This is the exact point at which Sabouraud leaves us for the moment; but he promises us before long another memoir containing the results of experiments not yet completed. If we retrace the ground covered by this analysis we find (1) that the microbial origin of baldness is certain; (2) that the micro-organism of baldness is the same as that of seborrhoea; (3) that it is identical with that of the seborrhoeic plugs of the orifices of the hair follicles in alopecia areata.

To render the whole work complete, to prove that these results are definitely allied to each other, it would suffice if the following could be added to the third conclusion: The bacillus which is the cause of both seborrhoea and loss of hair acts according to its greater or less virulence under varying conditions in a manner either violent and fulminating, producing what has hitherto been known as alopecia areata, or moderately gentle, giving rise to that progressive loss of hair which in ten or twenty years ends in baldness. This last deduction Sabouraud does not give, in spite of certain favourable experiments. For prudence sake he does not wish to state it yet, because he has till now perhaps been unable to produce in an animal the true patch of alopecia areata, that is to say, the typical disease with the club-shaped hairs. There is probably, however, but a shade of difference to overcome, and we are convinced that after what has gone before Sabouraud's last scruples will soon prove to be baseless. Thus from this work, besides all the other discoveries which it comprises, we hope to see arise at the same time the key to the much-sought solution of the problem of alopecia areata in the definitely and finally established discovery of the microbe of the disease.

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THE LUMLEIAN LECTURES

ON

SOME PROBLEMS IN CONNECTION WITH APHASIA AND OTHER SPEECH DEFECTS.

*Delivered before the Royal College of Physicians,
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[ABSTRACT.]

LECTURE III.

MR. PRESIDENT AND GENTLEMEN,—At the close of the last lecture I considered the different degrees of amnesia verbalis that may result from functional defects or partial lesions of the left auditory word centre. We must now consider the effects produced by more serious lesions of this centre.

(b) *Effects produced by Destruction of the Left Auditory Word Centre.*—Where, instead of partial or mere functional defects, we have to do with more or less complete destruction of the left auditory word centre by some organic disease a correspondingly complete word deafness is produced, so that the patient is no longer able to comprehend spoken language. Spoken words become to such a patient mere meaningless sounds. Wernicke, Déjerine, and Mirallié hold that paraphasia is the kind of speech defect resulting from destruction of the auditory word centre, and that it is invariably accompanied by alexia. In the former view Wyllie concurs.

The view that I entertained on this subject some years ago¹ were that loss of articulate speech should result from destruction of the auditory word centre, just as agraphia results from destruction of the visual word centre. Ballet² and Ross³ following me expressed very similar views, though the majority have rather followed Wernicke, and regarded paraphasia as the natural accompaniment of word deafness.

Now that more cases have been observed we are in a better position for throwing light upon this point. An examination of the recorded observations in which the lesion has been limited to the hinder part of the first, and perhaps also of the

second, left temporal convulsions (without perceptibly encroaching upon the visual word centre) shows surprisingly different results in regard to the nature and degree of the speech alteration met with. In some of the cases there has been aphasia; in others a more or less marked paraphasia; whilst in two or three of the cases the defect in voluntary speech has been less marked. Thus, looking to the same sixteen cases of destruction of the auditory word centre to which I previously referred⁴ when speaking of the frequency with which word blindness and agraphia occur as additional symptoms, I find that what is described as "motor aphasia" existed in six of them; in six of the cases also some amount of paraphasia existed. In one case both aphasia and paraphasia are said to have existed; while in the three remaining cases voluntary speech seems to have been rather less affected.⁴

Thus it will be seen that we have all been more or less wrong; and I must confess that the revelation of these apparently contradictory results in the way of speech disturbance in association with word deafness surprised me not a little. The meaning of such remarkable variations demands a careful consideration.

The three cases last referred to in which voluntary speech has been least interfered with are, I believe, altogether exceptional. One of them is a case recorded by Claus,⁵ concerning which, unfortunately, there are only a few meagre details.

The patient's hearing was good, but his intelligence weak; he understood nothing said to him, and always answered wrongly while he spoke correctly. A focus of softening was found involving the first temporal convolution except its anterior third.

It seems to be Case No. 20 in the "Table of Cases of Sensory Aphasia with Lesions and Symptoms," cited by Allen Starr,⁷ and there also no further details are given. No weight, therefore, can be attached to this example, the details of which are so incomplete. The second case is one that was originally recorded by Wernicke.⁸

The patient's spontaneous speech was extremely limited, and she made frequent mistakes in words and in their pronunciation. The statement contained in Amidon's abstract that her "spontaneous speech was very good," certainly goes far beyond what there is any warrant for. The whole of the upper temporal convolution and part of the supramarginal lobule were in a state of yellow softening due to thrombosis of some of the terminal branches of the middle cerebral artery. It is worthy of note, therefore, that the auditory word centre may not have been entirely destroyed, since the second temporal convolution seems not to have been at all involved. In the previous case, reported by Claus, it should also be observed that the second temporal convolution was only very slightly involved. It is merely said that its upper border was affected ("had a brown colour").

