

Language Disorders: Aphasia

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Glossary

Agrammatism – Characterized by articles, auxiliary verbs and some prepositions omitted or unrecognized in speech and writing.

Agraphia – Characterized by an inability to produce written language.

Alexia – Characterized by an impaired ability to recognize words and/or letters, thus affecting the ability to extract meaning from written text.

Anomia – Characterized by a struggle to find appropriate words in naming items and conversational speech.

Anomic Aphasia (also known as amnesic aphasia, nominal aphasia, and semantic aphasia) – Characterized by a failure to name or to retrieve names, and common and proper nouns in speech.

Apraxia (also called aphemia) – Characterized by errors in the selection or ordering of sounds such that resulting erroneous words sound like the target word but have the wrong meaning or are so distorted as to be non-words.

Broca's Aphasia (also known as verbal aphasia, expressive aphasia, and motor aphasia) – Characterized by speech output exhibiting poorly articulated words with missing, added, or transposed sounds, difficulty initiating speech, and reduced vocabulary.

Conduction Aphasia (also called central aphasia, repetition aphasia, and afferent motor aphasia) – Characterized by the repetition of individual sounds or words in speech.

Global Aphasia (also called total aphasia) – Characterized by equal impairment of all language modalities.

Wernicke's Aphasia (also called syntactic aphasia, sensory aphasia, and receptive aphasia) – Characterized by impairment in the understanding of spoken language.

Introduction

Kertesz (1979: p. 2) defined aphasia as “a neurologically central disturbance of language characterized

by paraphasias, word finding difficulty, and variably impaired comprehension, associated with disturbance of reading and writing, at times with dysarthria, non-verbal constructional, and problem-solving difficulty and impairment of gesture.” Goodglass and Kaplan (1983: p. 5) defined aphasia as “the disturbance of any or all of the skills, associations and habits of spoken or written language produced by injury to certain brain areas that are specialized for these functions.” Basso and Cubelli (1999: p. 181) defined aphasia as “a disorder of verbal communication due to an acquired lesion of the central nervous system, involving one or more aspects of the processes of comprehending and producing verbal messages.” Implicit in these definitions, of course, is a definition of language, itself a relatively complex concept.

The clinical syndromes of aphasia have been associated with particular anatomical loci in the central nervous system in the literature since the nineteenth century. Contemporary research, particularly aided with the use of modern brain-imaging techniques, suggests that in addition to the so-called classical language areas of the dominant (usually, left) hemisphere's cortex (Broca's area, Wernicke's area, etc.), many other areas of the central nervous system participate in language processing, including subcortical structures (including the basal ganglia, cerebellum and thalamus), other cortical areas of the dominant hemisphere (including inferior and mesial temporal lobe and insula) as well as various regions of the non-dominant hemisphere. This article focuses on aphasia as a behavior and not its anatomical substrate.

Approaches

In broad outline, there are two approaches to understanding the nature of aphasia. The first is concerned with functional components, what people do when engaged in language behaviors; the four modalities of language – speaking, listening, reading, and writing – are examples of functional components. Other functional components of language include being able to repeat what is said, initiating speech, speaking fluently, being able to name things and people, and being able to clearly articulate words. This approach to aphasia has typically been clinical; identifying impaired functional components of language in conjunction with impairments in one or more of the four modalities has led to a clinical classification of the aphasias that is used in assessment, rehabilitation, and research. An active area of

research in this approach has been correlating the loci of brain lesions with functionally identified syndromes of aphasia.

The second approach might be termed neuro-linguistic; it is concerned with the structure of language and which linguistic structures are impaired in aphasia. This approach begins with analyses of aphasic language in terms of linguistic levels – phonological, morphological, syntactic, semantic, and discourse – and then may take either an experimental or applied tack, correlating linguistic deficits, brain lesions, and clinical syndromes. The neuro-linguistic approach typically furnishes linguistic details to the functional-clinical approach, particularly in research applications.

