A Categorisation of Acquired Language Disorders with Special Emphasis on the Symptoms and Causes of Alexia

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1. Introduction

The importance of language in human behaviour has always been of the highest significance. This has especially become the case in recent times due to the heavy reliance on written communication. Hence, the effect of language disorders (and particularly reading and writing skills) on the modern western individual is particularly grave.

In this essay I have attempted to do two things. First, to present an ordered overview of the major disorders which affect language, and their symptoms: the various aphasias; oral apraxia; agnosia; various alexias; agraphia; and aprosodia. For each of these categories I present a definition, the key symptoms, related symptoms, and probable cause.

Secondly, the alexias and their causes are examined in detail. I have attempted to make this as up to date as possible and have touched on all the major issues relating to symptoms, recovery and causes.

2. Categorisation of Symptoms and Disorders[1]

Included in this section are the definitions of the major language disorders (excluding alexia, which is fully discussed in Section 3) and their symptoms. These categories are by no means mutually exclusive.

Various models of language processing attempt to account for the causes of these disorders. Acquired disorders (in contrast to developmental disorders which are primarily genetic in origin) virtually always occur after brain lesions. It is chiefly the location of these lesions which dictate the type and degree of language impairment.

2.1 Aphasia

Aphasia (literally “speechlessness”) is a disturbance in either the comprehension or production of speech. This does not include sensory or motor impairments (such as deafness, muscular deficiency), motivational lack or general mental deficiency (such as dementia).

In 90% of right handed people (and even most left handed people), it is the left hemisphere which controls speech. Hence, most lesions leading to aphasia are located in the left hemisphere.

Becker and Landau list some 40 types of aphasia (along with 40 alternate names). The more significant of these are discussed below.

2.1.1 Wernicke's aphasia

Wernicke's aphasia (aka acoustic aphasia) is defined by its cause rather than its symptoms: a lesion in Wernicke's Area (in the left posterior superior temporal lobe).

The key symptom is poor comprehension accompanied by fluent[2] but meaningless[3] speech. Paraphasia[4] is normally associated with Wernicke's aphasia, as is poor verbal repetition. There is usually a parallel impairment in reading and writing skills. Wernicke's aphasics commonly suffer hemianopia[5] and are often unaware of their disability.

It is suspected that Wernicke's Area contains memories of the sound of words. Thus, damage to Wernicke's Area would affect the ability to translate between phonology and the brain's internal representation of words (in both directions).

2.1.2 Pure word deafness

Sufferers of pure word deafness can produce speech (and can usually write), but fail to comprehend spoken language. They can recognise speech, but fail to extract meaning from the words they hear. This disorder is “pure” in that it is not accompanied by other disabilities.

Pure word deafness is caused by bilateral temporal lobe damage affecting the primary auditory cortex.

2.1.3 Broca’s aphasia

Damage to Broca’s Area leads to a “loss of ability to express thoughts in words even though the understanding of spoken and written matter is intact”[6]. Speech is slow, poorly articulated, agrammatic[7], anomic[8], with poor prosody (hence the synonym “non-fluent aphasia”).

Broca’s Area is situated in the left frontal lobe (which is thought to deal with language sequencing). This would explain why some Broca's aphasics have difficulty understanding subtleties in word ordering.

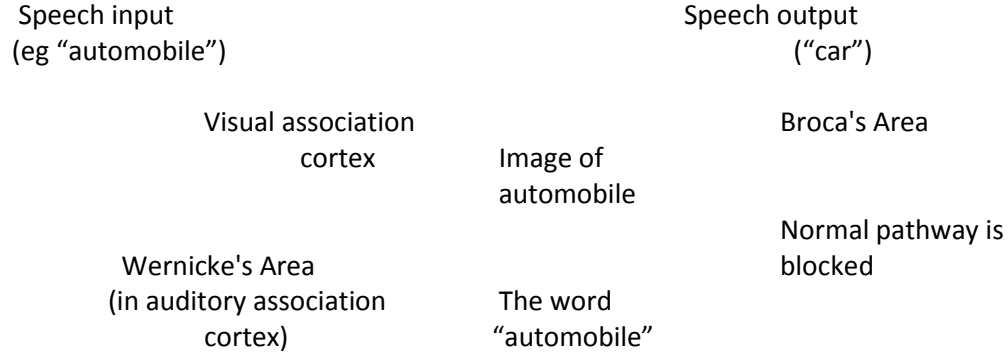
It is theorised that Broca’s Area contains memory of muscle movement, sentence structure and word endings.

