ON APHASIA

 Authorized Translation by
 E. STENGEL M. D.

LEIPZIG UND WIEN.
FRANZ DEUTICKE.
1891.
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Notes on creating a bilingual of Freud’s works on the topic Aphasia, especially his book *Zur Auffassung der Aphasien* (*On Aphasia*)

I used the English translation by Erwin Stengel (1902-1973) published as *On Aphasia* by the International Universities Press in 1953. For the German text, I used the new German Edition, *Zur Auffassung der Aphasien* published by the *Fischer Taschenbuch* in 1992. This particular new German edition did not use the original pagination of Freud’s original 1891 edition, which quite frankly left me speechless. However, the new German edition did notate the original pagination in the margins. So it did not faze me in the least to reconstruct the original 1891 edition and also to re-store the original page headings which Freud used at the top of every page to the English translation. I had to make a few decisions as to what to do with the copious footnotes of the new German edition. In order to include them all, I had to occasionally put a few of them at the bottom of the side of the English translation.

We must not forget that Freud’s concern with the mouth and speech is one of the threads running throughout his entire career. From this book on aphasia, to the talking cure, to the dream of Irma’s injection, to Freud’s cancer of the jaw. As Patrick J. Mahoney pointed out in his book, *On Defining Freud’s Discourse*, Freud’s first name Sigismund, later Sigmund, means “Victory-Mouth”. And I would further add that his whole name, Sigmund Freud, corresponds to Lacan’s formula for the fantasm, ($ <> a), Sigmund =Victory of the Mouth (the talking cure) <> Freud (joy, jouissance).

The title in German, *Zur Auffassung der Aphasien*, if written otherwise as: *Zur Auffassung Der Aphasien*
One can observe that the repetition of the A-f-s-n sound. The very pronunciation of it sounds like a stuttering. The metonymic forwardness of the title in some way collapses into a repetition.

Another peculiar thing is that on page 26 (Eng), page 27 (Ger) Freud quote’s one of Dr. Hammond’s Spanish speaking patients. “When Hammond asked him “Come sientes ahora?”(How are you now?) he repeated, “Come sien, sien, sien”, “ which happens to be close to the German word “Aphasien”.

Perhaps, unbeknownst to today’s experts on aphasia is that the Freudian concept of paraphasia (mistaken use of words) eventually gave rise to the Freudian concept of parapraxis (Fehlleistung) in psychoanalysis, they have sub-divided paraphasia into the three following categories:

“Literal/phonological paraphasia-more than half of the intended word is produced correctly. For example, a patient may say /pun/ instead of /spun/ in addition, transpositions of sounds can occur, e.g. tevilision for television This is also called phonemic paraphasia. The sound is close to the intended word. An example from Freud would be: “Butter“ for “Mutter”.

Neologistic parapaphasias-Less than half of the intended word is produced correctly. In some cases the entire word is produced incorrectly. Neologisms are also common in the speech of schizophrenics. An example from Freud would be “Vutter” for “Vater” or “Mutter”

Verbal paraphasias are those in which another word is substituted for the target word. (The substitution must be a real word. If it is not, the paraphasia is classified as neologistic). This is also called semantic paraphasia, where the substituted word is related in meaning. An example from Freud would be: “Potsdam” for “Berlin” “

The above three types are from Neuroscience on the Web Series SPPA 336, Neuropathologies of Language and Cognition, CSU Chico, Patrick McCaffrey PhD
The first type, Literal/phonological paraphasia corresponds to the rhetorical forms of: *Prosthesis, Epenthesis, Proparalepsis*, (adding a syllable or letter at the beginning, middle, or end of a word, respectively) or *Aphaeresis, Syncope, Apocope* (omitting a syllable or letter at the beginning, middle, or end of a word, respectively) or *Metathesis*, an exchange of letters in a word.

The second type, neologistic paraphasias, has no rhetorical counterpart as far as I know, unless one could call it a variation of *solecismus*, the ignorant misuse of cases, genders, and tenses.

The third type, verbal paraphasias corresponds to the rhetorical form of *metaphor*, substitution of one word for another often to suggest some sort of likeness between them.

Another topic of interest might be the question: is there is a relationship between Broca’s aphasia, (a continuity or metonymic speech disorder) and the Lacanian concept of holophrase (the holophrase is a word that expresses the meaning of a whole sentence or several sentences or the lack of separation between S₁ and S₂)?

The following books are very helpful in reading Freud’s *On Aphasia*:
Luis Riebel’s *Brain, Mind, and Language—Freud on Aphasia*, pages 81-97 of Papers of the Freudian School of Melbourne, Vol. 10: *Formations of the Unconscious*, Melbourne, 1989, which is now available on line: https://www.fsom.org.au/papers/?fbclid=IwAR3WBvenD4gO3gHJW2Zt6ZX6EdPDsikucoxOwf74vu_MLdIoPb3I-Ck-BY

Richard G. Klein
Summer 2020-- revision of my September 2002 Intro.
New York City
The term *aphasia* was coined by Armand Trousseau in 1864. (Valerie Greenberg Freud and His Aphasia Book)

**Aphasia** (ăf'zhə-ə). Path. [mod.L, a. Gr. ἀφασία, n. of quality f. ἀφατος speechless, l. d priv. + φανεροποιεῖν to speak (cf. φανερός speech).] Loss of the faculty of speech, as a result of cerebral affection.

1867 Chamb. J. 38. xxviii. 85 A musician, the subject of aphasia, who had lost the ability to read and write as well as to speak. 1878 A. Hamilton Nerv. Dis. 72 Embar- rassment of speech may vary from simple awkwardness of articulation to decided aphasia.

**Aphasic** (ăf'žik), a. and sb. [f. APHASIA + -IC.] Suffering from aphasia, having lost the power of speech.

1867 Chamb. J. 38. xxviii. 86 Most aphasic patients answer very well by signs. 1880 Bastian Brain xxix. 649 He had regained the power of speaking to a considerable extent, and now he had become Amnesic rather than Aphasic.

B. sb. = APHASIC (which is more analytical).

1867 Chamb. J. 38. xxviii. 85 We must... turn our attention to the writing of aphasics. 1869 Hunt in Eng. Mech. 7 May 147/l, I call him an aphasic in whom the signs of thought cannot manifest themselves.

Афазия, ἄφατος) speechlessness, caused by fear or perplexity, ἔπειται διὰν ἀφατον τε προστεθείς E.Hel.549; ἄ κτει id.Id.4837, cf. Ar. Th.994; ἄ ἀκτει λεξιγένει τοῦτο χρῆ λέγειν Pl.Lg.636e; els ἄ πώλ ἐμβαλλείν id.Philb.21d; els ἄ ἐμβαλλείν πράγματος inability to say anything about it, S.E. 2.111, cf. Dam.Pr. 7.

άφατος, ov, not uttered or named, nameless, ἄδερε διάκρισιν τε διάφορον τε Hes.Op.3. 2. unutterable, ineffable, λόγος E.Jon 7.82 (lyr.); ἄντα ἐγένος, Pl.N.1.47; κεφαλικός APl.112 (Pers.); ἐρμήματα ἐνιοῦδες, Hdt.7.190; ἀνέπότα, τιτόπα ἐπίνεος, S.Otv.314 (lyr.), OC.164 (lyr.); ἐνεργείαν ἀφατον (Schw. for —ταν) ἐμβαλείν Anaxandr.4.13; τάξαν ἀφατον τάξας IG.14.301.24 (Sulp. Max.); ἤποιηθή Phld.D.3.14; ἐπερ-βολῇ δυνάμεως Hermog.1nv.1.4; δύναμεις Plot.4.8.6; ἀφατον ἀς... there's no saying how... i.e. marvellously, immensely, Ar.Av.418, Ly.198. Adv. —τον Dsc.1.13.

(1891b) Zur Auffassung der Aphasien. Wien 1891. [Inhaltsangabe in (1897b).]

(1893–94a) Accessoriuskrampf; Accessoriuslähmung; Agraphie; Agraphie; Alexie; Amnesie; Anarthrie; Anosmie; Aphasie; Aphasic; Bradylalie; Bradyphrasie; Dysgraphie; Dyslalie; Dyslexie; Dysphasie; Echolalie; Paraphrasie [signierte Artikel], in: Diagnostisches Lexikon für praktische Ärzte, hrsg. von Anton Bum und Moritz T. Schnirer, Bd. 1 (1893) und Bd. 3 (1894), Wien und Leipzig. Nachdruck der Artikel Amnesie und Aphasie in: Köstle, Oswald Ulrich, Einige bisher unbekannte Texte von Sigmund Freud aus den Jahren 1893/94 und ihr Stellenwert in seiner wissenschaftlichen Entwicklung, Psyche, Bd. 41 (1987), S. 508–528. Vgl. Fichtner (1987).

(1897b) Inhaltsangaben der wissenschaftlichen Arbeiten des Privatdozenten Dr. Sigm. Freud (1877–1897), Wien 1897. G.W., Bd. 1, S. 461–488; S.E., Bd. 3, S. 227–257. FREUD'S ABSTRACTS OF HIS WORKS (1877–97)

Freud's abstract of that work (1897b, j. 240). XIX On the Interpretation of the Aphasias, a critical study. [1891b.]
On the Interpretation of the Aphasias, a critical study. [1891a]

After a firm basis for the understanding of cerebral disturbances of speech had been established by the discovery and definite localization of a motor and a sensory aphasia (Broca and Wernicke), the authorities set about tracing the more subtle symptoms of aphasia as well as factors of localization. In this way they arrived at the hypothesis of a conduction aphasia, with subcortical and transcortical, and motor and sensory forms. This critical study is directed against this view of speech-disorders and it seeks to introduce for their explanation functional factors in place of the topographical ones. The forms described as subcortical and transcortical are not to be explained by a particular localization of the lesion but by conditions of reduced capacity for conduction in the apparatus of speech. In fact there are no aphasias caused by subcortical lesion. The justification for distinguishing a central aphasia from a conduction aphasia is also disputed. The speech area of the cortex is seen rather as a continuous region of the cortex inserted between the motor fields of the cortex and those of the optic and auditory nerves—a region within which all communication and association subserving speech function takes place. The so-called speech-centres revealed by the pathology of the brain correspond merely to the corners of this field of speech; they are not distinguished functionally from the interior regions; it is only on account of their position in relation to the contiguous cortical centres that they produce more obvious signs when they become disordered.

The nature of the subject treated here called at many points for a closer investigation of the delimitation between the physiological and the psychological approach. Meynert's and Wernicke's views on the localization of ideas in nervous elements have had to be rejected and Meynert's account of a

[This book is generally regarded as Freud's most important contribution to neurology. (An English translation by Professor E. Stengel appeared in 1933.) Freud had lectured on aphasia some years before (in 1886 and 1887) and contributed an article on it to Villaret's encyclopaedia of medicine (1886b).]
representation of the body in the cerebral cortex has required revision. Two facts of cerebral anatomy, namely (1) that the masses of fibres entering the spinal cord are constantly diminished on passing upwards, owing to the interposition of grey matter, and (2) that there are no direct paths from the periphery of the body to the cortex—these two facts lead to the conclusion that a really complete representation of the body is present only in the grey matter of the cord (as a 'projection'), whereas in the cortex the periphery of the body is only 'represented' in less detail through selected fibres arranged according to function.
Aphasie {a privative} – φασις speech (φομι) (Fr. aphasie f; Engl. Aphasia, Aphasy; It. afasia f, alalia f); synonyms: aphemia, alalia—word deafness, word blindness—agraphia. By the term aphasie, one understands the abolition or impairment of the ability to express one's thoughts through conventional signs, or to understand such signs, despite the continuance of a sufficient degree of intelligence and despite the integrity of the peripheral sensory, nervous, and muscular apparatuses that are involved in the expression or comprehension of speech. Deaf-mutism, the speechlessness of idiots, the loss of speech in coma as well as through paralysis of the tongue and lips, therefore, do not fall under the concept of aphasie. Aphasie is a psychical illness, but it must be firmly grasped that it is not necessarily linked with intellectual disturbance; the latter is to be taken as a complication every time. One distinguishes between natural or emotional speech (gestural speech) and artificial or articulate speech, of which the latter succumbs to disturbances more frequently because it is acquired later. — The
manifold disturbances of articulate speech (true aphasia as opposed to amimia) only become comprehensible if one appreciates the following reflection on the normal course of speech: A 'word' is not a simple idea, but a complex that consists of four elements, two sensory and two motor. The two sensory [elements] are: the mnemonic image for the heard word (the auditory presentation) and the optical image for the seen word (in script or print). The two motor [elements] are: the movement presentation (of the speech instruments) for the spoken word and the movement presentation (of the right hand) for the written word. The second and fourth of these components only play a role in the educated. Speech is learned by way of hearing. Besides this, the connections that link the four elements of the word presentation with the idea of the object must be taken into consideration. Accordingly, there are two principal types of aphasias, motor and sensory, and four pure forms, namely, word deafness, word blindness, motor aphasia (aphemia), and agraphia. These pure forms are encountered now and then in the clinic, but complex speech disturbances in which all four aspects of the speech function have suffered to varying degrees are much more frequent. The part of the brain in which the material of speech presentations is connected, and where disease therefore leads to speech disturbance, is the island of Reil, with its surrounding convolution, which stretches from the frontal to the temporal ends of the hemisphere as the first frontal convolution, base of the central convolutions, inferior parietal lobule, and first temporal convolution. The speech field is therefore partially situated in the depths, and partially in the borders of the Sylvian fissure. It is not developed in both hemispheres, however; in most (right-handed) people it is the left, and in others (left-handers) it is the right hemisphere, which contains the speech field. There are individual areas in the speech field of the left hemisphere, the injury of which produces a pure form of aphasia. These carry the—incidentally misleading—name of centres. Their precise circumscription is impossible at present. Thus the 'centre' for motor speech capability lies in the posterior part of the first hemisphere, however; in most (right-handed) people it is the left, and in others (left-handers) it is the right hemisphere, which contains the speech field. There are individual areas in the speech field of the left hemisphere, the injury of which produces a pure form of aphasia. These carry the—incidentally misleading—name of centres. Their precise circumscription is impossible at present. Thus the 'centre' for motor speech capability lies in the posterior part of the first hemisphere, however; in most (right-handed) people it is the left, and in others (left-handers) it is the right hemisphere, which contains the speech field. There are individual areas in the speech field of the left hemisphere, the injury of which produces a pure form of aphasia. These carry the—incidentally misleading—name of centres. Their precise circumscription is impossible at present. Thus the 'centre' for motor speech capability lies in the posterior part of the first
frontal convolution (Broca’s area), the centre for writing capability in the posterior part of the second frontal convolution; the cortical region upon the integrity of which the understanding of heard speech depends is the first temporal convolution, and the corresponding cortical region for read speech signs is the inferior parietal lobule. Remarkably, these ‘centres’ for speech are the most outlying districts of the speech field and border directly on the centres of other functions (the tongue and lips, the arm, hearing and sight in general) whereas lesions lying between the centres of the speech field still seem to produce complex speech disturbances. The so-called centres for speech are therefore probably merely the radiation areas of association bundles that reach the speech field from other regions. The four pure forms of aphasia present clinically in the following fashion:

I. The sensory aphasias

(a) Word deafness. The patients no longer understand what one says to them, despite the preservation of hearing and good intelligence. To them speech sounds like a confused noise, though the vocabulary that the patients make use of themselves is unrestricted. With word deafness, however, one does nearly always find motor speech disturbance, so-called paraphasia, which consists in the patient’s use of inappropriate words for expressing his thoughts without realizing it. This paraphasia can go so far that the patient’s speech becomes entirely senseless and that patients are perceived as mentally disturbed.

(b) Word blindness (or preferably, writing blindness). The patients are not able to recognize the meaning of written or printed speech signs that they see very well; otherwise they have good speech ability. Therefore they cannot read (alexia) or they read with the help of a trick whereby they trace single seen, but unrecognized letters. This ‘blindness’ sometimes applies only to syllables, while single letters

Stirnwindung (Broca’sche Stelle), das Zentrum der Schreibfähigkeit im hinteren Teil der zweiten Stirnwindung; der Rindenort, von dessen Integrität das Verständnis der gehörten Sprache abhängt, ist die erste Schläfenwindung, der entsprechende Rindenort für die gelesenen Sprachzeichen das untere Scheitelläppchen. Diese „Zentren“ der Sprache sind bemerkenswerterweise die äussersten Bezirke des Sprachfeldes und stossen an Zentren anderer Funktion (der Zunge und Lippen, des Armes, des Gehörs und Gesichtes überhaupt) direkt an, während Läsionen des zwischen den Zentren gelegenen Sprachfeldes stets komplexe Sprachstörungen zu ergeben scheinen. Die sogenannten Zentren der Sprache sind also wahrscheinlich blosse Einstrahlungsstellen der von anderen Gebieten zum Sprachfeld kommenden Assoziationsbündel. — Die vier reinen Formen der A. charakterisieren sich klinisch in folgender Weise:


b) Die Wortblindheit (besser Schriftblindheit). Die Kranken sind bei sonst gatem Sprachvermögen nicht imstande, die Bedeutung geschriebener oder gedruckter Sprachzeichen, welche sie sehr gut sehen, zu erkennen. Sie können daher nicht lesen (Alexie), oder sie lesen mit Hilfe eines Kunstgriffs, indem sie die einzelnen gesehenen und nicht erkannten Buchstaben nachzeichnen. Diese „Blindheit“ besteht manchmal nur für Silben, während einzelne Buch-
are still recognized; at other times it applies to letters as well. Here it is not rare that numbers are still recognized. Word blindness is nearly always complicated by unilateral restriction of the visual field (hemianopsia).

II. The motor aphasia/s

(a) Actual motor aphasia (aphemia). This is by far the most common speech disturbance and is mostly encountered in [its] pure form. It is characterized by abolition or diminution of the vocabulary. In extreme cases the patient only has gestures, in others only individual syllables or words or even entire phrases, with which he answers everything. He is, however, very well aware of the insufficient character of these expressions and is visibly hurt by his inability to say more than his remaining stereotypical phrases. The speech remnants of the aphasic frequently have the character of interjection—"Yes", "No"—or they consist of individual syllables, 'tan-tan', and senseless combinations of these, like 'akoko', 'monomentive', eventually combined into entire, simply constructed sentences. All this speech is correctly articulated, which strictly distinguishes aphasia from paralysis of the speech instruments (alalia). If the reduction of the vocabulary is not severe, it mainly affects nouns and expresses itself insofar as the patient tries to transcribe them by indicating the actions. So, e.g., instead of 'Give me my hat', he says, 'Give me that which one puts on the-'. The speech capability of an aphasic fluctuates with his general condition, however, and under the influence of excitement he can frequently experience a sudden deterioration. It is to be noted further that many aphasics who are unable to articulate a specific word themselves, i.e. through their own train of thought, can repeat it if it is said to them first. This depends upon which of the manifold association pathways for speech are destroyed or preserved and finds its analogy in other forms of speech disturbance.—Motor aphasia frequently, but not necessarily, coexists with paralysis of the
right-sided extremities or with paralysis of the cerebral pathways that govern the tongue, lips, laryngeal, and pharyngeal muscles. It is common to find motor aphasia in the first days after a left-sided apoplectic insult and for as long as the entire hemisphere continues to suffer under the consequences of the insult. Usually the speech disturbance soon diminishes. In such cases it can be perceived as an indirect focal symptom.

(b) *Agraphia* can be designated as 'aphasia of the hand' after a fortunate expression of Charcot's. Agraphia is found comparatively rarely in pure form. If a right-sided paralysis is present, the question of the presence of agraphia is naturally left undecided. Otherwise agraphia accompanies motor aphasia as a rule but does not necessarily keep pace with it. It consists of the patients putting only senseless and disconnected strokes together when instructed to write. Now and then such patients can also still write properly to dictation or copy from a model, whereas they are unable to write without such a stimulus.—One must analyse the more complicated aphasic disturbances through careful investigation in such a way that one can ascertain which connections between the individual elements of the word presentation, and between these and the idea of the designated object, are preserved or interrupted. Because aphasia is an exquisite focal symptom, any lesion that affects the speech field can give rise to it; thus, brain haemorrhages, softenings, tumours, traumatic influences, abscess, etiological factors like heart and vascular disease, syphilis, morbus Brightii, acute infectious diseases (typhus, variola), diabetes melitus, etc., as with the other forms of brain disease, come into consideration here, without the one or the other contributing a particular causal relationship to the form of aphasia. The latter rather depends solely on the localization and extent of the established lesion in the speech field.—Aphasia is not always the consequence of a material brain process; rather, neuroses like hysteria and neurasthenia may also produce aphasic disturbances. Hysterical aphasia is purely motor as far as it


has been studied until now. However, it is characterized by its completeness or, rather, by its absolute character. It is not that the patients are restricted to the use of individual words, but that they are completely speechless, indeed voiceless; not a sound, not a cry comes about. Hysterical aphasia is therefore actually a 'mutism'. Writing capability is always preserved and increased, however. When questioned, these patients point at their mouths, then put pen to paper and write their thoughts down with unusual rapidity and certainty.—The speech disturbance resulting from neurasthenic brain-fatigue is limited to the forgetting of individual concrete words and to the confusion of similar-sounding words in speech and is thus similar to the paraphasia that occurs in healthy people.¹⁰—The prognosis and therapy for aphasia is the same as that for a paralysis and is directed to the basic complaint. Under favorable circumstances all forms of aphasic disturbance are capable of improvement or compensation. If a persistent defect of speech ability remains behind, then one can attempt to ameliorate it through the re-education of the patient. Thus the word blind (patient) learns, even if laboriously, to read again, and the agraphic learns to write again. The latter can make use of the left hand to learn to write, either in mirror-writing or in normal characters. Preserved intelligence is a precondition for such learning attempts.

EDITORS' ANNOTATIONS
1. Loss of the power to communicate by means of gesture or non-speech signs.
2. This and the following three sentences are relevant to the determination of the authorship of this paper. They read as a précis of Freud (1891), pp. 73ff. Freud's view that the word comprises a complex of four rudimentary ideational elements can undoubtedly be attributed to Charcot, under whom Freud studied in 1885–86; cf. Freud's translation of Charcot's *Leçons sur les maladies du système nerveux* (Charcot, 1886, p. 155).
3. Freud (1891b, p. 67) identifies this same continuous cortical area as the 'speech field.'
4. The localization of a unitary centre for this function was never 'definitely established', but Freud later (1891b, p. 63) identified the same region in this context.
5. The important point made in the last two sentences is more fully stated in *On Aphasia* (Freud, 1891b, pp. 62–64) and restated in Freud's abstract of that work (1897b, p. 240).

### Notes
6. This pseudo-reading manoeuvre is known as 'Wilbrand's sign'.
7. This was a recurrent utterance of Broca's famous (1861) patient, Leborgne. He came to be known by the name of 'Tan' by the Bicêtre hospital staff.
8. Freud's (1891b, p. 77ff) psychology of language and classification of the aphasias followed this exact scheme.
9. This claim was also made in the 'Dora' case study (Freud, 1905a [1901], p. 39) and in the 'Hysterie' article in Villarceau's dictionary (Freud, 1888b3, p. 47). The more general remarks on hysterical aphasia were also restated there and again later (Freud, 1893c, pp. 163, 164, 169).
10. Freud made a very similar point in *On Aphasia* (1891b, p. 13) and again in *The Psychopathology of Everyday Life* (1901b, p. 53); (cf. Stengel, 1953, p. viii).
11. This method was based on the traditional assumption, made in accordance with the teaching of others, that the intact right hemisphere can take over the speech function of the damaged left one in right-handed individuals. Freud did not question this teaching (see Freud & Rie, 1891a, p.126).
Diagnostisches Lexikon

für

PRAKTONISCHTÉ ÄRZTE

UNTE MITWIRKUNG DER HERREN


HERAUSGEBERN

von

Dr. ANTON BUM UND Dr. M. T. SCHNIRER

REDACTIUN DER WIENER MEDIZINISCHEN FRESSE

MIT 247 ILLUSTRATIONEN IM WOLSCHNITT

ERSTER BAND

Abasle — Ephemera

WIEN UND LEIPZIG

URBAN & SCHWARZENBERG

1893
Aphasia: (συναισθήματα, 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 deafmute (unless he has been taught to 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 them from genuine aphasia; it is discussed under anarthria. 

Since diagnosing aphasia requires judging a complicated psychological function, a few introductory remarks from this writer's study "On Understanding Aphasia" ["Zur Auffassung der Aphasien"] may not be superfluous. 

The speech function is an associative function acquired with a great deal of effort; it brings together psychological 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 results 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 image 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 latter; 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

![Diagram showing associations between words and objects](image)

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 asymtontic speech disorder (a case in HeinBNER). Aglossic 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 isid 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) the area where a lesion causes agraphia (area bordering the center for the hand); 2) Wernicke's area, where a lesion causes motor aphasia (adjointing the centers for speech and larynx); 3) Wernicke's area, where a lesion causes word deafness (adjointing the field for acoustic nerve or a 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

Bisher haben wir die Sprachfunction psychologisch betrachtet, wir wenden uns nun zur anatomisch-klinischen Erorterung der Sprachstörungen. Sectionsefunde haben uns gelehrt, dass die Verbalassociationen der Sprache sich innerhalb eines bestimmten Gebietes der Hirnrinde vollziehen (Associationsfeld der Sprache), welches nur in der linken Hemisphäre ausgebildet ist, ferner sind wir im Stande, ungefähr die Gegend der Hirnoberfläche anzuzeigen, deren Läsion eine rein asymptomische Sprachstörung verursacht (ein Fall von HeNBNER). Agonogue Sprachstörungen kommen dagegen nur zu Stande, wenn ausgedehnte Läsionen in beiden Hemisphären vorhanden sind, wobei das Sprachfeld selbst ganz intact sein kann. Im Sprachfeld der linken Hemisphäre, dessen Ausdehnung aus Figur 24 ersichtlich ist, haben nicht alle Stellen gleiche Bedeutung:

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 revolving areas.

The diagnostic task for a case of aphasia is two-fold: First, the symptomatology 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
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
are fully absent. The foregoing is found only in the rarest of cases; in general, all individual
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 which have 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 when it
is 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
forth. Clearly, this
that the number of
So forth. Clearly, this
he never repeats two or more words using the same innervation, that the number of
attempts to speak is the same as the number of syllables in the words he attempts, and so
intended meaning. He frequently makes mistakes, uses a word close to the meaning or sound
for 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 absentmindedness. Further observation of the same patient will reveal a second
symptom belonging to aphasia: amnesia (see above).

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symptom belonging to aphasia: amnesia (see above).
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 be nonsensical.
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, careless 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 agaphia (see above), though they do not exhibit paralysis of the right hand.

The remarks above have completed the topical diagnosis of aphasia. The more pronounced the verbal impoverishment, the more we must shift the lesion to the frontal

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

In the case of more serious lesions, the patient continues to speak through circumlocution, careful word choice and slow speech. With relative frequency, patients are able to conceal their speech deficiency though they do not exhibit paralysis of the right hand.

The third type of aphasia, optic aphasia, is characterized by a disturbance of the understanding of letters and an inability to read.
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 overwhelming
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
1. spontaneous
2. a) after hearing (repeating)
   β) after seeing (reading)

II. Writing
1. spontaneous
2. a) after hearing (dictation)
   β) after seeing (copying)

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
Verbal assoc.
1. reading out loud
2. copying
Symbol assoc.
3. understanding read material

IV. Hearing
Symbol assoc.
1. repeating speech
2. writing down
Symbol assoc.
3. oral comprehension (direct)
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
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 region creates dysarthric disorders, and when a case of
motor aphasia is accompanied by symptoms of anartria, 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.

an's frontale Ende des Sprachfeldes zu verlegen, jo mehr die Störung des Sprach-
verständnisses sich ausprägt, desto sicherer ist dieselbe im temporalen, und bei
deutlicher Störung im Verständnis der Buchstaben am parieto-occipitalen Ende
des Sprachfeldes zu suchen. Die topische Diagnose wird häufig noch durch die
discrepanz in den Symptomen bestätigt. So kann sich neben der motorischen A. rechtseitig
seitlicher Facialisparese oder halbseitige Körperlähmung die unbestimmte A.
kann von einer cerebralen Hemiaesthesie begleitet sein, die optische A. in reinster Form
(Alexie) findet sich überwiegend häufig neben einer rechtseitigen Hemianopsie.

Bei der akustischen A. ist darauf zu achten, ob nicht partielle Taubheit, halbseitige Einschränkung des Gehörs, Verlust der Wahrnehmung für gewisse Arten von Tönen gleichzeitig vorhanden ist.

Andere Symptome der A. bieten kein diagnostisches Interesse, erscheinen aber der Untersuchung wertig, weil sie ein Bild von der Selbständigkeit und Abhängigkeit der einzelnen Sprachleistungen, von deren Dissociation und von der Möglichkeit vicariierender Leistung geben. Will man sich eine Übersicht von diesen Verhältnissen verschaffen, so prüfe man jede der motorischen Sprachleistungen, inwieweit sie spontan und auf sensiblen Anreiz erfolgen kann. Man hat dann etwa folgendes Schema:

I. Sprechen
1. spontan
2. a) nach dem Gehörten (Nachsprechen), b) nach dem Gesehenen (Vorlesen).

II. Schreiben
1. spontan,
2. a) nach dem Gehörten (Dictat-
   b) nach dem Gesehenen (Vorlesen).

Es gibt dann die Regel, dass man zwar häufig eine Erhaltung der motorischen Sprachleistung auf sensiblen Anreiz (2a und 2b) bei Verlust der spontanen (1), aber niemals das Umgekehrte.

Bei der sensorischen Sprachleistung, die ja immer auf sensiblen Anreiz erfolgt, hat man die Verbalassociation und die Symbolassociation besonders zu prüfen.

III. Lesen
Verbalass. 1. Lautlesen,
2. Abschreiben,
Symbolass. 3. Verstehen des Gesehenen,
4. Verstehen des Nachgesprochenen,
5. Verstehen des Nachgeschriebenen.

IV. Hören
Aufsprechen,
1. Nachsprechen,
2. Nachschreiben.

Es kommt hier Fälle vor, dass die Verbalassociation noch möglich ist, die Symbolassociation aber nicht mehr. Wie man sieht, können die meisten Sprachleistungen als Associationen durch mannigfachen Sitz der Läsion gestört werden.

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 hemianopia, 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 (see below).

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 (see below) which are not the result of acquired diseases of the speech field in the cerebral cortex, are not covered here.

Freud.
Questions of terminology raise the question of translation, which is particularly problematic in the case of the aphasia book. Erwin Stengel’s translation made the book accessible to an English-speaking public but at the cost of accuracy to Freud’s text. Words, phrases, sentences are left out, and the final result, though careful on points of neuroanatomy, is in toto impressionistic, if not at points misleading. Thus I have found it necessary to provide my own translation of quoted passages, with the exception of the several pages translated by Strachey and published in the *Standard Edition*. More extensive quotation becomes necessary toward the end of my book, and I am hoping that by providing a version one stage closer to Freud’s text, I have assisted any future, improved translation of the whole.

Let me mention two particularly difficult challenges to translation which are also central to the aphasia book. One is the unresolvable conflict between “speech” and “language.” Both are rendered by *Sprache* in German, and it is difficult in individual instances to decide which Freud meant when he used *Sprache* and its compounds. Obviously, the distinction is important. My best judgment has led me to use “language” in cases where others have used “speech,” because I believe Freud had the more encompassing meaning in mind. The result can only be informed speculation. Another challenge is the German term *Vorstellung*, translated as “presentation” in the *Standard Edition*. In addition to being particularly amorphous and mutable according to context, *Vorstellung* entails a complex of historical and philosophic meanings that make it impossible to find one English equivalent. It has been appropriately translated into English as, among other things, “representation,” “image,” “idea,” “notion,” and “concept.” Strachey’s “presentation” is a less suitable translation for the aphasia text. Freud’s use of the term changes with context, so I have tried to indicate that while at the same time leaving the word in the German original or providing it parenthetically.

It is fortunate that the S. Fischer Verlag published its new German edition of *On Interpretation of the Aphasias*, edited by Ingeborg Meyer-Palmíedo (who continued the work begun by Paul Vogel). This is an invaluable resource in view of the extreme scarcity of the original edition. There are, however, some differences from the original: spelling has been modernized; paragraph indentations have been removed; page numbering is changed; and Freud’s running page titles have been deleted, although they are listed in the appendix. Thus the text has a different appearance from Freud’s, and the reader is deprived of the possibility of following along with Freud’s sense of the most important point on each page. In the bibliography there are several errors in page references to the text. Leuschner’s excellent introduction, the helpful notes, and the appendix of sources outweigh these minor failings. Nevertheless, it remains necessary for research purposes to look at the original.

**John Gach Books** Catalog 166 Part 2: Freud’s Original Writings

24. 1891 Zur Auffassung der Aphasien: eine kritische Studie. Leipzig/Wien: Franz Deuticke, 1891. [iv]+107+[1]pp. + 22 pages of inserted illustrated front ads foliated from page 3 and dated January 1891. 10 text woodcuts. Original printed green-gray wrappers. Edges a bit chipped, slight staining to spine and joints, small label removed from lower gutter of front wrapper, VG. Rare. Housed in a specially made cloth drop box with leather spine label. Rare in any condition and of the greatest rarity in original wrappers. We know of only one other copy with the ads intact. Grinstein #10352; GFB 2; NC F16 (this copy). HN’s copy with bookplate to the verso of the front cover. $6000.00

Freud’s first book other than translations and his first psychological book. 850 copies were printed, of which 257 were sold, with the remainder being destroyed. One of the great rarities in Freud collecting (we have had only two other copies in twenty-six years).
**CROSS REFERENCE**
for the three editions of the
Aphasia book

<table>
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<th>English OA 1953</th>
<th>German AA 1992</th>
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Zur Auffassung der Aphasien

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INTRODUCTION

Freud's book on aphasia is known to a small circle of experts only. Unobtainable for many years it has until recently been regarded as no more than an item on the list of Freud's "pre-psychoanalytical" publications and of no relevance to his later work. Freud himself, having turned to the study of the neuroses, hardly ever referred to his earlier writings. And yet, the period during which they originated was one of intensive and fruitful activity. Not only did Freud make valuable contributions to neurology but he laid the foundations of psychoanalysis. It has gradually been recognized in recent years that his anatomical, neurological and psychoanalytical works form a continuum. The book on aphasia demonstrates this clearly.¹ It

¹ In this introduction I have drawn on the writings of the following authors who discussed the significance of the book from various points of view:


I am indebted to Dr. Ernest Jones for the loan of a German copy of Freud's book and of a copy of Dorer's book.

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was the first of the author's studies dealing with mental activities and thus provides a link between the two apparently separate periods in his workinglife. It is worthy of serious attention to-day no less than it was sixty-two years ago. The neurologist will find it not only historically interesting, but also full of stimulating and original ideas concerning problems which are as topical at present as they were then. The psychoanalyst and psychiatrist will recognize it as the most significant forerunner of the author's later work.

As a contribution to the problems of the speech disorders due to brain lesions Freud's treatise stands out among the voluminous writings of the time. It appeared when neurologists were intensely preoccupied with the localization of cerebral functions. The discoveries of Hitzig and Ferrier were fresh in their minds. Broca and Wernicke had established the relationship of certain brain lesions to specific types of aphasia, and the exact localization of all the functions of speech seemed to be within easy reach. Freud was the first in the German speaking world to subject the current theory of localization to a systematic critical analysis. In challenging both a powerful scientific trend and its most influential representatives he showed himself an independent thinker of considerable courage.

Freud had been stimulated to the study of the subject by a paper by Exner and Paneth, this was a report on a piece of experimental research, by which the two physiologists were able to demonstrate that surgical isolation of a cortical area in dogs had the same effect as its excision. They attributed this to two factors: the cutting of the association fibres and "traumatism", thus invoking a localized lesion as well as a functional disturbance. The same combination was considered by Freud to be responsible for the peculiarities of certain types of aphasia. There can be little doubt that his contact with Charcot also contributed to the choice of the subject. But these were only accidental factors; it was almost inevitable for a neurologist with Freud's profound interest in mental processes to be attracted to the study of the aphasias.

