Language (Dr. Merchut)

1. The distinction between speech and language

Although the terms "speech" and "language" are often considered synonyms by lay people, they are truly distinct from the neurological viewpoint. Speech or speaking is the production of audible sounds which may or may not be used to communicate. **Speech basically consists of phonation and articulation. Phonation** is sound production by the moving vocal cords, the muscles of which are innervated by branches of the vagus nerve, which is controlled by its upper motor neurons. Abnormal phonation or dysphonia often sounds hoarse, whispering, or breathy. **Articulation** is sound production by actions and varied positions of the lips, tongue, palate, and pharynx, which are innervated by cranial nerves VII, IX, X, and XII, and controlled by their associated upper motor neurons. Connections with the cerebellar and extrapyramidal systems also provide the motor coordination needed for precise, clearly understood enunciation. Abnormal articulation or dysarthria often sounds slurred, choppy and indistinct. Both phonation and articulation depend on moving air from the lungs and thus a normally functioning respiratory system.

Language, neurologically speaking, **is communication by means of symbols**, and is not limited to production of audible sounds. Reading and writing are other means of communication, as are gestures, pictures, signing, Braille, and Morse code. Comprehension and expression or execution of language are functions of specific areas of cerebral cortex in the dominant hemisphere. The left cerebral hemisphere is dominant in almost all right-handed people and about half of all left-handed people. **The major language centers are in the vascular territory of the middle cerebral artery.** Abnormal language or **aphasia** may thus occur from an ischemic infarction, but may also be due to hemorrhage, tumor, trauma, or dementia. **Aphasia is** therefore **a disorder of previously acquired language ability due to a lesion in a critical language center.** A newborn infant born with a severely maldeveloped brain is not considered "aphasic" in the purest sense, since it will never learn to communicate. Although some aphasic patients may speak and produce sounds, certain aspects of their ability to communicate are impaired, and these same aspects are present to some degree in whichever method they communicate, such as reading and writing. Other patients may lose the ability to speak, as after extensive surgical removal of laryngeal cancer, but communicate normally by reading, writing, or other means, and thus have normal language function. A deaf person may be mistakenly considered unable to understand at first, but may exhibit normal comprehension by signing, reading and writing.

2. Clinical examination of language

Language function should be tested in several ways to ensure that deaf or visually impaired patients or those unable to speak are not erroneously considered to be aphasic. Usually the patient is first engaged in spontaneous conversation. More directed testing would include naming items, repeating phrases, following spoken and written commands, reading passages, and writing to dictation. Speech therapists use standardized tests of language that can be graded or scored.

Fluency refers to the ease, facility, and quantity of speech regardless of content or meaning. **Comprehension** should be evident when verbal or written commands are followed. **Repetition** is intact only if a phrase from the examiner is **perfectly repeated** by the patient. One of the most time-honored test phrases to repeat is: "no ifs, ands, or buts." **Imperfect repetition occurs with a lesion in either perisylvian language center (Broca's or Wernicke's area) or the connecting arcuate fasciculus.** An aphasic patient may utter a **paraphasia**, which is an abnormal word or syllable substitution. **Paraphasias are more common** with lesions in the posterior perisylvian language region, particularly **with Wernicke's aphasia**. The substitution errors may involve phonemes or syllables ("Open the boor" instead of "Open the door") or full words ("The grass is blue" instead of "The grass is green"). The most extreme type of paraphasia is a **neologism**, which sounds like a nonsensical or foreign-sounding word or phrase ("That is a blastorale" instead of "That is a ham sandwich").

3. Types of aphasia

Different types of aphasia are associated with lesions of specific language centers. Since several aspects of language may be affected, the old simplified terms of "expressive or motor aphasia" and "receptive or sensory aphasia" proved inaccurate, and Broca's and Wernicke's aphasias are best named after the physicians who first described them (Fig. 1).

