ON THE LOCALISATION OF THE AUDITORY CENTRE.

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ALTHOUGH the localisation of the auditory centre or sphere in the first or in the two upper temporal convolutions is generally admitted, this view has not received universal acceptance; therefore, the great value of the history and autopsy here recorded will be acknowledged.

The recent views with reference to auditory localisation mostly agree in placing the auditory centre or centres in the temporal lobe, but differ as to their exact position in this lobe or as to the extent of cortex included in the auditory field. According to Munk, whose theories have been based chiefly on experiments performed on dogs, the auditory sphere is at a position corresponding in the monkey and the man to about the middle temporal gyre, considerably lower in the temporal lobe than Ferrier's localisation. In the upper or posterior portion of the superior temporal convolution, to which Ferrier gives the preference as the seat of the auditory centre, Munk localises the area for sensibility of the auricle. Luciani includes in the auditory sphere the parts which correspond to the upper temporal gyres, but, in addition, a large part of the cortex which is concerned with other functions. Schafer repeated Ferrier's experiments in a number of monkeys, obtaining some of the results of this investigator, but failing to get others. In conjunction with Dr. Sanger Brown, he, in six cases, more or less completely destroyed the superior temporal convolution on both sides,

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and claims that in all, hearing was not only not abolished but was not perceptibly affected. In one of the six monkeys the whole temporal lobe of the one side was removed, and yet Schafer holds that the animal responded to all impressions of all the senses, but appeared to understand very imperfectly the meaning of such expressions. Sounds even slight in intensity were heard. He believes that this case militates strongly against the view that auditory perception is localised in the temporal lobes.

Ferrier has ably replied to the attacks of Schafer both in BRAIN, April, 1888, and in the Croonian lectures.[1] He also reaffirms the accuracy of his own experiments and the justice of his own conclusions. To clear this matter up, he reinvestigated the question. In one monkey in which he performed the operation of bilateral extirpation of the superior temporal gyre, with one month interval between the operations, the result was very striking. The following, in his own words, are the conclusions as to the condition of this animal after the second operation.

"At first it failed absolutely to respond to any of the tests which invariably attracted the attention of normal monkeys. To the last, with the single exception, perhaps, of the door of the laboratory from which it was always expecting something, it never realised the origin of sounds; it was altogether indifferent to sounds which formerly were full of significance to it, and all that could be said was that it was not, when otherwise occupied, insensible to sonorous vibrations. And it would have been difficult to make this out with any degree of certainty had it not been that the animal was one qualified to give oral testimony of the fact. Whether this form of auditory sensibility is to be attributed to portions of the cortical auditory centres undestroyed by the lesions described, or to the subcortical or mesencephalic centres, I am not yet in a position to decide."

He finds further confirmation of the localisation of the auditory centre in the superior temporal convolution in cases

[1] The Croonian lectures on Cerebral Localisation, delivered before the Royal College of Physicians, June, 1890. From these lectures the summary of the discussion of auditory localisation is chiefly taken.

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of auditory discharges or subjective auditory sensations in connection with irritative lesions implicating this gyrus. "Gowers has reported two cases of this nature. In one a tumor, of which the oldest part was beneath the superior temporal convolution, caused convulsions commencing with an auditory aura referred to the opposite ear. In the other, a tumor affecting the superior temporal gyrus caused unilateral convulsions preceded by a loud noise as of machinery. And Hughes Bennett has reported several cases of auditory sensory discharges followed by a temporary loss of hearing in the opposite ear or in both. Thus, a woman, subject to epileptic attacks preceded by a loud noise like the ringing of a bell in the left ear, became temporarily deaf in each ear after each attack. Both ears were deficient in hearing, but the left undoubtedly more so."

The present case should be ranked as conclusive in the decision of the question of auditory localisation, the specimens showing an insolated lesion in the two upper temporal gyres of the left hemisphere, which caused word deafness, and also a later lesion of the two superior temporals of the other hemisphere with a history of almost total deafness.

Ferrier speaks of the extreme rarity of bilateral lesions of the superior temporal convolution, but gives two important cases in which these double lesions simultaneously or successively occurred. One case, first recorded by Shaw, is that of a woman who suddenly lost power in the right arm, with also loss of speech and word deafness. The loss of power passed away, but she became incoherent and subject to delusions, and on testing her she was found to be deaf and blind. The *post-mortem* showed atrophy of both the angular and the two superior convolutions of both hemispheres. The other was a case of Wernicke and Friedlander, a woman who, after apoplexy, had right hemiplegia, aphasia, paraphasia, and word-deafness. A few months later she had a second attack, causing paresis of the left arm, and she became absolutely deaf. Extensive lesions were found in the superior temporal convolutions of both hemispheres.