The third case, curiously enough, has often been quoted as though it were a typical example of word deafness rather than one of an unusual character. It was recorded by Giraudeau,⁹ and some of the principal details are these.

The patient, a woman, aged 40 years, had previously enjoyed good health, though she had never menstruated. She had suffered for three months from headache. She had the greatest difficulty in understanding questions, only replying after they had been three or four times repeated. Even then, whatever subsequent questions were put to her, she followed her first idea, and her subsequent replies had no relation whatever to the questions put to her. Tactile sensibility was preserved, as well as the senses of taste and smell, and her motor power was unaffected; there was no word blindness. The psychical affection rapidly increased, and on the ninth day after her admission the word deafness was complete, and whenever she was addressed she invariably said, "I do not understand," and then began to weep. The next day she fell into a state of coma and died. At the necropsy a sarcomatous tumour of the size of a walnut was found occupying the posterior part of the first and second temporo-sphenoidal convolutions of the left hemisphere, the rest of the brain being healthy.

Here the details are fortunately more ample. Although this patient's speech was limited, there was clearly neither aphasia nor paraphasia, and this may, I think, be explained either by supposing that the lesion on the early days did not destroy the whole of the auditory word centre—which is more than possible, seeing that the lesion was a tumour and also that the word deafness was not absolute; or else that the patient was a strong "visual"—a person, therefore, in whom the excitation of Broca's centre might have been brought about directly from the visual word centre, both in spontaneous speech and in reply to written questions. And to the latter she is expressly said to have replied "verbally

or in writing, after a little reflection." This possibility might, of course, be applicable also in explanation of the other two cases.

There is an interesting point worth noticing here in reference to cases of partial word deafness, illustrating the increased power of recollection or comprehension when two or more bonds of association are simultaneously called into activity. Thus, in a case of incomplete word deafness observed by Fraenkel, when the patient was asked, "What is a fork?" he replied, "I don't know what you say." But when a fork was shown to him at the same time that its name was pronounced, he not only recognised the fork, but comprehended its name and repeated it, the visual image of the object here helping to revive the auditory image of the word. A somewhat similar case is recorded by Abercrombie in his *Inquiry into the Intellectual Powers*, seventh edition, 1837, p. 158.

This slight digression may serve to illustrate one of the two suggestions I have made in explanation of the comparatively slight defects in speech presented by the three altogether exceptional cases of word deafness to which I have above referred. But, before pursuing our attempt to explain the differences presented by the other cases, it will be well first of all to see what the results have been in two other sets of cases involving destruction of the left auditory word centre as one component of the existing lesions—that is to say, in the two next sets of cases already cited for consideration—namely: (2) the cases in which there have been combined lesions of the auditory word centres in each hemisphere: and (3) the cases in which the auditory and the visual word centres have been destroyed and severely damaged in both hemispheres.

DEFECTS RESULTING FROM LESIONS OF THE AUDITORY WORD CENTRE IN EACH HEMISPHERE.

Cases of double lesion in the upper temporal convolutions are extremely rare. Only four such cases are known to me, and concerning these clinical details in three are unfortunately extremely meagre, consisting of particulars obtained from friends or others, rather than from the examination by a skilled observer.

The first of these cases to which I will refer is one that has been recorded by Kähler and Pick,¹⁰ though the details are very scanty and of the kind above mentioned.

There was softening of both temporal lobes affecting all the convolutions except the basilar. There was complete loss of speech and hearing, without any paralysis or other motor affection. Broca's convolution was not involved.

The second case is one that has been recorded by C. K. Mills,¹¹ and although the details of the necropsy are full and all that could be desired, the patient only came under his observation a few days before her death, when she was very weak and ill, suffering from the effects of an old valvular disease of the heart.

A woman, aged 46, was admitted to the Philadelphia Hospital in August, 1891. Fifteen years before her death she had an apoplectic attack, which left her word deaf but not paralysed. Prior to the first attack of apoplexy her hearing had been good, but after it she could not by hearing understand anything that was said to her, though she could hear such sounds as a bell ringing and a clock ticking. She could write, but "sometimes mixed up her words in writing." Nine years before her death she had another and more severe apoplexy, after which her deafness increased for sounds as well as words, until it was almost total. This seizure left her also with partial left hemiplegia, chiefly affecting the arm. At the necropsy the posterior two-thirds of the left first temporal convolution was found to be shrunken to a thin strip, and the posterior fourth of the second temporal convolution was also atrophied. The other left temporal convolutions were healthy. On the right side there was an old and very extensive hæmorrhagic cyst, which had completely destroyed the first and almost completely the second temporal convolution, as well as the island of Reil and other parts in the immediate neighbourhood.

