THE CROONIAN LECTURES ON CEREBRAL LOCALISATION.

Delivered before the Royal College of Physicians of London.

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LECTURE IV.—THE AUDITORY CENTRE.

MR. PRESIDENT AND GENTLEMEN,—Among the reactions consequent upon electrical stimulation of the cortex there is one, or rather an assemblage of reactions, which might almost be considered of itself indicative of the existence of subjective auditory sensation—a reaction which guided me in my first attempts to define the auditory sphere by the destructive method. The reaction in question is that which occurs on irritation of the superior temporal convolution and its homologues in the lower vertebrates: namely, quick retraction or pricking of the opposite ear, associated frequently with opening of the eyes, dilatation of the pupils, and turning of the head and eyes to the opposite side. These are just the phenomena which occur when a shrill sound is suddenly made in a monkey's ear, as I have found by actual experiment. The reaction, however, varies somewhat in its completeness. After the first surprise is over, the repetition of the experiment always induces the pricking or retraction of the ear, but generally fails to induce the other parts of the reaction, namely, the intense look of surprise, and direction of the head and eyes to the supposed source of the sound. The results are still more characteristic on stimulation of the homologous region (Fig. 14) in those animals whose habits are such as to make their safety largely dependent on the acuteness of their hearing. The region in question is the posterior division of the third external or supra-Sylvian convolution. The reaction common to all these is pricking of the opposite ear, but the other factors vary in intensity. In the lop-eared rabbit, irritation of this region causes sudden elevation of the ear, as well as retraction and exposure of the mouth of the auricle towards the referred region of the sound. Occasionally the animal makes a sudden start and movement, as if to bound off the table. In the wild jackal, also, I observed on one or two occasions that the application of the electrodes to this region caused the animal to make a sudden spring or bound forward, pricking up both ears as if suddenly startled. If the movements of the eyeball on stimulation of the occipito-angular region are to be regarded as signs of arousal of subjective visual sensation, we have, I think, in the reactions under consideration, still more characteristic indications of the arousal of subjective auditory sensation. The determination, however, of affections of hearing is not so easy as that of sight in the lower animals. It is difficult to discriminate between mere reflex starting to sounds, and hearing proper. Nor is it easy to avoid mere coincidences, or to entirely avoid attracting the animal's attention by other channels of perception, such as sight, or smell, or sense of proximity conditioned by vibration, warmth, agitation of the air, and such like. The animal's present behaviour should be compared with its past, and with that of normal animals under conditions of variation of sound, all other things remaining the same. And even when all these precautions are taken, it is extremely difficult to avoid every source of fallacy. Hence it may happen, as it has actually happened, that different observers may arrive at different conclusions, and animals may be taken to be deaf which are not in reality so, simply because they do not respond to the test employed, or *vice versa*.

In one of my earlier experiments, in which the upper two-thirds of the superior temporal convolution were destroyed on both sides, the following note was made as to the condition of the monkey the day after the operation.¹ "Sight was good and tactile sensation unimpaired. Various experiments were made to ascertain the existence or not of hearing, but it was not easy to devise a test, as the animal was continually on the alert, and it was not easy to make a sound without in any way attracting its attention by sight. The following method was tried: while the animal was sitting quietly by the fire I retired to the other room, and, while watching through the chink of the half-shut door, called

loudly, whistled, knocked on the door, tinkled glass, etc., without ever causing it to look around or give any sign of having heard. I then cautiously approached the animal, and not till it saw me did it give any sign of consciousness of my presence. When the same experiment was repeated while the monkey and its companion were quietly seated at the fire enjoying the heat, it gave no sign of hearing, while its companion started with alarm, and came with curiosity to ascertain the cause of the sound. Ten hours afterwards, in the presence of Dr. Burdon Sanderson, I repeated the various tests with the view of eliciting signs of hearing. To all it remained without response. It seemed unconscious of my presence when speaking close to its ear, and only started when it caught sight of me."