I have recorded the case of a man, deaf for thirty years, whose brain showed marked atrophy of both superior

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temporal convolutions, particularly the left side.[1] Broadbent has recorded a similar case referred to by Ferrier.

Numerous cases of lesions of the superior temporal gyres of one side have been reported, and word deafness has most frequently been found an accompaniment of lesion of the left first temporal convolution. These facts are now well known, and have been put on record by various observers as Seppilli, Ferrier, Starr and others.

Word-deafness following an apopletic seizure—more complete deafness and partial left-sided paralysis following a second apoplexy—autopsy showing lesions of the first and second temporal convolutions of both hemispheres—a lesion of the first and second temporal convolutions of the left hemisphere, which probably accurately localises the centre for word-hearing.

The patient, a woman, aged 46 years, was admitted to the Philadelphia Hospital in August, 1891. She had a history of scarlet fever in childhood, of rheumatism many years ago, and also of old venereal disease. She had had five abortions or miscarriages, and had one living child, 23 years of age, who was deaf and weak-minded. For many years she had had valvular disease of the heart. She was right-handed. Some of the facts of her history were obtained from relatives who called to see her at the hospital, and others were procured after her death from a brother and sister-in-law, whom I sought out and carefully interviewed.

Fifteen years before her death she had an apoplectic attack which left her word-deaf but not paralysed. Prior to this first attack of apoplexy her hearing had been good, but after it she could not, by hearing, understand anything that was said to her. She could, however, hear music and sounds of various kinds ; for instance, when an organ or band had performed upon the street she at times had called attention to the fact; and she had also come down from the second or even the third story to open the front door in answer to a knock. She could hear such sounds as a bell ringing and a clock ticking. These facts were elicited from her relatives through various statements made by them, chiefly spontaneously.

When anyone wished to communicate with her it was done by means of writing or signs, as she had fully preserved her vision and was evidently not word-blind either for writing or printing.

[1] *University Medical Magazine*, November, 1889.

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She often read the newspapers, and could do so with intelligence up to a few weeks before her death. Her sister-in-law said that several times she had heard her try to read the newspaper aloud, and in so doing she had seemed to understand fully what she read, "but made a tangle of her words." From the time of the first attack she had never been able to speak well, her words becoming "jumbled" or "tangled." From the description given of her manner of speech the defect was evidently a serious form of paraphasia and paralexia.

Her relatives spoke positively of her deafness as having been due to the "stroke;" but the apoplectic attack, although it had at once caused this word-deafness and paraphasia, had not in any way, as far as could be ascertained, affected either motion or sensation. 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, and in this extremity, from the description, the paralysis was more marked below the elbow. Upon examination after admission to the hospital, she was found to have some contracture at the metacarpophalangeal articulations.

Six weeks before her admission she began to have pain in the left side and abdomen, which gradually increased in severity. The day before her admission she had a chill and pain in the left shoulder and arm. For five weeks she had involuntary evacuations of urine, and for about two weeks she had been unable to retain her fæces. For these six weeks she had been much more helpless than previous to this time, often letting things drop from her hands. She seemed to suffer considerably from headache, and also slept badly.

She was first examined by me on August 24, 1891. Her condition then was one of almost complete helplessness. It was impossible to make her understand what was said to her, and so far as could be determined by repeated tests she was totally deaf; but notwithstanding her weakness, helplessness, and deafness, her face had a somewhat intelligent expression. She looked about her as if she knew what was going on. She was very emaciated; her heart's action was excited, and examination showed the presence of marked murmurs, both mitral and aortic. She became feebler day by day, and died August 28.

Autopsy.—No disease of the bone or of the dura mater was

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found. Even before pia-arachnoid of the left hemisphere was removed, it was noticeable that the first temporal convolution was much smaller and thinner than usual, and that at the posterior extremity of it and of the second temporal, there was a depression covering a space about seven-eights of an inch in diameter.

The *left* first temporal convolution is remarkably small, narrow, and smooth, except at its anterior extremity. Its posterior two-thirds or three-fourths have shrunken to a thin strip. At a point about the middle of the gyre, the convolution has so disappeared as to leave only a notch and shred of tissue. Just anteriorly to this point a small annectant gyre runs to the second temporal. The attenuated appearance of this superior temporal convolution is such as to attract the attention of the most inexperienced observer of such appearances.

