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# B R A I N .

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Original Articles.

ON APHASIA.<sup>1</sup>

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WHEN we read the recent controversies concerning the symptoms of Aphasia and their explanation, we might readily come to the conclusion, that there exist differences between the methods of investigation adopted by the chief authorities, and that it would be, therefore, desirable first to reach unanimity upon this fundamental question of method.

Happily such an opinion would be erroneous, and we can have no doubt as to the way to follow in our investigations, as there is no such divergence. The method which has hitherto yielded the results does not differ from those used in the natural sciences. Starting from the observation of facts, it culminates in the explanation of these facts. The correctness of our explanations must be subjected to the control of further observations. Precisely the same course is followed in experimental research, with the exception that, in our present subject, the experiments are not instituted at the will of the investigator, but are

[<sup>1</sup> This paper has been translated by me from the German manuscript; but our limits required that the original should be condensed. The whole task was by no means an easy one; the careful revision bestowed by Professor Lichtheim on the proofs, and for which I beg to return him my best thanks, has, however, materially relieved me from the responsibility I had assumed.—A. DE W.]

supplied to him by nature, and that he thus depends for them upon a happy chance. The erection of the building can therefore proceed at a slow pace only, and must rise by degrees as the result of many toilers' work.

Nor do we meet with any divergence of opinion as to the end to be attained. Our task is to determine the connections and localisation of the paths of innervation subservient to language and its correlated functions. On the supposition of our having reached this end, we should then be able to determine the exact place of any solution of continuity in these paths, and account for its symptomatic manifestations with the same precision as we do for those of a motor or sensory paralysis depending on a lesion of the peripheral nerves.

Now, although all are agreed that we have by no means as yet reached this point, opinions differ as to how near we have arrived. We may, however, congratulate ourselves upon the simple fact of there being some agreement as to the fundamental meaning of disturbances of speech from cerebral causes. The amount of superstructure which will be raised on such a foundation must depend, in individual instances, upon the personal temperament of the architect. There is room both for the enthusiast and the sceptic, who both have their function to fulfil in the race for truth. The only necessary condition for the successful building up of the edifice, is that the one should not deny, the other not distort, acquired facts.

It follows from what I have already said, that every step which brings us nearer the fulfilment of the task before us must enable us to differentiate more accurately the clinical forms of aphasic disturbance. What to-day appears to us as a curiosity, as a case aberrant from the ordinary type, will to-morrow be classified as an instance of conformity to the law. This we shall find illustrated in the history of the previous researches into the nature of Aphasia. Broca was led, after many mistakes had been made, to bring into sharp relief aphasia in its narrower sense. Wernicke<sup>1</sup> was the first, to my knowledge, who distinguished between the latter symptom and those due to an interruption in the centripetal afferent paths. Besides these two chief forms, which he calls motor

<sup>1</sup> 'Der Aphasische Symptomencomplex;' Breslau, 1874.

and sensorial aphasia respectively, Wernicke describes a third, and designates it as commissural aphasia (*Leitungsaphasie*). The following discussion, the object of which is to establish a further differentiation, will bear upon this triple division of Wernicke's, and give me the opportunity of mentioning the other categories of aphasia. I abstain from any further reference to the historical aspect of the question, which has been treated by Kussmaul<sup>1</sup> in a way which leaves nothing to be desired.

The morbid types which I intend to discuss in the following remarks have been determined, in as far as they are new, deductively: it was the task of subsequent clinical observations to test the validity of the inferences. The necessity of differentiating still further the types of aphasia struck me on attempting to schematize the forms hitherto known, for the purposes of instruction. But I did not consider that my schema should be published until cases had been observed which coincided with the new types postulated therein. The important element of my task lies in the observations themselves and in their interpretation. Still I thought it advisable, in presenting my results, to follow the same path which I had myself trodden, giving in the first place the schematic representation from which I started in elaborating my views of aphasia. It will be seen that my conception is intimately connected with previous ones, especially with that of Wernicke: the points of difference will appear in the course of the argument.

The schema is founded upon the phenomena of the acquisition of language by imitation, as observed in the child, and upon the reflex arc which this process presupposes. The child becomes possessed, by this means, of auditory memories of words (auditory word-representations<sup>2</sup>) as well as of motor memories of co-ordinated movements (motor word-representations).<sup>3</sup> We may call "centre of auditory images" and "centre of motor images," respectively,<sup>4</sup> the parts of the brain where these memories are fixed. They are designated in the schema

<sup>1</sup> 'Die Störungen der Sprache;' Ziemssen's 'Cyclopedia,' vol. xiii. Leipzig, 1877.

<sup>2</sup> ['Wortklangbilder;' or auditory word-impressions.]

<sup>3</sup> ['Wortbewegungsbilder;' or kinæsthetic word-impressions.]

<sup>4</sup> ['Klangbildercentrum;' and 'Bewegungsbildercentrum.']

by the letters A and M. The reflex arc consists in an afferent branch  $a$  A, which transmits the acoustic impressions to A; and an efferent branch M  $m$ , which conducts the impulses from M to the organs of speech; and is completed by the commissure binding together A and M.

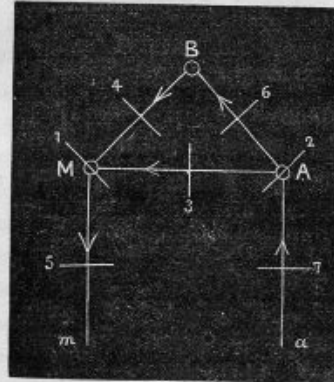


FIG. 1.

When intelligence of the imitated sounds is superimposed, a connection is established between the auditory centre A, and the part where concepts<sup>1</sup> are elaborated, B. All the diagrammatic representations of these phenomena agree so far, at least those given by German authors; beyond this, controverted points are found. The next step in the formation of volitional, or intelligent, speech involves a centrifugal connection between B and M. Our schema introduces, in consonance with most others, a direct commissure, whilst Kussmaul makes it pass from B through A to M. We shall soon return to this point. Leaving aside the point B for the present, we see from the diagram that seven different interruptions may occur in the paths therein assumed. It is manifest at first sight how variously the function of language may be disturbed by some of them; yet it is necessary, in order to gain a clear conception of the various types so produced, that we should include in our survey the disturbances of the functions of language involved in the acts of reading and writing. These are acquired in connection with the

[<sup>1</sup> 'Begriffe.']

exercise of speech, and are hence intimately connected with it; the same nervous paths are, to some extent, brought into play. Reading postulates the existence of visual memories of letters and of groups of letters. We may learn to understand writing through the connection between such visual representations (centre *o*) and auditory representations: by spelling aloud we bring the auditory centre into action, and thus establish a connection, through the path *o A*, between *o* and *B*; in reading aloud, the tract *o A M m* is thrown into activity.

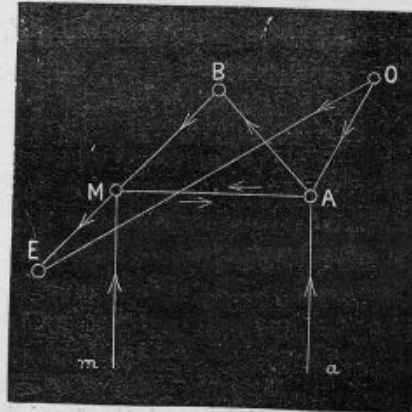


FIG. 2.

The problem with reference to writing is more complicated. The necessary movements have to be learnt, and associated with the visual representations; this is done through the commissure *o E*, designating by *E* the centre from which the organs of writing are innervated.

It is more difficult to determine the path through which volitional, or intelligent writing is executed. This tract must unite *B* with *E*, and clinical facts leave no doubt that it passes through *M*. There may be some doubt as to whether it leads directly hence to *E*, or passes round through *A* on its way thither. I shall return to this question presently; and adopt provisionally the former view, according to which Diagram 2 is constructed.

This figure makes it easy to derive the symptomatic type characteristic of each of the several possible interruptions in

the reflex arc. I have been in the habit of using it for several years past in my lectures, and have found that it greatly facilitates to beginners the mastering of an otherwise very complicated subject. But if the schema is to have any value beyond that of an aid to teaching, it must be shown that the seven derivable types do really correspond to existing forms of aphasia, and that clinical observations are fairly reducible to them. I shall begin my discussion with an *exposé* of the several types.

1. Interruptions in M—the centre of motor representations of words, or motor centre of speech—give rise to the following association of symptoms :

- Loss of (a) volitional speech ;
- (b) repetition of words ;
- (c) reading aloud ;
- (d) volitional writing ;
- (e) writing to dictation.

There still exist :—

- (f) understanding of spoken words ;
- (g) understanding of written words ;
- (h) faculty of copying.

This constitutes the true "aphasia" of Broca, as well as the "motor aphasia" of Wernicke, and the "ataxic aphasia" of Kussmaul. The interpretation usually given of this trouble of speech is the same as that involved in my plan. It is this aphasia which rests upon the firmest basis, and I need not adduce examples from the large number of those on record, such as, for instance, the two celebrated cases of Broca's. I believe I have seen some pure cases of this description, but I have no notes of them, and their investigation was not conducted with the fulness of detail necessary to clear up the only point among the symptoms which still remains to be elucidated. I am referring to the question, whether there is understanding of written language in such cases. According to the diagram, the power of reading aloud should be lost, but that of silent reading preserved intact. I am sure that in uncomplicated instances this must be so, but I much regret that I have not had such a case to observe during the last few years; for the study of the literature of the subject has not

afforded me any positive proof. In the majority of published cases no sufficient attention has been bestowed upon this question. It has indeed often been specified that the faculty of reading was intact: on the other hand, Trousseau<sup>1</sup> has shown that many 'aphasics' appear eager in their reading and yet do not understand what they read. He makes this observation precisely with reference to patients in whom the symptom of deficient understanding of writing was associated with the typical signs of Broca's aphasia. I shall return to this point, and show by personal observations how I think this contradiction may be explained.

2. If the continuity be broken at the point A, in the acoustic word-centre we find loss of:—

- (a) understanding of spoken language;
- (b) understanding of written language;
- (c) faculty of repeating words;
- (d) faculty of writing to dictation;
- (e) faculty of reading aloud.

There is preservation of:

- (f) faculty of writing;
- (g) faculty of copying words;
- (h) faculty of volitional speech.

This type corresponds with the "sensorial aphasia" of Wernicke, who has himself shown that though the faculty of volitional language is not lost, yet there are considerable disturbances in it. The latter is incorrect, inasmuch as wrong words are used, the words themselves are altered by the introduction of wrong syllables, occasionally to such an extent that the language becomes wholly unintelligible. This form has been called also "paraphasia." The explanation given by Wernicke of the fact, that in sensorial aphasia such disturbances occur in spite of the preservation of the tract for voluntary speech, appears at first sight rather forced. He assumes that the nervous influx descending along the path B M *m* sends a branch current to A, and that this *subconscious* innervation of the auditory memories of words secures the correct choice and expression of them; and that irregularities

<sup>1</sup> 'Clinique Médicale,' vol. ii.



occur as soon as the co-operation of these elements ceases to take place.

I accept this interpretation, but with a modification, namely, that the mere excitation of the auditory representation is not sufficient to secure correct speech, but that this representation must enter into relationship with the concept; that therefore the commissure  $A B$  must necessarily be intact for the same purpose. Paraphasia will be observed when in the arc  $B M A B$ , an interruption has occurred in such a way that language is not altogether arrested. The causes of such a modification will be pointed out presently.

