duction was in excess. There was a saccular bulging in the postero-superior wall of the canal, and caseous and cholesteatomatous matter was present in the inner end of canal, projecting from the attic of the tympanum. She came to the Ear Hospital complaining of alarming attacks of giddiness, along with great pain and constant buzzing in the affected ear. For a fortnight the giddiness had been so severe that she could not go out of doors, and she was in constant fear of falling. Even in bed she felt as if "tossing in a boat." When walking she was inclined to stagger to the affected side, and sudden motion of the head brought on these staggerings. One day while lifting a kettle from the fire she was impelled to wheel round, so that she fell on to the fire. Eight weeks previous to her coming to the hospital the giddiness was attended by severe sickness and vomiting, the attack lasting for several days. The caseous and cholesteatomatous matter was removed, and the ear was subjected to careful and prolonged antiseptic treatment, with the result that the painful symptoms passed quickly off, the giddiness subsiding more gradually. In May last she reported herself as having been quite free from the giddiness for a long time. The hearing was still very defective, and there was still slight discharge necessitating the occasional use of the syringe.

The simple exudative catarrhs of the middle ear without perforation of the tympanic membrane do not so frequently excite giddiness as the more acute purulent forms. Occasionally, however, we find striking exceptions, such as in the following case:

Giddiness and Sickness due to Simple Exudative Catarrh of Middle Ear.—Recently I had under my care a waiter from Perth, a man 40 years of age, who after a nasal catarrh became very deaf in both ears, with the development of subjective sounds in the ear, compared to that of blowing off steam. When these symptoms had continued for two weeks or thereabouts he began to have severe attacks of giddiness. On the way to consult me at the Glasgow Ear Hospital he had an alarming seizure in the street. He then became suddenly so giddy as to stagger and fall, followed immediately by sickness and vomiting; he had to be assisted into a place of shelter. I found on examination symptoms of exudative catarrh in both middle ears, which were occupied by a considerable amount of secretion, but without perforation. The deafness was extreme in both ears, while distressing subjective sounds were constantly present. I incised both tympanic membranes, giving exit to a large quantity of mucous secretion, with the result of effecting an instantaneous and marked improvement in the hearing. The giddiness also almost immediately disappeared and did not return. The use of Politzer's method of inflation for a few weeks restored entirely the hearing power. This patient informed me that he was "subject to biliousness and had a bad liver."

GIDDINESS FROM EXTENSION OF THE EAR DISEASE TO THE CRANIAL CONTENTS.

It is very important to remember that in the course of chronic purulent disease of the middle ear, and also, but much more rarely, in acute purulent disease of the middle ear, the giddiness may be due to the extension of the disease to the brain or other parts of the intracranial cavity. In such a case, however, the giddiness is associated with other grave symptoms, which point to involvement of structures more central than the organ of hearing. In this way meningitis, septic thrombosis of the sigmoid sinus, cerebral and especially cerebellar abscess may be associated with more or less disturbance of the equilibrium. It is also to be remembered, however, that grave disease of the cerebellum, such as a tumour, sometimes exists in a person who may be at the same time afflicted with an ear disease, although no connection may exist between the two. In this way there may be great disturbance of equilibrium, which at first sight is regarded as of aural origin, but subsequent events show that the disease causing the disturbance of equilibrium is entirely in the cerebellum, having possibly no connection with the ear. It is important, however, to remember that, as we have seen, marked giddiness, sickness, and vomiting may exist in connection with purulent disease of the ear, which has not extended beyond the cavities of the middle ear themselves. There is no doubt that practitioners sometimes take an unnecessarily gloomy view of the prospects of such cases, concluding, owing to these symptoms, that extension has taken place to the interior of the cranium.

THE Bishop of Bloemfontein, says the *Daily Chronicle*, is one of four Anglican prelates who were medical men. Dr. Hicks probably beats the record. He is a graduate of the University of London, as well as of Cambridge. Besides taking honours in science at Cambridge he took them at the University of London in chemistry, animal physiology, vegetable physiology, structural botany, biology, geology, and palaeontology. He took a first-class and obtained a gold medal in obstetric medicine, a third-class in forensic medicine, is a Fellow of the Royal College of Physicians, and was demonstrator of chemistry in the University of Cambridge.