At a position corresponding to the posterior fourth of the second temporal convolution and the parallel fissure, the brain presented a marked depression or cavity, at the bottom of which, when the specimen was in the fresh state, was a small mass of yellow, shrivelled, puckered tissue. This was evidently the remains of an old embolic softening. The subarachnoid cavity, or cyst, which was present before the inner membranes were removed, was formed by this old necrosed area and the widened parallel fissure, this widening having chiefly resulted from the atrophy of the first temporal convolution.

In connection with the question of the part played by the second temporal convolution in cerebral audition, it is, of course, important to carefully describe the condition of this convolution. From the position of the annectant convolution, it was seen to be decidedly atrophied, and in its posterior fourth, or perhaps third, it has practically disappeared, and has been replaced by the cavity or cyst just described.

The third, fourth, and fifth temporal convolutions were undoubtedly not involved in either the softening or atrophy, as the brain was carefully studied by me in both directions, from the second temporal convolution to the hippocampal and the reverse.

Around the ascending branch of the Sylvian fissure and at the bases of the two central convolutions, much atrophy has evidently also taken place. This ascending branch of the Sylvian, instead of being a mere indentation or narrow fissure as is usually the case, is wide and gaping. The hinder portion of the third frontal, and particularly the strip of convolution between the ascending Sylvian and precentral fissure, is markedly wasted.

The retro-insular convolutions are two in number, and the

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posterior of the two is very small. The anterior retro-insular presents the appearance of being a continuation of the anterior half of the first temporal, and the posterior retro-insular is continuous with the posterior much-shrunken half of the first temporal.

In the *right* hemisphere was an old and very extensive hæmorrhagic cyst which completely destroyed the first, and almost completely the second, temporal gyre, the island of Reil, retro-insular gyres, the lower extremities of the central gyres, and a large extent—but exactly how much could not be determined—of the ganglia and capsules. Examination from within showed that the caudate body and the thalamus had largely preserved their integrity, and the chief interior destruction was probably of the lenticular ganglion and the external capsule. In the posterior portion of the cystic area, some gyral substance is seen, but examination shows this to be the sunken in lower extremity of the inferior parietal convolution. As, on the other side, the supra-Sylvian bordering convolutions are much atrophied. The auditory nerves were atrophied. The acoustic striæ, usually so easily seen, and often so prominent, could not be made out with the naked eye.

Slight adhesions of the left pleura were present on both sides. The lungs were œdematous and exuded a turbulent fluid on pressure. The precardial sac contained an ounce and a half of fluid. The endocardium was thickened. The mitral valve was also much thickened, the orifice being so small that it would not admit the little finger. The aortic valves were in a similar but worse condition, and a clot was found in the left auricle resembling in appearance those seen in aneurisms. The spleen was small. The kidneys were small and presented on their surface large hæmorrhagic spots. On section the surface was found to be flabby, and the cortex and pyramids much distorted. The spots on the surface had irregular shapes, having the appearance of old and recent hæmorrhagic infarcts. The liver was small and moderately firm. The ovaries were dense and sclerotic.

A study of this case justifies the following conclusions:

(1) The centre for word hearing is situated in the hinder thirds of the first and second temporal convolutions; its exact position is in a line with, or just in front of, the posterior extremity of the horizontal branch of the fissure of Sylvius. Possibly it is restricted to the second temporal.

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(2) The third, fourth, and fifth temporal convolutions take no part in cerebral audition.

(3) A lesion confined to the posterior thirds of the first and second temporal convolutions of the left hemisphere will produce complete, or almost complete, word deafness, the corresponding regions of the other hemisphere remaining intact.

(4) The field or sphere for all auditory memories covers a much larger cortical area than that for word-hearing, including at least the posterior two-thirds of the first and second temporal convolutions.

(5) The auditory field and special auditory centres have their highest development in the left hemisphere, but destruction of the auditory areas of the two upper temporal convolutions of both hemispheres is necessary to complete brain deafness.

(6) A lesion limited to the centre for word-hearing and causing word-deafness will cause also paraphasia in attempts at speaking, and paralexia in attempts at reading.

(7) An isolated lesion of the centre for word-hearing, producing absolute, or nearly absolute, word-deafness, does not necessarily cause inability to recall words by other means, as, for instance, through their visual signs; in such cases probably the meaning of the word is understood, although the name cannot be properly verified in consciousness.

(8) A cerebral lesion or lesions causing word-deafness will, in time, lead to secondary atrophy of the speech and oro-lingual centres on the motor or emissive side of the brain, and also to atrophy of the association tracts between the sensory and motor hearing-speech centres.

(9) The retro-insular convolutions are anatomically and functionally closely related with subdivisions of the first temporal convolution, the most posterior of these retro-insular convolutions being continuous with the posterior two-thirds of the first temporal convolution.

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