropriate amounts of information. In the emotional story context, the performance of the right-brain-damaged patients was particularly impaired. That is, their speech had markedly less content than that of the normals.

One explanation for a set of the conversational problems that right-brain-damaged patients have is that they have impaired abilities to think about what is in the mind of the people they are talking to. This theory, the Theory of Mind, is built on the notion that children after a certain age develop a sensitivity to what their interlocutors know and what they do not. For example, around this time, the children start more appropriately using pronouns in instances when their interlocutor can know who is being referred to, and using proper nouns otherwise. For example, if I'm talking about two women, Henrietta and Jocelyn, I can't refer to first one and then the other in a sentence using only pronouns; "then she hit her on the head" can't be fully interpreted unless we know which pronoun refers to which person. Right-brain-damaged patients will have more difficulty than normals in seeing that referents are made clear. Also, starting in childhood, and extending through our work-lives, we learn how to choose the register of language we use so as to appropriately attend to the differences in power between, say, a boss and an employee. While normals are able to appreciate that one doesn't say "don't do that" to one's boss, right-brain-damaged patients might choose that response as often as "do you think it's possible for us not to do that?" when a similar situation is described (e.g. the boss has just planned a picnic on the day that a surprise party has been planned for her).

Not only do right-brain-damaged patients have difficulties in descriptive discourse and in conversation, generally; they also have difficulties with non-literal language. In a series of studies, Gardner and Brownell and their colleagues have tested right-brain-damaged patients' abilities to appreciate sarcasm and hu-

mor, as well as metaphoric meanings of words. (See Brownell et al., 1994, for a review of these studies.) The right-brain-damaged patients characteristically interpret expressions that are intended to be taken non-literally quite literally; by contrast, left-brain-damaged patients, even if they cannot comprehend all the words, can distinguish the two when given the appropriate contextual situations for interpretation. They looked at right-brain-damaged patients' ability to deal with metaphor and idioms and other examples of individual words that have more than one meaning (e.g. *blue*, the color and *blue* meaning sad). Right-brain-damaged patients again often select the literal meanings on these tasks, although under certain conditions the researchers have demonstrated that the patients do have access to the second, more abstract meanings of the words or phrases.

Accessing the abstract meanings of words can be demonstrated by giving the subjects a priming task. Because our mental dictionary is organized along semantic lines (as well as phonological, morphological, and orthographic ones), processing a word from a given semantic category gets the subject to the right "area" of the lexicon, making reactions to subsequent words in that same semantic area quicker. This quickening of reaction times is referred to as "priming." In such a task subjects must make some decision about the second word they see in a pair. For example, subjects may have to decide whether the word "blue" is a word. The first word is either related to it (e.g. *sad*) or not (e.g. *dog*). In the specific studies we are talking about here, the meaning of the first priming word crucially relates to the non-literal meaning of the second word or phrase. On such tasks, we see that the response time for the brain-damaged subjects to decide that the second word is a real word in English, or the idiom is a real idiom in English, is facilitated for the right-brain-damaged patients just as it is for normals and left-brain-damaged patients, indicating that the non-literal meanings of the second word in each pair are available to them. It seems one must conclude, then, as Brownell and colleagues (1994) do, that the problem lies not with the knowledge of non-literal features of language, but rather in applying that knowledge in the processing involved for performing such tasks.

Split-brain patients

Given that the right hemisphere is capable of acquiring language when the left hemisphere is removed or severely damaged, it is logical to consider the possibility that the right hemisphere has some linguistic abilities not normally in evidence due to the inhibitory effects of the dominant left hemisphere. In split-brain patients, the reader will recall, this interhemispheric communication is interrupted. Thus stimuli presented to the left visual field cannot be fully processed linguistically. The right hemisphere registers such stimuli, and may even react to them, however. In some studies (e.g. Zaidel and Peters, 1981) some split-brain patients have been able to extract linguistic information from stimuli presented to the right hemisphere. Some, for example, could pick a rhyming word from a list of choices. However, even for the majority of split-brain patients whose right hemispheres do demonstrate some rudimentary linguistic abilities, the isolated right hemisphere cannot comment on its perceptions.

In a review of studies of commissurotomy patients, Gazzaniga (1983) provides a description of the right-hemisphere linguistic abilities of the two (out of the 44 patients living in 1982) split-brain patients who had developed right-hemisphere speech (presumably after the surgery). For both of these patients, comprehension of stimuli presented to the right hemisphere was essentially normal. Both were able to name objects, carry out verbal commands and even detect semantic peculiarities in sentences presented to the right hemisphere alone.