Studying the localization of lesions that cause aphasia was at one time of major clinical importance; modern imaging techniques, applied to both brain-damaged aphasic patients and non-brain-damaged experimental subjects, have shifted emphasis to questions of which parts of the brain subserve which language functions and/or which language structures. A reasonable goal would be to identify a unique brain structure and its connections, damage to which caused a well-defined clinical aphasia syndrome and a well-defined linguistic impairment.

Functional-Clinical Aphasia Syndromes

Broca's Aphasia

Broca's aphasia has also been called verbal aphasia, expressive aphasia, efferent motor aphasia, and motor aphasia. The primary modality of language that is affected is speech production, but writing is often affected, too. Comprehension of spoken language and reading are usually much better preserved. According to the older scheme of dividing the aphasias into fluent and non-fluent, Broca's aphasia is the common variant of non-fluent aphasia. Speech output tends to exhibit poorly articulated words with missing, added, or transposed sounds, and there is usually difficulty in initiating speech. The range of vocabulary is often reduced from premorbid levels, and speech output tends to rely a great deal on basic, highly familiar, and thus overlearned speech patterns. Speech is likely to be in short phrases with fewer words than expected, punctuated with frequent pauses. Commonly seen in Broca's aphasia is agrammatism, speech and writing in which the small elements of grammatical structure, typically the so-called function words such as articles, auxiliary verbs, and some prepositions, are omitted or unrecognized. Anomia is also fairly common, causing patients to struggle with finding the appropriate word, both in conversational speech and in confrontational

naming. Although comprehension is noticeably better than production, some patients with Broca's aphasia have difficulty comprehending less frequent syntactic structures, although most demonstrate the ability to comprehend single nouns, verbs, or adjectives. Errors may occur in word order, which is called paragrammatism, but this is more common to Wernicke's aphasia (discussed next). Nouns tend to be preserved better than verbs and adjectives in Broca's aphasia, but grammatical function words are the most impaired. A concomitant of the shorter phrases and frequent pauses seen in Broca's aphasia is an impairment in prosody, alterations such as impaired inflection, pitch, and rhythm; this is commonly referred to as dysprosody. Repetition often shows the same impairments as in conversational speech. Writing in Broca's aphasia tends to be impaired analogously to speech output, but reading ability may be only mildly impaired; writing will exhibit misspellings, letter omissions, poor formation of letters, and agrammatism. Patients with Broca's aphasia are generally more aware that their speech and language is impaired than those with Wernicke's aphasia and thus may struggle to produce more correct responses.

The lesions typically leading to Broca's aphasia most often affect both the inferior frontal lobe and the anterior inferior portion of the parietal lobe; this is generally more extensive than the part of the third (inferior) frontal gyrus and surrounds that have been identified as Broca's area.

Wernicke's Aphasia

Wernicke's aphasia has also been called syntactic aphasia, acoustic aphasia, sensory aphasia, and receptive aphasia. The primary modality of language that is affected is speech perception, and reading may be affected, too. The ability to produce speech and the ability to write are usually much better preserved, although the content of speech and writing will likely be impaired. According to the older scheme of dividing the aphasias into fluent and non-fluent, Wernicke's aphasia is the common variant of fluent aphasia. The salient feature of Wernicke's aphasia is an impairment in understanding spoken language, particularly when the content of the spoken language is not predictable from the context or not otherwise highly familiar. In milder forms, comprehension may be contextually appropriate, e.g., a discussion of the weather, but lacking in details, e.g., unable to distinguish partially cloudy from overcast. On the other hand, the speech of the Wernicke's aphasic patient may appear articulatorily fluent but paraphasic, that is, an intrusion of non-words, words out of order, and word choices that are marginally related or unrelated

