

THE PSYCHOLOGICAL BULLETIN

APHASIA.¹

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The problem of aphasia, a few years ago, has been treated monographically by a number of writers, such as Wyllie, Bastian, Bramwell and Elder, and, in this country, Langdon and Collins, by Miraillié, Pitres, and Bernard, in France, and the treatises of Mills, v. Monakow, and Dejerine added much of importance to the standard descriptions of Kussmaul, Lichtheim, Ross, Gowers and the pupils of the Salpêtrière. Aphasia has since passed into one of those stages of self-sufficiency which are so apt to retard progress because of excessive faith in the theoretical constructions and the idea that far more is solved and proved than is really the case. Throughout the literature on aphasia certain 'elements' of psycho-physical correlation are taken for granted, often enough without much concern as to the strength of their foundation, merely for plausibility's sake. The appearance of a review of the field by Wernicke furnishes some material concerning the problem of elements supported by the available data of aphasia, because Wernicke is a decided localizer, and yet strongly enough opposed to reading and writing centers to subject them to an extremely laborious and searching discussion. Wernicke gives in this 'lecture' very interesting and clean-cut statements of points which should be within reach of every worker in this rather neglected field; and also psychologically instructive illustrations of his method of combination of analysis and reconstruction, and, with it all, he rouses a new hunger for further casuistic evidence and for greater clearness concerning the concepts with which one works, or which one has reason to think are aroused in most readers.

¹ Wernicke, C., *Der aphasische Symptomencomplex*. 13th lecture of Die Deutsche Klinik (Berlin and Wien, Urban & Schwarzenberg, 1903), Vol. VI., pp. 487-556.

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The central issue of Wernicke's lecture is the relation of spoken and written language and the bearing of the 'word-concept' or 'word-notion.' He begins with a brief statement of a case of so-called pure or isolated agraphia (reported in full, *Monatssch. f. Psych. & Neurol.*, April, 1903). The patient is a woman forty-six years old; the symptoms had developed within nine months, first slowly with increasing weakness of the right hand, then with three more acute exacerbations; the second one brought a transitory loss of speech, leaving slight anarthria, and the third one a permanent picture of right hemiplegia and profound sensory disorders of the entire right side, and complete loss of spontaneous writing; only once the patient had been able to draw, under dictation and special urging, the letter 'a' and the figures 2, 3 and 4. Understanding and speech were perfectly normal, even the reading and understanding of letters and figures and words and of outlines and pictures. Any attempt to write—with chalk and blackboard, *i. e.*, with such movements as the right hand had not lost in the hemiplegia—led to a striking perplexity and expression of exhaustion.

It would seem very tempting to assume that such a condition of 'pure agraphia' would be referable to the incapacity of a special writing center, a loss of the memory of how to write words, just as a lesion of Broca's center is supposed (also by Wernicke) to lead to a loss of the memory of the movements necessary for speech. Wernicke gives, however, good reasons why the accepted views about a speech-utterance center should not be generalized, and that the assumption of a writing center would be premature, if not really erroneous. He predicts a lesion largely of association-paths (the centrum ovale underneath the posterior angle of the island).

Wernicke's historical sketch illustrates splendidly individual differences in the psychology and methodology of investigation. Meynert is given a very prominent position. His teachings are said to have given the clinical and experimental data of Broca and Hitzig the real foundation and to have furnished Wernicke the material for the assumption of a sensory speech center in the first temporal gyrus. 'A happy coincidence soon corroborated this supposition by two autisies'—an excellent illustration of how constructive imagination sharpens the attention needed for discovery. The discrepancy between Broca and Trousseau became intelligible, and the data seemed sufficient to attempt, with the help of diagrams, a synthetic reconstruction of the functions decomposed by the experiment of nature.

Just what should be assumed as safely established elements for

such reconstructions was probably considered too easy a matter at first. Wernicke thought of explaining all the facts out of the function of two centers and their connection, out of some data from the method of learning a foreign language, and the acquisition of language by the child — unfortunately a field of speculation rather than of safe knowledge even to-day, — and out of the rough clinical and localizatory experiences in aphasia. The sensory word center is the place where ‘sound-images’ have their nerve-cells or cortical elements. He thinks that the sound-appreciation is the first acquisition, to be followed by the acquisition of appreciation of its sense, or ‘secondary identification.’ What happens in learning a foreign language supports this differentiation. In connection with the word-sound concept, the child acquires a word-utterance concept by manifold practice; and the firm connection of the two is identified with the possession of the word-concept or *word-notion*, or what the French call ‘internal language.’ (Special decomposition of the words into letters is considered a secondary process.) The acquisition of the word-notion or word-meaning is the most important process in learning a language, and, for correct use of language, the integrity of ‘both the sensory and the motor component’ of the word-meaning would seem indispensable. Wernicke does not, however, follow Bastian in assuming that all speech function is a recapitulation of how words are acquired (a view which makes the most of the supremacy of the word-hearing center); he claims that after destruction of the sensory center articulated language is preserved; the speech-impulses from the entire remaining cortex reach the ‘word-notions’ directly, and, where the latter are mutilated, the speech-movement images, so that articulated language remains, though defective owing to loss of the regulating influence. These data ‘should be sufficient to understand the clinical picture of sensory aphasia; the chief symptoms can readily be derived from them.’