This is undoubtedly a very important case, especially from the point of view of cerebral localisation, from which standpoint Mills principally deals with it. It is interesting to find that after the patient became word deaf, owing to the first lesion on the left side, there was no word blindness, as "she often read the newspapers, and could do so with intelligence," although some would have us believe that even the destruction of Broca's centre, which is so much further away from the visual centre, causes alexia. There was, however, paraphasia and paralexia. Unfortunately there is no definite statement as to what alteration took place in the patient's speech after the lesion occurred on the right side—that is, nine years before her death. I therefore wrote to Dr. Mills

to ascertain whether he could supply any further information on this important point, and his reply is as follows: "She was admitted to the hospital some little time (probably two or three weeks) before I examined her, and my recollection is that it was reported to me that she had not spoken during that time, but more than this I cannot say....and unfortunately I have no longer the means of verifying my recollection. She certainly was not able to speak, or at least did not speak, at the time of my examination." I will merely add that, from the fact of the word deafness in this patient not being associated with word blindness, it does not necessarily follow that the paraphasic speech should have been lost—that is to say, that there should have been speechlessness, immediately after the occurrence of the lesion in the right hemisphere. There is the possibility that she might have been one of those "visuals" capable of speaking, though in a disordered fashion, by the direct action of the visual word centre upon Broca's convolution. It seems pretty clear, however, from what Dr. Mills says, that at the time she went into the hospital she was incapable of speaking. Still, it is important to bear the above-mentioned possibility in mind, as it is one to which we shall have to recur.

The third case is one that was recorded by Wernicke and Friedländer,¹² and an abstract of it has been given by Ferrier.¹³

The fourth case has been described by Pick. It is one of great importance, but as it is also one of a very exceptional nature I reserve its consideration for the present.

DEFECTS RESULTING FROM DESTRUCTION OF THE AUDITORY AND VISUAL WORD CENTRES IN EACH HEMISPHERE.

There is only one case known to me in which these combined lesions have existed in the two hemispheres, although there is another with which it will be not uninteresting to compare it where complete blindness and deafness had been produced in infancy by peripheral lesions.

The case definitely belonging to this category was reported many years ago by J. C. Shaw,¹⁴ but unfortunately in this, as in other rare cases, many essential details are omitted.

The patient was deaf and blind, as the result of symmetrical patches of cortical atrophy involving the greater part of the upper temporal and angular gyri, the marginal and the upper parietal lobules on each side. Although unable to answer questions she was in the habit of talking and screaming unintelligibly for hours at a time.

It is interesting also to note that in the celebrated case of Laura Bridgman, who was totally deaf and blind from about the end of the second year, her power of uttering articulate sounds was not very different in kind, though less in degree, than that which was met with in the case above recorded.¹⁵

In both these cases the powerlessness of Broca's centre, when unaided, for the production of anything like intelligible speech, is clearly shown.

After the narration of these additional cases, in which there was a destruction of the auditory word centre in each hemisphere we may now with more advantage return to the question why, in a certain number of the cases of destruction of the left auditory word centre alone, there is (1) an aphasic loss of speech, in others (2) paraphasia, and in others still (3) a comparatively slight impairment in voluntary speech.

My original contention was that an aphasic loss of speech is what is naturally to be expected as a result of such a lesion, if we are to suppose, as seems highly probable, that the auditory word centre in the great majority of persons holds a relation to Broca's centre similar to that which the visual word centre holds to the writing centre. It would appear, therefore, that what stands in need of explanation is the occurrence of the second and third class of cases—namely, those in which more or less marked paraphasia, or even fair speech, has existed.

The evidence in favour of the posterior half of the upper, with perhaps the posterior part of the middle, temporal convolutions being the chief, if not the exclusive, seat of the auditory word centre must be regarded as very conclusive.¹⁶ The difference in voluntary speech power presented by the cases above referred to cannot therefore be explained by the supposition that the auditory word centre has not yet been properly localised.

The various possibilities that suggest themselves to me in explanation of the differences in the degree of impairment of speech in different cases in which lesions have been found in the left auditory word centre are as follows:

(a) There is the possibility that in some of the cases in which the word deafness has been complete whilst voluntary speech has been fairly good, the auditory word centre has been rather isolated from its afferent fibres than destroyed. These would be cases in which the amount of damage to the auditory word centre itself was small—cases approaching those named by Lichtheim "isolated speech deafness," which will be subsequently considered.