However, the left hemisphere in both patients remained the "more talkative," dominant hemisphere. This can be seen in the response of the patient P.S. to bilateral presentation of a brief story. The patient was presented a series of five slides each of which flashed one word into the right visual field (left hemisphere) and one word into the left visual field (right hemisphere). In normals, the information from each half of the slide is integrated and the story reads as follows:

MARY + ANN MAY + COME VISIT + INTO THE + TOWN
SHIP + TODAY

P.S. demonstrated an appreciation of the information presented both to the left and right hemispheres but reported the story quite differently, his left hemisphere at first reporting only what it saw and, only when pressed, did it struggle to integrate the verbal responses of the right hemisphere (Gazzaniga, 1983):

P.S. Ann come into town today. [NOTE: These were all, and only, processed by the left hemisphere]
E. Anything else?
P.S. On a ship.
E. Who?
P.S. Ma.
E. What else?
P.S. To visit.
E. What else?
P.S. To see Mary Ann.
E. Now repeat the whole story.
P.S. Ma ought to come into town today to visit Mary Ann on the boat.

Conclusion

In order to reach the conclusion that the right hemisphere normally participates in some way in the perception or production of language, we look for evidence of linguistic deficits following right-brain damage.

We have looked at the abilities of a surgically isolated right hemisphere and at experimental techniques devised for presenting linguistic stimuli only to the right hemisphere. The brains of commissurotomy patients are certainly not representative of the general population given the history of seizures. Yet, these commissurotomy studies, along with dichotic and tachistoscopic presentation studies, do provide evidence that the normal, adult, right hemisphere can, under special circumstances, demonstrate linguistic ability.

While the right hemisphere does not appear to have much responsibility in normal individuals for core linguistic processes such as phonology, morphology, and syntax, it contributes importantly to a set of paralinguistic phenomena. Intonation ap-

pears to be dealt with by the right hemisphere, whether it indicates syntactic structure or emotional communication. Some aspects of lexical selection appear to have substantial right-hemisphere involvement, like the ability to appreciate multiple meanings, especially non-literal ones, of words. Moreover, a host of pragmatic abilities appear to be impaired with right-hemisphere (but not left-hemisphere) damage. These include the abilities to appreciate humor, sarcasm, implication, discourse appropriateness, the interlocutor's knowledge, and the like.

8 Dementia

The term "dementia" is very broad. It refers to the results of a number of different diseases all of which lead to a loss of intellectual abilities. After we consider the relation between language and cognition, we will turn to the usual course of some types of dementia and their effect on the language of the dementing person. Dementia is caused by the deterioration of brain tissue. Different dementias affect different parts of the brain, but they do not result in obvious brain damage in distinctly localized areas of the brain the way aphasias do. Rather they appear as more generalized atrophy.

One crucial distinction for our purposes is the distinction between cortical and subcortical dementias. In the cortical dementias, the cellular changes associated with dementia are primarily in cortical areas; in the subcortical ones, conversely, the cellular changes are primarily in subcortical structures. The most commonly known cortical dementia is Alzheimer's disease. In this disease, the characteristic changes in cortical cells result in patients showing at least three of the following four symptoms: they evidence problems with language; they have memory problems; they have problems performing new tasks with knowledge they already know (e.g. spelling "world" backwards); and they have personality changes. Many patients become markedly more irritable, even belligerent; there is one instance where an adult daughter complained that her mother, who had previously been wonderfully critical, had become uninterestingly "nice" with Alzheimer's dementia.

The most common subcortical dementia occurs in perhaps

one-third of cases of Parkinson's disease. The difficulties in walking and in speech are remarkably similar. Patients may have trouble initiating each, and move quite slowly, at least at the beginning, however, they may speed up and, in walking, stumble. Speaking, their speech may end up muttered unintelligibly. The language changes in Parkinson's disease are more subtle as we discuss below.

Language and cognition

The language of dementing patients presents a unique opportunity for examining the relationship between language and cognition. The pattern of dissociation of abilities in dementia can yield information regarding the normal relationship – dependence or independence – between language and more general cognitive abilities. A patient with Alzheimer's dementia once told one of us that the woman in the Cookie Theft Picture (see Figure 4.1) was in a "turmoil of smin." Was he simply unable to say that the woman should have been upset because so many things were going wrong in her kitchen, or was he unable to figure out from the picture that indeed she was not upset, as most normal patients report? To study language production and comprehension abilities in dementing patients is to explore the boundaries between syntax and semantics and among semantics, real world knowledge, and reasoning abilities.

