The Lumleian Lectures

ON

SOME PROBLEMS IN CONNEXION WITH APHASIA AND OTHER SPEECH DEFECTS.

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LECTURE III.[1] (*concluded from p. 1137*).

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(5) DEFECTS RESULTING FROM ABNORMAL CONDITIONS OF THE LEFT VISUAL WORD-CENTRE.

THERE is not the same variety in the defects of speech caused by disease of the visual word-centre that is to be met with as a result of disabilities in the auditory word-centre. This is apparently due to the fact that in the great majority of persons voluntary revival of words occurs primarily in the auditory word-centre, while the visual word-centre is called into activity mainly by stronger stimuli—either by those coming to it through associational fibres or by still stronger incitations that come directly from without. It is only in comparatively rare cases, as we have seen, that the visual word-centre takes the place occupied in the great majority of individuals by the auditory word-centre, as the seat in which words are primarily revived in silent or in spoken thought. Consequently the speech defect known as "amnesia verbalis" occurs almost only as a result of a lowered functional activity in the auditory word-centre, and only in extremely rare cases (where the individual is a very strong "visual") as a result of mere lowered activity in the visual word-centre. Since a volitional stimulus is weaker than that which comes through an associational channel from a centre strongly aroused, and weaker still than that which comes to the centre directly from without, and since a lowered nutrition or diminished molecular mobility of a centre from any cause might lead only to a failure in the centre to respond to the weakest stimuli, it is to be expected that such results in relation to language would show themselves only in connexion with the centre accustomed to respond to such stimuli (namely, the auditory word-centre), and would be almost absent in connexion with the visual word-centre.

Consequently we know next to nothing about the effects of mere functional degradation in the visual word-centre—that is, of slight disabilities such as when occurring in the auditory word-centre are productive of the various degrees of amnesia verbalis already described. All that can be said is that when such functional degradation of the visual word-centre is present it would (*a*) in the rare case of the patient being an extremely strong "visual" of itself tend to produce amnesia verbalis, as was seen in a patient under the care of Charcot, whose case is referred to by Ballet[2]; or (*b*) even in an "auditive" tend greatly to aggravate any disability that may have been caused by a co-existing defect in the auditory word-centre, and also to hinder recovery therefrom; further (*c*) that where the functional defect involves both word-centres the amnesia ought to be more than usually bad and probably associated with more or less of paraphasia.

Effects produced by the destruction of the left visual word-centre.—We have to pass, then, at once to a consideration of the effects produced by destruction of the left visual word-centre, which is now generally supposed to be situated in the angular, and possibly in part of the supra-marginal, convolution. These effects will be found to vary in different individuals, just as variation was found to be the case when we had to do with the results of lesions in the left auditory word-centre.

One important difference between the effects of lesions of the visual word-centre as compared with those following upon lesions of the auditory is that speech is very little, if at all, interfered with. There is no well-marked aphasia or paraphasia, only a very slight amount of paraphasia in some of the cases.[3] That there should at times be such slight disturbance of speech is not to be wondered at if we bear in mind the close functional relationships of the visual and the auditory word-centres, and how easily therefore the functions of the latter may be disturbed by a lesion in the former.

It is often found that word-blindness is associated with right-sided homonymous hemianopsia—that is, on the same side as paresis of the limbs—for there is often no complete hemiplegia in these cases. The hemianopsia is generally due to destruction of the optic radiations of Gratiolet; but where the lesion is quite limited to the cortex these fibres will not be involved, so that hemianopsia is not a necessary accompaniment of word-blindness.

Agraphia also is a symptom sometimes present and sometimes absent in cases of word-blindness. The presence or absence of this symptom is, however, of much more fundamental importance than the presence or absence of hemianopsia. It is best, in fact, to divide cases of word-blindness into two categories—namely, (*a*) cases in which the word-blindness is associated with agraphia, and (*b*) cases in which there is no agraphia. In the latter group it is found that the individuals can write as well with the eyes closed as when they are open, and further that they are, after a brief interval, unable to read what they themselves have written.

(*a*) In the cases where agraphia is present it is commonly found that such patients are capable of writing their names correctly, although they can write nothing else. The signature is an emblem which may, by reason of its familiarity, be executed by the cheiro-kinæsthetic centre with the smallest amount of prompting; and it would appear that this prompting may come from the common visual centre in cases where the left visual word-centre is destroyed. Similarly a word-blind patient may be able to recognise his own name when he sees it, though apart from this there may be complete alexia. As Déjerine says[4]: "He recognises it by its general form, by its physiognomy, and not by the assemblage of letters of which it is composed"—just, in fact, as he would recognise a geometrical figure or any other drawing. Differences of degree are, however, met with in word-blindness. In some cases, though the patients are unable to recognise single words (word-blindness), they can recognise individual letters; whilst in others not even letters can be recognised (letter-blindness). These two forms were distinguished by Kussmaul, though he sought to make a more radical distinction between them—believing that they represented differences in kind rather than of degree. All intermediate forms, however, may be met with. Sometimes patients will recognise certain letters and not others; and in one case recorded by Batterham vowels only were recognised.

In illustration of this first group of cases, where word-blindness is associated with agraphia, I will quote one of a very typical character that has been reported by Déjerine.[5]

CASE 32.—A man, aged sixty-three years, seven years previously had a slight attack of right hemiplegia, not accompanied by speech troubles, which at the time of his entering the Bicêtre (Feb. 12th, 1890) had almost completely disappeared. Soon after his admission he found one morning that he could no longer read his newspaper. On examination it was ascertained that there was no trace of word-deafness: he easily understood all questions. He was unable to understand either print or handwriting, and could not even name a single letter of the alphabet. On the other hand, he could recognise his name and could name all objects shown to him. Right lateral hemianopsia was believed to exist. There was paraphasia both in his spontaneous speech and in his repetition of words, so that it was sometimes difficult to understand what he said. When asked to write he held the pen most awkwardly and could write nothing but his name, and that so badly as to be scarcely recognisable; and whether he attempted to write spontaneously, from dictation, or to copy he wrote only his own name, "Séjalon," in an indistinct fashion. On March 10th the initial paraphasia had almost completely disappeared, so that he could readily give particulars concerning his former life and his present condition. His writing was as bad as before, but he held the pen much better. He could not write a single word spontaneously or from dictation, nor could he copy—he made mere meaningless strokes only; but he could write from dictation a numeral or a number composed of not more than two figures. There was complete alexia, as before; he recognised his own name, but nothing else. On Nov. 5th, 1890, the

[1] Lectures I., II, and part of III. were published in THE LANCET of April 3rd, 10th, and 24th, 1897, respectively.

[2] Loc. cit., p. 161.

[3] For instance, in Nos. 44, 45, 47, 49, 50, and 52 in Mirallié's list of cases included under the heading "Cécité Verbale" (pp. 152–157).

[4] Comptes Rendus de la Société de Biologie, 1891, p. 200.

[5] Loc. cit., p. 197.

word-blindness was slightly less. He could then recognise "c" and "g" of the alphabet, but no other letters. He could not make out any word, but he recognised and pronounced numerals and numbers of not more than two figures. The agraphia was as complete as before, both for spontaneous writing, dictation, and copying. Death occurred on Nov. 20th, 1890, and at the necropsy a focus of softening having "the diameter of a five-franc piece" was found occupying the inferior three-fourths of the angular gyrus, all the rest of the cortex, including the foot of the third and also that of the second frontal convolution, being absolutely intact. Section of this left hemisphere showed that the softening extended inwards in a wedge shape as far as the posterior cornu, destroying the greater part of the optic radiations of Gratiolet. In the right hemisphere two small foci of softening of the size of a small nut were found—one in the putamen, the other in the anterior part of the thalamus, which destroyed by their union the genu of the internal capsule. (Figures of the lesion in the left hemisphere were given.)

