

Recent Works on Aphasia

Carl Wernicke

During the past few years a succession of new works on aphasia have appeared, and we shall attempt to review the most pertinent findings which have emerged from such studies. . . . Professional interest in this subject has yielded two major points of view. The first, which is essentially psychological in orientation – at least in its present state of development – tries to analyze the aphasia symptom-complex into its component parts. In so doing, it attempts for the first time to establish a precise theory of the existence of cortical centers in the human brain whose correlative connections and functions are achieved by means of open circuits, i.e. the so-called association pathways. The second concept proposes localization of these various circuits and centers to different areas of the brain. It can be dealt with only when the reader has been prepared by a review of the clinical analyses of individual case studies and has classified them according to the centers and conduction pathways which have failed.

1 The Symptomatology of Aphasia

1

. . . The cerebral centers under consideration are, it is true, anatomically preformed, but their functional content represents the unique acquisition of each individual, usually during childhood. The child learns to understand the speech of others and thereby develops a center for speech comprehension. He then learns to express himself aloud, thereby acquiring a center for complex motor speech patterns. Speech mimicry, which is of paramount importance in this training process, is dependent on the usage of a conduction pathway connecting these two centers in such a way that each acoustic image arouses the corresponding combination of movements. This leads to the following schema of the speech apparatus which was first submitted in my work of 1874 and which since that time has found almost universal acceptance among clinicians.

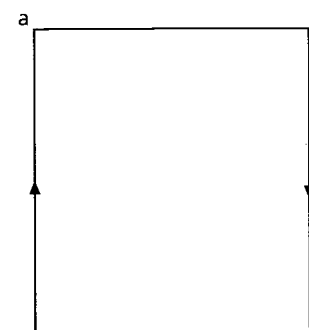


Figure 6.1 Schema of the psychic speech reflex. (a) Center of acoustic speech imagery. (b) Motor speech imagery.

A center (a), in figure 6.1 located in the central projection area of the acoustic nerve, contains the store of memory images of the speech sounds or the “acoustic images.” The center (b), situated in the so-called motor zone of the cortex, is likewise a depository of memory images of motor speech patterns which we may refer to as “motor speech-representation” or “motor speech images.” The latter cause activation of speech movements by means of a centrifugal pathway leading to the pertinent bulbar nuclei. A centripetal tract, that of the acoustic nerve, transmits to the first center. These centers are connected by means of an association pathway (a–b) which is utilized in mimicry of speech sounds. The destruction of center (b) causes motor aphasia, that is, loss of speech with intact speech comprehension, while destruction of center (a) results in sensory aphasia, that is, loss of speech comprehension with preservation of basic speech capacity. The disruption of the pathway (a–b) causes word-confusion in speech, a symptom to which Kussmaul has aptly applied the term “paraphasia,” and which I have designated conduction aphasia, provided that speech comprehension in center (a) and speech expression in center (b) are undamaged. . . .

[. . .]

One might even go a step further, as has already occurred in all quarters, and try to explain the reading and writing disorders so frequently observed in aphasia on the basis of two other completely analogous centers. Analogous to the speech comprehension center in the acoustic area is a center serving comprehension of written material in the visual cortical area. Analogous to the speech motor center a center for movements involved in writing. The symptoms of alexia and agraphia (to retain the old, established terminology), are explained on the basis of the failure of these centers.

The following schema, illustrated in figure 6.2, may then be formulated. Let χ be the location of visual memory images of written symbols, and let β be that of graphic

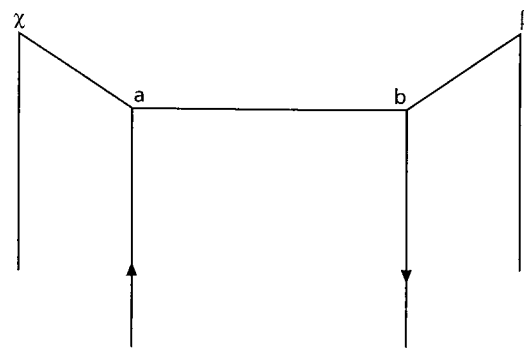


Figure 6.2 Schema of reading and writing. (a) Acoustic speech imagery. (χ) Visual letter imagery. (b) Motor speech imagery. (β) Graphic motor imagery.

