

24 Neurolinguistics

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The field of neurolinguistics has come to consist of two related areas of study: language disorders (which is sometimes called “aphasiology”) and the relationship between language and the brain. Aphasiology has made important discoveries about what goes wrong with language after brain damage, some of which have implications for what language is and how it is processed. Aphasiology is closely linked to the study of the relationship between language and the brain. The combined study of patients’ language deficits and neurological lesions provides evidence about the location and type of brain damage that affects language. This indirectly gives us information about how the brain is organized to process language normally. In fact, this traditional “deficit-lesion” or “clinico-pathological” correlational approach was the only way that we could learn about this subject for many years. This approach has been considerably refined as more detailed descriptions of language disorders have been produced that draw on linguistics and psychology and as advances in neuroimaging allow for much more precise and complete characterization of lesions than previously possible.

In recent years, the traditional deficit-lesion correlational approach to brain organization for language has been complemented by studies of brain function in normal subjects when they perform tasks that involve language. “Functional neuroimaging” uses positron emission tomography (PET) and functional magnetic resonance imaging (fMRI), which primarily measure regional cerebral blood flow (rCBF). Measurements can also be made on the scalp of electrophysiological potentials (“event related potentials” – ERPs) and small electromagnetic potentials (magnetoencephalography) to record neural activity that arises in relationship to language functions. In the late 1980s, most of these techniques were in their infancy, if they existed at all, and their evolution and application to the study of the relationship of language and the brain has made the present period one of the most exciting in the history of neurolinguistics. In this chapter, I shall present an introduction to this rapidly evolving field.

1 Aphasiology

1.1 *A very brief history of aphasiology*

Though it was known as far back as ancient Egypt that damage to the brain affected language, the scientific investigation of the relationship of the brain to language dates to the mid-nineteenth century. Speculative work by Brouillaud, Lordat, and others set the stage for the paper that started and defined both aphasiology and neurolinguistics – Paul Broca’s presentation to the Anthropological Society of Paris of the now famous case of Leborgne (Broca 1861). We shall review the “neurolinguistic” part of this paper below. The “aphasiological” part of the paper is fairly simple, but had profound effects on the field. Broca’s case was a 57-year-old man who had come into the hospital 21 years earlier unable to speak. He could only utter the syllable “tan.” He appeared able to understand what people said to him, and he could express his needs and emotions through gesture and by changing the tone of his voice. Over the years he developed a right sided weakness, and eventually became bedridden. He died of an infection that began in bed sores in his right leg, which had become ulcerated and infected as a result of having been lain on and improperly attended to. However, for the entire period of his hospitalization, his language capacities remained static – at least according to the information that Broca had and that he relayed to the Anthropological Society.

Broca’s analysis of the language disorder in “Tan” was that he had lost the “faculty of articulate speech” while retaining the faculty for language comprehension and those for production and recognition of meaningful gestures. With this analysis, Broca defined the approach to aphasia that dominated medical and scientific thinking for decades – to think of language disorders as disorders of the ability to use language in the usual tasks of speaking, understanding, reading, and writing. This approach focussed attention on the common tasks by which linguistically based communication takes place. This is a domain of human linguistic functioning that, though complicated, is much more tractable and much better understood than domains such as verbally based reasoning and planning, self-expression, verbal creativity, etc. From the neurolinguistic point of view, these basic functions of speech, comprehension, reading, and writing are related to a relatively small part of the brain. Thus, this focus allowed researchers to make some headway into the question of how the brain is related to language functions. However, the division of language functions into these large areas also had its drawbacks as well. For decades it inhibited investigation of the components of these psycholinguistic tasks, which has had negative effects on aphasiology, neurolinguistics, and approaches to therapy. Fortunately, modern aphasiology has begun the task of taking apart tasks such as speaking, comprehending, etc., into their components and describing language disorders in terms of these components. Before

turning to these modern studies, however, we should briefly review the “classic” clinically defined aphasic syndromes, since they still are commonly referred to by clinicians who diagnose and treat language disorders.

These “aphasic syndromes” are shown in table 24.1. “Broca’s aphasia” is a severe expressive language disturbance reducing the fluency of speech without an equally severe disturbance of auditory comprehension. “Wernicke’s aphasia” consists of the combination of fluent speech with erroneous choices of the sounds of words (phonemic paraphasias) and an auditory comprehension disturbance. “Pure word deafness” is the relatively pure case of an auditory receptive disorder in which the patient does not recognize spoken words, so spontaneous speech is normal but comprehension and repetition are disturbed. “Anarthria,” “dysarthria” and “apraxia of speech” are output speech disorders in which both repetition and spontaneous speech are misarticulated but comprehension is preserved. In “transcortical motor aphasia,” spontaneous speech is reduced but repetition is intact; in “transcortical sensory aphasia,” a comprehension disturbance exists without a disturbance of repetition. A disturbance in spontaneous speech and repetition without a disturbance in auditory comprehension is termed “conduction aphasia.” All of these syndromes were claimed to have been discovered in relatively pure form by researchers by 1885 (Lichtheim 1885). This classification was revived by Normal Geschwind in the 1960s (Geschwind 1965).

These syndromes reflect the relative ability of patients to perform language tasks (speaking, comprehension, etc.), not the integrity of specific components of the language processing system. For instance, the speech production problem seen in Broca’s aphasia can consist of one or more of a large number of impairments – disorders affecting articulation such as dysarthria or apraxia of speech, disorders affecting sentence form such as agrammatism, etc. Patients with Wernicke’s aphasia can have deficits affecting either the sounds of words or their meanings or both, as well as any number of other language processing deficits. There are at least two major deficits that underlie “conduction aphasia,” one affecting word production and one affecting verbal short-term memory. At the same time as patients with the same syndrome can have different deficits, identical deficits occur in different syndromes. For instance, certain types of naming problems can occur in any aphasic syndrome. For these reasons, knowing that a patient has a particular aphasic syndrome does not tell us exactly what is wrong with his or her language. For that, one has to turn to more detailed studies.

1.2 *Modern aphasiology*

The contemporary approach to language disorders provides such studies. It sees human language as a code that links a set of linguistic forms to a number of aspects of meaning. These forms are activated in the usual tasks of language

Table 24.1 The classic aphasic syndromes

<i>Syndrome</i>	<i>Clinical manifestations</i>	<i>Hypothetical deficit</i>	<i>Classical lesion location</i>
Broca's aphasia	Major disturbance in speech production with sparse, halting speech, often misarticulated, frequently missing function words and bound morphemes	Disturbances in the speech planning and production mechanisms	Primarily posterior aspects of the third frontal convolution and adjacent inferior aspects of the precentral gyrus
Wernicke's aphasia	Major disturbance in auditory comprehension; fluent speech with disturbances of the sounds and structures of words (phonemic, morphological, and semantic paraphasias)	Disturbances of the permanent representations of the sound structures of words	Posterior half of the first temporal gyrus and possibly adjacent cortex
Anomic aphasia	Disturbance in the production of single words, most marked for common nouns with variable comprehension problems	Disturbances of the concepts and / or the sound patterns of words	Inferior parietal lobe or connections between parietal lobe and temporal lobe
Global aphasia	Major disturbance in all language functions	Disruption of all language processing components	Large portion of the perisylvian association cortex
Conduction aphasia	Disturbance of repetition and spontaneous speech (phonemic paraphasias)	Disconnection between the sound patterns of words and the speech production mechanism	Lesion in the arcuate fasciculus and / or corticocortical connections between temporal and frontal lobes
Transcortical aphasia	Disturbance of spontaneous speech similar to Broca's aphasia with relatively preserved repetition	Disconnection between conceptual representations of words and sentences and the motor speech production system	White matter tracts deep to Broca's area

