# The Lumleian Lectures

ON

# SOME PROBLEMS IN CONNEXION WITH APHASIA AND OTHER SPEECH DEFECTS.

*Delivered before the Royal College of Physicians of London, on April 1st, 6th, and 8th, 1897,*

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## LECTURE II.[1]

*Delivered on April 6th, 1897.*

MR. PRESIDENT AND GENTLEMEN,—After the full discussion of physiological first principles in the last lecture, I now propose to limit myself to a consideration of certain parts only of the very extensive subject of speech defects, and I have selected those parts which are of most general interest and that have hitherto given rise to most discussion. I shall not deal at all with defects dependent upon lesions beneath the cortex—that is, neither with anarthria nor with aphemia; nor shall I consider, except incidentally, the effects of lesions of the commissures between the different word-centres. I shall confine my attention to the effects of lesions of the four word-centres themselves, both separately and in combination. And when I say lesions I mean for the most part gross lesions, for time would not permit of my dealing with the many varieties of aphasia due to transitory lesions or functional disabilities in Broca's region. And even when thus narrowing the subjects to be dealt with in this and the next lecture, I find myself over-burdened with matter—partly because of discordant views that must be discussed, and partly because in the present state of knowledge and opinion concerning these speech defects it would be impossible for me to attempt to substantiate my own opinions without quoting a number of cases the full details of which the time at my disposal will not permit me to read.

### STRUCTURAL DEFECTS IN THE GLOSSO-KINÆSTHETIC AND CHEIRO-KINÆSTHETIC CENTRES.

These kinæsthetic centres are, as I have already observed, concerned more with the expression of thought than with the thinking process. Their activity is in the main roused as thought is about to translate itself into action. These centres form the last outposts on the side of ingoing currents, and constitute at the same time the starting points for outgoing currents. They are situated at what psychologists have spoken of as "the band of the stream." Although with lesions limited to these regions the power of thinking may not be very greatly interfered with, still it is nearly always interfered with to some extent, so that patients having such lesions do not usually exhibit anything like the same amount of mental clearness as that shown by patients suffering from aphemia. It is true that the latter very frequently preserve their power of communicating their thoughts by writing, whilst aphasic patients do so only rarely. Any mental disability in the latter would therefore tend to appear greater than it really is. But this only very partially explains the apparent difference in mental power that is commonly met with. The fact that speech and writing are so frequently involved together in typical cases of aphasia is due partly to the proximity of the glosso- and the cheiro-kinæsthetic centres, and, as we shall subsequently see, perhaps not less to the proximity of the two sets of commissural fibres connecting these centres with the auditory and the visual word-centres respectively. Damage to these commissures may, in fact, be a cause of typical cases of aphasia and of agraphia, as I shall subsequently endeavour to show.

These modes of accounting for the co-existence of agraphia with aphasia undoubtedly hold good for certain cases, and especially for those in which, while there is no paralysis of the right hand and arm, the agraphia is complete—copying (except laboriously and slowly) being no more possible than writing spontaneously or from dictation. The destruction either of the cheiro-kinæsthetic centre or of the commissure connecting it with the visual word-centre ought to produce such decided results as this. On the other hand, if no other disturbing cause were in operation, there is no reason why such a patient should not be able to write well with separate wooden letters (*écriture typographique*) either spontaneously or from dictation. And in cases where such a patient is paralysed in the right hand there would be no reason why he should not, with practice, become able to write in all modes with his left hand.

Whether the combination of aphasia and agraphia is mostly due, as I think, to this simultaneous implication or functional derangement of the cheiro-kinæsthetic centre and the commissure uniting it with the visual word-centre or to some other cause must for the present be regarded as a moot point. Formerly a different view was taken. Thus some of the earlier observers of aphasic cases, such as Trousseau, Hughlings Jackson, and Gairdner, seemed to consider that the inability to write was as much a result of a lesion in Broca's centre as inability to speak, and contended, indeed, that there was almost always a parity between these defects. Trousseau,[2] for instance, says: "The inability to write is proportionate to the inability to speak"; and again: "Aphasics write as badly as they speak, and those who do not speak at all are absolutely incapable of writing." Gairdner expresses himself in almost similar terms. Hughlings Jackson is even more decided in his opinion. Thus he says[3]: "If a patient does not talk because his brain is diseased, he cannot write (express himself in writing). ..... I submit that the facts that the patients do not talk, and do write and do swallow, are enough to show that there is no disease at all."

One of the first to dwell upon the marked inequality that may be met with between these two disabilities was W. Ogle,[4] to whom we owe the introduction of the term "agraphia." More than ten years previously, however, Marcé[5] had insisted upon the independence of these two defects. Thus, speaking of inability to write, he said: "It is independent of the faculty of expression [by speech], since twice the patients could write freely when they were quite unable to speak. It is independent of the motility of the hand, since even when the hand has preserved all its power writing has been found to be impossible."

The dependence of ability to write upon the integrity of a cortical centre altogether distinct from that by which articulate speech is effected is, I think, practically certain, but this is but another aspect of the question previously put, and will have to be considered more fully a little later. For the present it is assumed that there are two such separate but contiguous centres; and if this be true, that they are frequently affected simultaneously (either structurally or functionally) in cases of aphasia may be fully recognised. Still in some cases either the glosso-kinæsthetic or the cheiro-kinæsthetic centre seems to be in the main involved, so that in one case speech may be principally interfered with, while in another it is more especially writing that is made difficult or impossible. This latter kind of defect is much less frequent, and usually less noticeable, than the former, because the patient is also often more or less paralysed in the right hand and arm. In such cases attempts at writing would only be possible, if at all, where the left hand and the right side of the brain have been more or less educated, and frequently no serious attempts have been made in this direction.

Though aphasic patients are unable to give voluntary and preconsidered expression to their thoughts; words, or even short phrases and oaths, may occasionally be uttered under the influence of strong emotion. We often find these patients able to make use of short familiar words like "yes" or "no" in response to questions addressed to them, though they may be often inappropriately employed. The articulation of such words, or "recurring utterances" as they are now commonly termed, is generally supposed to be brought about through the intervention of the comparatively uneducated right third frontal convolution, and this subject will be referred to more fully later. As Hughlings Jackson originally pointed out, such a patient is quite unable to repeat one of these words which he is continually bringing out, or, indeed, any other simple vowel sound, when he is asked to do so. He cannot utter it, that is, in a purely voluntary manner, in response to a request or command. Another interesting

[1] Lecture I. was published in THE LANCET of April 3rd, 1897.

[2] Lectures (Translation by Bazire), 1866, p. 261

[3] Brain, vol. i., 1878, pp. 320 and 329.

[4] St. George's Hospital Reports, vol. ii., 1867.

[5] Mémoires de la Société de Biologie, 1856.

peculiarity is also often seen when resident foreigners become aphasic. During recovery it is found that they are at first only able to express themselves in that language in which they are most thoroughly versed—namely, in their own native tongue. I have seen this in several patients. Two were Germans who had been long resident in this country; yet after an attack of right hemiplegia and aphasia each of them was for a long time unable to utter a word of English. When they began to speak they used German words only; and after they had further recovered, if occasionally in want of a word while speaking English, it was always a German equivalent that first presented itself.

There are many instances on record in which, though the aphasic condition itself has been complete and associated with more or less agraphia, the mental powers of the patients have been fairly well preserved. Many of such individuals have been able to read intelligently to themselves, and play games, like draughts or cribbage, perhaps better than their neighbours.

The powers of articulation and of speech remaining to aphasic patients are very various, but may generally be ranged under one or other of the following heads: (*a*) Cannot utter a sound, or only mutters inarticulately; (*b*) constantly repeats some meaningless sound or sounds such as "tan-tan" or "cousisi"; (*c*) uses some one or two single words such as "yes" and "no,"; and also mere sounds such as "ba-ba" or "poi-boi-ba"; (*d*) uses some short phrase habitually, such as "list complete," or "I want protection"; and (*e*) uses three or four words or expressions, though in themselves meaningless or irrelevant, in a constant and definite manner, as in Broca's case (Lelong), in which the words "oui," "non," "trois," and "toujours" were so employed.

An examination of the recorded cases in which aphasia has been produced by lesions more or less completely localised to Broca's region, or to it and the foot of the second frontal convolution (the supposed seat of the centre for writing), by no means suffices, as some suppose to establish the view that agraphia is to be regarded as one of the effects of a lesion in the foot of the left third frontal gyrus. It will rather be seen, on the whole, to lend considerable support to the opposite notion that co-existing agraphia in such cases is to be regarded as a result of a coincident damage to, or functional perturbation in, the foot of the contiguous second frontal convolution. The evidence afforded by such cases, however, is much less conclusive than it should be, owing to the fact that in so few of them have careful observations been made as to the ability of the patient to write some days after the occurrence of the lesion, when the mere perturbation of brain functions that it may have caused has had time to subside.

Of the first two cases of aphasia that were recorded by Broca,[6] in one the lesion was old and widespread, though the left third frontal convolution was one of the parts that were destroyed; in the other the lesion was limited to the posterior parts of the left third and second frontal convolutions, and there was as a consequence loss of speech and writing, without any paralysis of limbs. This was, therefore, altogether the most memorable case, since by means of it more especially the first attempt was made to limit the situation of the region in the left frontal lobe damage to which gave rise to aphasia. The following is an abstract of this classical case:—

CASE 1.—Lelong, aged eighty-four years, was admitted into the surgical ward of the Bicêtre, Oct. 27, 1861, under the care of Broca In the month of April, 1830, he had had a slight apoplectic attack, from which in a few days he was convalescent, no paralysis of limbs having been observed, though his speech was lost except for four or five words. His intelligence seemed unimpaired. He understood all that was said to him; and apart from his very scanty vocabulary he made use of expressive gestures. He remained in much the same condition for eighteen months, that is up to the time of his coming under Broca's care. To his questions he only replied by signs, accompanied by one or two syllables pronounced hastily and with visible effort. Broca says:—"These syllables had a meaning. They were French words, viz., *oui*, *non*, *tois* (for *trois*), and *toujours*. He had a fifth word, which he pronounced when one asked his name. He then replied, 'Lelo' for Lelong, his real name. . . . The first three words of his vocabulary corresponded each to a definite idea. In affirming or approving he said 'oui.' In expressing the opposite idea he said 'non.' The word 'tois' expressed all numbers, all numerical ideas. And lastly, whenever these three words were not applicable Lelong helped himself with the word 'toujours,' which in consequence had no definite meaning. I asked him if he knew how to write 'oui'; if he could do it. 'Non.' 'Try.' He tried, but he could not succeed in directing the pen." Broca gives details of his conversations with Lelong in illustration of these points. His sight and hearing were good. The tongue was protruded straight and was moveable in all directions. There was no paralysis of the limbs. This patient died twelve days after his admission, and at the necropsy the remains of an old hæmorrhage was found in the form of a small cavity filled with serum, and having its walls infiltrated with altered blood pigment, occupying the posterior third of the second and third frontal convolutions. This was evidently the lesion which occurred when he suddenly lost his speech about twelve months previously, as other parts of the brain showed no focal lesion of any kind.

Tamburini and Marchi[7] have also recorded a case of destruction of the second and third left frontal convolutions which was associated during life with aphasia and agraphia.

Another case has been recorded by Simon[8] in which there was a traumatism affecting these same convolutions.

CASE 2.—A perfectly healthy man fell from his horse. He got up at once, took hold of the bridle, and was about to vault into the saddle, when a physician, who by chance accompanied him, approached and examined him. He was unable to speak, but made himself understood by signs. No paralysis whatever was present. On the head there was a small wound with depression of the bone. Later, when death ensued from meningitis purulenta and inflammatory softening of the brain, a bit of bone that had been broken off was found in the left third frontal convolution which, together with the second and the island, was softened. In the skull there was only a perfectly round hole, no crack or fracture in any part.

Nothing is said here as to the presence of agraphia, but it would probably have manifested itself as soon as the inflammatory softening caused by the original traumatism had extended so as to involve the posterior part of the second frontal convolution. Probably the condition of the patient, however, was too serious to permit of this being tested.

A case has been recorded by Rosenstein[9] in which it is said that a hæmorrhagic softening involving the third frontal convolution only gave rise to both aphasia and agraphia.

CASE 3.—A woman, aged twenty-two years, was admitted into hospital suffering from a fever of intermittent type, and from nephritis. She was ill for several months, and whilst recovering from this affection she was suddenly seized with severe cerebral symptoms. The countenance was pale, the expression staring, the pupils dilated. She understood what was said to her, and put out the tongue when asked. She took a drinking-glass when it was offered to her. She tried to answer, but in reply to questions could only say "Ja, Ja." She nodded approvingly when one guessed what her wishes were. She had formerly been able to write well, but now when requested to do so she only made all sorts of scrawls, but no letters. She lived sixteen days longer without the aphasia disappearing, and then died from a thoracic complication. At the necropsy a clot the size of a hazel-nut was found in the third frontal convolution, surrounded by a limited area of secondary softening. The precise site of the clot in the third frontal convolution is not stated.