The absolute nature of the agraphia in this case is a noteworthy feature, and may perhaps be accounted for in part by the fact that writing was a comparatively unfamiliar exercise to this man, who was a mere day labourer. Damage to the visual word-centre under such circumstances might be expected to produce its maximum results, while a similar lesion in the case of an educated man much accustomed to write might lead either to no agraphia or to a much less pronounced form of this defect. Another point of interest is the temporary nature of the paraphasia that was at first set up as a consequence of this lesion limited to a part of the angular gyrus. We can only conclude, therefore, that it was due to a sympathetic disorder established for a time in the contiguous auditory word-centre. A third point of interest is to be found in the strict localisation of the lesion to the angular gyrus and its sufficing to produce complete alexia with agraphia.

Another very similar case, and almost equally valuable, has been recorded by Sérieux,[6] of which the following is an abstract.

CASE 33.—A woman, aged seventy-three years, entered the asylum of Villejuif on Sept. 29th, 1891. At the first examination it was ascertained that the patient heard and comprehended all questions well, but that there was some amount of amnesia verbalis. Reading and writing were impossible, although vision was good and she could recognise surrounding objects. After a time, being troubled by her inability to write, she practised much with her pen. At first she could only copy the letters whose outline is most simple, such as m, n, u, o, a. Her alphabet was almost composed of these letters, and they were sometimes grouped so as to give the appearance of words — such as um, aa, monon, mono, muosi. In the month of November the following observations were made: "Intelligence normal. No motor paralysis. No appreciable word-deafness. Slight paraphasia. No object blindness. Word-blindness is almost complete; she recognises a certain number of letters only. Hemianopsia absent. Agraphia is also complete for spontaneous writing, as well as for dictation and copying." On Nov. 23rd the patient had an abrupt apoplectic attack, she became comatose, and died. At the necropsy, in addition to a recent hæmorrhage into the right hemisphere, with extravasation into the ventricles, there was found one old lesion in the left hemisphere. This was a focus of yellow softening rather larger than a five-franc piece, which involved the whole of the angular gyrus and the greater part of the supra-marginal lobule. The contiguous convolutions of the superior parietal lobule as well as the posterior extremities of the first and second temporal convolutions were slightly yellowish and atrophied. All the other convolutions of this and of the other hemisphere were healthy.

Here, although the lesion was more extensive than in the last case, we find both the word-blindness and the agraphia rather less complete. The slight paraphasia, which in this case was persistent, may have been kept up by the slight degeneration of the posterior extremities of the two upper temporal convolutions. It is expressly stated that the patient did not suffer from hemianopsia, so we must conclude that the lesion was superficial and did not extend deeply enough to involve the optic radiations. Another case in which word-blindness and agraphia were produced by a patch of softening limited to the left angular gyrus has been published by Berkhan,[7] though under a very misleading title.

(*b*) In the second group of cases word-blindness is not associated with agraphia, the individuals being able to write, and as well with the eyes closed as open. As I have already said, a precisely similar group of symptoms may be produced in a totally different manner, quite independently of destruction of the left visual word-centre. These cases will be fully considered in the next section.

The effects of destruction of the left visual word-centre are liable to vary much in different individuals in accordance with their different sensorial aptitudes and different degrees of education, just as we have found the results of destruction of the left auditory word-centre to present marked differences in different cases. The results already described under group (*a*) are those which most frequently follow when the left visual word-centre is destroyed in ordinary individuals. But suppose a similar lesion to occur in a person who is a strong "auditive" but a weak "visual," and at the same time, perhaps, an educated person who has previously been in the habit of writing much. It may happen in such a person, after the stage of learning to write has well passed, that the activity of the visual word-centre may be reduced to a minimum during the execution of such acts. We have seen reason to believe that the words about to be written become nascent first in the auditory word-centre, and it seems probable that in individuals with the endowments above mentioned this centre, rather than the visual word-centre, may be the one which acts upon and coöperates with the cheiro-kinæsthetic centre, just as we have seen that in certain persons who are strong "visuals" the visual word-centre may be capable of coöperating directly with Broca's centre for the production of articulate speech. I contend, therefore, that the preservation of ability to write in cases where there has been word-blindness and where the visual word-centre has subsequently been found to be destroyed is to be explained in this way—that is, by supposing that the auditory word-centre, instead of acting, as it usually does, by rousing the visual word-centre to conjoint action, in these cases acts directly upon the cheiro-kinæsthetic centre by way of the commissure *f f* (Fig. 5).[8] The writing thus produced may be fairly good and without mistakes, though that of other patients may show defects of a paragraphic type. The handwriting itself, too, is usually larger than that which was previously customary to the patient. There is also the peculiarity that these patients can write as well with the eyes closed as when they are open, and the still further peculiarity that they cannot subsequently read what they have written except by a manœuvre which causes a stimulus to pass in the reverse way—that is, from the cheiro-kinæsthetic centre back to the auditory word-centre in the direction *f' f'* (Fig. 5). This is brought about by passing the tip of the finger over the outlines of the letters and so reading off the result. The possibility of this mode of reading by the "tip of the finger" seems to have been first noticed in a word-blind patient by Westphall.[9]

There are only two cases with necropsies that I can bring forward in illustration of this group, and unfortunately they are not very well defined cases, being rather complicated, not only clinically, but also by reason of the lesions found at the necropsy. The first of them is also, I believe, the earliest recorded instance of any such defect. It is a remarkable case that was published long ago by Broadbent.[10] The real significance of the case was not, I believe, recognised by him at the time, nor was it by myself some years afterwards when I first endeavoured to interpret it.[11] Now I think its leading peculiarities may be explained by supposing the existence of a weak condition, with lowered excitability of the auditory word-centre, together with a destruction of the visual word-centre. The case is well recorded with full details, but the essential particulars are these.

CASE 34.—The patient was a man, aged fifty-nine years, and of considerable energy and intelligence. His illness commenced early in 1870 with sickness, vomiting, and pains in the head. He was restless and delirious, and did not know his wife for a fortnight. He gradually improved, but after this attack remained unable to read either print or writing, though he could write quite well. Twelve months later he was no longer able to write voluntarily, though he could still write correctly from dictation. Soon afterwards when he came under observation he was found to be unable to read. He could see the words, but could not understand them; he could not even recognise or name single letters; the only exception to this being that he recognised his own name, whether printed or written. His voluntary speech was free except that he was amnesic and much at a loss for names. If a hand or an article of clothing, or any other familiar object, were shown him, he was quite unable to name it, while if the name came up in conversation he spoke it without hesitation. He also immediately recognised the names of common objects when he heard them. Thus, asked the colour of a card he could not give it. "Is it blue?" "No." "Green?" "No." "Red?" "Well, that's more like it." "Orange?" "Yes, orange." A square and a circle were drawn and he was asked to name either. He could not do it, but when the circle was called a square he said, "No, but that is," pointing to the proper figure. He wrote from dictation and took notes of instructions as to the day and hour of subsequent visits (which were seen to be correct), saying he was very forgetful and might make mistakes. He would be utterly

[6] Mémoires de la Société de Biologie, 1892, p. 13.
[7] Archiv für Psychologie und Nervenkrankheiten, 1891, p. 558.
[8] It is worthy of note that both Kussmaul and Spamer seem to think that this is the ordinary way in which writing is effected (see Kussmaul, loc. cit., p. 777).
[9] Zeitschrift für Ethnologie, 1874.
[10] Transactions of the Royal Medical and Chirurgical Society, 1872, p. 162.
[11] Brain as an Organ of Mind, 1880, p. 645.

unable shortly after to read his own writing, but relied on his wife making out the notes he took. He was a vestryman, and still always attended the meetings of the vestry. He was not able to take part in the discussions as he had formerly done, but it amused him, and he added, "But I dare say I am as useful as many of them still." Broadbent adds: "He was a remarkably intelligent man, cheerful and energetic, even under his affliction. He employed in conversation a very extensive vocabulary, usually speaking fluently, sometimes stopping for want of a word (usually a name), rarely using wrong words (for which I was on the look out), the only ones I have notes of being 'soup' for 'supper,' 'nephew' for 'grandson.'" He remained under observation from May 8th to June 1st, 1879. On June 21st he had a severe apoplectic attack, due to ventricular hæmorrhage, and died within forty-eight hours. The recent extravasation had torn its way into the left temporo-sphenoidal lobe, in which there was a focus of softening, though its extent could not be ascertained in consequence of this irruption of blood. Of principal significance, however, were two old blood-clots, the size and situation of which are thus described: "One about the size and shape of an almond was closely embedded in the infra-marginal gyrus between the deep parallel sulcus on one side and the secondary small gyri on the lower wall of the fissure of Sylvius on the other, about opposite the junction of the upper third with the lower two-thirds of the descending cornu. The other was further back and on a higher level, almost exactly corresponding in situation with the posterior end of the fissure of Sylvius externally and with the junction of the descending cornu with the body of the ventricle internally; in fact, it occupied the thickness of brain substance separating the extremity of the fissure from the ventricle. It was about the size of a bean, yellow, and very tough."