to the topic of the conversation. Paraphasias may appear in any variety of aphasia, but they are typically more prevalent in Wernicke's aphasia. As in Broca's aphasia, grammar may be affected, rather than function words being omitted, they are more likely to be used improperly or added extraneously. Grammatical word order constraints may be violated, a syntactic impairment called paragrammatism. Wernicke's aphasic patients are not likely to use complex sentence structure but rather will resort to simple, common declarative word order. Occasionally, patients with Wernicke's aphasia may talk excessively, even to the point of adding unrecognizable syllables, words, or phrases to their speech, a phenomenon known as jargon aphasia. Most researchers consider jargon aphasia to be a subtype of Wernicke's aphasia. In jargon aphasia, the jargon aphasic errors may be literal (single sounds), verbal (added words that are recognizable), or neologistic (added words that are unrecognizable). The patient with Wernicke's aphasia may be able to write letters and words correctly as a motor action, but the output reflects the patient's fluent paraphasic speech, including a disorganized and rambling style, occasional to frequent repetitions of words or phrases, jargon aphasic errors, and a lack of recognizable content. Reading tends to follow auditory comprehension ability, typically impaired.

The lesions responsible for Wernicke's aphasia are typically in the middle to posterior temporal lobe, particularly the superior gyrus, but frequently the middle temporal gyrus as well. These lesions often continue back to the junctions of the temporal lobe with the occipital and inferior parietal lobes, in the areas named the supramarginal and angular gyri.

Conduction Aphasia

Conduction aphasia has also been called central aphasia, disorganized execution of the encoding program related to disturbed auditory feedback, repetition aphasia, and afferent motor aphasia. The primary function of language that is affected is the repetition of speech, whereas comprehension and production tend to be much less impaired. Conduction aphasic patients frequently attempt to correct their repetition errors, implying a better preserved comprehension. The repetition errors may be at the level of individual sounds or words; repetition may exhibit anagrammatic character. The speech of the patient with conduction aphasia is more often like that seen in Wernicke's aphasia but sometimes is like that seen in Broca's aphasia. In addition to the repetition deficit, patients with conduction aphasia are often afflicted with anomia; literal paraphasias

(substitution of sounds) may intrude in both spontaneous speaking and in attempts to repeat what is heard. Within single words or very short phrases, articulatory fluency may be good, but patients with conduction aphasia typically display phonemic (or literal) paraphasias, a substitution of sounds. Although within-phrase syntactic patterns tend to be normal, many conduction aphasic patients have difficulty with sentences containing pronouns and grammatical function words, as well as polysyllabic words. Other characteristics that may be found include difficulties in writing, showing some forms of agraphia and ideomotor apraxia. Writing ability usually parallels speech output, showing deficits in spelling and letter omissions or substitutions. As is the case for Broca's aphasia, conduction aphasic patients are typically aware of their speech and language deficits.

The lesions leading to conduction aphasia tend to be located in and around the supramarginal gyrus and the arcuate fasciculus; the latter pathway connects the temporal lobe to the frontal lobe. Other lesions are along the border of the Sylvian fissure, extending to the subjacent white matter.

Anomic Aphasia

Anomic aphasia is also known as amnesic or amnesic aphasia, nominal aphasia, and semantic aphasia. The primary modality of language that is affected is speech production, restricted to the production of names, but it is most easily observed by asking an aphasic patient to name an object, so an input problem cannot be excluded. Anomia is described by the failure to name or to retrieve names, common and proper nouns; auditory comprehension is either unimpaired or only mildly impaired. One manifestation of anomic speech is a fluent output that lacks the nouns and verbs related to concepts. As a result, speech may be described as empty. Speech rate, articulation, and, surprisingly, grammar are typically normal, and the on-line deficits in word retrieval may be signaled by noticeable pauses. Accompanying impaired naming is the inability to comprehend nouns or verbs in isolation; however, object recognition is usually quite good. Although reading and writing are usually preserved, in severe cases of anomia there may be an anomic alexia or anomic agraphia. Some degree of anomia is found in virtually all varieties of aphasia; in part for that reason, no specific localization for the causative lesions has been or is likely to be documented.

