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Edited by Raymond D. Kent
Aphasia: The Classical Syndromes

Aphasia is an acquired disorder of language subsequent to brain damage that affects auditory comprehension, reading, oral-expressive language, and writing. Early observations by Broca (1861a, 1861b) and Wernicke (1874) suggested that aphasia might be classified into a variety of syndromes, or types, based on differences in auditory comprehension and oral-expressive language behaviors. Moreover, different syndromes were believed to result from different sites of brain damage. Revisions of early classification systems yield a contemporary taxonomy that comprises seven syndromes: global, Broca’s, transcortical motor, Wernicke’s, transcortical sensory, conduction, and anomic (Benson, 1988; Kertesz, 1979). Classification is based on the aphasic person’s auditory comprehension, oral-expressive fluency (phrase length and syntax), spoken repetition, and naming abilities. The seven syndromes can be divided into nonfluent, those with short phrase length and impaired morphosyntax (global, Broca’s, and transcortical motor), and fluent, those with longer phrase length and apparent preservation of syntactic structures (Wernicke’s, transcortical sensory, conduction, and anomic). An aphasic person’s syndrome may be determined by informal examination or by administering a standardized test, for example, the Western Aphasia Battery (WAB) (Kertesz, 1982) or the Boston Diagnostic Aphasia Examination (BDAE) (Goodglass and Kaplan, 1983). The following describes each syndrome and the assumed site of lesion associated with each.

Global Aphasia. This nonfluent syndrome is associated with a large left hemisphere lesion that may involve the frontal, temporal, and parietal lobes, insula, and underlying white matter, including the arcuate fasciculus (Dronkers and Larsen, 2001). It is the most severe of all of the syndromes. Auditory comprehension is markedly reduced and may be limited to inconsistent comprehension of single words. Oral-expressive language is sparse, often limited to a recurring intelligible—“bees, bees, bees”—or unintelligible—“doobe, doobe, doobe”—stereotype. Other automatic expressions, including profanity and counting, may also be preserved. Globally aphasic patients are unable to repeat words, and no naming ability is present. Reading and writing abilities are essentially absent.

Broca’s Aphasia. This nonfluent syndrome receives its name from the early reports by Paul Broca (1861a, 1861b). Classical localization of the lesion resulting in Broca’s aphasia is damage in the left, inferior frontal gyrus—Broca’s area (Brodmann’s areas 44 and 45) (Damasio, 1992). However, both historical (Marie, 1906) and contemporary (Mohr, 1976; Dronkers et al., 1992) reports question the classical lesion localization. Patients have been described who have lesions in Broca’s area without Broca’s aphasia, and other patients have Broca’s aphasia but their lesion does not involve Broca’s area. Auditory comprehension is relatively good for single words and short sentences. However, comprehension of grammatically complex sentences is impaired. Their phrase length is short, and they produce halting, telegraphic, agrammatic speech that contains, primarily, content words. For example, describing how he spent the weekend, a patient with Broca’s aphasia related, “Ah, frat, no Saturday, ah, frisk, no, fishing, son.” Repetition of words and sentences is poor. Naming ability is disrupted, and reading and writing show a range of impairment.

Transcortical Motor Aphasia. Lichtheim (1885) provided an early description of this nonfluent syndrome, and he observed that the site of lesion spared the perisylvian language region. Currently, it is believed that the lesion resulting in transcortical motor aphasia is smaller than that causing Broca’s aphasia and is in the left anterior-superior frontal lobe (Alexander, Benson, and Stuss, 1989). With one exception, language behaviors are similar to those in Broca’s aphasia: good auditory comprehension for short, noncomplex sentences; short, halting, agrammatic phrase production; disrupted naming ability; and impaired reading and writing. The exception is relatively preserved ability to repeat phrases and sentences. Essentially, patients with transcortical motor...
aphasia repeat much better than would be predicted from their disrupted, volitional productions.

**Wernicke's Aphasia.** This fluent syndrome received its name from the early report by Carl Wernicke (1874). The traditional belief is that Wernicke's aphasia results from a lesion in Wernicke's area (posterior Brodmann's area 22) in the left hemisphere auditory-association cortex (Damasio, 1992), with extension into Brodmann's areas 37, 39, and 40. However, Basso et al. (1985) have reported cases of Wernicke's aphasia resulting from exclusively anterior lesions, and Dronkers, Redfern, and Ludy (1995) have found Wernicke's aphasia in patients whose lesions also spared Wernicke's area. Spoken phrase length averages six or more words, and a semblance of syntax is present. However, the oral-expressive behavior includes phonological errors and jargon. One patient with Wernicke's aphasia described where he went to college, Washington and Lee University, by relating, "There was the old one, ah Frulich, and the young one, young hunter, ah, Frulich and young hunter or Brulun." A salient sign in Wernicke's aphasia is impaired auditory comprehension. These patients understand little of what is said to them, and the deficit cannot be explained by reduced auditory acuity. In addition, verbal repetition and naming abilities are impaired, and there is a range of reading and writing deficits.

