Sigmund Freud

Aphasia

The publication of Freud's APHASIA entry in the Diagnostic Lexicon for Practicing Physicians (Diagnostische Lexikon für praktische Ärtze) is published here for the first time in an English translation as part of my larger project of assembling bi-lingual texts of all of Freud's writings on the topic of aphasia. To quickly review the titles and publication dates of these texts, I offer the following list:

1888 Aphasie, in Villaret, Albert: Handwörterbuch der gesamten
Medizin, Bd. I, Stuttgart, l888, S. 88-90.

l891 Zur Auffassung der Aphasien, Leipzig und Wien, Franz Deuticke, 1891.

1893-94 Aphasie, in Diagnostische Lexikon für praktische Ärtze, edited by Bum, Anton and Moritz T. Schnirer, Erster Band, Wien und Leipzig, Urban & Schwarzenberg, l893.[this article]

l897 Inhaltsangaben der wissenschaftlicher Arbeiten der Privat- dozenten Dr. Sigm. Freud (l877-l897) Wien, l897. G.W. Bd. I, S. 472-473. (Freud's abstract of his l891 book)

All of the above: three articles and one book, have been translated into English and published before, except the l893-94 article which is published here in JEP for the first time.
Aphasia means the loss of the faculty of speech as a result of cerebral affection. It has been theorized that Freud's interest in it was at the time of a kind of nodal or turning point of his interest in seemingly purely organic medicine and his interest in hysteria (where there is no known organic lesion in the brain).
These articles on aphasia were published between l888 and l897.
The four case histories, that are his in Studies in Hysteria, took place from May of l888 or l889 to August of l893 and were published in l895.
If we discount a little bit, the last of his four publications on aphasia, as it is a one and a half page summary of his l891 book, that leaves us with one article before his book on aphasia, and this one which is after his book.
His book, which is very difficult to summarize, is a critique of the
localizationists, otherwise known as the diagram makers, who developed fanciful theories taking off from Broca's l861 discovery of lesions in the third left frontal cerebral convolution, which was posthumously discovered in cases of sensory aphasia (inability to understand) and Wernicke's discovery that lesions in the first left frontal cerebral convolution were posthumously discovered in cases of motor aphasia (inability to speak). From this, the localizationists or diagram makers, like Lichtheim, proceeded to hypothesize seven different types of aphasia corresponding to his beautiful diagram.
After succinctly summarizing their theories, Freud, in his book, countered these theories by showing contradictions in them, using their very own clinical examples to refute them, as well as their teacher's (Meynert's) theories. Then, using his own philosophical-psychological formulations of word-object association, combined with his own representation of the speech apparatus, he arrives at his own three part system of classification of aphasia types: verbal, asymbolic, and agonistic.

I cannot summarize his entire book in a few paragraphs.
Suffice it to say that there are hints of Ferdinand de Saussure's structural linguistics (arbitrariness of the sign, difference, and diachrony-synchrony) already here some twenty years before de Saussure's as well as Freud's theory of the forgetting of proper names.
In this particular article, the entry, Aphasia from the Diagnostic Lexicon for Practicing Physicians, which was written and published in 1893, two years after his book on aphasia, he seems to be more conciliatory toward the localizationists now that he has proven his points against them and has presented his own three part classification (verbal, asymbolic, and agnostic) of aphasia. He states a few general theses from them such as "we are able to approximately indicate the areas of the surface of the brain in which a lesion causes a purely asymbolic speech disorder (a case in Heubner). Agnostic speech disorders, on the other hand, occur only with extensive lesions in both hemispheres…". And, "On the other hand, destroying the peripheral area of the speech field generates specific aphasia."
You will see, also, that this article is a quick sketch for physicians as to what the speech function is; what aphasia is; a classification of speech disorders using psychological criteria; a clinical anatomy discussion of speech disorders; the diagnostic task for a case of aphasia symptoms and the problems of the clinical formation of aphasia. All of this is stated with a clarity and accurateness even by today's standards, which is all the more surprising because it was published exactly one hundred and ten years ago, when physicians did not have the benefit of an MRI!
Richard G. Klein

Aphasia (_____, speech).