Global Aphasia

Global aphasia has also been called total aphasia. All language modalities are affected in global aphasia to an equal degree, unlike the other aphasias, in which a

processing disparity among the modalities is evident. The causative lesions leading to global aphasia are typically very large, subtending all or most of language cortex.

Single-Modality Functional-Clinical Aphasia Syndromes

There are a number of aphasia syndromes that predominantly affect a single language modality.

Apraxia of Speech

Apraxia of speech has also been called aphemia, verbal apraxia, articulatory apraxia, and anarthria. Not all researchers agree that this is an aphasia syndrome; it is included here because, by definition, motor control of the speech musculature is not affected in apraxia of speech, in other words, it is independent of dysarthria. Apraxia of speech often accompanies Broca's aphasia but may be an independent, modality-specific impairment. The modality affected is speaking, and the problems are best described as errors in the selection or ordering of sounds such that the resultant erroneous words either sound something like the target word but have the wrong meaning or are so distorted that they are no longer words of the language.

Alexia with Agraphia

Alexia and agraphia are, respectively, input and output impairments of written language. Alexia and agraphia may occur independently or together; alexia with agraphia has also been called parietal-temporal alexia, central alexia, semantic alexia, angular alexia, and letter blindness. The primary modalities of language that are affected are reading and writing. Patients suffering from alexia with agraphia display impairments in both reading and writing skills. In general, their ability to copy words tends to be better preserved than their spontaneous writing ability. The inability to read and write extends into domains other than visual language: numbers, musical notation, and chemical formulas can also be impaired. Speech output and auditory comprehension may be somewhat impaired but typically only in a mild form of anomia. Both the location and the size of the lesion will strongly influence the manifestation of any aphasia, but these are particularly relevant in the impairment described as alexia with agraphia; the causative lesions are predominantly found in the region of the angular gyrus.

Pure Word Deafness

Pure word deafness has also been called auditory agnosia, isolated speech deafness, and subcortical

sensory aphasia; the modality affected is hearing. It causes patients to be unable to recognize speech sounds, while being able to hear non-language environmental noises, animal sounds, and music. Other language modalities – speech production, reading, and writing – generally remain intact. Lesions typically leave Wernicke's area undamaged, but destroy both Heschl's gyrus (primary auditory cortex) in the language hemisphere and the afferent auditory pathways coming from the non-language hemisphere. The functional result is that Wernicke's area behaves as though it is isolated from auditory language input; the patient can hear but cannot understand or repeat speech sounds. Except for the severely impaired input processing of speech, the patient with pure word deafness does not otherwise function like someone with Wernicke's aphasia. Auditory agnosia, which also reflects impaired processing of speech sounds, additionally reflects an impairment in processing non-language environmental sounds.

Agraphia

Agraphia, an inability to produce written language, has several neurolinguistic variants, which are discussed later in the neurolinguistic structures section. Functionally, it may appear as a written form of Broca's aphasia and, since written language so often mirrors spoken language, is typically associated with an aphasia. But, because writing also includes visuospatial skills as well as motor skills that differ from speech, impairments in spatial orientation or visual discrimination can cause agraphia without aphasia. Cases of pure agraphia frequently report damage within the frontal lobe, but a few cases have also shown damage within the left superior parietal lobe.