It seems pretty clear that the second of these blood-clots must have been situated in the angular gyrus, whilst the first of them was situated somewhere near the middle of the upper temporal convolution, and therefore that the patient's alexia was due to destruction of the visual word-centre. Notwithstanding the damage to the upper temporal convolution and the existence of an ill-defined amount of softening in the white substance of the temporal lobe, there was obviously nothing approaching to word-deafness in this patient. The only means, therefore, of explaining his ability to write with the right hand is by supposing that his cheiro-kinæsthetic centre must have received its stimulations from the left auditory word-centre, even though the trifling verbal amnesia showed that this centre was slightly weakened.

Another case which, as it seems to me, should find its place in this group has been recorded by Osler,[12] of which the most important details are these.

CASE 35.—A man, aged seventy-two years, applied at the Philadelphia Infirmary for Disease of the Nervous System on Nov. 14th, 1888, complaining of uneasy sensations in his head. He was a healthy, vigorous looking man, perfectly intelligent, and spoke well and clearly. It was not thought at first that there was anything the matter with him beyond slight headache; but it was noticed that he had occasional difficulty in getting the word he wished, and this circumstance led to a more careful examination. For some time past he had not felt so well as usual. On Nov. 1st, while at his supper in a restaurant, he found that he could not read the daily paper. He was sure that this came on quickly, and it had been his chief annoyance, as he was an ardent politician. He had no definite headache, but complained of a diffuse uneasy sensation. Though he spoke clearly and intelligently, and uttered some sentences without interruption, replying promptly and fluently to questions and evidently understanding everything, there was very distinct speech disturbance. Thus, for some time he could not give the address of his residence. He said he knew where it was, but could not utter the words. He gave the first name of the man with whom he lived, but could not give the second. He could not name his own occupation, but said, "Keep, keep, keep; Oh, you say it for me." When told "bookkeeper," he repeated it distinctly. He occasionally misplaced words In referring to a wetting which he had spoken of he said, "Deliberate attacks of wet dress." When a printed or written page was presented to him he did not appear to comprehend the words. The word "Philadelphia" at the head of a hospital blank he read "P, r, i, n, g, r, e, k." When told that it was Philadelphia he replied, "Oh, certainly it is, I've known it for sixty-five years." His age, "72," written on a slip of paper, he read "213." He did not recognise the words "Cleveland" and "Harrison" at the top of a newspaper column, but when they were read to him he said, "I know all about them" and began making some very shrewd observations. He could write his name, but said that since his failure to see distinctly he did so with difficulty. He wrote as well with his eyes shut as when they were open, but did so with hesitation. He wrote the name of the hospital, and the words "Philadelphia Record." He could not read the words of his name after he had written them. He named objects held before him quite readily. On examination, right lateral homonymous hemianopsia was found. For the first two weeks of his stay in the hospital there was no special change. On one occasion he wrote the word "record" when told to, but after he had written it he spelled it "freedom." On Dec. 8th he talked less freely. He spoke intelligently and plainly at first, but after a few minutes it was difficult to understand what he stated. There were no additional ocular changes. The grip in the hands was equal. He walked with a somewhat tottering gait, though there was no actual paralysis. Early in January, 1889, he became distinctly weaker, duller mentally and more restless, and on the morning of Jan. 15th he was found in a semi-comatose condition, completely paralysed in the right arm, and incompletely in the right leg. He died on the following day. At the necropsy softening was found of the left supra-marginal lobule and of the white substance of the lower part of the angular gyrus; also of the white matter of the posterior parts of the first and second temporal convolutions. There was likewise complete softening of the white matter between these convolutions and the lateral ventricle (posterior horn); while another area of softening two inches in thickness and an inch in breadth was found in the white substance of the temporal lobe, which externally touched the grey matter of the third and the base of the second temporal convolutions. Concerning the focus of softening in the substance of the left hemisphere Osler says: "Posteriorly the softening did not extend behind a line drawn across the level of the parieto-occipital fissure. The white matter of the occipital lobe was firm, and the grey matter of the cuneus was uninvolved." The right hemisphere showed no lesions.

It is, of course, impossible to say how much these areas of softening may have extended during the last four or five weeks of this patient's life, when he seems to have become gradually worse; or, in other words, we cannot say how much more limited the lesion may have been during the first week that he was under observation, to which period the above detailed clinical record pertains. It seems clear, however, that the clinical symptoms were of the type now under consideration—that is to say, that there was no word-deafness, but that the patient was affected with word-blindness without agraphia. On the other hand, it is equally clear that even at the end, when the pathological lesions had attained their maximum development, the softening had not extended into the occipital lobe. We seem, therefore, driven to the conclusion that the word-blindness must have been due to the softening of the angular and the supra-marginal gyri.

What I take to be another example probably belonging to this group is to be found in a patient under the care of Charcot, whose case has been recorded in great detail by Bernard [13] It is an extremely interesting case, and here, as in that of Sérieux, previously quoted, the destruction of the visual word-centre does not seem to have been complete, as the patient could still recognise individual letters. The following are some of the principal details.

CASE 36.—A man, aged thirty-five years, after unusual excitement, was suddenly seized with right hemiplegia and loss of consciousness. The next day, on recovering consciousness, he stammered and substituted one word for another. At the end of three weeks the disturbance in speech had almost entirely disappeared; the hemiplegia had been gradually subsiding, so that he could now hold a pen and write very legibly. About this time, wishing to give some order about his business affairs, he took a pen and wrote what he wished to have done; then, thinking he had forgotten something, he asked to see the letter that he had just written, but found he was unable to read it. He had been able to write, but could not read what he had written just before. He was also unable to read printed matter. He was seen five months after the onset of his illness by Charcot, who ascertained that there was no longer any hemiplegia or aphasia of motor type, but that in addition to the alexia there was a forgetfulness of a certain number of nouns and proper names. It was observed that he could write a long letter without any notable mistakes in orthography. "I write," he said, "as if I had my eyes shut; I do not read what I write." In fact he wrote quite as well with his eyes closed. Having written his own name he was asked to read it. "I know very well," said he, "that it is my name that I have written, but I can no longer read it." Nevertheless, when it was insisted upon that he should read some written words, it was observed while he was making efforts to do so that with the tip of his right index finger he traced one by one the letters that constituted the word, and he was thus able with much trouble to say what the word was. Or else when he was striving to read he traced in the air with his finger the letters placed before him. It was found that he had much more difficulty in reading print after this fashion than ordinary writing, apparently because of his lack of practice in tracing the former kind of characters. Again, if when his eyes were closed a pen were put into his hand and the latter guided so as to make it write certain words, he was able to say immediately and without hesitation what those words were. He knew separately all the letters of the alphabet, except q, r, s, t, and especially x, y, z. Though he could not make out these latter letters when they were isolated, he nevertheless wrote them easily when they formed part of a word. Numerals he recognised well, and was able easily to perform certain simple operations; but when the multiplications were a little complex he made mistakes. This patient suffered also from right lateral hemianopsia.

In the absence of a necropsy it cannot be definitely said that this case belongs to the group now under consideration rather than to that which is next to be considered. Looking to the fact, however, that a hemiplegic condition existed at first, that there was no hemianæsthesia or hemiachromatopsia, and to the very limited nature of the clinical defects subsequently remaining, it seems to me distinctly less probable that the patient's condition was due to lesion in the occipital region than to one further forward which also involved the visual word-centre itself. If that were the case, the reading by means of kinæsthetic impressions would mean that the excitations of that centre produced by the finger tracing successively the outlines of letters must have been conveyed backwards along the commissure *f' f'* (Fig. 5) to the auditory word-centre, there arousing the corresponding auditory word memories, and subsequently passing over to Broca's centre preliminary to articulation of the words.

