The Evaluation of Aphasia

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THIS article is based on the assumptions that the bedside physician takes a conversational clinical history from the patient; that he hopes to make a diagnosis of aphasia quickly and with the minimum of special test methods and materials; and that he is interested chiefly in those aspects of aphasia analysis which can help him predict the lesion site, size, clinical outcome, and cause of the aphasia.

The suggestions in this article are designed to emphasize clinicopathological correlation. The more traditional syndromes of aphasia are better described and assessed by methods discussed in some of the references at the end of this article.1-7

Before attempting to classify aphasia by a brief clinical examination, it is important to be aware of a few syndromes that interfere with this method of testing: abulia, medial frontal mutism, ideomotor apraxia, hemineglect, and dysnomia. Frontal polar lesions, depending on size, produce a spectrum of reduced rate of responding (abulia) and reduced apparent susceptibility to control by the examiner that ranges from a slight laconic tendency in monologue narrative, to delays varying from seconds to many minutes before brief replies are made, to the extremes of immobility and silence (akinetik mutism). These patients speak with normal clarity and language content in their sparse remarks; their reduction in all forms of motor activity parallels the speech, and they make none of the obvious effort at communication typical of most patients whose aphasia is due to left Sylvian disease.

Mutism also occurs acutely in some medial frontal lesions following aneurysm rupture, hematoma, or major anterior cerebral artery territory infarction. When the patient cooperates in testing (some have severe abulia), this location can be suspected if the functioning right limbs perform dictated commands properly (eupraxia) while the left limbs move in an amorphous, undirected fashion (ideomotor apraxia). Such a striking contrast is due to damage to the transhemispheral motor responses to verbal commands.

Thought to reflect an inability to relate an "idea" to a "motor" response, ideomotor apraxia remains poorly understood but is commonly encountered in lesions of varying size ranging all along the anterior half of the dominant hemisphere and in many lesions of the posterior half as well. Because of the frequency of faulty motor response to dictated or printed commands, the traditional commands that require a skilled motor response, such as "show me how to brush your teeth," are less useful as tests of language comprehension. They remain suitable as tests of comprehension and praxis only when the patient performs them correctly.

A similar problem to overcome in testing is hemineglect. A frontal lesion commonly causes hemineglect manifested as a failure to blink to threat in the contralateral visual field. Less well recognized is a similar failure to respond to the right-hand side of a language stimulus, such as a printed word or sentence, when there is a left hemisphere lesion. This neglect, which exists independently of any demonstrable hemianopia, can seriously interfere with the response to visual stimulus materials if the relevant parts of the stimulus are to the right. For example, "eight" is hard to read as anything else from left to right once the first two letters are noted, but "note" and "herd" each yield three different words depending on where the reading effort stops. Similarly, many multiword printed commands leave the main item to the end, e.g., "touch your . . ." and are better truncated to the main word only. The more severe the disturbance in language, the worse the right-sided verbal hemineglect. A deliberate use of easily misread words to test those who seem intact is a useful trick, but careful avoidance in the mild aphasics is necessary to prevent them from seeming worse than they are. Best of all, keep the materials constant on repeated examinations and note the actual materials used, not just the conclusion on judging the performance.

Finally, dysnomia may complicate the analysis of aphasia. Strictly speaking, this term refers to naming errors but does not distinguish between errors of mispronunciation or production of an incorrect word or silence because of failure to recall the name. Naming is the most difficult of language tests because it occurs under the duress of command, no help is provided, and the response is not simply constrained to one of a finite number of choices presented by the examiner. Little wonder that almost all aphasics have some form of "dysnomia," so much so that its separate description is of little localizing value. However, in lesions involving the undersurface or the depths of the temporal lobe, including tumors, abscess, encephalitis, meningitis, posterior cerebral artery occlusions, and Alzheimer dementia, a special form of dysnomia predominates as a failure to recall the names. This syndrome, also labeled amnestic aphasia, may exist separately from any other language disorder. It complicates the analysis of other aphasias, may occur as the first sign of a mass or the last residual of a more widespread distur-

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bance in language, and occurs to some degree in normals. When present in isolation, it is a sign of localizing value for the temporal lobe. Mixed with other speech and language disturbances, its localizing value declines.