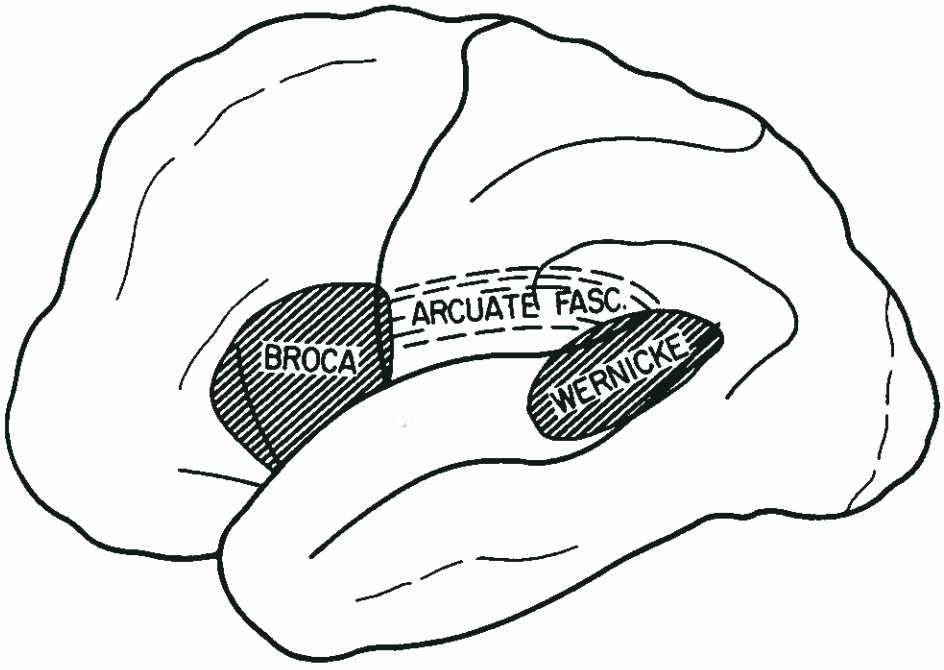

Fig. 1 Perisylvian language centers connected by the arcuate fasciculus (from Benson DF. Aphasia, alexia, and agraphia. New York: Churchill Livingstone, 1979)

Broca's aphasia is caused by a posterior inferior frontal lobe lesion in the dominant hemisphere. **Fluency is very impaired**, so speaking or reading aloud is very laborious and effortful. Few words are slowly spoken, often in telegraphic fashion. (In the old days of the telegram, wordier messages were costlier, so frugal customers sent very succinct, brief statements over the wire.) The patient may struggle to say over several minutes, "I . . . up . . . . early," intending to say, "I have to get up early tomorrow morning." **Comprehension is relatively preserved** and may be further evidenced by overt frustration in a patient aware of his language deficit. **Repetition is imperfect.** Attempts to read or write show these same difficulties. There often is an accompanying right hemiparesis since the lesion may also involve the primary motor cortex, which is just posterior to Broca's area.

Wernicke's aphasia is caused by a posterior superior temporal lobe lesion in the dominant hemisphere. **Fluency is preserved** such that the patient often speaks long phrases which make little sense, containing paraphasic errors and gibberish. The patient may say, "If you saw it, redness would schlodder over the galls anyway. I don't think it would much matter tweetch since fries are never made by keeters." **Comprehension is very impaired** and may instill paranoia in the patient who is unable to communicate with those around him. **Repetition is imperfect.** Reading and writing should likewise display these same difficulties, although the patient may not understand a request to read or write. In the absence of hemiparesis or other common stroke deficit, the diagnosis of Wernicke's aphasia may be difficult to make and the patient is often considered drugged or intoxicated or beset with a psychiatric illness.

Conduction aphasia occurs with a lesion of the arcuate fasciculus, the pathway connecting Broca's and Wernicke's areas. This aphasia has characteristics in-between those of Broca's and Wernicke's (Table 1), although more closely resembles the features of Wernicke's. Fluency is relatively intact with some milder impairment of comprehension but repetition remains imperfect. Some paraphasias are present.

Global aphasia occurs with an extensive lesion that virtually damages the entire perisylvian language region. The patient may appear awake but is unable to speak or communicate otherwise, follows no commands or gestures, and usually has a severe hemiplegia. Other types of aphasia may occur with lesions in the thalamus or in cortical areas just outside the perisylvian language centers. Lesions in posterior parts of the dominant hemisphere may affect specific aspects of language such as alexia or agraphia. **Alexia** refers to the impairment of reading when visual cortex is disconnected from language centers critical for reading. **Agraphia** occurs when a lesion disconnects the motor cortex for the dominant hand from language centers critical for writing.

