THE LUMLEIAN LECTURES ON SOME PROBLEMS IN CONNECTION WITH APHASIA AND OTHER SPEECH DEFECTS.

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[ABSTRACT.] LECTURE II.

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.

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. 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. 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-kinæsthetic 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.

These modes of accounting for the coexistence 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,¹ Hughlings Jackson,² 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.

One of the first to dwell upon the marked inequality that may be met with between these two disabilities was W. Ogle,³ to whom we owe the introduction of the term "agraphia." More than ten years previously, however, Marcé⁴ had insisted upon the independence of these two defects.

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 on. 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. Another interesting peculiarity is 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.

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 "tousjours" were so employed.

The evidence of recorded cases tends to show that agraphia is rather an effect of damage to the second frontal convolution than a result, associated with aphasia, of a lesion in Broca's region. Of the first two cases of aphasia that were recorded by Broca,⁵ 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:

L., aged 84, was admitted into the surgical ward of the Bicêtre, October 27th, 1861, under the care of Broca, eighteen months after a slight apoplectic attack, which left no paralysis, but deprived him of speech except for four or five words. Broca says: "They were French words, namely, *oui*, *non*, *trois* (for *trois*), and *tousjours*. 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 'trois' expressed all numbers, all numerical ideas. And lastly, whenever these three words were not applicable Lelong helped himself with the word 'tousjours,' 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." This patient died twelve days after his admission, and at the necropsy the remains of an old hæmorrhage were 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.

Similar cases have been recorded by Tamburini and Marchi,⁶ and Simon.⁷ In the latter, which was of traumatic origin, aphasia was associated with the presence of a spicule of bone in the left third frontal convolution.

A case has been recorded by Rosenstein,⁸ in which it is said that a hæmorrhagic softening involving the third frontal convolution, only gave rise to both aphasia and agraphia.

A woman, aged 22 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 initial lesion, the sudden irruption of a clot of blood 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.⁹ It will be of interest here, also, to refer to 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,¹⁰ but in which there was no necropsy. The patient was, as is very often the case in true agraphia, able with the left hand to write his own name, though nothing else.

Although this case 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 whose death the second left frontal convolution was found to be intact.¹¹

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.

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," "Poi-boi-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:

"Sir,—I am extremely obliged to you for the trouble you have taken about my affairs. You did not say whether (*etc.*) you have 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,

"ROBERT H. . ."

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.

In each of these cases, even when 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.

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,¹² 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.

A right-handed man, aged 36 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. His subsequent condition was as follows. There was no paralysis. He could write perfectly, either spontaneously or from dictation, and could answer written questions in writing. He understood perfectly both speech and writing, and made no mistakes either in syntax or orthography. He was, however, unable to pronounce a single syllable, or to repeat it when heard. 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 subcortical 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. For another very interesting case of aphasia in some respects similar to the last I am indebted to the kindness of Dr. Dickinson.

A sailor, aged 40 years, was admitted to St. George's Hospital under Dr. Dickinson's care, having had a fit in a neighbouring public-house. One hour and a-half 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 hemiplegic 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 understood perfectly all that was said to him. He retained the sense of music, and could whistle "God save the Queen." He eventually recovered his speech and the use of the leg, but not that of the 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."

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 Wadham¹³ is in some respects even more remarkable, and all the more convincing because it was followed by a necropsy. In it also before death the patient had regained his power of speaking. The patient was left-handed, and the lesion was found to be a large area of softening in the right hemisphere, involving part of the white substance beneath the Rolandic area, and completely destroying the island of Reil.

These cases show that the view held by authorities who maintain that destruction of the third frontal convolution alone in the leading hemisphere for speech gives rise to agraphia as well as aphasia is erroneous.¹⁴

The case reported by Wadham is further of special importance because it has been contended by Déjerine and also by Mirallié,¹⁵ 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 coexisting 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 cheiro-kinæsthetic centre.¹⁸

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.¹⁷

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 this 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.

1. *Does Destruction of Broca's Region alone entail Amnesia Verbalis?*—To this question I am disposed to give a negative answer; Déjerine, Wyllie, and Lichtheim¹⁹ take the opposite view. 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.

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 oneself 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.¹⁹ 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 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.²⁰ 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,²¹ 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, in which the patient was able to understand what she read; and a still more remarkable case recorded by Pick, with very similar lesions, 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 was the case of R. H., and two recorded cases by Broadbent,²² as well as others, pointing in the opposite direction.

Many of the cases in which there has been alexia coexisting 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,²³ which evidently influenced his opinion very much. Lichtheim²⁴ believes that many of these cases have at first word deafness, which subsequently disappears.