**Transcortical Sensory Aphasia.** This fluent syndrome may result from lesions surrounding Wernicke's area, posteriorly or inferiorly (Damasio, 1992). Oral-expressive language is similar to that seen in Wernicke's aphasia: longer phrase length and relatively good syntax. Auditory comprehension is impaired, similar to that in Wernicke's aphasia, and naming, reading, and writing deficits are present. The salient sign in transcortical sensory aphasia is preserved verbal repetition ability for words and, frequently, long and complex sentences. Essentially, transcortical sensory aphasia patients repeat better than one would predict based on their impaired auditory comprehension. However, transcortical sensory aphasia is not exempt from controversy. Geschwind (1965) proposed that conduction aphasia results from a lesion in the arcuate fasciculus that disrupts connections between the posterior language comprehension area and the anterior motor speech area. Damasio (1992) suggested that conduction aphasia results from damage in the left hemisphere supramarginal gyrus (Brodmann's area 40), with or without extension to the white matter beneath the insula, or damage in the left primary auditory cortices (Brodmann's areas 41 and 42), the insula, and the underlying white matter. Dronkers et al. (1998) reported that all of their patients with conduction aphasia had a lesion that involved the posterior-superior temporal gyrus, often extending into the inferior parietal lobe. The salient sign in conduction aphasia is impaired ability to repeat phrases and sentences in the presence of relatively good auditory comprehension and oral-expressive abilities. Although auditory comprehension is relatively good, it is not perfect. And, while oral-expressive language is fluent (longer phrase length and a semblance of syntax), patients with conduction aphasia make numerous phonological errors and replace intended words with words that sound similar. Naming, reading, and writing abilities are disrupted to some extent.

**Anomic Aphasia.** This fluent syndrome is the least severe. Anomia—word-finding difficulty—is present in all aphasic syndromes; thus, localization of the lesion that results in anomic aphasia is not precise. It can be found subsequent to anterior or posterior lesions (Dronkers and Larsen, 2001), and Kreisler et al. (2000) report anomic aphasia resulting from a lesion in the thalamus; medial temporal area; or frontal cortex, insula, and anterior part of the temporal gyri. Patients with anomic aphasia display longer phrase length and preserved syntax; mild, if any, auditory comprehension deficits; good repetition ability; and mild reading and writing impairment. Frequently, the anomic patient will substitute synonyms for the intended words or replace the desired word with a generalization, for example, "thing" or "stuff."

**Cautions**

The classification of aphasia into the classical syndromes is not exempt from controversy. Some (Caramazza, 1984; Caplan, 1987) have challenged its validity. Darley (1982) suggested that aphasic people differ on the basis of severity or the presence of a coexisting communication disorder, frequently apraxia of speech. He advocated viewing aphasia unmodified by adjectives. The relationship between the site of lesion and the corresponding syndrome is also controversial. The classical sites of lesion for most aphasic syndromes are challenged by exceptions (Basso et al., 1985; Murdoch, 1988; Dronkers and Larsen, 2001). Some of the inconsistency may result from the time post onset when behavioral observations are made. Improvement in aphasia over time results in approximately 50% of aphasic patients changing from one syndrome to another (Kertesz and McCabe, 1977). Thus, an acutely aphasic patient with an inferior left frontal gyrus lesion may display the expected Broca's aphasia; however, at 6 months after onset, the same patient's language characteristics may resemble anomic aphasia. Confusion may also result from the methods employed to classify the aphasias. For example, classifications made with the WAB do not always agree with those made with the BDAE (Wertz, Deal, and Robinson, 1984). Finally, controversy and confusion may result from misuse of the term syndrome (Benson and Ardila, 1996). The behavioral profile that constitutes a specific aphasic "syndrome" is characterized by a range of impairment and not by identical performance among all individuals within a specific syndrome. In many, certainly not all, aphasic people, impaired behavioral features—fluency, auditory comprehension, verbal
repetition, naming—tend to result in different clusters that represent different profiles. These have led to the development and use of the classical syndromes in aphasia.