Although the book is in many respects a period piece, it still has a message for the neurologist of to-day. Freud's insistence on the compatibility of the functional, i.e., dynamic, with the localizatory approach is still insufficiently heeded by many. In his view on localization he followed Hughlings Jackson. He rejected strict localization not only for the function of speech but also for individual muscles. However, the "speech apparatus" which, although not identical with the structural substrata of speech is in some way related to the latter, is a Freudian concept. The differentiation between a central speech area and the so-called speech centres bordering on the receptive and motor cortical areas functionally related to them, is a most interesting theory which has proved very fruitful.

The proposed division of the aphasias into three groups was a bold attempt at establishing a consistent psychological system based on the theory of associations applied to speech. Considering that the current classification was then, and still is, a confusing mixture of anatomical, physiological and psychological concepts, Freud's system had much to recommend it. However, it was too closely linked to a questionable theoretical framework to be acceptable to the clinicians, though part of it has survived. It was a forerunner of Head's classification which was also based on psychological criteria.

None of the leading authorities in the field of aphasia escaped Freud's criticism, with the notable exception of Hughlings Jackson for whom the author had nothing but praise and whom he pronounced his guiding spirit in the study of the speech disorders. He also appreciated Bastian's contributions, without however accepting his views on physiological speech centres. He quoted Jackson's warning against the confusion of the physical with the psychic and declared himself an adherent of the "Law of Concomitance" adopted by Jackson. He cited some of Jackson's most illustrative examples of "recurrent utterances" in the origin of which emotional factors had played a conspicuous part; in order to underline the importance of these factors in situations of stress he related an interesting self observation.

It is obvious that Hughlings Jackson had made a deep impression on Freud. The following passage (p. 87) shows how fully he had made Jackson's basic doctrine of the evolution and dissolution of function his own: "In assessing the functions of the speech apparatus under pathological conditions we are adopting as a guiding principle Hughlings Jackson's doctrine
that all these modes of reaction represent instances of functional retrogression\(^1\) (disinvolution) of a highly organized apparatus, and therefore correspond to previous states of its functional development. This means that under all circumstances an arrangement of associations which, having been acquired later, belongs to a higher level of functioning, will be lost, while an earlier and simpler one will be preserved. From this point of view a great number of aphasic phenomena can be explained\(^1\).

Here, then, we find for the first time in \textit{Freud's} writings the principle of regression which underlies all the genetic propositions of psychoanalysis. \textit{Freud} had probably come across this principle in some form or other earlier, possibly in \textit{Meynert's} writings; but nowhere had it been stated so clearly and its applications to psychopathology been pointed out so persistently as in the writings of \textit{Hughlings Jackson}, who himself had adopted it from \textit{Herbert Spencer}, the philosopher-psychologist of evolution. The close relationship of psychoanalysis to the theory of evolution was noted by \textit{Ernest Jones} long ago.\(^1\)

The important role played by \textit{Freud's} study of the aphasias in the foundation of psychoanalytic theory has been fully recognized by \textit{L. Binswanger}. He believes that, by acquainting \textit{Freud} with \textit{Hughlings Jackson's} genetic doctrine, it had a decisive influence on \textit{Freud's} thinking, and he goes so far as to state that without knowledge of this book a full historical understanding of \textit{Freud's} teachings is impossible. A study of the two articles by \textit{Hughlings Jackson} referred to by \textit{Freud} will convince the reader that \textit{Binswanger's} contention is not an overstatement. In these papers \\textit{Jackson} not only applied \textit{Spencer's} doctrine to the speech disorders but also adumbrated their importance for the study of "insanity". He also expressed the view that certain psychic states and utterances were the results of conflicting nervous discharges. All this must have been of absorbing interest to \textit{Freud} who was acquainted with concepts of psychodynamics through \textit{Herbert}, \textit{Fechner} and \textit{Brücke}.

The idea that disturbances of function similar to those caused by brain lesions occur in the healthy person under conditions of fatigue and lack of attention, was implicit in the theory of evolution and dissolution. It was to prove of far-reaching importance in psychopathology. It is therefore not surprising to find observations in this book which foreshadowed important psychopathological discoveries. What \textit{Freud} said about paraphasia, i.e. the mistaken use of words, reads like a prelude to the chapter on errors and slips of the tongue in "Psychopathology of Everyday Life". \textit{Freud's} observations on paraphasia are still up-to-date. This crucial problem of aphasia has hardly been advanced since.

The "speech apparatus" is the elder brother of the "psychic apparatus" to the working of which most of \textit{Freud's} later researches were devoted. Both terms obviously have their origin in \textit{Meynert's} writings. They demonstrate \textit{Freud's} lasting attachment to physiological concepts.

The book contains a number of other terms which have become household words in psychology and psychiatry. "Projection" and "representation" which were to play such an important part in psychoanalytical theory, are here used in their original sense. The term "Besetzung" and "besetzen" (occupation, occupy; catheasis, cathexis) had been used by \textit{Meynert} for the hypothetical process of the investment of unused cortical cells with new function. Although \textit{Freud} rejected \textit{Meynert's} hypothesis he later used these terms for the mechanism of the investment of objects with libido.

The concept of "over-determination" also was defined for the first time in relation to functions of speech which were supposed to be safeguarded against breakdown by a multiplicity of complementary mechanisms.

\textit{Freud's} preference for concepts implying dynamic processes rather than static conditions is conspicuous throughout the book. It is most clearly expressed in the remarkable passage concerning memories (p. 56). Considerations of this kind must have played their part in the discovery of unconscious mechanisms which was to become \textit{Freud's} most important contribution to psychology and psychiatry.

The book seems to have received little immediate attention
and its sale was disappointing. The author himself regarded this work with some pride and in one of his letters he spoke of it as something "really good", complaining at the same time that it was hardly taken notice of. This was not surprising; Freud occupied no official position such as was held by those whose theories he criticized so severely. He had not written about aphasia before nor did he pursue the subject. Besides, the book did not contain new clinical observations and was published as a monograph which soon went out of circulation. Possibly the fate of this study would have been different had it been published in one of the leading journals. However, it was not long before the tide of narrow localization theories subsided, and in the first decade of this century Freud's ideas were taken up by some students of aphasia. Storch based his interesting theory of inner language on them. He was followed by Kurt Goldstein who went back to Hughlings Jackson and Freud in evolving the most consistent and fruitful modern concept of aphasia. His differentiation of the central aphasias from the speech disorders due to disturbance of the instrumentalities of language derive directly from Freud. Some other writers also referred to him. The concept of the agnostic aphasias met with considerable interest and the term "agnosia" was generally accepted. Even now Freud's book is quoted with respect in some surveys of aphasia. Thiele, in an important monograph, frequently referred to it and remarked that it had remained a work of topical interest even to-day. Nielsen gave it its due place in his historical survey.

The book seems to have made little impression on the French neurologists and it has been unknown to most English and American writers. Jeliffe, and recently Ernest Jones took Head to task for completely ignoring Freud's book when he pronounced his wholesale condemnation of neurologists for their disregard of Hughlings Jackson. Obviously, Head had never read the book, although he quoted Freud as the originator of the term "agnosia". There can be no doubt that at the time of its publication Freud stood alone in his whole-hearted appreciation of Hughlings Jackson. If only for this historical fact, the book deserves to be saved from oblivion. But there is another reason, weightier than considerations of historical justice, which makes it desirable that this book should not remain unknown in the English speaking world: it appears that Freud's direct contact with the evolutionary theories emanating from England was a highly significant event in the development of psychoanalysis. The book bears witness to that encounter.

E. Stengel
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I

The subject I am going to discuss, without presenting new clinical observations, is one to the study of which the best brains of German and foreign neurology have already devoted their efforts (Wernicke, Kussmaul, Lichtheim and Grashey, Hughlings Jackson, Bastian and Ross, Charcot, and others). I therefore propose to state at once which aspects of the problem I hope to advance. I shall endeavour to demonstrate that the theory of aphasia jointly built up by the above-named writers, contains two assumptions which might profitably be revised. The first refers to the differentiation between aphasias caused by destruction of centres and aphasias caused by destruction of pathways. It has been accepted by almost all authors who have written on this subject. The second assumption is concerned with the topographical relationship between the individual speech centres. It was adopted mainly by Wernicke and those workers who have accepted, and enlarged upon, his views. As both hypotheses are important parts of Wernicke's theory of aphasia, my objections to them will take the form of a critique of the theory. They are also

I.

Wenn ich, ohne über neue eigene Beobachtungen zu verfügen, ein Thema zu behandeln versuche, an welches bereits die besten Köpfe der deutschen und fremdländischen Neuropathologie, wie Wer­nicke, Kussmaul, Lichtheim und Grashey, Hughlings Jackson, Ba­stian und Ross, Charcot u. a., ihre Kraft gewendet haben, so teue ich wohl am besten, sogleich die wenigen Punkte des Problems zu be­zeiehnen, in denen Erörterung ich einen Fortschritt einzuleiten hoffe. Ich werde mich also bemühen zu zeigen, daß in der Lehre von der Aphasic, wie sie durch das Zusammenwirken der eben genann­ten Forscher geworden ist, zwei Annahmen enthalten sind, welche man besser durch andere ersetzen kann oder welche zum mindesten vor diesen anderen Annahmen nichts Entscheidendes voraus haben. Die erste dieser Annahmen hat zum Inhalte die Unterscheidung von Aphasic durch Zerstörung der Zentren von solcher durch Zer­störung der Leitungsbahnen; sie findet sich bei nahezu allen Auto­ren, welche über Aphasic geschrieben haben. Die zweite Annahme betrifft das gegenseitige Verhältnis der einzelnen für die Sprach­funktionen angenommenen Zentren und findet sich hauptsächlich bei Wernicke und jenen Forschern, welche Wernickes Gedankengang angenommen und weiterentwickelt haben. Da beide Hypothe­sen als bedeutsame Bestandteile in der Wernickeschen Lehre von der Aphasic enthalten sind, werde ich meine Einwände dagegen in Form einer Kritik dieser Lehre vorbringen. Da sie ferner in inniger...
The current theory of aphasia.

intimately related to the idea of “localization”, i.e., of the restriction of nervous functions to anatomically definable areas, which pervades the whole of recent neuropathology. I shall have to consider the significance of this factor for the understanding of aphasia in general.

In doing so I have to turn to a famous chapter in the history of the knowledge of the brain. In 1861 Broca presented to the Société Anatomique of Paris the two post-mortem findings which enabled him to conclude that a lesion in the left third frontal convolution caused complete loss or severe reduction of articulate speech whereas the other speech functions and the intellect remained unimpaired. The qualification that this applied to right-handed subjects only, was added later, Broca's discovery has from time to time been disputed. This has no doubt been due to the tendency to reverse Broca's statements and to conclude that loss or impairment of articulate speech necessarily implies the presence of a lesion in the third left frontal convolution. Thirteen years later, Wernicke published the short essay on the symptoms of aphasia which brought him lasting fame. He described another type of speech disorder which forms the counterpart to Broca's aphasia, i.e., loss of understanding with preservation of the ability to use articulate language. He attributed this disorder to a lesion of the first temporal convolution which he had found in his cases. This discovery was bound to give rise to the hope that some day it would be possible to relate the various dissociations of the faculty of speech observed

1 P. Broca: Sur le siège de la faculté du langage articulé avec deux observations d’aphémie (perte de la parole), 1861.
2 Wernicke: Der aphasische Symptomencomplex (The aphasisie syndrome), Breslau, 1874.
in clinical practice to a corresponding number of well defined cerebral lesions. Wernicke's observations were only the first step towards this aim. He believed that he could see the way from the explanation of aphasia by localized brain lesions to an understanding of the physiological process of speech which appeared to him, in short, as a cerebral reflex: in his view, the speech sounds were conveyed via the acoustic nerve to an area in the temporal lobe, the sensory speech centre, whence the stimuli were transmitted to Broca's area, the motor speech centre, which sent the impulse to articulate speech to the periphery.

Wernicke had formed a definite idea as to how the word sounds were contained in the centre. This concept is of fundamental importance for the whole theory of localization. His answer to the question how far psychic functions could be localized was that this was permissible for the most elementary functions only; a visual perception could be related to the cerebral termination of the optic nerve, an auditory perception to the corresponding cortical area of the eighth nerve, etc. Everything beyond this, such as the association of various ideas to a concept, was a function of the association systems connecting different parts of the cortex; they could no longer be localized in one particular area. The sensory stimuli, however, which reached the cerebral cortex, left behind lasting impressions, each of which, according to Wernicke, was stored in a separate cell. "The cerebral cortex with its 600 millions of cells according to Meynert's estimation, offers a sufficiently great number of storage places in which the innumerable sensory impressions provided by the outer world can be stored one by one without interference. The cerebral cortex is populated with such residues of past stimuli which we propose to call memory images."

Wernicke's sensory aphasia.

[Wernicke (1874), S. 14]
1 [A.y. O., S. 4. Vgl. unten, S. 97]

3"
Nerve cells as storing places of impressions.

Such images of the speech sounds are supposed to lie enclosed in the cells of the sensory centre in the first temporal convolution, while Broca's centre contains the images of the speech movements, the "glosso-kinaesthetic impressions". Destruction of the sensory centre causes loss of the sound images resulting in inability to understand language, i.e., sensory aphasia, word deafness. Destruction of the motor centre eliminates the images of the speech movements, thus making the innervation of the cranial motor nerves for the production of speech sounds impossible, i.e., causing motor aphasia. In addition, the motor and the sensory speech centres are linked to each other by a tract of association fibres which Wernicke, in the light of anatomical studies and of clinical observation, located in the region of the insula. It is not quite clear whether in his view this association is provided by white fibres alone, or also via the grey matter of the insula. He mentions that fibrae propriae originate from the convolutions which encircle the Sylvian fissure. These fibres, he assumes, terminate in the insula which in consequence resembles a big spider concentrating on to itself the fibres converging from all parts of the surrounding cortex. This creates, more than anywhere else in the central nervous system, the impression of a real centre for certain functions. However, the only function attributed to the insula by Wernicke, is that of the association of "word sound image" and "kinaesthetic word impression", both of which are localized elsewhere in the cortex. A function such as this is usually attributed to white fibres only. The destruction of this fibre tract is supposed to cause a speech disorder consisting of paraphasia with normal comprehension and articulation, i.e., confusion of words and uncertainty in their use.

4 Die Bewahrung der Sprachvorstellungen in Zellen.


1 [Wernicke (1874), S. 17.]
2 [Diese Termine bei Lichtheim (1885 b), S. 207.]
3 [Wernicke (1874), S. 18 f.]

1 
2 
3
Conduction aphasia and centre aphasia. Wernicke designated this type of speech disorder "conduction aphasia" and differentiated it from the two other "centre aphasias" (Fig. 1).

I am reproducing another drawing from Wernicke's writings in which a schema of the process of speech is superimposed over a diagram of the brain, in order to show where his schema called for further elaboration:

Fig. 2

Fig. 3 in Wernicke: Der aphasische Symptomenkomplex (The aphasic syndrome)
F, T, O the frontal, temporal and occipital poles; S Sylvian fissure; a the central termination of the auditory nerve; b1 its entrance in the medulla oblongata; b area of the kinaesthetic impressions essential to articulate speech; b1 the centrifugal speech tract emerging from the medulla oblongata.

it presents the apparatus of speech without relation to the activities of the rest of the brain

5 Leitungs- and Zentrumaphasie.
Wernicke als "Leitungsaphasie"1 den bei den anderen "Zentrumaphasien" gegenüber. (Fig. 1.)

Fig. 1.

Fig. 3 in Wernicke, Der aphasische Symptomenkomplex.
F das frontale, O das okzipitale, T das temporale Ende eines schematisch gezeich- neten Gehirns. C die Zentralspalte, S die erste Umwindungsbogen um die fossa Sylvii herum. a das zentrale Ende des Nervus acusticus, b dessen Eintrittsstelle in die Oblongata, b1 Ort der zur Lautproduktion gehörigen Bewegungsvorstellungen, b, Austritt der zentrifugalen Sprachbahn aus der Oblongata.1

Das Schema von Wernicke stellt nämlich bloß den Sprachapparat außer Beziehung zur übrigen Hirntätigkeit.

1 [Wernicke (1874), S. 19. Dort sind die Zuordnungen der Buchstaben a und a, umgekehrt angegeben: innerhalb desselben seien a, das zentrale Ende des Nervus acusticus (in a dessen Eintrittsstelle in die Oblongata), b vertrete die zur Lautproduktion gehörigen Bewegungsvorstellungen in der Großhirnrinde, mit dem vorigen durch in der Inselrinde verlaufende Associationsfasern a, b verknüpft. In der zugehörigen Zeichnung jedoch fehlt der Strich bei a, so daß dieser Buchstabe zweimal ununterschieden erscheint. Freud hat jeweils die Ein- bzw. Austrittsstellen der Oblongata mit a, bzw. b, gekennzeichnet und die Zuschreibungen entsprechend umgetauscht.]

2 [A. a. O., S. 47, 63. Vgl. auch Wernicke (1881), S. 205.]
of Wernicke's theory, and might be applicable to the activity of repeating words heard. But if one takes into account the various other activities of the speech centres which are indispensable for spontaneous speech, a more complicated presentation of the speech apparatus is called for, which would hold out the prospect of explaining a greater number of speech disorders by localized lesions. Lichtheim\(^1\) (1884) took this step, and in a consistent elaboration of Wernicke's approach he arrived at the schema of the apparatus of speech which I am reproducing here (Fig. 3).

![Fig. 3 in Lichtheim: On Aphasia. "Brain", VII, p. 436.](image)

In this schema, M stands for the motor speech centre (Broca's area). A signifies the motor aphasia caused by its destruction. A represents the auditory speech area, 2 the auditory (sensory) aphasia resulting from its destruction, 3, 4, 5, 6 and 7 signify conduction aphasias.\(^1\) 3 indicates the conduction aphasia of the insula postulated by Wernicke. Point B has not the same significance in the schema as A and M which correspond to circumscribed areas of the cerebral cortex. B is no more than a schematic representation of the numerous parts of the cortex from which the speech apparatus can be stimulated into action. For this reason a speech disorder due to lesion of that point does not appear in the schema.


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6 Entwicklung der Wernickeschen

Tätigkeit des Nachsprechens in Betracht kommt. Berücksichtigt man die anderweitigen Verbindungen der Sprachzentren, welche für das spontane Sprechen könnten unerläßlich sind, so muß sich eine kompliziertere Darstellung des zentralen Sprachapparates ergeben, welche aber Aussicht bietet, eine größere Anzahl von Sprachstörungen durch Annahme von Läsionen an beschränkten Stellen zu erklären. Indem Lichtheim\(^1\) 1884\(^2\) diesen Schritt in konsequenter Weiterbildung des Wernickeschen Gedankenganges unternahm, gelangte er zu dem Schema des Sprachapparates, welches ich hier einschalte. (Fig. 3.)

![Fig. 1 in Lichtheim, On Aphasia. Brain VII (1885a), p. 436.](image)

In demselben bedeuten M das motorische Sprachzentrum (die Brocasche Stelle), 1 die durch Zerstörung desselben bedingte motorische Aphatie; A das akustische Sprachzentrum (die Wernickesche Stelle), 2 die durch Zerstörung derselben bedingte sensorische Aphatie. 3, 4, 5, 6 und 7 entsprechen Leitungsaphasien, 3 ist die von Wernicke aufgestellte Leitungsaphasie der Insel. Der Punkt B hat nicht denselben Wert im Schema wie A und M, welche anatomisch aufzeigbaren Regionen der Hirnrinde entsprechen, er soll vielmehr bloß eine schematische Vertretung der unzähligen Rindenstellen geben, von denen aus der Sprachapparat in Tätigkeit versetzt werden kann. Auch ist von einer Sprachstörung durch Läsion dieses Punktes keine Rede. Fig. 3.


\(^2\) \[Mitteilung auf der IX. Wunderversammlung der Südwestdeutschen Neurologen und Irrenärzten in Baden-Baden (Lichtheim, 1884). Die ausführlicheren Druckfassungen unter (1885a) bzw. (1885b).]
Lichtheim's elaboration

Lichtheim divided the seven types of aphasia postulated in his schema into centre aphasias (I, 2), peripheral conduction aphasias (5, 7) and central conduction aphasias (3, 4, 6). Wernicke later replaced this nomenclature by another which, although not without faults, has the advantage of having been generally accepted. According to the latter, Lichtheim's seven forms of aphasia are to be designated and defined as follows.

I. The cortical motor aphasia. Understanding of spoken language is intact, but the vocabulary is lost, or limited to a few words. Spontaneous speech and repetition of words heard are both impossible. This form is identical with Broca's well-known aphasia.

5. The subcortical motor aphasia. This differs from the above-named in one respect, i.e., by the preservation of the ability to write, and, allegedly, by another peculiarity to be mentioned later.

4. The transcortical motor aphasia. In this form spontaneous speech is impaired, but the ability to repeat words heard is preserved, which results in a peculiar dissociation of the motor component of speech.

2. The cortical sensory aphasia (Wernicke's aphasia). The patient fails to understand spoken language, nor is he able to repeat it, but he can speak spontaneously with an unlimited vocabulary. However, his spontaneous speech is not intact but shows "paraphasia". This feature, which is of far-reaching importance, will be discussed later.

7. The subcortical sensory aphasia, which differs from (6) by the absence of paraphasia in spontaneous speech.


6

7 | Lichtheim unterschied die durch sein Schema gegebenen sieben Formen von Sprachstörung als Kernaphasien (1, 2), periphere Leitungskernaphasien (5, 7) und zentrale Leitungskernaphasien (3, 4, 6). Wernicke hat diese Nomenklatur später durch eine andere ersetzt, welche gleichfalls nicht ohne Mängel ist, aber den Vorzug hat, zu allgemeiner Annahme gelangt zu sein. Wenn wir also letzterer folgen, müssen wir die Lichtheimschen sieben Formen der Sprachstörung folgendermaßen benennen und charakterisieren:

1. Die kortikale motorische Aphasie.3 Das Sprachverständnis ist erhalten, der Wortschatz aber aufgehoben oder auf wenige Worte beschränkt. Spontansprechen und Nachsprechen sind gleich unmöglich. Diese Form deckt sich mit der alten bekannten Brocaschen Aphasie.

5. Die subkortikale motorische Aphasie.3 Diese unterscheidet sich von der vorigen nur in einem Punkte (Erhaltung des Schreibvermögens), sowie angeblich durch eine andere – später zu erwähnende – Eigentümlichkeit.4

4. Die transkortikale motorische Aphasie.5 Bei dieser Form kann nicht spontan gesprochen werden, aber das Vermögen, Gehörtes nachzusprechen, ist erhalten und ergibt eine seltsame Dissoziation des motorischen Anteils der Sprache.

2. Die kortikale sensorische Aphasie.1 Der Kranke versteht nicht, was zu ihm gesprochen wird, kann es auch nicht nachsprechen, spricht aber spontan mit unbeschränktem Wortschatz. Daß seine spontane Sprache doch nicht intakt ist, sondern »Paraphasie« zeigt, ist eine Tatsache von weittragender Bedeutung, die später [S. 53 ff.] gewürdigt werden soll (Wernickesche Aphasie).

7. Die subkortikale sensorische Aphasie.2 Diese unterscheidet sich von der vorigen durch das Fehlen der Paraphasie beim Sprechen.


1 [In der deutschen Fassung (1885 b) auf S. 207.]
2 [Lichtheim (1885 b), S. 209ff.]
3 [A.a.O., S. 224 – 227.]
4 [Die Fähigkeit, die Silbenzahl anzugeben; vgl. unten, S. 58f., und S. 116.]
5 [A.a.O., S. 222 – 224.]
7 [A.a.O., S. 237 – 242.]
Lichtheim's seven forms of aphasia.

6. **Transcortical sensory aphasia.** This form presents the most unexpected dissociation of speech, but one that follows of necessity from Lichtheim's schema. The patient's spontaneous speech is paraphasic, he is capable of repeating but unable to understand what he is told and what he has repeated.

3. **Wernicke's conduction aphasia,** characterized by paraphasia in the absence of other symptoms.

I am reproducing another schema by which Lichtheim attempts to account for the impairment of written language by postulating a visual and a writing centre with their respective connections (Fig. 4). However, it fell to Wernicke, who closely followed Lichtheim's procedure, to complete this task in a later publication.

Lichtheim is said to have corroborated all forms of dissociation of speech function arising from his schema by cases which he actually observed clinically though their number was small. This makes the great success of his theory of aphasia appear well deserved. Lichtheim's schema which had been evolved by way of deduction, anticipated unexpected and hitherto unobserved forms of

Ich setze noch ein anderes Schema Lichtheims hierher, in welchem der Autor durch die Annahme eines visuellen und eines Schreibzentrums sowie deren Verbindungen den zu Aphasie gehörigen Störungen der Schriftsprache gerecht zu werden versucht. (Fig. 4.) Indes hat erst Wernicke in einer späteren Arbeit (Die neueren Arbeiten über Aphasie, Fortschritte der Medicin 1885 bis 1886) diese Aufgabe nach dem von Lichtheim gegebenen Beispiel vollends erledigt.

Wenn man erfährt, daß Lichtheim alle Formen von Dissoziation der Sprachfähigkeit, welche sich aus seinem Schema ergeben, durch wirklich beobachtete Fälle — wenn auch in geringer Anzahl — belegt hat, wird man den großen Beifall, den Lichtheims Auffassung der Aphasie fand, gewiß nicht für unberechtigt erklären. Lichtheims Schema war auf deduktivem Wege entstanden, es führte zu überraschenden und bis dahin nicht beobachteten Formen
speech dissociation. If these postulated forms could be confirmed by clinical observation, this was bound to appear as valid proof for the correctness of Lichtheim's premises. It is not a reproach against Lichtheim to point out that his schema must not be understood in the same way as Wernicke's. The latter can, as it were, be inscribed into the brain, as the localization of the centres and fibre tracts which it contains has been anatomically verified. Lichtheim's schema, however, postulates new tracts, the knowledge of which is still lacking. It is therefore impossible to say whether his centres and fibre tracts are related in space to each other in the way they are presented in the schema, or whether perhaps an "internal" and an "external" fibre tract connecting two centres merge into one for a long stretch. This would be totally irrelevant for the physiology of the speech function, though it would be highly important for the pathology of the cortical speech area. If Lichtheim's presentation was based on new anatomical findings any further opposition would be impossible and most of the observations to be presented here would be pointless. But there is an even weightier objection to Lichtheim's schema: whenever one attempts to fit an observed speech disorder into it, difficulties arise, because one finds the individual speech functions disturbed in various degrees, instead of one being completely lost and another having remained intact. Furthermore, the ease with which speech disorders that cannot be explained through one single interruption in the schema, can be attributed to combined lesions, opens the door wide to arbitrary explanations. But while these are shortcomings which more or less adhere to every attempt at schematizing, Lichtheim's schema fails to satisfy one important requirement: by its very nature
Objections to Lichtheim's schema

it must claim to be complete and to be able to account for every form of speech disorder observed clinically. Lichtheim already knew of a common instance which he was unable to fit into his schema, i.e., the combination of motor aphasia and alexia which is too frequent to be attributed to the coincidental interruptions of two fibre tracts. In trying to explain this syndrome Lichtheim made the assumption that these were cases with complete loss of all functions of speech in which the most easily reversible disability, i.e., word deafness, had already subsided; at that stage, therefore, only two other main disabilities, i.e., motor aphasia and alexia had remained. But this explanation does not seem to hold; Kahler 1 some time later reported the case of a patient who after his recovery from a transient aphasia maintained that at a time when he could only bleat instead of speak, and when he was unable to read because the letters appeared blurred, his understanding had been quite unimpaired throughout.

Such and similar observations may have caused Eisenlohr, 1 one of the soundest German neurologists, to concede to Lichtheim's schema no more than a "chiefly didactic" value.

II

All authors since Wernicke have, explicitly or implicitly, adopted the view that speech disorders observed clinically, if they have an anatomical basis at all, are caused by lesions of the speech centres or by

1 Kahler: Casuistische Beiträge zur Lehre von der Aphasie (Clinical observations concerning the theory of aphasia). Prager med. Wochenschr. Nr. 16 und 17, 1885.


10 Bedenken gegen Lichtheims Schema der Aphasie.

nach den Anspruch auf Vollständigkeit erheben, die Unterbringung einer jeden beobachteten Form von Sprachstörung ermöglichen wollen. Nun war bereits Lichtheim ein häufiger Fall bekannt, dessen Erklärung er aus seinem Schema nicht geben konnte, das Zusammentreffen von motorischer Aphasie mit Schriftblindheit (Alexie) 2, das doch zu häufig ist, um durch das zufällige Zusammentreffen zweier Unterbrechungen erledigt zu werden. Lichtheim machte zur Aufklärung dieses Symptomkomplexes die Annahme, daß es sich hierbei um Fälle von vollständigem Verlust aller Sprachfunktionen handle, bei denen die am leichtensten rückgängige Störung, nämlich die Worttaubheit, bereits überwunden sei, so daß in diesem Stadium nur die anderen Hauptstörungen: motorische Aphasie und Schriftblindheit, erübrigen. Aber diese Erklärung scheint nicht zuzutreffen, denn Kahler 1 hat späterhin einen Fall rasch vorübergehender Aphasie berichtet, in welchem der Kranke nach seiner Genesung versicherte, er habe nicht sprechen können, nur »gemeckert«, und nicht lesen können, weil ihm die Buchstaben wie »verschmiert« erschienen seien, habe aber alles verstanden, was man zu ihm gesprochen habe. Solche und ähnliche Erfahrungen mögen einen der besonnensten deutschen Neurologen, Eisenlohr 2, dazu veranlaßt haben, dem Lichtheimschen Schema der Aphasie doch nur einen »vorwiegend didaktischen« Wert zuzugestehe.

II.

Die Anschauung, daß die in der Klinik beobachteten Sprachstörungen, insofern sie überhaupt eine anatomische Begründung haben, von Unterbrechung der Sprachzentren oder

2 [Vgl. unten, S. 142.]
The differentiation between centre aphasia and conduction aphasia.

disruption of the speech association tracts, and that one is therefore justified in differentiating centre aphasias from conduction aphasias. It seems worth while to examine the validity of this distinction more closely as it is intimately related to Wernicke's afore-mentioned important concept of the rôle of the centres in the cortex and of the localization of mental functions.

If one recognizes the distinction between a "speech centre" and a mere connecting pathway consisting of a tract of white fibres, one 'has to expect a much more serious disturbance of function to result from destruction of a centre than from interruption of a conducting tract. Wernicke's presentation seems to bear this out. The only characteristic of his conduction aphasia caused by interruption of the tract a-b (Fig. 1) was confusion in the use of words, while in spontaneous speech the vocabulary was preserved and understanding unimpaired. The resulting disability, therefore, appeared to be much slighter than that in motor and sensory aphasia caused by lesions of the centres themselves.

However, there is something peculiar about Wernicke's conduction aphasia. The disturbance of function attributed to it cannot be deduced from Wernicke's schema. Wernicke states that interruption of a-b causes paraphasia; but if we ask what ought to be the result of this interruption the answer would have to be as follows: via the tract a-b the ability of reproducing perceived word sounds has been learned; its function is that of repetition of words heard; its interruption ought to result in a loss of that ability while spontaneous speech and understanding ought to remain intact. Yet every-
Critique of Wernicke's conduction aphasia.

body will admit that such a dissociation of speech functions has never been observed nor is it ever likely to be observed. The faculty of repeating is never lost as long as speaking and understanding are intact. It is absent only if (1) there is no speech at all, or (2) hearing is impaired. I know of only one single instance in which the ability to speak spontaneously and to repeat words perceived were not both intact: there are patients with motor aphasia who occasionally can produce a curse or a complicated expression which does not belong to their "speech remnants", yet they are unable to repeat on request what they have just said. But this is an entirely different situation: these patients are equally incapable of spontaneously repeating these isolated additions to their reduced vocabularies. The fact that there is no isolated loss of repetition of spoken language, and that this ability invariably remains intact as long as spontaneous speech is retained, is going to play an important part in the conclusion that one and the same tract serves speaking and the repetition of spoken words.

We are justified in denying the existence of Wernicke's conduction aphasia because it has proved impossible to find a speech disorder with the characteristics postulated. It was located by Wernicke in the insula. Lesions of this area must therefore produce a different type of speech disorder. In fact, Bastian,1 in his excellent presentation of aphasia, 1

1 The phenomena described by this term correspond to those called "recurrent utterances" by Hughlings Jackson (Transl.).

12 Kritik der Leitungsaphasie Wernickes.

12 [Vgl. unten, S. 59 f., 62, 64.]


aphasia which cannot be derived from the schema.

makes the definite statement that lesion of the
insula causes a typical motor aphasia. Unfortunately, the problem of the insular aphasia, which would be so very important for our considerations, has so far not been clarified by clinical observation. Meynert, de Boyer, Wernicke himself and others maintain that the insula belongs to the speech area, while Bernard and others, following Charcot, emphatically deny such a relation. Nothing decisive concerning this problem emerged from Naunyn's survey. Although it seems highly probable that lesions of the insula cause speech disorder (not only because of anatomical contiguity to the so-called centres), it is nevertheless impossible to state whether the speech disorder is of a specific type and if so of what type.

We propose to postpone discussion of the symptom of paraphasia (mistaken use of words), and also of the reasons which caused Wernicke to regard it as characteristic of an interruption between a and b. At this stage we only want to mention that the paraphasia observed in aphasic patients does not differ from the incorrect use and the distortion of words which the healthy person can observe in himself in states of fatigue or divided attention or under the influence of disturbing affects.—the kind of

4 de Boyer, Etudes cliniques sur la lésions corticales (Clinical studies of cortical lesions) Paris, 1879.
5 Bernard: De l’aphasie et de ses diverses formes (On aphasia and its various forms) Paris, 1885.
7 Bastian (On different types of aphasia, 1887) is inclined to regard the combination of aphasia with hemianesthesia described first by Grasset as the result of anatomical contiguity of the connections between Broca’s and Wernicke’s areas passing through the insula, and the posterior (sensory) third of the posterior peduncle of the internal capsule.

13 Sie läßt sich nicht aus dem Schema ableiten – Inselaphasie.

sicher auftretende Angabe, daß Erkrankung der Insel typische motorische Aphasie bedingt. Die Frage der Inselaphasie, die für alle unsere Erörterungen von großer Bedeutung wäre, ist leider durch die bis heute vorliegenden Erfahrungen nicht geklärt. Meynert, de Boyer, Wernicke selbst u. a. halten daran fest, daß die Insel zum Sprachbezirk gehört, während Charcots Schüler (Bernard) von einer solchen Beziehung der Insel nichts wissen wollen. Aus der 1887 vorgenommenen Zusammenstellung von Naunyn hat sich nichts Entscheidendes für diese Frage ergeben. Wenn auch eine überwiegende Wahrscheinlichkeit dafür spricht, daß Erkrankung der Insel nicht bloß der anatomischen Kontiguität wegen Sprachstörung macht, so läßt sich doch in keiner Weise angeben, ob dieser Sprachstörung eine bestimmte Form und welche Form ihr zukommt.

Wir behalten es einer späteren Erörterung [S. 54 ff.] vor, welche Bedeutung das Symptom der Paraphasie (Wortverwechslung) beanspruchen kann und wieso Wernicke dazu gelangte, es als charakteristisch für eine Unterbrechung zwischen a und b hinzustellen. An dieser Stelle sei nur erwähnt, daß die bei Kranken beobachtete Paraphasie sich in nichts von derjenigen Wortverwechslung und Wortverstimmung unterscheidet, die der Gesunde bei Ermudung, bei geteilter Aufmerksamkeit, beim Einfluß störender Affekte an sich
Paraphasia is not a focal symptom.

thing that frequently happens to our lecturers and causes
the listener painful embarrassment. It is tempting to
regard paraphasia in the widest sense as a purely func-
tional symptom, a sign of reduced efficiency of the
apparatus of speech associations. This does not exclude
that they may occur in most typical form as organic focal
symptoms. Allen Starr* is the only author of distinc-
tion who has taken the trouble of searching for the
anatomical causes of paraphasia. He arrived at the
conclusion that this symptom could be produced by
lesions of a great variety of localization; he found it
impossible to establish a consistent difference in the
pathology of cases of sensory aphasia with or without
paraphasia.