Wernicke himself was concerned about how thought and cognition related in his famous 1874 paper in which he distinguished Wernicke's aphasia from Broca's aphasia and predicted conduction aphasia. In fact, one of the two patients he asserted had Wernicke's aphasia was, our current analysis strongly suggests, a patient with dementia (Mathews et al., 1994). Because patients with both Wernicke's aphasia and Alzheimer's dementia include empty words (e.g. "this," "the thing") and nonsense phrases in their speech, it is hard to determine if there is underlying knowledge beneath what they say. To distinguish whether one is seeing the language changes of Wernicke's aphasia or the language-plus-cognitive changes of Alzheimer's dementia, it is not sufficient to rely on patients' language problems; we must consider whether

there is a history of a more generalized cognitive and behavioral decline. If so, we say that we are looking at Alzheimer's dementia, and that the language reflects both cognitive and linguistic impairments. If there was a sudden onset of language problems with no history of memory or other cognitive problems, we assume we are seeing Wernicke's aphasia.

Subcortical dementias

Most frequently seen among the subcortical dementias is Parkinson's disease which affects mostly subcortical brain areas. This kind of brain tissue deterioration has significant impact on the patient's ability to produce speech. Not only do patients with Parkinson's disease start walking slowly and with difficulty, and move slowly generally, this slow and halting behavior extends to control of the vocal apparatus. Parallel to the dysarthria or difficulty in articulating speech sounds seen in these patients, there is often a dysgraphia or disturbance in the ability to write. For example, writing samples exhibit both micrographia or a tendency to write very small letters, and an inappropriate use of space in writing. Of course, either of these symptoms might be attributed to loss of muscle control without any damage to brain representation for language.

Though these movement symptoms are the most striking for patients with Parkinson's disease, there are also cognitive changes in the one-third of patients with Parkinson's disease who also have dementia. This dementia consists primarily of memory problems and problems operating on the knowledge they already have. Moreover, a closer examination of the linguistic output of patients with subcortical dementia has revealed subtle effects on language itself. Consider the following example from a Parkinsonian patient's written description of the BDAE Cookie Theft Picture taken from Obler (1983; underlining added):

> the boy on the stoll tripp and the girl laugh at the boy and then she spill water on the floor.

Of the four underlined omissions or substitutions of letters, three involve morphological word-endings. This tendency to err more

frequently on inflectional endings is common in dementing patients. It cannot be explained by simple inattention. While it is true that the ability to pay attention to a task is impaired in the dementias and distraction is common, such problems in writing should distribute themselves randomly across words if attention or the ability to self-monitor were the only explanation of the writing problems. Here instead, it is the linguistic properties of words that appear to render inflectional endings particularly vulnerable to error. Murray Grossman and his colleagues (Grossman et al., 1994) have demonstrated additional lexical problems in patients with Parkinson's disease. When exposed to a new verb (*wamble*, meaning "to return home") and asked about it 10 minutes later, some patients learned nothing about the verb, while others picked up only semantic information but not information about syntactic use.

Further examples of linguistic disturbance in subcortical dementia are found in lexical-selection errors. Consider the following example from Dr.T., a patient with a subcortical dementing disease called *progressive supranuclear palsy* (a neurological disorder resulting in bodily rigidity as well as dysarthria – muscular problems at the end-stage of speech production – and dementia) whose language is reported more fully in Obler et al., 1980. In describing the Cookie Theft Picture, he writes:

> The young lady he is hang up in mischief.

Not only does he omit the affix "ing," he presumably intends "hanging up in" for "involves." In addition to such lexical and morphological disturbances, Grossman and his colleagues have demonstrated sentence-processing problems in patients with Parkinson's disease. They attribute these problems to memory and to attentional deficits resulting from poor distribution of dopamine to areas of the frontal lobe that are involved in cortical networks for sentence comprehension (e.g. Grossman et al., 1992).

Cortical dementias

In the *cortical* dementias, especially Alzheimer's disease, patients have symptoms much more like those of the aphasias. This is not surprising since the cellular damage to the brain, while wide-

spread, is primarily in the temporal and frontal lobes of patients with the disease. Consider the following speech sample from a patient with advanced Alzheimer's dementia cited by Bayles & Kaszniak, 1987:

> "No, for goodness sake. What is you doing? Coming home from a story, or playing? My parents is a has a present for you . . Ah, your parents has the house-cleaning, Timmy. We, we, no. Running out at three, then, the car wash, they, uh, fill, four, happy everyone, then can come back again."