2.1.4 Conduction aphasia

Conduction aphasia is marked by the inability to repeat words, without comprehension or voluntary production of speech being affected (though some paraphasia may be evident).

Interestingly, meaningful words and phrases can be repeated accurately (eg “blanch”, “up and down”) but not non-words or non-meaningful phrases (eg “blaynge”, “yellow big south”). Concrete nouns are more likely to be repeated than abstract words (and even then, the repeated word may be a synonym of the original).

The following schematic diagram indicating a lesion disconnecting Wernicke's Area from Broca's Area provides some explanation of these symptoms -



Normal repetition involves processing by both Wernicke's Area (recognition of the original word) and Broca's Area (production of repeated word). If these are disconnected, then this normal repetition is prevented. However, if the word represents a concrete object which may be visualised, then the visual association cortex may be able to pass the necessary information to Broca's Area.

One can imagine that Broca's Area may choose a different word to describe the visual information it receives (in the above example, “car” instead of the original “automobile”).

2.1.5 Anomic aphasia

Anomia is a difficulty in finding appropriate words (especially names of people and objects). Anomia is a common symptom in many aphasics, however, there are patients with “pure” anomia (aka anomic or nominal aphasia).

In anomic aphasia, speech is fluent and grammatical; comprehension and repetition are normal. However, the patients often cannot think of the words they want to use. This is not because they lack understanding of the concept they are attempting to express: they can usually describe what they want to say, but simply cannot remember the correct word.

In general, anomia may result from either anterior or posterior lesions (ie in both Broca's and Wernicke's aphasia), but pure anomia is always caused by posterior lesions.

2.1.6 Transcortical sensory aphasia[9]

The inability to convert between speech and meaning (in either direction). This causes patients to have very low speech output and apparently poor comprehension.

Although they neither initiate speech nor answer questions, it can be shown that they can both recognise words and speak understandably. They can repeat speech verbatim as well as complete known quotes which are commenced by an interogator. However, such use of language is devoid of meaning.

The cause of this disorder is hypothesised to be a disconnection between the speech areas and areas responsible for deeper understanding.

2.1.7 Transcortical motor aphasia

People with transcortical motor aphasia volunteer very little speech, though they exhibit good comprehension and repetition. Anomia is common.

The cause of this is damage to the supplementary motor area (in the superior frontal lobe), or a disconnection between this area and Broca's Area.

2.2 Oral apraxia

Oral apraxia is the inability to purposively control speech or mouth movements. This is not related to defective motor pathways. Rather, it is the conceptualisation of movements which is somehow impaired.

This maybe the root cause of the poor articulation evident in Broca’s aphasics.

2.3 Agnosia

Agnosia is the inability to recognise and interpret the meaning and significance of sensory information, even though perception is intact. Various types of agnosia relate to different sensory modalites. For instance, visual agnosia is that subset of agnosia which relates to visually perceived objects, pictures and written language.

Though this sounds similar to anomia, there is a clear distinction: while an agnosic cannot understand sensory input, an anomic can understand but cannot think of the words to describe it.

A good hypothesis of the cause of agnosic disorders would be the disconnection of the appropriate sensory cortex from the language areas.

2.4 Agraphia

The inability to write language (apart from motor or motivational causes) may fall into several categories: literal agraphia (can't write letters); verbal agraphia (can write letters but not words); mental agraphia (can't express ideas in writing); optic agraphia (can't copy written text); or sensory agraphia (due to impaired recognition of language).

2.5 Aprosodia

Prosody deals with the information communicated through intonation, rhythm, phrasing and punctuation. The aprosodic can neither convey nor comprehend meaning associated with these devices. Accordingly, they display monotonous speech.

Prosody is thought to be a right hemisphere function. Right frontal damage impairs production, while posterior damage impairs recognition of prosody. Of course, disconnection of right hemisphere from the left hemisphere language areas could have the same effect.

3. Alexia

Alexia is the inability to read, even though there is no lack in visual acuity. Sometimes this is related to visual agnosia, but often alexics can readily comprehend all visual perceptions apart from language.