Alexia

Alexia is an acquired reading problem exhibited as an impaired ability to recognize words and/or letters, thus affecting the ability to extract meaning from written text. There are several varieties of alexia, including literal alexia (also referred to as letter blindness because the problem is primarily with individual letters), verbal alexia (also referred to as word blindness because whole words are primarily affected), general alexia (which refers to reading impairments that affect grammatical and/or semantic processing more than letters or words), and hemialexia, more commonly called neglect alexia (which refers to the impairment of attending to only half of a word or a line of text). In so-called pure alexia, written language stimuli are seen but not recognized as letters, as words, or both. In these cases, lesions tend to compromise the visual association cortex in the

language hemisphere together with the callosal fibers projecting from the other hemisphere, effectively isolating central language brain areas from visual input. There is a variant of pure alexia called letter-by-letter reading; such patients seem to process words by reading one letter at a time aloud before the word is identified. In some cases of alexia patients may successfully comprehend words if they are spelled out loud or traced on the palm, thus bypassing visual input to access the language core brain regions.

Transcortical Aphasias

The transcortical aphasias are sometimes known as the echolalic aphasias; there are three types, transcortical sensory, transcortical motor, and mixed transcortical, the latter sometimes known as the isolation syndrome. The modalities of language that are affected are speech comprehension (transcortical sensory) and speech production (transcortical motor) in the context of a sometimes dramatic spared ability to repeat, thus contrasting with conduction aphasia. In discussing the transcortical aphasias, it is useful to consider the notion of the language core, the temporo-parieto-frontal cortex of the language-dominant hemisphere in which resides the ability to repeat what is heard as well as the ability to process the basic sounds and word and sentence structures of one's language. Originally, the term transcortical meant that an ability to reproduce the sound structure or representation of a word was preserved, in the context of being unable to construct its meaning; this could be considered analogous to an ability to repeat a word in a foreign language that one does not understand. The predominant anatomically distinguishing feature of these aphasias is that the causative lesions are largely extra-Sylvian in location, that is, outside the classic language core. The following provides a brief overview of the generally accepted classical forms of the transcortical aphasias.

Transcortical Sensory Aphasia Transcortical sensory aphasia is an uncommon form of aphasia that may occur when a lesion functionally isolates Wernicke's areas from the rest of the brain, leaving the reception-to-output sufficiently unimpaired that repetition is preserved; neither speech comprehension nor spontaneous speech remain intact. The simplest way to describe transcortical sensory aphasia is to think of it as a form of Wernicke's aphasia in which the patient exhibits a severe comprehension deficit, but in which repetition, and thus articulation, is well preserved. In spite of intact articulation, the repeated speech of the transcortical sensory aphasic patient

may be paraphasic, neologistic, anomie, and even echolalic. Typical output may appear to be uninhibited. Patients with transcortical sensory aphasia typically tend to be unaware of their impairment; as might be expected, their speech is occasionally misinterpreted as a psychogenic problem, such as schizophrenia. Writing ability is usually disturbed in a manner similar to that of patients with Wernicke aphasia.

Transcortical Motor Aphasia Transcortical motor aphasia, another form of the transcortical aphasias, is sometimes known as dynamic aphasia or anterior isolation syndrome. Functionally, the causal lesion separates the processing of speech from the mechanisms for initiating the action to speak. Patients with transcortical motor aphasia tend to appear mute, or nearly so, and may even have an associated general akinesia, an inability to initiate action. Although transcortical motor aphasia impairs the ability to initiate speech, once such patients begin talking, speech output is typically relatively intact. Comprehension will be relatively normal, as will repetition. Prosody, articulation, and grammatical structure remain quite preserved even if verbal output is interrupted by incomplete sentences, verbal paraphasias, or false starts. When asked to say something, or otherwise initiate a response without cues, these patients have a great deal of difficulty responding; however, when asked to repeat words, phrases, or sentences, performance is characteristically flawless. There is a range in ability in word retrieval, with some patients being able to perform well on tasks such as object naming. Verbal output may improve if related to common, repetitious material. The lesions that lead to transcortical motor aphasia are typically found on the mesial surface of the anterior left frontal lobe, near supplementary motor cortex, or along the lateral aspect of the left frontal lobe; in either case these lesions fall outside of what is traditionally thought of as Broca's area. Presumably the lesions impinge on an anterior cortical or subcortical site that forms part of a circuit linking the motor speech area with the supplementary motor area and certain limbic structures considered essential for the initiation of speech and other actions.