In terms of context, this discourse is clearly lacking in cohesion, with over-frequent topic shifts. It is also repetitive and fairly empty, reminiscent of the speech of a Wernicke's aphasic. In addition, there are frequent morphological and syntactic errors as well as lexical selection errors. The words *story* and *is* are misselected. In the case of "is," the patient repairs his error in selecting a verb for the sentence, but does not correct the form of the verb (i.e., my parents (pl.) is/has (sing.)). This difficulty with number agreement is seen again in "What *is you* doing?" and "Your *parents has* . . ." In addition, two sentence fragments are lacking subjects: "Running out . . ." and "can come back again." Only word order is relatively well preserved. One of the interesting questions is, then, is this patient's grammar affected by his dementing illness? Or is an intact grammar unable to function burdened by loss of memory, inattention and lack of self-monitoring? To begin to answer these questions, a closer look at some individual cases and specific linguistic deficits is necessary.

The lexicon

Patients begin to experience word-finding difficulty early in dementing illnesses. It is normal to have transient problems in finding words from the mental lexicon, of course. Everyone has experienced the "tip of the tongue" feeling that comes when the name for something once known cannot be retrieved. Both experiential and experimental evidence suggest that the representation of the missing word has not been erased from our mental dictionary. The most usual case is for the "missing" word to reappear after a

time, without outside help. Often, even during the time in which the word cannot be recalled, some information about the word is available. The number of syllables, some sound(s) or letter(s) or the stress pattern of the word may be recalled. Of course this partial information is not always completely accurate.

Picture a faculty member attempting to remember the name of a student. She says ". . . five letters, two vowels, three consonants – alternating." The name turns out to be "Morris." In this case the "mistake" in recollection might be explained by an appeal to a phonological (one [r] sound) rather than orthographic (two "r" letters) memory. Our partial recall of words we temporarily cannot access is not always even this accurate. However, the very phenomenon of remembering some information about "missing" words and eventually recalling them without re-learning them suggests that access to lexical representation is at issue and not the representation itself. Interestingly, aphasics can often tell information about words that they cannot recall, as can normal individuals, including elderly individuals after the age of seventy who have more trouble remembering the names of things than younger ones. However, with patients with dementia it is hard to get them to engage in this task, so we cannot use tip-of-the-tongue data to be sure whether their problem is with accessing the words or with loss of the actual words themselves. One way to judge which of these two is the case is to see if a given word consistently provides problems across modalities (for example, in using the writing system as well as the auditory system). There is still controversy in the field as to the extent to which patients with Alzheimer's disease exhibit a consistent loss of lexical items.

Another way to look at this issue is to determine when the inability to name things may even extend to common objects in some dementing patients such as WLP, the patient with Alzheimer's disease described in Schwartz et al. (1979). Although she was unable to name even five percent of common objects presented to her in pictures relatively early in her dementing illness, she was seventy percent accurate in choosing the correct answer from among several multiple-choice options. Multiple-choice naming tests are also designed in such a way that the pattern of *wrong* choices a person makes also provides information

about their knowledge of words. For example, given a picture of a fork, the multiple-choice answers might be:

(a) pork (similar in sound and spelling)
(b) knife (related in meaning)
(c) lamp (unrelated)
(d) fork (correct)

On ninety percent of the test items for which WLP did not choose the correct answer, she chose the semantically related item. This suggests that the picture was meaningful for her in spite of the fact that she could not consistently choose the correct lexical item: a question of difficulty in *access* rather than lost representation.

Later on in the course of her illness, WLP made even more naming errors. She also chose fewer semantically related words and more unrelated words. Her performance on picture-matching tasks began to evidence some breakdown in semantic categorization.

Research with normals has shown that semantically related words are linked to each other in the mental lexicon. Hearing one word in a semantic area (e.g. *nurse*) makes a person quicker in making judgments about the status of related words (e.g. *doctor*). WLP's choice of semantically related words on the picture-naming task demonstrates that in the early stages of her illness, these connections in the lexicon were still present and available to her for completing the naming task. As WLP's dementia progressed, however, her organization of semantic fields became impaired. Given pictures of dogs, cats and birds to associate with the labels "dog," "cat," and "bird," WLP frequently labeled pictures of cats "dog." She very rarely made this mistake with pictures of birds. Her sub-grouping of animals was principled but non-standard. This impairment extended beyond the purely linguistic level. When asked to match pictures of various dogs or cats to pictures of a typical dog, WLP always chose the cat.