It is by no means difficult to ascertain through self-observation, that such an innervation of the auditory word-centre

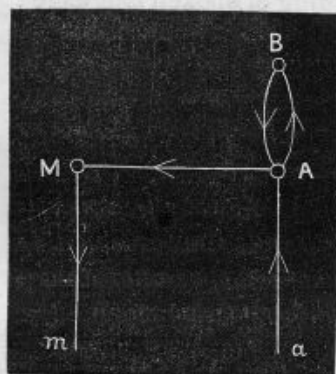


FIG. 3.

does really take place. When speaking aloud, we cannot control the fact, because the words are actually heard, and the innervation of  $A$  from  $a$  being much more powerful, conceals that of  $A$  from  $M$ . But if we perform only the movement of the mouth necessary to the emission of a monosyllable, without issuing it, we shall be most distinctly conscious of the corresponding auditory representation. This observation has perhaps given rise to the assumption of Kussmaul, that the path from the concept- to the movement-centre passes through the auditory centre. This is one of the points on which his schematic representation differs from the one given above. His diagram corresponds to the one given here (Fig. 3), and his path for voluntary language would be  $B A M m$ .

But it seems to me that this view does not correspond to the facts; the disagreement begins already in the case of Broca's aphasia, of which Kussmaul refers the seat, as we do, to the motor centre  $m$ . He assumes that the auditory representations are intact, and may be innervated from the concept-sphere; but this is, I believe, not the case. Trousseau, intending to prove that in aphasia words were forgotten, showed that in numerous cases the "inner speech" had disappeared. I have obtained evidence of this in a patient suffering from a form of the disease allied to Broca's type, and which will be adduced further on. It will generally be found a difficult task to elucidate this point satisfactorily; this is the method I use: I ask the patient to press my hand as often as there are syllables in the word to which an object corresponds. Those who have not lost the auditory representations can do this, even if their intelligence be limited, as I have been able to satisfy myself even under the least favourable circumstances. For instance, a patient who, besides a focal lesion of the right hemisphere, had had a hæmorrhage in the left half of the pons, and suffered, among other pseudo-bulbar symptoms, from complete speechlessness, preserved the faculty of fulfilling the test to the very last.

I have unfortunately, as was just said, not observed recently any pure cases of Broca's aphasia, but I have had a series of mixed cases in which this type predominated at least, and of pure cases of allied forms. I always found that the patients had lost the innervation of the auditory word-representations. These observations correspond so accurately to those of Trousseau, that I feel convinced that in ordinary cases of ataxic aphasia, the path from concept- to sound-centre must be interrupted.<sup>1</sup> Hence the diagram of Kussmaul cannot be correct, nor can a lesion in the path  $B \Delta$  (Fig. 3) explain what occurs, for then the arc  $a \Delta M m$ , and the power of repeating words would remain intact; whereas the latter, in Broca's aphasia, suffers as much as that of volitional language.

These considerations show that our schematic diagram may be considered as accurate, and that the co-operation of the

<sup>1</sup> A rare species of aphasia, included by Kussmaul under the ataxic, and to which we shall allude presently, differs in this particular.

auditory representations in speech is calculated to support the interpretation given by Wernicke of the paraphasic phenomena in his sensorial aphasia. I cannot refer to any typical instance of sensorial aphasia from my own experience; but Wernicke has given one,<sup>1</sup> carefully and minutely described, and in which the symptoms coincided<sup>2</sup> with the deductions from our schema.

3. The third form of aphasia, caused by the interruption of the commissure  $\mathfrak{M} \Lambda$ , has also been described by Wernicke, and corresponds with what we find in the diagram.

There are preserved :

- (a) Understanding of spoken language;
- (b) understanding of written language;
- (c) copying words.

Paraphasia and paraphagia are present; showing, in accordance with what has been said previously, the existence of:

- (d) volitional language; and
- (e) volitional writing.

Similar disturbances are observed in the

- (f) repetition of words;
- (g) reading aloud;
- (h) writing to dictation.

The path generally used in these acts (from  $\Lambda$  to  $\mathfrak{M}$ ) is interrupted; still they are not completely in abeyance, because the tract  $\Lambda \mathfrak{B} \mathfrak{M}$  may be substituted for it. These actions, therefore, are not those of repeating, reading, and writing to dictation, properly so called, but are the result of impulses from the concept-centre; hence they manifest the same disturbances as volitional speech.

Cases of this sort do not appear to be very rare, though I am not acquainted with any observation of the kind as complete as could be wished. This is the more to be regretted, that it is precisely here that I have some doubts as to whether the construction of the path of volitional writing is correct. I have mentioned previously that I hold it as doubtful whether the path  $\mathfrak{B} \mathfrak{M} \mathfrak{E}$  be really used in volitional writing, or whether

<sup>1</sup> Loc. cit. p. 39.

<sup>2</sup> Excepting a difference in the power of volitional writing. This point will be considered in detail further on.

the channel of innervation be not from B through M and A to E (Fig. 4). The circumstance, that in volitional writing we are conscious of the excitation of auditory representations, does not prove that the *direct* innervation current really passes through the centre for the latter. The conditions are the same here as for the relation existing between these word-representations and volitional speech; whilst the chief current flows along B M directly to E, a secondary current may diverge from

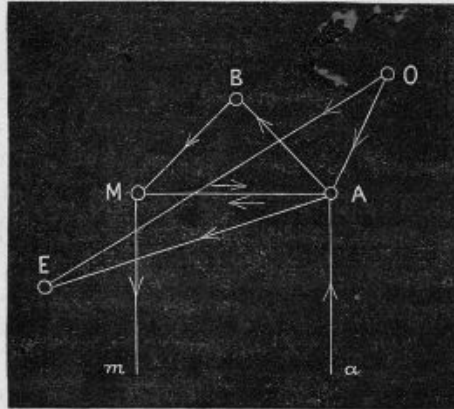


FIG. 4.

M through A to B, in order to secure the correctness of writing, as it does in the case of speaking. Clinical observations are necessary to decide on this point also; and the solution will be found in cases (described under 2) of Wernicke's sensorial aphasia, and of aphasia due to an interruption in the path M A. If localising the lesion of sensorial aphasia in A, we consider Diagram 4, the faculty of writing must have been lost too; whereas paraphasia only will be present, on the assumption that the relation of parts is as in Figure 2.

Though the question, when framed in these terms, appears a simple one, yet it is far from being easy to find an answer to it, because the published material at our disposal scarcely contains any sufficiently precise data to give us certainty on this point. The only case which we may adduce is the one already quoted of Wernicke's;<sup>1</sup> the patient could not

<sup>1</sup> Loc. cit. p. 39.

write: "When sitting at the table to write, she takes the pencil presented to her point upwards, looks at it, turns it round, holds it properly, but makes only up and downwards strokes. When the pen is given to her in the same way, she turns it round, dips it properly into the inkstand, holds it properly, but with no better result." The agraphia persisted throughout, whilst the disturbance in the expression and intelligence of speech had disappeared. This case points to the correctness of Diagram 4; but it is not safe to draw a definite conclusion from a single instance, and unfortunately all other published cases of word-deafness fail us here, as will be seen on reading the thesis of Skwortzoff.<sup>1</sup>

Precisely in the same way the form of aphasia arising from an interruption in the path  $M A$ , ought, according to Diagram 4, to be accompanied with a loss of volitional writing; whilst, according to Diagram 2, paragraphia ought to be present: but we cannot decide with absolute certainty from the observations at our disposal. In both of Wernicke's cases agraphia was present; whilst others, scarcely reported with sufficient accuracy however, leave room for doubts. In a case of my own the clinical aspects clearly corresponded to this type, whilst the post-mortem appearances coincided with Wernicke's description. I relate it here, because such autopsies are rare; but, owing to the insufficiency of the notes, it cannot serve in deciding on the point we are discussing.

<sup>1</sup> 'De la cécité et de la surdité des mots dans l'Aphasie,' Paris, 1881. On page 84 a Case of Broadbent is mentioned which could be opposed to that of Wernicke. It is one apparently of sensorial aphasia, for besides imperfect understanding of language, the speech consisted in a kind of unarticulated jargon. It is said that the writing was not involved. But apart from the fact, that it is doubtful whether this was really volitional writing, and not only that to dictation or to a copy, the whole description is given in such a fragmentary manner, that we cannot tell whether we have to do with true sensorial aphasia, or some variety of it. The case, therefore, cannot be brought as evidence on the subject before us.—Since this paper was in the press, I read a typical case of Wernicke's sensorial aphasia described by Grasset (*Contribution clinique à l'étude des Aphasies*. 'Montpellier médical,' Jan. 1884) and in which also there was complete agraphia. This case too, therefore, points to the correctness of Fig. 4.

CASE I.—*Right hemiplegia ; aphasic disturbances.—Obliteration of the Sylvian artery ; brown softening of the left insula and neighbouring portions of frontal and temporal convolutions.—Pachymeningitis interna.—Valvular heart-disease, &c.*

J. S. B., aged 46, a labourer in Thun, was brought into hospital at Berne on the 3rd of April, 1883. We failed to obtain any clue to the beginning and course of the disease.

*Sensorium* normal. *Speech* much altered. When asked to relate his history, he strings together in a fluent manner numerous words, of which scarcely one now and then can be made out. The following were noted: "Evening, five and twenty, and." Patient is aware of the incorrectness of his diction, and tries to assist himself by gestures. He succeeds better with single short words and answers; thus, in answer to the question, "What was there for supper?" he answered, "Bread, meat, potatoes," with only two mistakes. His own name he mutilates.

*Repeating*.—When he repeats connected sentences he manifests the same defects as in volitional speech; single short words are pretty correctly rendered. No note taken of the way the patient names objects.

*Understanding of Speech* entire.—*Understanding of Writing*, whether printed or hand-written, is preserved. On reading aloud he makes the same mistakes as in talking.

*Writing* very imperfect. He mixes up the order of the letters in a word; and usually stops after an attempt of short duration. He can copy what is set before him with absolute correctness.

*Motility*.—Slight paralysis of the right arm and leg; apparent on simultaneous action of both sides of the body. Paresis in buccal region of right facial.

*Sensation* normal. The tongue deviates to the right, and is the seat of tremors. We pass over the symptoms in the chest and other organs. Symptoms remained unaltered until the beginning of May. Then consciousness became obscured during the next few days, and he died in the night of the 8th to 9th of May.

The autopsy was made, and a full report drawn up, by Professor Langhans. The brain presented the following appearances:

Inflammatory membranous deposits on the inner surface of the dura-mater on the left side. Pia-mater somewhat opaque and whitish, especially over ascending, and apex of temporal convolutions, where it is adherent.

A good deal of serum at the base of the brain. There is a

considerable depression of the convolutions bordering the upper and posterior part of the left Sylvian fissure, as well as of the ascending frontal and parietal convolutions. The Island of Reil is sunken, forming a depression into which the second frontal convolution falls suddenly to a depth of  $1\frac{1}{2}$  ctm.; the third (lowest) frontal convolution terminates into it likewise. Here the pia is inflamed with yellow discoloration. The depression is bounded posteriorly by the fissure of Sylvius. The middle portion of the first temporal convolution is somewhat sunken opposite the depression of the Insula, in which the consistence of the cerebral matter is soft, with harder patches around.

It is difficult to follow the Sylvian artery beyond the point where it reaches the Insula, and presents on the length of  $1\frac{1}{2}$  ctm. a tough whitish appearance; it is occluded just before the division and a short way up the posterior branch.

The softened patch occupies the bottom of the Sylvian fissure and extends to about  $1\frac{1}{2}$  ctm. of the posterior part of the inferior frontal convolution, and to the neighbouring portions of the ascending frontal convolution. A fragment of the cortex of the ascending temporal is also wanting, but there is no yellow discoloration here.

The case just described is undoubtedly one of the third kind of aphasia; the symptoms tally with the theoretical postulates established previously. But the point which to us is most interesting, the capacity of the patient for volitional writing, is not established in the notes, because it is not sufficiently distinguished from writing to dictation.