THE LUMLEIAN LECTURES

ON

SOME PROBLEMS IN CONNECTION WITH APHASIA AND OTHER SPEECH DEFECTS.

*Delivered before the Royal College of Physicians,
April 8th, 1897,*

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[ABSTRACT.]

LECTURE III.

(Concluded from page 1034.)

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.

For the reasons given 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,⁴⁵ or (b) even in an "auditive" tend greatly to aggravate any disability that may have been caused by a coexisting 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 paraphasia.

Effects Produced by the Destruction of the Left Visual 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.⁴⁶ 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.

(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 says⁴⁷: "He recognises it by its general form, by its physiognomy, and not by the assemblage of letters of which it is composed." 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).

A very typical case of this character has been reported by Déjerine.⁴⁸ In this a lesion strictly localised to the left angular produced complete alexia with agraphia.

Another very similar case, and almost equally valuable, has been recorded by Sériex,⁴⁹ of which the following is an abstract:

The patient was a woman, aged 73, whose condition was as follows: 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. At the necropsy there was found a focus of yellow softening rather larger than a five-franc piece, which involved the whole of the left angular gyrus and the greater part of the supramarginal 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 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 Barkhan,⁵⁰ 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.

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 in an educated person who is a strong "auditive" but a weak "visual" the activity of the visual word centre may be reduced to a minimum in the act of writing. 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 may therefore be explained 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 $f'f'$ in Fig. 5).⁵¹ The writing thus produced may be fairly good and without mistakes, though that of other patients may show defects of a paragrammatic 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 Westphal.⁵²

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.⁵³ 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.⁵⁴ 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.

It seems pretty clear that the second of the two blood clots present in this case 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,⁵⁵ which seems to drive us to the conclusion that the word blindness must have been due to the softening of the angular and the supramarginal gyri.

What I take to be another example 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.⁵⁶ It is an extremely interesting case, and here, as in that of Sériex, previously quoted, the destruction of the visual word centre does not seem to have been complete, as the patient could still recognise individual letters.

In the absence of a necropsy it cannot, however, 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 hemichromatopsia, 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

Fig. 5.

auditory word centre, there arousing the corresponding auditory word memories and subsequently passing over to Broca's centre preliminary to articulation of the words.

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 accustomed to write, and 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 chiro-kinæsthetic centre directly at the instigation of the auditory word centre.

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 Wernicke⁵⁷ 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 rechristened by Déjerine (who is followed by Mirallié) as "pure word blindness." 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 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.

The first of the complete 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.⁵⁸ The following is the abstract that he gives by way of preface to the fuller details, which will, however, well repay a careful study.

This patient was a man, aged 68 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 2 cases.

The second case was recorded by Wyllie⁵⁹ two years later, some of the principal details being as follows:

There was word blindness, with at first letter blindness, which passed off; no hemianæsthesia, but hemianopsia, with marked contraction of the remaining halves of the visual field. There was no agraphia or paraphasia. The lesion was found to be atrophy of the white substance, from the tip of the occipital lobe to the cerebral peduncles in front. The angular gyrus and Broca's convolution were intact.

The third case was reported in the same year by Redlich,⁶⁰ and was very similar in nature.

In each of these 2 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.⁶¹ 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;⁶² while in the second of these cases, recorded by Batterham,⁶³ though there is no mention of hemiachromatopsia, the ability to read words by aid of kinæsthetic impressions was present, and word blindness was not complete.

The lesions found in each of the 3 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.

It is clear from the explanation that Déjerine advances of his case,⁶⁴ 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).

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 commissural 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.

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 centres 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 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.⁶⁵

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, *ceteris 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.

DEFECTS RESULTING FROM COMBINED LESIONS IN THE LEFT AUDITORY AND VISUAL WORD CENTRES.

Patients 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.

Taking the lists given by Amidon and Mirallié, and including one case by West⁶⁶ not contained therein and two of my own, I find 17 cases in all of this double lesion; of these, it seems desirable to exclude two—one by Chaffard, 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.⁶⁷ Of the remaining 15 cases, in 3 the lesion was complete in the visual and partial in the auditory word centre; in 2 it was complete in the auditory and partial in the visual word centres; in 4 cases it was incomplete in both word centres; whilst in 6 cases the lesion was pretty complete and equal in both word centres.