motor imagery. Destruction of χ would then result in alexia as an analog of sensory aphasia, while destruction of β would produce agraphia as an analog of motor aphasia. However, the hypothesis of the existence of such specific centers may be justified only on the basis of appropriate clinical case studies, for even if memory images of written signs are undoubtedly located in the visual cortical region, just as those of the speech sounds are contained in the acoustic area, it is nevertheless questionable, and, I believe, as yet unproven, if they always lie together and are localized at a particular point of the visual cortical region. The same is true of the graphic motor images. Undoubtedly, they are also contained somewhere in the cortical area involved in innervation of the upper extremities. However, confirmation by an adequate number of clinical studies is necessary before their deposition in a specific area can be definitely established. . . .

In my first work on aphasia I took pains to show that in such an interpretation of the speech process as has been reviewed above, we had probably found the scheme of cortical function as a whole, that memory images were the psychic elements populating the cortex in a mosaic-like arrangement as a functional development which may very well be localized according to the regions of the nerve-endings, so that the acoustic images find their abode within the cortical terminals of the acoustic nerve; the visual images, within the cortical endings of the optic nerve; and the olfactory images in that of the olfactory nerve and so on. Likewise, the motor memory images or movement-representation could be located in the cortical sites of the motor nerve origins. For example, the images of speech movements would then be found in the Broca gyrus and those of writing within the cortical area serving arm movements, etc. Apart from that I assumed only that the discharge-sites of voluntary movements and the depository of motor images were identical. Any higher psychic process, exceeding these mere primary assumptions, could not, I reasoned, be localized, but rested on the mutual interaction of these fundamental psychic elements mediated by means of their manifold connections via the association fibers. Since that time I have

become even more strongly convinced, particularly on the basis of clinical studies of aphasia, that we are not justified in going beyond this elementary hypothesis.

[. . .]

2

Now, before undertaking a discussion of the work of Lichtheim, that author who has been the most consistent in his support of our own theory, and who has, as I am bound to recognize, carried it further with great perspicacity, I must first explain to the reader my interpretation of the term "object-concept."

It can be readily seen that our interest in the speech mechanism, at least in the light of present knowledge, lies particularly in its role as an agent of consciousness. As was indicated at the conclusion of my last discussion, the cerebral hemispheres *in toto*, as the organ of consciousness, function as the executor of the motor speech center *b* in spontaneous speech production. Likewise, the organ of consciousness *in toto* receives the message which is first transmitted to the sensory speech center at *a*, functioning as a receiving station for acoustic messages. Therefore, it would seem that further localization within this single organ of consciousness is not the issue here. But as soon as we wish to point to a concrete example as a test of our schema, we can find other, more comforting results. For example, how might the process involved in comprehension and spontaneous expression of the word "bell" be explained? If we are to comprehend this word, the concept of a bell must be aroused within us by the acoustic message which has reached center *a*. The acoustic message must stimulate the memory images of a bell which are deposited in the cortex and located according to the sensory organs. These would then include the acoustic imagery aroused by the sound of the bell, visual imagery established by means of form and color, tactile imagery acquired by cutaneous sensation, and finally, motor imagery gained by exploratory movements of the fingers and eyes. Close association between these various memory images has been established by repeated experience of the essential features of bells. As a final result, arousal of each individual image is adequate for awakening the concept as a whole. In this way a functional unit is achieved. Such units form the concept of the object, in this case a bell. Thus when a spoken word is understood and provokes thought, these units are in a sense a second station, accessible to our own recognition, in the total activity of the hemispheres, a station which must be passed through if the spoken word is not to die away in our ears without having been understood. Moreover, our consciousness makes use of this same station when the word "bell" is to be articulated spontaneously, i.e. as the result of what may be highly complex processes within our consciousness.