The scrupulous reader could hardly share such a faith in the safety and definition of the ‘elements’ offered. For ‘sound-images’ he is referred to the ‘cortical elements of the sensory speech-field’ without an inkling as to how they would work. The word-notion is said to have its substratum in the connection of definite elements of the sensory center with the corresponding definite elements of the motor center — a far-reaching claim, considering that there is not, as yet, any evidence of subdivision of the ‘center.’ Something of a substratum with a ‘word-notion’ is admitted to persist outside of this complex of direct connections, because destruction of the sensory center

does not necessarily abolish articulated speech or utterance. Wernicke is not explicit as to the make-up and localization of the substratum and nature of the 'word-notion.' He merely says: "Such a firm connection of memory-images which belong together constitutes the essence of 'Begriff' (of the idea, concept or notion)." The question is whether that which lies outside of the speech-field should or should not have a definite share in the 'word-notion.' This is, undoubtedly, a crucial point for any attempt at explaining language in terms of activity of special cortical *elements* of clearly limited speech-centers. Considering the revival of difficulties of histological definition of 'nerve-elements,' and the logical or verbal rather than functional abstraction of 'elements' in speech function, it seems hazardous to promise the possibility of deduction of the picture of sensory aphasia from the few elementary conceptions given. It should certainly be clearly understood that, so far, we can only contrast very broadly the apperceptive and the emissive functions as Ross called them; and that a discrimination of the actual 'elements' and the concept 'word-notion' is a merely temporary contrivance.

Wernicke's characterization of the clinical types is lucid and definite, and rendered here for comparison with the claims of other writers. He begins with *sensory aphasia*: Although there is no deafness or not enough to account for the disorder, the patient presents a defect of understanding of the word-sound and of the word-sense, as far as the patient depends on the interpretation of sound, while gesture and non-auditory signs are easily grasped (and must be guarded against in tests!). Articulated speech is preserved and even excessive, 'perhaps owing to the numerous misunderstandings.' "For although the patient uses a fairly rich vocabulary and good form of speech, he frequently blunders in the choice of words, and even uses wrong or disfigured words without being aware of it; under affect he usually speaks better."

Objects shown are usually wrongly named, often with paraphasia. The confusion of words in spontaneous speech may reach the degree of true unintelligible jargon-aphasia. What the patient replies does not start from words heard; loss of understanding of the word sound necessarily also frustrates repetition. Written language, depending on the word-concept or internal language, is always strongly affected in sensory aphasia. It is, however, not well studied, since it is not common property of all persons. *The onset* is usually acute, through occlusion of a vessel, usually with very slight shock and often without any hemiplegia. As to prognosis, Dejerine thinks it to be a lasting

defect; Wernicke, with most writers, accepts *restitution by reëducation* from ordinary life; difficulty in the understanding of rare words — he mentions vertebral column, knee-pit, arm-pit — and also in the *naming of objects* is the most persistent residual. The restitution of *written* language is not sufficiently studied to allow of generalization.

With a little consideration one is struck by the haziness of the elements, loss of which should account for the variations in the extent of individual clinical differences, the varying affections of the word material or word-concepts, and the individual differences in restitution, and such matters as the difficulty of naming objects after recovery of spontaneous speech. It is also striking how sadly deficient the literature is in such a simple matter as a good description of the accurate extent of lesions in terms which should stand the critical attitude of a Flechsig. With all the observations collected by Miraillié and Bastian (Wernicke does not dispose of all the cases opposed to his views), we still are in a very vague position, far from being able to deduce the concrete symptom-complex from a simple scheme.