—Robert T. Wertz, Nina F. Dronkers, and Jennifer Ogar

References

Further Readings
A new concept in aphasiology was created when Wernicke (1874/1977) described ten patients with different forms of aphasia, and showed that two of the patients had fluent but paraphasic speech with poor comprehension (i.e., sensory aphasia). At autopsy of another patient, a lesion was found in the left posterior temporal lobe. This type of aphasia has been called by many names, including receptive, impressive, sensory, or more generally fluent aphasia. In most of the current classification systems, this type of syndrome is called Wernicke's aphasia. It affects 15%-25% of all patients with aphasia (Laska et al., 2001).

Although the exact boundaries of Wernicke's area are controversial, the typical lesion associated with Wernicke's aphasia is most often located in the posterior temporal area. The middle and superior temporal lobe posterior to the primary auditory cortex are affected in almost all cases. The primary auditory cortex is also often affected, as are the white matter subjacent to the posterior temporal lobe, the angular gyrus, and the supramarginal gyrus. In rare cases, restricted subcortical lesions may result in Wernicke's aphasia and hemiplegia, the latter being uncommon in cases with cortical lesions. Recent studies have not changed these classical views of the clinico-anatomical relations of initial aphasia.

Patients with Wernicke's aphasia are usually older than patients with Broca's aphasia. However, some rare cases of children with acquired fluent aphasia and a posterior temporal lesion have been described (Paquier and Van Dongen, 1991). Ferro and Madureira (1997) have attributed the age difference between patients with fluent aphasia and those with nonfluent aphasia to the higher prevalence of posterior infarcts in older patients. The most common etiological factor in vascular Wernicke's aphasia is cardiac embolus, which more often affects the temporal area, whereas carotid atherosclerotic infarctions are in most cases located in the frontoparietal area (Harrison and Marshall, 1987; Knepper et al., 1989). Coppins (1991), however, points to a higher mortality rate in older patients with stroke, which might cause a selection bias in studies showing a relationship between age and type of aphasia.

The typical clinical signs of Wernicke's aphasia include poor comprehension of spoken and written language and fluent but paraphasic (phonemic and semantic) speech. In some cases, neologistic jargon may occur. Naming is also severely affected, and phonemic or semantic prompting is of no help. Poor repetition distinguishes Wernicke's aphasia from transcortical sensory aphasia. Writing mirrors the speech output. Handwriting is usually well formed, but the text is without content, and jargonagraphia may occur. Because of posterior lesions, hemiparesis is present in rare cases, but visual field defects are more common. Many patients also show signs of anosognosia, especially during the acute stage of the illness. In most cases, the use of gestural communication or pantomime is affected as well.

Patients not traditionally classified as having aphasia may also show language disturbances resembling Wernicke's aphasia, such as patients with schizophrenia, dementia, or semantic dementia, a fluent form of primary progressive aphasia.

Some authors suggest that Wernicke's aphasia is not a unique entity but includes many variants. Forms of neologistic, semantic, and phonemic jargon and pure word deafness may all be grouped under Wernicke's aphasia. Pure word deafness is a rare disorder characterized by severe difficulties in speech comprehension and repetition with preservation of other language functions, including the comprehension of nonverbal sounds and music (Kirschner, Webb, and Duncan, 1981). However, when Buchman et al. (1986) reviewed 34 published cases, they were unable to find any really pure cases—that is, cases without any other more generalized perceptual disorders that could be classified as acoustic agnosia or mild language disorders such as paraphasia, naming difficulties, and reading and writing disorders. Most of the patients with "pure" word deafness have had bilateral temporal lesions, but some patients with unilateral left hemisphere lesions have been described (Takahashi et al., 1992).

Personality factors may play a role in the clinical expression of aphasia. In some views, jargon aphasia is not solely a linguistic deficit. Rochford (1974) suggested that a pathological arousal mechanism and lack of control were crucial to jargon aphasia. Weinstein and Lyerly (1976) suggested that jargon aphasia could emanate from abnormal adaptation to the aphasic speech disorder. They found a significant difference in premorbid personality between patients with jargon aphasia and those without jargon aphasia. Most of their patients with jargon aphasia had a strong premorbid tendency to deny illness or openly expressed fear of illness, indicating the importance of anosognosic features in jargon aphasia.