The first stage in this process then consists in the arousal of the concept of the object, "bell," and the second in the process of transmission to the pertinent motor memory images in *b*, the site involved in dispatch of the message. A schematic illustration of this process is indicated in the diagram in figure 6.3, in which (B)

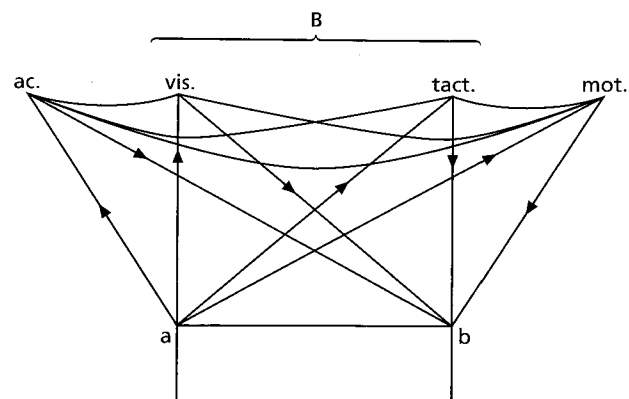


Figure 6.3 Composite schemata of language and conceptualization. (a) Acoustic speech center. (b) Motor speech center. (B) Conceptualization. (ac) Acoustic imagery. (vis) Visual imagery. (tact) Tactual imagery.

represents the concept of the object bell. The reader may find a very similar schema in Lichtheim's work. In the same way, if we attempt to construct the concept of the word or "symbol" as the name of an object is often called such as that of the word "bell" we find that, completely analogous to the object itself, it consists of the relevant firmly associated memory images in (a) and (b). These speculations then may suggest differentiation of speech comprehension into two stages, namely, (1) the arousal of the word and (2) arousal of the corresponding object-concept. The same process occurs in spontaneous speech, but in the reverse order, with the concept of the object emerging first, followed by that of the word.

This brief digression in regard to the word or symbol concept might seem unnecessary. However, we have an immediate need to use it. For we shall have to examine the extent to which such word-concepts are inseparable unities. Pathological research yields two lines of evidence relevant to this problem. On the one hand, if center *b* is destroyed, speech comprehension may remain completely intact; in other words, the acoustic imagery of the word is adequate for arousal of the concept of the object. If, however, center *a* is damaged, the independence of center *b* can be seen in the continued production of spontaneous speech. The latter, however, is characterized by inconsistent word-choice, with symptoms of word-transposition or paraphasia. Therefore, preservation of the word-concept is of greater significance in the active phase of the speech process than in the passive. Or, translated into terms of our schema, the association between the acoustic word image and the concrete object is firm and independent, but that between the object-concept and the pertinent motor word image is more fragile and not adequate to ensure accurate speech production. Presence of the word-concept in its entirety is necessary for production of spontaneous speech. This finding, gleaned from pathological studies, is comprehensible if

understood in the light of the mechanisms involved in speech acquisition. Undoubtedly the first knowledge of language which the child acquires consists of the comprehension of words, the association of acoustic images with concepts of concrete objects, while in many cases a further period of years is necessary for the development of the faculty of active speech. A preliminary stage of this last is the ability, by using the association pathway *a-b*, to imitate the speech sounds heard. On this basis I hypothesize that centrifugal innervation of the word-concept from the area (of sensory perception) of the concrete object follows a double path, namely the simple path *B-b* and the more complicated route *B-a-b*. If a portion of the latter is disrupted in any place incomplete activation of the word-concept will be reflected in the transposition of words. . . .

[. . .]

If we now return to our original schema in figure 6.3 and consider it in relationship to a concept center, which for the sake of simplicity we shall reduce to a point designated *B*, and if we also restrict the scope of aphasic symptoms to all such cases of speech disturbance in which the concept of the object itself is preserved, damage to the centers under question and their various conduction paths would yield seven different forms of aphasia.

Let us enumerate the causes of these, using Lichtheim's model but changing the order.

- 1 Damage to center *a*.
- 2 Disruption of the acoustic path ending in *a*.
- 3 Disruption of the centripetal pathway between *a* and the concept center.