In the absence of more definite data than those already adduced, whereby to decide which course the impulses follow in volitional writing, it appears to me more probable that Diagram 4 gives the more correct construction. There is one difficulty, however, and it is that in Broca's aphasia, writing to dictation is disturbed, a fact not deducible from Diagram 4, but with which Diagram 2 appears more consonant. On the other hand, as we shall see, in some cases where volitional speech is lost, but not the faculty of repeating words, the first syllables only are repeated correctly; when the patient is to repeat a sentence it is necessary to say it word by word. The explanation of this phenomenon is easy. If four or five words are given together, the patient must retain them in his

memory, and thus what he says ceases to be a repetition, properly so called, but is an effect of volition. This is still more the case with reference to writing, an act which takes a much longer time; but few letters are written really to dictation, and indeed one finds in many cases of Broca's aphasia that the first letter only of a word can be written down, the rest of the word being unreadable.

Further observations cannot fail to throw light on the question. This controverted point does not apply to the remaining forms of aphasia.

4. A variety of motor aphasia is created by interruption of the path B M, of which we have many examples. From the diagram we should expect the loss of

- (a) volitional speech;
- (b) volitional writing;

whilst there are preserved—

- (c) understanding of spoken language;
- (d) understanding of written language;
- (e) the faculty of copying.

So far the symptoms coincide with those of Broca's aphasia. They differ inasmuch as there is preservation of

- (f) faculty of repeating words;
- (g) writing to dictation;
- (h) reading aloud.

As an example of this kind of aphasia, characterised by loss of volitional speech and preservation of power of repeating words, I may mention a case of Hammond.<sup>1</sup>

Still more striking is the faculty of reading aloud. Most recorded cases are incomplete in this respect. I therefore give a case of my own which beautifully illustrates this point.

#### CASE II.—*Traumatic aphasia.*

Dr. C. K., a busy medical practitioner, had a carriage accident, and was carried home unconscious. Three hours afterwards he was bled, when consciousness returned. There were severe bruises on the right side of the body, and of the head, which was ecchymosed. The movements of the right arm and leg were never

<sup>1</sup> 'Diseases of the Nervous System,' 7th ed., chap. vii.



absent, but weakened, whether through paralysis or injury is not certain. Immediately after yencesection the right hand could be stretched firmly, only lifting it was difficult. Sensation normal. Difficulty of swallowing during the first two days; fluids readily got into the air-passages; mouth not easily opened; tongue difficult to pull out. Patient got up after about a week, when it was noticed that he dragged the right leg and swayed a little.

*Speech* was much affected: the first day he only said "yes" or "no," but quite appositely. Gradually, more and more words returned, at first imperfect. Whilst his vocabulary was still very meagre, it was observed that he could repeat everything perfectly. Soon after the accident he began to read with perfect understanding. It was established beyond doubt that he could *read aloud* perfectly at a time when he could scarcely speak at all. The statements of his wife are most positive and trustworthy on this point, though he himself does not remember what took place just after the accident. She states that after much difficulty in making himself understood by gestures, he obtained a newspaper, and to the great astonishment of all present he began to read fluently. She herself thought it most strange and inexplicable.

He could not write voluntarily at all; but this faculty returned slowly and imperfectly, as did speech. On the other hand, he could, soon after he had left his bed, *copy and write to dictation*. He spoke German and French fluently before the injury; but German rather the better of the two. As the aphasia diminished, German words returned before French.

Immediately after the accident the right pupil was dilated and immovable; no troubles of vision. I saw the patient six weeks afterwards, at a time when the loss of speech had to a great extent disappeared. One could observe a slight paresis in the right lower facial region; some weakness of the right leg, but nothing noticeable in the right arm. Sensation was normal, except that he did not so readily recognise objects with his right as with his left hand. The right pupil sluggish; no disturbance of vision nor hemiopia.

In every other respect he is in a normal condition, with the exception of speech. His vocabulary is copious, but he does not talk much, and speaks in a drawling manner. From time to time he misses a word or construction; he then tries to express himself with gestures. Speech much more defective when he must name objects shown to him; then many names escape him, and he also makes mistakes (*e. g.* for "Bild," he says "Milbe," then corrects himself and says "Portrait"; for "Stahlfeder" he says first "Bleifeder," then "Tintenfeder"). Many words are missed in

French also; he finds the French equivalents of the words he can say in German. Patient says himself that the auditory representations of the words he cannot find are missing; he cannot tell the number of syllables in them. If these words are spoken to him or written before him he says them at once, but forgets them immediately. He repeats correctly whole sentences, if not too long.

He understands spoken and written language perfectly. He can read aloud with the greatest fluency, and with scarcely any stoppage. Writing very imperfect. Asked to write the history of his illness, he puts down mutilated, meaningless words, in which it is impossible to discover any sense. Writing single words, names of objects shown, gives a somewhat better result. There is a parallelism here between writing and speech; such names as he cannot say, neither can he write.

On dictation, he writes fluently and without faults. The strokes are somewhat clumsy. He copies equally well. Intelligence normal. His wife assures me that he manages business perfectly, thinks of things in time, &c.

I saw the patient again a month later. Great improvement in his speech, which is fluent, and with almost no hesitation; he names objects with much less difficulty; words are rarely mutilated. Writing is still deficient, though much improved. What he writes is perfectly legible.

5. Still better known than the preceding is the type of aphasia arising in an interruption of the path *M m*. The diagram indicates loss of:—

- (a) Volitional speech;
- (b) repetition of words;
- (c) reading aloud.

Preservation of:—

- (d) Understanding of speech;
- (e) understanding of writing;
- (f) faculty of copying.

Again, and as a distinguishing feature between this and Broca's type, there remain:—

- (g) Faculty of volitional writing;
- (h) of writing to dictation.

There are numerous examples of patients who had lost the faculty of speaking, and could yet make themselves understood by writing. I have seen several cases of this description,

and possess the notes of one in which the patient was under my care several years ago, and wrote his history himself. But it would be useless to relate it here, in presence of the material already at hand.

According to Kussmaul, such cases present the true uncomplicated type of ataxic aphasia, that is, of that form of speechlessness, "in which patients, with freedom of intellect and of movement of the tongue, have the memory of words as acoustic signs, but are unable to emit them. The proof that they really possess these signs is found in the fact, that they can embody them in writing. When asked to articulate them as sounds or words, they cannot, even if they are shown how to shape their tongue and lips."

It is obvious that this description includes the type now before us, and Kussmaul gives a series of most pregnant instances in his chapter on ataxic aphasia. But the aphasia he so designates extends over a wider range of cases, such as those of Broca's aphasia, which we have discussed under our first heading. In these cases there is ataxic agraphia also, owing to the implication of the centre of co-ordination for written signs, which is distinct from, though connected with, that for spoken signs. The close relationship between these centres explains the fact, that the two symptoms so often co-exist.

Kussmaul's views rest on the assumption that, as the motor tract of spoken language is innervated by the left hemisphere only, so is the tract of written language; for it is only under such circumstances that a lesion of the left hemisphere could cause agraphia by an injury to the writing centre. I cannot accept this interpretation, but hold it more probable that the innervation of the movements of writing originates in both cerebral hemispheres.

When we learn how to write, we apparently employ the left hemisphere only; but we must assume that the right hemisphere is the seat of slight nervous action as well, for it can be shown that a certain amount of facility is also imparted for the same movements with the left hand. When the left hand executes "mirror-writing," it performs the same movements as the right hand in ordinary writing, and it is easy to

show that the left hand writes mirror-wise better than it does the usual type. In my own case, even, the strokes are more correct if I write in English characters. Now it could be objected that this innervation of the left hand flows from the left hemisphere; but this is improbable, for in some lesions of the left hemisphere it happens that this tendency of the left hand to execute mirror-writing becomes manifest. Moreover, the fact, that in complete right hemiplegia the power to write with the left hand is not diminished, is opposed to the unilateral innervation of the movements of writing. Therefore the point  $E$ , which in our diagram represents the centre of writing movements, must not be looked upon as single; in addition to the point  $E$  in the left hemisphere, there is a corresponding spot,  $E'$  in the right, to which commissures go, and which can

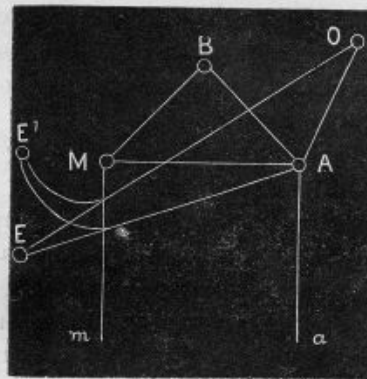


FIG. 5.

come into action when there is some lesion of  $E$ . There are other proofs that in agraphia the motor innervation of the movements of writing is not involved. In pure cases of Broca's aphasia the faculty of copying is preserved,<sup>1</sup> whilst that of reading aloud, and of every kind of repetition, is lost. On the other hand, these patients are not only unable to write, but cannot even compose words with letters placed before them. The real cause of the agraphia does not depend on any lesion of the motor apparatus, but on the impossibility of

<sup>1</sup> See example given by Wernicke, *loc. cit.*, p. 56.

reproducing voluntarily the auditory representations.<sup>1</sup> I have already shown how one can convince oneself that such patients are wanting in the faculty of volitional recall of the auditory representations, and have stated how far I had reached certainty on this point.

Since the above has been written, a case of Pitres<sup>2</sup> has been published, which gives direct evidence of the correctness of my views on agraphia. The patient was suffering from agraphia, the residual sequel of an apoplectic seizure; speech, understanding of language and writing were intact, whilst volitional writing and writing to dictation were impossible, the faculty of copying being preserved. The lesion would be referred by me in the path A E (Fig. 4). The interesting point about the case is, however, that this agraphia affected solely the right hand; the patient could write correctly with the left hand, and copy with his right what he had written with his left hand. This shows the correctness of the diagram, in which the centre E is represented as existing in both hemispheres, and the path A E as being double.

It is obvious that this agraphia has nothing in common with that occurring in Broca's aphasia. As could be expected, the patient could make up words from their component letters, a feat impossible to those suffering from Broca's aphasia.

Pitres adduces two more cases of isolated agraphia. The first, quoted from Ogle, is apparently much more complex. The patient could not even copy, nor make up his name with the letters, and had serious embarrassment of speech. The lesion was probably a more extensive one, and took its origin in an injury received eight years previously. In the second case (from Charcot) the conditions were likewise less simple. We are not told whether the agraphia was bilateral. I may add, that the paths A E and A C may be interrupted at the same time, as they appear (see Fig. 5) to run together in the brain for some distance before they separate to go into the two

<sup>1</sup> We learn to write with the co-operation of auditory representations, hence, we cannot write without it. Every lesion of the path B M A (Fig.) 4 destroys the innervation of the auditory representation from the concept-centre, and must necessarily result in agraphia.

<sup>2</sup> "Considérations sur l'agraphie à propos d'une observation nouvelle d'agraphie."—*Revue de Médecine*, 1884, 11.

hemispheres. Isolated agraphia will therefore, under certain circumstances, appear as bilateral.

The theoretical types with which we have hitherto been engaged corresponded closely with well-known varieties of aphasia. There remain two, due to lesions of the commissures  $A B$  and  $A a$ , which must present themselves as cases of sensorial aphasia. Owing to the rarity of examples of the latter, it would have been but natural had I had no actual examples of it to adduce, though it would obviously have weakened my whole position, the full confirmation of which requires a filling up of every gap. I have, however, found the two missing types, and their entire correspondence with the theory of aphasia here developed is a warrant for the accuracy of the hypothesis.