Aphasia type	Fluency	Repetition	Comprehension
Broca's	Nonfluent	Imperfect	Good
Wernicke's	Fluent	Imperfect	Poor
Conduction	Fluent	Imperfect	Intermediate
Global	Nonfluent	Imperfect	Poor

Table 1 Clinical Features of Aphasia

4. Language and the nondominant cerebral hemisphere

The analogous "mirror images" of language centers in the nondominant hemisphere are concerned with a more subtle aspect of spoken language called prosody. **Prosody** refers to the semantic and emotional meaning of language as conveyed by changes in vocal pitch, inflection, melody or tone. For example, the rising pitch or stress at the end of a spoken question ("You gave this to me?") distinguishes it from a spoken statement ("You gave this to me."). More complex changes in the prosody of spoken words can convey the affect or mood of fear, anger, sadness, joy, or surprise. Nondominant cortical lesions may create **aprosodia**, where these features are impaired.

A lesion in the nondominant posterior superior temporal lobe, opposite to Wernicke's area, produces a sensory ("receptive") aprosodia. Here the patient has trouble understanding the emotional content in the words spoken by others, and cannot repeat or mimic the elements of prosody which he or she hears. When that patient speaks, however, normal prosody and gesturing is present. A lesion in the nondominant posterior inferior frontal lobe, opposite to Broca's area, produces a motor ("expressive") aprosodia. Here the patient may fail to incorporate elements of prosody when speaking and is unable to repeat or mimic the elements of prosody which he or she hears. However, he or she is able to understand the emotional content or mood in the speech of others.

5. The experience of Wernicke's aphasia

The experience of Wernicke's aphasia

Article abstract—The authors induced a transient Wernicke's aphasia in a patient with left frontal arteriovenous malformation by superselective Wada injection exclusively into the lower division of the left middle cerebral artery. The patient was then asked to recall his experience, which the authors matched against his language during anesthesia. The patient's account showed that there was a more systematic attempt to respond appropriately than the authors could infer from his overt behavior. His narrative suggests that a thought process not measured by aphasia examinations may exist independent of language.

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The clinical characteristics of Wernicke's aphasia include fluent speech with literal and verbal paraphasic errors, neologisms and perseverative spoken responses, impaired comprehension, impaired repetition with paraphasias, and errors reading aloud.[1] Occlusion of the lower division of the left middle cerebral artery (LMCA) produces this syndrome, with infarction encompassing the whole posterior temporal, inferior parietal, and lateral temporo-occipital regions.[2,3] Because of the difficulty in evaluating severe Wernicke's aphasia, the experience from the viewpoint of the patient is rarely studied with adequate lesion localization immediately before and after naturally occurring cerebral events.

We had the opportunity to query a patient with a left frontal arteriovenous malformation (AVM) about his perceptions of his language abnormality on a

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task-by-task basis shortly after we deliberately induced a transient Wernicke's aphasia following superselective injection of anesthetics into the lower division of the LMCA.[4]

Case report. A 47-year-old right-handed man presented with minor sensory seizures of the right arm and lip. MRI revealed a 2-cm AVM in the left middle and inferior frontal gyri shown on angiogram to be supplied by the LMCA.

During angiography the microcatheter was placed in the lower division of the LMCA (figure), and the patient underwent serial aphasia examinations, evaluating fluency, comprehension, naming, repetition, and oral reading. Tasks were adapted so that the entire examination would take about 3.5 minutes, which was the approximate duration of the peak anesthetic effect.[5] For testing with anesthetic, 50 to 75 mg of amobarbital sodium (USP) and 20 mg of lidocaine opacified with metrizamide were injected, followed less than 1 minute later by repeat language assessment. Twelve to 15 minutes after injection, when anesthetic effects were presumed to have fully dissipated, the language examination was readministered to ensure that functioning had returned to baseline levels. After the entire examination the patient was asked to write his recollections, which were contrasted with the examiner's recorded observations.

At baseline and 15 minutes after anesthetic injection, all aspects of language function were entirely normal. The patient recalled that his speech throughout anesthesia appeared to be fluent and intelligible.

> "In general my mind seemed to work except that words could not be found or had turned into other words. I also perceived throughout this procedure what a terrible disorder that would be if it were not reversible due to local anesthetics. There was never a doubt that I would be able to recall what was said or done; the problem was that often I could not do it."