Forms 1-3 constitute the group of the sensory aphasias.

- 4 Damage to center *b*.
- 5 Disruption of the motor speech pathway.
- 6 Disruption of the centrifugal pathway *B-b*.

Forms 4-6 constitute the group of the motor aphasias.

- 7 Conduction aphasia (previously observed by us).

[. . .]

The following nomenclature of aphasia may then be formulated.

- 1 Cortical sensory aphasia
- 2 Subcortical sensory aphasia
- 3 Transcortical sensory aphasia

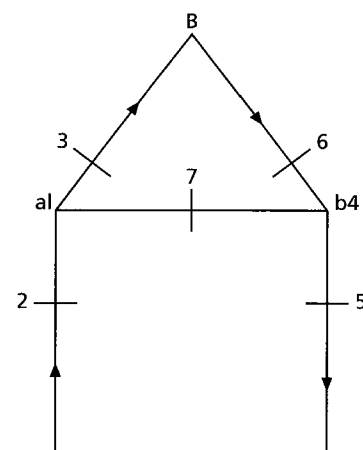


Figure 6.4 The Lichtheim aphasia schema using Wernicke's names. (B) Concept center, (1) Cortical sensory aphasia. (2) Subcortical sensory aphasia. (3) Transcortical sensory aphasia. (4) Cortical motor aphasia. (5) Subcortical motor aphasia. (6) Transcortical motor aphasia. (7) Conduction aphasia.

- 4 Cortical motor aphasia
- 5 Subcortical motor aphasia
- 6 Transcortical motor aphasia
- 7 Conduction aphasia

[. . .]

[Wernicke then elaborates on each of these forms of aphasia, using figure 6.4 to illustrate their origin.]

- 1 Cortical sensory aphasia is characterized by lack of comprehension of the spoken word and inability to mimic. However, the patient is able to speak spontaneously, although vocabulary is limited and characterized by frequent word-transposition, that is, paraphasia. See figure 6.4.
- 2 Subcortical sensory aphasia presents the same lack of comprehension of the spoken word and the same impairment in word mimicry. Spontaneous speech however is maintained, and the word-concept remains intact.

[. . .]

- 3 Transcortical sensory aphasia. Impairment in comprehension of the spoken word with preservation of mimicry. Symptoms of paraphasia are evident in sponta-

neous speech. See figure 6.4. It will be agreed that the features of forms 1-3 are complete without the necessity for consideration of disorders of written language. The assumption in all cases of sensory aphasia is that common deafness is not the basis of the lack of comprehension.

- 4 Cortical motor aphasia. Speech comprehension is intact, but the patient presents either muteness or a vocabulary limited to a few words. Spontaneous speech and mimicry as well as the voluntary mental sounding of the word are not possible. . . .
- 5 Subcortical motor aphasia. This form is differentiated from the preceding type by the complete integrity of the word-concept. See figure 6.4. The muteness is the same as that found in type 4. The patient is able to indicate the number of syllables contained in a word corresponding to an object presented to him.
- 6 Transcortical motor aphasia. . . . There is loss of spontaneous speech but no evidence of impairment in speech comprehension.

Of these three forms of motor aphasia, only the differentiation between forms 4 and 5, i.e. cortical motor and subcortical motor aphasia, presents problems which demand a critical discussion of written language. This function, writing, to anticipate a bit, is impaired in the first form of the aphasia mentioned, cortical motor aphasia, and is intact in the second, subcortical motor aphasia. Transcortical motor aphasia, however, may be identified without this feature. The cause of muteness in motor aphasia does not lie in paralysis of the speech musculature, just as common deafness is not the basis for impaired comprehension in sensory aphasia.

- 7 Conduction aphasia is primarily characterized by negative symptoms. If motor or sensory aphasia is not evident, but speech is paraphasic, presenting word-transposition, one may predict a disturbance in conduction between centers *a* and *b*.

I shall return later to the problem of so-called amnesic aphasia. This type is not related to the aphasia forms just reviewed, but is rather concerned with an actual memory disturbance.