In regard to this case it may be fairly said that the sudden irruption of a clot of blood of the size of a hazel-nut into the third frontal convolution may well have inhibited for a time the functions of the immediately adjacent portion of the second frontal convolution, and it is also by no means clear that the secondary softening may not have slightly extended into this part.[10] It will be of interest here, also, to quote a case of combined aphasia and agraphia in association with right hemiplegia, and therefore presumably the result of a much larger lesion. It is one of the cases recorded by Trousseau,[11] but in which there was no necropsy. The patient was, as is very often the case in true agraphia, able to write his own name, though nothing else. Trousseau says:—

CASE 4.—"In August, 1863, a lady came to consult me with her son, aged twenty-five years. Four years previously this young man had for several days complained of headache, when he suddenly called out to his mother one morning, 'Oh! I feel something extraordinary inside me.' These were the last words he spoke. His right arm and leg became numb, and after a few hours the hemiplegia was complete. After a short period he regained some power of moving first his leg and then his arm; but when he came to me he still walked with difficulty, and could only use his hand for very rough purposes. The aphasia, however, which had from the first day been complete, had not diminished. He could articulate two words only—'No' and 'Mamma.' 'What's your name?' 'Mamma.' 'What's your age?' 'Mamma, No.' He yet knew that he did not answer as he ought. He had taught himself to write with the left hand, but had not got beyond signing his own name, Henri Guénier. He wrote it very legibly on a piece of paper which I gave him. 'Since you write your name,' I then told him, 'say Guénier.' He made an effort, and said 'Mamma.' 'Say Henri.' He replied, 'No, Mamma.' 'Well, write Mamma.' He wrote 'Guénier.' 'Write No.' He wrote again 'Guénier.' However much I pressed him I could obtain nothing more. His mother informed me that he could play a pretty good game of cards or dominoes."

[6] Bulletin de la Société Anatomique, 1861.

[7] Rivista Sperimentale di Freniatria et di Medicina Legale, 1883, anno ix., p. 282.

[8] Quoted by Kussmaul, Ziemssen's Cyclopædia, vol. xiv., p. 735.

[9] Berliner Klinische Wochenschrift, 1868, p. 182.

[10] A case of aphasia in a boy five years old, resulting from a traumatic lesion limited to the third frontal convolution, has been recorded by Duval, and is cited by Bateman (On Aphasia, second edition, 1890, p. 196).

[11] Loc. cit., p. 237.

Here we do not know the extent of the lesion. In such a case, however, it would have been immaterial whether the second frontal was involved in the lesion or not, because, as I shall subsequently show, destruction of this centre together with Broca's centre would not prevent a patient learning to write with his left hand, provided the left auditory and visual word-centres were not damaged at the same time. No sufficient details are given by Trousseau to enable us to decide whether the visual word-centre was or was not affected. It certainly may have been, judging from the fact that this young man had been unable to do more than learn to write his own name during the four years that had elapsed since the commencement of his illness—this being generally the extent of the achievement in this direction of patients who are agraphic by reason of a defect in the visual word-centre. Although this case, therefore, may have influenced the opinion of Trousseau, the modern development of knowledge enables us to say that it furnishes no evidence in elucidation of the question whether or not agraphia as well as aphasia is a necessary result of a lesion limited to the left third frontal convolution. I say this with all the more confidence because W. Ogle has recorded a case in which there was right hemiplegia and aphasia, though the patient could write with the left hand, and at his death the second left frontal convolution was found to be intact.[12] The essential details in this case are these :—

CASE 5.—A man, who had suffered from rheumatism and was the subject of aortic and mitral disease, had three days before admission to hospital fallen down, without loss of consciousness, and had found himself hemiplegic and aphasic. On examination, his speech was found to be limited to the two words "yes" and "no." At first he had difficulty in deglutition and in putting out his tongue, but these symptoms passed away in a few days. He understood all that was said to him, and expressed himself well by pantomime. In regard to his writing W. Ogle gives us the following information: "He could write with his left hand with sufficient distinctness words which he could not pronounce when asked to do so. In his writing there was often a tendency to reduplication of letters. For instance, he wrote 'Testatament' for Testament. But I cannot say whether this was more than the result of defective education." The necropsy showed the brain to be healthy, except at two limited spots, of which the chief was the posterior part of the third frontal convolution on the left side. Here was a softened and almost diffluent patch, about three-quarters of an inch in breadth, reaching from the highest part of the third convolution backwards and downwards to the fissure of Sylvius. The softened part was not actually the most posterior part of the convolution, for there was a narrow unsoftened strip between it and the transverse (ascending) frontal convolution. On cutting into the brain a second small patch of softening was seen in the centre of the left hemisphere, external to and rather above the corpus striatum, and extending towards the posterior extremity of the fissure of Sylvius.

**Here, then, where we have the third left frontal affected whilst the second frontal convolution was proved to be healthy, there was aphasia, but no agraphia ; and we may, perhaps, assume that a similar exemption from disease of the second frontal convolution existed in a case of my own in which there was a similar combination of symptoms.**

CASE 6.—An active and intelligent man, a builder by trade, had an apoplectic attack associated with convulsions, which left him paralysed on the right side and quite speechless at first. His mind was also much confused for a time, but after two or three weeks he became able to understand what was said to him. The only articulate sounds he was able to utter for the first nine or ten months were "bi-bi-bi," "poy-boy-ba," and "no." After fifteen months he became able to say both "yes" and "no" appropriately; though whilst he continued under my care I never heard him utter any other articulate sounds than those above cited, and according to the statements of his wife this was the extent of his vocabulary. He had regained almost the complete use of his right leg, though the hand and arm still continued very powerless. He had practised writing with his left hand, and wrote fairly well with it. He was asked to write a letter to me, and the next time I saw him he gave me a note of which the following is a literal copy:—

"Jany. 5, 1869.

"Sir,—I am extremely obliged to you for the trouble you have taken about my affairs. You did not say thether *(sic)* you hav recovered from the severe cold and cough that you told me you were suffering from. I sincerely hope that you have quite recovered your health and strength.

"I am,

"_____."

This note was written, his wife informed me, in less than half an hour, and without any assistance. Yet since his attack this same patient had only been able to utter the two or three words and sounds above mentioned. He seemed to understand everything that was said to him, and appeared also to understand the newspaper equally well when he read it, which he did habitually. When told to count twenty by tapping on the table with the forefinger of the left hand, he did it rapidly and always correctly. . . He could add quite correctly columns containing five figures when the numbers were low, but often got puzzled and made mistakes when the numbers were higher. According to his wife's statement he had been much more irritable than he was before his illness. He had several epileptiform attacks after the one with which his illness commenced, and on each occasion the convulsions were almost limited to the right side of the body.

In each of these cases, even where there has been no agraphia, there has been a more or less distinct lowering of mental power, as compared with that which occurs in the class of cases due to subcortical lesions which I include under the term "complete aphemia." For this there are two causes. First, there is the fact that in these cases of aphasia the brain may have been more considerably and widely damaged by the various structural lesions that have occurred than has been the case in the aphemic group of cases. Secondly, there is the fact that in the cases of aphasia one kind of word-memory is blotted out, whilst in aphemia none of the forms of word-memory are interfered with. Although the kinæsthetic memory of words is, as I have previously said, much less important for the carrying on of thought processes than the revivals which occur in the auditory and in the visual word-centres, still the destruction of the glosso-kinæsthetic centre may interfere with the functional activity of the other two centres. And this interference with the customary completeness of the processes of association concerned with thought and speech might be expected to lead to more or less of mental deterioration.

The degree to which this interference with thought occurs may, however, in an uncomplicated case of disease limited to Broca's region be extremely slight—almost inappreciable in fact—and moreover associated with no impairment in the power of writing. A very remarkable case of this type has been recorded by Guido Banti,[13] which will well repay the most careful attention, because it speaks most authoritatively against the views of Trousseau and the other writers already referred to who have taught that agraphia, as much as aphasia, is a result to be expected from an isolated destruction of Broca's region.

CASE 7.—A right-handed man, aged thirty-six years, who was able to read and write correctly, had a sudden apoplectic attack in 1877. Recovering consciousness in a few minutes he was found to be suffering from right hemiplegia and loss of speech. The paralysis of the limbs disappeared almost completely during the following night, though the inability to speak persisted. The next day he was admitted into hospital, and on most careful examination his condition was found by Guido Banti to be as follows:—"The motility of the limbs on the right side had returned to their normal condition. There was no trace of paralysis of the face or of the tongue. The patient made ineffectual attempts to speak; *he could not articulate a single word*, not even isolated syllables. He was much affected by this mutism, and sought to make himself understood by gestures. I asked him if he knew how to write, and after he had made a gesture in the affirmative I gave him what was necessary and told him to write his name, which he did immediately. I put various other questions to him, to which he replied similarly by writing. I told him to give me a description of his illness, and *he wrote without hesitation* the details above reported. I showed him various objects, pieces of money, &c., telling him to write their names, and he did so without making any mistakes. Then instead of giving him these directions by word of mouth, I wrote them for him in order to thoroughly convince myself that he was able to understand writing. He replied to these questions with perfect correctness. He always wrote very rapidly and did not seem to hesitate to choose his words. He made no mistakes in syntax or orthography. He could understand equally well ordinary writing and print, and when one spoke to him he grasped at once the meaning of the questions, and never wished to have them repeated. I next wrote some most simple words, such as 'pain,' 'vin,' &c., and urged him ineffectually to read them aloud. I then pronounced myself some of the words, directing him to repeat them. He appeared to watch with great attention the movements of my lips whilst I spoke; he made some ineffectual efforts to obey, but he never succeeded in pronouncing a single word." This patient died in February, 1882, from an aneurysm of the aorta ; and a patch of yellow softening was found situated in the posterior third of the third left frontal convolution, and extending for some millimetres only into the white substance.

Here, then, we have a patient with a lesion destroying, but limited to, Broca's region, in whom there was loss of speech but no trace of agraphia, the patient being able to write well with his right hand. It is also of importance to note that there was here no more mental impairment than is found to exist where speechlessness is due to a sub-cortical or a bulbar lesion—that is, in a case of aphemia or of anarthria. But for the result of the necropsy I should have regarded this case as one of complete aphemia. It shows, therefore, that there may be no clinical differences present to enable us to make this differential diagnosis, even though in the one case we have destruction of Broca's region, and consequently no kinæsthetic revivals of words possible, while in the other this kind of word memory is in no way interfered with, and there is also less chance that the lesion would lead to any interference with the functions of the auditory and the visual word-centres. For another very interesting case of aphasia in some respects similar to the last I am indebted to the kindness of Dr. Dickinson.

[12] Loc. cit., p. 105.

[13] Afasia e sue Forme, Lo Sperimentale, 1886, lvii., Obs. II., p. 270, and quoted by Prévost in the Revue Médicale de la Suisse Romande, June 30, 1895.

CASE 8.—A sailor, aged forty-nine years, was admitted to St. George's Hospital under Dr. Dickinson's care, having had a fit in a neighbouring public-house. He had landed a few days before, and had been robbed in some place of resort for sailors, and had been much disturbed in mind in consequence. One and a half hours after admission he had recovered consciousness and intelligence, but not his speech. He could not say anything intelligibly except the word "no," but the remarkable fact about his case was that he could express himself perfectly in writing, which he did with his left hand. He was hemip egic on the right side, the right arm being completely powerless, the right leg partially so; there was also slight loss of sensibility over the whole of the right side of the body. For three weeks his speech was limited to the use of the word "no," a sound like "yong" which stood for "wrong," and the frequent repetition of the syllables "yah yah," which stood for everything. Told to repeat a sentence or phrase, "Great Britain and Ireland" or any other, he uttered the same "yah yah yah" for either. But he could express himself in writing as fluently and correctly as if his faculty of spoken language were uninjured. He wrote daily reports and an autobiography during the time that he was unable to speak. He understood perfectly all that was said to him. He retained his sense of music, and could whistle "God Save the Queen." On the twenty-fourth day after the seizure it was evident that his speech was improved; he could say "doctor" and "better." In the evening his full power of speech was rather suddenly restored. The limbs recovered more gradually, the leg sooner than the arm. He left the hospital six weeks after admission, able to walk, but not to lift his right arm. Dr. Dickinson adds:—"The perfect ease and fluency with which the man wrote with his left hand showed that he had been in the habit of doing so. He was in common phrase left-handed, though he said he was not to be called so, since he used both hands equally."