It is, of course, possible that this explanation may hold good for certain cases, but that it is not necessary to have recourse to it in order to explain these cases of apparently simple aphasia with which alexia coexists 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 coexistence of a more or less complete hemianæsthesia.

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 co-ordinated form to the motor centres in the cord by which the actual writing movements are effected.

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.

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 co-ordinating 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.²⁵ 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 Mirallie²⁶ to this evidence falls to the ground.

Another argument adduced by Déjerine²⁷ against the existence of a special centre for writing has been based upon an interesting case which he cites very briefly 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 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 case under the care of Déjerine however admits of an explanation quite different from what he has given to it, and which is also much more in accordance with existing knowledge—namely, that a lesion existed in

the hinder part of the hemisphere which damaged the visual word centre. 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.

On the other hand, 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 Bar²⁸ 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 be decidedly opposed to it. Another case regarded by Henschen²⁹ 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. Dutil³⁰ and is thought by Pitres to favour Exner's localisation.³¹ 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."³² 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. It can be shown 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 study of his memoir,

Fig. 3.

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.³³

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, nor of agraphia as an isolated symptom, certainly resulting from damage to some part of the visuo-kinæsthetic commissure.

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,³⁴ one of which 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.

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 two old focal lesions which were discovered lay in the supramarginal lobule over the posterior end of the Sylvian fissure, and would probably have destroyed the commissural fibres between the visual word centre and the cheiro-kinæsthetic not far from the former of these centres.

Nothing can be said as to the situation of the lesion 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.

J. S., aged 54, 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 misalled 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, misalled 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 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 JICMNO. 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 glosso-kinæsthetic centre (Fig. 2, *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 Féré,³⁵ it is easy to produce agraphia by suggestion in hypnotised persons.³⁶

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:

AGRAPHIA.		
Complete	Uncomplicated.	1. Destruction of cheiro-kinæsthetic centre. 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 audio-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).
		7. Isolation of the visual word centre from all afferent fibres.

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.

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 Symptomen-complex*, 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 contradistinction 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 subcortical 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." 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 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 radi-

cally 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.³⁷

The merit, however, of having determined the region of the brain at fault in cases of word deafness (namely, the hinder half of the upper terminal 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.

That it is not correct, however, to say that word deafness and word blindness were not well understood till Wernicke defined their nature can be seen by referring to my paper On the Various Forms of Loss of Speech in Cerebral Diseases, published in 1869.³⁸

At the date when this paper was written I knew nothing about what I now term "kinaesthetic centres," and had not fully realised the importance of kinaesthetic 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 kinaesthetic centres in the paper.

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 was 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 supramarginal 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.

Fig. 4.—Diagram showing the approximate sites of the four word centres in the left cerebral hemisphere.

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, 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 16 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 5 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 Roux³⁹) 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.⁴⁰

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.

We will now consider the most common combinations of symptoms, taking the various parts of the subject in systematic order.

1.—DEFECTS RESULTING FROM ABNORMAL CONDITIONS IN 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 constitutes the first stage in the processes of speaking or of writing.

(a) *Effects produced by Functional Degradation or Partial Damage of the Left Auditory Word Centre.*—In the slightest 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 kinaesthetic 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. 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."⁴¹

As Lichtheim points out,⁴² 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. Kussmaul says⁴³ that the ease with which a word is forgotten varies with the concreteness of the idea it expresses.

Ross's theory⁴⁴ is not incompatible with this. He points

out that as in the evolution of language nouns were the last words to appear, so in its dissolution they are the first to go.

In the slightest 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. Graves⁴³ placed on record an excellent illustration of this fact in a man who had entirely lost his memory for noun substantives and proper names, though he could remember the initial letter of every one of them.

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.⁴⁶ Hence many cases have been recorded in which the patient's speech has been so disordered that they could scarcely say more than three or four consecutive words, and could perhaps utter no nouns, in which when a book is placed before them they are capable of reading aloud correctly and with ordinary facility. I have seen three such cases, and a good instance of this degree of lowered excitability of the auditory word-centre was also reported by Ross.⁴⁷

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. He was able to read aloud almost perfectly at a time when he could hardly speak at all.

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.⁴⁸ 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 illustrating the same degree of amnesia has been recorded by Dr. P. J. Cremen,⁴⁹ which was also 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 coexists.

A defect of this kind (occurring in a woman who was hemiplegic from cerebral hæmorrhage) has been recorded by Professor Béhier.⁵⁰ 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.

REFERENCES.