Syntax and paragrammatism

Not only the lexicon is affected in Alzheimer's dementia. That other aspects of the grammar are not entirely spared is demon-

strated by the uneven effects of dementing illness on different aspects of speech production. If inattention were the sole cause of speech errors in dementing patients, as we said above, the distribution of the errors should not be skewed toward any grammatical category.

In the speech of LB, a dementing patient whose speech is described in Obler (1983), paragrammatic errors, that is, errors which evidence disturbed grammatical representation, or, at least, production processes, do occur regularly. Some errors, such as omissions, affect all parts of speech equally. When LB intended to refer to the name of the guy next door, he said "The guy next name," clearly a case of omission for "the guy next door's name." (Obler, 1983: 276.) However, other kinds of substitution errors and errors where the patient repeats an item said previously, now inappropriately (called perseverative errors), tend to occur more frequently with inflectional endings, suggesting some disturbance in the grammar itself.

Consider the following example of LB describing a war experience in which he perseverates in the past-tense form when he uses the word *lined*.

> Whenever they felt like it they would flip uh out machine guns and set them out and tell 'ems, us to *lined* up just to scare the devil[3] but we didn't know when they were going to pull them.

One famous example of a dementing person with an extreme dissociation between the functioning of formal grammar and the ability to produce meaningful utterances is described in Whitaker (1976). Her patient, HCEM, was a 59 year old woman who suffered from presenile dementia, a term used at that time for likely Alzheimer's disease starting before age 65. She had little or no spontaneous speech. She could not name even common objects nor accurately read numbers. Her speech during examinations was largely echolalic, that is, she repeated the examiner's utterances. However, many aspects of her language behavior in testing situations seemed to indicate a relatively intact grammar. If the examiner began a familiar song, HCEM would finish it. The examiner spoke a version of English strongly influenced by British English, yet HCEM's repetitions were all in her own American dialect.

Each of these behaviors is compatible with two possibilities: it may be that HCEM's grammar is intact and it guided her production of the utterances, or it may be that she has, like all of us, a separate store of over-learned material and that is how she was able to complete the song. However, HCEM would also correct intentionally deviant utterances. She would make phonological corrections if the named object was in her field of vision. For example, with a dollar bill on the table, the examiner (HW) would say "tollar bill" and HCEM would say "dollar bill." If the object was not present, HCEM reproduced the phrase with the phonemic "error." Perhaps most interestingly, HCEM corrected grammatically incorrect sentences in her repetitions. If HW said "I have hair gray," HCEM repeated "I have gray hair." She also corrected word order in major constituents, person and number agreement and tense usage. When reading morphologically complex words, she often read another complex word with the same root (e.g. *intuitive* for *intuition*). In short, she showed signs of having some phonological and semantic representation for words as well as some sensitivity to syntactic features of sentences. Whitaker referred to this case as an example of isolation of the language function. HCEM's grammar survived the damage to her brain but lost its ability to interface with other cognitive systems.

Spared language abilities

Interestingly, automatic aspects of language are spared until quite late in Alzheimer's disease. For a long time it was thought that patients remain able to read aloud with no difficulties. Recently Patterson and her colleagues (1994) report that irregularly spelled low frequency words (such as *yacht*) do pose problems for these patients. Nevertheless in the early and even middle stages the ability for patients to read aloud, when they make so many naming errors and have empty speech, is quite impressive.

The other automatic language abilities that remain with them quite late are the ability to appropriately produce phonology (although they make speech errors that often go uncorrected, (McNamara et al., 1992), unlike normal elderly who make markedly more speech errors than young, but correct them equally often to

young normals), and surface syntax (although as we said, paragrammatisms may occur). The patient who reported that the woman in the Cookie Theft Picture was in a turmoil of smin, also pointed out that "it's quite torrential here, although not torrential here," locating a low-frequency lexical item to describe the overflowing water, and using the relatively low-frequency functor "although." Indeed, this choice of low-frequency functors, even if they are not used semantically correctly, appears to distinguish patients with Alzheimer's disease from those with Wernicke's aphasia who manage to avoid them, choosing the logically simpler functors *and*, *or*, and *but* to phrase their correctly structured sentences.