Today, aphasia means a number of *acquired* disorders in admitting or producing articulated speech, which also meet the requirement of not being caused by a disorder of the peripheral apparatus for admitting speech (organ of hearing) or for producing speech (speech muscles), and not by a general clouding of the function of the brain (coma, psychosis). That is, we can speak of aphasia only if a person is not a deaf mute (unless he has been taught to use phonetic or sign language), and if the rest of his behavior indicates that he is not hindered from speaking and answering due to a delusional idea or a special condition of consciousness. Speech disorders resulting from motor paralysis of the speech muscles also do not deserve to be called aphasia, though it is no easy matter to sharply distinguish from genuine aphasia; it is discussed under "anarthria".
Since diagnosing aphasia requires judging a complicated psychic function, a few introductory remarks from this writer's study "On Understanding Aphasia" [*Zur Auffassung der Aphasie*] may not be superfluous.
The speech function is an associative function acquired with a great deal of effort; it brings together psychic elements of various origin. It is learned through listening and repeating, and for children too an intact sense of hearing is an indispensable condition for the formation of speech (deaf-mutism). For several years, the speech function is limited to associations between acoustic and motor elements (perception fragments, memory images), that is linking sound images with kinetic word images and reproduction of the latter. Reading and writing is learned at a later point in time. That result in adding new optical and motor memory images to the association whose meaning consists of their relation to the older acoustic and speech-motor memory images. Once this progress has occurred, an individual word represents a complex of four (or more) different psychological elements: the sound images of the heard word, the kinetic image of the spoken word, the optical image of the seen word and the kinetic image of the written word. Several other psychological elements, e.g. the sound image of the spoken word, the facial image of the written word are probably of secondary importance. We must imagine these psychological elements as being linked in such a way that each can be directly associated with the others, though the association with the sound image of the heard word has the primary role for the speech function. Single speech functions probably have a similar relationship as all association-based functions; depending on the intensity of the process, the association network is activated by individual elements or as a whole. In the final analysis, it is up to individual organization and exercise to assign a more prominent role to this or that association element in the actual performance of speech functions.

The speech function is, as it were, simply a special case of the general cerebral cortex function, which similarly consists of the association of different perception fragments (memory images). The “word” represents the association complex in the former case in the same way that the “object” represents it in the former, the only difference being that there is a limited number of “word associations,” while there is an indefinite number of “object associations!” The relationship between “object” and “word” is “symbolic” in nature. Each object is associated with a word as a “symbol” in order to gain a full picture of speech associations we must accept the association with the object.

It is quite probable that the association between object and word is not random between any given element of the object complex and any other given element of the word complex, but rather that the object selects the optical elements, while the word selects the acoustic elements. Accordingly, the schema of speech associations can be represented as follows (Fig. 23):

FIG. 23

The word presentation is shown as a closed complex of presentations, while the object presentation is an open one. The word presentation is not linked to the object presentation by all of its constituents, but only by its sound image. Of the object associations, the visual ones represent the object in a way similar to that in which the sound image represents the word. The links of word associations between each other (other than with the sound image) are represented by dotted lines; the connections of the word sound image with non-visual object associations are not shown.

This schema provides a classification of speech disorders using psychological criteria: disorders within word associations can be called *verbal* aphasia, while disorders in the association between word and object can be termed *asymbolic* aphasia, and those speech disorders which are generated by a disorder within the object associations can be called *agnostic* aphasia.

To date, we have viewed the speech function from a psychological perspective. We will now turn to a *clinical anatomy* discussion of speech disorders. Autopsies have taught us that the verbal associations of speech take place in a specific area of the cerebral cortex (association field of speech), developed only in the left hemisphere. Furthermore, we are able to approximately indicate the area of the surface of the brain in which a lesion causes a purely asymbolic speech disorder (a case in Heubner). Agnostic speech disorders, on the other hand, occur only with extensive lesions to both hemispheres, whereby the speech field itself can be fully intact. Not all areas of the speech field of the left hemisphere, whose size is shown in Figure 24, are of equal importance:

The Speech function is relatively unaffected by destruction of the central area (which contains the island convolutions, experiencing only indefinite aphasia. This aphasia consists of a general decrease in associative function; its specific characteristics have yet to be described.

FIG. *24.*

The hatched areas correspond to the speech field; the darkened areas above them are the so-called speech centers; specifically, 1 is the area where a lesion causes agraphia (area bordering the center of the hand); 2. Broca’s area, where a lesion causes motor aphasia (adjoining the centers for speech and larynx); 3. Wernicke’s area, where a lesion causes word deafness (adjoining the field for acoustic nerve or part thereof); 4. the area where a lesion causes alexia (directly adjoining the optic centers in the cortex). A large part of the central speech field lies deep in the sylvian fissure.