With the major syndromes of aphasia, an effort to separate speech from language disturbances is clinically worth while while in bedside testing because the more circumscribed the lesion, the more the production or comprehension of speech sounds and shapes is disturbed. Likewise, the larger the lesion, the more the language content conveyed in the speech is affected.

In global aphasia the patient is mute, fails to perform to dictated or printed commands, yet is usually alert enough to blink to threat, grimace to pain, and move some parts of the body. Observation of some spontaneous self-help activity with the unparalyzed limbs is a clinical point against abulia. Whether the acute lesion is superficial, deep, or both and whether from infarction, hemorrhage, other etiology, or even as a postictal state cannot be determined simply by detecting global aphasia.

When he is faced with this major aphasia, the most the physician can do is to try to establish a baseline of interaction with the patient for use at a later re-evaluation: determine if the patient turns his head reliably to sound, especially if to certain words; try to gain some control over his behavior by pantomime commands such as showing your tongue in hopes he'll show his, touching your finger to your tie, to your nose, and so forth. Most patients will engage in some mimicry. Later failures to mimic indicate worsening, while making these movements to spoken or printed commands documents improvement. Be aware that the alert but globally aphasic patient usually tries to respond correctly and will engage in mimicry if you provide examples. A common examination error is to demonstrate simultaneously the same command you say aloud; if the patient responds properly, you may assume he has understood your spoken command, whereas he may only be mirroring your movements. The hopeful family often makes the same error of interpretation when they report that the patient understands everything and is improving.

Global aphasia unchanged for a week or more has a dismal prognosis for functional language. Some patients initially utter a few stereotyped words, often so clearly that the family interprets the speech as a sign of normal language function. Whether it represents the scanty capabilities of the nondominant hemisphere is unknown, but the repetitive, unchanging character of this speech makes it a falsely hopeful sign.

An aphasia closely approximating global aphasia occurs acutely following a lesion of the insula, frontal, Rolandoic, and parietal regions supplied by the branches of the upper division of the middle cerebral artery. Extension of the lesion up the Rolandic gyri produces hemiplegia and a hemisensory syndrome, but these signs are supplemental and may not be present when a more restricted insular-opercular lesion produces only the aphasia. No proper descriptive term or eponym has yet become popular, but the terms global aphasia or total aphasia are often used. The patient is mute and unable to write as in global aphasia but is less severely affected initially in comprehension. He is often able to point to objects, pictures, or pictureable printed nouns to dictated or even to written commands. This initial skill is important to document as it bears on the prognosis and lesion size. The more complex the command to which the patient responds by pointing to the correct choice (matching to sample), the smaller the lesion and the better the outlook. Names of clothing and body parts are the first materials to try, starting with the shorter words as separate commands (i.e., "ear," "eye," "nose"), then multisyllabic words ("forehead," "knee-cap"). Tasks involving grammar or formal language are especially difficult for these patients, a point that can be brought out easily by spelling the body part name aloud as commands ("e-a-r"). Introduction of sentence structure to the command adds to the difficulty of the performance ("touch your n-o-s-e"), while passive voice, negatives, sequential commands, and the like are worth trying only if the patient seems flawless in his responses. The limits of the performance are easily characterized in a clinical note, for example: "mute, points to named body parts, not when spelled."

Patients who respond well to grammatical tasks usually start talking within hours or days despite their initial mutism. This early speaking is a sign of focal infarction, not just transient ischemia. The emerging speech is dysphonic and dysarthric. More troublesome is the unskilled coordination (dyspraxia) between the oropharyngeal, laryngeal, and respiratory apparatus which produces a jerky, dysmelodic speech (dysprosody) that interferes with intonation, stress, and phrasing of utterances. The language expressed in this dysphonic, dysarthric, dysprosodic speech usually mirrors the grammatical disturbances noted in language tests involving reading and auditory comprehension. The more severe forms of this late emerging syndrome are known as Broca aphasia, while the patients showing mainly dysprosody are labelled as speech dyspraxia. These labels imply a spectrum of dysfunction: at one end is agragmatism with dysprosody and at the other end almost no language disorder and only mild dysprosody. The dysprosody gives away the insular and/or opercular locus of the lesion, but the size of the lesion is best judged by the severity of the agragmatism, especially in the acute stage. Because this disturbance undergoes improvement with time, the later that agragmatism is a prominent sign, the larger the lesion involving the insula and operculum. The early absence of agragmatism may mean a lesion too small to be seen on conventional CT scanning.