Mixed Transcortical Aphasia Mixed transcortical aphasia, known also as the isolation syndrome, may be associated with Pick's disease or carbon monoxide poisoning affecting the so-called watershed region of the cerebral vasculature; the language core, the peri-Sylvian speech areas, are functionally isolated from other brain functions, particularly higher-order cognitive functions. The one remaining language

function is a striking ability to repeat words, phrases, and on occasion whole sentences. The isolation syndrome is most clearly the functional opposite of conduction aphasia; the former patient can only repeat speech, while the latter cannot repeat speech. Although articulatory fluency generally remains well preserved, the quasi-automatic repetition, often a frank echolalia, is prominent in a context of few if any other intact language functions. There is typically a complete alexia and agraphia, with an occasional ability to scribble meaninglessly. As pointed out by Benson and Ardila, other than the ability to repeat, patients with mixed transcortical aphasia exhibit the characteristics common to global aphasia.

Neurolinguistic Structures

The second approach, a neurolinguistic analysis of the aphasias, focuses on which linguistic components of language are affected by brain damage, within the framework of five, sometimes six, components of language: (1) phonology, or the sound system, (2) morphology, or the structure of words, (3) syntax, or the grammatical system, (4) semantics, or the system of meaning, (5) narrative or discourse, or the component that strings sentences together in coherent syntactic and semantic structure, and (6) the pragmatic or language use system. Linguistic-based descriptions of aphasic errors are neutral as to whether the errors are seen in speech production or comprehension or in written language. It is typical that the degree of impairment (percentage of errors seen on testing) of linguistic components is different in different language modalities; for example, a Broca's aphasic patient is likely to exhibit more severe agrammatism in speaking than in comprehending. It is occasionally documented that the linguistic impairments are overwhelmingly in one modality (e.g., being able to recognize grammatical errors but being unable to avoid producing them); this can lead to interesting theoretical issues regarding the nature of the language core brain areas, which are beyond the scope of this article.

Phonological Disorders

Phonological disorders are typically described in terms of phonemes, the minimal significant unit of sound in a language, or graphemes, the equivalent for the writing system. Errors may be described as substitutions (e.g., *bit* for *pit*), omissions (e.g., *cook* for *crook*), or sequencing errors (e.g., *cattle* for *tackle*, as sounds). Other phonological errors include problems with the control of prosody: syllables may be shortened or lengthened incorrectly, pitch contours may signal a question when a statement

was intended, or speech may be louder or quieter at inappropriate times.

Morphological Disorders

Morphological disorders affect a word's affixes, either (a) the inflectional affixes such as mark plural vs. singular, third person singular vs. first person singular, or past vs. present tense (e.g., *three cat* for *three cats*, *he will jumped* for *he will jump*) or (b) the derivational affixes such as mark words as nouns, verbs, or adjectives (e.g., *nationalness* for *nationality*).

Syntactic Disorders

Syntactic disorders, agrammatism, affect the grammatical structure of phrases and sentences. Obviously, if disorders of derivational or inflectional affixes discussed previously were to impact the grammatical correctness of a phrase or sentence (e.g., *he's a regularity guy* for *he's a regular guy*) one would speak of a syntactic deficit that manifested as a morphological error. The more common form of agrammatism is seen as the omission of grammatical function words (e.g., *boy hit ball* for *the boy was hit by the ball*), which clearly impacts the meaning of the sentence as well as its grammaticality; thus, as was the case with morphological disorders, syntactic disorders can overlap semantic disorders, too. A rarer form of syntactic disorder known as paragrammatism results in the inappropriate use and ordering of grammatical function words, rather than their omission (e.g., *in on a the by a flower pot* for *in the flower pot*). Additional syntactic disorders involve the simplification, ordering, substitution, or omission of phrase- and sentence-level structures that may interact with impairments in meaning. For example, if *what the pot put on the table was the boy* were substituted for *it was the pot that the boy put on the table*, or *what the boy put on the table was the pot*, one would note that the grammatical errors of substitution and ordering resulted in a change in meaning. As will be readily surmised, agrammatism is frequently, though not exclusively, seen in Broca's aphasia; paragrammatism is less frequently seen in Wernicke's aphasia because word order violations are infrequently observed syntactic disorders.