Looked at from another point of view, that is, as to the kind of speech defect presented by these 15 cases, I find that speech was more or less good in two; that there was more or less marked paraphasia or actual jargon-speech in 7, and more or less complete speechlessness in 6. 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, the visual word centre was only partially damaged, so that some help may also have come from its co-operation.

A few cases may now be referred to 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.⁶⁸

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 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.⁶⁹

In this case there was no speech at all for sixteen days from the onset of the seizure and for about eight days after the recovery of consciousness; and as the destruction of the left auditory and visual word centres was very complete, 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 recorded by d'Heilly and Chantemesse.⁷⁰

At the necropsy in this instance it was found that the left auditory word centre was 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:

A woman, aged 53, 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. 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 supramarginal 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.

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*.

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 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 over-weening 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 perceptual 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.

REFERENCES.

⁴⁵ *Loc. cit.*, p. 101. ⁴⁶ 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). ⁴⁷ *Comptes Rendus de la Société de Biologie*, 1891, p. 200. ⁴⁸ *Loc. cit.*, p. 197. ⁴⁹ *Mémoires de la Société de Biologie*, 1892, p. 13. ⁵⁰ *Archiv für Psychologie und Nervenkrank-*

heiten, 1891, p. 358. ⁵¹ It is worthy of note that both Kussmaul and Spamer seem to think that this is the ordinary way in which writing is affected (see Kussmaul, *loc. cit.*, p. 777). ⁵² *Zeitschrift für Ethnologie*, 1874. ⁵³ *Med. Chir. Trans.*, 1872, p. 162. ⁵⁴ *Brain as an Organ of Mind*, 1880, p. 645. ⁵⁵ *American Journal of Medical Science*, March, 1891, p. 219. ⁵⁶ *De l'Aphasie*, 1885, pp. 77-90. ⁵⁷ *Fortschritte der Medizin*, 1886, p. 463. ⁵⁸ *Mémoires de la Société de Biologie*, February 27th, 1892. ⁵⁹ *The Disorders of Speech*, 1894, p. 340. ⁶⁰ *Jahrbuch für Psychiatrie*, 1892, p. 242. ⁶¹ 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. ⁶² *Loc. cit.*, p. 191. ⁶³ *Brain*, 1888, p. 488. ⁶⁴ *Loc. cit.*, pp. 87-89. ⁶⁵ The tapetum may perhaps include the commissural fibres between the two visual word centres, and it is specially stated in Redlich's case that the fibres were in part degenerated, as also were the forceps major, which probably includes the fibres connecting the two half-vision centres with one another. ⁶⁶ *BRITISH MEDICAL JOURNAL*, 1896, 1, p. 1242. ⁶⁷ These are Nos. 5 and 31 of Mirallié's list. ⁶⁸ *Med. Chir. Trans.*, 1878, p. 147. ⁶⁹ *Comptes Rendus de la Société de Biologie*, 1891, p. 167. ⁷⁰ *Bulletin de la Société Anatomique*, 1882, pp. 324-338.

CÆSAREAN SECTION: RECOVERY.

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On October 15th, 1896, I saw the patient, aged 22, in consultation with Dr. Charles Knott, of Southsea. She had reached the full term of pregnancy, and had been in labour some hours. Her height was 4 feet 5 inches, forehead somewhat prominent, and teeth badly developed. She appeared a well-nourished woman, but mentally weak. The joints were normal; the limbs muscular, well developed, and free from distortion, except a slight antero-posterior curvature of both femurs. The well-marked lumbar curvature of the spine, with the convexity forwards, is exhibited in the figure. The

pelvis measured in circumference, just above the trochanters, 31 inches; the pubic symphysis was prominent and the arch compressed. On internal examination the pelvis was found considerably contracted; the antero-posterior diameter measured 2½ inches, and the transverse about 3 inches. Her mother stated that she had never had any serious illness.

The patient was at once admitted into the hospital, and my colleagues kindly met me in consultation. After an unsuccessful attempt to extract the child by craniotomy, Cæsarean section was performed without delay. Unfortunately at the commencement of the operation an accident occurred with the electric lighting of the hospital, and all the stages, after the abdominal incision, had to be done by the aid of candles. The placenta was attached to the fundus of the uterus, and its position was made out by palpation before the organ was incised. The uterus was not lifted out of the abdominal cavity, but flat sponges were placed between it and the