6. Lesions of the path  $A B$ , would give rise to the following symptoms. Loss of:

- (a) understanding of spoken language;
- (b) understanding of written language.

There are preserved:

- (c) volitional speech.

For the reasons stated above, there will be paraphasia. So far, the symptoms coincide with those of Wernicke's sensorial aphasia. Here, however, there subsist,

- (d) volitional writing,

which will also have the characteristics of paragraphia;

- (e) faculty of repeating words;
- (f) of reading aloud;
- (g) of writing to dictation.

Owing to the interruption of communication between  $A$  and  $B$ , there must be a complete loss of intelligence for what is repeated, read aloud, written to dictation by the patient.

There also subsists the

- (h) power of copying words.

I shall now relate a case of my own which presents all these characteristics.

CASE III.—*Aortic insufficiency. Cerebral softening. Sensorial aphasia.*

J. U. Schwarz was admitted into the Inselspital on the 19th of May, 1884. Sixty years of age—nothing noteworthy in antecedents.

He was well up to the 15th of May. The next night his wife noticed that he became restless, spoke in his sleep, rose and returned to bed, &c. At 4 in the morning it became apparent that something had happened; what he said had no sense. He did not answer to questions; he tried very hard to make himself understood, and the same mutilated expressions were constantly repeated. His wife thought he had lost his reason. His apparent intention was to complain of severe pains in the forehead and nape of neck. This state of things went on, and as he was losing strength, he was taken to the hospital.

Patient is a rather fat, pale, man. Features flabby; expression dull. On admission, he gave the impression of a subject with psychical deficiencies. He talked a good deal, but repeated the same phrases. He points to his head and says, "Oh how stupid I am, I cannot," &c. It is difficult to make anything out of him, for he understands little that is said to him; but it is easy to ascertain that he is in possession of his intelligence, and that the apparent psychical deficiency consists only in his difficulty in understanding others, and in making himself understood. His actions leave no doubt on this point.

*Spoken language.*—The disturbance in his power to understand it is very evident (verbal deafness). If one stands behind him and talks to him, he turns round and asks: "Do you speak to me?" The simplest requests, to show his tongue, shut his eyes, are answered by: "I don't know what one wants."

*Speech.*—There is no deficiency in his vocabulary. He talks a good deal in a flowing manner: he seldom is short for a word; he occasionally uses a wrong or a mutilated one. But this is unfrequent, and in this respect he has greatly improved on what his wife described he was. He is in great difficulty when he has to name objects shown to him; he finds the names with the greatest difficulty, and assists himself with descriptions. Instead of "wine," he says "that is strong;" for "water," "that is weak," &c.

*Repetition.*—He obviously could repeat correctly all that was spoken before him; but he apparently did not understand what he did repeat, as for instance the words, "My name is Peter Schwarz,

and I am already 4 years old," which did not draw any signs of denegation on his part.

*Written Language.*—He understands nothing printed, or hand-written. The simplest things placed before him he cannot read nor decipher. He knows the name of most letters, and gives it correctly; he only confounds the capital I.

*Reading aloud.*—He can make up letters into words, and he can read aloud by spelling; but it is evident that the sense of the word remains closed to him. Out of the sentence, "Will you have a glass of wine?" he goes correctly as far as "you" but has manifestly no idea of the meaning.

*Writing.*—His volitional writing is worse than his speaking—but he does write a few words correctly. In "Meine Frau" (my wife), he manages the first word, not the second.

*Writing to dictation* was not tested at the very beginning. Later it was found that he could do it well if each word was given singly; whole sentences are rendered inaccurately, words being missed.

*Copying.*—He copies perfectly, changing German into English characters, but does not understand anything of what he so writes.

*Motility and Sensibility.*—No difficulty in walking or standing. Movements of arms and legs quite normal; sensation likewise.

*Reflexes.*—Skin reflexes normal. Patellar and Achillis-tendon jerk absent.

*Cerebral nerves.*—1. Smell normal.—2. Nothing noteworthy.—3, 4, 6. Movements of the eye normal. Pupils small, react to light.—5. Normal.—7. No weakness of facial muscles.—8. Hearing not very acute; but present on both sides; he hears the tick of the watch only when close to the ear.—9. Taste appears to be normal. He can recognise and name acid; seems to recognise sweet, but cannot name it. He calls bitter "strong." Deglutition normal. 10. Voice natural.—11. Movements of the tongue free.

In the further evolution of the case, the word-deafness receded without disappearing altogether; on the 21st of May, the improvement was already very marked. On the 22nd the following conversation was held. "Shut your eyes."—He does it. "Take this glass of wine from the table."—He turns round and says: "Are you saying anything to me? I don't understand it." "Do you like red or white wine best?" "Here is some red wine." "Put your right leg out of the bed." "What shall I take?"—and he puts out the left. "Put your right forefinger to your nose."—"Some thing right? What must I do?" He puts the right leg out of bed. "Drink the Professor's health with this wine."—"As you wish. . ." The request is repeated. "Shall I take red wine for me or for you?" "Show your hand."—He does it. "Do you cough



much?"—"Only to-day, as the weather is so bad." "What costs the cheapest wine you buy?"—"I don't know the words any more," &c.

His writing improves also. On the 21st he could write the alphabet easily. German and English characters are used promiscuously. On the 29th he made the first attempt, on being asked, to write a letter.

"*Ich soll*<sup>1</sup> Herrn Professor etwas wissen, *aber was soll ich etwas gheides wissen.* Ich weiss garnichts davon."

He still fails to make out written language; but this does not hold for numbers. He reads the multiplication table, and tells the results, though with a little uncertainty.

At the beginning of June he understands what is said to him much better; his speech is to a slight degree paraphasic. But one notices now that conversation is easier, that he has difficulty in finding the names of objects shown to him, and that he immediately forgets them when they are told. Thus, he was shown a knife and asked "What is this?"—"I have seen something like it, but I can't remember the name." "Is it a knife?"—"Yes, knife, knife!" "What is this?" showing him a cap. He mutters, but cannot find the word. "Is it a coat?"—"No," pointing to his own coat. "Is it a cap?"—"Yes, a cap." "Tell us what you have been eating."—"Eaten something, this morning at 8 o'clock drank something, and again at 12 o'clock, but I can't tell more." "Take your pocket-handkerchief, and wipe your spectacles." Patient does it. "Take my purse, take five francs out of it, and give them to So-and-so." He takes the purse, and five francs out of it, but does not understand the rest.

His understanding of written language is very bad still, though when he reads aloud he makes out the meaning better, a fact which corresponds with his improvement as to spoken language. When he is not allowed to read aloud, he apparently spells inwardly.<sup>2</sup> Even what he copies to dictation is obscure to him. I dictated to him an L. O. U. for 20,000 francs, which he wrote down, and allowed me to put in my pocket without giving the least sign of emotion. Volitional writing is still accompanied with well-marked paraphasia, though there is improvement in this respect. He wrote the following letter to me on the 4th of June:—

<sup>1</sup> [The words in *italics* were written in English characters, the rest in German. The word "wissen" is apparently used for "schreiben." "Gheides" is meaningless.]

<sup>2</sup> When questions were asked, he, at the beginning of his stay in the hospital, used to repeat them, it seemed to facilitate his comprehension; but occasionally did not understand them even after repeating them.

“Juni Mittwoch d. 4ten.—Herr Professor Dr. Lichtheim. Ich möchte heute etwas schreiben. Ich weiss noch sehr wenig. Ich bin Arbergergasse No. 52, mit meiner meiner Lieben um 1 und  $\frac{1}{2}$  Stunde sehr gern gesehen. Ach wie tausend Mal so gern für seine liebes Herz. Ach gott wie gern sollte ich viel mehr wissen und mein liebes Herz müsste mal alle Tag, oft die weiss täglich hundert mal und ich weiss fasst noch nichts mehr was ich zu wissen sollte. O wie viel zu wenig daheim. Ich will am morgen Mittags daheim Arbergergasse zu meine Liebenfrau. Fünf mal heute noch. Ich will auch noch etwas schreiben.—Schwarz-Beer.”

Patient left the hospital on the 6th of June, but came to see us once every week. Every time he showed further improvement. On the 23rd the following notes were taken:

1. *Volitional speech*.—Paraphasia is very slight; word-amnesia, on the other hand, is considerable, though not so apparent in current conversation. He remembers the names of objects a little better; still he is very deficient in this respect.

2. *Volitional writing* is improved. There is less paraphasia.

3. *Understanding of spoken language* is much better. Patient understands almost everything he is told, even the longer sentences. Complicated tasks are executed correctly. Still, beyond a certain point, he still manifests deficiency in this respect; and it is impossible to appeal to his self-observation.

4. He *repeats words* correctly, and in longer sentences. He does it more quickly and intelligibly.

5. *Reading aloud*.—More ready; but he still mainly does it by spelling. He understands better.

6. *Understanding of writing* more deficient than that of speech, though unquestionably improved. He understands short words at once. Longer ones take him an interval, during which he seems to put them together; he used to repeat them, but this being forbidden he appears to spell them inwardly.

7. *Copying and writing to dictation* are carried out properly. He understands much of what he writes. The experiment with the I. O. U. does not succeed any longer.

The last time the tests were applied was on the 25th of July. He still talks somewhat slowly, the arrangement of words is often peculiar, but it takes time to discover that words are wanting in his vocabulary. He is easily stopped by proper names, and finds them with difficulty. The word-amnesia, however, is much less marked.

He seems to understand all that is said; one is occasionally obliged to repeat a question; and he himself repeats, as if with

astonishment, proper names. Much paraphasia still present when he writes of his own accord, though a letter he wrote to me on that day shows a marked improvement. Progress is also noticed in the other respects.

On the 30th of July I was called to see him. About midnight his wife noticed that he suddenly became restless. He tried to get up, and fell. The left arm appeared to be paralysed; but he can give no account of the state of the sensorium. He has been restless since, and talked nonsense.

I found his consciousness slightly affected; he is continually babbling as a delirious fever-patient. He reacts a little when called, but does not recognise people about him.

The eyes are chiefly turned to the right; it is difficult to make him fix them; when called he answers "yes," but turns them more to the right, and does not follow the finger. Pupils rather contracted.

He appears to have headache on the right side, points to it with his hand, and applies a sponge to it. Left arm flaccid and paralysed; left leg apparently sound, yet on rising he bends towards the left side.

Left naso-labial fold somewhat effaced. Sensation and reflexes on the left apparently diminished. No paraphasia noticeable in what he talks. There was constant vomiting; and the patient was taken to another hospital, owing to my removal into other wards, and is still living.

A few remarks on this case, in which the symptoms agree with those postulated by an interruption in the commissure A B, may not be out of place. It may be asked why he spelt the words, though the path O A M m was intact. I think this is explained by the fact, that for fluent reading it is necessary to understand the words; we guess the words before we have actually read them through, and gain much time thereby. When we have to say long words in a foreign language, we also must spell to a certain extent. Our patient shows also a phenomenon which has been observed in other cases of the kind; he repeated the question of which he did not understand the meaning. This condition is closely allied to the *Echolalia* of Romberg; and Spamer,<sup>1</sup> who also deduced the possibility of this symptom from his schema, used this name. In our case it did not appear as if the repetition necessitated the under-

<sup>1</sup> 'Ueber Aphasie und Asymbolie;' 'Archiv für Psychiatrie,' vi. 523.

standing of the words heard, by means of a centripetal path<sup>1</sup> from *M* to *B*. The circumstance that the patient supplied his lost understanding of written language by reading aloud, must be explained differently. The case shows, with many others, that disturbances in the use of written language are much slower to disappear than those connected with speech. Paragraphia here persisted much longer than paraphasia, and the patient understood writing but very imperfectly, whilst verbal deafness had practically passed away.