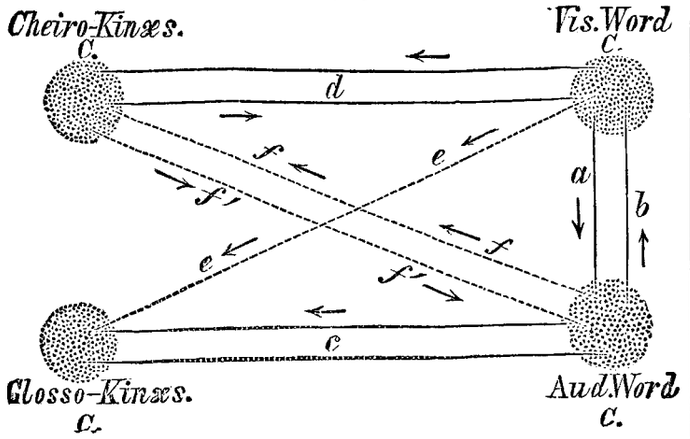
It will be observed that in each of these three cases, in which word-blindness has been present without agraphia the patients have been more or less educated persons well

[12] American Journal of Medical Science, March, 1891, p. 219.

[13] De l'Aphasie, 1885, pp. 77-90.

accustomed to write, and that in the first two, at all events, the incitations could not have come during their illness from the damaged visual word-centre. Therefore it seems only open to us to suppose that these patients were able to write spontaneously or from dictation simply because they were able to call up the familiar activity of the cheiro-kinæsthetic centre directly at the instigation of the auditory word-centre. It is well known that many voluntary movements during the

FIG. 5.



period that they are being acquired, and sometimes for long afterwards, require for their execution the active coöperation of the visual centre, though at a later period the guidance of the kinæsthetic centres may suffice for their production.

(6) DEFECTS RESULTING FROM ISOLATION OF THE LEFT VISUAL WORD-CENTRE.

Isolation of the visual word-centre is a term employed here in the same sense that isolation of the auditory word-centre was previously spoken of—it is not a complete isolation, but merely a cutting of the centre off from all its own afferent fibres, as well as from all communication with the corresponding centre of the opposite hemisphere. The clinical condition itself was first referred to by Wernicke[14] as a companion to the state described by Lichtheim as subcortical sensory aphasia or isolated speech deafness. Wernicke named this condition "subcortical alexia," while it has since been re-christened by Déjerine (who is followed by Mirallié) as "pure word-blindness." The question as to which nomenclature is to be adopted is therefore a little embarrassing. Seeing, however, that, as I have shown, each of these clinical groups of symptoms may be produced in a double way—that is, either by cortical or by subcortical lesions—it seems clear that the term "subcortical" as applied to these conditions by Lichtheim and by Wernicke would, if preserved, lead to great confusion. It seems best, therefore, to adhere to Déjerine's nomenclature and speak of "pure word-deafness" and "pure word-blindness" respectively, but with the understanding that these defects are not necessarily due to subcortical lesions.

The cases of pure word-blindness with which we are now concerned, in which the visual word-centre remains intact, are comparatively few in number. Three complete cases have been recorded with necropsies, whilst of two others the clinical details only have been published. Although the two latter cases probably belong to this type, there is, of course, the possibility that they may rather belong to the second group of cases included in the last section. I shall first give some brief account of the three complete cases, and subsequently discuss the mode in which the lesions found may be considered to be capable of giving rise to so remarkable an assemblage of symptoms.

The first of these cases to be recorded which was followed by a necropsy is one of great value; it was most carefully observed, and has been published in very full detail by Déjerine.[15] The following is the abstract that he gives by way of preface to the fuller details, which will, however, well repay a careful study.

CASE 37.—This patient was a man, aged sixty-eight years, of more than ordinary intelligence and culture. Word-blindness was complete, both verbal and literal. Musical blindness was also complete. There was preservation of ability to read figures and also to calculate. There was no trace of word-deafness, no trace of disturbance in articulate speech, and internal language was preserved. There was no object-blindness or optic aphasia; mimicry was perfect and very expressive; there was perfect preservation of spontaneous writing and of writing from dictation—the patient could thus write whole pages correctly. Writing from a copy was very difficult and defective. There was partial right lateral hemianopsia with complete hemiachromatopsia. There was integrity of motility, and of sensibility general and special, as well as of the muscular sense. The same symptoms persisted for four years. Sudden death occurred after the patient had presented for ten days total agraphia with paraphasia, without any trace of word-deafness. There had been perfect preservation of mimicry and of intelligence. At the necropsy recent lesions were found in the left hemisphere in the form of red softening in the inferior parietal lobule and in the angular gyrus. There were old lesions (*plaques jaunes*) situated in the lingual and the fusiform lobules, in the cuneus, in the white substance of the occipital lobe, and in the posterior extremity of the corpus callosum. The optic radiations were markedly atrophied. The right hemisphere was intact.

The notable features of this case are the absence of any hemiplegia; the combination of complete right lateral hemiachromatopsia with partial right hemianopsia; the absence of word-deafness or speech defect of any kind; the absolute word-blindness coupled with ability to write freely both spontaneously and from dictation, whilst ability to copy writing was very defective; and the length of time that these symptoms lasted without change. The subsequent sudden supervention of complete agraphia associated with paraphasic speech, followed by the discovery of a recent lesion in the angular gyrus and adjacent parts, were also very significant features of the case. Comments upon the old lesions found had better be reserved till some account has been given of the other two cases.

These cond case was recorded by Wyllie[16] two years later, some of the principal details being as follows.

CASE 38.—A man aged seventy-two years, formerly a clerk of the works, but then a collector of accounts for mercantile men, came as an out-patient to the Royal Infirmary of Edinburgh on Dec. 12th, 1889. About six weeks previously, when in the street, he had a very severe attack of vertigo, and ten days later a feeling of coldness and heaviness throughout the right half of the body. At this time also he discovered that he was word-blind. He could not read even his own name. At first he was even letter-blind, but before coming to the infirmary he had recovered sufficiently to be able to recognise individual letters, though he could read no single word without spelling it. On examination no right hemianæsthesia was found, but typical right hemianopsia, with marked contraction of the remaining left halves of the fields of vision. The contraction was so marked that he saw things as if looking at them through a tube. There was no agraphia; he could write well and made very few mistakes in spelling, such as he did make being evidently due not to his cerebral condition but to defective education. Although he expressed himself in writing with ease and fluency, he could read the writing only word for word, after spelling each word letter by letter. As to spoken speech, the patient expressed himself with great liveliness and volubility, being never at a loss for a word except in the case of proper names, which he was apt to forget. There was no forgetfulness of the names of things and no paraphasia. His intelligence seemed very good, but he complained of a sense of confusion in his head. This, together with his word-blindness, had made him quite unable to continue his occupation of collecting accounts. He died four years later (Jan. 23rd, 1893) of hepatic disease, with jaundice, and at the necropsy the following lesions were found: In the left hemisphere there was atrophy of the inferior surface of the occipital lobe, with a shrivelled condition of the whole lobe. Subsequent examination of sections showed softening of the white substance forming the floor of the posterior cornu of the lateral ventricle, the cornu itself being extremely dilated. The grey matter of the occipital convolutions was not involved in the softening, but the white substance was atrophied from the tip of the occipital lobe as far forwards as the cerebral peduncles. The convolutions affected were the lingual and the fusiform lobules, together with the posterior third of the gyrus hippocampus. The angular gyrus and Broca's convolution were intact.

The third case was reported in the same year by Redlich,[17] of which the following is an abstract.