Though in many respects different from the case previously recorded, this resembles it in the fact that we have to do with what certainly seems to be a simple case of aphasia without appreciable mental degradation and with the power of writing unimpaired. The case is also interesting since it constitutes an exception to the very general rule that left-handedness suffices to transfer the leading speech activity to the right side of the brain, and because of the further exceptional circumstance that being left-handed the patient had not been taught to write with his right hand. Another case that was recorded many years ago by Wadham[14] is in some respects even more remarkable, and all the more convincing because it was followed by a necropsy. It will be observed also that before death the patient had regained his power of speaking. The essential details are as follows:—

CASE 9.—A youth, aged eighteen, left-handed and ambi-dextrous, became partly hemiplegic on the left side and completely speechless after long exposure to cold, and was admitted to St. George's Hospital under the care of Dr. Wadham. Twelve days later, on being given a slate and pencil, he wrote readily the word "orange," and when asked his name also wrote that correctly with his right hand, although his mother asserted that she had never previously seen him do so. He and four of his brothers were left-handed. About a week after this, being still absolutely speechless, when asked whether he tried to speak and was unable he wrote "Yes." Asked if when well he wrote with his right or with his left hand, he wrote "Both," and then added, "Fight with left." In six weeks' time the left hemiplegia had much diminished, but he had still never spoken a word and continued to write all his wishes on a slate. His manner gave the impression of his "being very intelligent and rather facetious." At the expiration of three months he left the hospital, and when seen at his own home two weeks later Dr. Wadham says: "I found that at his mother's dictation he repeated after her various words and sentences with the intonation employed by one who endeavoured to speak without moving his tongue." This power was gradually increased until at last he was able to talk with sufficient distinctness to be perfectly understood by those accustomed to him. He subsequently suffered from necrosis of the jaw, and three months after his discharge was re-admitted to the hospital. After an operation on the jaw he became able to move his tongue naturally. His articulation subsequently was indistinct, but he "had no difficulty in producing words, and always used the right ones." He died twelve months after the onset of his illness. At the necropsy a large area of softening was found in the right hemisphere, involving part of the white substance beneath the Rolandic area, and completely destroying the island of Reil. The left hemisphere was found to be perfectly healthy.

It may be thought by some that these two cases have little bearing upon the question we have been considering. But this would be an erroneous impression. Certain authorities maintain, as I have said, that destruction of the third frontal convolution alone in the leading hemisphere for speech gives rise to agraphia as well as aphasia. They believe, for reasons that will be more fully specified presently, that destruction of Broca's centre hinders the revival of words also in the other word-centres, and thus prevents writing by stopping the process at its source. But in the cases of Ogle, of Guido Banti, and of Wadham—and possibly in that of Dickinson—we see that the ability to think and write was not interfered with by such a lesion in the leading hemisphere for speech, and therefore that the activity of the corresponding auditory and visual word-centres was neither suppressed nor gravely interfered with.[15]

[14] St. George's Hospital Reports, vol. iv., 1868, p. 245.

[15] Dally (Ann. Med. Psychol., 1882, p. 252) mentions the case of another left-handed man who had an apoplectic attack resulting in left hemiplegia and aphasia, who was not agraphic, though he wrote in a paragraphic fashion. Two years afterwards he had almost recovered from his hemiplegia, and he could write, speak, and read, though he was unable to spell properly. M. Mesnet mentioned also that he had formerly presented to the society a patient suffering from left hemiplegia and aphasia with preservation of ability to write.

The case reported by Wadham is further of special importance because it has been contended by Déjerine and also by Mirallié,[16] upon the basis of four cases (without necropsies) of aphasia with left hemiplegia occurring in left-handed persons, in whom the power of writing was lost simultaneously, that this loss is the rule and only what might be expected. The cases I have cited show, however, that it is not the rule, and leave us free to suppose that those of Bernheim, Parisot, Magnan, and Déjerine (where the power of writing was lost) were not cases of simple aphasia due to lesions in Broca's region only. The lesions in them may also have involved the visual word-centre in one or other hemisphere, or the commissure between them, and in either of these eventualities we might find an explanation of the co-existing agraphia.

In all the cases of left-handed persons in which the right hemisphere takes the lead in speech, whilst the left hemisphere executes the writing movements, it must be supposed that the right auditory word-centre is the initial centre for the revival of words either for silent thought or for speech, and therefore also for spontaneous writing. Thus in such a person we must assume that the left visual word-centre becomes educated concurrently during the process of learning to write (through the commissural fibres in the corpus callosum), and that the writing process is executed by this left side of the brain in the ordinary way by the passage of stimuli from the visual word-centre on to the corresponding che'ro-kinæsthetic centre.[17]

There is, therefore, no reason whatever why some, and perhaps the majority of, left-handed persons when they become the subjects of left hemiplegia and aphasia should not, after the shock attendant upon the brain lesion has passed, either be able to write at once, as was the case with Dickinson's patient and with the ambi-dextrous patient of Wadham, or else speedily learn to do so. Where there is not this ability after a time, as in the case of Trousseau and one subsequently to be referred to which has been recorded by Déjerine, there would, as I have said, be fair ground for suspecting that the lesion was not confined to Broca's convolution, but was also associated with some damage to the visual word-centre either on the same or on the opposite side of the brain.

Returning from this partial digression concerning the mode in which writing is performed in some aphasic persons, it will be seen that the case of Guido Banti, as well as that of Dickinson and of Wadham, is absolutely opposed to the views of Stricker and Hughlings Jackson. They show that for mere thought, apart from its oral expression, the memorial recall of kinæsthetic impressions may be even less necessary than might have been supposed by those who do not share their views. These cases are equally opposed to the views of Déjerine, Wyllie, Mirallié, and others, who, without looking upon this form of word-memory as the most important of all, nevertheless think that glosso-kinæsthetic revivals are necessary for silent thought, and that without such a process as an initiative there is an inability to recollect words (amnesia verbalis), together with an inability to read and write.[18]

Déjerine seems to have been led to these views in the first place by adopting as does Wernicke, the opinion expressed by Trousseau, Jackson, and Gairdner, that a lesion limited to Broca's region carries with it agraphia as well as aphasia, and that the two defects are almost always proportional to one another. The frequent co-existence and parity of the two defects cannot be denied, but this, as we have seen, may be due to the common simultaneous damage to the glosso- and the cheiro-kinæsthetic centres, or to the commissures connecting these centres respectively with the auditory and the visual word-centres.

[16] Loc. cit., p. 85.

[17] The process of learning to write here would, in fact, be the converse of what occurs when an ordinary right-handed person suffering from right hemiplegia and aphasia learns to write with the left hand. The only other possibility would seem to be that the incitation for writing should pass direct from the visual word-centre in the leading speech hemisphere (though in a diagonal direction) to the centre for writing in the opposite hemisphere, which seems to me much less probable.

[18] Thus Wyllie, who follows Déjerine, goes so far as to say (loc. cit., p. 314): "There is reason to believe that in every case of severe motor aphasia that is due to destruction of the motor images amnesia verbalis is extremely well marked—even more so, perhaps, than it is in severe cases of auditory aphasia."

A careful examination of the cases capable of throwing light upon these two interpretations, to some of the best of which I have above referred, tells distinctly against the view, which has been adopted by Déjerine and others, that agraphia is to be regarded as one of the consequences of an isolated destruction of Broca's centre. The fact, however, of Déjerine adopting this view has led, I venture to think, to his attaching an altogether undue importance to destruction of Broca's region, since he not only believes that it interferes with silent thought by hindering the revival of auditory word images, but that it leads to some amount of alexia, and is likewise a cause of agraphia. It has also induced him to bring forth a variety of arguments tending, as he thinks, to disprove the existence of any cortical centre having the same relation to writing movements that Broca's centre has to speech movements. All these positions have, moreover, been recently maintained by his pupil Mirallié in a valuable and interesting work entitled "De l'Aphasie Sensorielle." It will be necessary, therefore, to look at these various opinions in succession, in order to see how far they are warranted by existing knowledge.

*1. Does destruction of Broca's region alone entail amnesia verbalis?*—To this question I am disposed to give a negative answer. I rely upon the reasons previously adduced to show that words are primarily revived in the auditory word-centre rather than in the glosso-kinæsthetic centre. I can also refer to the case of Guido Banti, as well as to those of Dickinson, Wadham, and others, seeing that the ability of these individuals to write clearly showed that they could recall words and were not suffering from amnesia verbalis. It is true that Lichtheim also agrees with Déjerine and Wyllie in regard to this question. Thus he says[19]: "I think that in most motor aphasics—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 safe criterion, he thinks, of the preservation of the auditory symbols in these cases would be obtained by the existence of an ability to write, or, in default of that, the ability to realise the correct number of syllables in any word that cannot be uttered, and to signify this number by giving so many hand-pressures. Both these powers are, he thinks, usually lost in patients suffering from motor aphasia. It has been shown, however, that this is not so in uncomplicated cases of destruction of Broca's region, since, as we have seen, the power of writing persists, and so doubtless would the other test of minor significance to which he refers. I am quite prepared to admit, however, that exceptions to this rule may be occasionally met with. The functional relations of the auditory word-centre with Broca's region on the one side and the visual word-centre on the other are so close that the destruction of either of these centres may be easily conceived to derange the functional activity of the others for a longer or a shorter time. The degree of this derangement may be considered to vary in different persons, perhaps in accordance with the relative potency, original or acquired, of the several centres. Thus in some persons an isolated destruction of Broca's region may disturb to a very slight extent the functional activity of the auditory and the visual word-centres, whilst in others it is quite possible that the severance of this centre from the other two may greatly alter their functional readiness, so that the spontaneous revival of the auditory and visual images of words may in such persons be more or less hindered, and as a consequence the power of thinking and of writing proportionately lowered.

*2. Does destruction of Broca's region entail alexia?*—I believe that, as a rule, in reading a proper comprehension of the meaning of the text requires a conjoint revival of the words in the visual and the auditory word-centres, but that for this mere comprehension it is not necessary for the stimulus to pass on also to the glosso-kinæsthetic centre, as it must do in reading aloud. It may, however, be freely admitted that if the way is open, and this latter centre is in a healthy condition, it does commonly receive in reading to one's self a slight stimulus from the auditory word-centre, a fact which is often enough shown by the occurrence of involuntary half-whispered mutterings when reading. It may also be admitted that the rousing of all three centres does give assistance in the comprehension of anything difficult, as is shown by the common practice of reading aloud any passage the meaning of which may be at all obscure. By this proceeding aid is obtained, however, not alone by the full rousing of the glosso-kinæsthetic centre, as there is also the full activity of the auditory word-centre following upon the spoken words. This kind of aid is, indeed, commonly needed, as Wernicke pointed out, by an uneducated person for the full comprehension of almost all that he reads. Therefore it may easily happen when such a person becomes aphasic from destruction of Broca's region that alexia may go with the aphasia.[20] Similar aid may be necessary in cases in which there is functional weakness of the visual word-centre. Thus there is now under my care in the National Hospital for the Paralysed and Epileptic a woman, apparently suffering from a tumour in the left hemisphere, who is hemiplegic on the right side and understands readily all that is said to her, but who does not seem to gather the meaning of the simplest written or printed request unless she reads it aloud, which she is able to do without hesitation. She is quite unable to write spontaneously or to copy with the left hand, but then she has made very few attempts in this direction since her right hand became paralysed.[21] It would seem, however, that in some persons the comprehension of writing or printing does not even require the associated activity of the auditory word-centre, so long as the visual word-centre is in a healthy condition and its other associational fibres are intact. This is proved by the fact that in a case recorded by Wernicke and Friedländer,[22] in which there was aphasia associated with word-deafness (due to a lesion in Broca's region and also in the upper temporal convolution), the patient was able to read well and even to write; there is also a valuable case of word-deafness (with lesions in both upper temporal convolutions) recorded by Mills, and to be quoted further on (Case 26), in which the patient was able to understand what she read; and a still more remarkable case recorded by Pick, with very similar lesions (Case 30), in which, although the patient was word-deaf, he could write and comprehend perfectly what he read.

Long before I was aware of the crucial evidence afforded by these cases and that of Guido Banti I did not believe that the comprehension of writing or of print was appreciably interfered with by an isolated destruction of Broca's region—that such a lesion, in fact, did not lead to alexia. For independently of the general views discountenancing such a notion there were Case 6 and two recorded cases by Broadbent, as well as others, pointing in the opposite direction. Some particulars concerning one of these cases recorded by Broadbent may well be reproduced here.[23]

CASE 10.—A man, aged twenty-nine years, was admitted to St. Mary's Hospital on April 3rd, 1870. He had had fits when a child, the last at the age of seven years; and rheumatic fever twelve months previously to his admission. He had not been well, and had left off work for two days. During these two days he talked incessantly all sorts of nonsense. In the night his brother awoke and found him in violent convulsions, mostly of the legs, not more on one side than on the other. The attack lasted two hours; when it was over right hemiplegia with loss of speech was established. On admission the paralysis was complete, the loss of speech absolute, and he could not protrude his tongue. He gradually gained some power in the leg so as to be able to walk about, but the arm remained motionless and flaccid. He could now also say "yes" and "no," but was not always right in his use of these words. On one occasion he twice said "Here" to his sister, when impatient that she had not complied with his wish for her to come to him. He could write his name, but nothing else; and wrote it when asked to write an answer to a simple question. Broadbent adds: "The case presented no unusual features, and is here recorded partly as an example of a rather unusual type of attack in embolism; partly for the purpose of noting that the patient while in the condition described read the newspapers much with apparent interest, and at my request he pointed out a Crystal Palace advertisement of fireworks and an account of pigeon shooting at Hurlingham, the latter very promptly and with a pleased expression, as if he had already read and enjoyed it."