Motor aphasia or aphemia is 'equally easy to describe.' (1) Articulated language or the mechanism of articulation is 'forgotten'; hence there is mutism with but few residuals, often only of senseless syllables or words, and even these are not used at will but automatically (recurrent utterances). In emotion or in sleep, words not otherwise available may be produced. There is no bulbar palsy, but frequently a slight hemiplegia, or slight hypoglossal palsy, not sufficient to explain the aphemia; for some time the patient may be unable to show the tongue, to puff the cheeks, gnash the teeth or even open the mouth to order without sticking out the tongue; these disorders are, like the aphemia, a defect of the memory of the way to do things, and often exist only in the first period of the aphemia. (2) The *understanding* of speech is largely correct; orders are correctly carried out, and mimic well responded to; but Wernicke admits now that there is *at least some difficulty for longer sentences* as Dejerine has shown; but this difficulty is usually open to improvement. This disorder of understanding is explained by the fact that the acquisition of speech-concepts is a fundamental phase in the learning of a language, and that their loss, in turn, has a variable influence on language as a whole. In this respect there are evidently individual variations (not further specified). (3) *Writing* keeps pace with articulated speech. *The onset* of motor aphasia is usually with a more marked shock and more or less right-sided hemiplegia. *The prognosis* is on the whole unfavorable except where the insult is merely slight or the interfer-

ence merely indirect (due to a lesion of a neighboring part, or not very infrequently a very severe insult of even the *right* hemisphere with left-sided hemiplegia). Restitution usually leaves much exaggeration of motion and slowness; a certain awkwardness and exaggerated effort resembling that of deaf-mutes who have learned to speak, and syllabic stumbling always remain even in favorable cases. *Repetition* remains as deficient as spontaneous speech. *Training* by optic methods, as in deaf-mutes, seems to give very favorable results (in six weeks — Dejerine and Thomas). In relatively rare cases the motor defect is not so complete. The patient may succeed in repeating easy words, or short sentences, but *never* more complicated words or sentences; vowels or syllables without any resemblance will be substituted, or the patient's own name, or an 'ach Gott.' *Partial motor aphasia does not seem to exist* beyond these exceptions.

This presentation gives a much more exclusive definition of the functional picture and its clinical evolution than is suggested by most English and American writers, who describe several types of cortical motor aphasia, and in turn, are more hopeful about the anatomical focal subdivision of the motor speech-field into a propositionizing and utterance center. One of Wernicke's claims, especially worth reiterating and testing, is the non-existence of partial motor aphasia. The function of the 'center' evidently is considered one 'en bloc,' not a sum of many individual word utterance memories.

Wernicke still inserts here his hypothetical *conduction aphasia*. The available empirical data are admitted to be scanty and not consistent. Paraphasia is not sufficient evidence of the interruption of the connection between hearing and utterance center. It can result from more causes than Wernicke first assumed. Nor does repetition of words heard prove the integrity of this simple path. The sound-picture seems to be sufficient for an understanding at least of ordinary words (Wernicke explains the recovery from sensory aphasia on this ground, *i. e.*, without the creation of a new auditory word-center!), and motor utterances may be roused spontaneously without a previous rousing of the sound-picture; therefore, destruction of the connective path will not necessarily frustrate the repetition of words to order, as long as they are *understood*. *Some* paraphasia will then occur, realized by the patient. Evidence of the *integrity* of the oldest and original conductive path would be furnished by automatic echolalia and by *repetition of foreign or senseless words*. Wernicke, therefore, looks for a case in which *merely* echolalic repetition would be *destroyed*, with preserved understanding and execution of speech, and

a paraphasia with realization of the mistakes. The recorded cases of lesions of the island demand such a restriction of the theoretical deduction if they are not directly opposed to the whole conception. And what becomes of the simplicity of the notion 'word-concept'? In conduction-aphasia it should be destroyed; but Wernicke explicitly admits the existence of a long-circuit substitute.

Convinced of the anatomical and clinical demonstration of a sensory and a motor speech-center, Wernicke proceeds to the construction of pure or subcortical aphasias as 'a necessary logical consequence.' There must occur cases in which the projection-system of the one center or the other is destroyed without interference of the centers themselves and their connection; these cases too show a loss of understanding of the word-sound, or a loss of articulated speech, but 'preservation of the internal language or intact word-notions.'