On the other hand, destroying the peripheral areas of the Speech field generates specific aphasia. Depending on the location of the peripheral lesion, damage occurs to the motor, acoustic or visual element in the speech associations, which can be diagnosed as motor aphasia (and agraphia), acoustic (sensory) aphasia and/or visual aphasia (alexia), and located with virtual certainty. These peripheral areas of the speech field are therefore also known as “speech centers,” a name which could lead to the erroneous assumption that they alone are responsible for the functions of the speech associations. Rather, their significance is due solely to the fact that they are adjacent to the centers of other associations, so that when they are destroyed, the speech field is wholly deprived of the association with the one or other element (motor, acoustic, visual). At first

glance, Figure 24 shows the location of the speech field of the left hemisphere and the importance of the double hatched “speech centers” as border areas against the other motor, visual and acoustic centers of the same hemisphere. We cannot anticipate a sharp division between either speech centers and the other speech field, nor between speech field and the other cerebral cortex centers.

We are better able to judge the importance of this figure if we remember that the speech association field is developed on only one side, while the fields for the hands, speech muscles, sight and hearing are developed on both sides, and that the association paths from the latter cortex fields of the right hemisphere also join in these bordering areas.

The diagnostic task for a case of aphasia is two-fold: First, the symptomology of the case of the type of aphasia is used to determine the type and therefore localization of the lesion; secondly, to recognize the significance of the symptom of aphasia for the clinical description and process. The following remarks apply to the first part of the task:

An examination of a case of aphasia is not fruitful until the general symptoms have passed; the examination will have to be repeated frequently because the symptoms change often and a gradual recovery from the original damage is almost the rule. It is not possible to continue the examination for very long because in the case of a damaged function, fatigue plays a significant role, and can easily exaggerate the extent of the damage. It also cannot not be expected that some of the individual speech functions will remain fully intact while others are fully absent. The foregoing is found only in the rarest of cases; in general, all individual speech functions are damaged, only with one being damaged to a much greater degree than the others. Also, only the simplest tests can be used to test speech functions; on the other hand, a number of errors must be avoided in interpreting these tests. For the above reasons. examining a case of aphasia is quite difficult. Individual symptoms of aphasia are obvious, while others are very difficult to find. Some of the symptoms have topical significance for diagnosis, while others having no such significance can be traced to general functional damage of the apparatus.

Let us assume a case of a speech disorder which we cannot yet call aphasia: a patient demonstrates incorrect, difficult-to-understand speech behavior, he can barely be understood because he is unable to produce single consonants and articulates like a child learning to talk. The reason could, for example, be a partial destruction of the cores of the facial, hypoglossal and vagus nerves in the medulla oblongata, or a lesion of the peripheral nerves followed by atrophy of the muscles used for speech. Still, we are able to observe that this patient attaches an intention to every word, producing something at least similar so that he never repeats two or more words using the same innervation, that the number of his attempts to speak is the same as the number of syllables in the words he attempts, and so forth. Clearly, this patient has not forgotten the word, but is simply incapable of reproducing it correctly. We do not call this aphasia, but rather *alalia* or *anarthria* (see above). On the other hand, another patient is perfectly able to articulate correctly, one can clearly understand what he says, but one notes that the words he uses are probably not appropriate to his intended meaning. He frequently makes mistakes, uses a word close to the meaning or sound of what he means, notices this mistake and is able to correct it–or is not. We call this symptom *paraphasia*; it has no topical diagnostic significance, indicating instead a lower functional capability of the speech association apparatus. It is also found to a lesser extent in conditions of physiological fatigue, when the speaker’s attention is divided, as a symptom of so-called absent-mindedness. Further observation of the same patient will reveal a second symptom belonging to aphasia: *amnesia* (see above). We see that the patient frequently remembers a word he is totally incapable of finding as hard as he tries, which he knows, which he is finally forced to replace with a word of general meaning or a circumlocution, which reminds us of similar occurrences in our everyday speech in which we forget words. Amnesia primarily affects proper names and nouns; cases are known in which patients have to find a circumlocution for every noun.

We are correct in diagnosing aphasia in those cases in which paraphasia and amnesia are found together and are more than physiologic. Amnesia involves a greater or lesser limitation of the patient’s vocabulary. As long as this verbal impoverishment is not excessive, a case characterized by paraphasia and amnesia and an aggravation of all complex language functions (e.g. reading and writing) can be called *indefinite* aphasia. In such cases, we presume incomplete, central lesions of the speech field.