I recorded also four other experiments,² in which, along with destruction of other portions of the temporal lobe, the superior temporal convolution was invaded, unilaterally or bilaterally. In two of the cases (XI and XII), in which the temporal lobe was destroyed only on one side, there was impairment or total abolition of reaction to sound when the ear on the same side was plugged; and in the two other cases, in which the destruction was bilateral, no signs of hearing could be elicited during the short period which the animals were allowed to survive, though, in other respects, they were fully on the alert. In these experiments, however, the time that elapsed between the infliction of the lesion and the death of the animals was not sufficient to establish any very reliable data as to the permanency of the impairment of the sense of hearing, which was undoubtedly, however, affected in all for the time being. In my subsequent investigations, in conjunction with Professor Yeo, however, it was established in the first place by an extensive series of experiments on the temporal lobe³ that no signs of impairment of hearing could be detected when every part of the temporal lobe was destroyed, except the superior temporal convolution. What appeared to us, and most of those who saw it, conclusive evidence of the relation of the superior temporal convolution to the sense of hearing, was the behaviour of a monkey in which both superior convolutions were actually or potentially destroyed by means of the cautery. Where the cortex was not absolutely removed, the grey matter was undermined, and the medullary fibres destroyed by the action of the radiant heat.

This monkey was exhibited before the assembled physiologists at the International Medical Congress in London in August, 1881, and was admitted by all to be deaf, so far at least as that could be determined by comparing its behaviour with that of a normal animal, to the explosion of a percussion cap in the room.

As questions were raised in regard to the actual condition of this animal before and after the operation by the experiments of Professor Schäfer, to which I am about to allude, I have published a full account of my notes and observations on this animal.⁴ Briefly, these were as follows: in all other respects, except as to hearing, it was in a perfectly normal condition. During the first four days after the operation, no signs of hearing could be elicited by any sounds made in its vicinity, and which invariably attracted the attention of normal monkeys. While the other animals were observed eagerly listening to approaching footsteps, this animal made no sign of attention until the person came within range of vision. It was observed also that, at first at least, the ears of the animal did not twitch as they do in monkeys which undoubtedly possess the sense of hearing, but it was doubtful whether the absence of reaction in the ears continued during the whole time that the animal survived. Almost daily examination was made during the thirteen months that it was allowed to live. To sounds of various kinds, such as calling by name, to which it used always to respond, tapping, whistling, ringing of bells, shuffling of feet on the floor (a sound which normal monkeys are particularly quick to notice), this animal paid no attention. Occasionally, however, it seemed to start coincidentally with loud sounds made in its vicinity, so that doubts were constantly being raised as to whether it was absolutely deaf or no. These reactions were considered to be probably mere coincidences, for in general it did not even start when a percussion cap was exploded near it. Six weeks after the operation, while the animal was disporting itself before the assembled physiologists at the Medical Congress, it paid no heed to the explosion of a percussion cap, while another monkey, exhibited at the same time, started visibly, as, in fact, did the whole audience. From this time onward the tests were

² Experiments XI, XII, XIII, XIV, *op. cit.*, p. 462.

³ *Philosophical Transactions*, Part II, 1884.

⁴ *Brain*, April, 1888, p. 13.

¹ Experiment XV, *Phil. Trans.*, vol. 285, Part II, 1875.

constantly repeated and varied in every conceivable manner, but the results were essentially the same, and the conclusion seemed to be justified that the animal was essentially deaf, and that the occasional start which it made to loud sounds was either coincidence or merely of the character of the reflex start, which may occur in animals after removal even of the whole cerebral hemispheres.

Schäfer, in his earlier experiments, in conjunction with Horsley, did not arrive at any very definite conclusions with regard to the sense of hearing, though in one of their experiments⁵ in which they had removed the right temporo-sphenoidal lobe, the animal did not seem to hear slight sounds when the ear on the same side as the lesion was stopped. But, working with Sanger-Brown, he succeeded in removing all doubts which he might have entertained on the question, and in convincing himself that hearing was not in the slightest degree affected after complete removal, not only of the superior temporal gyri but of the whole temporal lobe itself on both sides. He thus describes the result of their experiments⁶:-

"In six monkeys we have more or less completely destroyed the superior temporal gyrus upon both sides. I say more or less completely because in one or two a small shred of grey matter belonging to this convolution was found *post mortem*, but practically the lesion was complete in all six, some of the grey matter within the fissures bounding the gyrus being all that could be taken to represent the convolution, and even this being deprived of its medullary centre. But in order to make assurance doubly sure we, in one monkey, a large female Rhesus, separated up the fissures bounding the gyrus and scooped it out entirely from the very bottom of the fissures, so that not a trace of the convolution in question should remain. In all six cases the result was the same. Hearing was not only not permanently abolished: it was not perceptibly affected. The animals, even immediately after recovery from the anæsthetic, reacted to slight sounds of an unusual character such as smacking of the lips or the rustle of a crumpled newspaper. Some of them were under observation for several months, and there never was any doubt in our minds as to the full possession of their auditory faculties. Nor could the reactions they exhibited to sounds be explained by supposing that they only responded in a reflex manner, for they gave every evidence of understanding the nature of different sounds, such as that caused by turning a door-handle or the differences between the footsteps of different people, varying emotion being exhibited according to the anticipations (of food, etc.) which the sounds called forth."