In *pure aphemia* (not infrequent, especially with hemiplegia), Wernicke finds some evidence of the correct intention of utterance, and the attempt at repetition is never so completely miscarried as with cortical destruction. Understanding is intact even for complicated sentences. *Written language* is quite *intact*. Dejerine published the first convincing cases with a lesion just beneath Broca's convolution, above the internal capsule, and demonstrated on them the fallacy of Charcot's view of a special writing center. Integrity of the word-concepts becomes the formula for the fact that the patient can write. In cases of illiteracy, Lichtheim has suggested the test of counting the syllables to demonstrate the integrity of the word-concept. The best sign, according to Wernicke, is the correct intonation of the speech rests, which should be in harmony with the rhythm of the intended utterances. Since the intonation-test necessarily fails where the patient has no speech rests, on which to produce the intonation, or where he does not grasp the issue of the test, one would welcome the simple contrivance of Onuf and Fraenkel, who merely depend on simple counting (turning over every sixth card of a pack) as evidence of integrity of internal language. I have, however, just recently had a chance to examine a patient of Doctor P. L. Murphy of Morganton, N. C., with motor aphasia in a state of partial reconstruction, who succeeded at once with the card test, *although* he has not recovered his writing as well as his speech, and gives no introspective evidence of knowing the words which he is not able to produce. This simple test can, therefore, no longer be accepted as sufficient evidence of what it claims to demonstrate clinically, not to speak of the danger of using it for anatomical inferences. The number of clinically and anatomi-

cally well established cases of subpictorial aphasia with really purely subcortical lesion is actually very small, and hardly larger than the number of cases in which the same clinical symptoms coexisted with destruction of the cortex itself. This is a serious objection to Wernicke's categorical attitude. It should be understood that the *collection and publication of such cases with all the clinical and anatomical detail is still urgently to be desired.*

Pure word deafness implies simple loss of understanding of words notwithstanding sufficient hearing, with integrity of word-concepts and all modalities of speech. Of this disorder Wernicke admits only one case of Liepmann as clinically and anatomically beyond doubt.¹

Wernicke next passes to a *plea for the transcortical aphasias* due to interruption of the 'connection of the motor or the sensory word centers with the concept regions.' Such a center for the 'word sense' or concrete concepts is here explained as a mere fiction, representing the firm connection of the visual, olfactory, auditory and tactile memories, which necessarily are complete inter- or transcortical complexes. (Wernicke declines to accept Flechsig's association or coagitation centers; all these fields have projection systems; 'the island and its claustrum might alone pass as association organ,' in faithful allegiance to Meynert!) *Transcortical sensory aphasia* is a loss of the word-sense with preserved appreciation of the word-sound — the active component of audition, repetition, is preserved (with but moderate para-

¹ The other spurious cases are made the basis of a discussion of some interest. Another of Wernicke's pupils, Freund, had tried to trace pure word deafness to a peripheral affection (of the labyrinth); this led to the utilization of Bezold's statement concerning the necessary range of sounds needed for the perception of words. A range between $b'-g''$ was found to be the necessary minimum, and at the most an octave below or above is used, according to Liepmann. Freund's case had this range, and *must* depend on a central lesion. Wernicke uses these data for further considerations: Our hearing covers eight octaves; only a small part is needed for the recognition of speech, and only about one fourth to one fifth of the projection fibers 'need' end in the speech center itself to make the hearing of speech possible. Hence its limitation to the posterior third or half of T_1 and the neighboring part of T_2 , whereas the rest of the temporal lobe must also very largely be a terminal auditory station (on what evidence?). The possibility of a pure auditory aphasia from a double-sided lesion (Pick, etc.) limited to the entrance zone for these sounds could not be excluded, if the above reasoning concerning localization of the tone-levels were correct. A patient might indeed lose both centers for the *tone-levels* of language. But Pick's cases really were completely deaf and would seem to belong to Bleuler's pseudo-word-deafness through insufficient hearing. The right-sided path would seem to play a rôle in *restitution* since it did not take place when both sides were affected (O. Berger).

phasia), and is enacted either on request, or as repetition in the form of a question, or in states of greater general reduction, wholly automatically, as echolalia. (Bastian speaks in such cases of isolation of the auditory word-center.)

Transcortical motor aphasia is a suspension or very considerable reduction of spontaneous speech, with correct repetition and understanding of language. The utterances are not always the same words or syllables as in cortical motor aphasia, but limited to expressions of displeasure, annoyance, helplessness, and the ability to recite memory material and to repeat spoken words shows the vocabulary to be unlimited. Articulation is perfect; yet no replies except an isolated simplest answer can be obtained.

In sensory transcortical aphasia reading is done correctly, only with occasional paraphasia, but without understanding.

In motor transcortical aphasia spontaneous writing is impossible, writing to dictation correct or slightly paraphasic, reading understood, though marred by paraphasia on reading aloud.