Table 24.1 Cont'd

<i>Syndrome</i>	<i>Clinical manifestations</i>	<i>Hypothetical deficit</i>	<i>Classical lesion location</i>
Transcortical sensory aphasia	Disturbance in single word comprehension with relatively intact repetition	Disturbance in activation of word meanings despite normal recognition of auditorily presented words	White matter tracts connecting parietal lobe to temporal lobe or in portions of inferior parietal lobe
Isolation of the language zone	Disturbance of both spontaneous speech (similar to Broca's aphasia) and comprehension, with some preservation of repetition	Disconnection between concepts and both representations of word sounds and the speech production mechanism	Cortex just outside the perisylvian association cortex

use – speaking, auditory comprehension, reading, and writing – by different “components” of a “language processing system.” For instance, a component of the language processing system might accept as input the semantic representation (meaning) activated by the presentation of a picture and produce as output a representation of the sound pattern of the word that corresponds to that meaning.

At least four levels of language representation are clearly identified in contemporary models of the language processing system: the *lexical level*, the *morphological level*, the *sentential level*, and the *discourse level*. Lexical items (simple words) designate concrete objects, abstract concepts, actions, properties, and logical connectives. The basic form of a simple lexical item consists of a phonological representation that specifies the sounds (phonemes) of the word and their organization into rhythmic structures (e.g., syllables). The morphological level of language allows the meaning associated with a simple lexical item to be used as a different syntactic category (e.g., the suffix *-tion* allows the semantic values associated with a verb to be used as a noun, as in the word *destruction* derived from *destroy*). This avoids the need for an enormous number of elementary lexical items in an individual's vocabulary. Morphology also signals grammatical information such as agreement (e.g., *I run; he runs*). The sentential level of language makes use of the syntactic categories to build syntactic structures (e.g., noun phrase, verb phrase, sentence, etc.) that define relationships between words relevant to the meaning of a sentence (its “propositional

content"). Sentences convey information about who did what to whom (thematic roles), which adjectives go with which nouns (attribution or modification), the reference of pronouns and other referentially dependent categories, etc. They make statements that can be true or false, and that therefore can be used in logical and planning processes and serve as a means for updating an individual's knowledge of the world. Discourse conveys information about temporal order, causation, the number of entities involved in an event, which of these entities is in the current focus of attention, and other similar aspects of meaning. The structure of a discourse depends upon a complex interaction between linguistic forms (such as whether an entity in the discourse is referred to by a noun or a pronoun) and non-linguistic functions, such as what a person knows and infers. For more details regarding the structure of language, see chapters 11 and 16.

These levels of the language code are all activated in parallel in the acts of speaking, understanding, reading, and writing. Consider speaking, for instance. A speaker selects words to convey concepts on the basis of his / her knowledge of a subject and also as a function of what words s/he thinks the listener will understand. S/he selects the sounds of each word and their proper order. S/he puts the words into the grammatical structures needed to convey the relationships between their meanings and finds the right morphological form for each word in each grammatical position. S/he selects intonational contours that express whether a sentence is a statement, a question, a command, or has some other illocutionary force. Intonational contours and syntactic structures are also selected to express the focus of the discourse. The speaker sends commands to the muscles of the chest, the diaphragm, the larynx, and the oral and nasal tracts to integrate the movements of all these regions in a way that produces exactly the right sounds for each word with the right degree of stress. All these operations go on unconsciously, at a speed of about 120 words per minute, or 2 words per second, or roughly 1 sound (phoneme) every 100 milliseconds or so. A speaker is not only fast at doing these remarkable computations and retrieval operations; s/he is also accurate. Estimates of the number of errors that a speaker makes are in the range of one semantic mistake every million words and a comparable number of sound-based errors (see Levelt 1989, for an extensive review of these processes).

Speaking is a remarkable act, but no more so than understanding spoken speech (where the words, structure, and meaning of an utterance are extracted from the acoustic signal), or reading or writing. All these functions are the result of the integrated activity of dozens of highly specific operations acting in parallel and in critically ordered sequences. More detailed discussion of language processing can be found in chapter 16.

Modern aphasiology tries to understand disturbances of speaking, comprehension, reading and writing as disruptions of specific language processing components. It is impossible to present a review of all the disturbances that have been described that affect each of the components of the language processing system in each of the tasks of speaking, comprehending, reading, and

writing. I shall instead sample from two areas of language – disorders affecting patients' abilities to understand the meanings of words and disorders affecting their abilities to understand sentences.

1.3 Disturbances of word meanings

Most recent research on disturbances of word meanings in brain damaged patients has focussed on words that refer to objects. The meanings of these words are thought to be stored in a specialized memory store, called "semantic memory" (Tulving 1972, 1983). Though semantic memory also is thought to house representations of entities other than objects (such as events, for instance), the concepts that correspond to objects have been the subject of the most extensive thought and investigation in philosophy and psychology. The representations of objects in semantic memory have traditionally been thought of as sets of features that list the properties that are necessary and sufficient for an item to fall into a given category, such as that for an entity to be a bird it has to have feathers and to fly. It is now appreciated that most concepts are not easily described in these terms, and that categorization must depend on probabilistic criteria (see Smith and Medin 1981, for discussion). For instance, penguins and ostriches are birds but do not fly. The concepts corresponding to concrete objects may be represented in both a "verbal" and a "visual" semantic memory system (Miller and Johnson-Laird 1976, Paivio 1971), though the evidence for this is controversial (Snodgrass 1984). The verbal semantic system may encode relations of one entity to another and functional properties of each item, such as the fact that some birds fly south in the winter. The visual semantic system may encode physical properties of an item that allow it to be recognized, such as the typical silhouette of a bird.

Disturbances of word meanings thus can be seen as problems affecting the ability of a patient to appreciate how an item is classified or what its necessary and typical features are. Such disturbances would be expected to affect a patient's ability to identify a word or a picture as an example of a specific concept, and therefore to cause poor performance on word-picture matching and naming tasks (Warrington 1975). The co-occurrence of deficits in word-picture matching and naming is not an adequate basis for diagnosing a problem affecting word meaning, however, because a patient may have separate disturbances that affect word recognition and production independently (Howard and Orchard-Lisle 1984). Co-occurring deficits in naming and word-picture matching are more likely to result from a disturbance affecting concepts when the patient makes many semantic errors in providing words to pictures and definitions, s/he has trouble with word picture matching with semantic but not phonological foils, s/he fails on categorization tasks with pictures, and when the same words are affected in production and comprehension tasks (Hillis et al. 1990).

It has been argued that brain damage may affect either the storage or the retrieval of word meanings. Shallice (1988a, 1988b), Warrington and Shallice (1979) and Warrington and McCarthy (1987) have suggested that there are five hallmarks of the loss of items in semantic memory – consistent production of semantic errors on particular items across different inputs (pictures, written words, spoken words), relative preservation of superordinate information as opposed to information about an item's features, relative preservation of information about higher frequency items, no improvement of performance by priming and cueing, and no effect of the rate at which a task is performed upon performance. These researchers have also suggested that disorders of *retrieval* of items from semantic memory is characterized by the opposite effects of these variables on performance. These authors have described patients with semantic impairments, whose impairments they say are of one or the other of these types. The criteria for distinguishing between storage and retrieval impairments remain controversial, however (Caramazza et al. 1990; see Caplan 1992: ch. 4, for discussion).