The first task required him to rapidly provide the names of animals. He said in fluent, prosodic speech, "one, abril, abril, magardy, amigal, brazair, try to get in, animal, animal, try and reach." He had no recollection of this task after the procedure.

In the second and third tasks, the patient was asked to follow simple commands and to respond "yes" or "no" following dictated questions (e.g., "Will a cork sink in water?"). He made no responses to these items during anesthesia and he had no recollection of them afterward.

The fourth task required him to label orally pictures from the Boston Naming Test.[6] He recalls:

> "Moving on to the pictures, I will never forget the item #1, the tennis racket. I tried extremely hard to state the name of the picture. I knew what I wanted to say, but clearly could not find the word—a very frustrating situation. With my right hand somewhat freed, laying on my chest, I even tried to show I was 'batting the tennis ball' with the racket in an attempt to demonstrate what I meant and hoping that doing so would encourage the word to come to mind."

The patient's response was "perkbull."

The patient then added:

> "What happened next I will never forget . . . I told (the doctor) in my frustration that I had just bought a tennis racket. But this was not true! What I explicitly meant to say was that I owned a tennis racket . . . My statement was explicitly false, and I knew that I had said it. But I think that no less than 3 times I stated that I had just bought a racket, and kept repeating it as a way of self-correcting my previous incorrect statement. This was even more frustrating as now incorrect words came from me. Having spent a lot of time on this first item, I then elected to move onwards."

Figure. The angiogram following injection of amobarbital sodium and lidocaine, superimposed on its approximate location in the left cerebral hemisphere. The catheter tip is shown to be in the lower division of the middle cerebral artery just distal to the bifurcation.

From our observations, the patient never said anything aloud about a tennis racket. On the next four pictures, he called a volcano "chemicol," had no response to the picture of a dart, responded "trying" to a globe, and said "want to charge him" to the picture of a beaver.

Repetition was tested via the presentation of four dictated sentences. When presented with *The vat leaks*, he responded, "No shook," and then he made no response to each of the next three sentences. He recalls, "Repeating words and phrases proved to be a disaster. . . . From my perspective, it appeared to be an issue of apparent retention."

Last, the patient was given 23 words to read aloud, presented in three text lines on a page. He made correct responses to *milk*, *tree*, *theory*, *grieve*, and *conspiracy*. He made the following errors: "help" for *himself*, "be" for *between*, "chead" for *chin*, and "esculate" for *escape*. Afterward, he stated:

> "Reading words aloud along a series of lines was also startling. The first few words and others throughout the reading, but not all, appeared to be a totally random group of letters from the English alphabet. My recall is also that in some cases, there appeared to be even a few more letters than had existed before. Just as an example:
> Printed word (made up): recollection
> My perception: mbldoyeexstyz
> I tried my best to pronounce them but it was clear even to me at the time that I was speaking gibberish. That, I was very sure about. I also recall that I had similar problems as I had with the tennis racket, in that I would see a word, pronounce it, but another word would emit from my mouth—and I was very well aware of it. My attempts to self-correct for these obvious errors only led to further repetitions of the same incorrect word."

Discussion. By clinical criteria, our patient had a dense Wernicke's aphasia indistinguishable from that produced by infarction. His recollections following the aphasia, however, suggest that there was greater functional competence during the peak anes-

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thetic effect than could be discerned by his overt behavior.

Our patient, for example, was able to derive enough information from the environment that he could determine the nature of the task, attempt to execute it, and monitor internally a decision-making process, albeit ineffectively. His description indicates not only that he could think, but that he could recall afterwards what it was he was trying to do. It has been well-established that Wernicke's patients can respond appropriately to extralinguistic cues,[7] but the notion that individuals with such profound receptive deficits can analyze their own behavior contradicts traditional assumptions.[8]

We infer from his description and from performance on our tests that visual stimuli exerted more control than spoken language. His report about thinking in the presence of pictures, for instance, suggests semantic categorization had not been completely disrupted but that the phonetic properties of auditory stimuli failed to elicit corresponding representations.[9] The linkage between meaning and his spoken responses also appeared disturbed, both with regard to semantic and to phonetic properties. His description, therefore, suggests that there may not be a unitary underlying abnormality, but rather several independent aspects of Wernicke's aphasia. More important, perhaps, is that information obtained in the conventional aphasia examination does not reveal the complexity of residual function.

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