Many of the cases in which there has been alexia co-existing with aphasia are I think clearly cases in which there have been rather wide lesions extending far beyond the third frontal convolution and involving also the visual word-centre. This is the interpretation that I attach to a celebrated case recorded by Trousseau, which evidently influenced his opinion very much. I allude to the wealthy patient whom he saw in consultation in the department of Landes.[24] On this point Trousseau says: "As he always answered 'Yes,'

[19] Loc. cit., p. 471.

[20] Kussmaul, loc. cit., p. 775.

[21] This patient died within a fortnight, when a tumour about two inches in diameter was found in the midst of the left centrum ovale, not extending into the cortex, but which may have caused some slight but undue pressure upon the left visual word-centre.

[22] Fortschritte der Medicin, No. 6, 1883.

[23] The other is recorded further on (Case 13) under the head of Agraphia.

[24] Lectures, Translation by Bazire, pp. 229-232.

I asked him whether he knew how that word was spelt, and on his nodding assent I took up a large quarto volume with the following title on its back, 'History of the Two Americas,' and requested him to point out the letters in those words which formed the word 'yes.' Although the letters were more than one-third of an inch in size, he could not succeed in doing as I wished. By telling him to seek for each letter in turn, and by calling out its name, he managed, after some hesitation, to point out the first two, and was very long in finding the third. I then asked him to point out the same letters again, without my calling them out first; but after looking at the book attentively for some time, he threw it away, with a look of annoyance which showed that he felt his inability to do as I wished him." Few, I think, would now suppose that we had to do here with a case in which there was no lesion about the posterior extremity of the Sylvian fissure as well as of the third frontal convolution.

Lichtheim is inclined to give much the same kind of interpretation to this class of cases. He points out that the examination of an old case is apt to be confusing in regard to its correct pathogenesis, owing to the fact that some of the defects originally existing have cleared up and disappeared. In this relation he calls attention [25] to the "usually rapid disappearance of word-deafness, and the greater persistence of the troubles connected with written language." He adds: "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 co-exists."

It is, of course, possible that this explanation by Lichtheim may hold good for certain cases, but that it is not necessary to have recourse to it in order to explain those cases of apparently simple aphasia with which alexia co-exists I have become convinced by two cases that have recently come under my observation. What we have to ask ourselves when in the presence of such cases is whether there is any evidence to show that the case is not one of simple aphasia, and whether a lesion exists also in the region of the posterior extremity of the Sylvian fissure. We may inquire, therefore, for the previous existence of word-deafness as Lichtheim suggests, and also examine the patient carefully for the present existence of some amount of hemianæsthesia. The finding of this latter would show two things: first, that the lesion was not limited to the third frontal convolution; and, secondly, that either it or a second lesion also existed not far away from the visual word-centre, which consequently may have been also involved. Since I resolved to apply this test two cases of aphasia combined with alexia have come under my care, and in both of them there was the co existence of a more or less complete hemianæsthesia, as may be seen from the abstracts of these cases which I now give.

CASE 11.—A woman aged thirty-two years, was admitted into the National Hospital for the Paralysed and Epileptic under my care on May 19th, 1896. She had been married fifteen years, and had eight children. She had a loud, mitral regurgitant bruit; the pulse was small, and irregular in force and frequency. She had been in good health till Oct. 25th, 1895, when she had a fit with loss of consciousness lasting three hours. For two or three days afterwards she seemed quite dazed and did not know anyone. The fit was followed by loss of power in the right arm and leg and loss of sight in the left eye. She has regained some power in the right arm and leg, but is still blind in the left eye. Ophthalmoscopic examination shows simple white atrophy of the left optic disc with evidences of past embolism of some branches of the central artery of the retina. She is naturally right-handed. There is distinct, though slight, right hemiplegia, including the face, arm, and leg. She can walk with dragging of the right foot and perform all movements with the right leg and arm, though much less forcibly than with the left limbs. The tongue is protruded straightly. There is incomplete right hemianæsthesia; sensation is dulled to touch and pain to the mid-line of the face, head, and trunk (back and front), also over the arm, hand, and upper two-thirds of the thigh. She is almost completely speechless. She can answer "Yes," "No," and say "Yes, I know" in answer to questions. Once she said "Good night." Her articulation of these few words is good. She cannot speak voluntarily, and cannot repeat the simplest word when bidden. The patient understands more or less complex matters spoken to her, and performs some spoken commands, but is slow at it, and often makes mistakes. When told to show her tongue she generally shows her teeth. There is almost complete alexia. She cannot recognise words or letters as a rule, except short names of external objects, such as "pen," "lamp," "cat," "dog." She will, when shown the name, point to such objects in the ward or in pictures. She does not recognise the word "man," and she cannot understand simple written commands. When shown four or five capital letters she can very rarely point correctly to one that may be named. She does, however, recognise and point out numerals; she can add two numbers together, but cannot subtract or multiply. She can also pick out objects named; she cannot name them, but shows by her expression that she recognises their names when she hears them. She cannot write letters with either hand spontaneously or from dictation, but she can copy single letters printed or written—the former better than the latter. She cannot do transfer copying.

[25] Loc. cit., p. 467.

This case, though interesting, is perfectly simple and needs no comment. The next, though equally cogent, is a little more complex.

CASE 12.—A young woman, aged seventeen years, was admitted into University College Hospital under my care on June 20th, 1896. She had had rheumatic fever when a child, since which she had been always ailing, principally by reason of a heart affection. She had a loud presystolic bruit and some hypertrophy of the left ventricle. On June 8th, 1896, on awaking she found herself paralysed on the right side of the body. During the following day, without the occurrence of any fit, she lost her speech. During the first week of her illness she was very dull mentally, and there was incontinence of urine and of fæces. She also suffered from pains in the left side of the head. When admitted, on June 20th, the paralysis of the right arm and leg was complete, together with slight paralysis of the lower half of the face. There was well-marked right hemianæsthesia, but no hemianopia. There was ankle clonus, with increased knee, wrist, and elbow jerks on the right side. The limbs were flaccid. She was almost speechless. She understood all that was said to her, looked intelligent, and often attempted to speak, but then and since her illness the only words she had uttered had been "yes," "no," and "oh, dear!" She often said "Yes" inappropriately when asked a question, and then immediately shook her head in correction. She could not repeat a single word when bidden. She recognised objects such as pencils, paper, and pens, but could not name them. There was complete alexia; she did not understand written or printed words or short sentences; nor could she point correctly to individual letters. On June 22nd she could repeat short words uttered before her in a slow, hesitating manner, and with indistinct utterance. On June 27th she was much better; she could count up to ten, and say the days of the week when started; she could also name most common objects which she was shown. She could also read aloud short words in print or writing, and understand their meaning. On July 2nd she talked a little in short sentences, though her pronunciation of words was still indistinct, and she had difficulty in recalling the words she wanted. On July 8th she was still improving. She could then move her right leg a little. She talked slowly and indistinctly, but still had difficulty in recalling words, though she did not utter wrong words. She read aloud a few lines of short words from a book slowly, passing over some and miscalling other words. She seemed to understand what she read. Some right hemianæsthesia still existed.

When first admitted this patient seemed to present a typical case of aphasia, but for the existence of the alexia. The fact of the presence of hemianæsthesia showed, however, that the lesion extended back to the posterior third of the hinder segment of the internal capsule, or else that another lesion existed there—that is, in a region very close to the visual word-centre. This, of course, carried with it the possibility that the visual word-centre itself might have been temporarily disabled. The subsequent progress of the case—the speedy clearing up of the aphasia, owing probably to a restoration of the circulation through the first cortical branch of the left-middle cerebral, which may have been temporarily blocked, and the revelation then of some amount of amnesia verbalis—made it all the more likely that the alexia was to be explained by some temporary disablement of the visual centre. That the auditory centre was only slightly (though more durably) affected was shown by the fact that there was never word-deafness and by the patient's subsequent ability to read even better than she could speak spontaneously.

The probability is, therefore, that in the cases of associated aphasia and alexia recorded by Trousseau and others the latter defect is not to be regarded as a consequence of the lesion in Broca's region, but rather as a result of some co-existing damage to the visual word-centre.

*3. Does destruction of Broca's region entail agraphia?*—The writers whom I have previously named say yes, that agraphia as well as aphasia is entailed by an uncomplicated lesion in the posterior part of the third frontal convolution. But with this doctrine, as already stated, I cannot agree. In writing spontaneously or from dictation there is, I believe, needed for most people a primary revival in the auditory word-centre and secondarily in the visual word-centre, whence combined stimuli pass to the cheiro-kinæsthetic centre, and thence well over in coördinated form to the motor centres in the cord by which the actual writing movements are effected. The evidence that I have already recorded is quite in harmony with this view, whilst it is entirely opposed to the doctrines on this subject formerly enunciated by Trousseau, Hughlings Jackson, and Gairdner, and which have been recently advocated by Déjerine, Wyllie, Mirallié, and others.

*4. Is it right to deny the existence of a special centre for the registration and regulation of writing movements?*—We are now brought to the consideration of Déjerine's objection to the very existence of a special centre for writing movements—a "cheiro-kinæsthetic centre" as I name it. His reasoning here seems to me rather surprising, and is of such a nature as to make it necessary to premise that the case of writing is almost exactly comparable with that of speaking—and, indeed, of all other habitual voluntary movements, whether

complicated or simple. Each set of movements must be associated with a set of ingoing impressions (kinæsthetic), which are registered in different portions of the Rolandic area of the cortex. This must be as inevitably true for writing movements as for speech movements; and, just as re-excitation of Broca's region under stimulation from the auditory word-centre is necessary for speech, so a re-excitation of the centre in which the impressions generated by writing movements are registered is, under stimulation from the visual word-centre, needful for the production of writing movements.

Yet both Wernicke and Déjerine say that the intervention of a special centre for writing movements is not necessary, on the ground that writing consists in a simple copying of the visual images stored up in the visual word-centres. This, however, is extremely vague. It must be asked, How is the copying achieved? That is, By what cerebral mechanism is it brought about? I maintain that the visual word-centre cannot act directly upon the motor centres in the cord, and that the conjoint activity (for coördinating purposes) of a kinæsthetic centre is quite as essential in the case of writing movements as is the activity of Broca's region for the production of speech movements. By parity of reasoning Wernicke and Déjerine might just as well say that Broca's centre is not needed, on the ground that speech consists in a simple copying of the auditory images stored up in the auditory word-centre, and hold that the auditory word-centre can act directly upon the motor centres in the bulb for the production of speech. But I contend that in each case another regulating centre must intervene, and that such a centre is just as essential for the translation of visual images into writing movements as it is for the translation of auditory images into speech movements.

Again, How can it possibly be alleged as a valid argument against the existence of a cheiro-kinæsthetic centre in a man accustomed to write with his hand that the same man is able to write on sand rudely with his foot, or that he can after a fashion write with his elbow, or with a pencil placed between his teeth? Clearly in these other crude modes of writing he would simply be dependent upon the activity of other parts of the general kinæsthetic centre. Such objections—for they are scarcely worthy of the name of arguments—are therefore altogether futile. The question that has to be considered is whether a man who has learned to write with his right hand, and who habitually writes in this fashion, calls into play a cortical centre the activity of which in the production of those movements is comparable with the activity of Broca's centre in the production of speech movements. To this question there can be but one answer. And the fact that in those cases of word-blindness in which the patient is able to write spontaneously he can subsequently succeed in reading what he has written, by tracing the outlines of the letters with his finger must also be taken as direct evidence of the existence of a cheiro-kinæsthetic centre, as I pointed out many years ago.[26] The impressions produced by the writing movements thus occasioned pass from their centre of registration back to the visual and auditory word-centres, and thus enable the writing to be read. Of course, a patient would not be able to read in this way if his visual and auditory word-centres were destroyed; therefore the objection of Déjerine and Mirallié to this evidence falls to the ground.[27]

Another argument adduced by Déjerine against the existence of a special centre for writing has been based upon an interesting case which he cites very briefly[28] of a patient, left-handed for all acts save those of writing, who became hemiplegic on the left side and aphasic, and at the same time lost the power of writing with his right hand. The patient continued in this condition for four years, and judging from the symptoms (there being no necropsy) Déjerine regarded it as a simple case of "motor aphasia." He thinks that the foot of the right third frontal was destroyed, and that this patient was as incapable of writing as of speaking because he had no longer a "notion of the word," owing to his supposed inability to revive verbal images. The fallacy of this inference has already been shown in part by the cases of Dickinson, Wadham, and others previously recorded; and in part by the evidence I have brought forward as to the non-occurrence of alexia as a consequence of destruction of Broca's region. This latter evidence goes to show that in many cases of what at first sight appear to be cases of simple "motor aphasia" with alexia there is reason for believing that a lesion exists in the hinder part of the hemisphere which may have damaged the visual word-centre. A lesion in this situation may therefore have existed in the case under the care of Déjerine, and thus have accounted for his patient's inability to write as he had been accustomed to do with his right hand. For if his previous power of thinking in words had depended upon the activity of the word-centres in the right hemisphere, then spontaneous writing would have required the activity of the auditory and visual word-centres of this side as a first stage in the process, and secondly that they should be in relation (through the corpus callosum) with the similar centres in the left hemisphere, so that the proper stimuli might be passed on to the left cheiro-kinæsthetic centre for the bringing about of writing movements by the right hand. A damage to the visual word-centre of the right side would therefore have caused this patient to lose his previous power of writing with the right hand. The case under the care of Déjerine therefore admits of an explanation quite different from what he has given to it, and which is also much more in accordance with existing knowledge. Clearly it cannot be deemed to support his notion that the kinæsthetic impressions of writing movements are not registered in a definite part of the cerebral cortex, and that this cortical region is not called into activity during the act of writing. That such impressions must be definitely registered in the cerebral cortex seems to me a self-evident truth.