Two of these animals, in which the destruction of the superior temporal convolution seems to have been most complete, were examined by several members of the Neurological Society. Two, who, however, did not record the tests they employed, were of opinion that the animal heard; while another thought on one occasion that one of the animals could hear, at another time that it seemed deaf on the left side, and, on still another occasion, that it did not seem to react to sound so readily and completely as a normal animal.⁷

As illustrating the difficulty of arriving at definite conclusions on this matter, one member of the Society thought that a perfectly normal monkey was quite deaf, and another that an animal in which both superior temporal gyri had been removed was also deaf. Schäfer⁸ believes that I also was satisfied as to the existence of auditory perception in his animals. Such examination as I made of them did not, I think, furnish any unequivocal evidence on the point. An unaccountable discrepancy between Schäfer's results and Yeo's and mine thus appeared to exist. If in his animals hearing was not perceptibly affected by the double lesion, this certainly was not the case in ours; for there was no question as to the profound difference between it and a normal monkey, and the only doubt was whether it had any real sense of hearing at all.

In order, if possible, to clear this matter up I have lately re-investigated the question. In one monkey I removed, first, the whole of the convex aspect of the left temporal lobe, including the superior temporal gyrus. The animal speedily recovered from the unilateral operation, and, for the first few days, though undoubtedly it heard, it seemed to hear less distinctly towards the right than towards the left. A fortnight afterwards a similar operation was performed on the right side, but though the animal

lived five days it remained in a state of great apathy. It took no notice of what was going on around it, and was altogether indifferent to the loudest sounds made in its vicinity. The experiment, therefore, on account of the short period of survival, was not quite a satisfactory one. Another monkey in which I performed the operation of bilateral extirpation of the superior temporal gyrus, with an interval of one month between the two operations, was specially well adapted for experimentation in regard to the sense of hearing. It was a remarkably tame dog-faced monkey, and its character and modes of behaviour under different circumstances were made the subject of careful study before it was operated upon. It was a noisy, talking monkey. It invariably responded when called to by name, and came immediately when called. It imitated smacking of the lips and other sounds of endearment. It always shouted vigorously and loudly when anyone approached or opened the door leading down to the laboratory in which it was kept. The rustling of a paper bag, from which it was accustomed to receive sweetmeats and nuts, was the signal for vociferous cries; as also any movement

Fig. 16.

Fig. 17.

of the handle of a drawer in which apples and fruits were kept. It had an insatiable appetite, and was always clamouring for food, of which it never seemed to have enough. It had also an insatiable thirst, and the sound of splashing of water, made by turning on the water-tap, caused it to shriek to be let out and put its mouth under the tap. Not a sound could be made in its vicinity, or a movement of its companion monkeys in the other cages, without exciting its active attention. It was full of fun and mischief, and in every respect an animal unusually adapted for determination of any alterations that might occur in respect of its auditory or other facilities. The left temporal superior convolution was scooped out on October 8th. (As will be seen in the photograph) a small portion also of the middle temporal gyrus was destroyed, (Fig. 16).

The next day, owing to the occurrence, as afterwards proved, of some recurrent hæmorrhage and tension in the wound, there were one or two slight epileptic seizures affecting the right side. These, however, ceased entirely when the wound was redressed, and the animal was speedily quite bright and active.

On the second day it turned its head to the dangling of keys close to its left ear, but it did not do so, or very doubtfully, to the

⁵ Experiment XXX, *Phil. Trans.*, 1880.

⁶ *Brain*, Vol. 10, p. 373.

⁷ See *Brain*, July, 1883, p. 164.

⁸ *Phil. Trans.*, 1888, B. 30, p. 325.

same test applied to the right. Next day, and from that time onwards, it appeared, as regards its hearing, in every respect as it had been before the operation. It was noted, however, that it had become completely hemipic to the right—a condition which lasted more or less completely until its death five months subsequently. This hemipia proved after death to be due, in all probability, to the recurrent hæmorrhage which occurred on the day after the operation, ploughing up the optic radiations in the occipito-angular region.