If the amnesia and the vocabulary impoverishment are more extensive then, of course, paraphasia must decline and we obtain the clinical description of *motor* aphasia in which the subject is limited to a small

number of words. In this case, we can assume a lesion of Broca's area. The motor aphasia patient can be completely mute; he may be able to utter 2 or 3 words, usually "yes" and "no," and other "speech fragments". If he is fully without speech, it is of course important to determine whether he understands, speech. The above is easily determined by observing the patient's psychological behavior and mimicry. A patient who neither understands speech nor motor speech functions will not be able to respond to requests directed at him; his features will remain unchanged when asked insulting or absurd questions. Such a patient we call *totally aphasic*. The symptoms of complete aphasia are often found in the first days after a serious injury of the speech apparatus; understanding of speech is later restored and the patient is spared *motor aphasia*. The motor aphasia patient is involved, anxious to communicate, he often uses the same word with different meanings, which he differentiates using appropriate gestures; in general, he is unable to repeat words recited to him. He is always able to produce a portion of his limited vocabulary, if he manages to reproduce another word, he repeats it in inappropriate contexts. He is unable to take apart his speech fragments, which are often complex sentences; that is, he has no conscious control over them. That is, his condition is characterized primarily by *poverty of speech*. He has an unlimited capacity to understand words, he is able to make the link between word and object as easily as a healthy person, that is, he recognizes every word spoken to him as a sound image and correctly links it to its object. As a rule, the motor aphasic cannot read, and is seldom able to write unless this function has not been eliminated by a concomitant paralysis of the hand.

If all of the above circumstances facilitate the diagnosis of motor aphasia, as a rule acoustic aphasia must be sought carefully and not confused with other conditions. The *acoustic* (sensory) *aphasic* is characterized by an inability to understand speech, that is, in social situations they act confused, do not respond to questions or give wrong answers, and only the contrast with their otherwise circumspect and appropriate behavior would draw attention to the fact that they may have a disorder understanding speech. Another symptom is that they imagine things from what they have heard; for example, they believe they have understood something and base their answer on that understanding, which appears to he nonsensical.

In a number of cases, it has been possible to force comprehension by insistently repeating a question. It is also common for individual short, familiar questions to be understood, while others are not. Acoustic aphasia (also known as *word deafness* is seldom found in its total form.

The symptom of acoustic aphasia leads us to move the lesion to the first and second left temporal convolution (Wernicke's area), although frequently this area can be destroyed without producing word deafness. Given the great significance of sound images as speech association nodes, it is understandable that acoustic aphasia creates the most serious disorders in the other speech functions. In individual, insufficiently explained cases, spontaneous speech remains intact. However, in general, as with other aphasics, this patient's speech is characterized by paraphasia and verbal impoverishment. Unlike motor aphasia, however, the abundance of the speech impulse does not suffer. That is, acoustic aphasics speak a great deal, though not correctly. Their vocabulary is very poor in nouns and words of specific meaning; it is rich in particles, emotional words, in repetition. In the case of more serious lesions, the patient continues to speak abundantly, though the result of the innervation is gibberish, a frequently endless stringing together of nonsensical, correctly articulated syllables. Since the association of word and object is communicated by sound images, acoustic aphasics are also incapable of every "symbolic" speech function. They are unable to find an association with one of the presentations of the word group for a shown object. They are neither able to spontaneously pronounce the name of the object nor to write it down, nor to put it together from letters shown to them. Reading is impossible; any existing optic aphasia is masked by acoustic aphasia, the more serious disorder. However, it is precisely in this case that we must reckon with the frequent presence of dissociation of individual speech functions as a result of lesion, for which there are no general rules, and we would do well to base our diagnosis on the principal trait, the loss of function or worsening of understanding speech.

The third type of aphasia, *optic* aphasia, is characterized by a disturbance of the understanding of letters and an inability to read. However, only the first of these symptoms, non-recognition of letters, can be used to diagnose optical aphasia. This is due to the fact that other types of aphasia also involve the inability to read, despite perfect recognition of individual letters, and this kind of behavior has no topical significance. Optic aphasia can only be diagnosed with certainty where no acoustic disorder, that is no disorder of speech

understanding, is present. Optic aphasia also involves paraphasia and poverty of speech, though the changes in spontaneous speech are so slight in this form that they must be sought. Frequently, patients are able to conceal their speech deficiency through circumlocution, careful word choice and slow speech. With relative frequency, optic aphasia takes the form of an isolated and intense partial disorder (see *alexia*)

In aphasia, writing disorders–writing having been learned in close connection with life–are revealed relatively independent of reading disorders, though they run quite parallel to motor aphasia disorders. It is highly unusual to find patients whose sole speech disorder is *agraphia* (see above), though they do not exhibit paralysis of the right hand.