(b) Then in some cases the auditory word centre may be damaged sufficiently to prevent perfect comprehension of speech, though it may still be sound enough with the aid of the visual word centre for the production of paraphasic speech. This might afford an explanation of certain cases in which there is only partial damage of the auditory word centre, together with incomplete word deafness, as in No. 7 in Amidon's list, and Nos. 2 and 48 of Mirallie's list, as well as in a case by West,¹⁷ which is not included in either of them.

(c) In other cases, where the individuals affected are strong "visuals," the paraphasic speech may be produced by the action of the visual word centre taking the place of the auditory word centre—that is, by the passage of the stimulus directly from the visual word centre to Broca's region across the commissure *c, c*, as represented in Fig. 3 (p. 900). Such an explanation may be applicable to some of the cases where the destruction of the auditory word centre has not produced word blindness as well as word deafness. And this, as we have seen, has been what has occurred in about two-thirds of the recorded cases.¹⁸

There is one extraordinary case that has been recorded by Pick¹⁹ which seems, moreover, quite incapable of being understood except by the supposition of the adequacy of the direct action of the visual word centre upon Broca's region even for the production of correct speech—as it is brought about, in fact, in all deaf-mutes who are taught to speak. This is the case of lesion in the upper temporal convolutions of each hemisphere, the consideration of which was deferred because of its altogether exceptional character. Although this patient was perfectly word deaf, he was able to speak correctly, to read correctly, and to comprehend writing perfectly, as may be gathered from the following details:

A day labourer, aged 24, was completely word deaf, and behaved like a deaf person, taking no notice of ordinary sounds near him. It was found that he only noticed loud calls, clapping or ringing of bells, and this not always readily. Yet if one shouted to him unexpectedly, he said, angrily, "don't shout at me so"; and he often said spontaneously, "I hear quite well, but I don't understand; I can hear a fly flying past me." His power of recognising airs previously known to him seemed to be also lost. His speech was perfectly correct. He spoke fluently, and only occasionally hesitated about the right word. He named objects shown to him correctly. He could not repeat words or phrases. Writing was executed slowly but quite correctly, though he could not write from dictation. With regard to his power of copying, nothing could be stated, as he could not be persuaded to make the attempt. He read aloud easily and quite correctly, and he understood both print and writing perfectly. Writing afforded the only means of communicating with him, apart from gestures. The upper parts of both temporal lobes were found to be shrunken, soft, and of a yellow colour. At the necropsy the posterior half on the upper left temporal convolution and the supramarginal gyrus were found to be softened. The island of Reil was intact. On the right side there was softening of the upper temporal convolution and a great part of the second temporal, as well as of the island of Reil together with some small foci in the lower part of the ascending frontal, and in the third frontal convolution.

It appears from the history of this patient that the first lesion occurred on the right side of the brain about ten years before his admission to the hospital. Six years later the lesion in the left hemisphere occurred, which resulted in the production of word deafness. His intelligence was distinctly weakened and his behaviour childish. It was difficult to fix his attention: he was very emotional, and spent much time in prayer. When so engaged no sort of noise seemed to disturb him, for, notwithstanding what he said as to his hearing, it appears that, like Lichtheim's patient,²⁰ he was also really very deaf to ordinary sounds. As above stated, there seems no way of explaining this case except by supposing that the left visual word centre was capable of acting directly upon Broca's centre, and without any aid from the left auditory word centre (the destruction of which had caused the word deafness four years previously) of acting so effectively that the patient was capable of reading aloud and of speaking, even without making mistakes in words. The word deafness was clearly caused by the lesion in the left auditory word centre itself, and it seems equally certain, therefore,

that this centre could not have been instrumental in enabling the patient to speak. It is superfluous, consequently, to look for a subcortical lesion.¹

(d) Another possibility in explanation of the preservation of slight or disordered speech after destruction of the left auditory word centre is that the auditory word centre of the right hemisphere acts, by means of commissural fibres passing obliquely through the corpus callosum, upon the left third frontal convolution. This is a possibility which has not, I believe, been previously suggested by any writer on speech defects, though it seems to me almost the only means of accounting for the existence of speech of a paraphasic type in those cases where, owing to its close functional and topographical relations to the visual word centre, the destruction of the left auditory word centre leads to word blindness as well as to word deafness—that is, in about one-third of the total number of such cases.