It is, however, altogether another question, and one which does seem open to discussion, whether the cortical centre in which the sensory impressions produced by writing movements are registered exists altogether apart, or whether its structural elements are inextricably mixed up with others pertaining to less special movements of the hand and arm. If there is a completely separate seat of registration, destruction of such a centre should produce loss of the power of writing, independently of paralysis of other less special movements of the limb. If otherwise, the loss of power of writing from damage to the cortical region in which its kinæsthetic impressions are registered would never be able to occur alone, but would always be merged in a more general paralysis of the limb. In other words, no agraphia of this type would be recognisable as such; it would not be looked for, because the co-existing paralysis of the hand and arm would naturally be supposed to be, and would in fact be, a sufficient cause of the inability to write. On the whole it must, I think, be said that no actual proof has yet been given that the centre for writing movements is topographically distinct—in other words, that Exner's or any other separate localisation of such a centre has yet to be definitely proved. The evidence that I have previously cited, though it has been sufficient to discountenance the idea that destruction of Broca's centre of itself gives rise to agraphia, has no pretence to be adequate for the localisation of a separate centre, and the cases that have hitherto been cited in support of Exner's localisation are all of them open to very legitimate objections. There is, for instance, a case recorded by Bar[29] in which an isolated lesion existed in the foot of the second frontal convolution, but, instead of giving rise to agraphia only, there were aphasia and agraphia, and the agraphia was not total (as it should have been if a topographically separate centre for writing had been destroyed, since the patient's penmanship was good); and, though he could not express his ideas either in writing or in speech, he wrote over and over again a certain phrase or word. This case, therefore, though cited by Exner in favour of his localisation, seems to medecidedly opposed to it. Another case, recorded by Henschen,[30] has been cited as favouring Exner's localisation, which has, however, no real value. Here the patient suffered from word-blindness and agraphia, and after death there was found not only a destruction of the foot of the second frontal, but also a softening which destroyed the angular gyrus.

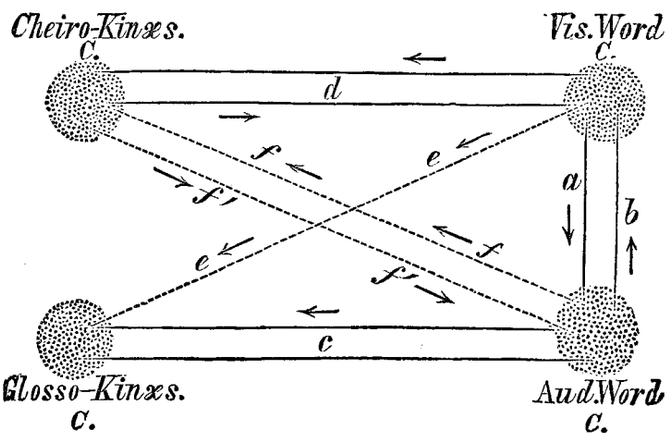
A third case has been recorded by J. B. Charcot and A. Dutil,[31] and is thought by Pitres to favour Exner's localisation. Here the patient was a woman sixty-four years of age, who twenty-nine years before her death had a slight cerebral attack which left her completely agraphic. Subsequently she had three other attacks, which led

[26] The Brain as an Organ of Mind, 1880, p. 646, note.
[27] Mirallié, loc. cit., p. 75.
[28] Mémoires de la Société de Biologie, 1891, p. 97.
[29] La France Médicale, 1878, p. 609.
[30] Klinische und Anatomische Beiträge zur Pathologie des Gehirns, Upsala, 1890, p. 273.
[31] Mémoires de la Société de Biologie, 1893, p. 129.

successively to embarrassment of speech, left hemiplegia, and finally to pseudo-bulbar paralysis. The agraphia persisted throughout her life without appreciable diminution. She could copy letters and figures after a fashion, but was absolutely unable to write spontaneously. There was no word-blindness or word-deafness. At the necropsy five small foci of softening were found in the right hemisphere and two in the left hemisphere, one of the latter being situated exactly in the foot of the second frontal convolution.[32]

Then, again, one of the causes which led Déjerine to deny the very existence of a centre for writing movements seems to have been based upon the characteristics of two cases that were brought forward by Pitres, and which, upon the basis of his authority, have been commonly cited by others as affording evidence of the correctness of Exner's localisation. They were cases in which the patients were unable to write spontaneously or from dictation, and yet were able to copy writing. If the centre is destroyed, says Déjerine, the possibility of executing any kind of writing should be abolished. That at first sight seems perfectly true; and it may be a valid criticism against Pitres for quoting any such cases as instances of "motor agraphia."[33] But Déjerine and his followers have overlooked the possibility that Pitres may not have been quite correct in his interpretation of these cases—in which there was no necropsy. They have not recognised the fact that this particular combination of symptoms may be present without the existence of what is called "sensory aphasia" in any of its forms. It can be shown, however, that such a combination of symptoms may be easily explained by a cutting across of the audito-visual commissure that transmits impressions from the auditory to the visual word-centre (Fig. 3, *b*). This I believe, after careful

FIG. 3.




study of his memoir, to be the explanation of the partial agraphic defects in the two well-known cases cited by Pitres as examples of the effects produced by damage to the cortical centre for writing movements.[34] It is obvious that damage to the commissure above mentioned would be capable of preventing the passage of stimuli from the auditory to the visual word-centre such as would be necessary in writing spontaneously or from dictation, whilst it would leave the power of copying writing intact so long as the visual word-centre, the commissure connecting it with the writing centre, and this centre itself remained undamaged.

At present, then, it would seem that there are no uncomplicated cases of agraphia upon record produced by a small lesion in the cheiro-kinæsthetic centre, or of agraphia as an isolated symptom certainly resulting from damage to some part of the visuo-kinæsthetic commissure. It seems clear, however, that if the centre for writing is topographically distinct, complete agraphia—that is, an inability to write spontaneously from dictation or to copy with anything like facility—should be producible in either of these ways, and that in each case the disability would be uncomplicated with word-blindness, whilst there would be nothing to prevent the patient learning to write with his left hand.

The reason for this extreme rarity of agraphia as an isolated symptom doubtless is that often in what might otherwise have been a simple case of inability to write this very special disability becomes hidden and unrecognisable, owing to the lesion being large enough to cause paralysis of many other movements in the hand and arm. Thus the mere inability to write is hidden by the production of a partial brachial monoplegia. No similar causes intervene to obscure the recognition of simple, uncomplicated aphasia, and yet it must be borne in mind that even this is decidedly rare.

If, then, there are no simple, uncomplicated cases of agraphia that can be quoted, either from disease of the cheiro-kinæsthetic centre or from damage to the commissure between it and the visual word-centre, all that can be done is to cite some of the best instances available in which agraphia has been associated with aphasia, but where the former defect has been more marked than the latter. This was, in fact, the class of cases originally described by William Ogle in his paper on "Aphasia and Agraphia." One of his examples (in which there was no necropsy), where right hemiplegia with aphasia was succeeded by a condition in which amnesia and agraphia were marked, still remains one of the most typical of this kind that can be cited. But two simpler and more typical cases have been recorded by Broadbent[35] to which I would first direct attention. One of them is notable also on account of the remarkable amount of intelligence and energy displayed by the patient, notwithstanding her very defective speech and inability to write. The following is an abstract of this important case.

CASE 13.—A lady, aged about seventy years, had an apoplectic attack on July 26th, 1867. She remained unconscious for nearly twenty-four hours, and was then found to be hemiplegic on the right side and quite speechless. After two or three months she had "quite recovered the use of her limbs and regained a few words." Her relatives treated her as if she were utterly incapable of looking after her own affairs, and especially of taking charge of her own money. This was a great grievance, and when her husband's relatives visited her she tried to make it known by showing her purse and repeating excitedly, "Oh, shameful! shameful!" She had an intense dislike to her attendant, and when she left the room would often look in the direction in which she had gone, shaking her head and exclaiming with great energy, "Oh! nasty! nasty!" following it up by a long story in which there was not a single word intelligible. At length she went to live near her husband's relatives. "A house was now taken and furnished for her, and she showed herself quite competent in making all arrangements about it, and when settled in it with a new housekeeper she seemed quite contented. Her principal topic of attempted conversation for several months was her niece's and brother's bad behaviour to her. She laboured hard to make the friends among whom she now was understand her grievances. She would point out her relatives' names or refer to their letters, or in some other way show to whom she referred, and then in great excitement would exclaim, 'Oh, shameful! shameful!'; then she would turn over letters and newspapers, and when she had tried in vain for hours to convey what she wished she would shed tears and say, 'Oh, pity! pity!' At length her banker's pass-book was obtained from her niece, and she scarcely knew how to express her joy. 'I tried, and I tried,' she said over and over again. But there was still something she wished to explain, and one day, while trying very hard as usual, she made the figures 40 quite distinctly, and it was understood at last that it was something about £40; but it was not till a friend came to see her and this happened to be mentioned that it was discovered she had put £40 into a bank before her attack, and believed her niece had the acknowledgement of it, and some promissory notes with other papers. These she wished to be sent for, and eventually they were obtained. This was nearly a year after her removal, and all the time she had persevered in her attempts at an explanation. During the same time she had been making a new will, and week after week would have her sister-in-law to write out the different clauses. It often took a long time to do it to her satisfaction, but she never rested till every particular was exactly as she wished it; when the right guess was made she showed her satisfaction quite unmistakeably by her gestures, and by saying, 'Yes, yes, that's it.' . . . . That she knew what word she wanted was shown by her resorting to letters and sometimes to a dictionary; and in one of my visits, wishing to recommend a linseed-meal poultice and failing to convey her meaning, she went for the housekeeping book and found an old entry of linseed-meal. She also pointed out to me newspaper paragraphs which interested her. She frequently read, but preferred being read to. . . . . She was quite unable to write or even to sign her name. She once wrote down the figures 40, as related, and sometimes wishing to divide money would make the proper number of marks to indicate the amount of a share." On Aug. 7th, 1871, she had another apoplectic attack, after which she was

[32] Two other cases which have been cited as favouring this localisation, namely—one by Balzer (Gazette de Médecine de Paris, 1884, p. 97 and another by Shaw (Brit. Med. Jour., February, 1892)—have in my opinion no value in this connexion.

[33] Pitres attempts to get over the difficulty standing in the way of his interpretation arising from the fact that the patients were able to copy writing in the following manner. He says (Congrès Francais de Médecine Interne, 1894, Rapport sur la Question des Aphasies, p. 10): "Copier, en effet, est toute autre chose qu'écrire couramment. C'est un acte de motilité générale qui n'a pas de centre d'exécution spécialisé, pas plus d'ailleurs que l'acte de tracer sur le sol avec le bout de sa canne, ou avec le pied, des traits ayant la forme de lettres. Ces actes là impliquent une attention soutenue, une surveillance constante de l'esprit, qui manque absolument dans l'écriture courante. C'est pourquoi l'agraphique dont les membres supérieurs ne sont pas paralysés, peut encore copier, bien qu'il ne soit plus capable d'écrire spontanément."

[34] Revue de Médecine, 1884, p. 864. These cases are also quoted more briefly by Wyllie (loc. cit., p. 357), though in the first of the two cases he omits the very important detail that the patient was able to copy writing easily.

[35] Transactions of the Royal Medical and Chirurgical Society, 1872, pp. 146 and 160.

found to be paralysed on the left side. She died on Aug. 12th, and at the necropsy no cause for the last apoplectic seizure was found, but two old apoplectic cysts were discovered in the upper border of the fissure of Sylvius. One "which might hold a nut" was in the situation at the lower end of the ascending frontal gyrus, "and a very small part of the adjacent posterior end of the third or inferior frontal gyrus." "Again, in the supra-marginal lobule over the posterior end of the fissure of Sylvius was another hollow filled with connective tissue, and when the fissure itself was opened out it was found to contain at this part a superabundance of connective tissue, and the convolutions of the posterior end of the island of Reil were completely atrophied." There was some atrophy also of the tail of the intra-ventricular portion of the corpus striatum.

In addition to the evidences of intelligence and energy displayed by this patient, there is also the fact that there was no alexia associated with the aphasia and agraphia. The latter defect was much more marked than the former; and the fact that the patient could use more words and sentences than are customary may perhaps be explained by the small extent of the lesion in the left third frontal gyrus. Although the paralysis of the right upper extremity had been quite recovered from, the agraphia was absolute. The posterior extremity of the second frontal gyrus was uninjured, but the posterior of the two old focal lesions discovered would probably have destroyed the commissural fibres between the visual word-centre and the cheiro-kinæsthetic centre not far from the former of these centres. In the next case there was a very similar inequality between the ability to speak and the ability to write.