Disorders affecting processing of semantic representations for objects may be specific to certain types of inputs. Warrington (1975) first noted a discrepancy between comprehension of words and pictures in two dementing patients. Bub et al. (1988) have analyzed a patient, M. P., who showed very poor comprehension of written and spoken words but quite good comprehension of pictures. These impairments have been taken as reflections of disturbances of "verbal" and "visual" semantic systems, though this interpretation is debated (Riddoch and Humphreys 1987).

Semantic disturbances may also be category-specific. Several authors have reported a selective semantic impairment of concepts related to living things and foods compared to human-made objects (Sartori and Job 1988, Silveri and Gainotti 1988, Warrington and Shallice 1984). The opposite pattern has also been found (Warrington and McCarthy 1983, 1987). Selective preservation and disruption of abstract versus concrete concepts, and of nominal versus verbal concepts, have also been reported (Miceli et al. 1984; Schwartz et al. 1979, Warrington 1981a, 1981b, Zingeser and Berndt 1988).

A very interesting feature of many aphasic disorders is that they may affect conscious and unconscious processing differently. Though it is far from clear exactly what it is about certain types of processing that makes them "conscious," it is clear that there is a difference between the way we accomplish functions such as speech production and the way we do other types of tasks, such as long division. The operations involved in speech planning are unconscious; those in long division are controlled and conscious. Milberg and his colleagues (Milberg and Blumstein 1981; Blumstein 1982) have described patients who cannot match words to pictures or name objects, but who show evidence of understanding words unconsciously. The evidence that these patients process meaning unconsciously is that they show "semantic priming effects." These effects consist of responding more quickly to a word when

it has been preceded by a semantically related word in a task that does not require the subject to process the word's meaning. For instance, if a subject is shown sequences of letters on a computer screen and asked to press a button to indicate whether a sequence is a word or not (a "lexical decision" task), s/he will respond faster to the letter string DOCTOR when it follows the string NURSE than when it follows the string TABLE. Milberg and his colleagues have found these priming effects in some patients who do not show evidence of understanding words when the task requires conscious processing of a word's meaning, suggesting that they are able to appreciate the meanings of words unconsciously but not process meaning in conscious, controlled tasks like word-picture matching. Conversely, Swinney et al. (1989) have shown that some patients who appear to understand words well may have abnormalities in tasks that examine unconscious processing of the meanings of words. These researchers reported that four aphasic patients who were good at word-picture matching performed abnormally in a priming task with ambiguous words (like *bank*). Unlike normals, who showed priming for both senses of an ambiguous word, the four patients studied by Swinney and his colleagues only showed priming for the most frequent sense of ambiguous words. This indicates that their unconscious processing of word meanings was abnormal, even if it did not affect their performance on untimed, controlled, conscious tasks such as word-picture matching.

1.4 Disorders of sentence comprehension

When a subject understands a sentence, s/he combines the meanings of the words into a propositional content in accordance with the syntactic structure of the sentence. There are many reasons why a patient might fail to carry out the operations that are needed to arrive at propositional content. Disturbances affecting comprehension of simple and morphologically complex words affect comprehension at the sentence level. In addition, there are disturbances affecting patients' abilities to understand aspects of propositional meaning despite good single word comprehension.

The largest amount of work in the area of disturbances of sentence comprehension has gone into the investigation of patients whose use of syntactic structures to assign meaning is not normal. Caramazza and Zurif (1976) were the first researchers to show that some patients have selective impairments of this ability. These researchers described patients who could match "semantically irreversible" sentences such as "*The apple the boy is eating is red*" to one of two pictures but not "semantically reversible" sentences such as "*The girl the boy is chasing is tall*." The difference between the two types of sentences resides in the fact that a listener can understand a sentence such as "*The apple the boy is eating is red*" because boys are animate and can eat, and apples are inanimate and can be eaten, whereas understanding "*The girl the boy is chasing is tall*" requires assigning the syntactic structure of the sentence since both boys and

girls are capable of chasing one another. Caramazza and Zurif concluded that their patients could not assign or use syntactic structure for this purpose.

Disorders of syntactic comprehension have since been examined in considerable detail (Caplan and Futter 1986; Caplan and Hildebrandt 1988; Hildebrandt et al. 1987, Linebarger et al. 1983, Martin et al. 1989, Schwartz et al. 1980, Tyler 1985). Patients may have very selective disturbances affecting the use of particular syntactic structures or elements to determine the meaning of a sentence. For instance, two patients we studied showed a double dissociation in their abilities to understand sentences with reflexive elements (*himself*) and pronouns (*him*) (Caplan and Hildebrandt 1988). Some patients can understand very simple syntactic forms, such as active sentences (*The man hugged the woman*), but not more complex forms, such as passive sentences (*The woman was hugged by the man*) (Caplan et al. 1985). Many of these patients use strategies such as assigning the thematic role of agent to a noun immediately before a verb to understand semantically reversible sentences, leading to systematic errors in comprehension of sentences such as "*The boy who pushed the girl kissed the baby.*" Other patients have virtually no ability to use syntactic structure at all. Most of these patients appear to rely upon inferences based upon their knowledge of the real world and their ability to understand some words in a sentence, as seems to have been the case with the original patients described by Caramazza and Zurif (1976).

As with simple words, some patients can assign and interpret syntactic structures unconsciously but not use these structures in a conscious, controlled fashion. For instance, Tyler (1985) reported a patient whose online word-monitoring performances indicated that he was sensitive to certain syntactic anomalies, but who could not make judgments regarding these same anomalies at the end of a sentence. Linebarger and her colleagues (Linebarger et al. 1983; Linebarger 1990) have reported that some patients who have syntactic comprehension problems (who cannot match reversible sentences to pictures, for instance) can make judgments as to whether or not a sentence is grammatical. For instance, some patients can indicate that the utterance "*The woman was watched the man*" is ill-formed and the utterance "*The woman was watched by the man*" is acceptable, despite not being able to match sentences such as "*The woman was watched by the man*" to one of two pictures. These researchers have interpreted these results as an indication that some patients can construct syntactic structures but not use them to determine propositional meaning (a so-called "mapping" problem – Schwartz et al. 1985). As with other areas of language functioning, it appears that patients may retain unconscious, online sentence comprehension processes but lose the ability to use the products of these processes in a controlled, conscious fashion in some tasks.

Disturbance of short-term memory can produce sentence comprehension impairments, but the connection between the two disorders is more complex than one might suspect. Martin and Ramoni (1994) have related short-term memory impairments to difficulties in understanding parts of sentences that consist of lists of words, such as *big, noisy, and aggressive*, in the sentence *The*

neighbor's dog was big, noisy, and aggressive. However, many case studies show that patients with short-term memory impairments can have excellent syntactic processing abilities (Caplan and Waters 1990, McCarthy and Warrington 1984, Butterworth et al. 1986, Waters et al. 1991). Short-term memory impairments may induce problems remembering the meaning of a sentence, not understanding that meaning in the first place (see Caplan and Waters 1998, for review).