CASE 39.—A man, aged sixty-five years, a copyist, had some slight cerebral symptoms for one month only in 1891. A year later he had an apoplectic attack, and on examination two days afterwards his intelligence was found to be slightly enfeebled and right lateral hemianopsia was detected. There was also slight paresis of the right arm and the right side of the face, together with slight diminution of sensibility throughout the right side of the body. There was no word-deafness, and spontaneous speech was unaffected. He had difficulty in naming objects shown to him. He could recognise the number of syllables in words. He sang perfectly both the words and the airs of popular songs. There was complete word-blindness, literal and verbal, both for print and manuscript. He could read numerals slightly. He could only write the first syllable of his name, and could not copy at all. At a second examination, one month later, the following facts were noted. Spontaneous speech was perfect, except that he confused certain words, and had sometimes a little difficulty in finding the right word. The word-blindness was still complete. He then wrote his name very correctly; he also wrote a great number of words slowly and with difficulty, both spontaneously and from dictation, sometimes forgetting a letter. The writing was tremulous, but correct. He copied with difficulty, letter by letter. Death occurred one year and a half after the date of this attack, and at the necropsy the following were the lesions in the left hemisphere: A focus of softening was found involving a great part of the calcarine fissure and the lingual and fusiform lobules, and extending to the posterior part of the occipito-parietal fissure; the cornu ammonis was atrophied;

[14] Fortschritte der Medicin, 1886, p. 463.
[15] Mémoires de la Société de Biologie, Février 27, 1892.
[16] The Disorders of Speech, 1894, p. 340.
[17] Jahrbuch für Psychiatrie, 1894, p. 242.

the cortical softening invaded the white substance of the occipital lobe and of the fusiform lobule, as well as the posterior part of the thalamus, the tail of the caudate nucleus, and the median portion of the corona radiata. Sections in series of the occipital lobe were made and examined microscopically, with the following results. At the tip of the lobe both the white and the grey substance were intact. The focus of softening corresponded with the fusiform lobule, and the inferior half of the lingual lobule. The forceps major was degenerated only in its median portion; its supero-external portion being intact. The tapetum was in part preserved. The inferior longitudinal convolution as well as the optic radiations were degenerated. The hippocampal gyrus was much altered, as well as a part of the third temporal.[18]

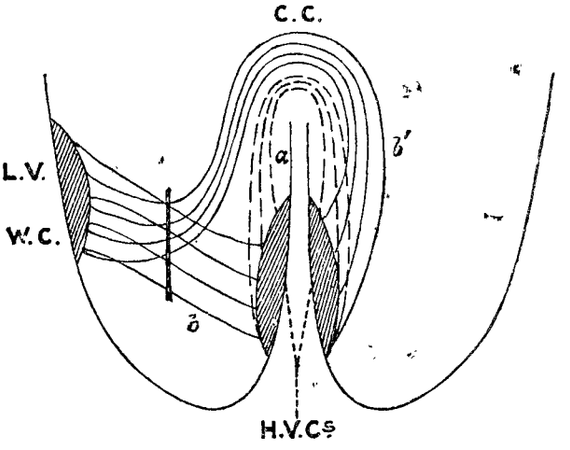
It will be observed that in each of these two cases, as in that of Déjerine, there was no motor paralysis, and only a very slight amount of right-sided hemianæsthesia. There was right hemianopsia in each, but no mention is made of hemiachromatopsia in the cases of Wyllie and Redlich, or of the ability to read words by means of kinæsthetic impressions, though both were present in the case of Déjerine.[19] It seems highly probable, however, that one if not both of these characteristics might have been met with had they been specially looked for. They both existed in one of the two incomplete cases before referred to—namely, in one observed by Gaucher, and recorded by Mirallié[20]; while in the second of these cases, recorded by Batterham,[21] though there is no mention of hemiachromatopsia, the ability to read words by aid of kinæsthetic impressions was present. Word-blindness was not complete in this case, and Batterham says: "When asked to spell out a word written in the 'round hand' of the copy books, she failed with several letters; but on being told to copy the unrecognised signs, or to run her pencil over them as if writing them, she in most cases recognised their name and significance. This experiment was repeated several times, and the patient was delighted to find that she could 'jog her memory' of letters in this way." Both these cases are well reported, and are worthy of careful study, although there is no record as to the pathological causes of the clinical condition.

The lesions found in each of the three cases where a necropsy was made have shown a striking similarity. In each there was softening and atrophy of the white substance of the occipital lobe, together with more or less damage to certain convolutions—that is, to the lingual and fusiform lobules as well as the hippocampal gyrus and the cuneus, or, speaking more generally, to some of the convolutions on the under and inner surface of the occipital lobe. It seems quite probable, however, that the lesions of the convolutions may be of little significance so long as there is the presence of extensive destruction of the white substance of the occipital lobe. Nothing more definite can be said on this subject at present. Déjerine attaches most importance to the destruction of portions of the white matter—namely, of that in which would be included the "optic radiations" of Gratiolet, the fibres proceeding from the left half-vision centre to the left visual word-centre, as well as the fibres from the right half-vision centre to this same word-centre. This he indicates in a diagrammatic figure of some complexity.

It is clear from the explanation that Déjerine advances of Case 37,[22] and also from what he says elsewhere, that he does not believe in the existence of auditory and visual word-centres in the right hemisphere in ordinary right-handed persons. On this subject he takes much the same view as Lichtheim, and now explains "pure word-blindness" in a fashion analogous to that by which the latter explained "pure word deafness"—namely, by supposing the severance from the left visual word-centre of the associational fibres connecting it with the general visual centre in each hemisphere (see Fig. 6).

I, however, believe in the existence of a visual word-centre in each hemisphere (though unequally developed), and that the two are brought into functional relation with one another by means of commissural fibres in the posterior part of the corpus callosum. I believe also that each of these visual word-centres would be in relation by other associational fibres with the half-vision centre of its own side (which both of us suppose to be commissurally connected with its fellow); and that Déjerine's form of pure word-blindness may be produced by severance of the associational fibres between the left visual word-centre and its corresponding half-vision centre, together with a lesion of the commissure between the

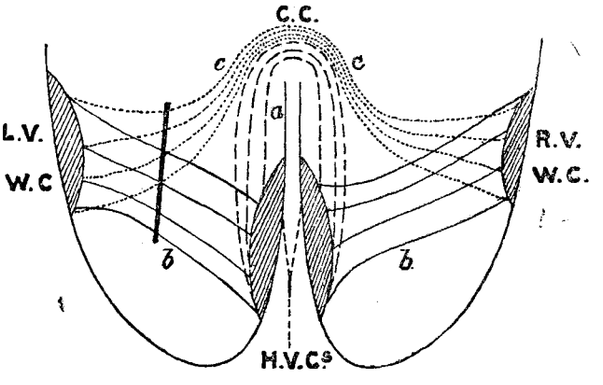
FIG. 6.



A simplified diagram representing Déjerine's view as to the mode of production of pure word-blindness. H. V. C., Half-vision centres. L. V. W. C., Left visual word-centre. C. C., Posterior extremity of corpus callosum, containing commisural fibres *(a)* connecting the half-vision centres, and also fibres *(b)* from the right half-vision centre to the left visual word-centre. (The "optic radiations" have been omitted.) The dark line indicates the site of a lesion which would cut off the left visual word-centre from the half-vision centre of each side.

two visual word-centres, in some part of its course. The isolation of the left visual word-centre would thus be complete; and brought about, moreover, in the same sort of way that I have postulated for the isolation of the auditory word-centre in the condition that Déjerine calls "pure word-deafness."

FIG. 7.



A diagram representing my view as to the mode of production of pure word-blindness. C. C., Posterior extremity of corpus callosum. *c*, *c*, Commissural fibres connecting the two visual word-centres. *b*, *b*, Fibres connecting each half-vision centre with the visual word-centre of the same side.

The commissure between the two word-centres may be damaged, in accordance with my interpretation, by the lesion in the white substance of the occipital lobe extending far enough forwards to involve them, just as Déjerine supposes those from the right half-vision centre to have been destroyed; or else by a separate lesion in the posterior part of the corpus callosum, such as was actually found in Déjerine's case, or in the part of it known as the tapetum.[23]

When we consider how one eye suffices for perfect vision, although only one-half of the proper amount of visual fibres goes to each half-vision centre, we may see all the more fully how complete and intimate must be the co-activity of these centres in vision, even when one eye only exists. That being so, it would be in vain with our present imperfect knowledge to attempt to define the actual mode of association of the half-vision centres with their outlying portions which we name

[18] Concerning some of the less familiar, though for us very important, structures mentioned above it may be well to refresh the reader's memory by quoting what Schäfer says in Quain's "Anatomy" (tenth edition, 1893, p. 129), when speaking of the posterior extremity of the corpus callosum. "The fibres," he says, "from the body and upper part of the splenium which curve over the lateral ventricle form the *tapetum*, whilst a large mass of fibres from the splenium proper curves round into each occipital lobe and is know as the *forceps major*."