Although mutism is a common acute sign, in some syndromes speech is preserved acutely. Upper division anterior branch occlusions are expected to produce mutism, but speaking is usually spared in parietal and temporal branch occlusions, whether of upper or lower division origin. The speech of patients with focal parietal infarctions has good prosody, phonation, and ar-
ticulation but sounds like such gross jargon that the listener is easily convinced that the patient has a severe language disturbance. However, some or all of the errors may merely reflect anatomical orofaryngeal malpositions which are thought to be due to disordered sensorimotor feedback. The sounds thus produced can be compared to the typing errors of a novice to the keyboard, i.e., close to the correct spot but yielding a wrong letter. These speech errors are hard enough to analyze that the hurried examiner often resorts instead to the contrast between the patient’s indecipherable jargon and his far superior auditory and visual language comprehension to delare it primarily a speech, not language, disorder. The patient is usually greatly annoyed by the errors. He makes fewer when speaking slowly or with familiar words. This clinical picture is known as afferent motor aphasia, literal paraphasia, or as one form of so-called conduction aphasia.

A liberal admixture of errors involving word meaning (verbal paraphasias) usually points to a lesion in the territory of the lower division of the middle cerebral artery, affecting posterior temporal, parietal, and/or lateral occipital regions. Verbal paraphasias are substitutions for the desired word by another belonging to the same class, i.e., another animal name, a verb describing a similar action, etc. They occur especially for words which are the key elements in a sentence. Verbal paraphasias may also be accompanied by literal paraphasias. These paraphasias are especially easily observed in tests involving a naming response, where the desired words must occur alone and have no chance to be spoken as part of a practiced series of words that usually occur in succession. These key predicative words fail to appear on cue even in running speech; the patient pauses, hesitates, blocks, and searches for the response. Verbal or literal paraphasias may occur together. Although the hesitancies in this syndrome occur on predicative words, the casual observer may mistake the hesitancies and paraphasias as a disorder similar to dysprosody. However, dysprosody differs in that it occurs throughout utterances and on all types of words. The ambiguity inherent in terms like fluency versus dysfluency, expressive aphasia, and dysnomia make them unhelpful as diagnostic descriptions of the patient’s speech. The terms dysprosody and paraphasia (verbal, literal, or both) are more meaningful.

The larger the lesion, the more the paraphasias disrupt not only the predicative words but even the word strings that make up frequently used clauses and phrases. The greater the language disturbance in spoken speech, the greater the same type of disturbance in comprehension. An accompanying disturbance occurs in behavior, disrupting flow of speech and conversational interactions. Large and deep lesions produce a press of speech (logorhea), a failure to engage in conversational pausing and listening, and even ocasionally anger toward the examiner. At times there is so much unregulated speech that the picture resembles a delirium.

The more discrete and focal the lesion or the later in the time course, the more the patient passes for normal in casual conversation. Such patients may reveal their syndrome only in detailed cross-examination and history taking. Here, extended narrative yields circumlocutions, non sequiturs, and other illogical utterances that mislead the unwary examiner toward a diagnosis of dementia, schizophrenia, or confusion.

This large complex of language disturbance is known as Wernicke aphasia. It usually requires a lesion involving most of the posterior half of the brain for its full development and persistence. Less severe infarction and, in particular, subcortical hemorrhage may be associated with rapid change toward normal within days. Rapidly improving Wernicke aphasia is a syndrome that should prompt CT scanning within the first few days to rule out the subcortical hematomas that can cause this syndrome.

Other focal disturbances only slightly affect spoken language but may disproportionately involve auditory or visual language function. Infarctions of the superior temporal plane or small hematomas interfere especially with auditory comprehension of language, a syndrome in older times characterized too stringently as pure word deafness. Likewise, alexia with agraphia was the term used to describe the posterior parietal and lateral occipital lesions that disrupted reading and writing. In either syndrome spoken speech contains enough paraphsisics to make it clear these are not pure deficits but are instead the auditory or visual forms of Wernicke aphasia.

Small hematomas involving the thalamus of the dominant hemisphere may disrupt the thalamocortical projections to the language zones. These lesions produce a striking picture that fluctuates wildly from minute to minute between normal language and gross logorrheic paraphasia. Metabolic delirium is an understandably frequent misdiagnosis, usually corrected by checking the CT scan.

References

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