1.5 *Comments on modern aphasiology*

This brief overview merely conveys some of the results of recent research into disorders affecting word meanings and sentence comprehension. Even this brief survey indicates that these disturbances are very complex, and vary in different patients. It shows that studies of brain damaged patients can suggest features of how language is structured, such as the suggestion that word meanings are organized into semantic categories, or that the assignment of syntactic structure is partially independent of the use of that structure to determine sentence meaning. The review indicates that modern aphasiology provides a list of representations and operations that ultimately need to be related to the brain – verbal semantic representations, visual semantic representations, category-specific semantic representations, specific syntactic operations, etc. Most of these representations and functions need to be better understood for us to be sure that what we are relating to the brain are the cognitive operations that are really those used by normal human beings. However, scientists have not waited for these cognitive functions to be completely understood before trying to see how the brain processes language; there are many studies of language–brain relationships that have been undertaken despite our current state of relative ignorance of the true nature of linguistic representations and psycholinguistic operations. Some of these studies will no doubt have to be revised as we discover that our models of language and language processing need changing, but others are quite solid. We now turn to this second aspect of neurolinguistics – the relationship of language to the brain.

2 Language and the Brain

2.1 *The overall organization of the brain for language*

The brain is perhaps the most highly differentiated organ in the body. It consists of a large number of regions, each of which contributes to sensation, motor function, thought, emotion, and other functions in special ways. Only a relatively small part of the brain is devoted to language (figure 24.1). This part

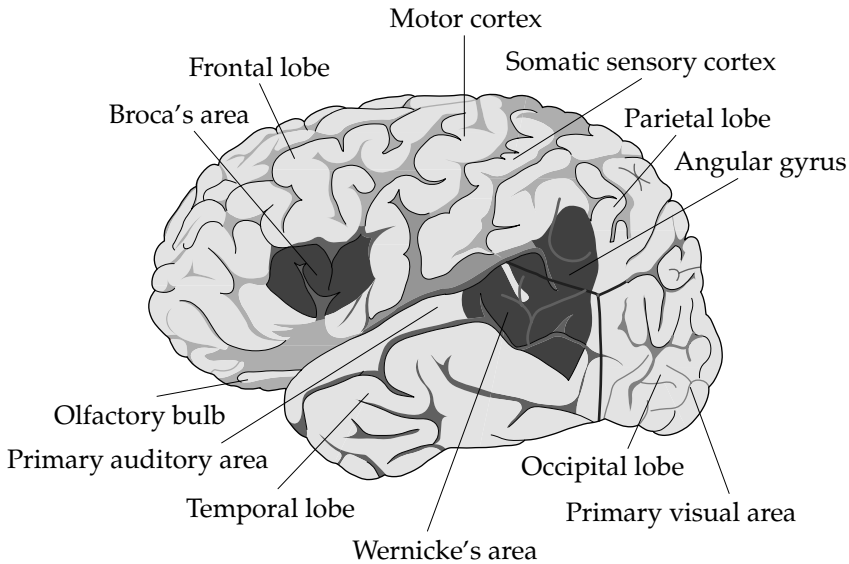


Figure 24.1 Depiction of the left lateral surface of the human brain showing perisylvian association cortex related to language
Source: Adapted from Carpenter 1983

lies in the cerebral cortex – a thin strip of neural cells and supporting tissue along the outermost edge of the brain – and consists of the association cortex in the region of the sylvian fissure (Luria 1970, Russell and Esper 1961, Basso et al. 1985, Weisenberg and McBride 1935, Brown 1972, Pick 1973). This region includes the pars triangularis and the pars opercularis of the third frontal convolution (Broca's area), the association cortex in the opercular area of the pre- and post-central gyri, the supramarginal and angular gyri of the parietal lobe, the first temporal gyrus from the supramarginal gyrus to a point lateral to Heschl's gyrus (Wernicke's area), and possibly a portion of the adjacent second temporal gyrus. On the basis of present knowledge, there is no other *cortical* area that can be confidently thought to subservise language functions. The supplementary motor area is the only other cortical structure that has been suggested to play a role in language processing. However, its primary function in language tasks appears to be to initiate vocalization, not to activate linguistic representations through subserving a component of the language processing system per se (Masdeu et al. 1978).

The cortex does not exist or function alone; it is connected by large white matter tracts to subcortical nuclei in the basal ganglia, the thalamus, and the cerebellum. These structures are part of a "neural system" that processes language. Lesions of both the white matter tracts that connect the language cortex to these subcortical regions and of these regions themselves produce language impairments (Naeser et al. 1982, Cappa et al. 1983, Damasio et al. 1982, Mohr

et al. 1975). However, the exact function of these structures is not completely understood. The white matter tracts presumably do not actually compute representations but only transfer the results of computations from one set of neurons to another. The basal ganglia, thalamus, and cerebellum may actually store and compute linguistic representations, and some researchers have suggested that these subcortical grey matter structures play roles in language processing (Damasio et al. 1982, Mohr et al. 1975, Crosson 1985). However, an alternative possibility is that the language disorders that follow subcortical lesions result from altered physiological activity in the overlying cortex, not disorders of the subcortical structures themselves.

The availability of patients with focal strokes that are visible only subcortically on CT or MR scans, in whom metabolic scanning is used to assess lesion site and size in both cortical and subcortical structures, provides an opportunity to investigate the role that both cortical and subcortical structures play in language. Across all the published cases of subcortical aphasia, there is a 1:1 correspondence between the presence or absence of cortical hypometabolism or hypoperfusion and the presence or absence of aphasic impairments in patients with strokes that are visible only subcortically on CT scans (Perani et al. 1987, Olsen et al. 1986). Moreover, studies correlating the degree of hypometabolism measured cortically and subcortically with the degree of language impairment indicate a much higher correlation of language impairments in aphasic patients with the indices of cortical hypometabolism (Metter et al. 1988, Metter et al. 1983, Kempler et al. 1988, Metter et al. 1987). There is not a single published case in which an aphasia has been documented in a patient in whom metabolic scanning, blood flow studies, and physiological measures have all shown normally functioning perisylvian association cortex. The conclusion that is suggested by these results is that subcortical structures are not themselves responsible for language processing but serve only to activate the cortically based language processing system and to transfer the results of psycholinguistic computations from one part of the perisylvian association cortex to another. The simplest model consistent with available data is that language processing is carried out only in the perisylvian association cortex. However, nature does not always do things in the simplest possible way and this conclusion may be modified as more information accumulates.

A very important and well-attested feature of neural organization for language is lateralization – the fact that language processing relies upon one hemisphere more than another in most normal individuals. In about 98 per cent of strong right-handers from right-handed families, the left perisylvian association cortex accomplishes most, if not all, language processing functions (Luria 1970, Milner et al. 1964, Milner 1974). In individuals with other handedness profiles (Geschwind and Galaburda 1987, Geschwind and Galaburda 1985, Annett 1985), language functions are far more likely to involve the corresponding regions of the right hemisphere (Luria 1970, Russell and Esper 1961, Goodglass and Quadfasel 1954), with different likelihoods of right and left hemispheric involvement in language functions in different subgroups within

this population (Subirana 1964). The data on differential lateralization as a function of sex are controversial (McGlone 1980).

A potentially important point is that many aphasic syndromes that follow either left or right hemisphere lesions in subjects who are not right-handed are often mild. Their occurrence suggests that many individual language processing components can be located in either hemisphere. Whether these language processing components are located in a given hemisphere in isolation from others can only be resolved by studies that establish whether the remaining intact language components are based in the intact portions of the lesioned hemisphere or in the undamaged hemisphere of patients with mild aphasias. In some cases (Kinsbourne 1971), intracarotid amygdala injections (Wada studies) indicate that the latter appears to be the case. This would suggest separate control of lateralization for individual language processing components, but very few data are available on this point.