The argument in favour of the possible action of the right auditory word centre in the manner indicated is, however, even stronger than that just stated, because we shall find in a future section, when dealing with cases in which there has been more or less complete destruction of both the auditory and the visual word centres of the left hemisphere, that out of 15 of such cases, in 2 speech is reported to have been fairly good, in 7 there was jargon speech or paraphasia, whilst in 6 it was more or less markedly aphasic. But when the auditory and the visual word centres of the left hemisphere are destroyed speech, if it is to be produced by that hemisphere alone, would have to be effected solely by Broca's centre. We have, however, already seen that with double destruction of the auditory word centre, as well as with double destruction of the auditory and the visual word centres, speech has always been rendered unintelligible, except in one single case—that of Pick, above recorded. It thus seems to stand out very clearly that Broca's centre alone is not capable of acting as an instigator and adequate guide for the production of intelligible speech.

We are therefore absolutely driven to look for aid or guidance from the right hemisphere if we are to account for the production of even paraphasic speech in cases of damage to the left hemisphere of such a nature that word deafness is associated with word blindness, whether the latter be of structural or of functional origin. This same hypothesis enables us to understand the unintelligible character of the speech in three out of the four cases cited of double destruction of the auditory word centre, as well as in the case in which the auditory and the visual word centres were destroyed in each hemisphere. The old notion that intelligible speech can be initiated and produced by the influence of Broca's centre alone must be absolutely renounced.

It is well known that each olfactory bulb and that each retina is connected with both cerebral hemispheres. With regard to hearing, Lichtheim²² assumes the opposite to be the case, and Foster²³ inclines to the same view, though he adds, "the matter needs further investigation."

The probabilities are, however, that each auditory nerve is indirectly connected with each hemisphere through the intermediation of commissural fibres between the two upper temporal and other convolutions in each temporal lobe, constituting the cortical seat of this endowment—such commissural fibres forming part of the corpus callosum.²⁴ In this way, therefore, a provision exists for the conjoint activity of the two hemispheres in connection with the sense of hearing. Had it not been for the existence of such a commissural word connection, word deafness would result from absolute deafness in the right ear (whether due to labyrinthine or to middle-ear disease), owing to the left auditory word centre being cut off from all afferent impressions, as I pointed out some years since.²⁵ We know, however, that this does not occur; therefore impressions must reach this centre through the left ear, the right auditory word centre, and thence across the commissure in the corpus callosum. And the fact that in such cases the comprehension of speech seems not to be diminished would point, I think, to the probability that the right auditory word centre is about as well developed as the left on its receptive side.²⁶ And if that be so, is it to be supposed that it would remain functionally inert, save in the case of the accidental supervention of right-sided deafness?

Clinico-pathological investigations have long since taught us in an unmistakable manner that the right auditory word centre usually exercises (in right-handed persons) little influence upon the glosso-kinæsthetic centre of its own side; and now I would urge that new clinico-pathological observations should strongly incline us to believe that it must to some extent co-operate with the left auditory word centre in the excitation of Broca's region for the production of articulate speech. We have seen that instances of paraphasic speech in case of word deafness associated with word blindness are only capable of being explained in accordance with some such hypothesis, and there is the additional fact that speech has been found to be so much worse (with one exception) as to be quite unintelligible when both the auditory word centres have been destroyed.

A difficulty that presents itself in connection with such views as I have just been expressing is to account for the non-production of any amount of word deafness from disease of the right auditory word centre alone. Extremely little evidence is available upon this subject, and it is one to which most careful attention should be given in the future.²⁷

We know, of course, that in left-handed persons the predominant activity of the auditory and the visual word centres is in the right hemisphere, and that consequently in them we may get as a result of lesions in this hemisphere either word deafness or word blindness or both, just as we may get aphasia either alone or in association with left hemiplegia. Such cases of so-called "sensory aphasia" due to lesions in the right hemisphere have been recorded by Kussmaul²⁸ and also by Bernheim,²⁹ and there are doubtless many others. There was also a typical case of amnesia verbalis due to right-sided lesion recorded by Trousseau.³⁰

In what I have said hitherto I have only been contending for the existence of another special illustration of a general principle which has previously been strongly advocated by others—namely, that of the double representation of movements in each hemisphere of the brain. Indeed, for many years Hughlings Jackson has gone further still, and has advanced the speculation³¹ "that all parts of both sides of the body are represented in each half of the brain." Definite evidence on this subject was brought forward by Goltz, who, at the Physiological Congress in Basel in September, 1889, showed a dog from which almost the whole of the left hemisphere had been removed, and from whose behaviour, as demonstrated to the Congress, he felt able to announce the following important conclusions³²: "Each cerebral hemisphere is in connection with the voluntary muscles of both halves of the body. It appears, however, that the connection between one cerebral hemisphere and the muscles of the opposite half of the body is more convenient than on the same side. Hence a dog whose left hemisphere is destroyed prefers to use the left paw, because the will impulses from the right hemisphere flow by more convenient paths to the left limbs." He showed that sensibility was also present, and only somewhat impaired, in the right limbs of this dog. On the death of the animal the brain was removed, and the absence of the left cortex was confirmed by Langley and Grünbaum.³³ So far as the production of movements is concerned, these results are harmonious with the well-known results obtained by Franck and Pitres.³⁴

