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, I.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
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...candy...well I don’t know but it’s writ...easy does it...slam...early...fall...men...many no...girl...dishes...soap...soap...water...water...falling pah that’s all...dish...that’s all.
cookies...can...candy...cookies...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 (“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
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 time 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 time took around here and saw me around in it it’s started with me no time and then ... 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 “log-off.” 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? 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 /hænd/) 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 agrammatists' linguistic abilities. Still others attempt explanations which would account for all of the observed symptoms.
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.

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*).

<table>
<thead>
<tr>
<th>Age</th>
<th>Usual language development</th>
<th>Effect on language of left lateral lesions</th>
<th>Other remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–3 months</td>
<td>- Emergence of cooing</td>
<td>- No effect in 50% of cases; 50% with delayed onset (but normal development)</td>
<td>- No lateralization of function</td>
</tr>
<tr>
<td>4–20 months</td>
<td>- from babbling to words</td>
<td></td>
<td>- Hand preference emerges</td>
</tr>
<tr>
<td>21–36 months</td>
<td>- Acquisition of language structure</td>
<td>- All language accomplishments disappear; language is reacquired with repetition of all stages</td>
<td>- Left hemisphere begins to assume sole responsibility for language</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Language appears to involve whole brain</td>
</tr>
<tr>
<td>3–10 years</td>
<td>- Grammatical refinement and expansion of vocabulary</td>
<td>- Aphasic symptoms: tendency for full recovery (except in reading and/or writing)</td>
<td>- Evidence for both hemispheres still active in language; right/left lateral-lesion disrupts language</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Possible to re-establish language in right hemisphere if left is damaged</td>
</tr>
<tr>
<td>11–14 years</td>
<td>- Foreign accents in 2nd language learning</td>
<td>- Some aphasic symptoms are not reversible, particularly in traumatic lesions</td>
<td>- Lateralization is formally established - usually irreversibly</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Language-free parts of brain cannot take over except where lateralization is incomplete (due to childhood pathology)</td>
</tr>
<tr>
<td>Mid-teens-senium</td>
<td>- Acquisition of 2nd language is increasingly difficult</td>
<td>- Aphasic symptoms may persist; symptoms present for more than 3–5 months are irreversible</td>
<td>- Language definitely lateralized in left hemisphere for 97% of population</td>
</tr>
</tbody>
</table>
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 hurtsilly 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 sap to /sæst/ and then added the syllabic plural to all the remaining items (e.g. /zæplz/). 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 went swimming’”) (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 aphasias 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.