3

The complex process involved in the acquisition of reading and writing may be more readily understood if one applies its obvious analogy to speech development.

One might then say that reading consists in the activation of the word-concept by the visual written image. Conversely, the process of writing involves the activation of the corresponding graphic motor image by the word-concept.

To produce a suitable schema (figure 6.5) let us use the same kind of device which we used earlier to denote the concept center *B*. In other words, let us write the word-

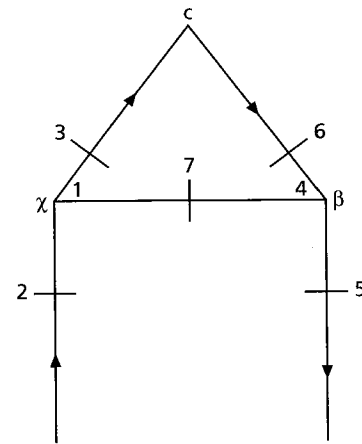


Figure 6.5 Schema of the alexias and agraphias. (c) Word-concept (acoustic and motor speech imagery). (χ) Visual written image. (β) Center of motor graphic imagery. (1) Cortical alexia. (2) Subcortical alexia. (3) Transcortical alexia. (4) Cortical agraphia. (5) Subcortical agraphia. (6) Transcortical agraphia. (7) Conduction agraphia.

concept as $a + b = c$, where the addition sign designates integrity of the association pathway $a-b$. This then leads to the schema formulated in the diagram in figure 6.5 in which χ denotes the visual written image and β the motor center of movements involved in writing. The pathway $\chi-\beta$, an analogue of pathway $a-b$ in the earlier schema diagrammed in figure 6.4, represents the crucial pathway by means of which writing is learned. Just as the critical process involved in speech-learning is repetition of the spoken word mediated by the pathway $a-b$, so the learning of writing is acquired under the controlling influence of the visual written image. The pathway $\chi-\beta$ therefore has the same significance for written language as the path $a-b$ for speech.

After these preliminary remarks, let us now turn to pathological case studies of written language disorders. The theoretical possibility of seven types of disorder therefore becomes immediately evident as was also true of disturbance in speech. Let us designate these disorders by the numbers 1 through 7, labeling disturbances of the centripetal pathway as alexia and those of the centrifugal path, agraphia. This provides us with the following summary.

- 1 Cortical alexia
- 2 Subcortical alexia
- 3 Transcortical alexia
- 4 Cortical agraphia
- 5 Subcortical agraphia
- 6 Transcortical agraphia
- 7 Conduction agraphia

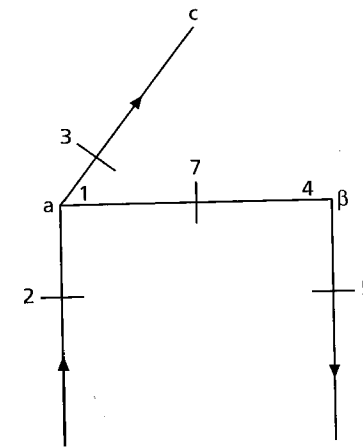


Figure 6.6 Schema of agraphias and alexias (with omission of tract $c-\beta$). (1) Cortical alexia. (2) Subcortical alexia. (3) Transcortical alexia. (4) Cortical agraphia. (5) Subcortical agraphia. [(6) Transcortical agraphia.] (7) Conduction agraphia.

A simplification of this schema is needed at this point, for reasons which shall be presented later, before we turn to a discussion of the symptom-pictures of the seven theoretically predictable forms, which is not written without practical interest. We can only briefly indicate here that there is much which contradicts the actual existence of a pathway $c-\beta$ and therefore the schema illustrated in the accompanying diagram in figure 6.6 alone may be used as a basis for our discussion. In this way an analogy to disturbances in oral speech can be readily demonstrated, although with certain simplifications.