On November 5th, the superior temporal gyrus of the right side was similarly exposed and scooped out. This time the lower extremity of the gyrus almost entirely escaped (Fig. 17). On the following day the animal made no response to any sounds made in its vicinity, nor when the door leading to the laboratory was slammed, nor to the noise of footsteps on the stairs which formerly used to excite lively demonstrations.

On the third day, though otherwise alert and bright, it made no reaction to sounds of any kind, did not reply to calls, did not notice the noise of footsteps on the stairs, exhibited no sign of perception when the water-tap was turned on, though it was evidently intensely thirsty, as it drank eagerly when water was presented to it; paid no attention to the cries of two animals which were placed in the adjoining cage, and, in general, responded to none of the tests of hearing which formerly aroused its active interest.

On the seventh day the condition was essentially the same. It paid no heed when called to, or when the door leading to the laboratory was slammed. The rattling of the drawer in which the food was kept or the rustling of the paper bag containing the sweetmeats were altogether unnoticed; the plashing of water was unheeded; and it went on quietly with its occupation, eating or searching for food on the floor of its cage, while various sounds, such as springing a rattle, loud barking, whistling, etc., were being made, which startled the other animals in adjoining cages. On the tenth day the condition was essentially the same. There was no reaction to calls, shuffling of feet, etc., which caused other two normal monkeys in the adjoining cage to peer curiously. On this day a test was employed to which it formerly invariably responded. It was late in the evening. I put out the light in the laboratory and ascended the stairs and shut the door. Several times I opened the door and called the animal by name. One of the other monkeys called back in reply, but no response was made by this animal. On former occasions this was the signal for shrill and loud outcries. On the following day the same tests were repeated, and with the same results. It was very fond of the laboratory attendant, and would invariably go to him if he called it. On this day, while it was on the table and watching my movements, the laboratory attendant came down the stairs and, standing behind it, called it repeatedly. The animal paid no attention whatever, and never once turned its head in his direction.

On November 20th (fifteen days after the operation) the following notes were made:—

"The animal well and active, occasionally uttering grunts of satisfaction while sitting quietly by the fire. It also cries loudly when it wants food, but it never responds when called to; pays no attention to the familiar sounds, such as opening the drawer containing the apples or turning on the water tap, which formerly excited in it lively demonstrations. To-day a box containing a whistle, capable of being blown by a long india-rubber tube in such a manner as to avoid attracting the animal's sight, was placed in its cage and the whistle repeatedly blown, but it was absolutely unmoved. The same experiment with three other monkeys caused signs of alarm and perturbation."

About this time the question began to be raised whether the animal did not seem to be aware, by a sense of vibration or otherwise, of the approach of footsteps descending the spiral staircase leading into the laboratory. It was quite certain, however, that while it was being watched, and its attention thus occupied, it seemed quite unconscious of any sound made to divert it. On November 30th, that is, three weeks after the operation, these observations were confirmed after careful study by myself and assistant for several hours. But when all was still it seemed to be aware, as evidenced by its cries, of the proximity of anyone walking on the floor overhead, or opening the door leading down into the laboratory. This was noticed more particularly in the early morning, when this and the other monkeys were eagerly looking towards the stairs expecting their breakfast. Repeated observations during the course of the following week succeeded in clear-

ing up the apparent irregularities in its reactions. It became evident that when the animal was left to itself it was aware of being called to, or of the sound of opening or slamming the door leading to the place in which it was kept, but when anyone was present in the laboratory, and the animal occupied in observing him, it showed no signs of hearing, and did not look round like the other monkeys when called to or when sounds of various kinds were made. If I waited in the laboratory within the range of the animal's vision, and my assistant went to the top of the stairs and called, it paid no heed whatever to anything he might do to attract its attention. Though it occasionally started when a percussion cap was exploded in its vicinity, it did not do so or look around if it happened to be engaged on anything else at the moment; whereas other monkeys would invariably start and look round towards the cause of the sound, and this condition remained practically unchanged until the death of the animal. In the direction of the laboratory door alone, towards which it was almost constantly looking, with expectation, did it seem able to refer sounds, but otherwise, especially when its attention was diverted in any way, it seemed entirely unaware of their origin, and did not turn its head in their direction. It either did not hear or wholly disregarded such sounds as scratching, tapping, rattling of keys, and so forth, which caused the other animals to peer eagerly through the bars of their cages. The plashing of water, rustling of paper, opening of the drawer from which it was accustomed to receive choice morsels never excited any notice, and caused none of the lively signs of interest which formerly were so characteristic. I did not observe in this animal, at least during the latter periods of observation, that absence of the twitching of the ears which I had noted in a previous experiment.