CASE 14.—A German, aged thirty-four years, was admitted into St. Mary's Hospital in February, 1870. He had suffered from rheumatic fever in 1885. On the 4th he fell off his stool whilst at work. Two days after he was found to have lost the use of his right hand and arm and to be unable to speak. On the evening of the 8th he was brought to the hospital, and was then found to be partially hemiplegic on the right side and unable to answer questions intelligibly. He soon began to improve, and Broadbent's notes on the 14th are as follows: "All his answers in a low, smooth tone, without modulation of voice; he could not be induced to utter loudly even a word he could say. Answered 'Yes' and 'No'; told me his age correctly and his name, but indistinctly. Asked how he is replies, 'Better' or 'Quite well,' rather indistinctly. If he had pain in the head since the attack? 'No.' Before? 'Yes, wonderful.' If he slept well? 'Nicht'. . . . (something or other which could not be made out). If appetite good? 'No' . . . It was impossible to understand him when he attempted a phrase or when he tried to say anything to which I had no clue." A strong German accent was distinguishable in all he said, whereas before the attack he spoke English particularly well. "A peculiarity of his attempts to read or speak was his persevering effort to master a word, syllable by syllable, by trying on and on again. For example, when asked to read the words 'dietcard' his attempt was something like this, 'Diget, dicht, dite, dite, dite car.' The next moment he pointed to his name, saying, though not very distinctly, 'Dat my name.' Up to this time and a week later, Feb. 21st, he could not sign his name or write the simplest word, such as 'yes' or 'no,' when asked to do so, or copy a word pointed out to him." He remained in the hospital some weeks longer, improving very slowly, and before he left he could "with great effort write his name."

Nothing can be said as to the situation of the lesion here or in the next case, which is the best of those which William Ogle was able to bring forward in his paper. We must be guided only by the clinical facts recorded.

CASE 15.—James Simmonds, aged fifty-four years, after a heavy blow on the left side of the head, seven years ago was obliged to give up his work. He spoke without difficulty or hesitation, but miscalled things strangely. He then had a fit one morning whilst dressing, which left him speechless and hemiplegic on the right side. For a fortnight he could not speak at all, though he was quite sensible. He could not say so much as "Yes" and "No." From this he gradually recovered, but always, as before, miscalled things. . . . A month ago he had a second fit, which left him with less power than before in his right side, but made little or no change in his speech. There is now partial paralysis of the right side, which does not prevent his walking. The facial muscles on that side are slightly affected as well as the limbs. His speech is very hesitating and imperfect. He often stops suddenly at a loss for a word and then frequently uses a wrong one, as, for example, he substituted "barber" for "doctor," "two-shilling piece" for "spectacles," "winkles" for "watercress." He can, however, pronounce any word perfectly when prompted. He says that he generally knows when he has used a wrong word, but not always. Before his illness he wrote a good hand and was above his lot as regards education. Now he cannot form a single letter. Even with a copy before him he makes only uncertain up-and-down strokes. I gave him some printed letters and asked him to pick out his name. After a long time he arranged JICMNOS. Clearly he had some slight notion of the letters which composed his name. According to his wife, before his illness he spelt well, and was very particular about the spelling of his own name, which is one admitting of many variations. When a copy was before him he quickly picked out and arranged his name correctly. He can read; but he says that reading makes him very giddy and causes great pain in the head. His general understanding seems good and up to the average of men in his class.

The conditions here recorded represent the remainders of an attack of aphasia, as it would seem that the amnesia was perhaps not much more than had previously existed. We may conclude that in this case also the most severe or durable lesion was in the track of the commissural fibres between the left visual word-centre and the cheiro-kinæsthetic centre (Fig. 3, *d*), but that in all probability there was also some damage to the former word-centre itself. This latter defect is indicated from the difficulty he had in spelling his name with separate printed letters placed before him; for inability to spell—that is, inability spontaneously to recall the letters composing a word—probably depends in the main upon some defect in the visual word-centre, and although he could read, what is said seems to imply that his powers were perhaps not great in this direction. It is therefore by no means a clear case, but one in which the defects were somewhat mixed.

Although we are unable as yet to cite any simple uncomplicated case of agraphia due to disease in the posterior part of the second left frontal convolution, yet, according to Binet and Fere,[36] it is easy to produce agraphia by suggestion in hypnotised persons. Speaking of their experiments Ballet[37] says: "They show that the loss of the motor memory for writing is of the same order as the abolition of other motor [kinæsthetic] memories, such as those which preside over the ability to smoke, to sew, or to embroider. For each of these acts which we are accustomed to perform there exists, in fact, a motor memory, so much the more easily disturbed, in all probability, as the acts themselves are more complicated and require more delicate muscular combinations. As an example of motor amnesia connected with special movement associations I may recall a curious case related by M. Charcot in one of his lectures: it was the case of a player on the trombone who had lost the memory of the associated movements of the mouth and of the hand necessary for playing on the instrument. All the motor memories in this patient were intact save this one. This musician had forgotten how to handle the trombone, as others had forgotten how to handle the pen." If we substitute in the above-quoted passage for "motor memory" the term "kinæsthetic memory" we may believe the loss of the different kinds of kinæsthetic memory referred to, which can be brought about in hypnotised persons, must be due to a temporary functional inertia produced under the influence of suggestion in the several parts of the general kinæsthetic centre in relation with this or that movement.

It seems best now, at the close of our discussion concerning aphasia and agraphia as combined and separate symptoms, to enumerate in tabular form the different modes in which complete or partial agraphia may be produced. Although I shall thus be anticipating results subsequently to be developed, it will be advantageous to show now how other different forms of agraphia are to be distinguished from those we have been considering and from one another.

AGRAPHIA.

| | | |
|---|---|---|
| *Complete.* | Uncomplicated. | 1. Destruction of the cheiro-kinæsthetic centre.<br>2. Destruction of the visuo-kinæsthetic commissure. |
| | Complicated with word-blindness. | 3. Destruction of the visual word-centre. |
| *Partial.* | No spontaneous writing or from dictation, but can copy; no word-deafness. | 4. Destruction of the audito-visual commissure. |
| | No spontaneous writing, or from dictation, but can copy; word-deafness present | 5. Destruction of the auditory word-centre. |
| | Can write spontaneously or from dictation, but cannot copy; word-blindness present. | 6. Destruction of the visual word-centre (in some educated auditives).<br>7. Isolation of the visual word-centre from all afferent fibres. |

The supposition is that in the large majority of cases the various lesions that have been above referred to as causes of complete or partial agraphia will be in the left hemisphere in right-handed and in the right hemisphere in left-handed persons. Of these various forms Nos. 1, 2, and 7 should occur in all individuals alike, in association with the lesions named; but Nos. 3 and 6 are two degrees of agraphia that may be met with in association with word-blindness according as the individuals in whom the lesions specified occur are "visuals" or strong "auditives" much accustomed to write. On the other hand, Nos. 4 and 5 are two cases in which the same degree of agraphia may be met with, but differing by the absence of word-deafness in the one case and its presence in the other. I say *may* be, because such symptoms might not occur in a strong "visual." It will be observed also that each of the partial forms of agraphia

[36] Les Paralysies par Suggestion, Revue Scientifique, 12 Juillet, 1884.
[37] Le Langage Intérieur, p. 134.

except the first is associated either with word-deafness or with word-blindness.

### DEFECTS IN THE AUDITORY AND IN THE VISUAL WORD-CENTRES.

Since the publication in the year 1874 of an important memoir by Wernicke entitled "Der Aphasische Symptomencomplex," it has been the fashion to speak of the defects of speech due to lesions in the auditory and the visual word-centres as "sensory aphasia," in contra-distinction to that produced by damage to Broca's region, which, in accordance with then prevalent notions, was and has since been very commonly spoken of as "motor aphasia." This mode of distinguishing these defects, though it has a certain convenience and has been widely adopted, is not in accordance with my views, as I hold the latter to be as much a sensory region of the brain as the former. It would, I believe, be much better if the term "aphasia" were restricted to the defects of speech produced by lesions in Broca's region, and the term "aphemia" to those dependent upon sub-cortical lesions in the course of the pyramidal fibres, leaving the speech defects produced by lesions of the convolutions around the posterior extremity of the Sylvian fissure to be grouped as so many varieties of "amnesia." Under this latter generic name would be included many forms of speech defect due to defective recall of the auditory and the visual images of words, and produced either by lesions of the auditory and the visual word-centres themselves or of the commissures by which they are united to one another and to corresponding centres in the opposite hemisphere. In all these cases there would be more or less interference with the recall of auditory and visual images of words. And whether we call the case one of sensory aphasia or of amnesia, in each instance alike the precise degree and nature of the defect or defects would have to be settled by a systematic examination, so as to determine whether we had to do with mere diminished recollection of words, with complete loss of their auditory or visual images, or with other combinations of symptoms pointing either to partial isolation of these centres from one another or to isolation from the general auditory or visual word-centres of which they form part. It is true that such a nomenclature involves a slight inconsistency, seeing that aphasia and agraphia are also, in accordance with my views, forms of amnesia due to the non-revival of glosso-kinæsthetic and cheiro-kinæsthetic images respectively. But these kinæsthetic images, as I maintain, play only a small part in thinking processes, and neither of them is subject to independent conscious recall like the auditory and visual images of words which constitute our habitual thought counters. The inconsistency is, moreover, much less than that which is entailed by speaking of "motor aphasia" and "sensory aphasia," as though the former belonged to a radically different category and really depended upon the lesion of a motor centre. The advantage would be great of confining the term "aphasia" to its original signification, and not including under it various types of speech defects which are radically different in nature and produced by lesions in totally different cerebral regions; this is especially desirable when the objectionable generic term "sensory aphasia" can be replaced by another having a very similar general connotation.

It has been commonly supposed, and repeated over and over again by workers in Germany and France, as well as by those writing in our own language both here and in America, that Wernicke was the first to call attention to and explain that form of speech defect that is now known as "word-deafness," this particular name having been given to the condition shortly afterwards by Kussmaul. It is admitted that the companion defect of the visual centre now known as "word-blindness" had been previously recognised by several writers, though it had not been clearly explained; but "word-deafness" was supposed to have escaped observation by writers on speech defects, the individuals suffering from it having been previously thought to be insane, or at least demented.[38]

The merit, however, of having determined the region of the brain at fault in cases of word-deafness (viz., the hinder half of the upper temporal convolution, with perhaps a portion of the hinder extremity of the middle temporal convolution) clearly belongs to Wernicke; though, as I shall subsequently endeavour to show, he was far from correct in saying that the complex of symptoms resulting from such a lesion was word-deafness, paraphasia, alexia, and agraphia. This view as to the symptomatology of the lesion was founded upon too narrow a basis of observed cases with necropsies. A wider experience and knowledge now necessitates its complete revision.

That it is not correct, however, to say that word-deafness and word-blindness were not well understood till Wernicke defined their nature may be shown by the following quotation from my paper "On the Various Forms of Loss of Speech in Cerebral Diseases," published in 1869:—[39]

"Most aphasic patients can *understand perfectly what is said* to them and can follow and feel interested when they hear others read aloud. In these cases we may presume that the afferent fibres connecting the auditory centres of the medulla with the auditory perceptive centres of the cerebral hemispheres, and also these latter centres themselves, are intact, so that the spoken sounds revive their accustomed impressions in the hemispheres, these being perceived as words symbolic of things or ideas, which, being duly appreciated by the individual as they are conjured up, suggest to him the thoughts which they are intended to convey. In certain of the severe cases of aphasia, however, as in that noted by Dr. Bazire, recorded at p. 16, and in Dr. Gairdner's case (Glasgow Medical Journal, May, 1866, p. 13, and Transactions of the Philosophical Society of Glasgow, 1866), it is distinctly stated that the patient either did not gather at all, or with difficulty and imperfectly, the import of words when he was spoken to, though he could be made to understand with the utmost readiness, by means of signs and gestures. Must we not suppose in such a condition either the communication of the afferent fibres with the auditory perceptive centres is cut off, or that this centre itself, in which the sounds of words are habitually discriminated and associated with the things to which they refer, is more or less injured? In either of these cases, though the sound is not appreciated as a *word* having its definite meaning, we must not expect that there would be deafness; the sound would be still heard as a *mere sound*, only it does not call up that superadded intellectual discrimination by the ingrafting of which upon it it can alone be made to serve as a symbol of thought. Hence the individual does not adequately comprehend when spoken to, though he may be quite capable of receiving and appreciating fully the import of signs and gestures which make their impression upon his visual perceptive centres. . . . . And where the individual *cannot read* I am inclined to think this must be owing either to some lesion of the afferent fibres to the visual perceptive centre, of the visual perceptive centre itself, or of the communications between the cells of this centre and those of the auditory perceptive centre. If lesions existed in either of the first two situations the visual impression could not receive its intellectual elaboration, and consequently it could not call up its associated sound (word) in the auditory centres, and hence no meaning would be conveyed by the hieroglyphic marks of the printed or written pages. They would be to the person mere meaningless strokes, just as we have assumed that if similar defects existed in the auditory perceptive centres, or in the afferent fibres with which they were connected, the individual could not appreciate the meaning of spoken words—these would be to him mere sounds."