Some pragmatic abilities, however, are spared until quite late in these patients. Thus in the early stages, when patients are unable to remember things or remember the names of things, they will comment on this, perhaps with a self-deprecating chuckle. In the middle stages, even when the patients are producing quite nonsensical discourse in large amounts, the firm examiner can interrupt them. One patient kept noting to one of us (LKO) that I was so "normal," suggesting, although he exhibited little distress, that he had some awareness of his problems. Eye contact is still quite well preserved until late stages, as is the ability to appropriately be quiet in conversation when the examiner interrupts one firmly, and then to speak, whether the speech is related or not, when the examiner has asked a question, or otherwise indicated it is time for the patient's conversational turn.

Conversation and other pragmatic abilities

In the chapter on right-brain damage, we discussed some of the principles involved in constructing appropriate discourse. In the course of dementing illness, patients are likely to develop difficulties in observing principles of appropriate quantity, relevance, and manner. These inabilities extend to non-verbal communication as well. Bayles, Kaszniak and Tomoeda (1987) studied this phenomenon in twenty severely demented patients: eighty-five percent of the patients made eye contact appropriately; sixty-four percent answered appropriately when thanked for their time. Half would correct an incorrect statement made deliberately by the examiner,

and verbally interrupt the examiner. Nearly half the patients could appropriately shake hands when the examiner stuck out a hand in the traditional gesture. But only a quarter would clarify one of their responses when the examiner requested that they do so, and fewer than one in twenty complimented the examiner after being themselves complimented.

Although not every aspect of conversational ability is equally impaired in these patients, all of the functions studied are significantly impaired. The authors do not discuss the extent of formal linguistic deficits in their patient population and it is important to bear in mind that these kinds of abilities are dissociable.

Hamilton (1994a) has documented the development of conversational breakdown in a patient with Alzheimer's disease over the course of several years. The patient's ability to initiate topics or to keep to Hamilton's topics increased progressively; by the later stages her role in "conversation" was virtually nil.

Subpatterns of decline in Alzheimer's dementia

In recent years, it is becoming clear that there are a number of subpatterns of decline, even within specific syndromes like dementia of the Alzheimer's type. Some patients get markedly more prominent language disturbances in the earlier stages, while others show markedly more predominant non-language decline. Many believe that predominant language disturbance is linked to an earlier onset of Alzheimer's disease. In some rare families in which Alzheimer's disease occurs in fifty percent of the children, onset of language problems may be as early as the mid-thirties or forties, although patients find ways of masking them; one patient LKO tested covered his mouth when speaking so completely that the listener could not be sure if the mumbling made sense or not. By contrast, patients with onset in their eighties are more likely to have substantially spared language abilities as compared to their other cognitive and daily self-care abilities. Why earlier onset should be associated with more severe language problems is unclear. Our focus in this chapter, of course, has been on the sizable group of individuals who do evidence language disturbance.

Comparison of the language disturbances of aphasia and dementia

A comparison of the language problems of particular patients with aphasia and with dementia allows for a comparison of the cognitive versus linguistic decline in each of the two forms of brain damage.

Both aphasic and demented patients produce some speech with disturbed form and some with unusual content. In general, it is the demented population that shows a greater weakening of the ability to encode meanings, the connection between cognition and language.

For example, both Wernicke's aphasics and demented patients experience difficulty with the use of functors. However, as explained in Obler (1983), the typical Wernicke's patient uses only a subset of the functors available in his or her language, with a marked preference for the "emptier" ones such as "and." Demented patients, on the other hand, are more likely to *misuse* a wide range of functors, as can be seen from the following examples from LA (Obler, 1983):

(a) I'd like a cup of coffee *but* I'd like one.
(b) The mother's *neither* caring, the son's *neither* caring *or* can't help it.

Stevens (1991) has suggested that this kind of difference in patterns of errors might serve as a diagnostic for differentiating linguistic disturbances in aphasia and SDAT (senile dementia of the Alzheimer's type). On a test battery with many subtests, the clearest differences were found in tasks involving descriptions of actions and the use of objects. SDAT patients were more likely to not respond, respond with something irrelevant, or perseverate on a previous response. Aphasics in this study, although sometimes giving incomplete responses, were more likely to circumlocute or produce phonemic paraphasias (substitutions of one or two phonemes in the word) or neologisms (strings of phonemes that sound like words but cannot be identified with any possible target word in English). The SDAT patients were exhibiting difficulties in communication that, while perhaps involving lexical or grammatical

disturbances, surely are centered more in the area of discourse and conversational skills.