Though not as important in language functioning as the dominant hemisphere, the nondominant hemisphere is involved in many language operations. Evidence from the effects of lesions and split brain studies, as well as experiments using presentation of stimuli to one or the other hemisphere in normal subjects, indicate that the nondominant hemisphere understands many words, especially concrete nouns (Gazzaniga 1983, Chiarello et al. 1990), and suggest that it is involved in other aspects of language processing as well. For instance, our studies have shown effects of right hemisphere stroke upon syntactic processing, although these are much more mild than those found after left hemisphere strokes (Caplan et al. 1996), and this result has its counterpart in at least one study that showed minor but reliable increases in blood flow in the right hemisphere that were associated with syntactic processing (Just et al. 1996). Some language operations may be carried out primarily in the right hemisphere. The best candidates for these operations are ones that pertain to processing the discourse level of language, interpreting non-literal language such as metaphor, and appreciating the tone of a discourse as is manifest in, for instance, humor (Brownell and Gardner 1988, 1989, Joannette and Brownell 1990, Molloy et al. 1990, Roman et al. 1987). Some scientists have developed models of the sorts of processing that the right hemisphere carries out. For instance, Beeman (in press) has suggested that the right hemisphere codes information in a coarse way compared to the left. This and other suggestions provide the bases for ongoing research programs into the nature of language processing in the right hemisphere.

Overall, the gross organization of the brain for language is such that language processing takes place in a relatively small region of the cortex – the association cortex surrounding the sylvian fissure – with supporting connections to grey matter elsewhere in the brain. Much remains to be learned about the role that these subcortical grey matter structures play in actual psycholinguistic computations. There is specialization for language in one hemisphere, the details of which differ as a function of handedness. Much also remains to be learned about the details of what is lateralized and in whom. But at least

the gross functional neuroanatomical facts are emerging. In comparison, the internal organization of the perisylvian cortex for language remains shrouded in mystery and steeped in controversy. To this, we now turn.

2.2 The organization of the perisylvian association cortex for language

Two general classes of theories of the relationship of portions of the perisylvian association cortex to components of the language processing system have been developed, one that maintains a distributed view of neural function (Jackson 1878, Freud 1891, Marie 1906, Head 1926, Mohr et al. 1978) and one that maintains a localizationist perspective (Luria 1970, Broca 1861, Wernicke 1974, Dejerine 1892, Lichtheim 1885, Henschen 1920, Neilson 1936, Geschwind 1965, Damasio and Damasio 1980, Luria 1973). Though theories within each of these two major groupings vary, there are a number of features common to theories within each class.

The basic tenet of distributed theories of the functional neuroanatomy for language is that linguistic representations and specific stages of linguistic processing are distributed widely across the perisylvian association cortex. Lashley (Lashley 1929, Lashley 1950) identified two functional features of distributed models that determine the effects of lesions upon performance – equipotentiality (every portion of a particular brain region can carry out a specific function in every individual) and mass action (the larger the neuronal pool that carries out a particular function, the more efficiently that function is accomplished). The features of equipotentiality and mass action jointly entail that lesions of similar sizes anywhere in a specified brain region have equivalent effects upon function, and that the magnitude of any functional deficit is directly proportional to the size of a lesion in this specified area. Computational models of lesions in “neural net” or “parallel distributed processing” (PDP) simulations of language and other cognitive functions have provided a mathematical basis for these properties of these systems (McClelland and Rumelhart 1986). Distributed models might also predict that activation studies in normal subjects would not find evidence for localized increases in blood flow or electrophysiological activity associated with particular psycholinguistic processes, although this prediction must be qualified by the consideration that some of these models suggest specializations within widely distributed neural nets, which may lead to such local increases in neural activity.

All of the traditional theories that postulate localization of components of the language processing system maintain the view that, discounting lateralization, the localization of components of the language processing system is invariant across the normal adult population. This is expected to result in localized increases in blood flow or electrophysiological activity associated with particular psycholinguistic processes, as well as to lesions in particular areas of the

perisylvian association cortex interrupting the same language processing components in all individuals. Many localizationist theories also maintain that the specific localization of language processing components results from a computational advantage inherent in juxtaposing particular language processing components to each other or to cortex supporting arousal, sensory, and motor processes (Luria 1970, Geschwind 1965, Luria 1973).

Because of the large number of specific theories within each of these two general camps, it is impossible to critically review the empirical basis of all theories that have present-day adherents (for a partial review, see Caplan 1987). I shall focus on the most widely cited theories within each class, and then turn to what is known about the localization of the two functions considered above – semantic memory and syntactic comprehension.

2.3 *Distributed theories*

Several lines of inquiry provide evidence for distributed theories, and all distributed theories suffer from similar inadequacies in accounting for certain empirical findings.

The first line supporting distributed theories consists of the ubiquity of general factors in accounting for the performance of aphasic patients. For instance, a statistical analytic technique known as factor analyses has been applied to analyze the performances of groups of patients both on general aphasia tests and on tests of specific language abilities. These analyses have almost always resulted in first factors (usually accounting for more than half of the variance in performance) that are roughly equally weighted for most of the subtests used to test the population (for general aphasia batteries, see Goodglass and Kaplan 1982, and Schuell 1957; for examples within a specific domain, see Caplan et al. 1985, 1996). Such factors are usually taken to reflect disruption of a single function that affects performance on all measures, such as a limited amount of mental resources available for psycholinguistic computations. The existence of such factors would be the immediate consequence of a system in which such functions were disrupted by lesions in a variety of locations, and they have therefore been widely taken as evidence for a distributed basis for language functions.

A second finding supporting distributed theories is the frequent observation of so-called “graceful degradation” of performance within specific language domains after brain damage. An example of such degradation is the strong tendency of certain dyslexic patients to read irregularly spelled words according to a regularization strategy (e.g., *pint* is read with a short *i*), a tendency which is inversely proportional to the frequency of the word (Bub et al. 1985). Graceful degradation reflects the preservation of the simplest (in many cases, the most commonly occurring) aspects of language processing after brain damage. Modern work with neural net models indicates that such patterns of performance can arise following focal lesions in systems in which information is

represented and processed in a distributed fashion (McClelland and Rumelhart 1986, Seidenberg and McClelland 1989).

A third source of empirical support for distributed theories comes from the finding of an effect of lesion size on the overall severity of functional impairments in several language spheres (Knopman et al. 1984, Knopman et al. 1983, Selnes et al. 1983, Selnes et al. 1984). This would follow from the principle of mass action (Lashley 1929). These results therefore are consistent with some form of distribution in the neural basis for linguistic representations and processes.

Against the complete adequacy of any distributed model is the finding that multiple individual language deficits arise in patients with small perisylvian lesions, often in complementary functional spheres. For instance, as noted above, in our studies of syntactic comprehension, we have documented patients who have trouble finding the referent of a reflexive form (e.g., *himself*) but perform normally on pronouns (e.g., *him*), and vice versa (Caplan and Hildebrandt 1988). The existence of these isolated complementary deficits in different single cases indicates that at least one abnormal performance cannot result from the relative complexity of processing required by one of these tasks. Double dissociations of this sort are common in the contemporary psycholinguistic aphasiological literature (see Shallice 1988). They indicate that the mode of organization of language in the brain must be one that allows focal lesions to disrupt specific aspects of psycholinguistic processing, not simply a mode of organization that produces complexity effects and degrades gracefully. Though some selective disruptions of function can occur when "lesions" are produced in simulated language processing systems that operate in parallel and distributed fashion (Wood 1982, Gordon 1982, Patterson et al. 1989), to date no mechanism of lesioning a distributed neural system has been shown to produce the range of specific patterns of language breakdown observed in patients.