This may be explained by assuming, that the recovery of the understanding of words is not effected by a new connection between *A* and *B*, but that the sound-representations are deposited at another point *A'* which is connected with *M*, *B*, and *a*. We should thus obtain a state of things as represented in

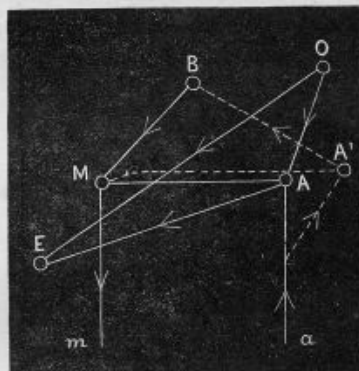


FIG. 6.

Diagram 6, where the destroyed path *A B* is not shown, but where the vicarious connections are depicted by dotted lines. With the assistance of the figure, one easily sees that the patient can easily understand by the ear what he reads aloud; whilst silent reading remains senseless. Silent spelling is already of assistance under these circumstances, because along

<sup>1</sup> I should not have mentioned this possibility, had not a case of Westphal's (cf. Spamer, p. 541) made such an interpretation probable.

<sup>2</sup> The rapidity with which this occurs may seem an objection to the explanation. But the sensorial aphasia of Wernicke through lesions of *A* goes on diminishing likewise, a fact not susceptible of another interpretation.

with the innervation of the movement-representations the words are revived in A' and make understanding possible; The commissure o A' and A' E, require to be used for a much longer time, before the disturbance in the writing can be overcome by their substitution.

The case I have just related is by no means an isolated one, but appears to be of the same category as that of Schmidt,<sup>1</sup> and that of Broadbent<sup>2</sup> mentioned by Skwortzoff as one of word-blindness. In other instances, the observations are not sufficiently full to allow us to decide whether the cases really belong to the same group.

7. The last interruption to be considered, that in the path A a does not really belong to the subject of aphasia, for the faculty of speech remains perfect. Still it is necessary to consider it here, because its symptoms cannot be properly understood except in connection with the present subject.

From the diagram we should conclude that there is a loss of:

- (a) understanding of language ;
- (b) faculty of repeating ;
- (c) faculty of writing to dictation.

The following are preserved intact :

- (d) volitional speech ;
- (e) volitional writing ;
- (f) understanding of writing ;
- (g) reading aloud ;
- (h) copying words.

There is no paraphasia nor paraphagia, because the path B M A B is whole; we could therefore designate such cases as "Isolated speech-deafness," as the incapacity for repetition and writing to dictation may be included under the term "speech deafness." But since there is a form of speech-deafness, in which, as we have seen, the faculty of repeating and writing to dictation is preserved, it is advisable to distinguish sharply these functions.

<sup>1</sup> 'Allg. Zeitschrift für Psychiatrie,' 1871, vol. 27, p. 304; quoted by Kussmaul, p. 176.

<sup>2</sup> "Cerebral Mechanism of Thought and Speech." 'Med. Chir. Transactions, 1872,' vol. lv.; quoted after Skwortzoff, loc. cit., p. 38.

I have a personal observation to record of such a case which has already been published by Burckhardt.<sup>1</sup>

CASE IV.—*Chronic bronchitis. — Arterio-sclerosis. — Two apoplectic seizures; after the first, paraphasia and paragraphia gradually receding; after the second, persistent word-deafness.—Incapacity of repeating and of writing to dictation.—No disturbance of speech.*

Mr. L., aged 55, formerly a teacher and journalist, remained healthy till 1877. He then had an apoplectic fit, which was followed by troubles of language only, with the exception of a slight weakness in the muscles of the mouth on the left side of the face. These are fully described in Burckhardt's observation, to which the reader is referred for particulars. They consisted in paraphasia during volitional speech, repetition of words, and reading; paragraphia in writing to dictation. Volitional writing was very faulty, but not abolished. Nothing is said of the intelligence of language; but from the context and information given by his wife, it appears to have been intact. Evidence as to his understanding written language is wanting.

This association of symptoms may be identified with the one described under 3, as due to a break in M A, and the case is one I had in view when I said that the conditions of voluntary writing agree best with Diagram 2.

The state just described gradually improved, and a slight defect of speech only remained, consisting in the occasional misuse of a word.

In June 1882 he had a sudden attack; the symptoms seem to have been slight. A medical examination made immediately after showed an increase in the facial paralysis; and his wife said the defect of speech was more accentuated; he could not read nor write. We have here a discrepancy between the information given by the wife and that furnished by Burckhardt, who does not mention any disturbance of speech, and says he could read and write. I have mentioned the former statements because they do not agree so well with my views; but as they were made long after the event they do not deserve the same confidence as the notes taken at the time by Burckhardt. He was completely word-deaf.

The disturbance of language rapidly improved, even beyond the state in which he was before the attack. He recovered reading

<sup>1</sup> "Ein Fall von Worttaubheit."—'Correspondenzblatt für Schweizer Aerzte,' 1882, No. 20.

and writing completely; but the word-deafness remained the same. Intelligence free: patient felt weak, and did not like to leave his bed; he suffers from chronic catarrh and asthma.

I saw him first in July 1883; and again in June 1884; on both occasions his condition was the same.

[Passing over the general examination of the body, which presented no features of importance, we come to the functions of language.]

*Understanding of speech.*—Patient gives the impression of an absolutely deaf man, and differs in this respect from other cases of word-deafness. One cannot have the least communication with him except through writing; even his wife cannot make herself understood otherwise, as he can read from the lips but very few words, and these imperfectly.

I should probably have taken him for really deaf had I not been assured that he was acute of hearing, and could perceive all noises, which he said hurt him; and had I not tested the fact myself. Having written for him to raise his hand as soon as he heard the sound of a bell, he did so correctly, even when it was rung most gently behind him. It is the deficient attention he pays to sounds which gives one the impression that he is deaf. Then a few minutes later I rang again, but he did not react to the noise by raising his arm a little, until it was very loud. The same want of attention is manifest when one talks to him. He does not make the efforts to understand observed in patients with word-deafness, but remains indifferent; one has to push or shake him in order to make him attend. He hears when one whistles or sings, but does not recognize melodies. When his children sing in his room quartettes, of which he formerly was fond, he tells them to stop, and that they make too much noise. When I played the national anthem before him, he said, "Once more, I shall perhaps recognize it." But he cannot do so.

*Speech.*—He speaks with absolute accuracy, but with a slight drawl; very rarely, not oftener than would a healthy man, he stops for a word. He finds substantives, even complex ones, and proper names. I showed him a picture, and he said without the least hesitation, "Winterthur." On neither occasion could I detect the least trace of paraphasia.

*Writing.*—He writes fluently and correctly, and composes long articles for journals, of which I have seen several, and possess one.

*Repeating* is impossible to him, even when he is told by writing to do so. Thus when I said, "Ich heisse," he fixed his eyes upon my lips and finally brings out an "Ich," but nothing more.

*Writing to dictation.*—Asked to do so, he said: "But I can't

hear." I nevertheless proceeded to dictate, upon which he remarked: "What a farce to dictate when one does not hear!"

*Copying and reading aloud* he carries out correctly and fluently.

*Intelligence of written language* is intact, as shown by what has already been said. He copies an I O U, written by myself, gives it to his wife, remarking, "You see you have got money."

*Intelligence* perfectly normal. The articles he writes are up to his usual standard and are often published in his newspaper. Advice from his medical attendant informs me that he has remained *in statu quo* up to the present date.

There is in this case only one point which needs discussion here. It is whether the word-deafness produced by the second attack really caused no disturbance of speech. The patient had preserved from his first attack a difficulty with reference to language and writing. If we follow the description of Burckhardt, the second attack brought no change into this condition, and the slight alteration of speech which remained receded completely in the subsequent evolution of the case before I saw the patient. This is in contradiction with the wife's account, who, in answer to my questions, said that the speech had been temporarily worse after the second attack, when writing and reading disappeared for awhile. I have already said that these statements were not trustworthy, having been made three years after the event, whilst the observations of Burckhardt were noted at the time; but I deemed it necessary to mention them precisely, because they seemed to militate against my view. However, even if they were correct, I would adhere to my interpretation, and assume that, in the second attack, the centre  $\Lambda$  of the diagram had been transitorily affected also. Thus Wernicke's type of aphasia, which includes, besides word-deafness, paraphasia, agraphia and alexia, might have come to be observed for a short time. This state of things would have passed away quickly, and word-deafness only persisted, complicated with a gradually receding trace of the disturbance of speech left after the first attack. This interpretation is obviously more probable than the assumption, that the disturbance of speech was a direct symptom of the focal lesion of the second attack, and was transitory only, whilst the verbal deafness remained. The



opposite course is indeed the usual one, verbal deafness disappearing very much more quickly than paraphasia.

In other respects also the case offers peculiarities. I have already adverted to the possibility of looking upon the patient as slightly deaf. The other cases of word-deafness I have seen were different in this respect; one was apt in them to overlook the symptom, the patients answering all questions, but not appositely, and the danger being then to diagnose confusion of ideas. Our patient, on the other hand, paid no attention to the questions, never returned any answer, and thus gave one the impression of being deaf.

Finally, the persistence of this symptom differentiates the present case from the others. Yet these peculiarities can lend only a high degree of probability to this differentiation; a single case does not warrant certainty, and further instances are required before we can decide. The case seems to be an isolated one; I have suspected from the description of others that they belonged to the same category, but the want of accuracy in the observations has not allowed me to reach a definite conclusion.

I think I have so far shown, that each of the seven forms of aphasia postulated by the diagram are found to exist; but there remains the question, as to whether all cases hitherto observed are reducible to these forms. If we examine a large number of the cases on record, it will be found that the majority present deficiencies of observation which allow the test to be applied to them only up to a certain point. In a large number of instances the probability is that they do really belong to one of the forms. But one readily obtains examples in which this is not the case; they seem to differ in one point or another from these morbid types. Do they constitute a serious objection to my theory? I do not think so; most of them can be shown to be reducible to the schema. I must first advert to the fact, that the seven forms hitherto discussed have their origin in *simple* interruptions; whilst there is no doubt that more than one of the paths may be affected simultaneously. A decisive proof of the reality of such combination types is to be found in "total aphasia," in which

there is complete incapacity to speak (the "logoplegia" of the French), with word-deafness. Here there must be a break in the centripetal as well as the centrifugal portion of the arc. We possess several observations to this effect, in which the autopsies have confirmed this assumption. Under the definition of total aphasia just given, this type will include all cases where there is loss both of speech and of understanding of speech; a combination which will arise not only when *M* and *A* are injured, but also when other combined breaks in the two branches of the arc do occur: it will be evident from the diagram that six such combinations are possible, each giving rise to the symptom. It is very doubtful, however, if these theoretical possibilities are all embodied in actual cases; their respective degree of probability can be arrived at only when we know more clearly the anatomical disposition of the nervous paths. We must assume, however, that some other combined lesions occur besides that of *A* and *M*; certain observations point to this conclusion. Thus, for instance, the celebrated case of Lordat, which, though I know only through the fragmentary account of Kussmaul, may be explained on the assumption of a simultaneous break in *M B* and *A B*. It appears that, like our patient Schwarz, he could read spelling-wise, though without understanding what he did read. Aphasia due to lesion in *M B* and *B A*, would be differentiated from that due to lesion of *M* and *A* by the preservation of the faculty of reading aloud, of repeating words, and of writing to dictation; but there is no mention made of these functions in the account just referred to.