Progressive aphasias

We usually think of the linguistic disturbances of aphasia as having a sudden onset and those of dementia, a more gradual onset. There are, however, cases of progressive aphasia brought on by brain disease rather than stroke or injury. Diagnosing such a patient as aphasic rather than demented is difficult because we associate progressive decline with the dementias. However if language is the only area impaired, and the patient continues to perform normally on all other cognitive tasks, the diagnosis must be progressive aphasia. Here we can see similarities as well as points of departure.

One patient with progressive aphasia without dementia is described by Parkin (1993). This patient, TOB, had great difficulty in word finding in certain circumstances. He had difficulty providing definitions of common words. His errors ranged from overly vague ("needle" defined as "more positive application to females") to wrong (for example, he considered "squirrel" to be "a bird that flies"). However, his comprehension was essentially intact. On a test of matching spoken words to pictures, TOB made no errors. He was also quite capable of naming described objects. Occasional instances of non-comprehension occurred mainly with low-frequency words such as "dissertation."

TOB had difficulty in reading words with irregular spelling, often regularizing the pronunciation, and a parallel difficulty in writing/spelling. Receptively, however, his performance was much better. On a lexical decision task he correctly identified all of the items that were deemed "pseudo-homonyms," that is irregularly spelled words that, when spelled regularly, are in fact not correctly spelled words in English (e.g. *goast*). In sum, the fact that his skills in processing "input" remained spared while those for "output" were impaired permits us to call this an aphasia rather than a dementia. The fact that it got progressively worse is what was surprising since he did not have a tumor. The causes of progressive aphasia remain unknown; only recently has it been distinguished from the cortical dementias.

Distinguishing the language changes of Alzheimer's disease from those of normal aging

In the early period, when the primary naming problem looks like the naming problem of anomic aphasia, it is not only important to distinguish the language changes of Alzheimer's disease from aphasia. It must also be distinguished, at that stage, from the naming problem of normal aging. Some scholars have argued that, indeed, the language changes associated with Alzheimer's disease are simply an exaggeration of what happens with normal aging. Conversational conventions can be broken in both normal adults as well as demented patients. These include the self-centeredness in discourse (which could result from substantial hearing loss that the speaker wants to mask from the listener) and repeating things one has said before, not remembering one has said them before, nor marking them as one is expected to pragmatically with a phrase such as "as I told you before."

Some behaviors, however, are seen in demented patients that are never seen in normals. One of these is the response to phonemic cues on a naming test. Such cues are very helpful in enabling even very old, normal, elderly patients to recover the correct lexical item that they can not remember the name of. Even when they do not help, normal elderly, unlike patients with Alzheimer's disease, will never simply blurt out a word that begins with the sound (a demented patient, by contrast, when given the cue /b/ for the word "beaver" might say *banana*, not recognizing the inappropriateness of such a response). Also, patients with Alzheimer's disease will sometimes blithely produce neologistic terms in their speech (like the example at the beginning of this chapter of the patient whose word "smin" had no obvious target); normal elderly never will.

Bilingual dementia

Although we discuss bilingualism in more detail in chapter 10, it is worth mentioning bilingual dementia here, as there are additional aspects of linguistic competence to be considered. Just as we see that bilingual aphasics might be found to have quite disparate

linguistic skills in each of their languages after brain injury, brain disease may have different effects on each of a patient's languages. Much more than in aphasic patients, the most spared language is likely to be the one learned first, even if it is not the language used most before the onset of the disease or the language of the post-onset environment. Table 8.1 from De Santi et al. (1990) provides an example of different linguistic deficits in the two languages of four Yiddish-English bilinguals.

A healthy bilingual has the ability to produce and/or comprehend utterances in more than one language. The healthy bilingual has an internal set of rules governing language choice. In the case of bilinguals who make use of the resources of both languages in conversations with other bilinguals, the languages are not mixed randomly. The choice of switch points is governed by a set of principles discussed in chapter 10. As mentioned above, research on language dissolution in monolingual dementia patients has shown that conversational skills such as knowing when to take one's turn are relatively well preserved in mild stages of dementia whereas other discourse skills, such as appropriately keeping to the topic (topic maintenance; Hamilton, 1994a), and repairing speech errors (Hamilton, 1994b, McNamara et al., 1992) are more severely affected early in the dementing process.