It could be objected that the above criticism of
Wernicke’s conduction aphasia is unjustified because it
does not allow for the following possibility: inability to
repeat spoken language need not be manifest because the
words heard which cannot be directly conveyed to the
motor centre might be repeated through the detour via
“understanding”, the connection A-B-M (Fig. 3) taking
the place of the interrupted tract A-M which normally
serves repetition. If this detour is really available
conduction aphasia would have to be characterized as a
condition in which understanding and spoken language
are intact, as well as repetition of comprehensible words,
but in which repetition of incomprehensible words, such
as those of a foreign language, is abolished. Such a
syndrome has not been observed, though it has so far
not been looked for. It may occur occasionally. In
admitting this possibility we have to stipulate another
condition arising from a strict differentiation between
speech centres and their

* Allen Starr: The pathology of sensory aphasia, with an analysis of
fifty cases, in which Broca’s centre was not diseased. Brain, XII, 1889.
The speech disorders associated with lesions of Wernicke's area.

The destruction of a centre naturally results in an irreplaceable loss of function: if, however, only a pathway is severed it ought to be possible to stimulate the intact centre via some undamaged fibre tract and to mobilize its stored memories. In searching for a case in which such a compensating mechanism could be found we came across an instance the discussion of which is of the highest importance for the whole theory of aphasia.

There are cases of loss of understanding (word deafness) without disturbance of spontaneous language. They are rare, but they do occur, and the development of the theory of aphasia might have taken a different course had Wernicke's first case of sensory aphasia been of that type. However, his patients showed, like most of those observed later, the picture of sensory aphasia with an impairment of spontaneous language which we propose to call paraphasia in accordance with Wernicke. Such a speech disorder could not, of course, be explained from his schema according to which the kinaesthetic word impressions are intact, as well as the pathways connecting them with the concepts. It is impossible, therefore, to understand why the words produced should not be correct. Wernicke had no choice but to base his explanation of paraphasia on the assumption of a functional factor which could not appear in his schema. He pointed out that the tract a-b, or A-M (Fig. 3), was the one via which speaking had been learned. Later on speech was produced directly from the concepts, but the tract a-b still retained a certain importance for speaking; whenever spontaneous speech was produced this tract was innervated also, and thus it exercised a continuous control over the flow of speech. The Association tract. The destruction of a centre naturally results in an irreplaceable loss of function: if, however, only a pathway is severed it ought to be possible to stimulate the intact centre via some undamaged fibre tract and to mobilize its stored memories. In searching for a case in which such a compensating mechanism could be found we came across an instance the discussion of which is of the highest importance for the whole theory of aphasia.

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Wernicke's explanation.

Wernicke supposed paraphasia to be caused by loss of this associated innervation.

Wernicke's ideas about this difficult problem are far from clear and, I believe, not even consistent, because in a later passage (p. 23 l.c. [1874]), the bloße Bestehen der Bahn a–b ohne intendierte Innervation derselben genüge schon, um die Auswahl der richtigen Bewegungsvorstellung zu sichern. Wie es zugehen kann, daß der bloße Bestand dieser Bahn, auch wenn sie nicht mitinnerviert wird, diese mächtige Einwirkung auf den motorischen Vorgang beim Sprechen äußern kann oder wie, wenn sie eine kollaterale Innervation beim Sprechen empfängt, diese sich äußern kann, ob das Zentrum b erst dann den Artikulationsimpuls aussendet, wenn die Erregung vom Zentrum a her angekommen ist, ob es viel mehr früher zu sprechen beginnt, Fehler macht und diese vermittelt der Erregung vom Wortklangzentrum her korrigiert; über all dieses kann ich mir nach Wernickes Darstellung keine anschauliche und widerspruchsfreie Vorstellung machen. Lichtheim hat diesen Mangel des Erklärungsversuches von Wernicke wohl gefühlt, denn er faßt die Bedingung zur Vermeidung der Paraphasie weit scharfer. Es genüge hierfür nicht, daß die Wortklangbilder intakt seien, sie müßten auch durch die Bahn a–b in Verbindung mit den Wortbewegungsbildern treten.1 Ein Schritt weiter hätte Lichtheim zur Annahme geführt, daß überhaupt nur auf dem Wege über die Klangbilder und die Bahn A–M gesprochen wird. Denn der Einfluß von A auf dem Wege A–M ist offenbar unnütz, wenn er erst anlangt, nachdem von M aus bereits gesprochen wurde; es wird also nicht eher gesprochen, als bis diese Erregung in M eingetroffen ist, und nun lösen sich alle Schwierigkeiten befriedigend, wenn wir die überflüssige Annahme weglassen: es bedürfe zum Sprechen noch einer besonderen Erregung von M vom Begriffe her.1

However this may be, according to Wernicke and Lichtheim spontaneous speech in sensory aphasia

16  Wernickes Erklärungsversuch derselben.

1  [Lichtheim (1885b), S. 211.]

2  [Wernicke (1874), S. 23 f.]

— der Bewegungsvorstellungen aus. Wegfall dieser Nebeninnervation von a–b bewirkt Paraphasie.
Clinical observation provides no evidence for the psychological significance of centres.

(destruction of A) becomes paraphasic because the sound images in A which normally have a controlling function have been destroyed. One would expect the clinical picture to be different if these important sound images remain intact and only the fibre tract connecting them with B has been destroyed. Such a difference would convince us that lesions of fibre tracts have a different significance from those of centres, and that images are contained only in the latter and not in the former. We should have to assume that the intact sound images exert their influence on speaking through the detour via the “concept centres” in a way similar to that contemplated earlier when repetition was discussed. In Wernicke’s conduction aphasia the centre is intact, but the association fibres are interrupted; and yet such a detour is not made. The interruption of A-M has the same effect as the destruction of A itself, i.e., paraphasia in spontaneous speech. This is another proof that Wernicke’s conduction aphasia is untenable. If we assume that interruption of a-b (A-M) cannot be compensated for by some detour of innervation, inability to repeat would be inevitable. If, however, we allow for the possibility of such a detour paraphasia ought not to occur. Consideration of all the conduction aphasias postulated by Lichtheim, and of the disorders of reading and writing not caused by lesions of the centres, leads to the following conclusions: the destruction of a so-called centre comes about only through simultaneous interruption of several fibre tracts; any assumption of a centre lesion can be replaced by one of a lesion of several fibre tracts.
Watteville's attempt at defining centre aphasia.

tracts, without abandonment of the theory of localization of psychic functions in the areas of the centres.

As 1 feel rather isolated in claiming that the alleged psychological status of the speech centre ought to manifest itself in the symptoms of the speech disorders in some way, I hasten to refer to a short but significant paper by de Watteville who expressed a very similar line of thought. "We have formed the idea", he wrote, "that these centres are storage places in which the various motor and sensory memory images are preserved. On the other hand, we must not search for the physiological substratum of mental activity in this or that part of the brain but we have to regard it as the outcome of processes spread widely over the brain. It follows from these two premises that certain lesions, the gross symptoms of which do not differ materially, must still differ in their psychological effects; let us take two cases of motor aphasia, one of them caused by destruction of Broca's centre itself, the other by interruption of the centrifugal tract originating from it. In the first case, the patient has lost control over his kinaesthetic word impressions, in the second this control has remained unimpaired. The effect of aphasia on intelligence and vice versa has been discussed frequently, yet in spite of good observations on both aspects the results have been full of contradictions. Might this not be due to the conditions mentioned above?... We feel justified in assuming that if aphasia has been caused by a central lesion the patient must have suffered intellectual damage

1 de Watteville: Note sur la cécité verbale (Note on word blindness). Progrès médical. March 21, 1885.

...}

Il nous semble donc que lorsque la lésion est centrale le malade doit, nécessairement, subir une dégradation de ses facultés intellectuelles; tandis que là où elle est commissurale ces dernières peuvent être conservées.«

Watteville's Versuch zur Auszeichnung der Zentrumaphasie.

| Lokalisation psychischer Funktionen in den Zentren vermisst wird.


1 de Watteville: Note sur la cécité verbale. Progrès médical. 21. März 1885. [Im Original, S. 227 f., lautet der von Freud übersetzte Text: »Or nous sommes arrivés, d'une part, à la conception que ces centres sont des points d'emmagement de mémoires diverses, motrices ou sensitives; d'autre part nous devons admettre comme substratum physiologique de l'âme, non l'action de telle ou telle portion du cerveau, mais une résultante de processus à siège beaucoup plus étendu. Il résulte de ces données que l'effet psychique de lésions dont les manifestations extérieures n'offrent pas de notables différences doit cependant être variable. Prevenons, par exemple, deux cas d'aphasie motrice, l'un causé par la destruction du centre même de Broca, l'autre par une interruption du faisceau efférent de ce centre. Dans le premier cas le malade aura perdu l'usage de la représentation motrice des mots, dans le second il l'aura conservée. On a souvent discuté l'effet de l'aphasie sur l'intelligence, et des opinions diverses ont été émises, appuyées par des observations bien faites. Ne trouverions-nous pas là la solution de cette contradiction apparente?
Doubts in the validity of a schema based on localization

also, while this need not be the case if the fibre tracts only have been damaged."

I do not think that anybody has taken the trouble to carry out the examinations necessary to prove Watteville’s hypothesis; I doubt that a more severe intellectual impairment will be found to be associated with “central” aphasia than with a conduction aphasia.

III

We have endeavoured to establish which clinical features bear out the alleged psychological significance of the speech centres and have for this purpose subjected Wernicke’s conduction aphasia to a critical examination. In doing so we discovered certain facts which gave rise to serious doubts in the fundamental correctness of a schema based mainly on localization, such as that of Wernicke and Lichtheim. It should not be overlooked, however, that both authors also invoke, without hesitation, functional factors in the explanation of speech disorders. A presentation which attempted to explain the variety of speech disorders observed by differences in the localization of the lesions only, would have to confine itself to assuming a number of centres and tracts functioning independently and equally liable to be put out of action. Wernicke and Lichtheim had to concede that the function of the motor centre M depended not only on its anatomical integrity but also on the maintenance of its connection with the sensory centre A. Indeed,
The significance of Lichtheim's syllable test.

Lichtheim made a surprising observation, the confirmation of which would reduce the significance of localization even further. He posed the question whether motor aphasics were in possession of "inner language," i.e., whether they could recall the sound of words which they were unable to express. He asked them to squeeze his hand once for each syllable of the requested word, and found that they were unable to prove their knowledge of the word in this way. This observation is bound to have a profound influence on the conception of the speech process for the following reason: the centre A is intact and its connection with the rest of the cortex unimpaired; the only part damaged is M, the centre of the kinaesthetic word impressions; and yet the patient is unable, because of a circumscribed lesion in the third frontal convolution, to elicit the word sound contained in the temporal lobe even with the help of some other cerebral activity such as the visual perceptions.

Unfortunately, this observation, which ought to be the corner stone of a new theory of the aphasias, has so far not been established beyond doubt. First of all, there are some objections to the way in which Lichtheim set out to prove it. His criterion of the availability of the word sound was the patient's ability to state the number of syllables of the word wanted; but it can be assumed that these patients had been in the habit of arriving at that number by transferring the sound to the motor speech tract. In this case the test would have been unsuitable because it implied the integrity of the very tract that is destroyed in motor aphasia. A similar objection against the validity of Lichtheim's

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Rejection of a direct tract for spontaneous speech.

...but there is still another objection: Lichtheim reported that he had been unable to apply his test in cases of typical cortical motor aphasia (with destruction of M) because he had had no pure cases of that type at his disposal for some time. He reported only a case of so-called transcortical motor aphasia in which the test was negative, although in this type of disorder not even M is supposed to be damaged but only its connections with B. However, I shall later demonstrate that these cases of transcortical motor aphasia call for a different hypothesis which is compatible with the loss of the sound images. The question whether or not the sound images are available in motor aphasia still appears to be undecided. Yet I should hesitate to advance a theory of aphasia before I had definite knowledge on this point.

Let us now return to the two other arguments on which we base our rejection of the functional independence of the centre M. (1) If there was a connection between M and B (tract for spontaneous speech) which was different from the connection between M and A (the tract which makes repetition of words heard and correct speaking possible), we ought to find disturbances of repetition without corresponding impairment of spontaneous speech. We have established beyond doubt that this does not happen. We therefore conclude that these two tracts are in fact one and the same.

(2) A lesion in A or in the tract A-M causes a speech disorder which compelled Wernicke and Lichtheim to adduce functional factors, without, however, enabling them to explain satisfactorily the occurrence of paraphasia in sensory aphasia. This difficulty, also, is resolved if one assumes that there is only the tract A-M, and that spontaneous speech takes place only through sound
The sensory speech disorder is more than paraphasia. This assumption is all the more suggestive as A-M is undoubtedly the first tract via which the child learns to speak. Wernicke assumed that after speaking via this tract had been sufficiently practised, another more direct tract which had no connection with the sound images came into use. But it is impossible to understand how practice in the use of a fibre system should result in its abandonment and in the choice of another. Almost all earlier writers, including Kussmaul, insisted that spontaneous speech took place via the same pathway as repetition, i.e., by means of the sound images, and a more recent author, Grashey, has reverted to this assumption. I have never been able to understand the arguments with which Lichtheim in his otherwise lucid presentation defends his thesis of a direct motor speech tract against Kussmaul.

If we suppose the pathway for spontaneous speech to go via the sensory centre A, the speech disorder resulting from a lesion of that centre naturally assumes particular interest for us. Indeed we have the impression that Wernicke and Lichtheim have not done full justice to it by calling it “paraphasia”. By paraphasia we are to understand a speech disorder in which the appropriate word is replaced by a less appropriate one, which, however, still retains a certain relationship to the correct word. Following the philosopher Delbrueck we may describe these relations as follows: In paraphasia words of a similar content, or linked by frequent association,

1 Kussmaul: Die Störungen der Sprache (Disorders of speech). 1877.

Die Sprachstörung bei sensorischer Läsion ist mehr als Paraphasie.

1 Kussmaul: Die Störungen der Sprache. 1877.
3 [Wernicke (1881), S. 206 mit Anm. Lichtheim (1885 b), S. 211, 214, 228.]
4 Delbrueck, Amnestische Aphasie, Jena’sche Zeitschr. f. Naturw. XX, Supplement II, 1886 [1887].
Definition of paraphasia. are used in place of one another, e.g., “pen” instead of “pencill”, “Potsdam” instead of “Berlin”. Furthermore, words of a similar sound are mistakenly used for each other, such as “Butter” for “Mutter” or “Campher” for “Pamphlet”; and finally, if the patient makes mistakes in articulation (literal paraphasia), single letters are replaced by others. It is tempting to differentiate between various types of paraphasia according to the part of the speech apparatus at which the mistake took place.

One also speaks of paraphasia when two intended words are fused into one malformation, such as “Vutter” for “Mutter” or “Vater”; by common consent circumlocutions by which a specific noun is replaced by a very general one (“dings”, “machine”, “chose”) or by a verb, have also been regarded as paraphasia. However, the speech disorder of sensory aphasia may go far beyond paraphasia. There are aphasics who do not produce any comprehensible words, but pour forth an endless sequence of senseless syllables (gibberish, jargon aphasia of any specific meaning, abundance of particles, interjections and other grammatical accessories, and frequent repetition of nouns and verbs are conspicuous. One of Wernicke’s patients, whose aphasia had already improved considerably, produced the following sentences in response to a present given to her: “Da lass ich mir viel viel Mal alles Mögliche, was Sie nur haben gesehen. Ich danke halb viel liebes Mal, dass Sie mir das alles gesagt. Na, da danke ich vielmals, dass Sie mir das alles gesagt. Na, da danke ich vielmal, dass Sie sind so gut gewesen, dass Sie sind so gütig gewesen.” (“There I leave for myself many many times everything which you have only seen. I thank many a good time that you told me all this. There I thank many times that you have been so kind, that you have been so kindly.”) I remember having myself seen a case of sensory aphasia in the Vienna General Hospital, a Mrs. E. who was

2 Ross: On Aphasia. London, 1887 (also Manchester Medical Chronicle).
3 [Richtig: Na, da danke ich viel Mal.]
4 [A. a. O., S. 43.]
The origin of Lichtheim's transcortical motor aphasia.

demonstrated to us as "encephalitic confusion"; her speech showed the same peculiarities: impoverishment in nouns, adjectives and verbs; abundance of all other types of words, and a tendency to reiteration. Wernicke regarded "an intact vocabulary with paraphasia" as characteristic of the sensory aphasia. I believe it can be more correctly described as "impoveryment of words with an abundance of speech impulses".

However, if we omit the tract for spontaneous speech, B-M, from Lichtheim's schema, how shall we explain the cases of transcortical motor aphasia which Lichtheim so easily explained with the interruption of that very pathway? These are the cases in which spontaneous speech is quite impossible, while repetition, reading aloud (i.e., speaking from visual images), etc., proceed unimpaired.

Fortunately we can arrive at an understanding of these cases in a different way. Heubner recently published an important observation to which we shall frequently refer. His patient had lost the capacity of speaking spontaneously, but he had retained the ability to repeat words spoken to him by others, and to read aloud. He also had lost comprehension of spoken and written language. The symptoms were those of a combination of a transcortical motor with a transcortical sensory aphasia. His case could not be explained by entering a single lesion in Lichtheim's schema, but rather by the

Heubner's case.

coincidence of two lesions: one in the tract B-M and another in the tract B-A. However, the post mortem examination revealed a cortical softening of a most interesting localization, at any rate as far as the sensory speech area was concerned; it encircled Wernicke's area in the first temporal convolution, thus isolating it from the rest of the cortex on its upper posterior and lower circumference. There was, in addition, a superficial cortical softening, the size of a lentil, on the tip of a gyrus belonging to the third frontal convolution (Fig. 5).

The post mortem finding in Heubner's case.

These findings seemed at first to corroborate Lichtheim's schema, but on closer consideration one must agree with Heubner that the lesion in the motor speech area was much too limited and insignificant to be charged with the "enormous and profound disturbance of speech". Besides, it was situated in the cortex and was in no way transcortical, and if it had caused disturbances they would have interfered with repetition as well as with spontaneous speech. The speech disorder has to be explained by the considerable lesion in the sensory area only, and we see from this case that isolation of the sensory centres from its cerebral connections, i.e., a
Magnan's case.

transcortical sensory lesion, can also cause loss of spontaneous speech. This means that the tract B-M is identical with the tract B-A, or that speech is produced only via the sound images.

We remember that Lichtheim, with the aid of his syllable test, established in his case of subcortical motor aphasia that the patient was unable to elicit sound images with the help of his "concepts". If we may draw inferences from Heubner's case to that of Lichtheim in which the speech functions were less severely disturbed, we may assume that in the former case the lesion was probably also situated in the sensory area. If this should be correct the negative result of Lichtheim's test would lose the significance it would have had if the lesion in the motor speech area had been verified.

It is admittedly unsatisfactory to base a decision on one single case, all the more so as there was a small lesion in the motor area. I have therefore endeavoured to find some other cases of so-called transcortical motor aphasia with post mortem findings, and I have arrived at the following unexpected conclusion: loss of spontaneous speech which is not coupled with inability to repeat words perceived, does not by itself indicate a lesion of the sensory area. This symptom, which is characteristic of the transcortical motor aphasia, can also be found in cases with a lesion confined to the motor area alone; but in one case only could the lesion be correctly described as transcortical. In this case (Magnan) a tumour was found which had originated from the inner surface of the dura. It had proliferated into the left hemisphere like a wedge the thin edge of which had reached the third left frontal convolution and the anterior third of the upper margin of the insula. The patient was unable to give information about

1 Magnan: On simple aphasia and aphasia with incoherence. Brain II, 1880.

2 [Wohl Verschreibung für: "transkortikaler".]
3 [Vgl. oben, S. 58 f.]

Läsion, auch Aufhebung der spontanen Sprache verursacht, d. h. daß die Bahn BM zusammenfällt mit der Bahn BA oder daß nur über die Klangbilder gesprochen wird.

Wir erinnern uns, daß Lichtheim bei seinem Falle von subkortikalerm motorischer Aphasie vermittels seiner Silbenprobe feststellte, daß der Kranke die Klangbilder der Worte nicht von seiner Gedankentätigkeit her erregen konnte. Wenn wir aus dem Falle Heubners auf den Lichtheims schließen dürfen, der jedenfalls eine geringere Schädigung der Sprachfunktionen repräsentiert, so läge auch in diesem Falle die Läsion auf sensorischem Gebiete, und der negative Ausfall der Probe verlor hierdurch die Bedeutung, die er in einem Falle von sicher motorischer Läsion gehabt hätte.

Es ist indes immerhin mäßlich, eine Entscheidung auf einen einzigen Fall zu stützen, zumal dieser doch eine kleine Läsion auf motorischem Gebiete aufweist. Ich habe mich daher bemüht, einige andere Fälle von sogenannter transkortikaler motorischer Aphasie mit Sektionsbefunden aufzufinden, und bin dabei zu folgendem, für mich unerwartetem Ergebnis gelangt. Die Unfähigkeit des spontanen Sprechens bei erhaltenem Nachsprechen läßt nicht mit Notwendigkeit auf eine Lokalisation im sensorischen Gebiete schließen. Dieses für die transkortikale motorische Aphasie charakteristische Symptom findet sich auch bei ausschließlichem Sitz der Erkrankung in der motorischen Region; aber nur in einem einzigen Falle war die Läsion wirklich als eine "transkortikale" zu bezeichnen. Es handelte sich in diesem Falle (Magnan) nämlich um einen Tumor, der auf der Innenfläche der Dura mater außergewöhnliche Ausdehnung und mit seiner Spitze bis zur dritten Frontalwindung vorstieß. Die Kranke war unfähig, Auskunft über sich zu geben, sprach

1 Magnan, On simple aphasia, and aphasia with incoherence. Brain II, 1880 [S. 120–123].
herself, being able to utter only single words and senseless syllables; but she could repeat words spoken to her.

In the two other cases in which post mortem findings were available the lesions were situated in the motor cortex itself. They could be called “transcortical” only if one used this term in a sense which makes it quite useless in the theory of aphasia. In one case the lesion consisted of a haemorrhage in the motor centre, in the other it had been caused by a bone fragment lodged within that centre. Both cases were observed by Hammond who described them as follows:

Case I. When Hammond, in summer 1857, was stationed in the Rocky Mountains with a group of soldiers and labourers, a Mexican was hit by a workmate on the left temple with a club and collapsed unconscious. When he recovered consciousness he had completely lost his memory for words but not the ability to articulate. He was incapable of spontaneous speech but able to repeat words spoken to him with correct articulation provided they were only a few at a time; e.g., when Hammond asked him “¿Cómo sientes ahora?” (How are you now?), he repeated, “¿Cómo sien, sien, sien”, and then burst into tears. The patient died on the following day; the post mortem examination revealed a “haemorrhage of the size of a half dollar piece involving the left frontal lobe on its postero-lateral margin” and a rupture of the right middle meningeal artery.

Hammond’s clinical examination of this case can hardly have been exhaustive; he added to his report: “I did not attribute any special importance to the lesion of the left frontal convolution at the time. Only after the


3 Hammond’s report of these two cases is not fuller than the one given here. Lichtheim, nevertheless, classified the first case as one of transcortical motor aphasia. I beg to venture the same opinion concerning the second case.
Transcortical motor aphasia
discussion in the Paris Academy in 1861 did I realize
that this patient’s amnesic aphasia had been caused by
the lesion.

Case II. During the winter of 1868-69 Hammond saw
a man who some months previously, when working in a
quarry, had sustained a knock against the left side of his
head from a machine. The patient appeared intelligent,
understood everything that was said to him, and made
the most desperate effort to express himself; but he was
unable to utter any words except “yes” and “no”. Hammond
asked him: “Were you born in Prussia?”—
“No”—“In Bavaria?”—“No”—“In Austria?”—“No”—
“In Switzerland?”—“Yes, yes, yes, Switzerland,
Switzerland.” When giving the last answer he laughed
and gesticulated wildly. Hammond assumed that the
accident had caused a fracture of the inner table of the
skull and that a bone fragment was pressing on the
third frontal convolution. On his advice trephining was
carried out and his diagnosis was confirmed. As soon as
the patient woke up from the anaesthesia his speech was
completely restored.1

In these cases Lichtheim’s transcortical motor aphasia
had been caused by lesions which had nothing whatever
to do with interruption of a tract B-M.

On closer consideration of these cases another
important aspect emerges which might be relevant for
other speech disorders also. It is generally known that
in the great majority of cases motor aphasia is caused by
softening. It seems a remarkable coincidence that in
each of the above-mentioned cases of so-called transcortical motor aphasia the lesions were of a different
nature, except for

28 Die transkortikale motorische Aphasic

der Diskussion in der Pariser Akademie 1861 bin ich
zur Überzeugung gelangt, daß die amnestische Aphasic dieses Falles
von dieser Verletzung herrührte.2

Fall II. Im Winter 1868-69 sah Hammond einen Mann, der einige
Monate vorher bei der Arbeit in einem Steinbruch einen Stoß gegen
die linke Seite des Kopfes von einer Maschine erlitten hatte. Der
Kranke schien sehr intelligent, verstand alles, was man zu ihm sprach,
machte die verzweifeltesten Anstrengungen, selbst zu sprechen, brachte aber nie andere Worte als “ja” und “nein” heraus. Hammond
fragte ihn: »Sind Sie in Preußen geboren?«—»Nein.«—»In Bayern?«
—»Nein.«—»In Österreich?«—»Nein.«—»In der Schweiz?«—»ja, ja,
ja, Schweiz, Schweiz.« Dabei lachte er und bewegte die Hand nach
nen Richtungen. — Hammond nahm an, daß bei jedem Unfall ein
Bruch der inneren Schädelkapsel stattgefunden habe und daß ein
Knochensplitter auf die dritte Frontalwindung drückte. Er riet zur
Trepanation, die auch ausgeführt wurde und seine Diagnose voll-
inhaltlich bestätigte. Sobald der Kranke aus der Narkose erwachte,
war seine Sprache wiederhergestellt.3

Wir sehen also, daß hier die transkortikale motorische Aphasic
Lichtheims durch Läsionen zustande kommt, welche mit der Unter-
brechung einer Bahn BM nicht das mindeste gemein haben.

Bei näherer Betrachtung dieser Fälle ergibt sich uns aber ein anderer
wichtiger Gesichtspunkt, der auch für andere Sprachstörungen in
Betrachtkommen dürfte. Es ist allgemein bekannt, daß die motoris-
sche Aphasic in der größten Mehrzahl der Fälle auf Erweichung
beruht. Nun ist es gewiß ein beachtenswertes Zusammentreffen,
däß die Fälle von sogenannter transkortikaler motorischer Aphasic,
die ich im vorstehenden erwähnt habe, durchwegs auf Läsionen an-
derer Natur zurückzuführen, bis auf

2 [Freuds Hervorhebung. — Ibid.: »At that time I attached no especial
importance to the injury of the left anterior lobe; but, since the debate in the
French Academy in 1861, I have had no doubt that to it the amnestische
aphasic was entirely due.«]

3 Die Beschreibung dieser beiden Fälle bei Hammond ist nicht vollständiger,
as ich sie wiedergeben habe. Da indes Lichtheim ([1885a], S. 222) den ersten
überselben als transkortikale motorische Aphasic anerkennt, wage ich dasselbe
für den zweiten.

a [Der zweite Fall (bei Hammond «Case III»), a.a.O., S. 193: »Were you
born in Prussia? — No. — In Bavaria? — No. — In Austria? — No. — In Switzer-
land? — Yes, yes, yes, Switzerland, Switzerland, at the same time laughing, and
moving his hands actively in all directions.«]
may be caused by lesion in the motor or sensory area.

Heubner's case which showed a sensory disturbance. Lichtheim's principal case was traumatic, and so were Hammond's two cases. Finally, in Magnan's case the speech disorder was due to a tumour.1

If lesions of the brain give rise to symptoms at all, conclusions as to the localization of the damage can be drawn, whereas we have to guess the diagnosis of the pathological process from special circumstances of the case or from the course of the illness. The speech apparatus, however, is exceptional, in having at its disposal such a wealth of symptoms that it may be expected to betray, by the type and manner of the disturbance of function, not only the site but also the nature of the lesion. Perhaps one day we shall be able to differentiate clinically aphasia due to haemorrhage from aphasia due to softening, and to recognize certain speech disorders as characteristic of specific pathological processes affecting the apparatus of speech.

It can be regarded as established that the occurrence of the so-called transcortical motor aphasia at any rate, does not prove the existence of a special pathway B-M for spontaneous speech. This type of speech disorder is caused either by lesions in the sensory speech area or by special affections of the motor speech region as the result of pathological processes.

1 In the case of transcortical motor aphasia referred to by Lichtheim (case of Farge, quoted by Kussmaul, p. 49, and in Nothnagel's Töpische Diagnostik, p. 358), a softening in the white matter adjoining the third left frontal convolution was found. Nothnagel denied that this case by itself proved anything about the significance of subcortical lesions for the origin of aphasia as the patient had died twenty days after the softening had set in; at that time, he argued, remote effects of the lesion on the third frontal convolution, which may appear anatomically intact, could not be ruled out.
It results from a lowering of the functional state of the motor center of which the motor center is functioning at a lower level.  

Charlton Bastian² with whose explanation of the so-called transcortical motor aphasia we agree, distinguishes three states of reduced excitability of a center. The smallest reduction manifests itself in a failure of the center to react to "volitional" stimulation while it still reacts to stimulation by association with another center and to direct sensory stimuli. If function is more severely disturbed the center reacts to direct sensory stimulation only, and finally, at the lowest level of functioning, that reaction also fails. For the transcortical motor aphasia one would, therefore, have to assume that the motor center can still be activated by direct sensory stimulation while volition no longer has this effect; and as this motor center is always stimulated by association with the auditory center, the cause of the change of excitability may be situated in either.

Our considerations have led us to attribute a certain clinical type of speech disorder to a change in the functional state of the speech apparatus rather than a localized interruption of a pathway. As this step is so very important for the whole theory of aphasia, we pro-

¹ The following is a list of the six cases of transcortical motor aphasia in which the etiology was established. (1) Lichtheim: traumatic contusion of unknown localization. (2) Farge: indirect interference with the motor speech area by a softening in its vicinity. (3) Heubner: softening in Wernicke's area. (4) Magnan: tumour involving Broca's area. (5) Hammond I: traumatic haemorrhage over Broca's area. (6) Hammond II: inhibition of the motor speech area through a bone fragment lodging in it.


30 Sie beruht auf einem herabgesetzten Funktionszustand des motorischen

[den normalen herabgesetzten Funktionszustand versetzt wird.³ Charlton Bastian⁴, der für die sogenannte transkortikale motorische Aphasia Lichtheims dieselbe Erklärung gibt wie wir, unterscheidet nämlich drei Zustände von vermindertem Erregbarkeit⁵ eines Zentrums. Die leichteste Herabsetzung zeigt sich darin, daß dieses Zentrum nicht mehr auf "willkürliche« Anregung reagiert, wohl aber noch auf Anregung auf dem Wege der Assoziation von einem anderen Zentrum hier und auf direkten sensiblen Reiz. Bei stärkerer funktioneller Schädigung ergibt es nur noch eine Reaktion auf direkten sensiblen Reiz, und endlich auf der tiefsten Stufe versagt auch dieser. Für die transkortikale motorische Aphasia mußte man also annehmen, daß das motorische Zentrum noch auf direkte sensible Erregung zur Tätigkeit zu bringen ist, während eine »willkürliche« Anregung dies nicht mehr vermag⁶, und daß dies motorische Zentrum immer durch Assoziation mit dem akustisch sensorischen an- geregter wird, kann die Ursache der Erregbarkeitsveränderung im sensorischen Zentrum ebensowohl wie im motorischen selbst gelegen sein.

Wir merken jetzt, daß wir dazu gelangt sind, eine klinisch beobachtete Form von Sprachstörung anstatt durch eine lokalisierte Bahnumterbrechung durch eine Annahme über eine Veränderung des funktionellen Zustandes zu erklären. Da dieser Schritt ein so wichtiger für die gesamte Auffassung der Aphasic ist, wollen wir uns zu


⁴ [Freud führt diese »diminution de l'excitabilité« als ein Beispiel von funktioneller oder dynamischer Veränderung in seiner französisch geschilderten Studie (1893c), S. 31, an; vgl. dazu auch die Anm. 1 auf S.70.]


⁶ [Vgl. unten, S.81, 85, 128, 134 ff.]
pose, in order to make sure of our premises, to restate that we were compelled to drop the localizatory explanation because the post-mortem findings (Heubner, Hammond) had failed to bear it out. The assumption made by Bastian and ourselves appears to follow without difficulty from the fact that repetition invariably remains intact longer than spontaneous speech. Later on we shall produce observations which will also demonstrate that the associative activity of a centre is less easily lost than the so-called spontaneous one.

Bastian's hypothesis seems at first somewhat perplexing; it appears irreconcilable with an approach aimed at the study of localized lesions and their effects. It may be argued that reduction of excitability of a centre, being a purely "functional" state, does not imply the presence of an actual lesion. This is correct, and there might be conditions similar to the transcortical motor aphasia which are the result of mere functional impairment without organic damage. However, if one considers the relationship between "organic lesion" and "functional disturbance" one must realize that a great number of organic lesions cannot manifest themselves otherwise than by disturbances of function, and experience shows that these lesions have, indeed, no other effect. For decades we have been endeavouring to advance our knowledge of the localization of functions by the study of clinical symptoms; we have got into the habit of expecting a lesion to destroy a number of units of the nervous system completely and to leave the rest completely intact, because only thus, we believe, can clinical experience be made to fit our preconceptions. Yet only few lesions comply with these postulates. Most lesions are not directly destructive and they have a disturbing effect on much larger a number of nervous units than those immediately involved.

1 [Auf die Frage nach der Natur einer solchen »bloß funktionellen Schädigung« (i.e. verminderte Erregbarkeit) geht Freud in seinem französischen Aufsatz (1893c) gegen Ende von Abschnitt III und in Abschnitt IV ausführlich ein; er folgert dort (S. 50f.), daß sie von anatomischer Lokalisation völlig unabhängig sein muß, und gelangt zu dem berühmten Satz: »[...] Lasion de la cordoncule de la quinte se comporte dans ses paralysies et autres manifestations comme si l'anatomie n'existant pas, ou comme si elle n'en avait nulle connaissance«, den er bereits 1888 in seinem Handbuchartikel 'Hysterie' so formuliert hatte: »[...] die Hysterie ist ebenso unwissend vom Bau des Nervensystems wie wir selbst, ehe wir's gelernt haben« (1888b (3), Nachtragsband S. 80f. mit editorischer Anm. 1).]

2 [Vgl. unten, S. 72; auch S. 128.]
Reaction of the speech apparatus to partially destructive lesions.

Furthermore, the impact of an only partly destructive lesion on the whole of the apparatus concerned must be taken into consideration. Two possibilities are conceivable both of which do in fact occur. Either some parts of the apparatus are put out of action by the lesion while the intact parts continue to function as usual; or it reacts to the lesion as an unitary whole, in which case there is no loss of part functions but a lowering of function in general. To an incomplete lesion it responds by a disturbance of function which could be caused also by non-structural damage. The central motor apparatus for the upper extremities, for instance, shows both modes of reaction: a small lesion in the anterior central gyrus may cause an isolated paralysis of the muscles of the thumb. More commonly, however, it results in a slight paresis of the whole arm. It appears that the speech apparatus shows in all its parts the latter kind of reaction to incomplete lesions; it responds to such a lesion with a disturbance of function. For instance, a small lesion in the motor speech area would never cause the loss of a hundred words the type of which would depend only on the site of the lesion. Partial loss can always be shown to be the expression of a general lowering of the functional activity of that centre. It is not, by the way, a matter of course that the speech centres should behave in this way; their reactions to damage suggest a certain concept regarding their organization which is to be discussed later.