At the date when this paper was written I knew nothing about what I now term "kinæsthetic centres" and had not fully realised the importance of kinæsthetic functions. At this period, moreover, the notion that definite "motor centres" exist in the cerebral cortex had not been mooted. This explains the reason of the absence of any reference to the needed activity of kinæsthetic centres in the above quotation.

Turning now to the question of the symptomatology of the condition named "sensory aphasia" by Wernicke, and referred by him to a lesion of the posterior extremities of the upper and middle temporal convolutions, it should be observed that Kussmaul shortly afterwards broke up into two sets of symptoms what were originally described as a single group by Wernicke. According to Kussmaul, what he first termed "word-deafness" is the primary and essential result of destruction of the hinder extremities of the upper temporal convolutions; while what he termed "word-blindness" holds a similar essential relationship to destruction of the angular and parts of the supra-marginal gyri. This is the view now commonly held, and both varieties are spoken of as forms of "sensory aphasia," though Wernicke's original doctrine as to the very complex results of a lesion in the auditory word-centre is held at the present day by Déjerine as well as by Mirallié. They both speak of word-deafness, paraphasia, alexia, and agraphia as the results of such a lesion.

It must be admitted that the functional relations of the auditory and the visual word-centres are so intimate, and their sites so close to one another, that a lesion occupying the one is apt more or less to interfere with the functions of the other for a longer or shorter period, which may vary with the nature and abruptness of the lesion, as well as with the different endowments of individuals. Disturbance to a very marked extent of the functions of the visual word-centre as a result of a lesion in the upper temporal convolutions is,

[38] This statement has been repeated by Wyllie in his recently published work on the Disorders of Speech (p. 295), although de Wattenville had previously pointed out in Brain (1885, p. 267) that I had five years before the appearance of Wernicke's memoir fully appreciated the nature of the defect as well as that of "word-blindness."

[39] British and Foreign Medico-Chirurgical Review, 1869, pp. 40 and 42.

however, by no means so universal as Wernicke's and Déjerine's statements as to the symptomatology of this lesion would imply. Thus, out of sixteen recorded cases of "sensory aphasia" in which the lesion was pretty closely limited to the hinder part of the first and more or less of the second temporal convolutions, in only five of them is there any mention of the existence of some amount of word-blindness. Doubtless had the patients been studied more minutely (after the manner practised by Thomas and Roux[40])

FIG. 4.

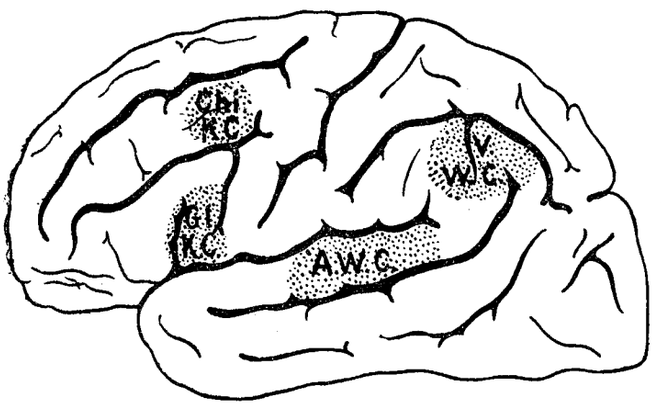



Diagram showing the approximate sites of the four word-centres in the left cerebral hemisphere.

some minor amounts of word-blindness might have been found in others; but that is not what Wernicke meant, and is not other than what might be expected if we look to the close functional and topographical relations of the two word-centres.[41]

It will subsequently be shown, moreover, that paraphasia is met with in only a little more than one-third of these cases, so that Wernicke's "symptomencomplex" does not prove to be at all in accordance with the data at present available, although his views are still adhered to by Déjerine and Mirallié.

It must, indeed, be admitted that the defects in the auditory and the visual word-centres giving rise to word-deafness and word-blindness respectively may occur separately or together, and in the latter case the defective action in the two centres may be unequally developed. Hence the very marked clinical variations that are met with as a result of lesions in the convolutions surrounding the posterior extremity of the Sylvian fissure. Another cause of clinical variations, even with similar lesions, is perhaps to be found in the different degrees of education of the persons attacked, and their consequent greater or less facility in reading and writing. Still another cause of variability is to be found in the varying individual endowments of patients in regard to the relative activity of their different word-centres—in other words, according as the patients are marked "auditives" or "visuals" respectively. Connate individual variations of this kind may give rise to notable clinical differences, even as results of similar lesions.

All that can be done here, therefore, is to point out the most common combination of symptoms, dealing with the various parts of the subject in the following order: (1) defects resulting from abnormal conditions of the left auditory word-centre; (2) defects resulting from destruction of the auditory word-centres in each hemisphere; (3) defects resulting from destruction of the auditory and the visual word-centres of each hemisphere; (4) defects resulting from isolation of the left auditory word-centre; (5) defects resulting from abnormal conditions of the left visual word-centre; (6) defects resulting from isolation of the left visual word-centre; and (7) defects resulting from combined lesions of the left auditory and visual word-centres, together with some remarks on the condition which has been named "psychical blindness" or "object blindness."

### 1. DEFECTS RESULTING FROM ABNORMAL CONDITIONS OF THE LEFT AUDITORY WORD-CENTRE.

Of all the word-centres the integrity of the auditory is of the most importance, and the defects due to different degrees of functional disability therein are the most varied, because this is the centre in which in the great majority of individuals words are first revived during thought, whether this be silent or whether it constitute the first stage in the processes of speaking or of writing. The effects of functional degradation or of partial damage to this centre may be considered first, and afterwards those resulting from its complete destruction.

(*a*) *Effects produced by functional degradation or partial damage of the left auditory word-centre.*—In the slighter degrees of damage and functional degradation we have to do with the most typical forms of verbal amnesia, in which various words fail to be recalled as they are needed in ordinary speech. The term "amnesia verbalis" applies especially to this defect, and the objections that have been raised to it on the ground that loss of auditory images constitutes only one kind of verbal amnesia are of no practical value because the two kinds of kinæsthetic images are, as I maintain, not spontaneously revivable as primary thought counters, and because it is only in rare cases that visual images of words are primarily revived. The term "amnesia verbalis" is therefore especially applicable to this particular functional disability of the auditory word-centre.

This failure to recall words is always most marked with the names of persons, places, and things, these being the most specialised units of speech. The most familiar type of this defect is that which occurs as a result of defective nutrition, either with advancing years or during convalescence from prostrating diseases. Such persons are often noticed to halt in their speech, owing to their inability to recall some such words. Occasionally, however, a similar or more marked defect of the same order occurs as a result of some more or less distinct lesion of the brain. A good example of amnesia of this type, where the "volitional" recall of names was impossible though their "sensory" recall was preserved, is thus referred to by Trousseau in his "Lectures."[42]

CASE 16.—"The patient does not speak because he does not remember the words which express ideas. You recollect the experiment that I often repeated at Marcou's bedside. I placed his nightcap on his bed and asked him what it was. After looking at it attentively he could not say what it was called, and exclaimed, 'And yet I know well what it is, but I cannot recollect.' When told that it was a nightcap he replied, 'Oh! yes, it is a nightcap.' The same scene was repeated when various other objects were shown to him. Some things, however, he named well, such as his pipe. He was, as you know, a navvy, and therefore worked chiefly with the shovel and the pickaxe, so that these are objects the names of which a navvy should not forget. But Marcou could never tell us what tools he worked with, and after he had been vainly trying to remember, when I told him it was with the shovel and the pickaxe, 'Oh! yes, it is,' he would reply, and two minutes afterwards he was as incapable of naming them as before."

As Lichtheim points out,[43] evidences of amnesia are "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." There is general agreement as to the fact that in amnesia words are lost in a tolerably definite order. First there are failures in the recall of proper names, then of other nouns; and only much more rarely of verbs, adjectives, and pronouns. Of this fact different explanations have been given. I will quote two of the best. Kussmaul says:—[44]

"The more concrete the idea the more readily is the word to designate it forgotten when the memory fails. Probably the only reason for this is that the conceptions of persons and things are more loosely connected with their names than the abstraction of their circumstances, relations, and properties are. We easily represent persons and things to ourselves without names; the image of sense is here more important than the symbol—i.e., the name—which conduces but little to our comprehension of personages or objects. More abstract conceptions, on the contrary, are attained only with the aid of words, which alone give them their exact shape. Hence verbs, adjectives, pronouns, and, still more, adverbs, prepositions, and conjunctions, possess a much more intimate relation to thought than nouns. We can conceive that the processes of

[40] Bulletin de la Société de Biologie, 22 Février, 1896.

[41] The cases to which I refer where the lesion has appeared to be pretty completely limited to the hinder part of the first or of the first and second temporal convolutions are those which in Mirallié's list (De l'Aphasie Sensorielle, 1896, p. 135) are numbered, 1, 3, 4, 6, 7, 10, 12, 14, 19, 21, 25, and 32; together with those in Amidon's list (New York Medical Journal, 1885, pp. 113 and 181) numbered 7, 14, 17 and 18. Of these, the five cases in which some amount of word-blindness co-existed are those which in Mirallié's list are numbered 12, 14, 19, 21, and 25. In Amidon's No. 11, moreover, there was the co-existence of aphasia and word-deafness, owing to simultaneous lesions in Broca's region and in the upper temporal convolutions; nevertheless the patient showed neither alexia nor agraphia. I have purposely alluded to these two lists, which include, I believe, almost all the complete cases with necropsies of "sensory aphasia" hitherto published (many of them being included in both lists), because they are both fairly accessible, and a reference to them will thus enable anyone interested in the matter to verify the correctness of my statements and criticisms with comparative facility.

[42] Translation by Bazire, 1866, Part I., p. 237.

[43] Brain, January, 1885, p. 473.

[44] Ziemssen's Cyclopædia, vol. xiv., p. 759.

excitation and the combinations in the cellular network of the cerebral cortex must be much more numerous for the creation of an abstract than of a concrete conception, and that the organic tracts which connect the former with its name must be correspondingly much more numerous than those of the concrete."

The other explanation, which may be considered as supplementary to, and not incompatible with, Kussmaul's, is that which was given by Ross. He said :—[45]

"The science of language teaches unmistakeably that the language of aboriginal man consisted almost entirely of verbs, demonstrative pronouns, and a few adverbs of time and place, and that the names of even common objects are always derivative, and consequently of much later growth than the roots themselves, and it is only what might have been expected that in the dissolution of language caused by disease nouns should disappear from the vocabulary of the patient before the parts of speech which have been first developed, and therefore most deeply organised."

In the slighter forms of amnesia the efforts at recollection of a person who is "at a loss for a word" tend also to call the visual word-centres into an incipient activity. Graves[46] placed on record an excellent illustration of this fact, though he quotes the case merely as "a remarkably exaggerated degree of the common defect of memory observed in the diseases of old age, in which the names of persons and things are frequently forgotten, although their initials are recollected."

CASE 17.—"The man was a farmer, fifty years of age, who had suffered from a paralytic attack, from which he had not recovered at the time of observation. The attack was succeeded by a painful hesitation of speech. His memory was good for all parts of speech except noun-substantives and proper names; the latter he could not at all retain. This defect was accompanied by the following singular peculiarity: he perfectly recollected the initial letter of every substantive or proper name for which he had occasion in conversation, though he could not recall to his memory the word itself. Experience had taught him the utility of having written on manuscript a list of the things he was in the habit of calling for or speaking about, including the proper names of his children, servants, and acquaintances; all these he arranged alphabetically in a little pocket dictionary, which he used as follows: if he wished to ask anything about a cow, before he commenced the sentence he turned to the letter C, and looked out the word 'cow,' and kept his finger and eye fixed on the word until he had finished the sentence. He could pronounce the word 'cow' in its proper place so long as he had his eyes fixed upon the written letters; but the moment he shut his book it passed out of his memory and could not be recalled, although he recollected its initial and could refer to it when necessary. He could not even recollect his own name unless he looked out for it, nor the name of any person of his acquaintance; but he never was at a loss for the initial of the word he wished to employ."

The fact, moreover, that in those cases where we cannot "get out" a particular word we often seem to know something of its length, and can say that it consists of about so many letters, also seems to testify to an abortive revival of the word in the visual centre. But the fact that this partial revival is not associated with full consciousness of the word and does not enable it to be written is one of considerable significance, because it seems to show how all-important in the majority of cases is the primary revival in the auditory centre, not only for the accomplishment of speech, but also for that of writing, the visual word-centre being probably called into play in writing spontaneously as well as in writing from dictation through the intermediation of the auditory word-centre.

It seems reasonably certain that in the great majority of cases in reading aloud there is first the excitation of the visual word-centre, then the passage through commissural fibres of stimuli to related portions of the auditory word-centre before the stimulus passes on to the glosso-kinæsthetic centre.[47] This affords the explanation of another peculiarity in the class of cases of which we have been speaking, as well as of others in which the amnesia has been even more profound. Many cases have, in fact, been recorded in which the patients' speech has been so disordered that they could scarcely say more than three or four consecutive words, and could perhaps utter no nouns; yet when a book is placed before them they are capable of reading aloud correctly and with ordinary facility. I have seen three such cases. One of them was a typical instance, and the following are some details concerning it.