Another major problem for distributed models is the finding that regional cerebral blood flow increases in specific parts of the perisylvian cortex when subjects do different types of language tasks (see discussion below). As noted above, this suggests at least some degree of cerebral specialization within any distributed system.

2.4 *Classical localizationist theories*

Though many localizationist models exist, the "connectionist" model of language representation and processing in the brain revived by Geschwind and his colleagues (Geschwind 1965) in the 1960s and 1970s probably remains the best-known localizationist model of the functional neuroanatomy of language, at least in medical circles in North America. This model is based upon the aphasic syndrome that we reviewed above, that was described over a century ago (Broca 1861, Wernicke 1974, Dejerine 1892, Lichtheim 1885).

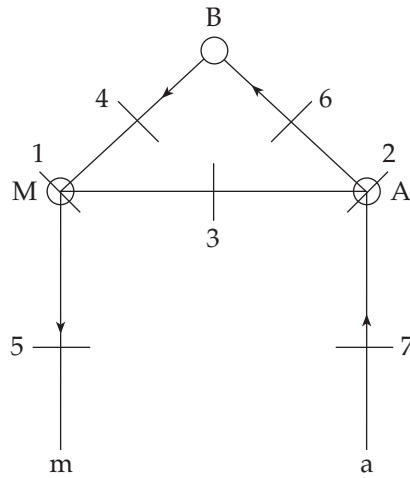


Figure 24.2 The classical connectionist model

Note: A represents the auditory center for word processing. M represents the motor center for speech planning. B represents the concept center. Information flow is indicated by arrows. Numbers indicate the location of lesions said to produce the classical clinical aphasic syndrome. For discussion, see text.

Source: From Moutier 1908, Lichtheim 1885

Figure 24.2 represents the basic “connectionist” model of auditory-oral language processing and its relation to areas within the dominant perisylvian cortex. This model postulates three basic “centers” for language processing, all in cerebral cortex. The first (A), located in Wernicke’s area, stores the permanent representations for the sounds of words (what psycholinguists would now call a “phonological lexicon”). The second (M), located in Broca’s area, houses the mechanisms responsible for planning and programming speech. The third (B), diffusely localized in cortex in the nineteenth-century models, stores the representations of concepts. A major innovation proposed by Geschwind is in the location of one aspect of the concept center (Geschwind 1970). Geschwind proposed that the inferior parietal lobule – the supra-marginal and angular gyri – are the location at which the fibers projecting from somesthetic, visual and auditory association cortices all converge, and that as a consequence of this convergence, associations between word sounds and the sensory properties of objects can be established in this area. Geschwind argued that these associations are critical aspects of the meanings of words and that their establishment is a prerequisite of the ability to name objects.

According to this model, language processing involves the activation of linguistic representations in these cortical centers and the transfer of these representations from one center to another, largely via white matter tracts. For instance, in auditory comprehension, the representations of the sound patterns of words are accessed in Wernicke’s following auditory presentation of

language stimuli. These auditory representations of the sounds of words in turn evoke the concepts associated with words in the "concept center." Accessing the phonological representation of words and the subsequent concepts associated with these representations constitutes the function of comprehension of auditory language. In spoken language production, concepts access the phonological representations of words in Wernicke's area, which are then transmitted to the motor programming areas for speech in Broca's area. In most versions of this model, the proper execution of the speech act also depends upon Broca's area receiving input directly from the concept center. Repetition, reading, and writing are modeled as involving similar sequences of activation of centers via connections.

The principal evidence in favor of this model is said to be the occurrence of specific syndromes of language disorders that can be accounted for by lesions of these centers and the connections between them, shown in table 24.1 above. A lesion in Broca's area said to be associated with Broca's aphasia; a lesion of Wernicke's area with Wernicke's aphasia; a lesion of the input pathway to Wernicke's area with pure word deafness; a lesion in the outflow pathway from Broca's area with dysarthria, apraxia of speech or another form of a motor speech disorder; a lesion between the concept center and Broca's area with transcortical motor aphasia; a lesion of the pathway between the concept center and Wernicke's area with transcortical sensory aphasia; and a lesion of the pathway connecting Wernicke's and Broca's area with conduction aphasia. All of these syndromes were claimed to have been discovered in relatively pure form by Lichtheim (Lichtheim 1885), and to have resulted from lesions in the appropriate cortical and subcortical areas of the brain. Recent studies using modern imaging technology have provided additional evidence of the correlation of these lesion sites with these aphasic syndromes (Basso et al. 1985, Damasio and Damasio 1980, Kertesz et al. 1982, Kertesz 1979, Naeser and Hayward 1978, Kertesz et al. 1979, Barat et al. 1978, Hayward et al. 1977, Mazzocchi and Vignolo 1980, Naeser and Hayward 1979, Naeser et al. 1981, Noel et al. 1977, Yarnell et al. 1976, Benson and Patten 1967).

Despite these data, there are many inadequacies in the database that supports this theory of the functional neuroanatomy of language. On the neurological side, problems exist with several aspects of the published data. Lesions have often been described in very general terms (Basso et al. 1985, Naeser and Hayward 1978). The techniques used to localize lesions are often imprecise and have at times been inconsistently applied to imaging data (Kertesz 1982). Ignoring these technical problems, the correlations between lesion sites and aphasic syndromes are far from perfect even in patients with stroke, and they become less reliable in other neurological diseases (Kertesz 1979). As early as 1908, François Moutier documented large numbers of stroke patients with lesions in Broca's area without Broca's aphasia and patients with the syndrome with lesions elsewhere (Moutier 1908, Lecours and Joanette 1984). Recent work has confirmed the failure of small Broca's area lesions to produce Broca's aphasia (Mohr et al. 1978), leading to various modifications of the simplest

version of the connectionist localizationist model (Levine and Sweet 1982). The variability in lesions associated with Wernicke's aphasia has been well documented (Bogen and Bogen 1976).

From the psycholinguistic point of view, the principal problem with this correlational evidence for the classical localizationist theory is its dependence on the interpretation of the clinical aphasic syndromes. As we have seen, these syndromes reflect the relative ability of patients to perform language tasks (speaking, comprehension, etc.), not the integrity of specific components of the language processing system, and therefore they cross-classify patients with respect to their underlying functional deficits in language processing components and reflect disturbances of different language processing components in different patients (Schwartz 1984). Accordingly, knowing that a particular aphasic syndrome tends to be associated with a lesion in a particular part of the perisylvian cortex does not tell us what components of the language processing system are located in that area of cortex and does not guarantee that these same components are not also damaged by lesions in different cortical areas (that are associated with different syndromes).

Both lesion-deficit correlations in patients with more specific functional impairments and activation studies in normal subjects using functional neuroimaging and electrophysiological approaches have begun to provide data relevant to the localization of specific components of the language processing system within the perisylvian association cortex. I shall illustrate this work with a short review of some of studies in the areas of lexical semantics and syntactic comprehension.