Before leaving the subject of motor aphasia it seems appropriate to consider two points: if transcortical motor aphasia is symptomatic of a state between normality and complete loss of excitability

32 Reaktion des Sprachapparates auf unvollständig destruierende Läsionen.

32 Ferner ist das Verhältnis einer unvollständig destruierenden Läsion zu dem Apparat, den sie befallen hat, ins Auge zu fassen. Es sind hier zwei Fälle denkbar, die sich auch in Wirklichkeit vorfinden. Entweder der Apparat zeigt sich durch die Läsion in einzelnen Teilen verstümmelt, während die erhaltenen Teile desselben in unveränderter Weise funktionieren, oder er reagiert als Ganze, solide, auf die Läsion, läßt nicht den Ausfall einzelner Teile erkennen, sondern weist sich in seiner Funktion geschwächt; er antwortet auf die unvollständig destruierende Läsion mit einer Funktionsstörung, die auch durch nicht-materielle Schädigung zustande kommen könnte. Der zentrale Apparat für die obere Extremität zeigt uns z. B. beiderlei Reaktionsweisen. Wenn sich eine kleine organische Läsion in der vorderen Zentralwindung befindet, so kann deren Wirkung in der isolierten Lähmung, etwa der Daumenmuskeln, bestehen. Gewöhnlicher ist es aber, daß sich die Wirkung als Parese mäßigen Grades des ganzen Armes offenbart. Der Sprachapparat scheint nun in allen seinen Teilen die zweite Art der Reaktion gegen nicht-destruktive Läsionen zu zeigen, er antwortet auf eine solche Läsion solidarisch (wenigstens partiell solidarisch) mit einer funktionellen Störung. Es kommt z. B. nie vor, daß infolge einer kleinen Läsion im motorischen Zentrum hundert Worte verlorengehen, deren Natur bloß vom Sitze der Läsion abhängt. Es läßt sich jedesmal zeigen, daß der partielle Verlust Ausdruck einer allgemeinen funktionellen Herabsetzung dieses Zentrums ist. Es ist übrigens nicht selbstverständlich, daß die Sprachzentren sich in dieser Weise verhalten, und wird uns später zu einer ganz bestimmten Vorstellung vom Baue dieser Zentren verhelfen.

Ehe ich diese Erörterung über die motorische Aphasie abbreche, muß ich zweier Punkte gedenken, die hier die passendste Erledigung finden. Wenn die transkortikale motorische Aphasie das Symptom eines Zustandes ist, welcher zwischen der Norm und der vollen Unerschließbar-

1 [Beispiele für solche nicht-materiellen Schädigungen gibt Freud wiederum in der französischen Arbeit (1893 c), S. 52 f.]

2 [Siehe unten, S. 81, 85, 134. - Vgl. dazu die grundsätzlich entgegengesetzte Position und Argumentation Meynerts, beispielsweise in (1867/68), S. 83 f.: Fassen wir die Rinde als ein solideres, wirkungsmäßiges Organ auf, dann ist mit der Erkenntnis, daß sie die psychischen Vorgänge überhaupt vermittelt, die funktionelle Kenntnis von derselben in psychologischer Hinsicht abgeschlossen, die Weiterbildung einer Gedankenskala über dieselbe unmöglich und unnötig geworden. —] Ähnlich ibid., S. 103, 213 f.; auch in (1884),
Logoplegia. one would have to expect it to occur when motor aphasia is in the process of subsiding, i.e., one would anticipate motor aphasics to pass through a phase when they are better able to repeat words heard than to speak spontaneously. I believe that a case described by Ogle bears this out. I have not been able to find other instances which would confirm my expectation. I daresay that clinicians have not directed their attention to this question.

Secondly, I have to consider an objection which has no doubt occurred to every reader: if spontaneous speech takes place via the sound images by the route B·A·M, every sensory aphasia ought to result in loss, and not only in a disturbance, of speech. How is it to be explained that, on the contrary, in this type of aphasia speech is still so abundant though incorrect? I can do no more than recognize the difficulty and point to another one in reply.

There are cases of logoplegia, i.e., of simultaneous loss of understanding and of expression, where our postulate of the loss of spontaneous speech in sensory aphasia seems to be met. However, in these cases the disability is caused by multiple and extensive lesions involving both motor and sensory areas. These cases seem to take a characteristic clinical course: the sensory disorder subsides and in a later stage the patient presents the picture of a pure motor aphasia. It may also happen that the patient presents a motor aphasia from the beginning, and post-mortem examination may reveal a lesion affecting not only Broca’s region but also a large part of the remaining speech region including Wernicke’s

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33 Logoplegie.

...keit liegt, so muß man erwarten, daß sich dieses Symptom bei motorischer Aphasie einstelle, wenn dieselbe in Besserung übergeht, daß also motorisch Aphasische früher und besser nachsprechen lernen, ehe sie wieder spontan sprechen. Ich glaube, daß ein Fall von Ogle diesem Charakter erkennen läßt; im übrigen war ich nicht imstande, zahlreiche Bestätigungen für meine Erwartung zu sammeln. Ich darf sagen, daß die Aufmerksamkeit der Beobachter sich diesem Punkt nicht zugewendet hat.

Ferner muß ich einen Einwand berücksichtigen, den gewiß jeder der Leser bereits bei sich gemacht hat. Wenn das spontane Sprechen auf dem Wege B·A·M über die Klangbilder vor sich geht, so müßte ja jede sensorische Aphasie den Verlust der spontanen Sprache, nicht bloß eine Störung derselben nach sich ziehen. Wie ist es zu erklären, daß bei sensorischer Aphasie noch so reichlich, wenn auch nicht richtig gesprochen wird?


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1 [Vgl. S. 69.]


3 [Auf der Seite 33 befindet sich im Original noch eine weitere Anmerkung Freuds, für die im Text jedoch eine entsprechende Fußnotenziffer fehlt, so daß nicht deutlich wird, worauf sich dieser Hinweis bezieht:] 2 Vgl. Ross l.c. 

4 [Vgl. dazu den Aufsatz ·Zur Aphasielehre Sigmund Freuds· von Paul Vogel (1954), in welchem die Besonderheiten der Freudschen Auffassung der Aphasien herausgearbeitet worden sind. (P. V.) – Siehe auch unten, S. 129.]
Destruction of Wernicke's area without word deafness

area. Kahler\(^1\) has reported a case of this type which is far from rare, and has collected similar cases from the literature. The occurrence of cases with lesions of the sensory centre but without word deafness, at least without permanent word deafness, has been established beyond doubt, although every word deafness is to be related to a lesion of that centre. How this apparent contradiction can be resolved I am at present unable to state; but I believe that its clarification will also provide the answer to the earlier question why sensory aphasia does not always entail a complete loss of speech. It is important to realize that the extension of the centre A has not been finally established.

There are cases of sensory aphasia without any disturbance of spontaneous speech, cases with only slight paraphasia and marked impoverishment of language and cases with distortion of speech amounting to gibberish. According to Allen Starr,\(^1\) it is impossible to explain these variations by differences in the localization of the lesions within the sensory area. Perhaps some observations to be presented later in this book will contribute to the clarification of this difficulty.

IV

The publication of Lichtheim's paper which presented the localizatory theory of aphasia with such consistency, coincided with an address by Grashely\(^2\) which was soon hailed as a fundamental contribution to the understanding of aphasia, although hardly anybody has

1 Kahler: Casuistische Beiträge zur Lehre von der Aphasie (Clinical observations concerning the theory of aphasia). Prager med. Wochenschr., Nr. 16 and 17, 1885.

2 Allen Starr: The pathology of sensory aphasia, with an analysis of fifty cases in which Broca's centre was not diseased. Brain, XII, 1889.


34 Zerstörung der Wernickesen Stelle ohne Worttaubheit.


IV.

Etwa gleichzeitig mit jener Arbeit Lichtheims, welche die lokalisateurische Erklärung der Sprachstörungen so konsequent durchführte, wurde ein Vortrag von Grashely\(^1\) bekannt, welchem man bald eine fundamentale Bedeutung für das Verständnis der Aphasie nachrühmte, ohne daß übrigens seither viele auf den so geschaffenen Grundlagen

1 Kahler, Casuistische Beiträge zur Lehre von der Aphasie. Prager med. W., Nr. 16 und 17, 1885.

2 Allen Starr, The pathology of sensory aphasia, with an analysis of fifty cases in which Broca's centre was not diseased. Brain, XII, 1889.


Amnesic aphasia.

continued on his lines. Grashey’s case showed no special features, except one; the patient was a man aged 27, who as the result of a fall had sustained a fracture of the skull; he was almost completely deaf in the left ear, had lost his sense of smell and taste, was able to see with his right eye hand movements only, while the visual acuity of his left eye was reduced to 2/3 and his visual field was contracted. The facialis and hypoglossus, as well as the whole musculature of the right side of the body were paretic. In addition, the patient had a speech disorder which immediately after the injury showed the features of word deafness. When he came under Grashey’s observation his speech had greatly improved and showed on some of the common residual disturbances. The patient was able to speak coherently, he used all prepositions and conjunctions and also some verbs and adjectives without difficulty; in spontaneous speech he produced an occasional noun, but usually resorted to circumlocutions ("Dingsda"). He recognized every object which he had known before his illness yet was unable to name any. Understanding of spoken language was intact. The inability to use nouns in spontaneous speech and to name objects, though they were recognized, is known to be one of the most common symptoms of the so-called amnesic aphasia which was distinguished from the atactic aphasia by earlier writers.1

The relationship of amnesic aphasia to the types of speech disorder which can be attributed to interruption of pathways has always been a difficult problem. This is not surprising as the concept of amnesic aphasia was based on psychological, and that of the other forms on anatomical considerations.

1 The distinction between amnesic and atactic aphasia was proposed by Sanders in 1866.

34 Amnestische Aphasie.


Die Unfähigkeit, im Redefluß Substantiva zu gebrauchen und erkannte Gegenstände mit Namen zu bezeichnen, ist wie gesagt eines der gemeinsten Symptome der sogenannten amnestischen Aphasie, die von älteren Autoren neben der ataktischen Aphasie unterschieden wurde.2

Das Verhältnis dieser amnestischen Aphasie zu den Arten von Sprachstörung, welche man durch Bahnunterbrechung charakterisieren konnte, hatte der Auffassung immer Schwierigkeiten bereitet. Allerdings begreiflicherweise, da die eine Aufstellung auf einem psychologischen, die andere auf einem anatomischen Gesichtspunkte beruhte.

2 [Grashey, a.a.O., S. 655.]
3 Die Unterscheidung von amnestischer und ataktischer Aphasie ist 1866 von Sanders aufgestellt worden. (Zit. bei Bastian (1869), S. 214, Anm. 1: »In his paper, in the 'Edin. Med. Journ.' for March, 1866, he pointed out the distinction between what he termed the amnesic and the ataxic forms of aphasia [...].«]
Grashey's case. Lichtheim regarded it as incorrect to list amnesic aphasia together with the other speech disorders. In his view it was a common accompaniment of the typical aphasias and their residual states; it was not a focal symptom and occurred in cases with more diffuse pathological processes, such as generalized vascular lesions, or as a sign of senile reduction of cerebral functions.

The suggestion that the principles of localization which had been declared all-important for one class of speech disorders, should not be applied to another class, seems at first quite unconvincing. Grashey undertook an analysis of the symptoms of his case of amnesic aphasia with the help of the schema reproduced in Fig. 6. He considered the possibility that the pathway from the area of the sound images to that of the object images was intact, while the pathway in the opposite direction was interrupted. Under these circumstances the patient might have been capable of correctly relating a word to the appropriate object.

Der Fall von Grashey.

Lichtheim hielt es für unstatthaft, die Amnesien den anderen Formen von Sprachstörung gleichzustellen; er meinte, Amnesie sei eine häufige Begleiterscheinung der von ihm beschriebenen Typen und deren Rückbildungszustände, sie sei aber kein Herdssymptom und zeige sich bei diffuseren krankhaften Prozessen, bei allgemeiner Zirkulationsstörung im Gehirn oder als Zeichen der senilen Rückbildung der Hirntätigkeit.

Fig. 6

The schema with the help of which Grashey explained the disturbance of function observed in his patient.

A centre for sound images; B centre for object images; C centre for symbols, i.e., written and printed letters, words and numerals; D centre for the kinaesthetic impressions of articulate speech; F nuclei of the nerves serving phonation and articulation; G centres for the kinaesthetic impressions of writing movements; H nuclei of the motor nerves serving writing movements.

1 [Grashey (1885), S. 656. Dort ist allerdings auch noch eine mit Richtungspfeil versehene Verbindungslinie vom Scheitelpunkt des rechten Winkels bei »Auges« zum Punkt C eingezeichnet, die in der Wiedergabe bei Freud fehlt.]

2 [Vgl. dazu Freuds Lexikonartikel »Amnesie« (in 1893–94a), in dem auch die beiden Formen der amnestischen und der ataktischen Aphasie erwähnt werden.]
Rejection of localization as explanatory principle.

yet unable to find the sound impression for the object presented to him. Grashey, very much to his credit, dismissed this hypothesis with the following words: "After all, in this way one could explain any symptom. . . . I was therefore not satisfied with the arbitrary insertion and elimination of conducting tracts, but examined the patient more carefully. I found that the functions of the apparently normal centres were considerably disturbed."

His patient showed a conspicuous inability to retain "object images, sound images and symbols" over any length of time. When shown an object which he was able to recognize, and requested after a few moments to touch it, he had in the meantime forgotten which object he had been shown; when two words were said to him in succession he was unable to repeat the first when requested to do so; he had invariably forgotten it and had retained the second word only. For the same reason, he was unable to synthesize, and to perceive as wholes "object images, sound images, impressions of touch, and symbols". If the picture of an object known to him was covered by a piece of paper with a slit in the middle, and if this paper was moved about so that only successive parts of the picture became visible, he was unable to put the visual impressions of these parts together; yet when the paper was removed he could at once see and recognize the picture as a whole. When a written or printed word was covered in the same way, letters being exposed singly in succession, he read them one by one:

1 [Der genaue Wortlaut bei Grashey (1885), S. 669: "Auf diese Weise ließe sich schliesslich jedes Symptom erklären. Von dieser Überzeugung ausgehend, habe ich mich mit der willkürlichen Ein- und Ausschaltung leistungsfähiger Verbindungsbahnen nicht begnügt, sondern den Kranken eingehender untersucht und gefunden, dass die Centren für Objectbilder, Klangbilder und Symbole nur scheinbar normal, in der That aber in ihren Funktionen erheblich gestört sind."]

2 [Ibid.]

1 [A. a. O., S. 673.]
The effect of shortening of the perception time, but was unable to read the word; having arrived at the last letter he had forgotten the preceding ones.

Grashey explained his patient's speech disorder by this general impairment of perception, without postulating a localized lesion. An object, he argued, could be perceived visually even if exposed only for a brief fraction of time. A sound image he believed to require a longer period to be perceived because it needed time to develop from successive stimuli. If the time allowed for the visual perception of an object was as low as 0.06 sec., it could still be perceived as a whole, while of its respective sound impression, i.e., its name, only the first letter could be perceived auditorily in that space of time. However, the parts of the object images and of the sound images did not correspond; the sound of the word “horse” had no relation to any part of the object “horse”; the sound image had to be complete before it could be related to the object. “If therefore, an object image is to elicit a sound image, the former must be complete and continue long enough to allow the successive parts of the sound image to emerge. If the time allowed for exposition of the object image ‘horse’ drops below 0.06 sec., only a fraction, i.e., a single letter of the sound image can be elicited.”—“If, on the other hand, an image of the object is to be elicited from the sound image no part of the emerging sound image can elicit a part of the visual object image because the parts of the images do not correspond to each other. The sound image must be complete and continue long enough to enable the image of the object to emerge.” As the object image required only a moment to emerge a sound image of very short duration sufficed.

Grashey concluded that one and the same disorder was apt to alter the transition from images of objects to the sound images, while the transition of

Erklärung desselben durch Verkürzung in der Dauer der Perzeption.
aber das Wort nie lesen, weil er beim letzten Buchstaben alle früheren vergessen hatte.²

Aus dieser allgemeinen Schädigung der Perzeption erklärte nun Grashey die Sprachstörung seines Kranken, ohne eine lokализierte Läsion annehmen zu müssen. Ein Objekt, führt er aus, kann vom Auge auch bei momentaner Einwirkung des Lichtes wahrgenommen werden; ein Klangbild braucht zu seiner Auffassung eine längere Zeit, weil es für unser Ohr ein werdendes, sukzessive entstehendes Objekt ist. Sinkt die Dauer des Objekteindruckes auf 0,06 Sekunden herab, so kann dieses noch als Ganzes erfasst werden, während das zugehörige Klangbild in derselben Zeit nur in seinem ersten Buchstaben erfasst werden kann.³ Objektbild und Klangbild entsprechen einander aber nicht Teil für Teil, vom Worte ‘Pferd’ entspricht z. B. der Klang ‘P’ keinem Teil vom Objekte Pferd; das Klangbild muß erst fertig geworden sein, ehe es eine Beziehung auf das Objekt erfahren kann. »Soll also von einem Objektbild ein Klangbild hervorgerufen werden, so muss das Objektbild fertig sein und so lange dauern, bis successive die einzelnen Theile des Klangbildes entstanden sind. Sinkt die Dauer des fertigen Objectbildes Pferd auf den Werth von 0,06 Sekunden, so kann von diesem Objectbild aus höchstens noch ein einziger Theil, ein Buchstabe des Klangbildes hervorgerufen werden.«¹ — »Soll umgekehrt von einem Klangbild ein Objectbild hervorgerufen werden, so kann ebenfalls kein Theil des entstehenden Klangbildes irgend einen Theil des Objectbildes erregen, weil die Theile dieser Bilder einander nicht entsprechen. Das Klangbild muss vielmehr fertig sein und so lange dauern, bis das Objectbild entstanden ist.« Da das Objectbild zu seiner Entstehung aber nur eines Momentes bedarf, so kommt es auch bei verkürzter Dauer des Klangbildes zustande.

»Man sieht also«, schließt Grashey, »dass durch eine und dieselbe Störung [...] der Uebergang von den Objectbildern zu den Klangbildern alterirt, der Uebergang von den

2 [A. a. O., S. 672.]
3 [A. a. O., S. 674 f.]
1 [Wörtlich bei Grashey, a. a. O., S. 676 f., so: »Soll von einem Objectbild ein Klangbild hervorgerufen werden, so kann nicht ein bestimmter Theil des Objectbildes einen bestimmten Theil des Klangbildes erregen, son dern das Objectbild muss fertig sein und so lange dauern, bis successive die einzelnen Theile des Klangbildes entstanden sind. Bedarf also das Klangbild zu seiner Entstehung 0,3 Sekunden, so muss das fertige Objectbild mindestens ebenso lang im Bewusstsein vorhanden sein. Sinkt aber die Dauer des fertigen Objectbildes z. B. auf den Werth von 0,06 Sekunden, so kann von diesem Objectbild aus höchstens noch ein einziger Theil, ein Buchstabe des Klangbildes hervorgerufen werden.«]
Naming with the help of the first letter of the required word sound images into object images remained unchanged. We add: without the assumption of a lesion in any tract or centre.

Grashey's patient showed yet another peculiarity. He was able to recall the names of objects by writing them, but only when allowed to look at the object at the same time. He would glance at it and write down the first letter of its name, read that letter and repeat it several times; then he would look at the object again, write the second letter, pronounce the first two letters and continue in this manner until he had produced the last letter and with it the required name. This peculiar procedure could be satisfactorily explained with an abnormally short duration of the single perceptions if one presumed that the writing down and reading of the letters were means of fixating the fleeting perceptions. Grashey was justified in concluding from this observation that successive parts of the sound images, of the cheiro- kinaesthetic and the visual word images corresponded to each other, and that their association could help in eliciting the word even when the duration of the single perception had declined considerably.

It therefore appeared proven that there were cases of aphasia in which no localized lesion needed to be assumed and the symptoms of which could be attributed to an alteration of a physiological constant in the speech apparatus. "Grashey's aphasia" could be clearly distinguished from the aphasias described by Wernicke and Lichtheim and caused by localized lesions. It seemed possible that discovery of further functional mechanisms other than the reduction of the time of exposition of sensory impressions may result in clarification of other forms of "amnesic aphasia".

However, Wernicke himself subjected Grashey's analysis to a trenchant criticism and demolished its basic
Critique of Grashey's explanation.

assumptions. He pointed out that the sound image was not perceived as a sequence of letters. The word sound was a whole, which only later in life could be broken down into sounds of letters to meet the requirements of writing. Nor did Wernicke overlook another weighty objection to Grashey's hypothesis: if the patient had built up the word sound from the sounds of the constituting letters, his hearing could not have been better than his reading, and he would have been unable to understand one single word without putting it down in writing. Wernicke expressed this objection thus: “The same patient who, when shown various successive objects or letters, invariably forgets the preceding ones, can read fluently, understands everything said to him and can write to dictation. To understand a word or sentence, the sound of several successive letters and words respectively must be retained by the patient long enough to enable their meaning to be comprehended. The sound images, therefore, are in this case retained much longer than visual object images and the memory disorder is in a certain sense localized, i.e., it concerns chiefly the area of visual activity.” (p. 470.)

We note that Wernicke was unable to explain Grashey's case without assuming a localized and select functional disturbance. However, the emphasis on the visual dysfunction cannot satisfactorily explain the peculiarities of Grashey's case. We also remember that Grashey established that the sound images, too, were of extraordinarily brief duration in his case. Furthermore, unless the duration of the sound images had been markedly reduced, it would be impossible to understand why the patient needed to fixate them, by writing and reading, once they had emerged;

1 [Im Original: »außerordentliche«.]

Kritik der Erklärung Grasheys.

macht darauf aufmerksam, daß man das Klangbild ja nicht als aus Buchstaben bestehend hört. Der Klang ist etwas Ganzes, dessen Zerlegung in Buchstabenklänge erst später im Leben zum Zweck des Einvernehmens mit der Schriftsprache erfolgt. Es entging Wernicke auch nicht, daß die Auffassung Grasheys, einem anderen gewichtigen Bedenken ausgesetzt war. Wenn der Kranke darauf angewiesen war, den Klang des Wortes aus den Buchstabenklängen zusammenzusetzen, so konnte sein Hören nicht besser sein als sein Lesen, er hätte unfähig sein müssen, auch nur ein Wort zu verstehen, ohne es durch Schreiben zu fixieren. Wernicke drückte diesen Einwand folgendermaßen aus: »Derselbe Kranke, der, wenn ihm verschiedene Objecte oder auch Buchstaben nach einander gezeigt werden, jedesmal über dem zweiten den ersten vergisst, kann fließend lesen, versteht Alles, was zu ihm gesprochen wird, kann Wörter auf Dictat schreiben. Um ein Wort, einen Satz zu verstehen, muss der Klang mehrerer Buchstaben, bei Sätzen der Klang vieler Wörter dem Patienten so lange im Gedächtniss haften, bis der Sinn des Satzes verständlich zum Ausdruck gekommen ist. Die Klangbilder haben also hier eine viel längere Dauer als die optischen Objectbilder, und die Gedächtnissstörung ist in gewissem Sinne localisirt, indem sie [so] vorzugsweise das optische Gebiet betroffen hat.« (p. 470 [469 f.].)

Wir nehmen es zur Kenntnis, daß Wernicke den Fall Grasheys nicht anders als durch eine lokalisierte (also ungleichmäßige) Funktionsstörung zu erklären weiß. Allein wir können nicht zustehen, daß die Versetzung dieser Störung ins optische Gebiet die Eigentümlichkeit der Grasheyschen Beobachtung befriedigend aufklärt. Wir erinnern uns z. B., daß Grashey die außerordentlich kurze Dauer auch der Klangbilder für seinen Fall direkt erwiesen hat. Ferner wäre, wenn nicht die Dauer der Klangbilder in maßgebender Weise verringert ist, nicht zu verstehen, wozu der Kranke der Fixierung des gefundenen Buchstabens durch Schreiben und Ableseen bedarf;
The need for assuming a localized lesion.

he ought to have arrived at the whole of the sound image without special help if the perception of the object was renewed often enough.

Grashey's case therefore, calls for a different explanation and I hope that the one to be presented here will prove unassailable. The general reduction in the duration of sensory impression obviously cannot result in a speech disorder such as the one under discussion. Rieger,1 carefully examined a patient with a very similar memory disorder, also caused by a trauma, and he paid due attention to the patient's speech. He had difficulties in finding nouns and adjectives in spontaneous speech and required constant coaxing to produce the names of objects shown to him. He succeeded in doing so only after a long interval. This was not used for building up words by way of spelling, but they were uttered explosively. All this suggests that in Grashey's case a localized lesion must have been present in addition to the general impairment of memory, and that the lesion was situated in the centre for the sound images. The case presented an example of Bastian's second level of reduced excitability when a centre fails to respond to normal, associated with the sound image. The case of Grashey's centre for the sound images could no longer be stimulated directly through object associations, but still permitted the conduction of the stimuli to the visual word images associated with the sound image. Of the former, the first part (letter) could be recognized during the fraction of the moment, as the sound image without special help, but is still reacting to association and sensory stimulation. In Grashey's case the centre for the sound images could no longer be stimulated directly through object associations, but still permitted the conduction of the stimuli to the visual word images associated with the sound image. Of the former, the first part (letter) could be recognized during the fraction of the moment.

1 Rieger: Beschreibung einer Intelligenzstörung infolge einer Hirnverletzung nebst einem Entwurf zu einer allgemein anwendbaren Methode der Intelligenzprüfung (Description of a disorder of intelligence due to a brain injury, together with a design for a generally applicable method of testing intelligence). Wiesbaden, 1888.
Grashey's case illustrates of time when the stimulus arising from the seen object took effect; through repetition of this process the other parts emerged. The letters of the visual word image thus assembled elicited the sound image which could not be activated through the object associations alone.

My interpretation is strongly supported by the fact that Grashey's patient was at first word deaf, which implies the presence of a gross damage in the very area a moderate lesion of which would explain the speech disorder described by Grashey. I am, of course, still of the opinion that the auditory part of the speech apparatus reacted as a whole to this lesion, in the same way as it did in the transcortical motor aphasia discussed earlier.

Cases such as that described by Grashey have been known before. A patient reported by Graves had, following a stroke, lost his memory for nouns and adjectives, but was able to remember their first letters with unfailing certainty. He found it helpful to draw up an alphabetic list of the most commonly used nouns which he always carried with him. When he required a word he looked it up under its first letter; having recognized the wanted noun by its visual word image he was able to pronounce it as long as he kept his eyes fixed on the written word. As soon as the book was closed he forgot the word. It is obvious that this patient, too, could make missing words available by establishing an association with their visual word images.

In the study of speech disorders it has often been observed that, for speech to be produced, the activity of a centre requires to be assisted by the activity of another...
associated with it. The visual centre (area of the letter images) shows this need most frequently, and in such cases reading is impossible unless the individual letters are copied, or drawn in the air. Westphal was the first to report such an observation in an aphasic patient who could read only when carrying out writing movements at the same time. In Charcot's \(^3\) *New Lectures* which I have recently translated, we find the full history of another word blind patient who availed himself of the same mechanism. Thus the aphasias simply reproduce a state which existed in the course of the normal process of learning to speak. As long as we were still unable to read fluently, we endeavoured to make sure of the visual word images by arousing all other images associated with them, and equally, when learning to write, we used to stimulate the sound images and the motor kinaesthetic images, in addition to the visual word images. There is only one difference; when learning, we are restricted by the hierarchy of the centres which started functioning at different times; the sensory-auditory first, then the motor, later the visual and lastly the graphic. In pathological cases, however, the centre which has suffered least is the one the assistance of which is sought first. The cases of Graves and Grashey were peculiar only in that the centre of the sound impressions (images) required support from centres which otherwise are dependent on it.

Although Grashey's study has not proved as important for the elucidation of amnesic aphasia without localized lesions as it seemed to be at first, it can still claim lasting

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Anmerkung des Übersetzers.*


* [Vgl. auch unten, S. 131 ff.]
The significance of Grashey's observation for the understanding of alexia.

value by virtue of several subsidiary findings. It was the first study to investigate the mutual relationship of the speech centres and their dependence on the centre of the sound impressions, and the first to give us a glimpse of the complicated and often devious course of the associations underlying the processes of speech; finally, by proving that reading is accomplished by spelling, it has established the correct approach for the evaluation of the reading disorders. With regard to the last statement, a qualification might be called for. It is probable that for certain types of reading, especially for certain words, the object image of the whole word contributes to its recognition. This explains why persons who are "letter blind", i.e., unable to read single letters, can nevertheless read their own names or some words very familiar to them, such as the name of a town or a hospital, and why one of Leube's patients was occasionally able to pronounce a word which she had in vain endeavoured to spell, as soon as it was withdrawn from her sight, i.e., as soon as she was stopped in her attempts at spelling it. Leube suggested that in this case the object image of the printed or written word had by then imprinted itself deeply enough to be read as a whole.

Our starting point was a concept of speech disorder according to which some forms of aphasia could be explained solely as the effects of circumscribed lesions of tracts and centres, while the rest could be fully understood as functional changes in the apparatus of speech. We pointed out that in the case of the transcortical motor aphasia the former explanation alone was not applicable.

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The significance of Bastian's modifications

and that functional changes had to be assumed for this particular disorder. On the other hand, a critical appraisal of Grashey’s case led us to the conclusion that amnesic aphasia could not be explained without the assumption of a localized lesion. We proposed to reconcile these apparently incompatible assumptions by means of the hypothesis that the centres of the speech apparatus reacted as wholes with an alteration of function to partial lesions, and we adopted as such modifications of functions Bastian’s three levels of reduction of excitability; i.e., a centre may be (1) no longer excitable at all; (2) excitable only by sensory stimulation; and (3) excitable in association with another centre. We expect to find an interruption of fibre tracts as well as a modification of function in any given case of speech disorder. The question arises, therefore, which features of an aphasia are attributable to the one and which to the other cause. Our approach calls for a concept of aphasia which is not open to the objections discussed earlier.

V

In one of the preceding chapters it was pointed out that Wernicke’s theory of the speech process was based on a specific assumption concerning the role of “centres” in the cerebral cortex, and that clinical observation had failed to bear out certain anticipations which such an assumption would warrant. It appears necessary, therefore, to subject Wernicke’s theory to a closer scrutiny. According to him, there are certain fairly well defined areas in the cortex, such as Broca’s and
Wernicke’s areas, the nerve cells of which contain the images (impressions) essential for the process of speech. These images are residues of impressions which reach the brain via the visual or auditory nerves, or which originate as sensations of innervation or of perceptions of movements carried out in the act of speaking. Depending on their origin from one of these sources, they lie together in the cerebral cortex: one area is assumed to contain all “word sound images”, another all “glosso-kinaesthetic word images or impressions”, etc. These cortical centres are interconnected by white fibre tracts (association tracts) and in between the centres there is unoccupied cortical territory, i.e., the “functional gaps” according to Meynert. With the latter term we have introduced a part of Meynert’s teaching into Wernicke’s concepts. Wernicke, who never fails to mention that his theory of aphasia is only an application of Meynert’s basic teachings, tended at first to deviate from them in his writings on the speech centres. In his essay on the aphasic syndrome he still regarded the whole of the convolutions around the Sylvian fissure as speech area; later on, however, in his textbook on the diseases of the brain, only parts of the first frontal and first temporal convolutions were presented as speech centres (Fig. 7).

It appears appropriate at this point to consider Meynert’s doctrine of the organization and the functions of the brain. My presentation of them here, and the objections raised against them, will be only cursory and sketchy; it cannot possibly do justice to their great importance. However, a full discussion of these theories

Meynert's theory

would be beyond the scope of this study which is intended to deal with the concepts of aphasia only. But as the latter cannot be viewed independently of a broad theory of cerebral activity I cannot avoid at least touching upon the general problem of brain function. Meynert's theory of the organization of the brain deserves to be named "cortico-centric". In his far-reaching speculations on anatomical conditions, which are so typical of him, Meynert expressed the view that the cerebral cortex, by its superficial situation, was particularly suited for the reception and retention of all sensory stimuli. He also compared the cerebral cortex to a complex protoplasmic organism which expanded over an object it wanted to incorporate by taking

2 In speaking of the cortical origin of the spinal cord Meynert referred to efferent as well as afferent tracts. Freud has occasionally followed this usage in this book. (Transl.)
The projection of the body in the cortex.

In addition, the nuclei pontis and the fibrae arcuatae provide a connection with the cerebellum which is otherwise somewhat neglected in Meynert's map of the brain.

1 In what way, then, is the body represented in the cerebral cortex which is connected with the periphery by means of these tracts? Meynert calls this representation a "projection", and some of his comments indicate that he actually envisages a projection, i.e., a point by point representation, of the body in the cerebral cortex. This is suggested by the frequent analogy he draws between cerebral cortex and retina. The latter is an end organ which several writers have called an "end organ", which several writers have called an "end organ", while morphologically one would expect it to correspond to a piece of spinal grey matter.

The following is one of several passages which suggest that Meynert did think of projection in the strict sense of the term: "It is most improbable that every single fibre tract representing different groups of muscles, areas of the skin, glands and bowels should disperse sufficiently to be represented, by projection, over the whole surface of the cortex"; or "a cross section through the cerebral peduncles contains as it were the whole organism though without the senses of smell and sight". However, other parts of Meynert's teachings contradict such an assumption and I hesitate to attribute it to him. However, there can hardly be any doubt that Munk and other workers who have accepted the principles of Meynert's teachings, have more or less explicitly propagated the concept of a complete and topographically exact representation of the body in the cerebral cortex.

1 Meynert: *Bau der Grosshirnrinde* (Organization of the cerebral cortex), l. c., p. 83.

2 Idem: *Riickenmarksursprung* (Origin of the spinal cord), l. c., p. 488.

Abbildung des Körpers in der Hirnrinde.

| werden. In der Brücke findet sie über- | 49 |
| dies vermittelst der grauen Substanz der Brückenkern, welche durch solche Bahnen mit der Peripherie verbunden ist? | 49 |
| Meynert nennt diese Abbildung eine "Projection"1, und einige seiner Bemerkungen lassen schließen, da | 49 |
|ß er in der Tat eine Projektion, d. h. eine Abbildung Punkt für Punkt, des Körpers in der Hirnrinde annimmt. | 49 |

1 [Meynert (1884), S. 105, 160. Auch (1869b), S. 550.]
2 [Meynert (1867/68), S. 83, und (1869a), S. 447, 450. Vgl. auch (1869b) und (1884), S. 37 u. 6.]
3 [Etwa Meynert (1884), S. 127, 140, 167; (1867/68), S. 79, 80, 206, 105.]
4 Meynert, Bau der Grosshirnrinde l. c. [1867/68], p. 83. [Hervorhebung von Freud.]
5 Meynert, Rückenmarksursprung l. c. [1869a], p. 488 [richtig: 448].

I wish to draw attention to the fact that the recent advances in the anatomy of the brain have necessitated considerable changes in Meynert's concept of cerebral organization and have thrown doubt on the role attributed by him to the cortex. In particular, his views on the course of the most important and best known pathway from the cerebral cortex to the skeletal muscles had to undergo far-reaching revisions. The first assumption to be dropped was that of the corpus striatum as a ganglion within the motor tract. The clinicians, under Charcot's leadership, have shown that a lesion of this nucleus affects motor function only because of its vicinity to the internal capsule, while lesions of the ganglion which spare the inner capsule are incapable of causing paralysis. Wernicke has demonstrated that this so-called ganglion of the peduncles lacks any substantial connections with the cerebral cortex. Thus the first internodium had been torn out of the course of Meynert's projection tract. The study of the successive phases of myelination confirmed Wernicke's view and revealed a new gap in Meynert's concept of the structure of the brain. Flechsig was able to demonstrate that the motor pathway runs without interruption from the cortex through the internal capsule into the peduncles and that there is no connection in the pons between the motor pathway and the cerebellum. The pyramidal tract is now regarded as the direct connection between the anterior horns of the spinal cord and the cortex. Meynert's idea of the relationship of the cerebellum to the motor tract has been dropped. Of the large subcortical nuclei, only the optic thalamus is connected with the cerebral cortex; it is atrophic in cases of congenital malformation of cerebral lobes; the striate body, however, remains intact.

in cases of lobar degeneration, while it is found to be atrophic in patients with congenital cerebellar atrophy. Thus a formidable portion of the brain, i.e., the corpora striata, the pons and the cerebellum, can be differentiated, as an organ of unknown function, from the rest of the brain with which it has a great many connections, though developmentally and functionally it is fairly independent of it. Although Meynert’s hypothesis of the two levels of the cerebral peduncles can no longer be maintained, no alternative hypothesis has so far taken its place. If there be any question of a double origin of the spinal cord, it can only mean one origin in the cortex and thalamus (cortico-thalamic origin) and another which is striato-cerebellar. The whole organization of the brain seems to fall into two central apparatuses of which the cerebral cortex is the younger, while the older one is represented by the ganglia of the forebrain which have still maintained some of their phylogenetically old original functions. Another important part of Meynert’s theory, i.e., the assumption of a double sensory tract, one direct and the other reflexory, seems to have remained unconfirmed. Investigations carried out so far have shown that no fibre tract reaches other parts of the brain without having entered into some connection with the grey matter of the spinal cord or similar structures, and that the reflex tracts always originate from sensory fibres.