The effects of dementing illness on bilinguals' language-choice pragmatics are quite striking (Hyltenstam and Stroud, 1989). De Santi et al. (1990), for example, report on testing and conversation with the same four bilinguals whose linguistic difficulties are summarized in the table below. Each patient was interviewed in English by a monolingual interviewer and in Yiddish by an interviewer who, although bilingual, used only Yiddish throughout the interviews. Language choice by the demented patients ranged from almost entirely appropriate to almost never appropriate. One patient, patient D, who did have problems in both of his languages, nevertheless always chose the right language for the interviewer. Patient C, the most severely demented of the four, often chose the wrong language in both the Yiddish and English contexts. Patient E sometimes spoke Yiddish with the monolingual English examiner, a more marked error than that of Patient B whose only

Table 8.1. *General language behavior of four bilingual demented subjects*

Language Behavior	Patient B		Patient C		Patient D		Patient E	
	E	Y	E	Y	E	Y	E	Y
Naming problems	+	n/a	+	+	+	—	+	+
Paraphasic errors	+	—	+	+	+	—	+	+
Neologisms	—	—	+	+	+	—	+	+
Circumlocutions	—	—	+	+	+	—	+	+
Perseveration	+	—	+	+	+	+	+	+
Illogical responses	—	—	+	+	+	+	+	+
Topic loss	—	—	+	+	+	—	+	—

Note: Key: E = English; Y = Yiddish; n/a = not available; + = The problem listed was evidenced in the sessions; — = The problem listed was not evidenced in the sessions.

Source: From De Santi, S., L. K. Obler, H. Sabo-Abramson, J. Goldberger. Discourse abilities and deficits in multilingual dementia, in Joanette, Y. and H. Brownell, eds., *Discourse ability and brain damage*, New York: Springer-Verlag, 1990: 224.

Table 8.2. *Code-switching with bilingual and monolingual interlocutors*

With bilingual interlocutor

Subject	C-S	+EQ	−EQ	−FM
Patient B	4	4	0	0
Patient C	87	72	15	3
Patient D	24	23	1	0
Patient E	57	50	6	1

With monolingual interlocutor

Subject	C-S	Utter	+EQ	−FM
Patient B	0	350	0	0
Patient C	68	888	68	0
Patient D	0	944	0	0
Patient E	23	1664	21	0

Note: Key: C-S = Number of code-switches; Utter = number of utterances; +EQ = number of code-switches that follow the equivalence constraint; −EQ = number of code-switches that do not follow the equivalence constraint; −FM = number of code-switches that do not follow the free morpheme constraint.
Source: From De Santi, S., L. Obler, H. Sabo-Abramson, J. Goldberger, Discourse abilities and deficits in multilingual dementia, in Joanette, Y. and H. Brownell, eds., *Discourse ability and brain damage*. New York: Springer-Verlag, 1990: 224.

"wrong" choice was to speak English in the Yiddish interview which was conducted by a bilingual.

The subjects in De Santi et al. (1990) also differed in the extent to which they code-switched during the interviews. Although the Yiddish interview was intended to be entirely in Yiddish, the interviewer's bilingualism created an appropriate context for code-switching and all four subjects did switch to some extent. Two of the subjects, patients C and E, also inappropriately switched into Yiddish with the monolingual interviewer.

Examining the code-switches themselves according to the criteria outlined in chapter 10, De Santi et al. found that the vast majority of code-switches were of the linguistic type made by normal bilinguals. These linguistic-rule-governed aspects of bilingual language behavior seem to be more resistant to the effects of dementing illness than are the pragmatic abilities of these patients which are presumably governed by "executive" functions associated with the frontal lobes.

Conclusion

In subcortical dementias, the problems lie most obviously with speech rather than language. However, subtle problems can be seen with naming, lexical selection, list generation, affixal morphology (in writing, at least), and sentence comprehension.

In the cortical dementias, especially those which begin at a relatively young age, language problems are more evident. While phonology, surface-level syntax, and reading aloud are relatively spared, lexicon and semantics are severely impaired. Simple pragmatic abilities, such as responding when a question is asked and keeping eye-contact, are spared until relatively late in the course of the disease, but more complex ones such as inference or monitoring what the other participant in a conversation already knows, disappear. Non-language aspects of cognition (in particular declines in memory, attention, and the ability to manipulate ideas) contribute heavily to the apparent linguistic decline. These are linked to the frontal, parietal, and temporal lobes where cellular changes are most severe. Although language can be impaired independent of thought, as we have seen in the chapters on aphasia, it seems that thought cannot be impaired without impairment in language performance.