Semantic Disorders

Semantic disorders can take any number of forms, depending upon what aspect of the semantic system is impaired. Substitution of similar-meaning words is common (e.g., *concert* for *orchestra*) as is the interchange of superordinates and subordinates (e.g., *animal* for *dog*). A common strategy for anomic patients is to substitute an indefinite noun for an inaccessible

one (e.g., *something* or *stuff* in place of *shirt* or *clothes*). Semantic disorders may also impair knowledge of features of objects (e.g., being unable to indicate that a fire truck is red or that grass is green) or attribute incorrect features to objects (e.g., something inanimate is given attributes of being alive).

Narrative Disorders

Narrative or discourse disorders will affect the coherent stringing of sentences or phrases together in conversation, for example, changing a pronoun so that it no longer refers back to the person who is being discussed or inappropriately changing the time frame of a narrative. Pragmatic disorders refer to impairments in language use, for example, no longer understanding that the statement “I could use some salt on my roast beef” is an indirect request to another person to pass the salt shaker to the speaker. It will be immediately apparent that, just as with morphological and syntactic disorders, higher level problems with discourse and pragmatics may be described in terms of semantic or syntactic errors. The different linguistic components of language, levels, are simultaneously present when language is being used.

Alexias

The analysis of neurolinguistic structures has led to a syndrome classification of reading and writing disorders as follows. Three alexias have been defined in terms of the putative locus in a psycholinguistic model of reading: deep alexia, phonological alexia, and surface alexia.

Deep Alexia Patients with deep alexia usually have sustained a lesion sufficiently large to produce an aphasia, frequently a Broca’s aphasia; their reading is characterized by semantic errors in reading aloud (the error is semantically related to the target word) and may also show visual errors (the error is visually similar but otherwise unrelated to the target word), morphological errors (the error is a morphological variant of the target word), a concreteness effect (concrete words are easier to read than abstract ones), and difficulty in reading grammatical function words. Word frequency and word length may also impact the prevalence of reading errors. Patients with deep alexia are impaired in grapheme-to-phoneme conversion; as a result, they have a pronounced inability to read non-word letter strings that could be possible words in the native language (e.g., for English, the string *vib* or *phite*).

Phonological Alexia Phonological alexic patients can read real words but because they also have a

major problem with grapheme-to-phoneme conversion, they have difficulty reading pronounceable nonwords, as is the case with deep alexic patients. Patients with phonological alexia typically do not make the semantic errors seen in deep alexia; otherwise, the boundary between deep and phonological alexia is not always sharp and some patients seem to have many characteristics of both types. It has been reported that deep alexia may evolve to phonological alexia in the course of recovery.

Surface Alexia Patients with surface alexia have a reading impairment characterized by their ability to read orthographically regular words (note that about 75% of the English lexicon is orthographically regular, e.g., words such as *top*, *jelly*, *sing*) but a pronounced difficulty reading orthographically irregular words (in English, words such as *pint*, *come*, *bury* are orthographically irregular). Errors made by surface alexic patients on irregular words tend to be regularizing errors, that is, pronouncing them as though they were orthographically regular. Surface alexic patients are able to read pronounceable nonwords, e.g., *heaf* would be read to rhyme with the word *leaf*, which is regular, but not *deaf*, which is irregular.