Thus the theory of the dominant role of the cerebral cortex has been disproved. On the other hand, some processes previously regarded as sub-cortical, have now been allocated to the cortex. The question arises in what way the body is represented in the cerebral cortex. I believe that the assumption of a projection of the body into the cortex in the strict sense, i.e., of an image that is complete and topographically similar, can be rejected.

begleitet sein sollte. Wir besitzen kein Kriterium, welches einen psychischen Vorgang von einem physiologischen, einen Akt in der Großhirnrinde von einem Akt in den subkortikalen Massen exakt zu trennen gestattete [...]. Zur Fortführung dieser Gedanken bei der Erörterung der Frage nach der Lokalisation seelischer Vorgänge vgl. unten, S. 97, Anm. 1.]
I propose to take Henle's concept of the reduction of fibres through grey masses as my point of departure. If one compares the total number of fibres entering the spinal cord from the periphery with that of the fibre tracts leaving the cord to connect it with the brain, the latter proves to be only a fraction of the former. According to Stilling's count, in one instance 807,738 fibres of a nerve root corresponded to no more than 365,814 fibres in a cross section of the upper cervical cord. It follows that the relationship of the spinal cord to the body is different from its relationship to the grey masses of the brain. Only in the spinal cord, and in analogous grey areas, do the prerequisites for a complete projection of the body periphery exist. For each peripheral unit of innervation there exists a corresponding area of grey matter in the spinal cord, and in the extreme case one single nervous element. Owing to the reduction of the projection fibres through the grey matter of the spinal cord, a unit of grey matter belonging to a higher level can no longer correspond to one peripheral unit, but must be related to several such units. This also holds for the cerebral cortex, and it is therefore appropriate to use different terms for these two types of representation in the central nervous system. If the way in which the periphery is reflected in the spinal cord is called a "projection", its counterpart in the cerebral cortex might suitably be called a "representation", which implies that the periphery of the body is contained in the cerebral cortex not point by point, but through selected fibres, in a less detailed differentiation.

This simple argument requires further elaboration in a different direction; not all the fibres of the highest cross section of the spinal cord serve the connection with the cerebral cortex.

1. [Stilling (1859), S. 602, 606. – Mit den grundlegenden Werken von Henle und Stilling hatte Freud sich schon früher bei seinen ersten wissenschaftlichen Forschungen über das Nervensystem des Petromyzon (1877 a) und (1878 a) beschäftigt.]

1. [In seinem französischen Aufsatz (1893 c), S. 41, bezieht sich Freud auf die obige Argumentation und schlägt analog dazu die Unterscheidung zwischen "paralysie de projections" (Projektionslähmung) und "paralysie de représentation" (Repräsentationslähmung) vor. – Der Gedanke, daß der gesamte Organismus (i. e. Eindrücke [impressions] und Bewegungen von allen Körperteilen) auf verschiedenen hierarchischen Evolutionsebenen des Zentralnervensystems mehr oder weniger "direkt" bzw. "indirekt" "repräsentiert" wird, spielt im Werk Hughlings Jacksons eine zentrale Rolle; vgl. u.a. Jackson (1884), (1887 a) und (1887 b).]
Reduction of fibre tracts through grey masses.

A considerable portion, especially those forming the short pathways, exhausts itself within the ventricular grey matter between the nuclei of the medulla, while another portion enters the cerebellum. Only of the pyramidal tract can it be said with certainty that its size in the brain is the same as in the cervical cord, and this tract is no doubt a greatly reduced continuation of the fibres which connect the muscles of the body with the spinal cord. On the other hand, the reduction of the projection fibres is not as great as it might appear from these considerations; if we take the example of the posterior tracts, a certain proportion of their fibres do not reach the cortex, but in their place the latter receives the fibres of the fillet which after various interruptions in the nuclei of the posterior tracts, the oblongata and the thalami, finally represent the posterior tracts in the cerebral cortex. It is not known whether the fibres of the fillet are equal in number to those of the posterior tracts; probably they are much less numerous. In addition, the cerebrum receives fibres from the cerebellum which could be regarded as an equivalent for the cerebellar connections of the spinal cord. It is therefore quite possible that the cerebral cortex receives at least as many fibres from the periphery, though by devious routes, as were required for projection in the spinal cord.

There is yet another aspect which has not been made sufficiently clear in Meynert's presentation. For Meynert, who in describing pathways is mainly concerned with their cortical connections, a fibre or a fibre tract retains its identity even after having passed through an unlimited number of nuclei. This is indicated by his phrase: "the fibre passes through a grey substance". This naturally gives rise to the impression that the fibre remains the same on its long way to the cortex, apart...
The change of the functional significance of fibres on their way to the cortex.

...from the fact that it has possibly entered into several connections. This view can no longer be maintained. If we observe how in the course of individual development myelinization proceeds piecemeal from one nucleus to another, and how for one afferent fibre tract three or more efferent tracts emerge from one single nucleus, these grey masses, and not the fibre tracts, appear to be the organs of the brain. If we follow the course of a sensory afferent tract as far as we know it, and if we regard its frequent interruptions in grey nuclei and its arborizations through them as characteristic, we cannot but assume that the functional significance of a fibre on its way to the cerebral cortex has changed each time it has emerged from a nucleus. Let us take one of the better understood examples; a fibre of the optic nerve conveys a retinal impression to the anterior quadrigeminal body; here it terminates, and in its place another fibre goes from the ganglion to the occipital cortex. However, in the grey substance of the quadrigeminal body the retinal impressions become associated with a kinaesthetic oculomotor impression; it is extremely likely that the new fibre between quadrigeminal body and occipital cortex no longer conveys a retinal stimulus, but the association of one or more such impressions with kinaesthetic impressions. The complexity of this change in the functional significance of the fibres must be even greater in the fibre systems serving skin and muscle sensations; we are still quite ignorant.

1 See Edinger's, Bechterew's and the present author's investigations into the course of the posterior tracts and of the auditory nerve.

54 Bedeutungsänderung der Fasern auf ihrem Wege zur Rinde.


1 Vgl. unten, S. 134f.

The body is not represented topographically in the cerebral cortex, which contribute to the functional changes that conducted stimuli undergo. We can only presume that the fibre tracts, which reach the cerebral cortex after their passage through other grey masses, have maintained some relationship to the periphery of the body, but no longer reflect a topographically exact image of it. They contain the body periphery in the same way as—to borrow an example from the subject with which we are concerned here—a poem contains the alphabet, i.e., in a completely different arrangement serving other purposes, in manifold associations of the individual elements, whereby some may be represented several times, others not at all. If it were possible to follow in detail the rearrangement which takes place between the spinal projection and the cerebral cortex, one would probably find that the underlying principle is purely functional, and that the topographic relations are maintained only as long as they fit in with the claims of function. As there is no indication that this rearrangement is reversed in the cerebral cortex to produce a topographically complete projection, we may suppose that the representation of the body periphery in the higher parts of the brain, and also in the cortex, is no longer topographical but only functional. The animal experiment is bound to obscure this fact because it cannot reveal any other but a topographical relationship. However, I believe that those who seriously look for a cortical centre for the musculus extensor pollicis longus, or for the musculus rectus oculi, or for the sensibility of a certain area of the skin, are labouring under a misconception of the function of the cortex, as well as of the complicated conditions which make this function possible.1

1 I should like to point out that this concept of the cortical representation of the body challenges Munk’s views of the point to point projection of the retina in the occipital lobe, and that a study of the cortical hemianopias ought to confirm or refute it.
The localization of psychic elements is the result

After this digression we now return to the problem of aphasia. We remember that, under the influence of Meynert's teachings, the theory has been evolved that the speech apparatus consists of distinct cortical centres; their cells are supposed to contain the word images (word concepts or word impressions); these centres are said to be separated by functionless cortical territory, and linked to each other by the association tracts. One may first of all raise the question as to whether such an assumption is at all correct, and even permissible. I do not believe it to be.

Considering the tendency of earlier medical periods to localize whole mental faculties, such as are defined in psychological terminology, in certain areas of the brain, it was bound to appear as a great advance when Wernicke declared that only the simplest psychic elements, i.e., the various sensory perceptions, could be localized in the cortex, the areas concerned being those of the central terminations of the sensory nerves. But does one not in principle make the same mistake, irrespective of whether one tries to localize a complicated concept, a whole mental faculty or a psychic element? Is it justified to immerse a nerve fibre, which over the whole length of its course has been only a physiological structure subject to physiological modifications, with its end in the psyche and to furnish this end with an idea or a memory? Now that "will" and "intelligence", etc., have been recognized as psychological technical terms referring to very complicated physiological states, can one be quite sure that the "simple sensory impression" be anything else but another such technical term?

The relationship between the chain of physiological events in the nervous system and the mental processes is probably not one of cause and effect. The former do not cease when the latter set in; they tend to continue,

2 [Wernicke (1874), S. 4. Vgl. oben, S. 41.]
of a confusion of "psychic" and "physical".

but, from a certain moment, a mental phenomenon corresponds to each part of the chain, or to several parts. The psychic is, therefore, a process parallel to the physiological, "a dependent concomitant".

I am well aware that the writers whose views I am opposing here cannot have been guilty of thoughtless mistakes in their scientific approach. They obviously mean only that the physiological modification of the nerve fibre through sensory stimuli produces another modification in the central nerve cells which then becomes the physiological correlate of the "concept" or "idea". As they know a lot more about ideas than of the physiological modifications, which are still undefined and unknown, they use the elliptic phrase: an idea is localized in the nerve cell. Yet this substitution at once leads to a confusion of the two processes which need have nothing in common with each other. In psychology the simple idea is to us something elementary which we can clearly differentiate from its connection with other ideas. This is why we are tempted to assume that its physiological correlate, i.e., the modification of the nerve cells which originates from the stimulation of the nerve fibres, be also something simple and localizable. Such an inference is, of course, entirely unwarranted; the qualities of this modification have to be established for themselves and independently of their psychological concomitants.

1 [Auf den psych-o-physischen Parallelismus kommt Freud auch in seinem Entwurf einer Psychologie von 1895 im Abschnitt über das Bewußtsein (1900, S. 403) zu sprechen: "Es handelt sich nur darum, die uns bekannten Eigenschaften des Bewußtseins durch parallel veränderliche Vorgänge in den Nerven zu decken. Auch in (1915; Studienausgabe, S. 126f.) taucht der Begriff wieder auf.]


3 [Von der physiologischen zur psychologischen Betrachtungsweise. (Anm. der Hrsg. der Studienausgabe.)]
Hughlings Jackson has most emphatically warned against such a confusion of the physical with the psychic in the study of speech: "In all our studies of diseases of the nervous system we must be on our guard against the fallacy that what are physical states in lower centres fine away into psychical states in higher centres; that for example, vibrations of sensory nerves become sensations, or that somehow or another an idea produces a movement." Brain I, p. 306.
The impossibility of a separation of idea and association.

What then is the physiological correlate of the simple idea emerging or re-emerging? Obviously nothing static, but something in the nature of a process. This process is not incompatible with localization. It starts at a specific point in the cortex and from there spreads over the whole cortex and along certain pathways. When this event has taken place it leaves behind a modification, with the possibility of a memory, in the part of the cortex affected. It is very doubtful whether this physiological event is in any way associated with something psychic. Our consciousness contains nothing that would, from the psychological point of view, justify the term “latent memory image”. Yet whenever the same cortical state is elicited again, the previous psychic event re-emerges as a memory. We have, of course, not the slightest idea how animal tissue can possibly undergo, and differentiate, so many various modifications. But that it is able to do so is proved by the example of the spermatozoa in which the most varied and highly differentiated modifications lie dormant and ready to develop.

Is it possible, then, to differentiate the part of “perception” from that of “association” in the concomitant physiological process? Obviously not. “Perception” and “association” are terms by which we describe different aspects of the same process. But we know that the phenomena to which these terms refer are abstractions from a unitary and indivisible process. We cannot have a perception without immediately associating it; however sharply we may separate the two concepts, in reality they belong to one single process which, starting from one point, spreads over the whole cortex. The localization of the physiological correlates for perception and association is, therefore, identical, and as localization of a perception means nothing else but localization of its correlate, we cannot possibly have

dargelegten Ausführungen über die Funktion des Sprachapparates formuliert hatte: »Diese Auffassung erfuhr eine Ausgestaltung und Abänderung, nachdem man als den wesentlichen Charakter einer vorbewussten Vorstellung die Verbindung mit Wortvorstellungsernten erkannt hatte (!Das Unbewusste!, 1915[e]).« (Vgl. oben, Ende der Anm. 1, S. 97.)]

1 [Vgl. hierzu und zum folgenden die Erörterungen über den psychischen Apparat wiederum in der Traumdeutung (1900a; Studienausgabe, S. 512ff.).]

3 [Vgl. den Niederschlag dieser Gedankengänge und ihre Weiterschauung im Zusammenhang mit dem Problem des Bewußtwerdens unbewußter Gedankengänge im 7. Kapitel der Traumdeutung (1900a; Studienausgabe, S. 578), wo ebenfalls von «Vorgängen oder Ablaufarten der Erregung» die Rede ist, denen Annahme uns […] nahegelegt wurde». Anstelle lokalisatorischer Gleichnisse setzen wir ein, was dem realen Sachverhalt besser zu entsprechen scheint, daß eine Energiebesetzung auf eine bestimmte Anordnung verlegt oder von ihr zurückgeworfen wird […]]. Wir ersetzen hier wiederum eine topische Vorstellungsweise durch eine dynamische; nicht das psychische Gebilde erscheint uns als das Bewegliche, sondern dessen Intervallation. In einem Zusatzz von 1925 führt Freud diesen Gedankengang dann zusammen mit dem Konzept, das er inzwischen in Weiterentwicklung der unten auf S. 116–122
The association tracts are situated in the cortex itself.

a separate cortical localization for each. Both arise from the same place and are nowhere static.

With this refutation of a separate localization for ideation and association of ideas we have disposed of an important reason for differentiating between centres and pathways of speech. In every part of the cortex serving speech we have to assume similar functional processes, and we have no need to call on white fibre tracts for the association of ideas within the cortex.

There is a post-mortem finding which proves that the association of ideas takes place through the fibres situated in the cortex itself. I am again referring to Heubner's case which has already taught us an important lesson. Heubner's patient showed the form of speech disorder called by Lichtheim transcortical sensory aphasia and attributed by him to interruption of the fibre tracts from the sensory speech centre to the areas serving the association of concepts. According to his theory a lesion in the white matter underlying the sensory centre was to be expected in this case. Instead, post-mortem examination revealed a superficial cortical softening which separated the otherwise intact sensory centre, which was functioning normally, from most of its cortical connections outside the speech area. Heubner did not fail to emphasize the importance of this finding, and Pick drew the same conclusions from it as we do, i.e., that the speech association tracts seem to go through the cortex itself.

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1 Pick: Ueber die sogenannte Re-evolution (Hughlings Jackson) nach epileptischen Anfällen nebst Bemerkungen über transitorische Worttaubheit (On the so-called re-evolution [Hughlings Jackson] following epileptic seizures, with observations on transitory word deafness). Arch. f. Psychiat., XXII, 1891.

The hypothesis of "functionless gaps".

This does not, of course, exclude the possibility of subcortical association tracts contributing to this function.

Our conception of the speech apparatus will undergo a thorough transformation when we finally consider the third stipulation of the Meynert-Wernicke theory, i.e., that the areas functioning as speech centres are separated by "functionless gaps". A statement such as this, apparently based on morbid anatomy, seems at first unassailable. However, if one examines the way in which distinct centres were inferred from post-mortem findings, it becomes clear that morbid anatomy is incapable of deciding this question. One only needs to glance at the diagram in which Naunyn entered the extent of the lesions in seventy-one cases of aphasia. The areas which showed the greatest overlapping of lesions were regarded as the speech centres, i.e., as the areas the intactness of which was indispensable for speech to function normally. There might, however, be other cortical areas also serving speech, though their destruction can be tolerated more easily. If there are such areas we shall not be able to detect them from the study of Naunyn's tables. Possibly a speech disorder caused by lesion of areas other than the speech centres, may be due to a remote effect which such lesions exert on the centres; it is also possible that the areas which in the table appear less frequently involved, are also speech centres but not indispensable and constant ones.

Let us now turn to the question which functions have been allocated to the so-called functionless cortex between and beside the speech centres? Meynert states quite clearly (Psychiatrie, p. 140): "It follows that in the physiological process of the occupation of the cerebral cortex by memory..."
The role of the "functionless gaps"

images an increasing number of cortical cells are thus involved; on this process the growth of the child's range of imagery depends. Very probably the receptivity of memory, which is the basis of all intellectual achievements, is limited by the number of cortical cells available." The latter sentence can be taken to mean that not only the intellectual development in childhood but also the acquisition of later knowledge, such as the learning of a new language, depends on the occupation of hitherto unoccupied cortical areas, similar to the way in which a town expands by people settling in areas outside its walls.

In an earlier note, Meynert had allocated the task of taking over the functions of the speech centres, in the case of experimental or other lesions, to the unoccupied areas within their neighbourhood. This theory derived support from experiments carried out by Munk whose hypotheses had been based entirely on Meynert's teachings.

Having revealed the trend underlying the assumption of the "functionless gaps" in the cerebral cortex we can now proceed to examine its usefulness for the understanding of the aphasics. In doing so we find that the exact opposite of what would have to be expected according to the above theory, actually takes place. The function of speech presents the most perfect examples of new acquisitions. Learning to read and to write is one of them, compared with the primary activity of speaking; this acquisition can be impaired by lesions localized outside the speech centres because additional sensory functions, i.e., the visual and the cheiro-motor, are involved in these activities. But if I learn to understand and to speak several foreign languages, or acquire the knowledge of the Greek or Hebrew alphabet in addition to the one I learned first, or practise shorthand and other

1 [Vgl. Meynert (1884), S. 134ff.]
2 [Ibid.]
Association and superassociation.

forms of writing beside my cursive handwriting, I am acquiring abilities which may demand memories many times more numerous than the original language; all these new acquisitions of the faculty of speech are obviously located in the same areas which we know as the centers for the first learned language. It never happens that an organic lesion causes an impairment affecting the mother tongue and not a later acquired language. If in the case of a German who understands French, the word sounds of the latter language had a different localization from the German word sounds, it ought to happen occasionally that following a cerebral softening such a patient would cease to understand German while still understanding French. In fact, the opposite is invariably the case, and this applies to all functions of speech. In reviewing relevant case material which, unfortunately, is much too small considering its great theoretical interest, I could find only two factors determining the character of the speech disorders in the polyglot; (1) the influence of the age of the acquisition, and (2) that of practice. These two factors work as a rule in the same direction. It is noteworthy that if they differ, the earlier acquired language may survive the one in greater use. But their relationship can never be explained by an unusual localization but only by the two functional factors mentioned. Obviously a new set of associations is capable of being superimposed on the established associations involved in speaking; we are clearly aware of this process as long as the new associations can be carried out only with difficulty. The superimposed set of associations is impaired before the primary one, wherever the site of the lesion. Perhaps there is no better illustration of the degree to which a moderately severe modification of the speech apparatus can persist in spite of damage, contrary to all theories of localization of ideas, than the following which I am borrowing from

1 [Vgl. unten, S. 132.]
2 [Diese Tatsache verwendete Freud als eines der Kenntzeichen, durch welche sich die hysterischen von den organischen Sprachstörungen unterscheiden. Vgl. (1893 c), S. 44: »Du syndrome de l’aphasie organique [...] ce qui est chose inouïe dans l’aphasie organique, elle [l’hystérie] peut créer une aphasie totale (motrice et sensitive) pour telle langue, sans attaquer le moins du monde la faculté de comprendre et d’articuler telle autre [...]«]
3 [Vgl. unten, S. 132 f.]
4 [Vgl. unten, S. 132, aber auch S. 120, Punkt 5.]
5 [Vgl. unten, S. 133, Punkt 3.]
Hughlings Jackson. This author, on whose views I have based almost all the arguments which I have advanced in refuting the localizatory theory of the aphasia, discussed the not unusual case of the motor aphasic who, apart from "yes" or "no", has retained a residue of speech which otherwise would represent a complex activity of language. This residue frequently consists of a vigorous curse (sacre nom de dieu, Goddam, etc.); Hughlings Jackson points out that even in normal persons such an utterance belongs to the emotional and not to the intellectual language. In other cases, however, this recurrent utterance is not a curse, but a phrase of special significance; one might regard it as very amazing indeed that exactly these cells or these memory traces should have escaped the general destruction. Some of these cases, however, permitted a very plausible interpretation. For instance, a man who could say only "I want protection", owed his aphasia to a fight in which he had been knocked unconscious by a blow on the head. Another patient had the curious speech remnant: "List complete": he was a clerk who had a stroke immediately after he had laboriously completed a catalogue. Such instances suggest that these utterances are the last words produced by the speech apparatus before injury, or even at a time when there already existed an awareness of the impending disability. I am inclined to explain the persistence of these last modifications by their intensity if they happen at a moment of great inner excitement. I remember having twice been in danger of my life, and each time the awareness of the danger occurred to me quite suddenly. On both occasions I felt "This is the end", and while otherwise my inner language proceeds with only indistinct sound images and slight lip movements, in these situations of danger I heard these words as if somebody was shouting them.

The speech region as a continuous cortical area.

into my ears, and at the same time I saw them as if they were printed on a piece of paper floating in the air.

We have rejected the assumptions that the speech apparatus consists of distinct centres separated by functionless areas, and that ideas (memories) serving speech are stored in certain parts of the cortex called centres while their association is provided exclusively by subcortical fibre tracts. It only remains for us to state the view that the speech area is a continuous cortical region within which the associations and transmissions underlying the speech functions are taking place; they are of a complexity beyond comprehension.

How can such a theory explain the existence of the speech centres, especially of the areas of Broca and Wernicke established by morbid anatomy? A glance at the convexity of a left hemisphere will enlighten us; the situation of the so-called speech centres suggests an explanation which fits well into our theory. These centres lie far apart; according to Naunyn, they are situated in the posterior part of the first temporal convolution, the posterior part of the third frontal gyrus and in the inferior part of the parietal lobe where the angular gyrus merges into the occipital lobe; the site of a fourth centre, for writing, does not seem to be definitely established (?posterior part of the middle frontal gyrus). These areas are situated in such a way that there lies between them a large cortical region, i.e., the insula with the convolutions covering it; the lesion of any part of this area is probably always associated with speech disorder. Although the extent of this area...
The centres are the corner stones of the speech region. One cannot be exactly delineated from a survey of lesions found in cases of aphasia, one can nevertheless say that the so-called speech centres form the outlying parts of the speech area assumed by us, and that speech disorders occur when lesions are situated within the external boundaries of these centres, i.e., towards the centre of the hemisphere, while lesions in cortical areas lying outside them are of a different significance. Thus the "centres" appear as the corner stones of the speech territory. Next we have to consider the areas adjoining these centres from outside. Broca's area is immediately adjacent to the centres of the bulbar motor nerves. Wernicke's area is situated in a region which also contains the acoustic termination, the exact localization of which is unknown; the visual speech centre borders on those parts of the occipital lobe in which we know the optic nerve to terminate. An arrangement such as this, though meaningless from the point of view of the centre theory, has for us the following significance:

The association area of speech, into which visual, auditory, and motor (or kinaesthetic) elements enter, extends for that very reason between the cortical areas of those sensory nerves and the motor regions concerned with speech. If we now imagine a movable lesion of constant size within this association area, its effect will be the greater the more it approaches one of these cortical fields, i.e., the more peripherally it is situated within the speech area. If it borders immediately on one of these cortical fields it will cut off the association area from one of its tributaries, i.e., the mechanisms of speech will be deprived of the visual, or auditory, or some other element, as every association of that nature used to come from that particular cortical field. If the lesion is moved towards the interior of the association area its effect will be more indefinite; in no event will it be able to destroy all possibilities of one particular category of associations. Thus the parts of the speech region bordering on the cortical fields of the optic, auditory and motor cranial nerves.
Consequences of the unilaterality of the speech region.

has the described effect on the speech function only because it has severed the connections with the respective sources of association, the destruction of these receptive and cortical areas themselves ought to have the same result. This, however, is contrary to clinical experience which has established that all such lesions cause localized symptoms without speech disorder. This first objection can easily be disposed of if one considers that all these cortical areas are bilateral, while that of the association area of speech is organized in one hemisphere only. Destruction of one visual cortical area, for example, will not interfere with the utilization of visual stimuli for speech, i.e., with reading, because the speech area retains its connections with the contralateral visual cortex which, in this particular case, is provided by crossed white fibres. If, however, the lesion moves to the boundary of the visual receptive area, alexia ensues, probably because the connection not only with the homolateral, but also with the contralateral visual area has been severed. We therefore have to add to our theory: the appearance of centres is also created by the fact that the fibres from the cortical receptive fields of the other hemisphere enter at the same place, i.e., on the periphery of the speech area where, in case of lesion, the connection with the homolateral receptive areas is also effected. This is plausible, because for the function of speech association the presence of a bilateral origin of visual, auditory and other stimuli is physiologically irrelevant.

The assumption, by the way, that the speech region is connected with cortical areas of both hemispheres is not new but has been taken over from the theory of the centres. The precise anatomy of these crossed connections has not yet been established, but when it is known it might explain some peculiarities in the localization and

Konsequenzen aus der einseitigen Ausbildung des Sprachfeldes.

| Optikus, Akustikus, der Hand, Zunge etc.) anstößt, die geschilderten Folgen für die Sprachfunktion hat, bloß weil dadurch die Verbindungen mit den optischen, akustischen und anderen Assoziationsanregungen unterbrochen ist, so müßte ja die Zerstörung dieser Rindenfelder selbst dieselbe Folge für die Sprache haben. Dies würde aber direkt unseren Erfahrungen widersprechen, welche uns die Lokalsymptome aller solcher Läsionen ohne Sprachstörung nachweisen. Dieser erste Einwand erledigt sich leicht, wenn man in Betracht zieht, daß alle anderen Rindenfelder doppelseitig vorhanden sind, das Assoziationsfeld der Sprache aber nur auf einer Hemisphere organisiert ist. Die Zerstörung des einen optischen Rindenfeldes z.B. wird die Verwertung der visuellen Erregungen für die Sprache (das Lesen) nicht stören, weil das Sprachfeld dabei seine (diesmal durch gekreuzte weiße Fasern) hergestellte Verbindung mit dem optischen Rindenfeld der anderen Seite behält. Rückt die Läsion aber an die Grenze des optischen Rindenfeldes, so tritt Alexie auf, weil nicht nur die Verbindung mit dem gleichseitigen, sondern auch die mit dem gekreuzten optischen Rindenfeld unterbrochen sein mag. Wir haben also die Annahme hinzuzufügen, daß der Anschein von Zentren weiterhin dadurch entsteht, daß die gekreuzten Verbindungen von den Rindenfeldern der anderen Hemisphere an denselben Stellen, nämlich an der Peripherie des Sprachfeldes, hinzukommen, wo auch die Verbindung mit dem gleichseitigen Rindenfeld vor sich geht. Dies ist plausibel, weil ja für die Leistung der Sprachassoziation das doppelte Vorhandensein der optischen, akustischen und anderen Anregungen keine physiologische Bedeutung besitzt.

Es ist dies übrigens keine neue Annahme, sondern eine der Zentrentheorie entlehnte, daß solche Verbindungen des Sprachbezirks mit den beiderseitigen Rindenfeldern existieren. Die anatomischen Verhältnisse dieser gekreuzten Assoziation sind übrigens noch nicht sichergestellt und dürften, wenn bekannt, manche Eigentümlichkeit in Lage
The speech region has no special efferent pathways

extent of the so-called centres, as well as some of the individual features of the speech disorders.

(a) The question may be raised what advantage there be in denying the existence of special centres for speech while we have to assume cortical fields, i.e., centres, for the visual and auditory nerves, and for the motor organs of speech. The answer is that there is no reason why these areas should not be subjected to similar considerations. However, their existence cannot be disputed; their extent is defined by the anatomical fact of the termination of the sensory nerves and the origin of the pyramidal tract in circumscribed areas of the cortex. The region of speech associations, however, lacks these direct relations to the periphery of the body. It certainly has no sensory and most probably no special motor “projection fibres”.

VI

Our concept of the organization of the central apparatus of speech is that of a continuous cortical region occupying the space between the terminations of the optic and acoustic nerves and of the areas of the cranial organs of speech. The answer is that there is no reason why these areas should not be subjected to similar considerations. However, their existence cannot be disputed; their extent is defined by the anatomical fact of the termination of the sensory nerves and the origin of the pyramidal tract in circumscribed areas of the cortex. The region of speech associations, however, lacks these direct relations to the periphery of the body. It certainly has no sensory and most probably no special motor “projection fibres”.

VI

Our concept of the organization of the central apparatus of speech is that of a continuous cortical region occupying the space between the terminations of the optic and acoustic nerves and of the areas of the cranial organs of speech.

1 I reported the main contents of this study in a paper read to the “Wiener physiologischer Club” (Vienna Physiological Club) as early as 1886. However, the statutes of this club do not allow for a claim to priority to be based on its proceedings. In 1887, Naunyn and Naunyn presented their well-known review of the localization of brain diseases to the Congress of Internal Medicine at Wiesbaden. Their views agree with those presented here in several important points. Naunyn’s observations on the concept of cerebral centres as well as Naunyn’s remarks on the topography of the speech area are likely to make readers suspect that my study was influenced by their highly significant view. This was not the case; the stimulus to this study came, in fact, from papers published by Exner jointly with my late friend Josef Paneth in Pflüger’s Archiv.

68 Das Sprachfeld hat keine besonderen zuführenden Bahnen.

1 und Ausdehnung der scheinbaren Zentren sowie manche individuelle Ausprägungen der Sprachstörungen erklären.

2. Man könnte fragen, welchen Wert es wohl hat, besondere Zentren für die Sprachfähigkeit zu bestreiten, wenn wir dabei doch genötigt sind, von Rindenfeldern, also Zentren, des Optikus, des Akustikus und der motorischen Sprachorgane zu reden? Darauf läßt sich erwidern, daß ähnliche Betrachtungen auch für die anderen sogenannten motorischen und Sinnesszentren der Rinde zu wiederholen wären, daß man aber Rindenfelder, selbst besser abgegrenzte, für die anderen Funktionen nicht bestreiten kann, weil solche durch die anatomische Tatsache der Endigung des Sinnesnerven oder des entsprechenden Anteiles der Pyramidenbahn in bestimmten Hirnrindengebieten charakterisiert sein mögen. Das Assoziationsfeld der Sprache aber entbehrt dieser direkten Beziehungen zur Peripherie des Körpers, es hat sicherlich keine eigenen sensiblen und höchst wahrscheinlich auch keine besonderen motorischen »Projectionsbahnen«.

1 [Meynert (1867–68), (1869a), (1884)] passim: »Projektionsbündel«; (1884), S. 37 u. ö.: »Projektionsfasern«, auch: »Projektionsysteme«.


Viert. Unsere Vorstellung vom Aufbau des zentralen Sprachapparates ist also die eines zusammenhängenden Rindengebietes, welches den Raum zwischen den Endstätten des Nervus opticus, acusticus und der motorischen Hirn- und:
All aphasia are due to interruption of conducting fibre tracts.

and certain peripheral motor nerves in the left hemisphere. It probably covers, therefore, the same area which Wernicke was inclined to allocate to speech in his first paper, i.e., all the convolutions forming the Sylvian fissure. We have refused to localize the psychic elements of the speech process in specified areas within this region; we have rejected the supposition that there were areas within this region which were excluded from the speech function in general and kept in reserve for the acquisition of new knowledge of speech. Finally, we have attributed the fact that pathology has demonstrated centres of speech, though of indefinite delimitation, to the situation of the adjoining receptive and motor cortical areas and of the crossed fibre tracts. Thus the speech centres are, in our view, parts of the cortex which may claim a pathological but no special physiological significance. We feel justified in rejecting the differentiation between the so-called centre or cortical aphasias and the conduction (association) aphasias, and we maintain that all aphasias originate in interruption of associations, i.e., of conduction. Aphasia through destruction or lesion of a centre is to us no more and no less than aphasia through lesion of those association fibres which meet in that nodal point called a centre.

We have also asserted that every aphasia is directly, or through some remote effect, caused by disturbance within the cortex itself. This implies that the speech area has no afferent or efferent pathways of its own extending to the periphery of the body. This statement is proved by the fact that subcortical lesions of any location are incapable of producing aphasia, provided anarthria is excluded by definition. Nobody has ever been known to

1 [Im Original: *kennen lernen*]
The subcortical sensory aphasia.

become word deaf as the result of a lesion in the auditory nerve, in the medulla oblongata, in the posterior corpora quadrigemina or in the internal capsule unless he had been deaf already; nor has anybody ever been made word blind by a partial lesion of the optic nerve, or of the diencephalon, etc. However, Lichtheim differentiates a subcortical word deafness and a subcortical motor aphasia, and Wernicke postulates subcortical alexia and agraphia. They do not attribute these types of speech disorder to lesions of subcortical fascicles of association fibres, which in our view cannot be differentiated from association fibres within the cortex itself, but to lesions of radial, i.e., afferent or efferent speech tracts. It is therefore necessary to analyse these subcortical aphasias more precisely.

The characteristic features of a subcortical sensory aphasia can easily be deduced from Lichtheim’s schema which postulates a special auditory tract \( \alpha A \) (Fig. 3) for speech. The patient is supposed to be unable to perceive word sounds, yet capable of availing himself of previously acquired sound impressions and of carrying out all other speech functions faultlessly. Lichtheim actually found such a case; although the early stages of this patient’s illness had not been fully elucidated, his final state entirely conformed to the picture supposed to be caused by interruption of \( \alpha A \). I confess that in view of the importance of the sound images for the speech function I have found it exceedingly difficult to find another explanation for this subcortical sensory aphasia which would make the assumption of an afferent auditory tract \( \alpha A \) unnecessary. I was already inclined to explain Lichtheim’s case by assuming that individual speech might be independent of the sound images; the patient was a highly educated journalist. But such an explanation would quite rightly have been regarded as a mere subterfuge.
Giraudieu's case of partial word deafness.

I therefore searched the literature for similar cases. Wernicke, in reviewing Lichtheim's paper, stated that he had made a similar observation which he was going to publish in the regular reports from his Klinik. Unfortunately I have not been able to find this report in the literature. However, I found a case described by Giraudieu which closely resembled Lichtheim's patient. Giraudieu's patient (Bouquinet) was able to speak perfectly well but she showed a severe word deafness without being deaf. However, the data concerning her hearing ability were incomplete. She could understand questions addressed to her but only after they had been repeated several times, and even then she failed frequently. Once a question had been understood and answered, all following replies would continue in the same train of thought, the patient not taking any notice of later questions. The two patients appear even more alike if we consider that the behaviour of Lichtheim's patient differed from that commonly observed in cases of word deafness. He did not make any effort to understand questions addressed to him; he gave no reply nor did he appear to pay any attention to what he heard. Perhaps the patient, by this apparently 'purposive' behaviour, gave the wrong impression of being completely word deaf, while possibly repeated and urgent requests might have made him, like Bouquinet, understand. Word deaf patients, as a rule, perceive language in the same way as persons with normal hearing, giving the wrong impression of being completely word deaf, while possibly repeated and urgent requests might have made him, like Bouquinet, understand.