[Whilst the present article was going through the press, I had the occasion to read the original account of the case of Lordat given by himself.<sup>1</sup> What I have just said has been confirmed, so far that Lordat could repeat words; but no mention is made of any trouble of writing. Certain statements, however, make it doubtful as to whether he was suffering from aphasia, and not from paraphasia only. He says: "There was not only amnesia, but also what I would call paramnesia, that is, an erroneous use of known and

<sup>1</sup> Grasset, "Contribution clinique à l'étude des aphasies."—'Montpellier Médical,' Janvier 1884.

remembered sounds. Thus when I wanted a book, I asked for a pocket-handkerchief. True that immediately after having pronounced this word, I withdrew it; I felt that another was wanted. Another mode of paramnesia consisted in interverting the letters of the syllables in a compound word, of which I had recovered possession. Thus for 'raisin' I said 'sairin'; for 'musulman,' I felt inclined to say 'sumulman.'"

These data are somewhat opposed to those which precede them, and which leave no doubt that, with the exception of a few words, Lordat was quite speechless. I am therefore disposed to assume, that this paraphasia appeared only later, in the period of regression. The explanation given above would thus be perfectly justified; for I have already shown, that paraphasia and amnesia are observed during the healing stage of aphasic lesions in B M.

This supposition is the more plausible, that the quotation from Lordat shows that the word-deafness had already left him; he was conscious of the mistakes he made in speaking.

If the case of Lordat had been from the very beginning one of paraphasia and not of aphasia, it would agree in the main with that of our patient Schwarz (Case I.); and we should explain it simply on the assumption of a break in the path A B.]

Besides such combinations, we may conceive others due to the simultaneous implication of paths on the same side of the arc. The types thus obtained would coincide, to a great extent, with those arising from single breaks; the one due to interruptions in M A and A a, however, would present a peculiar clinical picture, distinguished from Wernicke's sensorial aphasia, inasmuch as the understanding of writing would be preserved. I have found no case illustrating this form. Wernicke, however, in his explanation of his type of aphasia, expressly specifies that the faculty of reading may be intact, but the reasons he gives for this fact are quite different from mine.

Further examples of combination may arise from the implication of the nerve-paths interested in the production of written language only, A o, A E, and o E (Fig. 4). It is probable that the path o E, at least, is sometimes involved,

for there are cases in which it is expressly stated that the power of copying was affected, as for instance in that of M. Valentin.<sup>1</sup> To judge from the symptoms, this must have been a case of total aphasia caused by a lesion of  $\pi$  and  $\Lambda$ , with a break in  $\circ$  E.

It is not necessary for me to go through all the possible combinations; many of them are very unlikely to occur in reality.

A second, and very important circumstance which must be kept in view when we deal with cases which appear to militate against our views, is that the several components of the various types disappear at very different rates. A large number of "aphasics" fall under our notice at a variable period after the attack has taken place, when some of the symptoms have already begun to disappear. It is easy to show, by examples, how even a few days may be enough to efface some of the characteristic symptoms of the type; this is what happened, for instance, in our third Case.

We must always bear this fact in mind, when in presence of complicated morbid types of unrecent date. Our task is obviously made more difficult, though we must not despair of elucidating the problem; for there is no doubt that there is a law governing the order of retrogression of the various symptoms, at least when they are not obscured by the interference of indirect focal symptoms. These laws are to be derived from a sufficient number of exact observations. To our small stock of knowledge on this point belongs the fact of the usually rapid disappearance of word-deafness, and the greater persistence of the troubles connected with written language. This fact appears to me to throw light upon a not unfrequent type which does not readily fall in with our classification; I mean that of motor aphasia in which alexia<sup>2</sup> co-exists.

A break in the motor tract can never in itself produce a disturbance in the intelligence of writing; and according to the schema there must then be a lesion of the path  $\Lambda$   $\circ$  also. Such a combination, exclusive of the simultaneous implication of  $a$   $\Lambda$   $B$ , is obviously not impossible, but, as we shall presently

<sup>1</sup> 'Revue Médicale de l'Est,' xii. 6. Quoted by Skwartzoff, p. 63.

<sup>2</sup> By "Alexia" is meant here, not only the impossibility of reading aloud, but also the deficient understanding of written signs.

see, all our actual notions concerning the localisation of aphasic phenomena compel us to admit that it must be a purely accidental one. Now the type I am referring to is so common, according to my own experience, and as shown in Trousseau's *Clinique Médicale*, that we can scarcely admit of an accidental occurrence. An explanation must therefore be found for what would otherwise be a serious objection to the theory proposed in this article.

These cases appear to me to have been instances of total aphasia in which the symptom of word-deafness has already been recovered from; I have actually observed this course of events in a case where the initial total aphasia evolved into the type we are now considering. I have already referred to this case, of which I unhappily do not possess the notes. In other instances a careful investigation has led me to discover remains of word-deafness, consisting in a deficiency manifested by the patient when given more complicated orders to carry out, and corresponding exactly to the similar imperfections noted in the later stages of our Case III. Here is an example of the kind.

CASE V.—*Aphasia appearing during typhoid fever. Doubtful traumatic cause.*

S. T., aged 49, a cabinet-maker, was admitted into the Insel hospital on the 24th of January, 1884. It is almost impossible to hold communications with the patient, owing to the disturbance of his faculty of language. All that could be made out of his previous history, was that he fell ill with typhoid during a great epidemic at Meyringen. One day he fell out of bed upon his head, he remained several weeks (?) unconscious, and was speechless when he came to himself again.

Patient is a fairly strong man; his intelligence appears to be perfectly normal; he tries with a certain degree of success to express himself by gestures. He occupies himself in the wards with the general service and with woodwork. He has made with skill several boxes, and plays dominoes and cards very well. The nurse, however, thinks he is deficient in some ways, because he has failed to carry out certain directions.

*Speech.*—He is absolutely speechless, with the exception of a few words such as, "yes," "no," "now," "but," "too," "just;" and the sound "gock," which he substitutes on every possible occasion.

Now and then he comes out with a longer exclamation, such as "Herr Jesu Gott," or "Min Gott im Himmel."<sup>1</sup>

*Repeating.*—He cannot repeat anything said to him except "yes" and "no."

*Intelligence of speech.*—On a superficial examination the patient appears to understand everything. He points to the right objects when they are named, and carries out simple orders perfectly, shows his tongue, closes his eyes, stands on a chair, shuts the stove, &c. But when the directions are more complicated it becomes evident that he does not understand so well. When asked, for instance, to bring a large bottle standing near a basin, he takes the basin. If simple but unusual things are told him, he does not do them correctly. I asked him one day, on coming near his bed, to show me his nose, and he pulled out his tongue. Suggestive questions are rightly understood by the patient.

*Intelligence of writing.*—He formerly read and wrote fluently, but now never takes up a book or newspaper, and manifests on careful examination a great loss in his power to understand written language. He apparently manages a few words; for instance, "soldier," "sheep," "horse," "pigeon." He signifies by gestures that he understands the words "wine," "Albert" (the name of his son); short sentences likewise, such as "eyes shut," "mouth open." But beyond this he is incapable of carrying out plain directions written in large characters, though he points to the letters as if to indicate that he knows them. He has great difficulty, however, in picking them out of a box, and seems to have no idea of their alphabetical order. His difficulty in understanding writing is greater than that in recognising spoken words, though the disturbance is obviously analogous in both cases.

*Writing.*—The patient writes his name correctly, but he cannot write anything else.

To *Dictation* he makes illegible scrawls, except for his own name, which he writes correctly.

*Copying.*—He copies everything, but in a servile manner, and with all the mistakes made in the model.

*Reading aloud* absolutely impossible. He knows the numbers, can write them also, even when with two digits. He often makes mistakes in the multiplication table.

In other respects patient is quite well. No abnormality dis-

<sup>1</sup> We tried to determine whether the word-representations could be excited from the higher centre. Objects were shown to him, but he could not give the number of syllables of their names, nor indicate it by pressure of the hand, according to the plan previously described. He did it correctly if the name was given to him, so that he must have understood what he was told to do.

coverable about the head, all functions depending upon cerebral and spinal innervation intact, excepting a weakness of the right hand, which gives 36 on the dynamometer whilst the left gives 55. Patient indicates by signs that this has been the case since his fall. No such difference exists in the lower extremities.

We have to do here with an apparent case of Broca's aphasia in which the behaviour of the patient led one to suppose that he could not read at all; on closer examination a considerable diminution of the intelligence of written language was shown to exist. The understanding of spoken language appeared at first sight undisturbed, but on investigation defects appeared which were obviously remains of a transitory word-deafness; so that the case must be considered as one of total aphasia.

I have observed another case, quite analogous to this, with reference to the troubles in the functions of language; and I believe, as has been already mentioned, that Trousseau's cases must be interpreted in the same way. The inability of the patients to read was here assumed on the evidence of their not taking to books though their intelligence was good. Though they sometimes thought they could read, they persisted in ever using the same volume or the same page; similar phenomena were observed in my two patients. Trousseau, however, does not adduce any more thorough investigation of their intelligence of writing or speech.

If we take into consideration the facts, that the individual disturbances in the functions of language may occur more or less incomplete or partial<sup>1</sup>; and that more or less considerable remains of them may persist and thus complicate the task of elucidating the type to which a case belongs, it will not appear strange that many instances are difficult or impossible to explain. But this forms no insuperable objection to the theory. On the other hand, our hypothesis would be gravely compromised if a whole category of common aphasic disturbances could not find room within its boundaries; and thus

<sup>1</sup> I abstain from entering here into details about such partial disturbances, so as not to complicate a subject already complex enough. Their study must, however, elucidate many points connected with the mechanism of language. A remarkable start in this direction has been made by Broadbent ('Medico-Chir. Trans.' June 1884).

it becomes necessary for us to consider what place the "amnesic aphasia" of authors must occupy.

The first who distinguished between aphasia from loss of memory of words and aphasia from disordered co-ordination of word-movements, or disturbed conduction of volitional impulses, was James Russel.<sup>1</sup> Most English writers, and in Germany Biermer<sup>2</sup> and Kussmaul, have taken this view. For the first form Ogle<sup>3</sup> preserved the name of amnemonic, for the second that of *ataxic* aphasia; but the word *amnesic* has since displaced the former. Our task is to throw light upon the distinctive clinical symptoms by which these two forms of aphasia can be recognised.

Russel gave as a characteristic test that patients with preserved memory of words, but disturbed conduction of volitional impulses, still make notable efforts at speaking. According to this criterion a great number of cases which are now denoted as of amnesic aphasia would not have been considered as such by him, for the patients try hard to speak, and often possess a goodly vocabulary. Nor can I believe that efforts to talk by aphasics who have but a few words left, justify the conclusion that they have the sound-representations of others at their command. Moreover, I think I have shown that in Case V., which belongs to this category, these representations were missing.

Ogle has described as a pathognomonic sign of his amnemonic aphasia that patients can name nothing, or next to nothing, but can usually repeat words spoken before them. On this assumption, the fourth form described by us as depending upon an interruption of the path B M, would rank as amnesic. But it is certain that this definition does not exhaust all that is understood by amnesic aphasia. Most cases of it must receive another explanation.

Let us consider how, from our point of view, an aphasia due to obliteration of the memory of words is distinguished from one due to disturbance in the motor apparatus. As stated above,

<sup>1</sup> 'British Medical Journal,' 1864. Quoted after Spamer.

<sup>2</sup> 'Correspondenzblatt für Schweizerärzte, I. 1871.

<sup>3</sup> 'Aphasia and Agraphia.'—'St. George's Hospital Reports,' ii. 83-121.—See Virchow's 'Jahresbericht,' 1867, p. 40.