Finally Wernicke mentions combined forms: total aphasia with loss of comprehension and utterance, usually with loss of internal language, *always with hemiplegia*; or much more rarely with fairly preserved internal language, as a summation of the two transcortical forms, at times *without* hemiplegia; further mixtures of subcortical and transcortical forms — even more frequent than the pure forms.

Reviewing briefly what Wernicke claims for the speech-function as such, we find an 'auditory word-center' and a 'motor word-center,' and a direct and an indirect connection of the two are referred to under the common term 'word-concept' or 'word-notion.' Partial defects (loss of only a limited number of words) are not admitted in motor aphasia. In auditory aphasia, there is no record of any dropping out of special sounds. The occasionally reported loss of special languages or dialects evidently does not command Wernicke's attention. There are several word-functions; several degrees of identification (from appreciation of the word-sound to that of the meaning), and of verbal elaboration (from recurrent utterance through automatic echolalia to paraphasic utterances and finally free spontaneous speech); but for all of these we are merely given the 'word-concept,' in one place used as that word-function which allows of decomposition of the word into letters (where the ability to write is made the criterion between cortical and subcortical aphasia), in another place as the word-function sufficient for automatic echolalia (which need not even be understood).

We now follow Wernicke to the second part of his discussion

dealing with the question whether the occasional occurrence of isolated agraphia or of isolated alexia warrants the assumption of special reading and writing centers (with Charcot and Bastian and others), or the restriction to a reading center (Dejerine), or neither (Wernicke and v. Monakow).

Written language (symbolization by written signs) is acquired late and not common property of everybody, and therefore not provided with a uniform brain-mechanism such as we assume in the whole race for symbolization by word-sound. Hieroglyphs would have a mechanism different from the method with letters, which makes of reading a process of spelling, as Wernicke maintains with Grashey and Goldscheider, with visual memories only for letters and not for words, except for a small number of very common words (especially one's name). He therefore declines the identification of a visual word picture with an object without some intermediary 'thinking' in which the letters cease to be essential and of direct meaning. Charcot's case of thinking in written words is an extreme exception not fit for generalization, as little as the hypothetical types of 'moteurs, auditifs and visuels.' Nor would it be right to generalize from deaf-mutes. Wernicke does not know visual word-memories, but only twenty-five letters and a few ready-made compounds. He does not think it likely that there should be a visual duplication of what is already available in the sound-formula. *Disorders of reading and writing* are fundamentally distinguished as *either verbal, i. e.,* depending on disorder of the word-concept, *or literal,* independent of any such disorder, but due to non-recognition of the form of the letter. Wernicke specially considers the two cases of Rieger and Sommer in which cerebral traumatism led to imperception of a limited number of letters. The patient of Grashey, who could find the words for objects in no other way but by writing and only after the entire word was written, shows according to Wernicke merely a peculiar trick, and moreover that the letters and even combinations of letters are not directly related to the object but become so only when the material for the sound-equivalent is complete. *Written language*, being merely spelled language, is a *transcortical function* subordinated to the centers of speech, dependent on its integrity, and, in return, the best criterion of the integrity of word-concepts and of internal language.

In the main, the disorders of written language (as far as they are verbal) go parallel with those of spoken language. The understanding of what is read vanishes with that of what is spoken (or at least formulated), and the ability to write spontaneously with the ability to

speaking spontaneously, and paraphasia in reading aloud and in writing to dictation keeps pace with paraphasia on trying to repeat spoken words. Writing may be especially difficult because it depends not only on the ability of finding the word but of finding also the letters belonging to it.

In *cortical motor aphasia* the word-concept is, as a rule, profoundly disturbed, as shown by the *lasting alexia and agraphia* (which Bastian does not accept as due to lesion of the Broca center). The recognition and the copying of letters (even from print into writing) is, however, not involved. Yet, Thomas and Roux found that, in recovery, the patient first re-learns to read complete words, then simple syllables and, at last, single letters. Writing is apt to improve slowly, about as articulated speech, but more slowly for dictation than for spontaneous speech (Dejerine). v. Monakow errs when he minimizes the special importance of the integrity of the word concept for writing and when he claims that the motor aphasic is often able to write better than he speaks. This holds only for exceptions (Banti's case) — and v. Monakow claims for these cases disease of only the opercular lip of the Broca convolution.

Cortical sensory aphasia does not occur without very profound disorder of written language, especially agraphia is apt to be persistent, perhaps partly on account of the neighborhood of a path very essential for the motor act of writing. *Conclusive records are, however, scarce.*

The schematic presentation of the function of written language differs from the Lichtheim scheme of spoken language in the fact that evidently the motor execution cannot be roused directly from the concept-mechanisms (as the motor-speech utterance can be, without the help of the auditory center); it seems that writing always demands the rousing of the optic memory of the letters. On the other hand, the motor component is not essential for the recognition of letters, as is shown by our reading of printed letters.