There is likewise actual evidence tending to show that in man also all movements are to some extent represented in each hemisphere of the brain.³⁵ In support of this double cerebral representation there is the well-known fact that in hemiplegia some amount of paresis exists also on the opposite side of the body (that is, on the side of the brain lesion). There is also the well-known fact that in cases of large lesions in one hemisphere occurring in early life defects of sensibility are often even more thoroughly recovered from than the motor defects.³⁶

What has already been said tends to show that bilateral arrangements of different kinds exist in the brain more or less analogous to that which I now postulate, namely, that Broca's centre is connected with both auditory word centres—very intimately with the centre of the same side and less intimately with that of the opposite side. The same is probably true of the half-vision centres of either side. It would seem, therefore, as if in each case we have double auditory or double visual centres exercising their influence

upon a kinæsthetic centre in the left hemisphere, in which speech movements have been registered in the one case and writing movements in the other.

The existence of the kind of nervous arrangements that I have been endeavouring to indicate in the two hemispheres would clearly necessitate the presence of numerous fibres in the corpus callosum having an oblique direction, in addition to those that are more directly transverse. Sherrington has found³⁷ in contradistinction to Meynert's views that not only similar but dissimilar parts of the two hemispheres are connected through this commissure. In regard to some inquiries of mine concerning this subject Sherrington writes: "My evidence was in part, as you suggest, the obliquity of the fibres, and also in part the spreading of them.....I found the degenerations from even circumscribed lesions of the fronto-parietal cortex spread considerably, especially after crossing the corpus callosum. I saw nothing at all incompatible with what you urge, but anatomically everything in favour of it."

The fact pointed out by Marshall³⁸ that the bulk of the corpus callosum in proportion to the size of the brain is twice as great in man as in manlike apes such as the chimpanzee, may well be due to the acquirement of language by the former.

DEFECTS RESULTING FROM ISOLATION OF THE LEFT AUDITORY WORD CENTRE.

A clinical condition is produced by the isolation of the left auditory word centre—that is, by the cutting it off from all its afferent fibres, which was first described by Lichtheim in 1884³⁹ upon the basis of a single case of word deafness that seemed to differ from all others that had been previously recorded. To this condition Lichtheim gave the name of "isolated speech deafness." It has been spoken of also by Wernicke as "subcortical word deafness," whilst lately a similar condition has been described by Déjerine (who seems to have been oblivious or unaware of Lichtheim's case) under the name of "pure word deafness." The state itself is simple enough, though an extremely interesting one. It is the condition of a partially deaf man, the deafness being not general, but limited to word deafness. The patient can hear ordinary sounds, though more or less badly, he can talk correctly, can write correctly, can read aloud, and understand what he reads. His three disabilities are that he is unable to comprehend spoken words, and that he is consequently unable either to repeat words or to write from dictation.

I cannot agree with the pathology of the affection as given by Lichtheim,⁴⁰ or with that advanced by Déjerine. The former attributes it to a lesion in the white matter of the temporal lobe cutting off the afferent fibres of both auditory nerves on their way to the left auditory word centre; while the latter speaks only of a severance of one set of afferent fibres proceeding to this centre.⁴¹

In regard to these explanations it may be said that the view of Déjerine is certainly insufficient, since, as I have already observed, it is well known that the cutting off of the afferent impressions going to the brain from the right ear alone does not give rise to word deafness. Lichtheim's explanation, on the other hand, is unsupported by any anatomical facts showing the distribution that he assumes to exist, which would really be that of a semi-decussation of the auditory nerves, since, after speaking of his imaginary arrangement on the left side of the brain, he adds in a footnote, "A similar distribution must obtain in the right hemisphere."

There was no necropsy in Lichtheim's case of "isolated speech deafness," and from the particulars given it seems clear that the patient was generally rather deaf, altogether apart from his word deafness. It seems to me probable that there was pretty complete deafness on the right side, cutting off the left auditory word centre from its proper incitations, whilst the slight sudden brain attack that is recorded to have occurred in June, 1882, may in some way have destroyed the commissural fibres connecting the left with the right auditory word centre. This view has since been rather confirmed by the record of another case by Sériex,⁴² in which it is definitely stated that the patient was absolutely deaf on the right side, as a sequence of an old otitis.

Strangely enough it has been found that a very similar

clinical combination may be produced in quite a different way, as may be seen by reference to a very remarkable case, previously cited from Pick, of destruction of the auditory word centre in each hemisphere.