I think that in most motor aphasias—in those, among others, of the true Broca's type—the patient has lost the auditory word-representations, that is to say, cannot awake them voluntarily by an action of their higher centres. The only criterion of the fact is the preservation of the faculty of writing; or else the power of putting together words from letters; or of counting syllables, their number being expressed by signs, or pressure of the hand. These actions are possible to but a small number of the patients, who are described as ataxic aphasics. Kussmaul<sup>1</sup> also considers the power of writing as a sign of the possession of auditory word-images; but as most ataxic aphasics have lost it, he assumed in their case the presence of an ataxic agraphia, that is, of a lesion of the motor-writing tract. I have already shown why I cannot adopt this view; nor can I accept the criterion by which the same author pretends to recognise the faculty of exciting sound-representations in themselves by those ataxic aphasics who have lost the faculty of written language. He says: "The best, but not the only proof of the existence of their faculty is given us by written language. We must assume its existence where aphasics who have never learnt to write, or have lost the faculty of writing, use expressive signs to manifest their thoughts and to answer our questions correctly. Fallacies are obviously dangerous here, and accurate investigations must repeatedly be made in order to ascertain whether the patient actually understands. No signs especially must be made to accompany what we say."

I believe that we must distinguish sharply between the power of resuscitating sound-representations of words voluntarily (i.e. from the concept centres), and the faculty of doing so acoustically (i.e. from the ear), and putting them in communication with the concept-centres. The former involves efferent, or motor, actions; the latter afferent, or sensorial, effects; involving the paths of the diagram B M A and  $a$  A B respectively. The test of Kussmaul has reference only to the understanding of words (i.e. to the state of the path  $a$  A B); and which may be lost without destruction of the auditory word-representations, as is shown by Case IV., where we find absolute word-deafness with no disturbance of writing. I have also

<sup>1</sup> Loc. cit. pp. 157 and 158.

found, that though a patient understood speech perfectly, he might have lost every power of voluntarily bringing back the word-sounds (Case II.). In most ataxic aphasias, therefore, I consider that there is, besides the impossibility of expressing any words, a loss of word-memory, that is of the power of sound-revivifications.<sup>1</sup> If we turn to Ogle's amnemonic aphasia, where the faculty of repeating is possible, we find that the power of speech proper is gone.

Loss of voluntary innervation of the auditory word-centre, but not of the motor word-centre, would occur in cases of lesion of the centre  $\Lambda$  itself, or of the commissure  $\Lambda M$ . The corresponding types (sensorial and commissural aphasias of Wernicke) described under 2 and 3, do manifest the disturbance of language which has been called amnesia or amnesic aphasia. This trouble we have, like Wernicke, associated with a lesion of the auditory word-representations; and, as the reader will remember, we find in recent cases that the vocabulary is rich (as one would expect when the path  $B M m$  is intact), but that speech is incorrect, the words used being mutilated or inverted. This symptom we name, after Kussmaul, paraphasia; when it is disappearing, or is but slightly developed, there occur evidences of amnesia, that is more easy to demonstrate when the patient is made to name objects than when he is engaged in ordinary talk; names which occur without effort in fluent speaking, arrest him when he has to find them for objects or persons shown to him. If the path  $M A$  be interrupted whilst  $\Lambda$  remains intact, the auditory word-representations may be called up through the auditory, as well as through the optic nerve; and it is enough to say or show the word to restore it, either for an instant or for a longer period.

These phenomena may be brought into harmony with our schematic representation, by assuming, that for fluent talking the innervation of the auditory word-centre is not necessary, the latter being connected to the path  $B M m$  only.

The process of finding the name of persons and objects is different. It is easy to convince ourselves by self-observation

<sup>1</sup> Ogle admits that in ataxic aphasia, the memory for words may fail, as well as the power to express them.

that we first seek the auditory representation of the word, from which starts the excitation for the mechanism of articulation; we are not conscious of such a process in fluent speaking. Experience further shows that the same word-amnesia occurs in the type which we have seen to depend upon a break in the path A B (see Case III.). In explanation we must assume that the nervous current, starting from the auditory representation in A, must pass through B to reach M.

The reader may find this very improbable and too complicated. But it would appear as if, in naming objects, the auditory representations once found had to react in consciousness. This variety of language is a much more "conscious" one than fluent speaking, in which we are aware of the sense of what we are saying, rather than of every word we say. Under certain circumstances conversational language is carried on in a similar way to naming, as, for instance, when we use an idiom not quite familiar to us. Here we must seek the words by the complicated process just mentioned; the direct communication between concept and motor centre without co-operation of sound-representation does not exist; the sub-conscious act of speaking is not yet possible. A greater psychological exertion is obviously required, and consequently more fatigue is entailed.

It seems to me questionable, however, to place amnesia on a par with the other phenomena of aphasic disturbance. At any rate, it is a pretty constant accompaniment of the types above mentioned, and of the regressive forms of motor aphasia which pass through a stage where amnesia is observed, as has been shown in the second of the cases we have adduced and in other analogous ones. But it is not a sign of a focal lesion, and appears in more diffuse morbid processes, or where the cerebral circulation is deficient, or as an early sign of the natural involution of the brain.<sup>1</sup> Its occurrence may be explained on

<sup>1</sup> The various forms of amnesia, the senile as well as those observed in aphasic disturbances, do not offer essential differences; they always involve proper names chiefly, then substantives; very rarely verbs, adjectives and pronouns. Kussmaul explains this fact thus: "The more concrete the idea, the more readily the word which expresses it escapes the memory when it is failing. The reason of it is that the representations of persons and things are more loosely connected with their names than the abstractions of their conditions, relations

the assumption, that the resistance offered by the complicated nervous path can be overcome only when the latter is in a normal condition, and when the functions of the cerebrum are fully carried out. Thus amnesia may be found as a residuum after lesions in B M A B, and appear as a symptom of diminished cerebral activity. At any rate, it cannot be accepted as a basis for the classification of aphasias, and the word "amnesic" aphasia had better be abandoned. The expression "ataxic aphasia" is equally unsatisfactory; we have not to do with disturbed co-ordination of word-movements.<sup>1</sup> The misnomer has probably originated in the fact, that movements other than those of speech can be performed by the patient with his vocal organs; but from our point of view, aphasic speechlessness is more of a *paralytic* nature.

We have thus reached the subject of nomenclature, and should advert to the best way of designating the seven simple forms of aphasia we have described. The best plan is to adhere, as far as possible, to the names already accepted and in use. In a preliminary notice I gave last June, I followed the division of Wernicke, and designated aphasia from breaks in the efferent path B M *m* as *motorial*, and that from break in the afferent B A *a* as *sensorial*.

Further consideration led me to a different result. As long as one thought, with Wernicke, that disturbances in the understanding of language were always associated with troubles of speech, the former would be called "aphasias"; but this is not the case when we deal with morbid types in which speech is perfectly intact, such as that described under No. 7. On the other hand, it is obviously necessary to bring together lesions

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and properties. We represent persons and things to ourselves as easily without as with their names; the sensory representation (Sinnenbild) is more important here than the idea (Sinnbild), the name contributing but little to the knowledge of the person or of the object. Abstract ideas, on the other hand, necessitate the aid of words, which form their sole embodiment. Hence verbs, adjectives, pronouns, and still more adverbs, prepositions and conjunctions, are far more intimately associated with thought. We may imagine that there is a much greater number and combination of excitations of the cerebral cells required for the genesis of an abstract than of a concrete idea; and that there are in consequence far more numerous organic connections between the former and their corresponding word-forming process, than in the case of the latter."

<sup>1</sup> As Sander has already shown. 'Archiv für Psychiatric,' II. p. 40.

in B A *a* under one appellation. Hence it seems to me more correct to accept the nomenclature of Kussmaul, and group together those affections, designating them as *word- or speech-deafness*. The imperfection in the understanding of language is, of course, but a partial symptom in this type; but the same is the case for the speechlessness, or aphasia, symptomatic of a break in B M *m*, and word-deafness bears to the symptoms of a lesion in B A *a*, the same relations as aphasia to those of a lesion in B M *m*. It constitutes the dominating and necessary sign thereof, around which the others are grouped, just as agraphia and the other disturbances centre round the aphasia, in lesions of B M *m*.

We shall therefore do well to reserve the name *aphasia* for the latter class; and, as just mentioned, designate the former by the term *speech-deafness*, or *logokophsis*. Each of these groups fall into three subdivisions: lesions of the centres (M and A in the diagram), might best be said to give rise to *central* aphasia or word-deafness; whilst the others would be distinguished as (*inner and outer*) *commissural* aphasia or word-deafness. The disturbance arising from a lesion in M A could provisionally receive the name of *commissural paraphasia*, until its localisation be fixed with certainty in the Island of Reil, when it would be called *insular aphasia*.

I am well aware of the objections which may be raised against this nomenclature. It would have been more rational not to give the name of a prominent symptom to the leading morbid types, and to find new names for them; but I shrank from an attempt involving the substitution of new names for old and familiar ones, and feared only to create more confusion thereby.

Some readers will object, that the nomenclature rests upon a still hypothetical basis; but, in my opinion, it is of assistance to make it rest upon the assumptions involved in the explanation of the problem. Should this explanation, perchance, prove to be erroneous, no harm will have resulted thereby, and we shall have only to alter the names.

We cannot conclude without having previously considered how far we can proceed with the cerebral *localisations* of the various forms of aphasia. We must obviously start from those

data which may be considered as most certain and best defined. The origin of Broca's aphasia in disease of the inferior frontal convolution will scarcely meet with any doubt, and Wernicke's explanation of his sensorial aphasia by a lesion of the neighbouring temporal convolution has been confirmed by subsequent experience. We may thus assume that the points M and A of our diagram occupy these parts respectively. The path M A has been referred by Wernicke, on anatomical grounds, to the region of the insula; and it is of common occurrence to see lesions of this spot cause disturbances of speech; but we cannot as yet affirm that the latter corresponds to our type (through lesion of A M), though Case II. gives support to this view. There we see the clinical association of symptoms accounted for by the destruction of the insula; but the lesion extended to the neighbouring frontal and temporal convolutions, and no data could be obtained concerning the severity of the apoplectic seizure. It is, however, probable that the trouble of speech was an immediate effect of the lesion, because it persisted unaltered more than a month, till death supervened, whilst the other symptoms underwent a more or less considerable retrogression. A case<sup>1</sup> of Meynert's, though somewhat fragmentary, might be adduced here also.

We shall further have to deal with the course of the inner and outer commissures of M and A. Though in the diagram B is represented as a sort of centre for the elaboration of concepts, this has been done for simplicity's sake; with most writers, I do not consider the function to be localised in one spot of the brain, but rather to result from the combined action of the whole sensorial sphere. Hence the point B should be distributed over many spots; and the commissures M B and A B, would not form two distinct and separate paths, but consist of converging radiations from various parts of the cortex to the points A and M (see Diagram 7, p. 478). This admission does not do away with the possibility of interruptions in the commissures B M, B<sup>1</sup> M, B<sup>2</sup> M, &c.; but leads us to expect, that any simultaneous break in them must occur close to their entrance into the lower centres M and A.

Hence we shall have to look for the lesion which produces

<sup>1</sup> 'Wiener medicinische Jahrbücher,' 1876.

inner-commissural aphasia in the white matter of the hemispheres, namely at the base of the third frontal convolution. The anatomical localisation of inner-commissural word-deafness will likewise have to be sought near the first temporal convolution. We have already had to observe more than once,

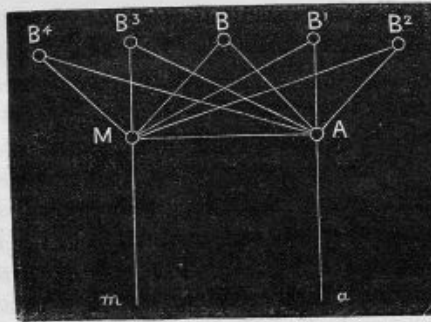


FIG. 7.

that in most published cases, the description of the symptoms is deficient; their morbid anatomy likewise leaves room for many doubts.<sup>1</sup> But though we are still in want of demonstrative evidence, we occasionally find indications in support of our views. Thus in a case of Farge,<sup>2</sup> we find that the patient had right hemiplegia after a severe apoplectic seizure. He could say only a very few words; but could repeat correctly what was said, at least the first words of a sentence. He died, as will be seen in a reference by Nothnagel,<sup>3</sup> twenty days after the fit. The autopsy revealed integrity of Broca's convolution; but in the white matter beneath was found a patch of softening, the size of a small egg.