The above remarks have completed the topical diagnosis of aphasia. The more pronounced the verbal impoverishment, the more we must shift the lesion to the frontal end of the speech field; the stronger the disturbance of understanding speech, the more certainly it is located in the temporal lobe, and in the case of a distinct disorder in understanding letters, in the parietal-occipital end of the speech field. Topical diagnosis is frequently reinforced by concomitant symptoms. For example, in addition to motor aphasia, there are right facial paresis or unilateral body paresis, indefinite aphasia can be accompanied by cerebral hemianesthesia, while optic aphasia in its pure form (*alexia*) is overwhelmingly present next to dextral hemianopia. In acoustic aphasia, we must make sure that partial deafness, one-sided hearing impairment, loss of certain kinds of sounds are not also present.

Other symptoms of aphasia have no bearing on diagnosis, though they are worth studying because they provide a picture of the dependence and independence of individual speech functions, of their dissociation and of the possibility of substitute functions. To obtain an overview of these conditions, each of the motor speech functions can be examined for their *spontaneous reaction to sensory stimulus*, which can be broken down into the following schema:

I. Speaking	II. Writing
1. spontaneous	1. spontaneous
2. _) after hearing (repeating) _) after seeing (reading)	2. _) after hearing (repeating) _) after seeing (reading)

The rule also applies that it is common to retain motor speech function with sensory stimulus (2a and 2b) with loss of spontaneous speaking (1), though never vice versa.

In the case of the sensory speech function, which is always the result of sensory stimulus, special attention must be paid to testing verbal and symbolic associations.

	III. Reading	IV. Hearing
Verbal assoc.	1. reading out loud	1. repeating speech
1. spontaneous	2. copying	2. writing down
Symbol assoc. (direct)	3. understanding read material	3. oral comprehension
		4. oral comprehension (indirect)
		5. writing comprehension

There are cases in which verbal associations are still possible while symbolic associations are not. As can be seen, as associations most speech functions can be disturbed by lesions in a variety of sites.

The *clinical significance* of aphasia symptoms is quickly defined. Aphasia, regardless of its form, is a deficiency symptom of the cortex of the left hemisphere. This statement requires only a few reservations. It can be stated that subcortical lesions do not result in aphasia. However, such a cortical lesion can be so close that it damages tissue and integrity and function. In this case, aphasia is a symptom of an indirect effect, which must be of the same type as a direct one. The only exception to the above is a subcortical lesion below Broca’s area. A disturbance of this area creates dysarthic disorders, and when a case of motor aphasia is

accompanied by symptoms of anarthria, it is possible to conclude that the disorder goes from the cortex deep into the medullary substance. A second exception concerns the side of the lesion. In the overwhelming majority of cases, aphasia is associated with a disorder of the left hemisphere. However, for left-handed persons we should be prepared to find the same symptoms in the right hemisphere. That is, stated more correctly: aphasia is a deficiency symptom of the cortex and bordering medullary substance of the dominant hemisphere.

As such it has precisely the same clinical significance as the other motor and sensory paralyses due to diseases of the cortex, the brachial and crural monoplegia, the hernianopia, etc., with the difference that aphasia is in all certainty a cortex symptom, meaning that it is crucial in an otherwise topically ambiguous paralysis complex.

Aphasia is not especially important in diagnosing the course of an illness. With our current state of knowledge, the clinical formation of aphasia does not by itself permit us to determine whether it is the result of a vascular disorder, embolism, hemorrhage, thrombosis, from a tumor, an acute inflammatory process and the like. Aphasia remains the local symptom of the speech field of the dominant hemisphere, and in weighing the clinical importance of a case it should be assumed that the existing lesion would have produced motor or sensory paralysis if it had been created in another location. Therefore, for the rest, please consult the diagnostic evaluation of paralysis.

Since the speech field falls almost within the area supplied by the sylvian artery, this explains the frequency of aphasia in diseases that favor this artery, such as embolism, luetic arteritis. The best description of partial aphasic disorders is provided by occlusion of individual branches of these arteries.

It should be expressly noted that a number of speech disorders, which are not the result of acquired diseases of the speech field in the cerebral cortex, are not covered here.