The very foundation for writing is the existence of notions of direction, since we can write with any part of the body. A special center for writing movements of the right hand does not appear plausible, and is, so far, based 'on material uncritically used.'

A definite one-sided localization of the memories for letters has, however, been claimed with more appearance of justification and is upheld by Dejerine, Bastian, Pick, etc. Wernicke opposes this view with v. Monakow, as he did in his classical review of 1886, reprinted in his *Gesammelte Aufsätze*. Wernicke, to begin with, feels sure that

a visual *word-center* is not to be thought of, but at best a center for letters. Letter-images are distinguished above all other optic images by being: (1) Two dimensional and therefore 'having only one visual form,' not innumerable ones as the three dimensional objects (Storch); (2) used extremely often; and (3) devoid of a direct connection with concrete concepts, and devoid of associations apart from being connected with the one-sided speech-center, especially its auditory part. This alone does not, however, guarantee one-sided localization. Any special localization within the visual sphere is difficult to prove. There is not even a demonstration of any special cortical locality for sharpest vision, and of another locality for the most differentiated oculomotor directive concepts. The functional acquisition of letter concepts does not point solely to the left hemisphere either. Macular vision, which is almost alone concerned in the recognition of letters, is represented in either hemisphere, and large letters are equally soon recognized when approached in the right and the left visual field. Right hemianopsia may cause difficulty in reading, but it does not imply letter-blindness (even directly after the shock), although the latter is always combined with right hemianopsia. Bastian and Dejerine resort to the explanation that callosal fibers reach the specialized 'visual word-center' from the right hemisphere; but Wernicke sees in this an unjustified extension of the afferent optic path-way beyond its projection-field and a disregard of Meynert's fundamental law of the exclusively associative nature of the callosum. He claims that otherwise even the right hemianopsia would be covered up by callosal fibers from the normal hemisphere to the visual center cut off from the tract of its side (which might be relatively true if Dufour's distinction of hemianopsia with *vision nulle* or *vision noire* holds), and that the relation is quite different from that of the auditory afferent path to the auditory word-center, concerning which he says (p. 519, below) that the functional interruption of the auditory path to the left temporal lobe is the cause of the subcortical sensory aphasia, while in the only conclusive case of Liepmann he admits the importance of the participation of callosal fibers. "The facts of pathology refute the unilaterality and narrow localization of a visual word-center; what then creates the appearance to the contrary?" Evidently the close commissural relation with the one-sided speech-field, especially its auditory part, for which two possibilities are to be considered: 1. v. Monakow's view, that the focus underneath the angular gyrus cuts the afferent optic radiation of the left side, and the crossed visual-auditory commissure. The left-sided memories are not reached by stimulation. The right-sided ones

cannot be used because they cannot rouse the sound-component; reading by spelling would be lost, and reading would be limited to a few words read as a whole. Objects would be identified because their cortical representatives are connected with more than the auditory projection field; the frequent difficulty in naming objects will be discussed on p. 276. The recognition of forms, and among them the forms of letters (identification of the same letter in different alphabets and free copying) would remain: Yet in many of these cases copying is reduced to drawing; and could this be the effect of a simple interruption of the crossed visuo-auditory commissure?' The sound-component alone gives the signs their sense; and, with its loss, the sense is lost.

Alexia would therefore be a mixture of a left-sided subcortical lesion and the cutting off of the visuo-auditory commissure of the right visual center, whereas the preserved left visuo-auditory connection would remain sufficient for writing.

2. Dejerine's view assumes the principle of economy also to hold in a one-sided presentation of letters, in the left angular gyrus which alone would have a connection with the auditory speech-field.

However the future will decide this dilemma (after a reliable definition will be found for what constitutes the angular gyrus!), the denial of a unilateral letter-center is necessary to formulate the problem of inquiry of the callosal radiation. It would seem that the assumption of a visual word-center would make it easy to explain the agraphia in the case reported at the outset. A lesion just beyond this center, cutting the fibers to both arm-centers, would explain it. But why should the patient have lost at the same time the ability *to draw* the simplest figure?