- ¹ Lectures, translation by Bazire, 1866. ² Brain, vol. i, 1878, pp. 320 and 329. ³ St. George's Hospital Reports, vol. ii, 1867. ⁴ Mémoires de la Société de Biologie, 1856. ⁵ Bulletins de la Société Anatomique, 1861. ⁶ Rivista Sperimentale di Freniatria e di Medicina Legale, 1883, p. 282. ⁷ Quoted by Kussmaul, Ziemssen's Cyclopædia, vol. xiv, p. 735. ⁸ Berl. Klin. Woch., 1868, p. 182. ⁹ A case of aphasia in a boy 5 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, 2nd edition, 1890, p. 96). ¹⁰ Loc. cit., p. 237. ¹¹ Loc. cit., p. 105. ¹² Afasia e sue forme, Lo Sperimentale, 1886, livi. Obs. II., p. 270, and quoted by Prévost in the Revue Médicale de la Suisse Romande, June 30th, 1895. ¹³ St. George's Hospital Reports, vol. xx, 1868, p. 245. ¹⁴ Dally, Ann. Méd. 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 paragrammatic 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. ¹⁵ Loc. cit., p. 85. ¹⁶ 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. ¹⁷ 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." ¹⁸ Loc. cit., p. 471. ¹⁹ Kussmaul, loc. cit., p. 775. ²⁰ 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. ²¹ Fortschritte der Medizin, No. 6, 1883. ²² One of these is referred to further on under the head of "Agraphia." ²³ Lectures, translation by Bazire, pp. 229-232. ²⁴ Loc. cit., p. 467. ²⁵ The Brain as an Organ of Mind, 1880, p. 646, note. ²⁶ Loc. cit., p. 75. ²⁷ Mémoires de la Société de Biologie, 1891, p. 97. ²⁸ La France Médicale, 1878, p. 603. ²⁹ Klinische und anatomische Beiträge zur Pathologie des Gehirns, Uffers, 1890, p. 273. ³⁰ Mémoires de la Société de Biologie, 1893, p. 129. ³¹ 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 (BRITISH MEDICAL JOURNAL, February, 1892)—have in my opinion no value in this connexion. ³² Pitres attempts to get over the difficulty standing in the way of his interpretation arising from the fact that the patients were unable to copy writing in the following manner. He says (Congrès Français 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 spécialisée, 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'agraphe qui dont les membres supérieurs ne sont pas paralysés, peut encore copier, bien qu'il ne soit plus capable d'écrire spontanément." ³³ 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. ³⁴ Med. Chir. Trans., 1872, pp. 146 and 160. ³⁵ Les Paralyties par Suggestion, Revue Scientifique, July 12th, 1814. ³⁶ Ballet, Le Langage Intérieur, p. 134. ³⁷ This statement has been repeated by Wyllie in his recently published work on the Disorders of Speech (p. 295), although de Wateville 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." ³⁸ British and Foreign Medical-Chirurgical Review, 1869, pp. 40 and 42. ³⁹ Bulletin de la Société de Biologie, February 22nd, 1896. ⁴⁰ See the lists given by Mirallié (De l'Aphasie Sensorielle, 1896, p. 135), and Amidon, New York Medical Journal, 1885, pp. 113 and 181. ⁴¹ Translation by Bazire, 1866, part i, p. 267. ⁴² Brain, January, 1885, p. 473. ⁴³ Ziemssen's Cyclopædia, vol. xiv, p. 759. ⁴⁴ Loc. cit., p. 112. ⁴⁵ Dublin Quarterly Journal, 1851. ⁴⁶ 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). ⁴⁷ 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." ⁴⁸ 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, Med. Chir. Trans., vol. lxvii, 1884, p. 249. His explanation is also more in accordance with that of Lichtheim than with mine. ⁴⁹ BRITISH MEDICAL JOURNAL, January 2nd, 1886, p. 14. ⁵⁰ Gazette des Hôpitaux, May 16th, 1867.

PRESENTATION.—Dr. St. John Wright, who has just resigned the position of Medical Superintendent of St. Mary's Infirmary and Workhouse, Islington, was last week presented with a handsome silver candelabrum and illuminated address on vellum by the guardians, justices of the peace, and the indoor and outdoor staff. The nurses also presented Dr. Wright with a silver salver and an address. The presentation was made by Mr. Elliott, L.C.C., the Chairman of the Board, who paid a high tribute to the ability Dr. Wright has shown during the fifteen years he had been Medical Superintendent.

ANTIRABIC INOCULATION AT CRACOW.—In the institute for antirabic inoculations in the Lazarus Hospital, Cracow, which is under the direction of Professor O. Bujwid, the number of persons treated during 1896 was 146. Of these, 44 were bitten by animals experimentally proved to be rabid, 50 by animals shown to be so by post-mortem evidence, 52 by animals in which the disease was suspected. No death occurred.