[19] Déjerine is inclined to think that achromatopsia may be produced by lesions in the lingual and fusiform lobules. If the hemianopsia were complete, however, from destruction of the "optic radiations," there could be no achromatopsia.

[20] Loc. cit., p. 191. [21] Brain, 1888, p. 488.

[22] Loc. cit., pp. 87–89.

[23] The tapetum may perhaps include the commissural fibres between the two visual word-centres, and it is specially stated in Redlich's case that these fibres were in part degenerated, as also was the forceps major, which probably includes the fibres connecting the two half-vision centres with one another.

visual word-centres. All I can say is that such an arrangement as I have previously indicated seems to me to be much more in accordance with all known probabilities than that of Déjerine, who assumes that the right angular gyrus has practically no visual functions,[24] just as he assumes that the posterior half of the right upper temporal convolution has no auditory functions. What I have said towards the end of the last section but one of the first lecture, as well as what we know as to the modes in which recovery takes place in various forms of speech defect, may be considered to lend support to my view.

As I have already indicated, the two modes of producing pure word-blindness are pretty strictly comparable, *cæteris paribus*, with two modes of production of pure word-deafness. One of these forms, which for the sake of distinction may be termed the parietal type of pure word-blindness, is brought about by destruction of the left visual word-centre in persons by whom writing can be executed under the instigation and guidance of the left auditory word-centre, while the other (Déjerine's form), which might be called the occipital type, is due to isolation of the left visual word-centre from the half-vision centre of its own side as well as from the opposite visual word-centre.

At present there seems no very definite means of diagnosing these two types of pure word-blindness from one another during life. The following considerations may, however, afford some help:—

Parietal Type of Pure Word-blindness.	*Occipital Type of Pure Word-blindness.*
Possibly some right-sided paresis.	Probably no right-sided paresis, but possibly some slight right hemianæsthesia.
Speech often slightly paraphasic.	Speech not affected, or slightly amnesic only.
May be no hemianopsia or hemiachromatopsia.	Hemianopsia always, and when incomplete probably also hemiachromatopsia.

These seem to me to be the only approximations to differential characteristics that can be suggested at present, some of them being based upon the fact that destruction of the visual word-centre may be associated with a lesion in the parietal region, and consequently may be associated with some amount of right-sided hemiparesis; while in the cases of occipital type, if the lesion of the occipital lobe extends sufficiently far forwards, we may get more or less marked hemianæsthesia without any motor paralysis. The association of alexia without agraphia in a person whose speech is not appreciably interfered with, and who has slight hemianæsthesia without paralysis, would, in fact, afford strong presumptive evidence that the pure word-blindness was of the occipital type.

(7) DEFECTS RESULTING FROM COMBINED LESIONS IN THE LEFT AUDITORY AND VISUAL WORD-CENTRES.

It will have been seen from the cases already recorded that destruction of the left auditory word-centre or of the left visual word-centre alone, and especially in the last case, may be quite compatible with the preservation of a fair amount of intelligence. The result is, however, altogether different when both these centres are badly damaged at the same time. The unfortunate individuals thus affected are generally reduced to a most deplorable condition, seeing that they can usually neither speak nor write intelligibly, and that they are unable to understand the speech which they hear or the language they may see either in writing or in print. They can mostly communicate with others, and be communicated with, only by means of signs and gestures; and at the same time they must necessarily suffer a very distinct amount of mental impairment, owing to the blotting out of the principal linguistic symbols by means of which all but their most elementary thinking processes are carried on.

Although this is the kind of condition that has been met with in the great majority of these cases, yet a careful examination of their published records shows that, as in cases where there is destruction of the left auditory word-centre, so here with combined lesions of the auditory and the visual word-centres, a considerable variation is met with in different cases. These variations depend partly upon differences in the relative completeness of the lesion in one or other of these centres; and in cases where one of the centres is incompletely destroyed probably also to a considerable extent upon individual variations in original endowment—that is, upon the question whether the patients are "auditives" or "visuals," as well as in part upon their degree of education. Where the destruction of both the left word-centres has been complete, moreover, variations in symptoms may depend upon the degree of development of the corresponding centres in the opposite hemisphere.

Taking the lists given by Amidon and Mirallié, and including one case by West[25] not contained therein and two of my own, I find seventeen cases in all of this double lesion; of these, it seems desirable to exclude two—one by Chauffard, as the patient only lived three days, and one by Shaw, seeing that the patient was demented and that the clinical record is very incomplete.[26] Of the remaining fifteen cases, in three the lesion was complete in the visual and partial in the auditory word-centre (in Amidon's No. 4, Mirallié's No. 38, and in West's case); in two it was complete in the auditory and partial in the visual word-centres (in Amidon's Nos. 1 and 2); in four cases it was incomplete in both word-centres (Mirallié's Nos. 8, 23, 24, and 48); whilst in six cases the lesion was pretty complete and equal in both word-centres (Mirallié's Nos. 5, 16, 26, and 29, and my own two cases).

Looked at from another point of view—that is, as to the kind of speech defect presented by these fifteen cases—I find that speech was more or less good in two (Amidon's Nos. 1 and 2); that there was more or less marked paraphasia or actual jargon speech in seven (Amidon's Nos. 5, 16, 23, 29, 38, and 48, and West's case); and more or less complete speechlessness in six (Amidon's No. 4, Mirallié's Nos. 8, 24, and 26, and my own two cases). These very different results may at first seem very astonishing to others, as they did to me. But the more one thinks of cases presenting this double lesion, in which speech was only slightly paraphasic or fairly good, the more it seems necessary to suppose that the auditory word-centre of the right hemisphere must have been able to act upon and with the left glosso-kinæsthetic centre. These cases cannot be explained by supposing that speech was produced by the right auditory word-centre acting with the right glosso-kinæsthetic centre, because all present knowledge goes to show that this could only be brought about after a long interval, during which these centres were educated to act together. In the cases in question, on the other hand, there was evidently no such interval, seeing that the modified speech was initiated in each case just after the brain lesion occurred. It is worthy of note also that in the two cases in which speech was best preserved (Amidon's Nos. 1 and 2) the visual word-centre was only partially damaged, so that some help may also have come from its coöperation.

A few cases may now be given in illustration of the different degrees of speech defect associated with this double lesion with which we are now concerned. The first is one that was published nearly twenty years ago by Broadbent in which there was well-marked jargon-speech.[27] It is given here only in abstract, and is No. 5 of Mirallié's list.

CASE 40.—A man, aged sixty years, previously very intelligent and able to read and write well, but of intemperate habits, had had some sort of fit two weeks before admission to St. Mary's Hospital. From that time he had kept his bed and had been unable to speak intelligibly. When seen there was no hemiplegia, but slight paresis of the right side of the face and some amount of right hemianæsthesia. His speech was an inarticulate jargon and preserved the same character throughout. When questions were asked he attempted to reply, but as a rule nothing resembling a word could be detected in what he said. The voice was inflected and he appeared quite unconscious that his speech was mere gibberish. He seemed to have some idea in his mind and to think that he was giving expression to it. Besides speaking after questions, he would go on talking, addressing himself to one or another of those present, the whole of what he said being an incomprehensible ramble, though at times a distinct word or phrase would slip out. When excited the phrase, "If you please," was several times heard distinctly. He seemed to understand nothing that was said to him, and when told to shut his eyes or give his hand there was never any attempt at compliance. He was similarly quite unable to read writing. [Nothing is said about his ability to read print or his capability of writing, but he was probably incapable of doing either.] He would sit up in bed and appear to watch with interest what was going on in the ward. When the meals were brought in he looked for his portion, and ate his food

[24] One result of this supposition is that in endeavouring to account for the writing of an aphasic person with the left hand he assumes that the right hand and arm centres are stimulated from the one and only visual word-centre, the left—in fact that the left hand and arm are in part under the direction of the left hemisphere for writing movements, while for all other kinds of movements of the left hand and arm the right visual word-centre would, as usual, coöperate with the kinæsthetic centres of the same side. I must confess that this does not seem to me to be very probable.