CASE 18.—A lady, aged eighty-one years, had a slight stomach derangement, followed by diarrhœa, in the spring of 1885. She had been rather deaf for many years, but after this slight illness an amnesic difficulty in speech very gradually became more and more pronounced, and after about eighteen months became associated with marked paragraphia. Two years after the commencement of this speech trouble I found this patient capable of understanding all that was said to her in a sufficiently loud and distinct voice. She was thoroughly capable, also, of superintending her household affairs, correct in her accounts, and always knew exactly what change she ought to receive after having given money for the purchase of food or other necessaries. She made use of brief sentences, generally stopping short when she came to a noun, or she would then substitute the word "things" for any such part of speech. Thus, she said, "I will go up and see what we can," meaning that she would go upstairs and see what she could find; or "Will you have the things?" meaning some refreshments for a journey. At this time she was accustomed to read novels with large print for several hours daily. I found that she could read aloud as well as ever, and she did actually read to me, with only two or three slight mistakes in pronunciation, nearly half of a column of a newspaper. I was able to satisfy myself, also, that she understood the meaning of what she was reading; her laughter when I gravely gave her a short amusing paragraph to read left no doubt as to this point. During the next six months her condition became steadily but markedly worse. Here is a letter written to her son about the end of June, in which for the first time she omitted to put the name of the month and his Christian name at the commencement of the letter. "29, -87.—MY DEAR PAPA.—I hope you are well, and think you are well for something for the Queen, and too things for the Queen [evidently referring to two papers which had been sent to her with details of Jubilee proceedings.] Pills for the father [an intimation that she wished some pills to be sent to her.] I hope you are well.—Yours affectitory." [The surname was spelt wrongly; she had previously always signed with the Christian name alone.]

Here are a few details concerning another of these cases, in which, however, the amnesia was much less severe.

CASE 19.—A lady, aged sixty-five years, was sent to me on Feb. 18th, 1893. She had had no fit of any kind, and no pains in the head—only a feeling of "confusion in the occiput" for several years. She had suffered much from throat affections in early life, and there had been some deafness for nearly thirty years, which had slowly increased. She did not bear the ticking of a watch on either side even with contact. She heard a tuning fork on both sides with contact, and also for about two inches away from the ear on each side. Her husband died four years previously; she then became weak and suffered from dyspeptic symptoms for several weeks. There was no illness after this, but eighteen months ago she began to suffer from difficulty in expressing herself. There was no defect of articulation, but a difficulty in finding words, and this trouble had become much more marked for the last five months. There had been no unilateral weakness and there is no trace of any now. She speaks in broken sentences, omitting smaller words, and is often at a loss for a noun. Articulation is distinct. She reads from a book fluently, correctly, and with no appreciable hesitation.

A good instance of this degree of lowered excitability of the auditory word-centre was also reported by Ross.[48]

Lichtheim has likewise recorded an extremely interesting case of this type. It occurred in the person of a busy medical practitioner who met with a carriage accident and was carried home unconscious. There was paresis of the right arm and leg, and he was confined to his bed for about a week. The following are some of the particulars given by Lichtheim as to the patient's condition.

CASE 20.—Speech was much affected; the first day the patient only said "Yes" or "No," but quite appositely. Gradually more and more words returned, at first imperfectly. 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 upon this point, though he himself does not recollect 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 left his bed, copy and write from dictation.

The meaning of this ability to read aloud in such a case is that though the auditory word-centre is so much damaged as to be unable to act spontaneously (that is, under volitional stimuli), it is still capable of responding to the associational stimuli coming to it as a result of strong excitation of the visual centre.[49] Persons so affected are also quite capable of responding to sensory stimuli passing direct to the auditory centre itself—that is, they can at once repeat words uttered before them.

Another very interesting but more complicated case

[45] Loc. cit., p. 112.

[46] Dublin Quarterly Journal, 1851.

[47] Several of the writers on aphasia, however, in their diagrams assume that the stimulus passes direct from the visual word-centre to Broca's region (See Ballet, loc. cit., p. 164).

[48] On Aphasia, 1887, p. 40, Case 8. I am a little puzzled, however, in view of the simple explanation that can be given of this patient's defect, to find that in different parts of his work Ross describes cases essentially similar under different names—see the case of a boy referred to on p. 68 as an example of what he terms the "second degree of aphemia," and also his reference to another example (on p. 72), which he terms, after Kussmaul, "aphasia of recollection."

[49] Lichtheim's interpretation of this case (Brain, 1885, p. 447) is wholly different from mine. He accounts for it by supposing a damage of commissural fibres to exist, which pass between his postulated "centre for concepts" and Broca's convolution, which for him also is a motor region rather than one of sensory type. These are the cases which Lichtheim includes in his Type IV. A defect closely allied to this which I have just been considering has been described by Broadbent in a paper "On a Particular Form of Amnesia; Loss of Nouns" (Transactions of the Medico-Chirurgical Society, vol. lxvii., 1884, p. 249). His explanation is also more in accordance with that of Lichtheim than with mine.

illustrating the same degree of amnesia has been recorded by Dr. P. J. Cremen,[50] the essential details of which are as follows.

CASE 21.—The patient, a man of intemperate habits, was admitted to the Cork North Infirmary on April 10th, 1885. He was the subject of aortic and mitral disease, fully compensated, which had followed an attack of rheumatic fever in childhood. About October, 1883, he suddenly became giddy, with loss of speech, but no paralysis of limbs. He continued to work during the same day, and during five months his speech slowly improved very much. "He now commenced to do some light work, being able to make himself understood fairly well, occasionally using one word for another." About Christmas, 1884, on awaking one morning, he was surprised to find that he had lost the sight of his left eye. (This was subsequently found to be due to embolism of the central artery of the retina.) "He continued otherwise the same as regards speech, until about four weeks before admission, when, after a hard day's work, whilst in the act of holding a candle, he suddenly allowed it to drop and commenced to cry. His speech again became very imperfect and his face was slightly drawn, but there was no appreciable paralysis of limbs." Since then no change had taken place in his symptoms. On admission volitional speech was much affected. When asked of what he complained he pointed to his left temple and said he had a pain there, and on being asked if it was constant he said not. He also pointed to the left eye, and indicated that he could not see with it. His memory of names, places, and things was very defective. He did not recollect the names of his father, mother, or other near relatives. When asked to try, he made an effort to do so, sometimes repeating his own name instead, evidently knowing that he was wrong. When corrected, he repeated the name distinctly, having no difficulty whatever in articulation, the latter being very distinct. When asked to name the organs of sense he called the ear a "hair-pin," and named correctly the nose and tongue; he did not recollect the name of the eye, and called this also the "tongue"; but when corrected said, "Yes, eye; that's right." When further questioned he became confused, calling nearly everything that was shown to him "tongue." His vocabulary was, however, liable to variation from day to day. When asked to repeat the Lord's Prayer he failed to do so, but repeated another prayer instead; however, when given the first sentence he repeated it through correctly. His understanding of spoken and written language was perfect. He could repeat accurately and quickly words spoken before him. Notwithstanding this great defect of memory of words, he could read aloud clearly and distinctly, without hesitation, a page of a book from beginning to end. The following composition, in which it is impossible to discover any meaning, was written by him at my request as the history of his case: "Cork Molens.—I noscent nountg ani ammbys gosisbyoyey imitwats yab I bet yas you me sent sml me good me much cocleped." He wrote his own name and residence accurately, and could write numerals to any extent without dictation. The following will exemplify defects in writing from dictation. When asked to write the word "just" he wrote "fugl," for "subject" "supfect," for "speak" "sery," for "found" "spunt." Strange to say, when told to spell those words he did so accurately in every instance, and when asked why he did not write them he explained that he had forgotten how to make the letters. He remained in hospital for about three months, and left much improved in his speech, being able to carry on a conversation fairly well, though occasionally using one word for another. He could name objects better. Volitional writing was not improved; writing from dictation was slightly better.

Apart from the marked amnesia with preservation of ability to read aloud, this case is remarkable for the gibberish character of the patient's writing, combined with an ability to spell correctly—two characters that do not often go together. In this relation it may be mentioned that it sometimes happens that the speech of patients is entirely limited to a mere imitative repetition of words spoken in their hearing, while they are without the power of volunteering any statement—that is, their auditory word-centres respond only to direct sensory incitations, and not at all to those of an associational or volitional order. In these cases (usually included under the term "echolalia") a marked general mental impairment almost invariably co-exists.

A defect of this kind (occurring in a woman who was hemiplegic from cerebral hæmorrhage) has been recorded by Professor Béhier.[51] She was born in Italy, and had resided both in Spain and France; of the three languages she had thus acquired she had completely forgotten the Italian and Spanish, and had only retained a most limited use of French. In this latter language *she only repeated like an echo* the words pronounced in her presence, without, however, attaching any meaning to them. But in the case of a woman seen at the Salpêtrière by Bateman the mimetic tendency was much stronger. She even reproduced foreign words with which she had never been familiar. It is clear that in such a case as this there must have been a mental degradation of a much wider kind than that which occurs when the auditory word-centre alone is reduced to its lowest grade of functional activity.

[50] Brit. Med. Jour., Jan. 2nd, 1886, p. 14.
[51] Gazette des Hôpitaux, May 16th, 1867.

THE Farnham Rural District Council on April 1st granted an extra donation of £20 to the medical officer for his special services during the recent outbreak of diphtheria.

# ON FUNCTIONAL MURMURS: A FURTHER CONTRIBUTION IN ILLUSTRATION OF THE MIMICRY OF ORGANIC HEART DISEASE.

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SINCE my paper on Functional Heart Murmur appeared in THE LANCET of July 27th, 1895, I have met with a number of additional cases illustrating the difficulties attending the diagnosis of heart disease. A few of these I have selected in order to call attention once more to the frequency with which functional disturbance simulates organic mischief. The subject is of great importance, and I am the more disposed to pursue it because of the interest shown by a number of thoughtful clinical observers who noticed the paper. I ventured to refer to three varieties of functional murmur—namely, the cardio-hæmic or anæmic, the cardio-muscular or neuro-typtic (so-called), and the cardio-respiratory. To this nomenclature I am disposed to adhere, though I am quite free to admit that the explanation offered of the murmurs may not be entirely correct. In some respects the appended cases resemble closely those alluded to in my first paper; in others they differ, and chiefly in point of time that the murmurs occupy in the cardiac cycle.

CASE 1.—A man, aged fifty-two years, a life-long neurotic, who lived in dread of the development of organic disease, complained of dyspepsia, pain after food, flatulency, loss of appetite, some reduction in weight, palpitation, and a sense of general weakness. The heart rate averaged 85; the apex beat was in the fifth interspace, about half an inch to the left of the usual position. On auscultation at the apex the first sound was reduplicated; the second part of the double sound was little more than an echo, and was followed immediately by a well-marked blowing murmur, which ran up to the second sound. This post-systolic murmur was audible from the apex to the sternum and up to the lower border of the third rib, was lost over the aortic valves, and was scarcely, if at all, conducted beyond the apex towards the left. It disappeared when the patient held a deep inspiration, was intensified when he was lying on the left side, and materially diminished when on the right side. It was also diminished on resting, but did not entirely cease.

CASE 2.—A young man, aged twenty-one years, was examined by a medical man and passed for an office under a public body in June, 1996, when no murmur was detected. He was examined by another practitioner within the next few weeks, and a murmur found, and again with the same result six weeks later. He was advised to consult his own medical man, who at the first examination detected the murmur; but on seeing the patient a few days later when at rest failed to do so, and sent him to me. The patient was thin and pale, and was not conscious of palpitation or shortness of breath. He had never had rheumatism, and was able to lie on either side at night. When sitting the pulse was 126, small, regular, and compressible. When lying on the back a voluminous pulsation in the precordium was visible, with apparent displacement of the apex-beat downwards and outwards. This was, however, more apparent than real, as the nipple was three-quarters of an inch above its natural point. The apex was in the fifth interspace two and a half inches from the border of the sternum. On auscultation, when standing or sitting, a short, loud, blowing, systolic murmur could be heard at the apex and half an inch to the left of that point. This was conducted along the fifth rib to the centre of the sternum. The murmur was intensified during inspiration and was at a minimum at the end of expiration. On drawing a long breath as the end of inspiration was approached it became very high in pitch and resembled the sound caused by squeezing air through a hole in an india-rubber ball. When he held his breath at the end of a deep inspiration the heart became suddenly slower and two or three deliberate beats occurred which were unaccompanied by murmur. Fifteen minutes' rest in the recumbent position brought the heart down to 110, when the murmur could no longer be heard so long as he held his breath at the end of a deep inspiration, and the area over which the murmur was heard under other conditions was much reduced—that is to say, it was only to be detected over the immediate apex after rest.

CASE 3.—A woman, aged about twenty-eight years, was seen for headaches and nervous depression about four months