Agraphic Disorders

As with reading impairments, there are three main linguistic forms of agraphic disorders. The first is phonological agraphia, which is an impairment in writing pronounceable pseudowords to dictation, with a much better preserved ability to write real words and occasional difficulties with grammatical function words and abstract words. A second form is semantic agraphia, which can occur with focal lesions but is more commonly found in the early stages of senile dementia of the Alzheimer type; such patients may write real words and pseudowords normally to dictation, but they make frequent semantic errors in written confrontation naming or written descriptions. A third form is lexical agraphia; these patients preserve the phonological form of the word when writing, but produce spelling errors that normalize spelling to approximate to how the word sounds.

Conclusion

In conclusion, the main recommendations today for continued use of the functional/clinical aphasia syndromes, the so-called classical syndromes, reviewed here are convenience and consistency. A great deal of modern research in neurolinguistics, clinical neuropsychology, and the cognitive neurosciences employs the classical aphasia syndromes for identifying patient groups, notwithstanding the research in the late twentieth century that casts doubt upon the

validity of the consistent location of causative lesions, other research that questions the logical coherence of the symptomatology of syndromes, and still other research that questions the possibility of studying groups of patients classified in terms of these syndromes. The most widely used test battery in the United States, the Boston Diagnostic Aphasia Examination (BDAE), from which the major aphasia syndrome typology originates, claims to be able to classify only about three-fourths of all patients with language impairments. One may still argue, and many contemporary publications will attest, that the patients who can be so classified into syndromes can, sensibly and statistically, be grouped under these headings for research purposes. In contrast, a linguistic typology of aphasia, a classification of neurolinguistic impairments, would only be challenged by a better linguistic theory. Such a classification does characterize aphasic impairments even if it does not neatly align with functional and clinical categories, nor does it neatly align with particular brain regions as revealed by lesion localization or imaging techniques. Caveat emptor.

See also: Language and Communication in Aging; Language Disorders: General; Speech and Communication (speech styles).

Further Reading

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Language Disorders: General

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Glossary

Aphasia – Acquired, usually abrupt-onset language impairment resulting from brain damage.

Dementia – Acquired disorder of cognitive and self-management abilities, that may be characterized by progressive decline of memory, judgment,

and abstract thinking, as well as changes in personality.

Discourse Skills – Skills allowing the appropriate use of pronouns, intonation patterns, and meeting social expectations in conversation.

Lexical Skills – Skills that relate to the production and comprehension of individual words.

Syntactic Skills – Skills that enable a person to understand and use conventional rules of grammar.

Overview of Language Disorders in Older Adults

Language disorders in older adulthood can be generally divided into progressive impairments of

Aphasias are a group of language disorders caused by brain damage. Unlike other kinds of language disorders, such as alexia, an aphasia affects both spoken and written language. A person with aphasia has an impaired ability to use language itself, both in understanding and in producing language, even if he or she has no perceptual or motor problem that would prevent him or her from hearing or seeing well or moving the muscles in the mouth to speak. What causes aphasia? Aphasia is defined as a disorder of language acquired secondary to brain damage. This definition, adapted from Alexander and Benson (1997), separates aphasia from several related disorders. First, aphasia is distinguished from congenital or developmental language disorders, called dysphasias. Second, aphasia is a disorder of language rather than speech. Speech is the articulation and phonation of language sounds; language is a complex system of communication symbols and rules for their use. Aphasia is a disorder that results from damage to portions of the brain that are responsible for language. Learn about its types, causes, and more. On this page: What is aphasia? Who can acquire aphasia? What causes aphasia? What types of aphasia are there? How is aphasia diagnosed? How is aphasia treated? What research is being done for aphasia? Where can I find additional information about aphasia? What is aphasia? Aphasia is an acquired language disorder with a diverse set of symptoms that can affect virtually any linguistic modality across both the comprehension and production of spoken language. Partial recovery of language function after injury is common but typically incomplete. Rehabilitation strategies focus on behavioral training to induce plasticity in underlying neural circuits to maximize linguistic recovery.