- 1 Cortical alexia. Loss of reading and writing. Disruption of the centrifugal pathway for writing at χ .
- 2 Subcortical alexia. Inability to read. No disturbance in writing with the exception of writing from a model.
- 3 Transcortical alexia. Loss of ability to read and write with preservation of the mechanical copying of printed and script material.
- 4 Cortical agraphia. Loss of fine motor patterns requisite to writing. Copying is not possible. No impairment in reading.
- 5 Subcortical agraphia. Essentially the same picture. (Further comment will be made later.)
- 6 Transcortical agraphia. This form is nonexistent.
- 7 Conduction agraphia. Reading unimpaired. Loss of writing.

If one assumes the existence of a direct pathway $c-\beta$, transcortical agraphia, form 6, would be characterized by the exclusive loss of spontaneous writing. In form 7,

conduction agraphia, writing would still be possible, but paraphasia, analog of paraphasia observed in oral speech disorders, would be present.

Therefore, the control exerted by means of visual imagery may be considered much more indispensable to the writing process than that of acoustic imagery in respect of the speech process. Writing demands greater conscious activity than speech. In the latter, distracting factors may be predominant: the influence of the acoustic image a is always only slight, while the writing process, on the contrary, demands at least enough concentration to produce a series of letters.

Paraphasia, which is the chief issue here, therefore probably amounts to the same as complete agraphia; with impairment in formation of the letter itself. This can be seen when attempts to write produce only scribbles and lines which depart from the correct letter form. These samples of writing also occasionally contain correctly formed letters. Such, however, are infrequent, occurring only here and there among a group of incomprehensible scribbles. If correct and completely formed letters can be produced, and the disturbance is limited to transposition and interpretation of letters, one is dealing not with agraphia or paraphasia, as we interpret it, but rather with the symptoms of paraphasia, which may also be revealed in writing: in a word, written paraphasia.

A second point which must be briefly touched on here concerns the building of words by the combining of individual letters. The visual memory images deposited in χ and the motor memory images at β consist of single letters of the alphabet. The word-concept, on the contrary, always includes at least one syllable, and often several syllables, and is comprehended as a series of association of letters in a specific sequence. If we should take a very simple example of a one-syllable word such as *hand*, it can be seen that only the four letters in sequence, *h-a-n-d*, are able to call up the correct meaning. This is also true of multi-digit numbers. In alexia it will therefore be possible to determine whether whole words or only individual letters can be read, i.e. recognized. Likewise, in agraphia we must see whether only letters, but not whole words, can be written, or whether whole words, too, can be produced. For both the reading and the writing of individual letters it is necessary only for the concept of the letter to be intact, and this consists of χ , β , and the area between χ and β which I need not again describe in detail.

The fact that man, however, can learn to read letters without necessarily learning to write them – one need only point to the reading of printed characters – indicates the greater autonomy of χ in contrast to that of β .

We may now complete the classification of the individual forms of alexia presented earlier within the framework of this hypothesis. In cortical alexia, the letters presented to the patient apparently cannot be recognized but appear strange and foreign, a fact which may be confirmed by the use of leading questions. Furthermore, he is unable to copy, and in so attempting does not actually write, but merely draws the letters, and even then, later recognition is not possible.

Moreover, subcortical alexia spares spontaneous writing alone, and letters presented to the patient appear unfamiliar and must be laboriously copied from the model. Copying, in fact, is not possible. Occasionally letters may be recognized

during such attempts to copy, and under certain conditions may even be read, a trick which has long been observed, since it often occurs in aphasia.

In transcortical alexia, on the contrary, the letters appear to be recognized and can be copied without difficulty. Reading aloud is not possible since disruption of the path $\chi-c$ results in inability to arouse the associated acoustic image of the letter. In none of the three forms of alexia is the reading of words intact. This is readily understood, since reading of individual letters is not possible. Nevertheless, one is impressed by the striking ability demonstrated by such patients in their immediate reading of the word itself, as soon as the letters of which it is composed can be read. Therefore, it would seem that impairment of the word-concept must be postulated. A similar situation exists in the writing of letters and words. Only in impairment of the word-concept itself does it happen that the letters alone, but not the word, can be written without aid.

[. . .]