Redich has found 27 cases of simple word-blindness (literal blindness?) or subcortical alexia. Wernicke adds the case of a man of 62, intelligent, without speech-disorder, who also writes quite well, but who cannot read anything, neither letters nor words, nor numbers (the latter are exempt in some cases of alexia). The patient sees, and is able to copy, letters and drawings, and even then does not understand the letters, while some patients of this type actually gain an understanding by going through the motion of writing. There is right hemianopsia. The patient recognizes objects, but occasionally has some difficulty about finding the right name (without a similar difficulty on palpation?), although he recognizes it at once from among a number of names mentioned to him. (In one of Wernicke's earlier observations a similar patient could not name any objects and

had also difficulty about finding names of concrete things in spontaneous speech—evidence of a true aphasic disorder.) At first the patient had even some difficulty in correctly *recognizing* objects seen—evidence of mind-blindness as a remote symptom of the focus implying the lasting alexia—and also a similar difficulty about recognizing objects merely palpated. Mind-blindness usually implies alexia; isolated alexia is, however, usually not complicated by additional mind-blindness. The lesion in the case is probably embolic; hemiplegic symptoms disappeared again in two to three weeks; but then the patient was found unable to read the paper.¹

Isolated simple alexia would depend on a deep seated lesion beneath the angular gyrus, with destruction of a subcortical (and a transcortical?) path and integrity of a transcortical one, passing nearer the cortex of the angular gyrus. Dejerine saw indeed an extension from the deep lesion beneath the angular gyrus (with simple alexia) extend to the cortex and to alexia + agraphia.

Rieger's patient, a sculptor of 32, developed, six months after a fracture of the skull, loss of *p*, *x* and *y* from the small German alphabet, these and *d*, *h*, *k* and *v* from the small Latin alphabet, and 14 capital letters from both alphabets: the above with the exception of *D*, and *B*, *E*, *F*, *M*, *N*, *R*, *T* and *W*. He could neither write nor identify these; also no numbers besides 0, 1, 2 and 3. He could use the available letters on dictation and copying only, and what he read was without understanding. Otherwise there was an occasional difficulty in finding a noun in spontaneous speech; he also found it difficult to name objects on vision and palpation, but always succeeded after a long while; for letters it took him about half as much time as for objects, but for the above letters there was complete abolition. A defect of retentive memory in all sensory domains was not less marked than in Grashey's case. The ability to draw was also gone; also the recitation of series. With all this there was no reduction of intelligence in a practical sense.

Sommer's case had a similar partial alexia and agraphia after an apoplexy. For several other letters there was a variable difficulty. Moreover, he could not compound even the preserved letters into words. Writing was practically abolished, but the writing of single letters was in many respects better preserved than the reading. The patient was hemiplegic for two weeks, had right hemianopsia, but,

¹ Dyslexia (Berlin) is probably akin to alexia, but merely a great fatigability of the reading-capacity leading to Lesescheu (Bruns), and due to atheroma or syphilitic vascular disease. Hemianopsia is not a *condition*, as in alexia.

apart from the alexia and agraphia and a casual difficulty in finding a word, he was perfectly normal.

The loss implicates not only the rare letters. The constancy of the defect 'seems to exclude purely functional factors,' — we should rather say stamps the cases as quite exceptional, with but one further analogy in the literature of cerebral pathology, a case of sensory-motor aphasia with only partial but constant vocabulary, following a psychosis (Heilbronner, *Z. f. Psych. & Phys. d. Sinnesorgane*, XXIV., p. 83). Rieger himself mentions a case of hysteria with loss of the letter *H*, and keeps aloof of localizing conjectures, whereas Wernicke suggests a lesion of paths belonging to the path *ca*, from the 'word-notion' to the visual memories of letters.

Pure isolated agraphia seems to be present in the case reported at the outset. But the internal language is not intact, and the original disorder appears to have been a transcortical motor aphasia. What is left of disorder of word-concepts does not wholly explain the strikingly motor character of the symptoms. The patient has her visual memories of letters but cannot transfer them to the motor apparatus, and since these memories are bilaterally located, there should be a *bilateral* interruption of the path between receptive and emissive centers for letters and drawing. The temporary presence of left-sided symptoms might indeed speak for a bilateral lesion; but other cases seem to have depended on left-sided lesions only; some diffuse damage may, however, have suspended the function not only of the affected but also of the opposite side. Evidence of such diffuse damage would lie in the slightly indicated transcortical disorder of *speech*. Pitres' case had at first general agraphia, but after a while it was limited to the right hand (which had otherwise recovered motility); the right hemiplegia with its profound disturbances had disappeared, leaving behind right hemianopsia, with good visual acuity. The remaining agraphia of the right hand should hardly be called a 'pure (motor) agraphia'; but it is a distinctly one-sided disorder of writing, such as, in *Liepmann's* case, was simply *part of* the right-sided apraxia (the patient wrote in mirror-writing with the left hand). The permanent absence of mirror-writing in Wernicke's patient favors to his mind the possibility of bilateral lesions.