Linguistically, patients with Wernicke's aphasia speak with normal fluency and prosody without articulatory distortions. They often provide long and fluent answers (logorrhoea) to simple questions. In fact, patients with Wernicke's aphasia produce an equal number of words as persons without aphasia in spontaneous speech. However, they show less lexical variety, a high proportion of repetitions, and empty speech (Bates et al., 2001). This may give an impression of grammatically correct speech, but the meaning of the utterances is lost because of a high proportion of paraphasias and neologisms (Lecours and Lhermitte, 1983). This type of speech error is called paraphrasmatism. Patients with Wernicke's aphasia show morphological errors, but less so than
patients with Broca’s aphasia (Bates et al., 2001). However, there is some evidence that in highly inflected languages such as Finnish, the number of errors is higher on inflected words than on the lexical stems (Niemi, Koivuselkä-Sallinen, and Laine, 1987). At least in spontaneous speech, distorted sentence structure in utterances of patients with Wernicke’s aphasia is related to the lexical-semantic difficulties rather than to morphosyntactic problems (Helasvuo, Klippi, and Laakso, 2001). The same has been found in sentence comprehension. Patients with Wernicke’s aphasia performed correctly only on sentences that did not require semantic operations (Pinango and Zurif, 2001). According to these findings, the deficit in phonemic hearing does not explain the nature of comprehension problems in patients with Wernicke’s aphasia.

Most patients show skill in pragmatic abilities, such as using gaze direction and other nonverbal actions in conversation. Unawareness of one’s own speech errors usually occurs initially in Wernicke’s aphasia, but some degree of auditory self-monitoring develops after onset, and patients then begin to use various self-repair strategies to manage conversation (Laakso, 1997). In contrast to self-repair sequences in nonaphasic speakers, these sequences are very lengthy and often unsuccessful.

The initial severity of the aphasia is considered the most important single factor in predicting recovery from aphasia. Wernicke’s aphasia is usually tantamount to severe aphasia. In a study by Ross and Wertz (2001), all patients with aphasia, those with Wernicke’s aphasia and global aphasia showed the most severe impairment in language functions and communication. These patients showed only limited recovery when measured at the impairment level by the Boston Diagnostic Aphasia Examination (BDAE) and at the disability level by CADL. In addition to initial severity of aphasia, supramarginal and angular gyrus involvements seem to relate to poor recovery in comparison with cases without extension to the posterior superior temporal gyrus (Kertesz, Lau, and Polk, 1993).

Patients who have recovered from Wernicke’s aphasia have shown a clear increase in activation in the left perisylvian area, suggesting a functional reorganization of the language with the help of the right hemisphere (Weiller et al., 1995). However, Karbe et al. (1998) reported that increased activity in the right hemisphere was present in patients with poor recovery and reflected the large lesions in the left hemisphere. Patients with good recovery showed increased activation in the left hemisphere surrounding the damaged area.

The classification of aphasia depends strongly on the methods used in the assessment. The major diagnostic tests, such as the BDAE, the Western Aphasia Battery (WAB), or the Aachener Aphasia Test (AAT), have slightly different criteria for classification. For example, whereas the WAB assigns all patients to some aphasia classification, up to 70% of patients examined with the BDAE might be designated as having unclassified aphasia. Another issue that confuses classification is the time after onset at which the evaluation is done. Depending on the sample studied, more than half of patients with aphasia will show evolution to another type of aphasia during the first year after the onset of illness (Ross and Wertz, 2001). Patients with initial Wernicke’s aphasia will usually evolve to have a conduction or transcortical type of aphasia, and may evolve further to have anomic aphasia (Pashek and Holland, 1988). On the other hand, the condition of elderly patients with initial global aphasia tends to evolve to Wernicke’s aphasia during the recovery period, and the condition of younger patients evolves to Broca’s aphasia. This could explain why only one-third of patients with fluent aphasia and lesions in Wernicke’s area have a persisting aphasia, and only slightly more than half of patients with chronic Wernicke’s aphasia have lesions in Wernicke’s area (Dronkers, 2000).

——Matt Lehtihalnes

References


Further Readings


Aphasia Treatment: Computer-Aided Rehabilitation

The role of technology in treating clinical aphasiology has been evolving since studies first demonstrated the feasibility of using computers in the treatment of aphasic adults. This journey began with remote access to treatment in rural settings using large computer systems over the telephone. There followed the introduction and widespread use of personal computers and portable computers, with the subsequent development of complex software and multimedia programs. This changing course is not simply the result of technological progress but represents greater understanding by clinicians and researchers of the strengths and limitations of computer-aided treatment for aphasia and related disorders.

Four common types of treatment activities are appropriate for presentation on a computer: stimulation, drill and practice, simulations, and tutorials. Stimulation activities offer the participant numerous opportunities to respond quickly and usually correctly over a relatively long period of time for the purpose of maintaining and stabilizing the underlying processes or skills, rather than simply learning a new set of responses. It is easy to design computer programs that contain a large database of stimuli, and then to control variables (e.g., word length) as a function of the participant’s response accuracy. Drill and practice exercises teach specific information so that