[25] Brit. Med. Jour., vol. i., 1896, p. 1242.

[26] These are Nos. 5 and 31 of Mirallié's list.

[27] Transactions of the Royal Medical and Chirurgical Society, 1878, p. 147.

naturally. When the bowels were about to act he called the attention of the nurse by knocking on his locker and then pointing to the commode. There was never anything extraordinary in his behaviour. He died about three weeks after his admission to the hospital. At the necropsy softening of the posterior part of the left hemisphere was found in the region supplied by the third and fourth branches of the Sylvian artery. The convolutions softened were the supra-marginal lobule and the angular gyrus throughout their whole extent, and also the posterior half of the first temporo-sphenoidal gyrus, together with some portions of adjacent occipital and posterior parietal convolutions. Broca's region and the anterior half of the brain as a whole were unaffected.

This is a fairly typical case, the destruction of both the word-centres being complete and speech reduced to an unintelligible jargon. In the next case (No. 29 of Mirallié's list) the lesion was equally complete, though the derangement of speech was less marked—a very bad form of paraphasia being present rather than jargon-aphasia. It has been described at length by Déjerine,[28] but I give it here only in abstract.

CASE 41.—A man, aged sixty-three years, was admitted to the Bicêtre on July 3rd, 1890, having had an apoplectic seizure on the previous evening. The patient was of vigorous appearance and intelligent face, and was said to have been able to read and write previously to his illness. When examined on July 4th he was found to be in a semi-comatose condition, from which he could only be roused momentarily with difficulty. He remained in this condition for four days, and then gradually recovered consciousness. On July 20th he walked about the ward and showed no appreciable hemiplegia. He began to speak two or three days before, but his speech was much altered and he understood nothing of what was said to him. On saying to him, "Comment vous appelez-vous?" he replied, "Je suis et, surtout c'est-à-dire, c'est-à-dire, non, je ne peux pas po pa." "Quel métier faisiez-vous?" "Mon père se nommait, non, peux pas." The only words that he pronounced in a suitable manner were "bonjour" when one approached his bed, and "merci" when one gave him something to eat or drink. When a newspaper or some manuscript was shown to him he looked at the paper and then at the person who had given it to him, and it was evident that they had no meaning for him. When a pen was given him he held it correctly as if he were going to write, but made only meaningless marks on the paper, either when he was left to himself or when a phrase was dictated to him in a loud voice. If one gave him a phrase in manuscript to copy he copied the letters one after the other, but very badly and the words were illegible. Nevertheless, the general form of the letters was preserved, while in his spontaneous writing or that from dictation he was incapable of tracing even the rough outline of a letter. The patient recognised, however, quite well all the objects and persons around him. His hearing was also intact, and the slightest sound behind him would cause him to turn his head. On Dec. 4th, 1890, the word-deafness remained about the same, but he could now recognise one question, "Comment vous appelez-vous?" and gave his surname correctly. But to any question that anyone put to him immediately afterwards he still answered by giving his name. If after an interval one came back to him again he no longer replied as before with his name to all questions, but by words or phrases that had no relation to the question. For example: "D.: Qu'avez-vous fait hier? R.: Mon père était marchand de vins.—D.: Dans quel hôpital êtes-vous? R.: Je ... vou ... je voudrai ... non ... papapa ... tou.—D.: Quel age avez-vous? R.: J'avais cent soixante-trois.—D.: Quel métier faisiez-vous? R.: Trois ans, six six ans, trente trois, jamais trente un ans jamais, trente, trente, trente, trente-trois ans, jamais trente, trente, trente-trois ans." The patient was still unable to read, but for the last few days he had recognised his name and been able to pronounce it aloud, though he could do this with no other word. Right lateral hemianopsia seemed to be present. The agraphia remained complete as before. The patient remained in about the same condition for another three weeks; then his whole condition changed, he gradually sank into a semi-comatose state (owing to the establishment of a large area of softening in the right hemisphere), and about a month later he died. At the necropsy, in addition to a large area of recent softening in the right hemisphere, there was found a large area of yellow softening completely destroying the visual and the auditory word-centres of the left hemisphere, which also extended forwards into the parietal region and backwards into the occipital. Section of the hemisphere also showed that the lesion extended inwards through the white substance as far as the posterior cornu of the lateral ventricle.

It is worthy of note that there was here no speech at all for sixteen days from the onset of the seizure and for about eight days after the recovery of consciousness; and, looking to the completeness of the destruction of the left auditory and visual word-centres, it is difficult to suppose that the speech which was subsequently possible could have been effected without the aid of the right auditory word-centre. Much the same thing may be said in reference to the next case, although the speech trouble was more like that met with in incomplete motor aphasia. It is No. 8 of Mirallié's list, and has been recorded by d'Heilly and Chantemesse.[29] The following is a brief abstract.

CASE 42.—A woman, aged twenty-four years, became suddenly speechless on Oct. 12th, 1881. She entered a hospital at once. To all questions put to her she would repeat five or six times with different intonations, "Because, because, because." Sensibility and motility seemed perfectly intact. On Oct. 15th, in reply to a question, she answered, "Thank you, sir, I am better"; though, as Bernard points out, she was never able to repeat it. She looked very intently at the speaker, but words seemed to wake in her no image, no remembrance. When told to put her hand to her head she hesitated an instant and appeared to be trying to remember something, but she remained motionless. When the command was accompanied by a suggestive gesture she quickly obeyed. Hearing and vision were both preserved, Her power of calculation seemed preserved, as she could play écarté skilfully, making no errors as to colour or value. When asked, she called her knife, tumbler, food, wine, and plate each "du plan." When an orange was held out to her she answered all questions, "Yes, sir," and reached for the orange. Once when her soles were tickled she said, "Please don't, sir." She could neither copy, write from dictation, nor read. She died on Nov. 3rd from marasmus. There was found at the necropsy a softening from thrombosis of the fourth branch of the left Sylvian artery. This softening involved the upper posterior half of the first temporal convolution, most of the inferior parietal lobule, the supra-marginal gyrus, and some of the sigmoid gyrus. The softening affected only the cortex, and on section was seen to implicate only the extreme posterior hidden part of the insula.

It seems clear from the records of the necropsy that the left auditory word-centre was here only partially destroyed, so that perhaps we need not suppose that help came from the right auditory word-centre for the production of the few short sentences that this patient was capable of uttering.

In a patient recently under my own care, suffering from a softening of the same region of the brain, during the three weeks that elapsed before death occurred there was absolutely no speech. The following are a few notes concerning this case.

CASE 43.—A woman, aged fifty three years, was admitted to University College Hospital on April 28th, 1896. Whilst suffering from inflamed varicose veins in the left leg she began to feel ill on April 22nd, suffering from headache and irritability. On the morning of April 26th these thrombosed veins were rubbed with an embrocation. She went to bed on the night of the 26th with no change in her symptoms, but the next morning she fell from the bed in an unconscious condition. She twitched her left arm and muttered indistinctly for a time, and remained in a stuporous state up to the time of admission (9 A.M. on the 28th). She was found to be semi-conscious and paralysed on the right side, showing some irritability on examination, throwing her left arm and leg about, and occasionally uttering some inarticulate sounds. There was no cardiac bruit; the respirations were natural. Her temperature was 100·8° F. She remained in much the same condition, semi-conscious, yawning frequently, rather restless at times, till May 5th, when she became more conscious, but made no attempt to speak or even utter any sound. She could not be induced to protrude her tongue. She did not attempt to respond or show that she understood any simple request, and appeared to be perfectly word-deaf. She took no notice of written sentences held before her. The right arm was rather rigid. On May 11th she seemed more conscious than she had been but did not speak, nor did she seem to understand anything. One-fourth of albumin was found in the urine. On May 14th she began to suffer from sickness and some diarrhœa (the albumin in the urine persisting), together with lung complications, and she died about midnight on May 18th. At the necropsy the third frontal and the lower part of the ascending frontal convolution were found to be healthy, both externally and on section; but the lower part of the ascending parietal, together with the whole of the supra-marginal and angular gyri and the two upper temporal convolutions, were completely softened throughout. The softening extended to, and partly involved, the outer part of the thalamus and the corpus striatum; it also extended backwards slightly into the occipital lobe. The right hemisphere showed no focal lesion of any kind.