These are the only 3 cases of "isolated word deafness" known to me.¹³ It will be seen from what I have said that this rare group of symptoms is capable of being induced in two very different modes—just as we shall find subsequently that what Déjerine has called "pure word blindness" may also be produced in two different modes, and that the modes are analogous in the two cases. Pick was fully aware of the very unusual nature of his case. He recognised that it could not be a case of subcortical word deafness of Lichtheim's and Déjerine's type, though he was not prepared to advance any other interpretation. Mirallic, however, has been so very uncritical as to cite it¹⁴ as an example of Déjerine's type, notwithstanding the fact of the existence of the double cortical lesions occupying the sites of the auditory word centres.

REFERENCES.

- ¹ *The Brain as an Organ of Mind*, 1880, p. 685. ² *Le Langage Intérieur*, 1886, pp. 91 and 166. ³ *On Aphasia*, 1887, p. 119. ⁴ Excepting that I omit Mirallic's Case 10, and include instead his Case 2, where the only lesion beyond the destruction of the first temporal convolution was a very minute "plaque jaune," no more than a millimetre square, situated in the upper border of the foot of the third frontal convolution. ⁵ Numbers of the cases of "sensory aphasia" hitherto recorded are comparatively valueless for the purpose of any critical study of their symptoms, either on account of the nature, extent, and complication of the lesions, or from the paucity of the clinical details, or perhaps from two or three of these causes in combination. Among such more or less vague cases I include thirteen in Mirallic's list, and three in Amidon's list. In four other cases there was a lesion in Broca's centre as well as in the auditory word centre. ⁶ *Der Irrsinnfreund*, No. 6, 1883. ⁷ *Brain*, 1890, p. 100. ⁸ *Der aphasische Symptomen-Complex*, 1874, p. 43. ⁹ *Revue Médicale*, 1882, p. 448. ¹⁰ *Vierteljahrsschrift für praktische Heilkunde*, No. 1, 1879, p. 6. ¹¹ *Philadelphia University Medical Magazine*, November, 1891; and *Brain*, 1891, p. 468. ¹² *Fortschritte der Medizin*, Band i. No. 6, 1883. ¹³ *Lectures on Cerebral Localisation*, 1890, p. 89. ¹⁴ *Archives de Médecine*, New York, 1882, p. 80. ¹⁵ *Loc. cit.*, p. 634. ¹⁶ On this subject see Ferrier on *Cerebral Localisation*, 1890, and C. K. Mills in *Brain*, 1891. ¹⁷ *BRITISH MEDICAL JOURNAL*, i, 1885, p. 1242. ¹⁸ The next explanation is, however, equally applicable to this class of cases as well as to those in which word blindness coexists with the word deafness. ¹⁹ *Archiv für Psychologie*, 1892, p. 909. ²⁰ *Loc. cit.*, p. 460. ²¹ Yet Mirallic actually quotes it as an example of a sub-cortical lesion. ²² *Loc. cit.*, p. 483. ²³ *Physiology*, fifth edition, 1890, p. 1089. ²⁴ The anterior commissure seems mainly to connect the convolutions of the tip and under surface of the temporal lobe, so that it is more probable that its upper and posterior regions should be brought into structural and functional relation through fibres of the corpus callosum. ²⁵ *Paralyses: Cerebral, Bulbar, and Spinal*, 1886, p. 267. ²⁶ On this subject see what has already been said towards the end of the last section but one of the first lecture. ²⁷ What has just been said concerning the right auditory word centre applies almost equally strongly to the right visual word centre. We want to know also to what extent and for how long the comprehension of written and printed words is impaired by destruction of the right angular gyrus and supramarginal lobule. ²⁸ *Ziemssen's Pathologie und Therapie*, Band xii, 1876, p. 168. ²⁹ *Revue de Médecine*, 1885, p. 625. ³⁰ *Lectures*, translation by Bazire, pp. 253, 267. ³¹ *BRITISH MEDICAL JOURNAL*, 1884, i, p. 593. ³² *Journal of Physiology*, 1890, p. 658. ³³ *Loc. cit.*, p. 634. ³⁴ *Archives de Physiologie*, August 5th, 1883. ³⁵ It has been shown by Sherrington and others, in their studies of "secondary degenerations," that some of these double motor effects may be accounted for by the distribution in the cord of the fibres of the pyramidal tracts. It is known also that Broadbent explains some of the bilateral movements by reference to commissures existing between functionally related bilateral centres in the cord. But the voluntary movements of the right fore-paw in Goltz's dog must have implied the development in the only existing right hemisphere of the animal of all the nervous arrangements needful for the "conception" of separate movements in this right limb—that is, the passage to it and registration of kinæsthetic impressions from this limb, as well as the bringing of this centre into relation with the visual centre, in the manner that would be needful for the production of its voluntary movements. ³⁶ *Paralyses: Cerebral, Bulbar, and Spinal*, 1886, p. 262. ³⁷ *Journal of Physiology*, 1886, p. 429. ³⁸ *Natural History Review*, vol. i, p. 296. ³⁹ *Brain*, January, 1885, p. 460. ⁴⁰ *Ibid.*, January, 1885, p. 482. ⁴¹ *Mémoires de la Société de Biologie*, 1891, p. 112. ⁴² *Revue de Médecine*, 1893, p. 733. It is quoted also by Mirallic (*loc. cit.*, p. 190). ⁴³ Though Wernicke says a few words concerning a case that had come under his observation (see *Fortschritte der Medizin*, 1886, p. 474). ⁴⁴ *Loc. cit.*, p. 171.