1 A private enquiry at the Breslau Klinik revealed that the cases mentioned in this context by Wernicke have not yet been published.
2 Giraudieu: Revue de médecine, 1882; also quoted by Bernard, l. c.
Difficulties in
result, tend to give inappropriate answers.

The post-mortem examination of Giraudeau's patient revealed a lesion of the first and second temporal convolution, such as has so frequently been found to underlie ordinary sensory aphasia. Nobody, looking at the drawing attached to Giraudeau's communication, would have thought that this lesion had caused anything but the common form of sensory aphasia. But there is another aspect to be considered. The lesion in Giraudeau's case was again an unusual one, i.e., a tumour (gliosarcoma). In discussing transcortical motor aphasia, I ventured the opinion that probably a lesion of the speech apparatus did not only cause localizing signs, but that the special nature of the disease process might be revealed by a functional modification of its symptoms. Giraudeau's case, therefore, does not prove the existence of the subcortical fibre tract \(\alpha A\). The tumour found at the post-mortem examination had not proliferated from the white matter outwardly, having perhaps in an earlier state caused a subcortical lesion only. On the contrary, it was attached to the meninges and could easily be lifted from the softened white matter. I therefore feel justified in assuming that the subcortical sensory aphasia is not due to a lesion of a subcortical pathway \(\alpha A\), but to damage of the same localization as found in cases with cortical sensory aphasia. However, I am unable to throw light on the specific functional state of the area thus affected.\(^1\)

\(^1\) Notwithstanding these considerations, I still find it very difficult to explain the subcortical sensory aphasia, i.e., word deafness

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\(\text{Nachschrift bei der Korrektur: Ich bin trotz der obenstehenden Erörterungen unter dem Eindrucke verblieben, daß die Erklärung der subkortikalen sensori-}

1 [A.a.O., S.451.]

2 Schwierigkeiten und wahrscheinliche und unpassende Antwort. Giraudeau's Kranke gelangte nun zur Sektion, und es erwies sich als Ursache ihrer Sprachstörung eine Läsion der ersten und zweiten Schlafenwindung, wie sie so häufig als Ursache gemeiner sensorischer Aphasie gefunden worden ist. Niemand, der einen Blick auf die Zeichnung wirft, die Giraudeau Miteilung beigegeben ist, wird vermuten können, daß diese Läsion etwas anderes als die gewöhnliche Form der sensorischen Aphasie mit schwerer Sprachstörung verursacht habe. Es kommt aber noch etwas anderes in Betracht. Die Läsion im Falle Giraudeau ist wiederum eine ungewöhnliche, ein Tumor (Gliosarkom). Wir erinnern uns dabei einer Vermutung, die wir bei Besprechung der transkortikalen motorischen Aphasie geäußert haben [S.68], daß der Sprachapparat wahrscheinlich nicht bloß Lokalanzeichen gebe, sondern auch eine besondere Natur des Krankheitsprozesses durch eine Abänderung seiner funktionellen Symptomatik verraten dürfte. Wir sehen also, daß der Fall Giraudeau nichts für die Existenz einer subkortikalen zuführenden Bahn \(\alpha A\) beweist. Der Tumor, den die Sektion aufdeckte, war nicht etwa von der weißen Substanz her nach außen gewachsen, so daß er in einem früheren Stadium eine bloß subkortikale Läsion ergeben hätte. Er war vielmehr mit den Hirnhäuten verwachsen und aus der erweichten weißen Substanz leicht ausschälibar. Ich glaube also für die subkortikale sensorische Aphasie annehmen zu können, daß sie nicht auf der Läsion der subkortikalen Bahn \(\alpha A\), sondern auf einer Erkrankung derselben Region beruht, welche sonst für die kortikale sensorische Aphasie verantwortlich gemacht wird. Für den besonderen funktionellen Zustand, den ich in der so erkrankten Stelle voraussetzen muß, kann ich allerdings keine volle Aufklärung geben.\(^2\)
the explanation of the subcortical sensory aphasia and a theory of its origin.

Subcortical motor aphasia can be dealt with more briefly. According to Lichtheim—it is characterized by intact writing ability in the presence of symptoms of cortical motor aphasia. Wernicke, who without impairment of spontaneous speech, while Lichtheim's schema disposes of the problem by simply assuming interruption of a tract \(\alpha A\). It was therefore of great value to me to come across a paper by Adler in which a similar case was described as "a combination of subcortical and transcortical sensory aphasia" (Beitrag zur Kenntnis der selteneren Formen der sensorischen Aphasie. [A contribution to the knowledge of the rarer forms of sensory aphasia.] Neurol. Centralblatt, May 15 and June 1, 1891.)

A comparison of Adler's case with those of Lichtheim and Wernicke contributes to the understanding of the so-called subcortical sensory aphasia. Two points in particular are illuminating. (1) Lichtheim mentioned the possibility that his patient may have been slightly deaf as the data about his hearing ability were incomplete. Wernicke's patient had a defect for higher tones. Adler's patient had definitely diminished hearing, which according to the paper by Adler in which a similar case was the case in Arnaud's patients to be referred to later. (2) More decisive still is the following conformity which can hardly be incidental. Both cases (Lichtheim and Adler; Wernicke's brief note is silent on this point) developed the picture of subcortical sensory aphasia only after repeated cerebral accidents of which at least one had involved the minor hemisphere; Lichtheim's patient had a left-sided facial palsy, Adler's a left-sided hemiplegia.

Adler mentioned this coincidence without recognizing its significance for the explanation of pure word deafness. I feel justified in assuming that subcortical sensory aphasia is caused not, as postulated in Lichtheim's schema, by a simple tract interruption, but through incomplete bilateral lesions in the receptive field of hearing, perhaps combined with peripheral deafness, as was the case in Arnaud's patients. Such complicated conditions for an apparently simple speech disorder fit better into my conception of the sensory aphasias than into that of Lichtheim.

73 Erklärung der subkortikalen sensorischen Aphasie.

Für die subkortikale motorische Aphasie können wir uns kürzer fassen. Lichtheim charakterisiert sie durch die erhaltene Schreibfähigkeit bei sonstigem Verhalten wie die korticale motorische Aphasie. Wernicke, der die...
Subcortical motor aphasia and anarthria.

made a careful analysis of the disorders of written language, refused to accept this criterion. To him the one characteristic feature of the subcortical motor aphasia is the patient’s ability to state the number of syllables. The controversies over Lichtheim’s test have been mentioned earlier in this book. Some observations made by Dejerine have in the meantime confirmed the significance of Lichtheim’s syllable test for the diagnosis of subcortical motor aphasia. However, this particular speech disorder could with equal justification be classified as anarthria rather than aphasia.

Several well observed cases, the most recent one by Eisenlohr, suggest that damage underneath Broca’s area causes a speech disorder which can be described as literal paraphasia and which represents a transition to dysarthria. For the motor part of the speech apparatus alone, therefore, a special pathway to the periphery may have to be conceded. However, in attributing a special efferent tract to the motor speech area, we want to point out that the deeper the lesion is situated the more closely the disability resembles anarthria. Aphasia still remains a cortical phenomenon.

Therefore, the speech apparatus as conceived by us, has no afferent or efferent pathways of its own, except for a fibre tract the lesion of which causes dysarthria. We shall refer to the so-called subcortical reading and writing disorders later on.

We now propose to inquire what kind of hypotheses have to be made about the causation of aphasia following lesions of a speech apparatus thus organized; or, in

1 Dejerine: Contribution à l’étude de l’aphasie motrice sous-corticale et de la localisation cérébrale des centres larynges (muscles phonateurs). (A contribution to the study of the subcortical motor aphasia and to the cerebral localization of the centres of the speech muscles.) Compt. rend. de la Soc. de Biologie, 1891, No. 8.
2 Eisenlohr: L. c. 72

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The word concept.

other words, what does the study of the aphasias teach us about the function of this apparatus? In doing this we shall endeavour to separate the psychological from the anatomical aspect of the problem as much as possible.

From the psychological point of view the “word” is the functional unit of speech; it is a complex concept constituted of auditory, visual and kinaesthetic elements. We owe the knowledge of this structure to pathology which demonstrates that organic lesions affecting the speech apparatus result in a disintegration of speech corresponding to such a constitution. We have learned to regard the loss of any one of these elements as the most important pointer to the localization of the damage. Four constituents of the word concept are usually listed: the “sound image” or “sound impression”, the “visual letter image”, the “glosso-kinaesthetic and the cheiro-kinaesthetic images or impressions”. However, this constitution appears even more complicated if one considers the probable process of association involved in the various speech activities.

(1) We learn to speak by associating a “word sound image” with an “impression of word innervation”. When we have spoken we are in possession of a “kinaesthetic word image”, i.e., of the sensory impressions from the organs of speech. The motor aspect of the “word” therefore is doubly determined. Of its two elements the former, i.e., the impression of word innervation, seems to be the least important psychologically. Its existence as a psychological element may even be disputed. We also perceive, after having spoken, a “sound image” or “sound impression” of the spoken word. As long as we have not perfected our speech, the second sound image, though associated with the first, need not be identical with it. At this stage, which is the phase of speech development in childhood, we use a language built up by ourselves; in associating various

75 Die Wortvorstellung.

| deren Worten, was uns das Stud- |
| dium der Sprachstörungen für die Funktion dieses Apparates lehrt. |
| Dabei wollen wir die psychologische und die anatomische Seite des Gegenstandes möglichst voneinander trennen. |

1 [Im Original: »einer«.]
2 [Welches wir nachgeahmt haben.]
The process of association:

other word sounds with the one produced by ourselves we behave like the motor aphasics.

(2) We learn the language of others by endeavouring to equate the sound image produced by ourselves as much as possible to the one which had served as the stimulus for the act of innervation of our speech muscles, i.e., we learn to “repeat”. In “continuous speech” we produce a series of words by waiting with the innervation of the speech muscles until the word sound, or the kinaesthetic word impression of the preceding word, or both, have been perceived. The safeguards of our speech against breakdown thus appear over-determined, and it can easily stand the loss of one or the other element. However, the loss of the correcting function of the second sound image and of the kinaesthetic word image explains some peculiarities of paraphasia, both physiological and pathological.

(3) We learn to spell by associating the visual images of the letters with new sound images which inevitably recall word sounds already known. We immediately repeat the word sound characteristic of the letter. Thus, in spelling aloud, the letter, too, appears determined by two sound impressions which tend to be identical, and two motor impressions which closely correspond to each other.

(4) We learn to read by linking up with each other, according to certain rules, a succession of word innervation impressions and kinaesthetic word impressions perceived in enunciating individual letters. As a result, new kinaesthetic word images originate, but as soon as they have been enunciated we detect from their sound images that both kinaesthetic and sound images so perceived have long been familiar to us, being identical with those used in speaking. Next, we associate with those word images acquired by spelling the significance attached to the original word

76 Der Assoziationsvorgang

wie motorisch Aphatische, indem wir verschiedene fremde Wortklänge mit einem einzigem selbst produzierten assozieren.


3. Wir lernen buchtabieren, indem wir die visuellen Bilder der Buchstaben mit neuen Klangbildern verknüpfen, die uns indes an die bereits bekannten Wortklänge erinnern müssen. Das den Buchstaben bezeichnende Klangbild sprechen wir sofort nach, so daß der Buchstabe uns wiederum durch zwei Klangbilder, die sich decken, und zwei motorische Vorstellungen, die miteinander korrespondieren, bestimmt erscheint.

4. Wir lernen lesen, indem wir das Nacheinander der Wortinnervations- und Wortbewegungsvorstellungen, die wir beim Sprechen der einzelnen Buchstaben erhalten, nach gewissen Regeln verküpfen, so daß neue motorische Wortvorstellungen entstehen. Sobald letztere ausgesprochen sind, entdecken wir nach dem Klangbild dieser neuen Wortvorstellungen, daß uns bereits die neue Wortklänge vorliegen, die sich durch zwei motorische Vorstellungen, die miteinander korrespondieren, bestimmt erscheinen.

in speaking and reading.

sounds. Now we read with understanding. If we have originally spoken a dialect instead of a literary language, we have to super-associate the kinaesthetic and sound impressions perceived in spelling aloud over the original ones, and we have to acquire a new language in this way; this process is facilitated by the resemblance between dialect and literary language.

This presentation shows that the process of learning to read is very complicated indeed and entails a frequent shift of the direction of the associations. It also suggests that the defects of reading in aphasia originate in various ways. Impairment of reading letters is characteristic of a defect of the visual element. The assembling of letters to a word takes place in the process of transmission to the speech tract; it will therefore be abolished in motor aphasia. The understanding of what has been read is effected only with the aid of the sound images produced by the words uttered, or through the kinaesthetic impressions produced in speaking. Reading with understanding thus proves to be a function which disintegrates as the result not only of motor but also of auditory defects, furthermore a function which is independent of the act of reading itself. Everybody knows from self observation that there are several kinds of reading some of which proceed without understanding.

When I read proofs with the intention of paying special attention to the letters and other symbols, the meaning of what I am reading escapes me to such a degree that I require a second perusal for the purpose of correcting the style. If, on the other hand, I read a novel, which holds my interest, I overlook all misprints and it may happen that I retain nothing of the names of the persons figuring in the book except for some meaningless feature, or perhaps the recollection that they were long or short,
and that they contained an unusual letter such as $x$ or $z$. Again, when I have to recite, whereby I have to pay special attention to the sound impressions of my words and to the intervals between them, I am in danger of caring too little about the meaning, and as soon as fatigue sets in I am reading in such a way that the listener can still understand, but I myself no longer know what I have been reading. These are phenomena of divided attention which are of particular importance here, because the understanding of what is read takes place over circuitous routes. It is clear from the analogy with our own behaviour that understanding becomes impossible once reading itself has become difficult, and we must beware of regarding this as an indication of a lesion in a fibre tract. Reading aloud is not to be regarded as a different function from reading to oneself, except that it tends to distract attention from the sensory part of the reading process.

5. We learn to write by reproducing the visual images of the letters with the help of kinaesthetic impressions received from the hand (cheiro-kinaesthetic impressions) until we have obtained identical or similar pictures. As a rule, the pictures produced in writing are only similar to, and super-associated over those perceived in reading, as we learn to read print but have to use different characters in handwriting. Writing is comparatively simpler and less vulnerable than reading.

6. It can be assumed that the various speech activities continue to be performed by way of the same associations by which we learned them. Abbreviations and substitutions may be employed, but their nature is not always easy to recognize. Their significance is still further reduced by the consideration that in cases of organic lesion the speech apparatus as a whole probably suffers some damage and is forced into a return towards the primary and secure, though more cumbersome modes of associations. In the case of the experienced reader the influence

78 Der Assoziationsvorgang


5. Wir lernen schreiben, indem wir die visuellen Bilder der Buchstaben durch Innervationsbilder der Hand reproduzieren, bis gleiche oder ähnliche visuelle Bilder entstanden sind. In der Regel sind die Schriftbilder den Lesebildern nur ähnlich und superassoziert, da wir Durchschrift lesen und Handschrift schreiben lernen. Das Schreiben erweist sich als ein verhältnismäßig einfacher und nicht so leicht wie das Lesen zu störender Vorgang.


1 [Auf das Verhältnis zwischen Aufmerksamkeit und Vorlesen geht Freud auch bei der Erörterung der Bedingungen der Sprech-, Lese- und Schreibfehler in der Psychopathologie des Alltaglebens (1901 b), S. 145 ff., ein.]  
2 [Vgl. unten, S. 119.]  
3 [Vgl. oben, S. 60.]  
4 [Vgl. unten, S. 131 ft., 141.]
in reading and writing.

of the “visual word image” makes itself felt, with the result that single words, especially proper names, can be read even without recourse to spelling.

The word, then, is a complicated concept built up from various impressions, i.e., it corresponds to an intricate process of associations entered into by elements of visual, acoustic and kinaesthetic origins. However,

the word acquires its significance through its association with the “idea (concept) of the object”, at least if we restrict our considerations to nouns. The idea, or concept, of the object is itself another complex of associations composed of the most varied visual, auditory, tactile,

Psychological schema of the word concept.

The word concept appears as a closed complex of images, the object concept as an open one. The word concept is linked to the concept of the object via the sound image only. Among the object associations, the visual ones play a part similar to that played by the sound image among the word associations. The connections of the word sound image with object associations other than the visual are not presented in this schema.

Das Wort ist also eine komplexe, aus den angeführten Bildern bestehende Vorstellung, oder anders ausgedrückt, dem Wort entspricht ein verwirkelter Assoziationsvorgang, den die aufgeführten Elemente visueller, akustischer und kinästhetischer Herkunft miteinander eingehen. Fig. 8.

Das Wort erlangt aber seine Bedeutung durch die Verknüpfung mit der »Objektvorstellung«, wenigstens wenn wir unsere Betrachtung auf Substantiva beschränken. Die Objektvorstellung selbst ist wiederum ein Assoziationskomplex aus den verschiedenartigsten visuellen, akustischen, taktilen, kin-

[Fig. 8. Psychologisches Schema der Wortvorstellung. 1


Das Schema hat Freud mit geringfügigen Abänderungen in seinen Lexikonen (Aphasie (in 1899–94, Fig. 23, S.171 des Erstdrucks) aufgenommen. In der Legende fehlt dort die Überschrift, und am Anfang des letzten Satzes wurde folgender Wortlaut dazwischengefügt: »Die Verbindungen der Wortassoziationen untereinander (außer mit dem Klangbild) sind punktiert.«]

[Dieser Begriff erscheint später, allerdings mit geändertem Sprachgebrauch, wieder in der Schrift »Das Unbewusste« (1915), Vgl. dazu die oben, S. 116 f., Anm. 6, erwähnte editorische VORBEMERKUNG. Zehn Jahre wodurch hatte Freud (1905 c; Studienausgabe, S.113) die Besonderheit der Beziehungen zwischen akustischer Wortvorstellung und Dingvorstellung (= »Sachvorstellung« bzw. im obigen, alten Wortgebrauch »Objektvorstellung«) zur Erklärung einer bestimmten Gruppe von WITZEN herangezogen.]
kinaesthetic and other impressions. According to philosophical teaching, the idea of the object contains nothing else; the appearance of a "thing", the "properties" of which are conveyed to us by our senses, originates only from the fact that in enumerating the sensory impressions perceived from an object, we allow for the possibility of a large series of new impressions being added to the chain of associations (J. S. Mill). This is why the idea of the object does not appear to us as closed, and indeed hardly as closable, while the word concept appears to us as something that is closed though capable of extension.

In the light of observations in speech disorders we have formed the view that the word concept (the idea of the word) is linked with its sensory part, in particular through its sound impressions, to the object concept. In consequence, we have arrived at a division of speech disorders into two classes: (1) verbal aphasia, in which only the associations between the single elements of the word concept are disturbed; and (2) asymbolic aphasia, in which the association between word concept and object concept are disturbed.

I am using the term asymbolia in a different sense from that given to it by Finkelnburg because "asymbolic" seems more appropriate a designation for the relationship between the word and the idea of the object than for that between the object and its idea. For disturbances in the recognition of objects, which Finkelnburg called asymbolia, I should like to propose the term "agnosia". It is quite possible that agnostic disturbances which occur only in cases of bilateral and extensive cortical lesions,  

1 J. S. Mill: Logic I, Chap. III, and "An examination of Sir William Hamilton's philosophy".  

80 Wort- und Objektvorstellung.  


3 [Vgl. unten, S. 127, 128, 135.]  
2 (Finkelnburg 1870), S. 461, re. Spalte.  
3 (Ibid.)  
4 (Diese von Freud vorgeschlagene Bezeichnung für die Störungen des Objekt­erkennens hat sich in der Neurologie völlig eingebürgert. Allerdings ist der meisten gar nicht mehr bekannt, daß dieser Terminus auf Freud zurückgeht. (P. V.)}
The three kinds of aphasia.

may also entail a disturbance of speech as all stimuli to spontaneous speech arise from object associations. Such speech disorders I should call the third group of aphasias, or “agnostic aphasias”. Clinical experience has in fact acquainted us with several cases which call for such a concept.

The first case of agnostic aphasia is that of Farges which was inadequate observed and most inappropriately interpreted as “aphasie chez une tactile”; but I hope the clinical facts will speak for themselves. The patient was a case of cerebral blindness, probably due to bilateral cortical lesions. She did not reply when addressed, and when one tried hard to contact her she kept on repeating, “Je ne veux pas, je ne peux pas!” in a tone of extreme impatience. She was unable to recognize her doctor by his voice. However, as soon as he felt her pulse, i.e., as soon as he provided her with the opportunity of a tactile association, she at once recognized him, called him by his name and chatted with him without any sign of aphasia, until he let her hand go and thus again became inaccessible to her. The same happened in relationship to objects when she was given the opportunity of producing associations of touch, smell or taste by being offered the respective sensory stimuli. As long as they lasted she had the necessary words at her command and behaved in a purposeful manner; however, as soon as she was deprived of them she resumed her monotonous expressions of impatience or uttered incoherent syllables and proved unable to understand what was said to her. This patient therefore had a completely intact speech apparatus which she was unable to utilize unless it was stimulated by those object associations which had remained intact.

1 Farges: Aphasie chez une tactile (Aphasia in a tactile personality type). L’Encephale, 1885, No. 5.
Agnostic aphasia.

A second observation of this type caused C. S. Freund to postulate the category of “optic aphasia”. His patient showed difficulties in spontaneous speech and in naming objects very similar to those observed in sensory aphasia. The following is a sample of his reactions: he called a candlestick “spectacles”, and on looking again he said, “It is for putting on, a top hat” and immediately afterwards: “It is a stearin light”. If, however, he was allowed to take the object into his hands with his eyes closed he quickly found the correct name. His speech apparatus, therefore, was intact, but it failed when stimulated by way of visual object associations only, while it worked correctly when stimulated through tactile object associations. However, in Freund’s case the effect of the disturbance of the object associations had a less severe effect than in Farges’ case. Freund’s patient deteriorated and later became completely word deaf. The post-mortem examination revealed lesions involving not only the visual but also the speech area.

The disabling effect which disturbances of the visual object associations may have on the speech function can be explained by the special importance that they assume in certain cases. In an individual whose thought processes depend largely on visual images, a peculiarity which according to Charcot is determined by individual predisposition, bilateral lesions in the visual cortex are bound to cause in addition disorders of the speech function which go far beyond what can be accounted for by the localization of the lesions. “Aphasia chez une visuelle” would have been a far more appropriate description for Farges’ observation than “aphasia chez une tactile”.

While agnostic aphasia was in these cases caused by a remote functional effect in the absence of an organic lesion of the speech apparatus itself,

Anatomical schema of the area of the speech associations demonstrating how the appearance of speech centres is created. The auditory and visual receptive fields and the motor areas for the muscles serving articulation and writing are represented by circles. The association tracts connecting them with the interior of the speech area are represented by radiating fascicles. The area in which the latter are crossed by the corresponding fascicles from the other hemisphere becomes a centre for the respective associative element when the tracts are cut off from the fields represented by circles. The crossed connections of the auditory receptive field have been omitted from the schema to avoid confusion, and also because of the uncertainty of the connections between the auditory receptive field and the sensory speech centre. The separation of the connections with the visual receptive field into two fascicles is based on the consideration that the eye movements play an important part in the associations contributing to the act of reading. Functional from the topographical factors in the analysis of these speech disorders.

We have designed a schema (Fig. 9) which is meant to illustrate the relations between the various elements of speech associations without taking into account anatomical details. In this schema the verbal and asymbolic aphasias are manifestations of such a lesion. We shall endeavour as far as possible to differentiate the

Anatomisches Schema des Sprachapparates.

Alaer und asymbolischer Aphasie die Läson des Sprachapparates selbst zum Ausdrucke kommen. Wir werden uns jetzt bemühen, hier die funktionellen wie die topischen Fig. 9.

Anatomisches Schema des Sprachassoziationsfeldes.


Wir entwerfen uns ein Schema, welches von den genauereren anatomischen Lageverhältnissen absieht² und nur die Beziehungen der einzelnen Elemente der Sprachassoziationen darstellen soll (Fig. 9). Wir stellen in demselben durch

2 [In seinem Lexikonartikel „Aphasie“ (in 1893—94a, Fig. 24, S. 172 des Erstdrucks) hat Freud dann die Ausdehnung des Assoziationsfeldes der Sprache in die Abbildung einer linken Hemisphäre eingezeichnet und ihre Beziehung zu den sog. „Sprachzentren“ durch unterschiedliche Schraffierung verdeutlicht.] ¹ [Vgl. unten, S. 130, Anm. 1.]
the circles do not represent the so-called speech centres but show the receptive and motor cortical fields between which the speech associations take place. The parts of the speech area bordering on these cortical fields acquire the significance of centres by virtue of their crossed connections with the other hemisphere: those connected with the cortical fields for the hand, the speech muscles and the optic nerve can be seen in the schema. It follows that in the case of a verbal aphasia, three symptoms can be accounted for by the localization of the lesion: if the latter is situated within the “speech centres”, the following functions will be impaired: (1) transmission of stimuli to the tracts serving the speech muscles and (2) to the tract serving the muscles employed in writing, and (3) recognition of letters. The resulting disorder is a typical motor aphasia with agraphia and with alexia for letters. The further the lesion moves towards the centre of the speech region the less likely it is to cut off any single element of the speech associations, and the more the features of the aphasia will depend on functional factors to which the speech apparatus is subject, independently of the site of the lesion. In verbal aphasia, therefore, only the loss of individual elements of the speech associations can be related to and explained by the localization of the lesion. It will help the diagnosis of the site of the lesion if the latter does not extend more centrally into the speech area, but rather into the adjoining receptive or motor cortical fields, i.e., if the motor aphasia is accompanied by a hemiplegia, or the alexia by a hemianopia.

The asymbolic aphasia may sometimes exist in a pure state resulting from a circumscribed lesion lying across the path of the association tracts. This had happened in Heubner's patient who presented an almost ideal example of the separation of the speech region from its associations by a vascular lesion encircling the auditory area which is a nodal point of the speech region.

1 [Im Original: ‘daß die: 1. Übertragung‘.]
2 [Vgl. oben, S. 63 f., 101.]
Asymbolic speech disorder without complications, i.e., without disturbance of the word associations, may also result from a merely functional state of the speech apparatus as a whole; there are some indications that the link between word associations and object associations is the most easily exhaustible component of the speech function, its weakest point as it were. This was illustrated in an interesting paper by Pick who had noted transient word deafness following epileptic fits. The patient observed by him showed an asymbolic speech disorder in the course of her recovery from the fit. Even before the understanding returned she was able to repeat words spoken to her.

The phenomenon of echolalia, i.e., the repetition of questions asked, appears to belong to the asymbolic disorders. In some of these cases, e.g., in those observed by Skwertzoff (case X) and Fränkel (quoted by Ballet), echolalia proved to be a means of overcoming the difficulties in relating the words perceived to the object associations by reinforcement of the word sounds. These patients failed to understand questions at first, but were able to understand and answer them after they had repeated them. This phenomenon also calls to mind Bastian's thesis that a speech centre, the function of which is impaired, loses first the ability to respond to "volitional" stimuli, while still able to react efficiently to sensory stimulation and in association with other speech centres. Every "volitional" excitation of the speech centres, however, involves the area of the auditory images and results in its stimulation by object associations.

3 [Vgl. oben, S. 122, und unten, S. 135.]
1 [Vgl. unten, S. 137.]
2 Skwertzoff, De la cécité et de la surdité des mots dans l'aphasie. Paris 1881.
4 [Vgl. oben, S. 68f.]
5 [Vgl. unten, S. 135.]
Mixed asymbolic-verbal aphasia.

This goes to show that, while the so-called trans-cortical sensory aphasia can be caused by a lesion, there are functional factors which tend to produce a similar clinical picture.

The mixed asymbolic-verbal aphasia due to impairment of the auditory element of speech is more common than the pure asymbolia. As all other word associations are linked to the sound image, any substantial lesion of the speech area adjoining the auditory field is bound to result in disorder within the word associations themselves as well as in disturbances of their connections with the object associations. The resulting clinical picture is that of Wernicke's sensory aphasia which also comprises disturbances in the understanding of written language, in spontaneous speech and in repetition. The area in question is probably so large that smaller lesions may lead to clinical pictures in which either the verbal or the asymbolic disorder is more pronounced. Detailed anatomical knowledge of the points at which the various fibre tracts enter the auditory speech area would be necessary for more exact localization. Such knowledge is not available at present.

We may assume that the most important source for the association of symbols is the visual area of the cortex, because the visual images usually play the most important part among the object associations. If they are excluded, the speech region can still receive impulses from the remaining cortex, i.e., tactile, gustatory and other associations, and it can still be sufficiently stimulated for speech to be produced. This explains the marked poverty of words of special significance, especially of nouns and adjectives, in patients with asymbolic-verbal aphasia, although spontaneous speech is not abolished even in the most pronounced cases. The words used are spoken mainly on visual stimulation. When stimulated by the other object associations entering the auditory

86 Die gemischte asymbolisch-verbale Aphasie.


Wir dürfen nur annehmen, daß die wichtigste Assoziationsrichtung für die Symbolasoziation die zum optischen Rindenfeld ist, da unter den Objektassoziationen die optischen Erinnerungsbilder gewöhnlich die Hauptrolle spielen. Sind diese Assoziationen unmöglich, so kann das Sprachfeld allerdings noch Impulse von der übrigen Rinde, nämlich von den taktilen, gustatorischen und anderen Assoziationen her erhalten, es kann überhaupt noch zum Sprechen angeregt werden. Wir erklären uns so, daß das spontane Sprechen bei noch so ausgeprägter asymbolisch-verbaler Aphasie nicht aufgehoben ist, aber die Charaktere der Verarmung an Redeteilen von enger Bedeutung zeigt.1 Diese (Hauptwörter, Eigenschaftswörter) wurden meist auf optische Anregung hin gesprochen. Auf die Anregung von den anderen Objektassoziationen her, welche wahrscheinlich an

1 [Vgl. oben, S. 62, 72, 73, 74, 80, 82, und unten, S. 133f., 135.]
The so-called optic aphasia.

area, the speech region produces a mutilated language, or it transmits all possible stimuli which do not require special object associations, such as particles or senseless syllables (jargon) to the motor pathway serving speech.

We remember that between the large area of the visual cortex and the cortical field of the acoustic nerve there pass not only the association tracts connecting word and object concepts, but also the tract which serves the understanding of visual letter images. A lesion of a certain localization can therefore, as the result of anatomical contiguity, cause alexia besides asymbolic aphasia. Clinical experience shows that such a combination of alexia with asymbolia of varying degree can actually be observed in cases of lesions of the angular and supramarginal gyrus. These symptoms need not, however, coexist, as has been pointed out before. As a rule, lesions of this region cause a purely verbal speech disorder which takes the form of alexia; for asymbolia to occur in addition, bilateral lesions of the visual cortex must be present. On the other hand, even a unilateral lesion suffices to cause asymbolia if situated closely to the auditory speech area, because of the connection of this "speech centre" with the visual association fibres from both hemispheres. The combination of asymbolia with word deafness, therefore, comes about more easily than that of asymbolia with alexia; the former requires only a unilateral lesion close to the auditory receptive cortical field, while for the latter to occur bilateral lesions are necessary which, however, need not be situated close to the receptive cortical field.1

1 [Vgl. oben, S. 61 f., 73, 74.]
2 Es ist wahrscheinlich nicht ohne Bedeutung, daß die reine (nach Wernicke subkortikale) Alexie so häufig bei Läsion des parietalen Randes der ersten Urwindung (Gyrus angularis und supramarginalis) gefunden wird. Wir erinnern uns, daß die Läsion des unteren Scheitellappchens eine dauernde Seitenwendung beider Augen hervorrufte, jene Art der Augenbewegung, die beim Lesen mit den visuellen Buchstabenbildern assoziiert wird. [Vgl. den letzten Satz der Legende zu Fig. 9, oben, S. 126; auch S. 107 mit Anm. 1.]
The reaction of the speech apparatus

C. S. Freund, in designating the combined speech disorder under discussion as optic aphasia has, it seems to me, failed in separating the agnostic aphasic from the asymbolic disorder under discussion. 1

This is as far as we can go in tracing the influence of the topographical factor of a lesion on the symptoms of speech disorder. Our main finding has been that this influence shows itself under the two following conditions: (1) when the lesion is situated in one of the speech centres in our sense, i.e., in the most peripheral areas of the speech region, and (2) when it puts this centre out of action completely. The lesion then results in a loss of one of the elements entering into the speech associations. In all other cases functional factors will play a part in addition to the topographical factor, and we have to decide which of the two conditions mentioned has been lacking. If, however, the lesion is situated in one of the centres, without destroying it, this particular element of the speech associations will react as a whole with a change of its mode of function, and Bastian's modifications will come to the fore. If the lesion is situated in the interior of the speech area it will, however destructive, only cause such reductions of functions as I shall

1 It is probably not without significance that pure alexia, named "subcortical" by Wernicke, is so frequently found in cases with lesions of the angular and supramarginal gyrus. We remember that a lesion of the inferior part of the parietal lobe causes permanent deviation of both eyes, i.e., the kind of eye movement which in the act of reading becomes associated with the visual letter images.

2 Siemerling, in his paper "Ein Fall von sogenannter Seelenblindheit nebst anderweitigen cerebralen Symptomen" (A case of so-called mind blindness, combined with other cerebral symptoms), Arch. f. Psychiatrie, XXI, 1890, thought that "it is possible to produce experimentally a condition resembling mind blindness merely by reducing visual acuity and by monochromasy". However, such an experimentally produced state is not entirely identical with the picture of visual agnosia. Besides, the patient tends to produce illusions because of his indistinct perceptions while the healthy subject simply feels undecided. Aphasic patients with alexia or word deafness also produce illusions. A patient described by Ross (I. e.) could read his newspaper for hours without understanding what he was reading; he was amazed at the nonsense put into the papers nowadays. Word deaf patients usually reply to a question because they believe they have understood it.
presently enumerate. They follow from the general organization of an apparatus of association. In this case the possible extent of the lesion is limited by the stipulation that it must not touch a centre anywhere.

In assessing the functions of the speech apparatus under pathological conditions we are adopting as a guiding principle Hughlings Jackson’s doctrine that all these modes of reaction represent instances of functional retrogression (dis-involution) of a highly organized apparatus, and therefore correspond to earlier states of its functional development. This means that under all circumstances an arrangement of associations which, having been acquired later, belongs to a higher level of functioning, will be lost, while an earlier and simpler one will be preserved. From this point of view, a great number of aphasic phenomena can be explained.

(1) The loss, through damage to the speech apparatus, of new languages acquired as super-associations, while the mother tongue is preserved. Next, the nature of the speech remnants in motor aphasia which are so frequently only "yes" and "no" and other words in use since the beginning of speech development.

(2) Furthermore, it can be stated that the most frequently practised associations are most likely to resist destruction. This is why patients suffering from agraphia are still capable of writing their names, if anything at all, just as illiterates are capable of writing nothing else. (In motor aphasia, however, the patient’s own name is not spared, nor is this to be expected as we only rarely pronounce our names.) The above statement implies that the patient’s occupation may conspicuously manifest itself in his symptoms; e.g., Hammond reported the case of a ship’s captain with asymbolic aphasia who could name things only by nouns referring to naval objects. Also, whole functions of speech will, according to this principle, prove more or less resistant in the case of lesions situated centrally in the speech region.

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Functional factors inherent in

of a lesion. I am inclined to agree with Maréc1 who attributed the fact that in an aphasic barrister the ability to write to dictation was hardly impaired, to the patient’s special skill in taking down information. It is to be expected that the manifestations of aphasia will be different in the highly educated from those in the illiterate. This has to be studied in each individual case.

(3) On the other hand, a rare product of speech may prove highly resistant if it had acquired great force by being associated with great intensity. I referred to such instances earlier when discussing speech remnants (“recurrent utterances”) which, according to Hughlings Jackson, are last words.

(4) It is also noteworthy that series of words are better preserved than single ones, and that words remain the more easily available the more widespread their associations are. The former rule applies to series such as successive numbers, days of the week, months, etc. Grashey’s patient was unable to state a certain number directly, but he got round this difficulty by counting from the beginning until he arrived at the requested number. Sometimes the whole series of associations can be recited, but not one particular part in isolation. Kussmaul a.o. have reported numerous instances of this type. It even happens that people who are incapable of uttering a single word spontaneously, are able to sing a song perfectly correctly.