Here, then, there was complete loss of speech as a result of destruction of the auditory and the visual word-centres in the left hemisphere, together with some adjacent tracts of brain tissue, which lasted continuously for just over three weeks. We have seen that Déjerine's patient (Case 41) also remained speechless for sixteen days after the onset of his seizure, and then began to talk in a badly paraphasic fashion. It seem probable, therefore, that my patient might also have developed some sort of power of speaking had she lived longer.

My second case belonging to this group is one of a very extraordinary nature in many respects, the patient having lived over eighteen years after his seizure, and his speech defects having remained constant throughout almost the whole of this period. His spontaneous speech was limited to a few words, and, though he was neither word-deaf nor word-blind, both the auditory and the visual word-centres of the left hemisphere were completely destroyed. The record of this remarkable case, together with illustrations of the brain, will be published in the forthcoming volume of the Transactions of the Royal Medical and Chirurgical Society for 1897.

Time does not permit of my referring either to "object-blindness" or so-called "optic aphasia"—interesting conditions, one or other of which may sometimes be met with in association with such cases as we have just been considering, where lesions producing speech defects extend into the occipital lobes. These conditions, together with other parts of the subject, I shall hope to deal with before long in some other way.

My object in these lectures—after laying down some general principles needful, as I consider, for the proper understanding of speech defects, and culminating in my non-recognition of any distinct and separate "centre for concepts," such as

[28] Comptes Rendus de la Société de Biologie, 1891, p. 167.
[29] Bulletin de la Société Anatomique, 1882, pp. 324-338.

many postulate—has been to limit myself to a definite set of problems concerning the functional relations of the four cortical word-centres. I have striven to show that the overweening importance attached by many to the functions of Broca's centre is not justified by facts—that too many disabilities have been supposed to result from its destruction by some, and that the power of independent activity ascribed to it by others does not exist. I have tried to show the way in which these views have influenced others to deny the existence of a cheiro-kinæsthetic centre and the fallacious nature of many of the arguments on which they rely. Again, while alluding to the unsatisfactory nature of the nomenclature at present in vogue, I have tried to show that the so-called "sensory aphasia" of Wernicke has no claim, as he supposed, to an independent existence, but really includes a large number of distinct conditions, each of which has to be studied separately and in detail. The great importance of the auditory word-centre has been dwelt upon, as well as the variety of defects produced by functional and structural derangements of this region, and also by its isolation. A study of these results, taken in conjunction with that of the less varied defects resulting from disease and from isolation of the visual word-centre, led to some novel conclusions. We have seen the enforcement of the view to which we had previously been driven as to the comparative powerlessness of Broca's centre alone; we have found evidence for the existence of an unexpected possible amount of functional substitution between the visual and the auditory word-centres for the production of speech and writing respectively; and, still more surprising, we have found reason for believing that both auditory word-centres are accustomed to act upon Broca's region for the production of speech—as well as much evidence tending to favour the view that even in comparatively simple perceptional and intellectual operations very extensive cortical areas in both hemispheres of the brain are called into simultaneous activity.

I can only say, in conclusion, that my best thanks are due and are given to the College for the opportunity that has been afforded me of submitting the results of my labours to its consideration, and to you all for the patient manner in which you have listened to minute details which, I fear, must have been often wearisome and somewhat difficult to follow.

THE MALIGNANT TUMOURS OF INFANCY, CHILDHOOD, AND YOUTH.

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DURING the last half century vast additions have been made to our knowledge of the malignant neoplasms of early life. Unfortunately, most of these precious acquisitions are still buried in the archives of learned societies, where they are beyond the ken of practitioners. In this country not a single endeavour has hitherto been made to make known to the profession the general results thus attained; and in the standard text-books—even in those that deal specially with the diseases of children—little or nothing is to be found on the subject. Hence these diseases are frequently overlooked, and the real nature of the malady is often never even suspected. I was particularly struck with this deficiency; when, desiring to compare my own experience of this particular class of tumours with that of others, I found to my surprise, that no standard of comparison existed, for my predecessors had recorded nothing but isolated cases and small groups of such cases. Under these circumstances I determined to supply the deficiency by collecting cases and reviewing the chief publications in the light of my own experience. Of this undertaking the present article is the outcome. The subject having proved much larger than I at first anticipated, I have endeavoured to make my review representative rather than exhaustive.

Notwithstanding the large number of instances of malignant disease in early life lately recorded, it will be gathered from the subjoined data that fewer cases originate during infancy and childhood than at any other periods of life. Of 941 consecutive malignant neoplasms under my own observation only 25—i.e., 2·6 per cent.—commenced under the age of twenty years; of these, 8 started in the first quinquenniad, 2 in the second, 6 in the third, and 8 in the fourth. This subject is further illustrated by the following table from the Registrar-General's Report for 1886, which shows a lower proportion of cases at early periods—viz., 1·15 per cent. under twenty years of age:—

TABLE I.—*Showing the Deaths due to Malignant Disease at Different Age Periods.*

Sex.	All ages.	Under 1 year.	1-2	2-3	3-4	4-5	Total under 5 years.	5-10	10-15	15-20	20-25	25-35	35-45	45-55	55-65	65-75	75-85	Over 85	Total both sexes.
Males	5754	8	6	6	6	5	31	18	18	33	50	145	426	1095	1727	1555	591	65	16243
Females ...	10489	7	6	6	6	6	31	15	16	25	41	321	1313	2164	2922	2330	910	101	

It is sad to have to relate that during the last decennium the mortality due to malignant disease in early life, for the first time, showed a decided increase, although the increment of increase is proportionally much less than at subsequent age periods. This is illustrated by the following table from the Registrar-General's recently issued decennial supplement.

TABLE II.—*Showing the Number of Deaths from Malignant Disease in Early Life per Million Persons Living at each Age Period.*

Age periods.	Under 5.	5-10	10-15	15-20	20-25	All ages.
1861-70	13	7	7	7	17	384
1871-80	12	7	7	15	27	468
1881-90	20	10	11	20	35	589

It will be gathered from Table I. that the relative sex liability to malignant disease in early life shows no such marked differences as are noticeable at more advanced ages.

I have been greatly impressed, as I shall subsequently have occasion to point out, with the large proportion of the neoplasms of early life that obviously arise in connexion with embryonic developmental aberrations. This gives to the whole group a certain peculiarity, but it seems to me undesirable on this account to make a special morbid entity of these neoplasms, as Birch-Hirschfeld and others have proposed. I have found that the proportion of cases in which a history of heredity is recorded is considerably smaller than in the corresponding neoplasms of adult life, consequently I cannot corroborate Hutchinson's dictum that "malignant disease in young persons is generally inherited." The newly-born infants of mothers themselves the subjects of malignant disease are hardly ever thus affected.

Nearly all the malignant tumours of early life are of a sarcomatous nature; true cancers—i.e., malignant epithelial neoplasms—are at this period exceedingly rare.

Of my 941 neoplasms 806 were cancerous and 136 sarcomatous; in only one of the former did the disease begin under twenty years of age, whereas the latter furnished 24 cases.

With regard to the localisation of the disease, this depends largely upon the age at onset. Duzan[1] collected the records of 182 cases in early infancy, which were distributed as follows: Eye in 70 cases, kidney 45, testis 11, prostate 8, bones 5, tongue 5, brain 5, abdomen 5, lung 4, dura mater 4, pancreas 3, liver 2, tonsil 2, rectum 2, and the stomach in 1. During the third and fourth quinquenniads certain of the long bones, together with the superior

[1] For bibliography see end of article.