There is no other case, besides my own, of inner-commissural word-deafness published in sufficient detail to allow

<sup>1</sup> In Hammond's 'Diseases of the Nervous System,' there is a clear case of aphasia, without loss of power to repeat words, quickly followed by death. The case was one of injury, and then were found (1) an ecchymosis the size of  $\frac{1}{2}$  dollar in the right frontal lobe; (2) a tear of the right middle meningeal artery, with large hæmatoma. It is obvious that such an observation cannot be utilised for our purposes.

<sup>2</sup> 'Gazette hebdomadaire, 1865,' 44. See Ku-smul, p. 99.

<sup>3</sup> 'Topische Diagnostik,' p. 358.

of a certain diagnosis. The one by Broadbent<sup>1</sup> furnished an autopsy, but as it came under observation several months after the attack, and as a portion of the symptoms had certainly retrograded by that time, there is some difficulty in identifying the type. There were slight word-deafness and paraphasia present; understanding of writing was highly deficient. The patient could write to dictation, copy and repeat words; he died a month afterwards from another attack, when an extensive hæmorrhage took place in the left hemisphere and broke into the lateral ventricle. This fresh lesion made it difficult to recognise the older one; but it appears that the left temporal convolution bordering the Sylvian fissure was intact. A small hæmorrhage was found in the lowest temporal convolution. The focus, the appearance of which made Broadbent connect it with the symptoms, was in the white matter of the temporal lobe, between the posterior end of the Sylvian fissure and the lateral ventricle near the root of the cornu ammonis. The case proves nothing positively, and all which may be said is that it rather harmonises with our hypothesis.

The difficulties are still greater when we turn to the localisation of the paths *m m* and *A a*. We know the terminal points of the former; the third frontal (Broca's) convolution, and the motor nuclei of the medulla oblongata, which enter into activity during the act of speaking. But we know practically nothing of the actual course of this commissure, whether it is bilateral, or runs exclusively between the left hemisphere and the basal organs. The usual view is that the bilateral movements of the lips, tongue, palate and larynx, which enter into play during speech, may be excited from each hemisphere; but that the connection between this process of innervation, and the intelligent concepts and auditory word-representations necessary to the combination of those movements into articulate speech, occurs on the left side only. It thus comes to pass that only the left third frontal and its efferent fibres are engaged in the movements of speech, whilst the corresponding organs on the right side govern the movements of the organs of speech, but have no share in co-ordinating them into the expression of

<sup>1</sup> "Cerebral Mechanism of Thought and Speech," in 'Med. Chir. Trans. ;' quoted after Skwartzoff, p. 88. Virchow's 'Jahresbericht,' 1872, II. 45.



words. The consequence is that a lesion on the left side gives rise to a disturbance of speech without apparent loss of motility, whilst a bilateral lesion is required to produce a paralysis of the motor organs.

Now if the fibres from Broca's centre reached the basal organs down the left side only, the usual persistent aphasic symptoms would arise from lesions of the left peduncle or internal capsule as uniformly as they do from those of the centre itself. But we know that this is only exceptionally the case; hence there must be a partial decussation of the speech-tract from left to right hemisphere within the brain itself, so that the left internal capsule does not contain the whole bundle of those fibres. Diagram 8 is intended to represent this fact, and

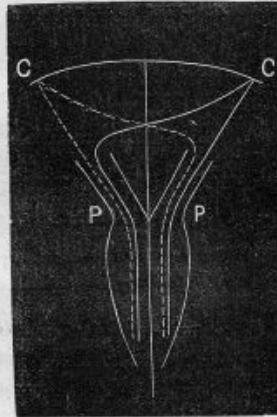


FIG. 8.

shows why the usual lesions of the left internal capsule are not followed with permanent troubles of speech. We see that both hemispheres partake in the function of language,<sup>1</sup> and that bilateral disease is necessary to set up disturbance of speech, combined with deficient movement of the tongue, lips, &c.; such cases, however, are no longer looked upon as instances of

<sup>1</sup> This function is more frequently interfered with by lesions of the deeper portions of the *left* hemisphere, a fact which may be explained on the assumption, that the direct fasciculus is more considerable than the crossed one. The great individual differences observed with reference to this point may rest on corresponding divergences in the relative distribution of fibres in these fasciculi.

aphasia, but have been classified under the name of "Pseudo-bulbar paralysis."

I do not think Wernicke justified when in his last paper<sup>1</sup> he includes under aphasia the speechlessness occurring in the terminal period of progressive bulbar paralysis through total destruction of the motor nuclei. The term aphasia should, I think, be strictly limited to those cases of speechlessness which are unaccompanied with any disturbance of the mechanism of articulation, though I am aware of the difficulty of always accurately defining aphasia from anarthria. When an aphasic is deprived by a fresh right-sided hæmorrhage of the movements of the tongue, lips, &c., we are enabled to recognise the aphasia among the symptoms of pseudo-bulbar paralysis only by our knowledge of how the affection was developed. But occasional difficulties of this sort cannot make me abandon the secure ground afforded by the discrimination between aphasic and anarthric phenomena.

On the assumption that a portion of the fibres decussate, as just suggested, in the hemispheres themselves, an explanation is afforded of all the phenomena which led Wernicke to assume that the right cortex has a share in the production of speech. I hold that no other fact referring to this function is more solidly established than that of the origin of the speech-tract from the left side only. We are not justified in attacking it, as long as we can reconcile with it the data of experience. Now all the facts adduced by Wernicke harmonise perfectly with my view; but whilst his explanation does not account for the regular occurrence of aphasia in disease of Broca's convolution and its non-occurrence in lesions of the left peduncle and internal capsule, these phenomena are the natural consequences of my hypothesis.

Lesions of the deeper portions of the brain, followed by disturbance of speech, moreover, do not bring about aphasia with its characteristics as a "verbal" trouble; the formation of sounds is more or less prominently interfered with. This verbal characteristic of the cortical symptom is a natural

<sup>1</sup> "Ueber die motorische Sprachbahn und das Verhältniss der Aphasie zur Anarthrie."—'Fortschritte der Medicin,' 1884.

consequence of the functions now usually attributed to the cerebral centres in general, and of the innervation of those concerned in language especially. If we assume that Broca's sphere is the seat of acquired motor word-representations (kinæsthetic centre), and that its lesions obliterate these memories, and so give rise to aphasia, it follows that its destruction must affect *words*, and not *sounds*; for those representations acquired during childhood are associated with words only. It is at a much later period of development that we have learnt to decompose words into sounds. As the elements for the formation of words are deposited in the cortical centres of speech, so the dispositions for the production of sounds are made in the bulbar nuclei. The paths, therefore, between Broca's centre and the nuclei will have to undergo a corresponding conversion. The question is now where this conversion takes place; whether low down in the pons, as Wernicke supposes; or, as I think, much higher up, basing my opinion on the fact that disturbances of speech, arising from lesions of the deeper cerebral regions, do not, as just mentioned, partake of a purely "verbal" character.

All these considerations compel us to assume, that only a short extent of the efferent tract from Broca's centre is so constructed as to give rise, on being injured, to real aphasic disturbances. We shall therefore have to look also for the lesion of aphasia without agraphia in the white matter of the hemispheres, though we can as yet scarcely conjecture its localisation. Two new contributions,<sup>1</sup> the results of which, however, are scarcely concordant, point not to a simple radiation of the fibres from the Broca's convolution towards the internal capsule, but to a more complicated arrangement.

The course of the outer commissure *A a* is still more problematical, though one may, in a certain measure, circumscribe the region which it must occupy.

Both ears are capable of receiving and transmitting excitations of speech, but intelligence of language is bound up with

<sup>1</sup> Wernicke, 'Fortschritte der Medicin,' 1884. No. 1. Bitot, "Du siège et de la direction des irradiations capsulaires chargées de transmettre la parole." — 'Archives de Neurologie,' 1884, 22, &c.

the activity of the left hemisphere only; so both acoustic nerves must enter into relation with the latter.<sup>1</sup>

The affection due to a lesion in  $\Lambda a$ , characterised by word-deafness, can obviously come into existence only if the irradiations of both acoustic nerves in the left temporal region are broken through.

With reference to the point where the auditory tracts combine to form the path  $\Lambda a$ , we might place it low down in the bulb or cerebellum, and assume that the fibres ascend already, distributed as they are in the cerebrum itself. Such a view would be opposed to the clinical evidence we possess; there are numerous cases of hemianæsthesia through lesion of the posterior part of the internal capsule, in which a diminution of hearing of the affected side existed. Hence the union of the two tracts does not seem to be effected below this point. A unique observation by Vetter<sup>2</sup> of complete deafness of one ear with hemianæsthesia on the same side, of cerebral origin, lends further support to this view. The union, therefore, must take place in the cerebral white matter, and we shall have to look for the localisation of the path  $\Lambda a$  and its lesions in this region, and probably in the temporal lobe.

It is obvious that lesions of the cerebral white matter may not always give rise to the associations of symptoms we have described. Apart from indirect lesions of the cortical centres, complex interruptions in the commissures  $M B$  and  $M m$  on the one hand,  $\Lambda B$  and  $\Lambda a$  on the other, will give rise to clinical types corresponding exactly to those of Broca and Wernicke.

I have still to advert to the phenomena of *word-blindness*. As we have already seen, this symptom arises in common with others from breaks in different tracts subservient to the function of language. If we were to give to it the same extension as does Skwortzoff, for instance, we obviously could not speak of its being uniformly localised. This condition might rather be

<sup>1</sup> Since a similar distribution must obtain in the right hemisphere, crossed deafness would not be produced in man by destruction of one auditory sphere (as Munk says is the case for dogs). In fact there is no record of observations showing left deafness to be due to a lesion of the right hemisphere.

<sup>2</sup> "Ueber die sensorischen Funktionen des Grosshirns;" "Deutsches Archiv für klin. Medicin," xxxii. p. 498.

fulfilled in cases of isolated word-blindness. Here we have not to do with a simple obliteration of the visual representations of the written signs, with a lesion of the point  $O$  in the diagram. These patients can copy, hence the path  $O E$  must be intact; and from what we know of the cortical distribution of the optic tract in man, it is certain that the visual memories of words must be stored up in bilateral spheres. Isolated word-blindness is, as has been said by other observers, the result of a break between the visual and the auditory centre of word-representations; that is to say, of a break in the path  $O A$  after the union of the converging tracts from both occipital lobes. The observations bearing on the point are few, but they support this conclusion; the anatomical substratum of word-blindness has been found to be a focal lesion in the neighbourhood of the left inferior temporal lobe.

Though I have ventured the above remarks concerning the probable localisations of aphasic disturbances, I am well aware of the restricted foundations on which we may safely build, and what a space theoretical reasoning has still to occupy in the discussion. If I have, nevertheless, not kept my views to myself, it was on the principle, that we must not recoil from the consequences deducible from our hypotheses. In proportion as we draw these conclusions, we shall obtain the necessary data whereby to correct, or if need be abandon, them. Again, no simple observer can collect material enough to accomplish this task, which requires the co-operation of many; and under these conditions even erroneous assumptions may prove of advantage in the search for truth.