(5) In the speech disorders resulting from asymbolia, it is obvious that the words most likely to be lost are those with the most specific meaning, i.e., those which can be elicited by only few and definite associations. Names of persons are forgotten most easily, even in the physiological amnesia; in asymbolia nouns are affected first, adjectives and verbs later.1

1 Quoted by Bastian. On the various forms etc., 1869.

1 See Broadbent: A case of peculiar affection of speech with commentary. Brain, 1, 1878-1879, p. 494.
inherent in an apparatus of association.

(6) The effects of fatigue after prolonged associative activities, of the reduction in the duration of the sensory impressions, and of fluctuating and erratic attention are important for the individual features of a speech disorder, but too obvious to require special proof.

Most of the factors enumerated here are inherent in the general properties of an apparatus equipped for association; similar factors play a part in the functions of other regions of the brain under pathological conditions. Perhaps the most striking counterpart of speech regression is the total loss of all cortical associations acquired up to a certain early period of life, occasionally observed after head injury.

The three levels of reduced functional efficiency of the speech centres postulated by Bastian, have been mentioned several times already. We can accept them, although we reject the notion of physiological speech centres. We prefer the formulation that the visual, auditory and kinaesthetic parts of the speech apparatus are still able to function under certain specifiable conditions. We also have to realize that Bastian’s modifications apply mainly to incompletely destructive lesions, especially of the “speech centres”: if the lesion does not affect all elements of one particular speech function, such as is the case when the damage is situated in the nodal points, the activities of the intact portion of the nervous tissue will compensate for that of the damaged part and cover up the defect. A statement such as this implies, of course, that no single nerve fibre and cell can serve one single function of speech only, but that the conditions are in fact more complicated than this.

Wir haben bereits mehrmals die drei Stufen verminderter Funktionsfähigkeit besprochen, welche Ch. Bastian für die Zentren der Sprache aufgestellt hat.2 Wir können dieselben annehmen, auch wenn wir von Zentren der Sprache im physiologischen Sinne absehen, indem wir sagen, der optische, akustische, kinästhetische Bestandteil des Sprachapparates sei noch unter diesen oder jenen Bedingungen leistungsfähig. Dabei wollen wir noch im Auge behalten, daß die Bastianschen Modifikationen hauptsächlich für Läsionen, die des beschädigten Natur3 gerade unserer Zentren Geltung haben werden, denn wenn die Läsion nicht alle Sprachelemente einer Herkunft betrifft, wie dies bei ihrem Sitz an den Sammelpunkten der Fall ist, wird die Funktion des intakt gebliebenen Nervengewebes die des beschädigten ersetzen und deren Schädigung verdecken. Hinter einer solchen Behauptung steckt natürlich die Anschauung, daß eine einzelne Nervenfaser und Nervenzelle nicht für eine einzige Sprachassoziationsleistung in Anspruch genommen wird, sondern daß hier ein komplizierteres Verhältnis obwaltet.

2 [Vgl. oben, S. 68ff.]
3 [Vgl. oben, S. 71, 85.]
1 [Vgl. oben, S. 94.]
Functional modifications of the acoustic element.

Bastian's modifications represent, in a sense, also levels of disinvolution, i.e., of functional retrogression. I regard it, however, as profitable to discuss them for each element of the speech associations.

(1) The acoustic element is the only one which responds to three different types of stimulation. The one called "volitional" by Bastian, consists of stimulation from the object associations, or more exactly from the activities of the rest of the cortex. This type of association is liable to break down as the result of comparatively slight lesions in the auditory speech centre causing a partial asymbolic aphasia. The latter manifests itself by impairment of spontaneous speech and of the ability to name objects. In the mildest form there is only some difficulty in finding nouns of strictly limited significance and with a small range of associations.

The associative activity of the acoustic element is the central part of the whole speech function. Grashey's and Graves' cases illustrate a disorder of voluntary speech with intact ability of association with the visual element. I have been unable to find examples of failure of the acoustic element to associate spontaneously, while still functioning on direct stimulation. Such a state would probably imply a complete loss of function, as the activity of the acoustic centre consists in association and not in transmission to an efferent tract. On the other hand, it may happen that the acoustic element may still be able to produce verbal associations on peripheral stimulation, but no longer associations of symbols. Such a disorder would again manifest itself as asymbolia, i.e., as Lichtheim's transcortical sensory aphasia. From this we are inclined to infer that the latter type of speech disorder can be caused by a lesion in the auditory centre as well as by one situated between the latter and the central part of the whole speech function. Grashey's and Graves' cases illustrate a disorder of voluntary speech with intact ability of association with the visual element. I have been unable to find examples of failure of the acoustic element to associate spontaneously, while still functioning on direct stimulation. Such a state would probably imply a complete loss of function, as the activity of the acoustic centre consists in association and not in transmission to an efferent tract. On the other hand, it may happen that the acoustic element may still be able to produce verbal associations on peripheral stimulation, but no longer associations of symbols. Such a disorder would again manifest itself as asymbolia, i.e., as Lichtheim's transcortical sensory aphasia. From this we are inclined to infer that the latter type of speech disorder can be caused by a lesion in the auditory centre as well as by one situated between the latter and the central part of the whole speech function.


2 [Vgl. oben, S. 131 ff. mit 132, Anm. 1.]
3 [Vgl. oben, S. 128.]
Partial word deafness.

visual receptive cortical field. In the former case it would be caused through functional, in the latter through topographical factors.

It seems that loss of excitability of the acoustic element, which manifests itself as word deafness, is invariably to be interpreted as a local symptom. The only exception is the rather obscure group of cases mentioned by Arnaud, which might suitably be called "partial word deafness". It is essential for their understanding that they invariably show a considerable degree of ordinary bilateral deafness. These patients speak quite correctly, but they can understand only with difficulty and have to be spoken to slowly and distinctly. Under this condition their comprehension is immediate and complete, which argues against the assumption of a lesion in the so-called auditory speech centre. The only difference between the behaviour of these patients and that of ordinary deaf people lies in the fact that the latter can understand, i.e., associate, while listening, whereas the former begin to understand only when the strength of the peripheral stimulus is above certain thresholds.

Understanding of spoken words is probably not to be regarded as simple transmission from the acoustic elements to the object association; it rather seems that in listening to speech with understanding, the function of verbal association is stimulated from the acoustic elements at the same time, so that we more or less repeat to ourselves the words heard, thus supporting our understanding with the help of kinaesthetic impressions. A higher measure of attention in listening will entail a higher degree of transmission of speech heard on to the tract serving the motor execution of language. One may suppose echolalia to occur if there is an obstacle in the

1 Arnaud: Contribution à l'étude de la surdité verbale (A contribution to the study of the pure word deafness). Arch. de Neurol., Mars 1877.
Modification of the visual element.

connection with the object associations; under these circumstances the whole of the excitation may be discharged by way of an even stronger, i.e., an audible repetition of words heard.

(2) The visual element is not directly linked to the object associations, as our letters represent sounds and do not symbolize concepts like those of certain other peoples; we do not therefore need to consider spontaneous stimulation here. This element is activated mainly by peripheral stimulation and, in the case of spontaneous writing, by mere association with other elements of the word concept. Inability to recognize letters is the only manifestation of impairment of the visual element, as “reading” is a much more complicated function which can be impaired by a great variety of lesions. Here the exceptional case of an element no longer responding to peripheral but still to associative stimulation can apparently occur: there are patients who are unable to recognize letters but can write well. Wernicke calls this disability subcortical alexia and explains it with the localization of the lesion. He differentiates three disorders of reading whereby the word concept (C) is intact (Fig. 10): (1) Cortical alexia characterized by loss of reading and writing; (2) Subcortical alexia; loss of the ability to read; writing is unimpaired except for inability to copy; (3) Transcortical alexia; there is loss of reading and writing, but the ability to copy printed or written words mechanically is preserved.

There is a simple objection to this schema for the disorders of letter reading. If in subcortical alexia the lesion is situated in the peripheral fibre tract leading to \( \sigma \), no impression of the letters presented can reach the cortex; they are not seen and therefore cannot be copied, unless each letter is normally seen via two pathways,

1 [(1886), S. 644.]
2 [(1886), S. 464.]
The so-called subcortical alexia.

the one perceiving it as an ordinary visual object and the other as speech symbol. The same objection could not be raised for the so-called subcortical word deafness, because the word that is not heard is not repeated either. But the letter that is not recognized can be copied, and therefore the failure of recognition cannot be due to a lesion before \( \alpha \); we are not dealing with a disorder of perception but with one of association. Wernicke, it is true, distinguished between "copying" and "drawing from a model", thus trying to save his theory. However, I believe that interruption before \( \alpha \) must impede both motor functions, unless we assume that every letter image reaches the brain by two different peripheral tracts, as an ordinary object and as an object for speech.  

Copying differs from drawing from a model only by the degree of facilitation derived from the comprehension of the model;

It could be argued that this does in fact happen, because this type of alexia is usually accompanied by right-sided hemianopia, the letter being perceived as object for speech by the left and as ordinary object by the right hemisphere. However, if this were so, every right-sided hemianopia would have to be accompanied by alexia, which is not the case.

93 Die sogenannte subkortikale Alexie.

95 Bahnengesehen wird, von denen ihn die eine als gewöhnliches visuelles Objekt, die andere als Sprachsymbol auffäßt. Bei der sogenannten subkortikalen Worttaubheit konnte dieser Einwand nicht gemacht werden, denn das nicht gehört Wort wird auch nicht nachgesprochen. Da der nicht erkannte Buchstabe aber nachgehakt werden kann, ist die Annahme, daß er infolge einer Läsion vor \( \alpha \) nicht erkannt wird, ausgeschlossen; es handelt sich um keine Störung in der Wahrnehmung, sondern um eine Störung in der Assoziation. Wernicke unterscheidet allerdings zur Rettung seines Erklärungsversuches »Copieren« von »Nachzeichnen«. Aber ich halte dafür, daß für beide motorischen Leistungen die Unterbrechung vor \( \alpha \) ein Hindernis abgibt, wenn wir nicht in der Tat annehmen, daß ein Buchstabenbild auf zweierlei peripherischen Bahnen ins Gehirn gelangt, als gemeines Objekt und als Objekt für die Sprache.

Fig. 10.

Wernickes Schema der Lesestörungen
(Die neueren Arbeiten über Aphasie, Fortschritte der Medizin, 1886, p. 464.) \( \alpha \) das optische Schriftbild, \( \beta \) das motorische Zentrum der Schreibbewegungen, \( c = a + b \) der Wortbegriff.

Das Kopieren unterscheidet sich vom Nachzeichnen entweder bloß graduell durch die größere Leichtigkeit, die das Verständnis der Vorlage mit sich bringt,

1 [A.a.O., S. 466.]
2 Man könnte den Einwand erheben, daß dieser Fall tatsächlich vorliegt, da diese Alexie meist neben rechtseitiger Hemianopsie gefunden wird. Der Buchstabe würde mit der linken Hemisphäre als Objekt für die Sprache, mit der rechten als gemeines Seheobjekt aufgefaßt werden. Allein, dann müßte jede rechtseitige Hemianopsie mit Alexie kompliziert sein, was nicht der Fall ist.
Explaination of the subcortical alexia.

otherwise they are identical and use the
same pathway. Everybody requires a high degree of
attention for copying incomprehensible symbols; this is
as a rule difficult to obtain in aphasics. There is, how­
ever, an alternative explanation: copying consists in a
transposition of printed letters into letters of script. This
transposition is possible because we learn to read, but
not to write, normal print or italics, and it makes no
difference whether or not the words read are understood.
A child patient observed by Bernard (Obs. V) showed a
remarkable facility in faultlessly carrying out this trans­
position in copying though he was quite unable to read
what he had copied.

In my opinion the so-called subcortical alexia calls
for a different explanation. In writing as well as in
speaking we receive kinaesthetic impressions from the
movements carried out by the muscles involved. How­
ever, the impressions coming from the hand are more
distinct and intensive than those coming from the
speech muscles, either because we are used to attributing
great value to the perceptions of the hand also in relation
to functions other than writing, or because they are
associated with visual impressions: we can see ourselves
writing but not speaking. Therefore we are able to write
directly from the sound images with the aid of kine­
aesthetic impressions without depending on the visual
element.

The lesion underlying subcortical alexia can be
assumed to be situated on the periphery of the speech
region as it is so frequently accompanied by hemianopia.
In this type of aphasia, therefore, the whole motor
apparatus may be intact and writing is possible through
direct associations with the word sounds. In some cases
of subcortical alexia reading is aided by writing as
mentioned earlier; the letter images incapable of direct
association with the acoustic element, are nevertheless
associated with it by means of the kinaesthetic impressions

96 Erklärung der subkortikalen Alexie.

I es ist sonst dieselbe Handlung und voll­
zieht sich auf derselben Bahn. Jeder von uns wird zum Nachzeich­
nen ihm unverständlicher Zeichen einen hohen Grad von Aufmerk­
samkeit brauchen, der bei den Aphasischen im allgemeinen schwer
zustande kommen wird. Oder aber das Kopieren besteht in einer
Umsetzung der Druckbilder der Buchstaben in Schriftbilder. Die­
selbe erklärt sich daraus, daß wir Druck- und Kursivschrift lesen,
aber nicht Druckschrift schreiben lernen?, und zeigt sich unabhän­
gig von dem Verständnis des Gelesenen. Ein kleiner Patient Bern­
ards ([1885] Obs. V) war durch die Leichtigkeit und Sicherheit auf­
fällig, mit der er beim Kopieren diese Umsetzung vollzog, ohne im
mindesten lesen zu können, was er kopierte.

Ich glaube die Erklärung der sogenannten subkortikalen Alexie ande­
erswo suchen zu müssen. Wir erhalten beim Schreiben wie beim
Sprechen kinaesthetische Empfindungen von den Bewegungen, wel­
che die betreffenden Muskeln ausführen. Die kinaesthetischen Emp­
findungen der Hand sind aber deutlicher und intensiver als die der
Sprachmuskulatur, sei es, weil wir diesen Empfindungen der Hand
auch für andere Funktionen einen großen Wert beizulegen pflegen,
sei es, weil sie noch mit visuellen Eindrücken verknüpft sind. Wir
sehen uns nämlich schreiben, sehen uns aber nicht sprechen. Wir
sind darum imstande, direkt von den Klangbildern aus mit Hilfe der
kinaesthetischen Empfindungen zu schreiben und das visuelle Ele­
ment dabei zu umgehen.

Bei der subkortikalen Alexie dürfen wir annehmen, daß es sich um
eine extreme Läsion im Sprachfelde handle, da sie so häufig mit He­
mianopsie zusammen vorkommt. Der gesamte motorische Teil des
Apparates kann also bei ihr intakt und das Schreiben auf direktem
Wege von den Klangbildern her möglich sein. In einigen dieser Fälle
subkortikaler Alexie wird, wie bereits früher erwähnt [S. 82 f.],
schreibend gelesen; die der direkten Assoziation mit dem akusti­
sehen Element unfähigen Buchstabenbilder werden durch die beim
Nachzeichnen geweckten kinaesthetischen

3 [Vgl. oben, S. 120.]
Disorders of reading.

aroused in the process of "drawing from a model", and in this way they are recognized.

Almost all authors who reported instances of mixed aphasia with writing and reading disorders state that in these cases the degree of the writing disorder was proportionate to the motor impairment of speech rather than to the disorder of reading. This would be impossible if writing had not become independent of the letter images after some practice. Self observation, I believe, also shows that in writing spontaneously one does not rely on the visual element, except when writing foreign words, proper names and words which one has learned by way of reading only.

Impairment in the recognition of letters naturally implies inability to read. However, it is possible for a reading disorder to be present without loss of the ability to recognize letters. This may result from a variety of lesions and conditions such as can be readily understood from earlier remarks about the intricate processes of associations which enter into the act of reading. A reading disability may be no more than the result of increased exhaustibility of the visual function, whereby motor aphasia or disturbance of auditory associations need not be present. This applied to a case of Bertholles mentioned by Bernard, and to the so-called dyslexia described by Berlin. In such a case the failure to read will be preceded by an attempt at spelling which may at first be successful. This may mean that the impaired visual element be still capable of the simpler function of associating visual images with the acoustic and kinesthetic elements while no longer able to cope with the numerous repetitions and the proper arrangement of this

1 I believe that some physiological and individual peculiarities of memory can be explained by the changing role of its individual elements. One may have a very good memory yet be unable to retain proper names and numbers. Individuals who excel in remembering names and numbers belong to the visual type, i.e., they have a predilection for recalling the visual images of objects even if they think in sound images.

* Berlin: Eine besondere Art der Wortblindheit: Dyslexie (A special type of word blindness: dyslexia), 1887.
activity which, if performed with a certain speed, makes up the process of reading. This is an instance of the loss of a complicated function while the simpler one is preserved.

Alexia may also be the result of damage to the motor or auditory element of speech; this naturally deprives it of diagnostic significance. I think it can be maintained that generally motor aphasia abolishes the understanding of written language as well as the so-called mechanical reading because the understanding of read material is accomplished only after the transmission of the stimulus from the visual to the motor elements through the association of the latter with auditory impressions. In cases of auditory impairment, however, as well as of asymbolia, the purely mechanical type of reading may be preserved. The explanation of reading disorders, which I do not intend to discuss in detail here, presents certain difficulties which cannot be disposed of by reference to the localization of the lesion, nor by assuming the familiar functional changes. In complicated cases one or the other part of the reading function remains intact, depending on the specific elements of association having been spared to a greater or lesser degree in individual patients.

(3) The motor element, i.e., innervation impressions and kinaesthetic images, presents fewer difficulties. We assume that volitional and associative stimulation of this element usually coincide as spontaneous speech is activated via the sound images. The so-called stimulation from the periphery is also an association, being activated either through the auditory element, as in repetition of spoken words, or through the visual element, as in reading aloud. It seems that occasionally the latter succeeds, but not the former, and vice versa. In the so-called transcortical motor aphasia we have an activity which, if performed with a certain speed, makes up the process of reading. This is an instance of the loss of a complicated function while the simpler one is preserved.

The motor element of the speech associations.

Die Lesestörung kann ebensowohl Ergebnis einer Schädigung des motorischen und anderemal des akustischen Sprachelementes sein, wobei natürlich eine diagnostische Bedeutung derselben wegfällt. Ich glaube, man kann im allgemeinen behaupten, daß motorische Aphasie sowohl das Leseverständnis wie das sogenannte mecha­
nische Lesen aufhebt, da das Leseverständnis erst nach der Übertragung der Erregung von den visuellen Elementen auf die motorischen durch die Assoziation letzterer mit den akustischen Elementen zu­
stande kommt. Bei akustischer Läsion dagegen sowie bei Asymbolie kann das rein mechanische Lesen erhalten bleiben. Im übrigen be­
reitet die Erklärung der Lesestörungen, auf die ich in einzelnen ein­
zugehen nicht beabsichtige, manche Schwierigkeiten, die weder durch bloß topische Momente noch durch die Annahme bekannter Funktionsveränderungen zu erledigen sind. Es bleiben in komplexen Fällen bald diese, bald jene Stücke der Funktion erhalten, wahrschei­
nlich je nachdem von den Elementen, die zur Assoziation nach einer bestimmten Richtung dienen, hier oder dort eine größere An­zahl leistungsfähig geblieben ist.

3. Das motorische Element (Innervations- und Bewegungsbild) bie­
tet unserer Betrachtung geringere Schwierigkeiten. Wir nehmen an, daß für dasselbe willkürliche und assoziative Anregung meist zu­
sammenfällt, da beim spontanen Sprechen über die Klangbilder ge­
sprochen wird. Auch die sogenannte periphere Anregung ist eine Assoziation, da sie entweder (beim Nachsprechen) von dem akustischen oder (beim Lautlesen) vom visuellen Elemente her er­
folgt. Es scheint der Fall vorzukommen, daß letztere Anregung Er­
folg hat, während die erstere versagt, und umgekehrt. In der soge­
nannten transkortikal en motorischen Aphasie haben wir den Fall kenne gelernt, daß das

4 [Vgl. oben, S. 120, 132.]
Problems of the motor aphasia.

instance of the motor element still being capable of stimulation by peripheral association, but failing to respond to volitional association.

Nevertheless, the understanding of motor aphasia, which has been known better and for a longer time than any other type, offers more difficulties than one would expect. We have already referred to the uncertainty as to whether in motor aphasia the function of symbolic association, i.e., the volitional stimulation of sound images, is really intact. If the opposite could be established it would mean that the loss of the motor element impairs the function of the auditory element in the same way as we have long known it to happen in reverse. Furthermore, there are the inexplicable cases of motor aphasia with letter blindness which one can hardly attribute to a chance coincidence. Finally, it has never been satisfactorily explained why cases of total loss of motor speech are so common, while reduction of speech to half or a third never occurs. Cases of the latter type invariably turn out to be sensory aphasias on closer analysis. It seems that a lesion capable of disturbing the motor function of speech destroys it completely in most cases, apart from the well-known scanty remnants.

There is, as it were, no paresis, but only paralysis. Also, the failure of most cases of motor aphasia to improve, deserves attention. This is in striking contrast to the rapid and complete return of speech in other cases. It is hardly necessary to point out that speechlessness in the first days after the onset of an illness has no diagnostic significance. It may occur irrespective of the site of the lesion, and is obviously caused by the shock to the apparatus which had previously been working with all its resources.

1 Bernard has reported a case of this type. I. e. p. 125.
Charcot's theory of

(4) I do not intend to enter into a similar discussion of the cheiro-motor element. Some important considerations regarding it have been advanced earlier, when the function of the visual component of speech was discussed.

I now have to consider an interesting and significant idea introduced into the theory of aphasia by Charcot. Its acceptance would impose considerable limitations on our hypotheses in this field. We have assumed that in spite of equal potentialities of the associations between the various elements of the speech function, nevertheless certain kinds of associations have preference over the rest; in the speech disorders, therefore, not all and sundry, but only a limited number of associations between the elements of speech need be taken into account. We have postulated that they are those which play a leading part in the learning of speech. In Charcot's view, no such general rule of preference of routes of associations exists; all links between the elements of speech appear at first to be endowed with equal functional rights, and it is left to individual practice or organization to make one or the other element of speech the central co-ordinating factor for the rest. According to this theory one individual speaks, writes and reads predominantly or exclusively with the help of kinaesthetic sensory impressions, while another may employ the visual element for the same purpose, etc. This would rule out an over-all dependence of the function of speech association on the contribution of the acoustic element.

It can easily be seen that identical lesions would result in different speech disorders, if such a relationship

1 Charcot: *New Lectures*, 1886. See also the papers of his pupils Ballet, Bernard and Marie.

2 Charcot, Neue Vorlesungen etc., 1886 [1887]. – Außerdem die Arbeiten seiner Schüler Ballet [1886], Bernard [1885] und Marie [1883].
individual variations in the associations of speech

exists. A "motor" speaker could suffer a lesion of the acoustic or visual element with hardly any ill-effect, while damage to his motor element would deprive him of almost all speech functions in addition to the motor one. Damage to the visual element would render a "visual" speaker not only letter blind, but nearly or completely incapable of using his speech apparatus at all. The diagnosis of aphasia would be liable to the grossest errors if inferences concerning the site and extent of the lesions were drawn from the loss of functions sustained, before the individual's preference for a single associative element had first been ascertained; such knowledge could be obtained only in the most exceptional cases.

Nobody has so far wanted to reject Charcot's approach completely. However, its significance for the theory of aphasia is open to doubt. Extreme claims, such as those advanced by Stricker\(^1\) for the paramount importance of the motor element of speech, have been refuted by Bastian with the remark that he was waiting until he was shown a case of a person who had been made word deaf through destruction of Broca's area. I believe that the study of the speech disorders has so far provided no reason for attributing any great importance to Charcot's speculations regarding the main aphasic symptoms.

However, the possibility cannot be excluded that as long as the speech apparatus is in possession of all its resources, such a habitual preference for one or the other speech association may exist, but that in case of illness, i.e., of lowering of associative activity, the pre-eminence of the first used lines of associations is re-established.

\(^1\) Stricker: *Studien über die Sprachvorstellungen* (Studies of the speech concepts) 1880.

1 Stricker, Studien über die Sprachvorstellungen 1880
It would certainly be wrong to dismiss Charcot's idea completely and to allow oneself to be misled into a schematic rigidity in the interpretation of the speech disorders. "Different amounts of nervous arrangements in different positions are destroyed with different rapidity in different persons", says Hughlings Jackson.

Summary and Results

We can now survey the route we have travelled in this treatise. Our starting point was Broca's discovery which for the first time related a certain form of speech disorder, i.e., the motor aphasia for which he proposed the term aphemia, to lesion of a certain area of the cerebral cortex. When Wernicke repeated this feat for another type of aphasia, the way was open for explaining differences of speech disorders by differences of localization. Wernicke distinguished sharply between speech centres and fibre tracts; he characterized centres as storing places of impressions and postulated a conduction aphasia (commissural aphasia) in addition to the two main forms of speech disorder mentioned above. Lichtheim, in considering the possible connections of the speech centres with the rest of the cortex, increased the number of conduction aphasias and attempted to interpret other varieties of speech disorders as subcortical and transcortical aphasias. Thus the contrast between central aphasias and conduction aphasias was assumed to be the key for the understanding of the speech disorders. On the other hand, Grashey, in his theory of the amnesias, completely discarded the basic explanation by localization and, in an ingenious analysis, attributed a certain type of disorder to alteration of a functional constant in the apparatus of speech. According to him, disturbances of speech fall into two groups: the one due to localized lesions, and the amnesias due to functional change not localized anywhere in particular.
We set out with the intention of examining whether the principle of localization could really offer as much for the explanation of the aphasias as has been claimed, and whether one is justified in differentiating between centre and pathways of speech and between the respective types of speech disorders. We first analysed Wernicke's conduction aphasia, and found that according to his schema it ought to have different features from those he had attributed to it, features such as nobody is ever likely to find in reality. We then turned to one of Lichtheim's conduction aphasias, the so-called transcortical motor aphasia, and we established with the help of several post-mortem findings that it was due to a lesion in the motor and sensory centres themselves and not in the fibre tracts, and that the pathway the lesion of which Lichtheim regarded as the cause of this aphasia, probably did not exist at all. In the course of our study we also discussed other subcortical and transcortical aphasias, and we found on every occasion that the lesions had been situated in the cortex itself. Only to the transcortical sensory aphasia, for which we proposed the name asymbolia, did we have to concede a specific localization. Our views were strongly supported by a case reported by Heubner. However, the fact that cortical lesions of the same localization could cause such different clinical pictures called for an explanation. We put forward the assumption that the so-called speech centres as wholes reacted to partial damage with a modification of function. Regarding the kind of modification, we followed Bastian who recognized three pathological conditions of a centre: (1) Absence of excitability to volitional stimulation, with preservation of excitability through association and to sensory stimuli. (2) Loss of excitability except by sensory stimuli. (3) Complete loss of excitability.
While thus resorting to functional factors in the explanation of the so-called cortical aphasias, we found ourselves unable to accept as satisfactory Grashey’s attempt to explain a case of amnesia by functional changes only. We proved that the topographical factor, too, had been of importance in this case and we explained its clinical features by referring, in addition, to one of Bastian's modifications.

Having rejected the differentiation between centre and conduction aphasia and between aphasias and amnesias, it was incumbent on us to evolve another conception of the organization of the speech apparatus, and to state how in our view topographical and functional factors manifested themselves in the disorders of this apparatus. After a critical digression to Meynert's doctrine of the organization of the brain and of the localization of concepts in the cortex, we successively rejected the following assumptions: that the impressions with which the speech function works could be localized separately from the process by which they were being associated; that association was carried out by way of subcortical fibre tracts; and that between well-defined centres there extended a functionless region waiting to be occupied by new acquisitions. Our concept of the structure of the speech apparatus was based on the observation that the so-called speech centres border externally (peripherally) on parts of the cortex which are important for the speech function, while interiorly (centrally) they enclose a region not covered by localization which probably also belongs to the speech area. The apparatus of speech therefore presented itself to us as a continuous cortical area in the left hemisphere extending between the terminations of the acoustic and optic nerves and the origins of the motor tracts for the muscles serving articulation and arm movements. The necessarily ill-defined parts of the speech region which border on these receptive and motor cortical fields, have acquired the significance of speech centres from the point of view of morbid anatomy but not in respect of normal function; their lesions...

Results

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cut off one of the elements of speech association from its connections with the others. A lesion situated centrally in the speech region can no longer have this effect. We have made the additional assumption that this speech region is connected with the cortical fields of the right hemisphere via the corpus callosum, and that the connecting crossed fibre tracts also enter into the most peripheral parts of the speech region, i.e., the speech centres. Within the speech region we recognized conduction aphasias only, i.e., aphasias due to interruption of associations, and we denied any subcortical lesion the ability to produce aphasia, because there is only one pathway to the periphery, i.e., the tract which runs through the knee of the internal capsule, and its lesion causes anarthria.

In considering the effects of lesions on this apparatus we found that they could result in three types of aphasia: (1) purely verbal, (2) asymbolic and (3) agnostic aphasia. The discovery of the last named was a necessary corollary of our theory according to which destruction of both cortical areas for one of the elements involved must have the same effect as unilateral destruction of the nodal point for this element.

From the psychological point of view we recognized the word as a complex of concepts (impressions, images) which through its sensory part (its auditory component) is connected with the complex of object associations. We defined verbal aphasia as a disturbance within the word complex, asymbolic aphasia as a separation of the latter from the object associations, and agnostic aphasia as a purely functional disorder of the speech apparatus.

Finally, the following factors have proved to be decisive for the effect of lesions on the speech apparatus so organized: the degree of destructiveness of the lesion, and its situation relative to
Conclusions

If situated on its periphery, i.e., in one of the so-called speech centres, its symptoms are related to its localization; depending on whether it causes complete or incomplete destruction it either results in a loss of only one of the elements of speech associations, or it alters the functional state of this element in a way described as Bastian's modifications. If the lesion is situated centrally in the speech region the whole apparatus of speech suffers functional disturbances such as arise from its character as an instrument of association, and which we have attempted to enumerate.

Conclusions

I am well aware that the considerations set out in this book must leave a feeling of dissatisfaction in the reader's mind. I have endeavoured to demolish a convenient and attractive theory of the aphasias, and having succeeded in this, I have been able to put into its place something less obvious and less complete. I only hope that the theory I have proposed will do more justice to the facts and will expose the real difficulties better than the one I have rejected. It is with a clear exposition of the problems that the elucidation of a scientific subject begins. I should like to formulate the essence of my views briefly in a few sentences. Previous writers on aphasia who knew only of one cortical area with a special relation to speech disorder, found themselves compelled by the incompleteness of their knowledge to seek for an explanation of the variety of speech disorders in functional peculiarities of the apparatus of speech. After Wernicke had discovered the relationship of the area called after him to sensory aphasia, the hope was bound to arise that this variety could be fully understood from...
the circumstances of localization. It appears to us, however, that the significance of the factor of localization for aphasia has been overrated, and that we should be well advised once again to concern ourselves with the functional states of the apparatus of speech.

107 Schlußwort.

Lokalisation zu verstehen. Es scheint uns nun, daß hierbei die Bedeutung des Momentes der Lokalisation für die Aphasie überschätzt worden ist und daß wir recht daran tun werden, uns wiederum um die Funktionsbedingungen des Sprachapparates zu bekümmern.


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3 Wernickes sensorische Aphasie.
4 Die Bewahrung der Sprachvorstellungen in Zellen.
5 Leitungs- und Zentrumpaphasie.
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7 Lehre von der Aphasie durch Lichtheim.
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99 Charcots Gesichtspunkt der
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BIBLIOGRAPHISCHE NOTIZ

Zur Auffassung der Aphasien

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APPENDIX B
PSYCHO-PHYSICAL PARALLELISM

[It has been pointed out above (p. 163) that Freud's earlier views on the relation between the mind and the nervous system were greatly influenced by Hughlings-Jackson. This is particularly shown by the following passage extracted from his monograph on aphasia (1891b, 56-8). It is especially instructive to compare the last sentences on the subject of latent memories with Freud's later position. In order to preserve a uniform terminology, a new translation has been made.]

After this digression we return to the consideration of aphasia. We may recall that on the basis of Meynert's teachings the theory has grown up that the speech apparatus consists of distinct cortical centres in whose cells the word-presentations are contained, these centres being separated by a functionless cortical region, and linked together by white fibres (associative fasciculi). The question may at once be raised whether a hypothesis of this kind, which encloses presentations in nerve cells, can possibly be correct and permissible, I think not.

The tendency of earlier periods in medicine was to localize whole mental faculties, as they are defined by psychological nomenclature, in certain regions of the brain. By contrast, therefore, it was bound to seem a great advance when Wernicke declared that only the simplest psychological elements, the different sensory presentations, could legitimately be localized—localized at the central termination of the peripheral nerve which has received the impression. But shall we not be making the same mistake in principle, whether we are trying to localize is a complicated concept, a whole mental activity, or a psychical element? Is it justifiable to take a nerve fibre, which for the whole length of its course has been a purely physiological structure and has been subject to purely physiological modifications, and to plunge its end into the sphere of the mind and to fit this end out with a presentation or a mnemonic image? If 'will', 'intelligence', and so on, are recognized as being psychological

ANHANG B
DER PSYCHO-PHYSISCHE PARALLELISMUS

[Es wurde schon weiter oben (S. 122) darauf hingewiesen, daß Freud frühe Ansichten über die Beziehung zwischen Psyche und Nervensystem stark von Hughlings Jackson beeinflußt waren. Dies zeigt sich ganz besonders in der folgenden, seiner Monographie über die Aphasien entnommenen Passage (1891b, 56-8). Aufschlußreich ist zumal ein Vergleich der letzten Sätze zum Thema der latenten Erinnerungen mit Freuds späterer Position.]


Gegenüber der Neigung früherer medizinischer Epochen, ganze Seelenvermögen, wie sie der Sprachgebrauch der Psychologie abgrenzt, an bestimmte Bezirke des Gehirns zu lokalisieren, mußte es als großer Fortschritt erscheinen, wenn Wernicke erklärte, daß man nur die einfachsten psychischen Elemente, die einzelnen Sinnsvorstellungen lokalisieren dürfe, und zwar an die zentrale Endigung des peripherischen Nerven, der den Eindruck empfängt hat. Im Grunde aber begeht man nicht denselben prinzipiellen Fehler, ob man nun einen komplizierten Begriff, eine ganze Seelentätigkeit oder ob man ein psychisches Element zu lokalisieren versucht? Ist es gerechtfertigt, eine Nervenfasern, die über die ganze Strecke ihres Verlaufes bloß ein physiologisches Gebilde und physiologischen Modifikationen unterworfen war, mit ihrem Ende ins Psycheische einzutauchen und dieses Ende mit einer Vorstellung oder einem Erinnerungsbild auszustatten? Wenn der 'Wille', die 'Intelligenz' u. dgl. als psychologische Kunstworte erkannt sind, denen in der
technical terms to which very complicated states of affairs correspond in the physiological world, can we feel any more sure that a "simple sensory presentation" is anything other than a technical term of the same kind?

It is probable that the chain of physiological events in the nervous system does not stand in a causal connection with the psychical events. The physiological events do not cease as soon as the psychical ones begin; on the contrary, the physiological chain continues. What happens is simply that, after a certain point of time, each (or some) of its links has a psychical phenomenon corresponding to it. Accordingly, the psychical is a process parallel to the physiological—"a dependent concomitant." ¹

I know quite well that I cannot accuse the people whose views I am here disputing of having executed this jump and change in their scientific angle of approach [i.e. from the physiological to the psychological] without consideration. They obviously mean nothing else than that the physiological modification of the nerve fibres which accompanies sensory excitation produces another modification in the central nerve cell, and that this latter modification becomes the physiological correlate of the "presentation." Since they can say a great deal more about presentations than about the modifications, of which no physiological characterization whatever has yet been reached and which are unknown, they make use of the elliptical statement that the presentation is localized in the nerve cell. This way of putting matters, however, at once leads to a confusion between the two things, which need have no resemblance to each other. In psychology a simple presentation is something elementary for us, which we can sharply distinguish from its connections with other presentations. This leads us to suppose that the physiological correlate of the presentation—i.e. the modification that originates from the excited nerve fibre with its termination at the centre—is something simple too, which can be localized at a particular point. To draw a parallel of this kind is of course entirely unjustifiable; the characteristics of the modification must be established on their own account and, independently of their psychological counterpart. ²

¹ [In English in the original. The phrase is from Hughlings-Jackson.]
² [Hughlings-Jackson has given the most emphatic warning against confusions of this kind between the physical and the psychical in the process of speech: 'In all our studies of diseases of the nervous system physiologischen Welt sehr komplizierte Verhältnisse entsprechen, weiß man von der "einfachen Sinnesvorstellung" denn mit größerer Bestimmtheit, daß sie etwas anderes als ein solches Kunstwort ist?