(To be concluded.)

On April 2nd the University of Vienna, for the first time in its history, conferred the degree of Universæ Medicinæ Doctor on a lady, the Baroness Gabriele Possaner von Ehrenthal. The Baroness, who is 37 years of age, and who already possessed a medical degree obtained from Zurich, passed all the examinations required by the Vienna University. The Rector, Professor Reinisch, in conferring the degree, specially congratulated the new Doctor.

OSTEOTOMY OF THE FEMUR AS A TREATMENT FOR TUBERCULOUS DISEASE OF THE HIP IN ITS EARLY STAGES.*

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HAVING in a few cases made, with successful results, a departure—new at least to me—in the treatment of tuberculous disease of the hip, I beg leave to submit it for your consideration. I shall take it as granted that in the early stages of morbus coxæ the direct removal of the affected parts yields unsatisfactory results, and that the problem of cure is narrowed into one of giving rest to the joint, combined, of course, with constitutional treatment. The difficulty of this problem lies, it seems to me, in the fact that the position of greatest ease for the joint is one of great awkwardness for the limb, which is flexed, abducted, and everted.

Experiments on the dead body show most clearly that any attempt to straighten the limb when the capsule is distended increases the tension of the joint. Mr. Swan distended the capsule with glycerine through a hole drilled into it through the ileo-pectineal eminence, whereupon the limb assumed what is called under such conditions the natural position of rest; he fixed the limb in that position and the capsule retained the injected fluid; he next straightened the limb, whereupon the fluid was forcibly squirted out.

The chief means of treatment at present in use seem to ignore this injunction: extension by a weight and pulley does so, in so far as it is used to straighten the limb; for this appliance does not act in the way it is represented as doing in the plates in handbooks of surgery. In these you see a patient with a weight attached to the leg, lying supine, his back and knee flat and both touching the mattress. What happens in reality is this. If the weight is heavy enough to keep the leg straight, the spine is dragged into a position of lordosis; if it is less heavy, there is less straightness of leg and less lordosis; in each case the lordosis is an indication of force exerted on the joint, to its discomfort. As to Thomas's splint, it is of course, as a support for the limb, an excellent contrivance, and when properly fitted and carefully worn it does all that a splint can do to make a distorted position easy for a patient; but it does not get over all the drawbacks of that position both by night and day.

I do not mean to say that many excellent cures are not effected by the treatment at present in use. Its general adoption by the profession proves it to be efficient in some degree. But I do say that it leaves much to be desired, that it not seldom fails, and that always the process of cure is a very protracted one, requiring throughout most careful and judicious supervision. This last-named condition alone puts it beyond the reach of very many patients. The number is large for whom hospital accommodation cannot be provided from start to finish, and who cannot carry out in their own homes any treatment that is not simplicity itself. Going back only on my own experience I am ashamed to say how many patients I can recall whom I have treated for two or three months in my wards with a weight and pulley, and fitted with a Thomas's splint, who were then unavoidably discharged and lost sight of for a time, and who at a later period turned up with a suppurating joint, due in part, no doubt, to the Thomas's splint having been thrown aside as soon as it had got in any way awry.

Being then convinced that the awkward position the thigh assumes in cases of morbus coxæ is a great obstacle to obtaining satisfactory results from treatment by rest, I have in some recent cases met the difficulty in the following way. I have, in the early stages of the disease, done an osteotomy of the femur on a level with the lesser trochanter (Gant's operation), and put the limb in a straight position as regards the body generally, leaving the upper fragment to retain its position of ease. The following case is illustrative:

P. R., aged 11, was admitted into St. Vincent's Hospital on February 11th, 1896, with tuberculous disease of the hip. The disease, which was of some months' standing, had come on insidiously, and he was sent for treatment only when lameness was very pronounced. There was no history of accident. All the usual signs of morbus coxæ in its first stage were present. On February 18th I did an osteotomy of the femur on a

* Read before the Dublin Biological Club.