2.5 *Localization of semantic memory*

Semantic memory is not easy to localize, since it consists of knowledge that we have about items, and such knowledge has many components. For all their localizing zeal, the nineteenth-century founders of aphasiology and neurolinguistics had no idea where the meanings of words were located in the brain. Broca (1861) did not deal with this issue at all and Wernicke (1974 [1872]) barely mentioned it; Lichtheim (1885), who thought he had codified all the aphasias, said of "the concept center" that it was located in an area of the brain above the temporal-to-frontal lobe system that was responsible for word recognition and word sound production. Some modern researchers have adopted theories that also deny a localization to semantic memory. For instance, Damasio (1989), like Geschwind (1965), has argued that the meanings of words consist of the characteristic features associated with the corresponding items – shape, color, texture, odor, taste, actions performed on the object, etc. Unlike Geschwind, who thought of the inferior parietal lobe as the place where these associations came together to produce meaning, Damasio maintains that the neural substrate for meaning consists of "retroactivation" of sensory and motor cortices in which these properties were encoded by sensory and motor

experience. In Damasio's model, meaning changes as a function of context, so that the meaning of the word (or concept) "scissors" involves activation of stored motor representations to a greater degree if the word (or concept) is being activated in connection with the actual act of cutting than if the word is mentioned because someone is telling someone else that a piece of paper is lying under a pair of scissors. In this model, the location of meaning changes as a function of the mindset of the speaker or listener.

As opposed to these theories, which either ignore the location of meaning or explicitly deny that the meaning of a word or a concept can be localized for principled reasons, there are other researchers who have advanced hypotheses about the localization of the cortex involved in representing the meaning of words and concepts. For instance, as we have seen, Geschwind (1965) articulated a specific hypothesis regarding the location of word meanings within the inferior parietal lobe.

What does the evidence suggest? The best data from deficit-lesion correlational studies usually comes from patients with no neurological history who have a first stroke that is relatively small. These patients have normal brains up to the moment of injury, and the injury is circumscribed and maximal at its onset. The deficits that follow these strokes reflect disruptions of the functions that are supported by the area of the brain that is affected by the stroke. However, strokes do not usually produce isolated semantic impairments. It is often the case that patients with strokes cannot name objects or understand words, but the deficits seen in most of these cases usually involve disruptions to the mechanisms that activate word sounds and recognize words from the acoustic waveform or print. Patients with Wernicke's aphasia are typical in this respect – these patients almost certainly have semantic deficits, but their other problems make it hard to document these deficits clearly. Nonetheless, there are a few stroke patients in whom the deficits appear to affect meaning in isolation, and these few cases have by and large had lesions whose centers lie in the left temporal lobe (Warrington 1987). A temporal lobe location for the representations of the meanings of words is also suggested by the study of patients with herpes encephalitis, some of whom have been shown to have disorders of semantics without equally severe disruptions of the processes underlying word sound activation and word recognition (Warrington 1981b). In these cases, the brunt of the illness has been shown to affect the temporal lobes.

An important caveat about most of these cases relates to a feature of aphasia we reviewed above – the distinction between deficits that are seen in conscious, controlled tasks, and those that appear in tasks that do not require conscious controlled processing. As we noted above, Milberg, Blumstein and their colleagues have reported that some patients with Wernicke's aphasia show evidence of automatic, unconscious activation of meaning. Conversely, some patients with Broca's aphasia show some evidence of abnormalities in these automatic processes relating to manipulation of semantic information. However, there is too much variability in the performances of these patients and too little detailed study of their lesions to be able to draw any firm conclusions

about the localization of deficits in either conscious or unconscious semantic processing from these studies.

Degenerative disease often presents a less clear picture than stroke because it is often diffuse. But this is not always the case. Diseases such as Parkinson's disease and Huntington's disease, which affect specific parts of the basal ganglia, have been very informative about the neural basis for motor control and, more recently, procedural memory. Recently, several researchers (Hodges et al. 1992) have described a degenerative disease that selectively affects semantic memory. The patients with this disease lose knowledge of the meanings of words but retain the ability to repeat and to recognize words. MR, SPECT, and PET studies of these patients have predominantly shown a left temporal lobe locus of atrophy and hypometabolism, though other parts of the brain – notably the left frontal lobe – have been implicated in some studies in some of these patients. It is unclear what the etiology of this degenerative condition is; Alzheimer's or Pick's disease are the leading possibilities (see Hodges et al. 1992, for discussion).

While the evidence from the effects of brain damage suggests a temporal lobe location for semantic knowledge about words, a group of researchers using functional neuroimaging have come to a different conclusion. Posner et al. (1988) reported a now-famous study in which they compared PET activity associated with subjects' generating a verb that conveyed the use to which a noun is put (e.g., "sweep" for "broom") with PET activity associated with subjects' repeating the noun. There was an increase in PET activity, representing increased regional cerebral blood flow (rCBF), in the left dorsolateral frontal cortex, just in front of Broca's area, when the repeat task was subtracted from the generate task. This result is very robust and has been replicated many times. The authors interpreted it as reflecting semantic processing associated with accessing the noun's meaning and generating an action associated with that meaning. Posner et al. (1987) reported a similar increase in rCBF in the same region in a task in which subjects listened to a list of animals and indicated which were dangerous (a semantic task) compared to their simply passively listening to the same list and making no response. These authors also found that, in the "ferocious animal detection task," there was no effect of the number of positive examples (fierce animals) in the list upon the presence of the rCBF increase in the left frontal region, suggesting that the increase in blood flow is a result of subjects' monitoring the meaning of each word, not confirming that an animal is fierce.

There are other interpretations of these PET and fMRI effects, however. One, which I favor, is that this increase in blood flow may represent subjects' switching from one category to another. Lesions in the dorsolateral region are well known to disturb the ability to switch one's mental set (Luria 1973). An argument against the view that the activation seen in Posner's studies is due to semantic processing is that patients with lesions in the dorsolateral frontal cortex do not have semantic deficits when tested on standard tests of word comprehension. Posner and his colleagues point to the fact that patients with

lesions in this region have trouble producing lists of animals, which they consider a semantic task. These authors also point to the Milberg and Blumstein studies that document abnormal semantic priming in some Broca's aphasics as evidence for a role of left frontal cortex in semantic processing. But neither of these arguments is convincing. Deficits in semantic verbal fluency can be the result of many cognitive problems, including switching from one subset within a category to another. Normal subjects typically generate animals in subsets in this task (domestic animals, farm animals, fierce wild animals, reptiles, etc.). Difficulty switching from one of these subcategories to another may result in trouble generating lists of items within a broader category. As we have seen, the data on both semantic priming and lesion location in Broca's aphasia are too fragmentary and inconsistent to be interpreted as clearly showing that there is a semantic deficit in Broca's aphasia; moreover, the lesions in Broca's aphasia more often extend posteriorly from Broca's area, not anteriorly into the dorsolateral frontal cortex where rCBF increased in the Posner et al. (1988) studies.

There are also activation studies that do not find a frontal increase in rCBF associated with semantic processing. Murtha et al. (in press) reported the results of a task in which subjects had to say whether the animal designated by a presented word had hoofs, horns, or antlers – a task that requires visualization of features of the animal that are thought to be part of its semantic representation. When compared to passive listening to the same words, these authors found a left frontal increase in rCBF. However, the authors also used an interesting paradigm in which they subtracted out a dummy scan in which blood flow was measured while the subjects waited to begin the task. This was intended to subtract out the effects of anticipation and mental set associated with the semantic but not the baseline task. With this additional subtraction, the frontal activity disappeared and rCBF increased from the baseline to the experimental condition only in the left temporal lobe – exactly where the studies by Hodges and others suggest it should.

In summary, what evidence there is suggests that there are regions of the brain that increase their blood flow as a function of semantic processing and in which lesions disturb such processing. These regions do not seem to include the inferior parietal lobe, as Geschwind claimed, nor to depend upon the context within which an object is mentioned for a deficit to be observed, as Damasio's theory would suggest. The best candidate for the location of semantic memory for objects and concrete nouns is the cortex of the left temporal lobe, with the left dorsolateral frontal cortex also being in the running. But there is much more uncertainty than hard knowledge about this localization.