There remains some overlap in the use of the terms “dyslexia” and “alexia”. Pedantic use of “alexia” would be limited to total lack of reading skills, forcing the use of “dyslexia” to cover all cases of partial lack. However, a more useful distinction is to apply “alexia” to the acquired disorder and “dyslexia” to the developmental disorder[10].

Alexics are often poor copiers of written text; are normally (but not always) hemianopic; and normally (but not always) exhibit colour anomia[11].

3.1 Categorisation

There appears to be several ways to categorise the alexic disorders. Becker and Landau list at least seven types: alexia with agraphia; alexia without agraphia (aka pure word blindness); aphasic alexia (words can be recognised but not their meaning); motor alexia (text can be understood but not read aloud); semantic alexia (text can be read aloud but not understood); musical alexia (musical script cannot be read); tactile alexia (Braille text cannot be read).

Other authors[12] (particularly describing aphasic alexia I suspect) emphasise the differences between literal alexia (inability to read letters), verbal alexia (inability to read words, though letters can be distinguished) and sentence alexia (inability to recognise words presented in a group, though individual words can be understood).

H□caen and Kremin are unsatisfied with these distinctions and present a classification based on three contrasts. Firstly, recognition (i.e. identification of linguistic units (letters, words, sentences)) versus reading (comprehension). Secondly, meaningfulness versus non-meaningful-

ness of the units being read. Thirdly, combinatory versus global comprehension (by which they mean whether the unit is interpreted by combining sub-units or by seeing the unit as a whole).

However, it is the categorisation of Benson on which I will concentrate. This categorisation is based on the three possible locations of lesions: anterior, central or posterior. Though even Benson admits that this is oversimplified, it provides a good framework for examining the causes of alexia.

3.1.1 Central alexia

Central alexia (aka alexia with agraphia) is a common disorder, caused by damage to the dominant angular gyrus (in the parietal lobe). The key symptom is impairment of both reading and writing ability.

Both literal and verbal alexia is present, and usually comprehension of numbers and written music are also reduced. Both reading aloud and comprehension are reduced, and spelling aloud makes little improvement.

Writing skill is almost as disturbed as reading: though letters may be produced, words cannot. Copying is mechanical (as though copying an unknown language), and transposition between cursive and printed writing cannot be acheived.

Other symptoms commonly present in central alexics are aphasia, right side sensory loss, right hemiparesis[13] (in early stages), right hemianopia, Gerstmann syndrome[14], and decreased overall intellectual capability.

The central location of lesions leading to these symptoms seem to imply that the left angular gyrus plays a crucial role in the interpretation of written language.

3.1.2 Posterior alexia

Posterior alexia is much less common than central alexia and is characterised by major comprehension difficulties without any writing difficulty (hence it is commonly called “alexia without agraphia” or “pure alexia”).

Typically, the patient can read some letters and a few common words (eg their name). However, they would be unable to read normal printed text or even text they may have just written. Because they can't verify their writing, it naturally deteriorates over time.

Comprehension can be improved by reading letters aloud or by tracing letters with the finger. Posterior alexics can write well from dictation, but not so well by copying.

Co-disturbances commonly present include right hemianopia, colour agnosia (though no general aphasia), mild anomia, and difficulty with number reading.

The chief cause is damage to the visual cortex (in the left occipital lobe). There is often additional damage to the splenium of the corpus callosum[15].

These lesions tend to give credence to the classical disconnection hypothesis, but it should be noted that this hypothesis does not adequately explain all cases. For instance, how is it that some patients cannot name words, but can name objects, colours and numbers?[16]

3.1.3 Anterior alexia

Anterior alexia is that form of alexia caused by damage to the left frontal gyrus (commonly extending into the anterior insula). Such damage leads to a failure to keep track of word sequencing, and inability to take account of significant relational words. They understand some words (especially nouns, action verbs and significant modifiers), yet have severe literal alexia.

When reading aloud, many words are ignored. Spelling aloud is of no assistance (due to literal alexia). However, any word which can be read aloud can be understood.