Die Kette der physiologischen Vorgänge im Nervensystem steht ja wahrscheinlich nicht im Verhältnis der Kausalität zu den psychischen Vorgängen. Die physiologischen Vorgänge hören nicht auf, sobald die psychischen begonnen haben, vielmehr geht die physiologische Kette weiter, nur daß jedem Glied derselben (oder einzelnen Gliedern) von einem gewissen Moment an ein psychisches Phänomen entspricht. Das Psychische ist somit ein Parallelvorgang des Physiologischen ("a dependent concomitant").


³ [Der Ausdruck stammt von Hughlings Jackson.]
⁴ [D. h. von der physiologischen zur psychologischen Betrachtungsweise.]
What, then, is the physiological correlate of a simple presentation or of the same presentation when it recurs? Clearly nothing static, but something in the nature of a process. This process admits of localization. It starts from a particular point in the cortex and spreads from there over the whole cortex or along certain tracts. When this process is completed, it leaves a modification behind in the cortex that has been affected by it—the possibility of remembering. It is highly doubtful whether there is anything psychical that corresponds to this modification either. Our consciousness shows nothing of a sort to justify, from the psychical point of view, the name of a 'latent mnemic image'. But whenever the same state of the cortex is provoked again, the psychical aspect comes into being once more as a mnemic image...

we must be on our guard against the fallacy that what are physical states in lower centres fine away into psychical states in higher centres; that, for example, vibrations of sensory nerves become sensations, or that somehow or another an idea produces a movement. ' (1878, 306.)
APPENDIX C

WORDS AND THINGS

[The final section of Freud's paper on 'The Unconscious' seems to have roots in his early monograph on aphasia (1891b). It may be of interest, therefore, to reproduce here a passage from that work which, though not particularly itself, nevertheless throws light on the assumptions that underlay some of Freud's later views. The passage has the further incidental interest of presenting Freud in the very unusual position of talking in the technical language of the 'academic' psychology of the later nineteenth century. The passage here quoted follows after a train of destructive and constructive anatomical and physiological argument which has led Freud to a hypothetical scheme of neurological functioning which he describes as the 'speech apparatus'. It must be noted, however, that there is an important and perhaps confusing difference between the terminology Freud uses here and in 'The Unconscious'. What he here calls the 'object-presentation' is what in 'The Unconscious' he calls the 'thing-presentation'; while what in 'The Unconscious' he calls the 'object-presentation' denotes a complex made up of the combined 'thing-presentation' and 'word-presentation'—a complex which has no name given to it in the Aphasia passage. The translation has been made specially for this occasion, since, for terminological reasons, the published one was not entirely adapted to the present purpose. As in the last section of 'The Unconscious', we have here always used the word 'presentation' to render the German 'Vorstellung', while 'image' stands for the German 'Bild'. The passage runs from p. 74 to p. 81 of the original German edition.]

I now propose to consider what hypotheses are required to explain disturbances of speech on the basis of a speech apparatus constructed in this manner—in other words, what the study of disturbance of speech teaches us about the function of this apparatus. In doing so I shall keep the psychological and anatomical sides of the question as separate as possible.

ANHANG C

WORT UND DING

[Es hat den Anschein, daß der Schlussabschnitt von Freuds Abhandlung 'Das Unbewußte' seine Wurzeln in der frühen Monographie über die Aphasie (1891b) hat. Es mag daher von Interesse sein, hier eine Passage aus jener Arbeit anzuführen, die zwar in sich nicht gerade leicht verständlich ist, aber doch Licht auf die Annahmen werfen kann, die gewissen späteren Ansichten Freuds zugrunde liegen. Die Passage ist überdies deshalb aufschlußreich, weil sie uns, gänzlich ungewohnt, einen Freud zeigt, der sich in der Fachsprache der akademischen Psychologie des ausgehenden neunzehnten Jahrhunderts ausdrückt. Der hier zitierten Stelle geht eine ganze Folge negativer und bestätigender anatomischer und physiologischer Argumente voran, die Freud schließlich zu jenem hypothetischen Schema neurologischer Leistungen führte, das er dann als 'Sprachapparat' beschreibt. Es sei jedoch angemerkt, daß zwischen der hier verwendeten Terminologie und dem Sprachgebrauch in 'Das Unbewußte' ein wichtiger und vielleicht verwirrender Unterschied besteht. Was er hier 'Objektpresentation' nennt, heißt in 'Das Unbewußte': 'Sachvorstellung'; was dagegen in 'Das Unbewußte' mit 'Objektpresentation' bezeichnet wird, meint einen aus 'Sachvorstellung' und 'Vorsatzvorstellung' kombinierten Komplex. Dieser Komplex hat in der hier zitierten Passage noch keinen Namen. Die angeführte Stelle steht in der deutschen Originalausgabe auf den Seiten 74–81.]

Wir wollen nun nachsehen, welcher Annahmen wir für die Erklärung der Sprachstörungen auf Grund eines solchen Aufbaues des Sprachapparates bedürfen, mit anderen Worten, was uns das Studium der Sprachstörungen für die Funktion dieses Apparates lehrt. Dabei wollen wir die psychologische und die anatomische Seite des Gegenstandes möglichst voneinander trennen.
From the point of view of psychology the unit of the function of speech is the ‘word’, a complex presentation, which proves to be a combination put together from auditory, visual and kinaesthetic elements. We owe our knowledge of this combination to pathology, which shows us that in organic lesions of the apparatus of speech a disintegration of speech takes place along the lines on which the combination is put together. We shall thus expect to find that the absence of one of these elements of the word-presentation will prove to be the most important indication for enabling us to arrive at a localization of the disease. Four components of the word-presentation are usually distinguished: the ‘sound-image’, the ‘visual letter-image’, the ‘motor speech-image’ and the ‘motor writing-image’. This combination, however, turns out to be more complicated when one enters into the probable process of association that takes place in each of the various activities of speech:—

(1) We learn to speak by associating a ‘sound-image of a word’ with a ‘sense of the innervation of a word’. After we have spoken, we are also in possession of a ‘motor speech-presentation’ (centripetal sensations from the organs of speech); so that, in a motor respect, the ‘word’ is doubly determined for us. Of the two determining elements, the first—the innervatory word-presentation—seems to have the least value from a psychological point of view; indeed its appearance at all as a psychical factor may be disputed. In addition to this, after speaking, we receive a ‘sound-image’ of the spoken word. So long as we have not developed our power of speech very far, this second sound-image need not be the same as the first one, but only associated with it. At this stage of speech-development—that of early childhood—we make use of a language constructed by ourselves. We behave in this like motor aphasics, for we associate a variety of extraneous verbal sounds with a single one produced by ourselves.

1 [It was once supposed that actively initiated movements involved a peculiar sort of sensation connected directly with the discharge of nervous impulses from the motor areas of the brain to the muscles. The existence of this “innervation-sense”, or sense of energy put forth, is now generally denied.] Stout (1938, 258). This last remark is confirmed by Freud a few lines lower down.

2 [The second sound-image is the sound-image of the word spoken by ourselves, and the first one is that of the word we are imitating (the sound-image mentioned at the beginning of the paragraph).]

Für die Psychologie ist die Einheit der Sprachfunktion das »Wort«, eine komplexe Vorstellung, die sich als zusammengesetzt aus akustischen, visuellen und kinästhetischen Elementen erweist. Die Kenntnis dieses Zusammensetzung verdanken wir der Pathologie, welche uns zeigt, daß bei organischen Läsionen im Sprachapparat eine Zerlegung der Rede nach dieser Zusammensetzung eintritt. Wir werden so撵arauf vorgelegt, daß der Wegfall eines dieser Elemente der Wortvorstellung sich als das wesentlichere Kennzeichen erweisen wird, welches uns auf die Lokalisation der Erkrankung zu schließen gestattet. Man führt gewöhnlich vier Bestandteile der Wortvorstellung an: das »Klangbild«, das »visuelle Buchstabenbild«, das »Sprachbewegungsbiild« und das »Schreibbewegungsbiild«. Diese Zusammensetzung erscheint aber komplizierter, wenn man auf den wahrscheinlichen Assoziationsvorgang bei den einzelnen Sprachverrichtungen eingeht:


2 [Das zweite Klangbild ist dasjenige des Wortes, das wir selbst sprechen; das erst (am Anfang des Absatzes erwähnte) Klangbild ist dasjenige des Wortes, das wir nachahmen.]
(2) We learn to speak the language of other people by endeavouring to make the sound-image produced by ourselves as like as possible to the one which gave rise to our speech-innervation. We learn in this way to 'repeat'—to 'say after' another person. When we juxtapose words in connected speech, we hold back the innervation of the next word till the sound-image or the motor speech-presentation (or both) of the preceding word has reached us. The security of our speech is thus overdetermined, and can easily stand the loss of one or other of the determining factors. On the other hand, a loss of the correction exercised by the second sound-image and by the motor speech-image explains some of the peculiarities of paraphasia, both physiological and pathological.

(3) We learn to spell by linking the visual images of the letters with new sound-images, which, for their part, must remind us of verbal sounds which we already know. We at once 'repeat' the sound-image that denotes the letter; so that letters, too, are seen to be determined by two sound-images which coincide, and two motor presentations which correspond to each other.

(4) We learn to read by linking up in accordance with certain rules the succession of innervatory and motor word-presentations which we receive when we speak separate letters, so that new motor word-presentations arise. As soon as we have spoken these new word-presentations aloud, we discover from their sound-images that the two motor images and sound-images which we have received in this way have long been familiar to us and are identical with the images used in speaking. We then associate the meaning which was attached to the primary verbal sounds with the speech-images which have been acquired by spelling. We now read with understanding. If what was spoken primarily was a dialect and not a literary language, the motor and sound-images of the words acquired through spelling have to be super-associated with the old images; thus we have to learn a new language—a task which is facilitated by the similarity between the dialect and the literary language.

It will be seen from this description of learning to read that it is a very complicated process, in which the course of the

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1 [In German 'überbestimmt'. The synonymous term 'überdeterminiert' is the one used so frequently in Freud's later writings to express the notion of multiple causation. Cf. Standard Ed., 2, 212 n.]
associations must repeatedly move backwards and forwards. We shall also be prepared to find that disturbances of reading in aphasia are bound to occur in a great variety of ways. The only thing that decisively indicates a lesion is a disturbance in the reading of separate letters. The combination of letters into a word takes place during transmission to the speech-tract and will thus be abolished in motor aphasia. An understanding of what is read is arrived at only through the medium of the sound-images produced by the words that have been spoken, or through the medium of the motor word-images that arose in speaking. It is therefore seen to be a function that is extinguished not only where there are motor lesions, but also where there are acoustic ones. Understanding what is read is further seen to be a function independent of the actual performance of reading. Anyone can discover from self-observation that there are several kinds of reading, in some of which we do without an understanding of what is read. When I am reading proofs with a view to paying special attention to the visual images of the letters and other typographical signs, the sense of what I read escapes me so completely that I have to read the proofs through again specially, if I want to correct the style. When, on the other hand, I am reading a book that interests me, a novel, for instance, I overlook all the misprints, and it may happen that the names of the characters in it leave only a confused impression on my mind—a recollection, perhaps, that they are long or short, or contain some unusual letter, such as an ‘x’ or a ‘z’. When I have to read aloud, and have to pay particular attention to the sound-images of my words and the intervals between them, I am once more in danger of concerning myself too little with the meaning of the words; and as soon as I get tired I read in such a way that, though other people can still understand what I am reading, I myself no longer know what I have read. These are phenomena of divided attention, which arise precisely here because an understanding of what is read only comes about in such a very circuitous way. If the process of reading itself offers difficulties, there is no longer any question of understanding. This is made clear by analogy with our behaviour when we are learning to read; and we must be careful not to regard the absence of understanding as evidence of the interruption of a tract. Reading aloud is not to be regarded as a process in any way different from reading to oneself, apart

from the fact that it helps to divert attention from the sensory part of the process of reading.

(5) We learn to write by reproducing the visual images of the letters by means of innervatory images of the hand, till the same or similar visual images appear. As a rule, the writing images are only similar to, and super-associated with, the reading images, since what we learn to read is print and what we learn to write is hand-writing. Writing proves to be a comparatively simple process and one that is not so easily disturbed as reading.

(6) It is to be assumed that later on, too, we carry out these different functions of speech along the same associative paths as those along which we learnt them. At this later stage, abbreviations and substitutions may occur, but it is not always easy to say what their nature is. Their importance is diminished by the consideration that in cases of organic lesion the apparatus of speech will probably be damaged to some extent as a whole and be compelled to return to the modes of association which are primary, well-established and lengthier. As regards reading, the 'visual word-image' undoubtedly makes its influence felt with practised readers, so that individual words (particularly proper names) can be read even without spelling them.

A word is thus a complex presentation consisting of the images enumerated above; or, to put it in another way, there corresponds to the word a complicated associative process into which the elements of visual, acoustic and kinaesthetic origin enumerated above enter together.

A word, however, acquires its meaning by being linked to an 'object-presentation', at all events if we restrict ourselves to a consideration of substantives. The object-presentation itself is once again a complex of associations made up of the greatest variety of visual, acoustic, tactile, kinaesthetic and other presentations. Philosophy tells us that an object-presentation consists in nothing more than this—that the appearance of there being a 'thing' to whose various 'attributes' these sense-impressions bear witness is merely due to the fact that, in enumerating the sense-impressions which we have received from an object, we also assume the possibility of there being a large number of further impressions in the same chain of associations (J. S.

1 The 'thing-presentation' of the paper on 'The Unconscious' (p. 201 ff.).

APPENDIX C

Aufmerksamkeit von dem sensorischen Teil des Lesevorganges abziehen hilft.

(5) Wir lernen schreiben, indem wir die visuellen Bilder der Buchstaben durch innervationsbilder der Hand reproduzieren, bis gleiche oder ähnliche visuelle Bilder entstanden sind. In der Regel sind die Schriftbilder den Lesebildern nur ähnlich und supersoaziiert, da wir Druckschrift lesen und Handschrift schreiben lernen. Das Schreiben erweist sich als ein verhältnismäßig einfacher und nicht so leicht wie das Lesen zu störender Vorgang.


Das Wort ist also eine komplexe, aus den angeführten Bildern bestehende Vorstellung oder, anders ausgedrückt, dem Wort entspricht ein verwickelter Assoziationsvorgang, den die aufgeführten Elemente visuell, akustischer und kinästhetischer Herkunft miteinander eingehen.

Das Wort erfährt aber seine Bedeutung durch die Verknüpfung mit der »Objektvorstellung«, wenigstens wenn wir unsere Betrachtung auf Substantiva beschränken. Die Objektvorstellung selbst ist wiederum ein Assoziationskomplex aus den verschiedenartigsten visuellen, akustischen, taktile, kinästhetischen und anderen Vorstellungen. Wir entnehmen der Philosophie, daß die Objektvorstellung außerdem nichts anderes enthält, daß der Anschein eines »Dinges«, für dessen verschiedene Eigenschaften jene Sinneserindrücke sprechen, nur dadurch zustande kommt, daß wir bei der Aufzählung der Sinneserindrücke, die wir von einem Gegenstande erhalten haben, noch die Möglichkeit einer großen Reihe neuer Eindrücke in derselben Assoziationskette hinzunehmen

[Die »Sachvorstellung« in der Abhandlung »Das Unbewußte« (oben, S. 159 ff.).]
The object-presentation is thus seen to be one which is not closed and almost one which cannot be closed, while the word-presentation is seen to be something closed, even though capable of extension.

The pathology of disorders of speech leads us to assert that the word-presentation is linked at its sensory end (by its sound-images) with the object-presentation. We thus arrive at the existence of two classes of disturbance of speech: (1) A first-order aphasia, verbal aphasia, in which only the associations between the separate elements of the word-presentation are disturbed; and (2) a second-order aphasia, asymbolic aphasia, in which the association between the word-presentation and the object-presentation is disturbed.

I use the term 'asymbolia' in a sense other than that in which it has been ordinarily used since Finkelnburg, because the relation between word [-presentation] and object-presentation rather than that between object and object-presentation seems to me to deserve to be described as a 'symbolic' one. For dis-

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1 Cf. J. S. Mill, *A System of Logic* (1843), Book I, Chapter III, also *An Examination of Sir William Hamilton’s Philosophy* (1865).

2 Quoted by Spamer (1876).

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(J. S. Mill) 2. Die Objektvorstellung erscheint uns also nicht als eine abgeschlossene, kaum als eine abschließbare, während die Wortvorstellung uns als etwas Abgeschlossenes, wenngleich der Erweiterung fähiges erscheint.
APPENDIX C

Turbances in the recognition of objects, which Finkelnburg classes as asymbolia, I should like to propose the term 'agnosia'. It is possible that 'agnostic' disturbances (which can only occur in cases of bilateral and extensive cortical lesions) may also entail a disturbance of speech, since all incitements to spontaneous speaking arise from the field of object-associations. I should call such disturbances of speech third-order aphasias or agnostic aphasias. Clinical observation has in fact brought to our knowledge a few cases which require to be viewed in this way...
I, without having new observations of my own, I attempt to treat a topic to which the best minds in German and foreign neurope-thology—such as Wernicke, Kussmaul, Lichtheim and Grashey, Hughlings Jackson, Bastian and Ross, Charcot, and others—have devoted their efforts, I had best immediately indicate the few aspects of the problem which I hope to advance through my discussion" (AA 1).
Freud quotes from a reported case, and the opinion of "one of the most prudent German neurologists," Carl Eisenlohr, who finds Lichtheim's theory inadequate, terming it "only of primarily didactic value" (AA 10).

"Wernicke's conduction aphasia does not exist because a form of speech disturbance with its characteristics cannot be found" (AA 12).

Freud here adds, in a footnote to Bastian, that Bastian "is inclined to explain" one syndrome "as the result of the anatomic contiguity of the connections between Broca's and Wernicke's areas passing through the insula" (AA 13).

Freud concludes, "paraphasia might well be regarded as a purely functional symptom, a sign of reduced efficiency of the apparatus of speech association" (AA 13).

"However, one meritorious author, Allen Starr, took the trouble of investigating the anatomic foundations of paraphasia. He came to the conclusion that paraphasia can be produced by lesions in very different regions. He found it impossible to discover a consistent difference in the pathology of cases of sensory aphasia with or without paraphasia." (AA 14).

Freud even enlists Wernicke's follower Lichtheim as an ally: "Lichtheim must have sensed the flaw in Wernicke's explanatory effort, because he defined much more concisely the conditions under which paraphasia did not occur" (AA 16). "Only one step more," Freud writes, and Lichtheim would have arrived at the proper insight. Freud's own conclusion at this stage is that "the destruction of a so-called center is characterized only by simultaneous interruption of several fiber tracts [pathways]" (AA 17).

"Since I know that I am quite isolated in claiming that the special psychic status attributed to the speech centers ought to reveal itself in some way in the clinical symptoms of speech disorders, I do not want to fail to mention that Wattville expressed a very similar line of thought in a short but substantial paper" (AA 18).
We must understand paraphasia to be a language disturbance whereby the appropriate word is replaced by a less appropriate one that always maintains a certain relationship, however, to the correct word. (AA 22)

"In other cases, such as that of Wernicke himself, at least the poverty of word formations with any more specific meaning, the overabundance of particles, interjections, and other linguistic embellishments, the frequent repetition of nouns and verbs is noteworthy" (AA 23).

Freud suggests naming the phenomenon "word impoverishment with copious speech impulses" instead of Wernicke's name "conservation of vocabulary with paraphasia" (AA 24); “impoverishment of all more specific parts of speech, nouns, adjectives, and verbs, an abundance of indifferent parts of speech, along with repetition of those words she had once managed to say" (AA 24).

He writes that the lesions were "transcortical" only in the sense of the term that makes it so unsuited for use in the theory of aphasia. [The lesion] consisted in one case of a hemorrhage over the motor center, in another of a bone fragment which had lodged in it" (AA 27).

We know that the portions of the brain whose pathology reveals itself in any way as symptoms, will always only produce local symptoms, whereby it is up to us to guess the diagnosis of the process on the basis of accessory circumstances [Nebenumständen] or from the course of the disease. However, the language apparatus [Sprachapparat] disposes over such a wealth of means of expressing symptoms that we can expect from it alone that it will betray not only the site but also the nature of the lesion by the type and manner of disturbance of function. Perhaps we will succeed one day in clinically distinguishing aphasias caused by hemorrhage from those caused by softening, and in recognizing a series of speech disturbances as characteristic for special processes in the language apparatus. (AA 25)
We now note that we have arrived at the point where we can explain a clinically observed form of language disturbance by a change in functional condition instead of by a localized interruption of a pathway. Since this step is so important for the entire understanding of aphasia, we want to repeat, for the sake of our own reassurance, that we were compelled to drop the localizing explanation because the autopsy findings (Heubner, Hammond) contradicted it. The assumption we have decided to make, along with Ch. Bastian, appears to us to derive naturally from the fact that [the capacity for] repetition always remains intact longer than [the capacity for] spontaneous speech. Later we will become acquainted with facts that will also demonstrate to us that the associative action of a center is less easily lost than the so-called "spontaneous" [action]. (AA 30)

To begin with, one would think that a reduction in excitability of a center would not need to be explained by a lesion; it appears to us as a purely "functional" condition. That is correct and there may be conditions similar to transcortical motor aphasia which result from mere functional impairment without organic lesion. However, if one considers the relationship of "organic lesion" to "functional disturbance," one must understand that a whole series of organic lesions can only manifest themselves through functional disturbances, and experience shows that these lesions in fact do just that. For decades we have been guided by the attempt to utilize the disturbances we see in clinical practice for understanding the localization of functions and have become accustomed to expecting an organic lesion to completely destroy a portion of the elements of the nervous system, while leaving the other parts completely undamaged, because only then is it [the lesion] exploitable for our purposes. Only a few lesions fulfill these requirements. Most are not directly destructive and draw a larger number of elements into the region of their disturbing effects.

Furthermore, one must consider the relationship of an incompletely destructive lesion to the apparatus which it has afflicted. Two cases are conceivable which are also found in reality. Either individual parts of the apparatus are maimed by the lesion while the intact parts continue to function in the usual manner, or it [the apparatus] reacts to the lesion as a whole in solidarity, does not reveal the loss of individual parts, but rather proves itself to be weakened in its function; it responds to the incompletely destructive lesion with a disturbance of function which could also come about through nonmaterial damage. (AA 31-32)

The language apparatus appears to show in all its parts the second kind of reaction to nondestructive lesions; it responds to such a lesion in solidarity (at least partial solidarity), that is, with a functional disturbance. For example, it never happens that consequent to a small lesion in the motor center a hundred words are lost whose nature depends only on the site of the lesion. It can be shown every time that the partial loss is the expression of a general functional curtailment of that center. It is not, by the way, a matter of course that the language centers behave this way. The fact that they do will later assist us in arriving at a very specific idea of their structure. (AA 32)
It was the first to explore again the true relationship of the speech centers to one another and their dependence on the center for sound images; it was the first to convey to us an idea [Vorstellung] of the complicated and multiply geniculated [vielfach in seiner Richtung geknickten Ablauf] course of associations in the speech process; finally, by providing proof that all reading is done by spelling, it established unshakably the correct point of view for judging reading disturbances. (AA 44)

These Vorstellungen are residues of impressions which arrived via the pathway of the visual and auditory nerves, or which originated in speech movements as a sensation of innervation or perception of the movement that was carried out. Depending on their origin from one of these sources, they lie together in the cerebral cortex, so that one site includes all "word sound images," another all "word movement images," [such as of the mouth and tongue] etc. These distinct cortical centers are connected by masses of white fibers (association bundles), and between the centers is "unoccupied territory" of the cortex, called "functional gaps" by Meynert. (AA 46)
If one were to follow a nerve pathway on its multiply altered and branching course through gray matter, one would have to conclude, according to Freud, "that a fiber on the way to the cerebral cortex has changed its functional significance [or "meaning," Bedeutung] after every new emergence from gray matter." He follows with an example of the course of the optic nerve in relation to retinal impressions, proposing that the changed (he writes "new") version of the nerve fiber has assumed more complex functions, or a "change in significance [or "meaning"; Bedeutungssänderung]." With regard to the transfer of skin and muscle sensation, the change in meaning, he speculates, must be even more complex; "we have as yet no idea which elements join together in the new content of the transmitted stimulus" (AA 54-55).

We can surmise only that those fibers which arrive in the cortex after permeating the gray matter still contain a connection to the periphery of the body but can no longer produce a topically similar image, just as—to take an example from the subject that occupies us here—a poem contains the alphabet in a rearrangement [Umordnung] that serves other purposes, in manifold connections among the individual topical elements, whereby some may be represented repeatedly, others not at all. (AA 55)

If one could follow in detail this rearrangement that takes place from the spinal projection on to the cerebral cortex, one would probably find that the principle is a purely functional one and that the topical aspects are maintained only insofar as they coincide with the requirements of function. Since there is no evidence that this rearrangement is reversed in the cortex in order to produce a topographically complete projection, we may surmise that the periphery of the body is not at all contained topically, but only according to function in the higher parts of the brain as well as in the cortex. (AA 55)

There follows a biologic analogy that inflects the discussion toward complexity, in particular, expanding Freud's point about the complexity of physiological phenomena: "Of course we do not have the slightest idea how animal tissue is able to manage undergoing and keeping separate such manifold modifications. But that it is able to do so is proved by the example of the spermatozoa,27 in which the most varied and detailed of such modifications lie ready to develop" (AA 58).

These moments seem inflected by and toward language—as Freud wrote, "the subject that occupies us here."

Freud included a footnote with a quotation from Jackson, which he introduced by noting that "Hughlings Jackson warned most sharply against such a confusion of the physical with the psychic in the language process" (AA 58).

With this refutation of a separate localization for das Vorstellen [a verb used as a noun] and the association of the Vorstellungen, a primary reason for us to differentiate between centers and pathways of speech has been ruled out. At every site in the cortex that serves the language function we can presume similar functional processes, and it is not necessary for us to call upon white fiber masses to which we delegate the association of the Vorstellungen found in the cortex. We even have an autopsy finding which proves to us that association of Vorstellungen occurs through pathways that lie within the cortex itself. I refer again to Heubner's case, from which we have already learned one important lesson. (AA 58-59; my italics except for Freud's "one")

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Accordingly, we reject the assumptions that the language apparatus consists of separate centers which are divided by cortical regions that are without function, furthermore that the Vorstellungen (mnemonic images) [Erinnerungsbilder] that serve speech are stored at particular cortical sites which are to be called centers, while their association is provided exclusively by subcortical white-fiber tracts [Fasernzassen]. After that, it only remains for us to state the view that the language area [Sprachgebiet] of the cortex is a coherent cortical region within which the associations and transmissions upon which the language functions are based take place with a complexity that is beyond comprehension. (AA 64)

The association area of language which visual, auditory, and motor (or kinesthetic) elements enter extends for that very reason between the cortical fields of those sensory nerves and the related motor cortical fields. If we now imagine a lesion that can be moved within this association field, its effect will be greater (given unchanged dimensions) the closer it approaches to one of the cortical fields, thus the more peripherally it is located within the language area. If it abuts directly on one of these cortical fields, it will cut off the association area of language from one of its tributaries: the language mechanism will be deprived of the visual, the auditory element, etc., since every stimulus for association of this kind originated from the relevant cortical field. If one moves the lesion further toward the interior of the association field, its effect will be indistinct; by no means will it be able to destroy all the possibilities for any one kind of association. In this way the sections of the language field that abut the cortical fields of the visual, auditory, and motor cranial nerves gain the significance attributed to them by pathology, which has led to their being put forth as centers for language. This significance, however, holds only for the pathology and not for the physiology of the language apparatus, since one cannot maintain that different or more important processes take place in them than in those parts of the language field which can better tolerate lesions. This view follows directly from the refusal to separate the process of Vorstellung from that of association and localize both processes at different sites. (AA 65–66)
I confess that in view of the importance of the "sound images" for the use of language, it has been extremely difficult for me to attribute to this subcortical sensory aphasia another explanation that would dispense with the assumption of an afferent auditory pathway [a reference to Lichtheim's diagram]. I was already on the verge of explaining this case of Lichtheim's through individual independence of other speech elements from the sound images, since Lichtheim's patient was a highly educated journalist. This, however, would rightly have been seen as nothing but an evasion.

Thus I searched the literature for similar cases. Wernicke, in reviewing the Lichtheim paper, stated that he had made a completely analogous observation and would communicate it in the regular reports from his clinic. However, I have been so unskilled as to be unable to find this communication in the literature. On the other hand I did come across a case of Giraudeau's that at least strongly resembles the Lichtheim case. (AA 70-71)

munication would be able to suspect that this lesion caused anything other than the usual form of sensory aphasia with serious speech disturbance. But there is something else to be considered." One knockout blow is followed by a second: his observation about the nature of the tumor's growth and its easy separability from surrounding tissue. Thus Freud adds together the external and the internal evidence to arrive at a conclusion that supports his thesis: "I therefore believe I am justified in assuming that subcortical sensory aphasia is due not to a lesion of the subcortical pathway but rather to damage to the same area that is otherwise considered responsible for cortical sensory aphasia. However, I cannot completely explain the specific functional condition that I must presume to exist at the site thus affected" (AA 72).

The object-presentation itself is once again a complex of associations made up of the greatest variety of visual, acoustic, tactile, kinaesthetic and other presentations. Philosophy tells us that an object-presentation consists in nothing more than this—that the appearance, of there being a 'thing' to whose various 'attributes' these sense-impressions bear witness is merely due to the fact that, in enumerating the sense-impressions which we have received from an object, we also assume the possibility of there being a large number of further impressions in the same chain of associations (J. S. Mill). The object-presentation is thus seen to be one which is not closed and almost one which cannot be closed, while the word-presentation is seen to be something closed, even though capable of extension. (SE 14:213-214; AA 79-80)
The word-presentation is shown as a closed complex of presentations, whereas the object-presentation is shown as an open one. The word-presentation is not linked to the object-presentation by all its constituent elements, but only by its sound-image. Among the object-associations, it is the visual ones which stand for the object, in the same kind of way as the sound-image stands for the word. The connections linking the sound-image of the word with object-associations other than the visual ones are not indicated. (SE 14:214.) From Sigmund Freud, Zur Auffassung der Aphasien: Eine kritische Studie (Leipzig: Franz Deuticke, 1891), 79.

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In order to explain the appearance of language centers, the cortical fields for acoustic, optical, and arm muscles are schematized by circles; the association pathways that reach from them to the interior of the language field are represented by pencils of rays. Wherever the latter are crossed by pencils that have been cut off from their origins, a "center" for the relevant association element is created. For the acoustic field, the double-sided connections are not indicated, partly in order not to complicate the diagram, partly because of the lack of clarity that exists about precisely the relationship between the auditory field and the acoustic language center. -- Dividing the connections with the optical field, also, spatially into two pencils permits taking into consideration the fact that eye movements are enlisted in a special way in the association process of reading. From Sigmund Freud, Zur Auffassung der Aphasien: Eine kritische Studie (Leipzig: Franz Deuticke, 1891), 83.

"functional effect over distance (Fernwirkung)" without an organic lesion of the language apparatus" (A.A 83), as opposed to cases of verbal and asymbolic aphasia, in which there are lesions of the language apparatus. He wants to separate the two types clearly, and to that end has drafted the diagram, "which disregards the anatomic situation" (A.A 83).

When Freud next mentions Pick, it is again in association with Heubner's findings, and in reference to a case of "transitory word deafness following epileptic attacks" (A.A 85).

Spontaneous speech is not abolished, no matter how marked the asymbolic-verbal aphasia is, but it is characterized by a poverty of parts of speech that have a narrow meaning. These (nouns, adjectives) were usually spoken in response to visual stimulation. In response to stimulation from other object associations which probably enter at other sites in the auditory field, the language field produces mutilated speech, or it transmits to the motor speech pathway all possible stimuli which do not require the more narrow object associations, such as particles, syllables (gibberish). (A.A 86-87)
Freud wrote: "It is to be expected that some symptoms of aphasia will be manifested differently in highly educated persons from the way they are manifested in those who are less articulate" (AA 90)

"We can accept these [stages] even if we disregard the centers of language in a physiological sense by saying that the visual, auditory, kinesthetic component of the language apparatus remains functional under such and such conditions" (AA 91)

"no single individual nerve fiber and nerve cell is enlisted for a single language association function; rather, a more complicated relationship reigns" (AA 91).

"The Bastianian modifications, in a certain sense, also constitute degrees of dis-involution, or functional retrogression." Freud next takes this amalgam as relevant for "every element of speech association activity," analyzing first the auditory, then the visual (reading and writing), then the motor elements. As do other aphasiologists, Freud places the "associative activity of the auditory element" (AA 92) at the heart of the entire language operation.

"The visual element is not directly connected to object associations (our characters are not, like those of other peoples, direct symbols of concepts, but rather of sounds)" (AA 94).

This case will be recognized by the fact that the inability to read is preceded by a briefly successful attempt to spell, and its interpretation will be that the damaged visual element, while capable of the simpler activity of associating the visual images one time with the auditory or kinesthetic element, is, however, incapable of multiple repetitions and correct sequencing of these activities which, in order to lead to reading, must in addition proceed with a certain amount of speed. This is a case of loss of the more complex ability while maintaining the simpler one. (AA 97-98)

Reading comprehension comes about only after transmission of the stimulus from the visual to the motor elements through association of the latter with the auditory elements. On the other hand, in the case of an auditory lesion, as well as of asymbolia, purely mechanical reading [capacity] can be retained. In other respects the explanation of reading disturbances—which I do not intend to consider in detail—causes several difficulties which can be disposed of neither by localizing factors nor by the assumption of the familiar functional changes. In complex cases sometimes some, sometimes other pieces of the [reading] function are retained, probably according to which of the elements that serve association in a particular direction have remained functional here or there in a large enough number. (AA 98)
I need to acknowledge an interesting and important point of view whose introduction into the theory of aphasia we owe to Charcot, since its acceptance would require us to modify our explanatory efforts to an even greater degree" (AA 100).

Nobody has so far wanted to completely dismiss Charcot's view. It remains to be seen, however, to what extent it has significance for the theory of language disturbances. Extreme claims, such as, for example, those by Stricker, for the predominance of the motor element for speech, were refuted by Ch. Bastian with the comment that he would first wait until a case was shown to him where a person had become word deaf after destruction of Broca's area. (AA 101)

"Different amounts of nervous arrangements in different positions are destroyed with different rapidity in different persons," [quoted by Freud in English] says Hughlings Jackson. (AA 102)

The consequence of these developments was that "the key to understanding language disturbances" was considered to lie in the "contrast between center and conduction aphasias" (AA 102).

Thus the language apparatus revealed itself to us as a coherent portion of the cortical area in the left hemisphere between the cortical terminations of the acoustic and optical nerves, and the fibers for motor control of speech and arms. The portions of the language field adjoining these cortical fields—with necessarily indeterminate boundaries—acquire the significance of language centers in the sense of pathological anatomy, not of function, because their lesion prevents one of the elements of language association from connecting with the others, which does not occur when a lesion is centrally located in the language field. (AA 104–105)

"It appears to us that with these theories the significance of the factor of localization for aphasia was overestimated and that we would be well advised to pay attention once again to the functional conditions of the speech apparatus" (AA 106–107).

"If the lesion is centrally located, however, the entire language apparatus suffers functional disturbances that result from its nature as an association mechanism, and which we have attempted to enumerate" (AA 106).