2.6 Localization of syntactic comprehension

The first studies of the neural basis for syntactic comprehension were deficit-lesion correlations. Caramazza and Zurif (1976), Heilman and Scholes (1976),

and other researchers described disorders of syntactic comprehension in several groups of aphasic patients, including Broca's aphasics, "mixed anterior aphasics," and conduction aphasics. The theoretical emphasis in this literature was on the co-occurrence of an impairment in syntactic processing in comprehension with agrammatic speech in agrammatic Broca's aphasics. This co-occurrence led several researchers to suggest that the area that is usually affected in Broca's aphasia – Broca's area: pars triangularis and opercularis of the left third frontal convolution, Brodmann's areas 44 and 45 – play a special role in syntactic processing (Zurif 1982).

This area of research and theory development has been pursued along two lines: (1) more specific characterization of the syntactic structures that are affected in Broca's aphasics (Grodzinsky 1990, 1995, Hickok and Avrutin 1995, Hagiwara 1995), and (2) description of certain aspects of online syntactic processing in Broca's aphasics (Zurif et al. 1993, Swinney and Zurif 1995).

Grodzinsky, Hickok, Avrutin and others have argued that Broca's aphasics have selective disturbances affecting a restricted set of syntactic operations that can be characterized in terms provided by Chomsky's (1986, 1995) theory of syntactic structure. In this framework, the disturbances these patients are said to have affect the ability to "co-index traces." Traces are one of several sets of items that, according to this theory, are understood but physically unexpressed. The best examples of traces are found in sentences like *Who do you like [t]?*, and *John saw the man who the police were searching for [t]*. In both these sentences, the "wh-word" (*who*) is understood to be the object of the verb (*like*, *search for*). In Chomsky's theory, there is a trace (*t*) that is mentally but not physically present in these sentences after the verb, and the *wh*-word is co-indexed with it. Grodzinsky and his colleagues have documented disturbances on the part of Broca's aphasics in understanding sentences such as *Who do you like?* and *John saw the man who the police were searching for*, which they attribute to impaired abilities to accomplish the necessary co-indexation. (The exact characterization of the deficit varies somewhat in different reports, as does the description of normal syntactic structure, but this is the essence of these analyses.) Grodzinsky and his colleagues have argued that the brain region that is affected when these operations are impaired – Broca's area – is responsible for this aspect of syntactic processing in normals.

Zurif, Swinney and their colleagues investigated the online processing of these structures in Broca's and Wernicke's aphasics. They reported that Wernicke's aphasics – but not Broca's – showed priming to semantic associates of the antecedents of traces (*the man* in *John saw the man who the police were searching for*) at the point at which the trace occurred. They argued that this indicated that Broca's aphasics had a deficit in online processing of traces. This analysis is closely related to those of Grodzinsky, Hickok, and Avrutin in that it describes in processing terms a deficit that the previously cited authors describe in structural linguistic terms.

Some electrophysiological and metabolic data are consistent with this model. Researchers have identified several event related potentials (ERPs) that may

reflect syntactic processing. The two leading candidates are the P600 or “syntactic positive shift (SPS)” in the central parietal region (Hagoort et al. 1993, McKinnon and Osterhout 1996, Neville et al. 1991, Osterhout and Holcomb 1992, 1993) and the “left anterior negativity (LAN)” that arises roughly over Broca’s area (Kluender and Kutas 1993a, 1993b, Munte et al. 1993, Rosler et al. 1993). The more posterior wave (the P600, or SPS) appears to be elicited at the point at which violations of syntactic structure can be identified (McKinnon and Osterhout 1996, Osterhout and Holcomb 1992, 1993). The LAN appears to arise when subjects process sentences with traces that have moved over an intervening noun phrase (Kluender and Kutas 1993a, 1993b). The existence of this wave is broadly consistent with the localizationist model outlined above.

Recent work with PET has also provided evidence for localization of this same aspect of syntactic processing to Broca’s area (Stromswold et al. 1996, Caplan et al. in press). In these studies, PET activity associated with making plausibility judgments about simpler object–subject sentences (e.g., *The award thrilled the actress that praised the producer*) was subtracted from that associated with making judgments about the same propositions phrased in syntactically more complex subject–object forms (e.g., *The actress that the award thrilled praised the producer*). The subtraction showed an increase in rCBF in the pars opercularis of Broca’s area. The difference between the more and less complex sentences resides in the complexity of the trace coindexation operation; thus these studies are also consistent with the specific localizationist model discussed above.

However, there are contradictory data. Patients with aphasic syndromes other than agrammatic Broca’s aphasia, whose lesions lie largely outside this region, often show impairments of syntactically based sentence comprehension that are indistinguishable from those seen in Broca’s aphasics (Berndt et al. 1996, Caplan 1987, Caplan and Hildebrandt 1988, Caplan et al. 1985, 1996, 1997, Dronkers et al. 1994, Tramo et al. 1988). Conversely, patients with agrammatic Broca’s aphasia often show good syntactic comprehension on sentence-picture matching tests (Berndt et al. 1996). Studies that have looked at lesion sites – rather than aphasic syndromes – have also found no relation between the presence of a lesion in particular parts of the perisylvian cortex and the presence of a disorder of syntactically based sentence comprehension (Caplan 1987, Caplan et al. 1985, 1996). In addition, the neuroimaging data that have been reported in these studies largely consist of CT, and some MR, images – often analyzed by quite subjective techniques – and the studies of patients with degenerative disease cited above (e.g. Hodges et al. 1992) show that areas of hypometabolism are important to consider when looking for the relationship of deficits with lesions.

Nor is the picture derived from functional neuroimaging studies perfect from the localizationist point of view. Using fMRI, Just et al. (1996) reported an increase in rCBF in both Broca’s area and in Wernicke’s area of the left hemisphere, and smaller but reliable increases in rCBF in the homologous regions of the right hemisphere, when subjects read and answered questions about sentences that were very similar to those used by Stromswold et al. (1996) and

Caplan et al. (1998). We have repeated our PET study with elderly subjects (between the ages of 70 and 80). We found a very specific increase in rCBF – but in the inferior parietal lobe, not Broca’s area. Since the age of the patients in the studies by Swinney, Zurif, and their colleagues is much closer to that of our elderly than our young normal group, this finding makes the localization of this aspect of syntactic processing in Broca’s area less secure. And we must remember that what is being proposed to be localized in Broca’s area is only one particular syntactic operation related to an aspect of the structure and meaning of relative clauses. Detailed online studies of deficits in other operations and neuroimaging studies in normal subjects that highlight such operations are lacking.

3 Conclusion

Contemporary studies of language disorders and the relationship of language to the brain are beginning to deal with the hard problems: how do we represent linguistic information? what are the component processes that activate this information in the acts of speaking, listening, reading, and writing? What is the nature of disorders of these representations and processes? Where are these representations and processes localized in the brain? Research into these questions is now at a point where we can propose hypotheses and support (and attack) them empirically. This is a real advance over the level of description and modeling of language disorders and the neural basis for language of the late 1970s. I have outlined the types of studies that characterize this field, within a historical perspective, and illustrated some of the results of these studies. The reader will appreciate that these are new questions, being approached with new psycholinguistic paradigms and new technologies to describe the brain, and there are no sure answers at present to many of these basic questions. Nonetheless, however confusing parts of this field may at times appear to be, this research is clearly in the direction of a more detailed and accurate understanding of language disorders and the neural mechanisms that support normal language knowledge and use.

NOTE

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