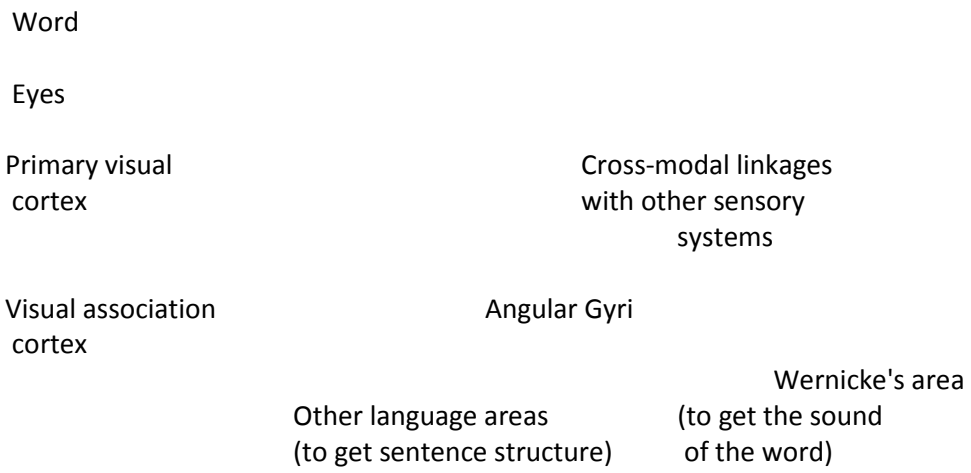
Agraphia normal accompanies this disorder, and hence even copying text is poor. Broca's aphasia is nearly always evident; right hemiplegia[17] is usually present; and some patients show sensory loss.

3.2 Recovery

Relatively little is known about the recovery of language disorders[18]. The level of recovery varies from none to almost total[19]. In general, the skill of processing visually perceived text is not regained. However, the brain finds ways to compensate for this loss by involving non-visual modalities in the language interpretation process.

For instance there are cases where tracing letters with the finger provides an alternative sensory impression that enables a patient to read. The general principle is to use some intact cognitive apparatus to substitute for the visual reading deficiency.

Benson explains a theory based on cross-modal association (the process of transfering information between sensory modalities) to account for this[20].



The angular gyrus (in the parietal lobe) has connections to the associative cortices of the visual, somesthetic[21], and auditory modalities. These connections are essential to the association of written and verbal language.

Both left and right angular gyri are involved in this process (the right handles nonverbal visual input).

However, these cross-modal connections are not sufficient for the task of reading. There must also be communication with Wernicke's Area to allow correlation of written language with phonological memories. And there must be communication with the parietal-temporal language areas and the frontal language area for the understanding of grammatic structures.

The theory suggests that damage to any part of the visual processing pathway will affect reading ability, but that the damage will be offset by the cross-modal connections.

4. Bibliography

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[1] Content of this section is compiled from Carlson (ch 16), and Becker and Landau.
[2] “Fluent speech” flows as though it were normal in terms of speed, intonation and sentence structure (notably the correct use of connecting words).
[3] “Meaningless” implies the use of many incorrect or nonsense words which cause the overall intent of a snetence to be obscured.
[4] “Paraphasia” is the insertion or transposition of syllables, words or phrases.
[5] “Hemianopia” is the inability to see in one visual field.
[6] Becker and Landau.
[7] “Agrammatic” implies the failure to comprehend or use grammatical constructs such as word endings, correct word order and connecting words. Though vocabulary is intact, syntax is faulty.
[8] See Section 2.1.5.
[9] Becker and Landau indicate that transcortical sensory aphasia is synonymous with conduction aphasia. However, my description follows Carlson.
[10] Benson, p. 69.
[11] Ajax.
[12] For instance, Benson.
[13] “Hemiparesis” is the partial paralysis (affecting motor movements but not sensory perception) of the one side of the body.
[14] “Gerstmann syndrome” is characterised by finger agnosia, right-left disorientation, acalculia (inability to do numeric calculations) and agraphia.
[15] Geschwind claims that both lesions are required. He explains that the lesion in the left visual cortex implies that only words in the left visual field are perceived (and these are transmitted to the right cortex). But the right hemisphere has only limited language capability and therefore must send a visual message to the left hemisphere. However, this is prevented by the damage to the splenium.
[16] Rosati et al. discuss such problems and suggest alternative explanations.
[17] Paralysis of the right side of the body
[18] Pirozzolo and Lawson-Kerr, p319
[19] Pirozzolo and Lawson-Kerr, p327
[20] The following schematic is my reconstruction of Benson's discussion on pp87-89.
[21] “Somesthetic” - dealing with bodily sensations.