The rare cases of *isolated literal agraphia* would seem to be analogous to conduction-aphasia; all forms of agraphia in which letters can still be shaped are, however, *verbal agraphia*, a consequence of disorders of spoken language, or of connection with the 'word-concepts.' Since writing is an additional task, it may occur that sen-

sory aphasia may recover just far enough to leave out this most difficult reaction, the translation of the word into letters. This is in harmony with the observation that paraphasic disturbances are usually exaggerated in writing, or may persist in writing when they have disappeared in spontaneous speech.

True paraphasia with disfigurement of the individual letters occurs oftenest in general paralysis or other diffuse loss of memory of the forms of letters.

It is easy to understand that Wernicke brings the problem of 'Wortfindung,' *i. e.*, finding the word or naming, in close connection with reading, the finding of the word or sound for letters. The naming of letters, like that of unisensual visual perception, such as colors, took only about one half the time taken by other objects, and in Grashey's case, letters could be named at once, even without the motor help of writing. The path for naming letters would probably be the inferior longitudinal fasciculus for the left and the crossed forcepts-tapetum tract for the right hemisphere. To incriminate the same path in Freund's optic aphasia, is probably incorrect. The naming of an object presupposes its secondary identification or recognition, *i. e.*, the association of the optic memory at least with the corresponding tactile memory, which is not necessary with letters. It is certainly necessary to test the naming for all sense-qualities, and to consider whether a concept does not anyhow depend largely on one sense (thunder, waltz on the auditory, and wind, warm, cold on the tactile sense). In Grashey-Wolff's case, the visual projection field seems to have been relatively best preserved; this might perhaps explain the unique fact that he found his words by the way of the letter-compounds.

In the cases of Rieger and Sommer the concepts for certain letters are lost. This has probably nothing to do with the fact that occasionally a patient can read, *i. e.*, name words, but not single letters. One of Bastian's cases could not name a solitary letter, and misread on account of marked paraphasia, but understood what he read. This may in part be an exaggeration of the difficulty caused by unaccustomed attention to a detail act, and in part to the facilitation by secondary identification (reading manuscripts from sense).

Lately Pitres has yielded to a practical need of recognizing a provisional picture of amnesic aphasia (without disorder of understanding, reading and writing). This heterogeneous group would include most cases of isolated word-blindness, and the cases of Grashey and Rieger (which are not explained simply by their defect of Merk-

fähigkeit), and a large number of cases in which the 'amnestic aphasia' is merely a residual of various disorders. In eight of ten cases the inferior parietal lobule was affected similarly to the lesion of predilection of Naunyn's 'indefinite aphasias'; lesion of the Broca convolution is probably least represented in such difficulty of naming. Amnestic aphasia consists solely in a lack of connection between concept and word; it has no definite localization and may be simulated by diffuse memory disorder. It is essential that cases with additional paraphasia should be distinguished from cases with mere difficulty in finding a word, such as a noun designating an object. What may be normal with rare words or words of a foreign language marks a symptom of defect when it occurs in the mother-tongue. In the systematic aphasia of polyglots, a stage of amnestic aphasia is apt to precede restitution. The difficulty of finding words is a special form of transcortical motor aphasia. Its climax may be reached in the actual loss of concepts as in Rieger's and Heilbronner's case, and in other cases there is at least a relative retardation of the rising of the concepts. Only the concept as a whole, not the individual sense-memory, is capable of rousing the word, with the exception of unisensual concepts. It is obvious that difficulties in the sensory spheres are most apt to so diminish the efflux to the concept mechanism as to leave it relatively inefficient in rousing the names. For the tracing of such defects Rieger's scheme is recommended.

Lack of space forces me to put off to another occasion the review of the anatomical considerations of Wernicke. What has been rendered of his general discussion cannot fail to rouse a wholesome desire for convincing observations of patients sufficiently capable of introspection to give more directness to the discussions, and with such anatomical examination as will put an end to the regrettable tendency of so many clinicians to consider the white matter of the hemispheres the cornucopia of all the desirable conduction paths.

The great advances in the studies of asymbolia and apraxia will do their share in shaping new problems for the elaboration of sensory impressions into speech- and writing-reactions, and in this connection Storch's work promises fair to do away with much of the brain-cell mythology with which the theory of aphasia is afflicted, and also the hazy dogmatism about the relation of concept and word.