The condition of this animal may, therefore, be stated briefly as follows. At first it failed absolutely to respond to any of the tests which formerly excited active reaction, and 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 subcortical or mesencephalic centres, I am not yet in a position to decide. I have not yet been able successfully to carry out observations on animals in which the temporal lobes have been destroyed on both sides; but if, as Schäfer's experiments seem to indicate, the whole of the temporal lobes, like the other cerebral lobes, may be removed without entirely abolishing reaction to sounds, we should have reason for believing, with Longet, Goltz, etc., that in monkeys, as well as lower mammals, a crude and simple form of auditory sensation is still possible through the agency of the lower centres.

Munk, from his experiments on dogs, states that destructive lesion of the point B (Fig. 14), situated towards the lower extremity of the supra-Sylvian and adjacent extremity of the second external convolution, causes a condition as regards hearing similar to that resulting from destruction of A (Fig. 14) in the sphere of vision; a condition which he terms "psychical deafness" (*Seelen-taubheit*). The dog seems to hear, but is unable to interpret the sound that it hears. This condition, however, lasts only for a few weeks at most. The animal again learns the meaning of sounds, and becomes restored to its normal state. In this region are stored up, according to him, the auditory pictures, similar to the visual pictures in the region A (Fig. 14). Frequently, however, before the secondary disturbances created by the primary lesion have subsided, the animal appears to be totally deaf, so that it exhibits no reaction to any kind of sound, however loud. Occasionally when the whole of the cortex of both temporal lobes had been destroyed, he has observed persistent "cortical deafness" (*Rindentaubheit*), as he terms it, but as none of the animals on which he has performed this operation seem to have lived beyond a few days, his experiments do not afford data for the determination of the duration of this complete deafness. He, however, assumes that the auditory area of the cortex embraces a much larger extent than B, and includes, according to his figures, the whole of the posterior half of the third, and also of the posterior or lower divisions of the first and second external convolu-

tions. With respect to the auditory sphere in the brain of the monkey, he assumes—for he does not appear to have performed any experiments bearing on this point—that it is situated at the lower extremity of the middle temporal gyrus (B., Fig. 18) a region which I have completely extirpated without causing the slightest affection of auditory sensation. And he considers that the upper portion of the superior or temporal gyrus (G, Fig. 18) is the sensory sphere of the ear; of this I can obtain no evidence, for neither after unilateral nor bilateral extirpation of this region have I been able to verify any affection of the sensibility of the auricle.

Fig. 18.—Cortical centres of the monkey, according to Munk. A, Visual area; c to J, tactile sensory areas (*Fühlsphäre*) of Munk's psychomotor areas of other authors; D, fore-limb region; c, hind-limb region; E, head region; F, eye region; G, ear region; H, neck region; J, trunk region. B is placed upon that portion of the cortex which, from experiments on dogs, is assumed to be the auditory area.

Luciani and Tamburini⁹ found that unilateral destruction of the upper and posterior part of the third external convolution in dogs caused deafness in both ears, but to a greater extent in the ear of the opposite side. The difference in auditory sensibility on the two sides greatly diminishes, and after a few days disappears altogether, though it cannot be asserted that perfect recovery ever takes place. After comparative equalisation of the power of hearing on the two sides, removal of the corresponding region of the other hemisphere is followed by almost total deafness, approximately equal on both sides. This bilateral deafness passes off gradually, but data do not yet exist, according to them, for determining whether perfect recovery takes place. When the destructive lesions are confined to the posterior part of the second external convolution, and do not implicate any part of the third, there does not occur the slightest impairment of hearing, which, indeed, sometimes appears to be unusually keen. These authors believe that semi-decussation of the auditory nerves exists similar to that of the optic nerves, and that both ears are represented in each cerebral hemisphere. This latter is undoubtedly the case; for unilateral extirpation never gives rise to permanent deafness of the one ear; but, though I have on many occasions, after extirpation of the auditory area in one hemisphere, observed loss or impairment of hearing in the opposite ear, I have never been able to detect the slightest impairment of hearing on the ear of the same side. Luciani¹⁰ states: "The effects of extirpations in the province of the parietal lobe confirm what was recognised by Ferrier, and afterwards by Tamburini and myself, that the bend of the third external convolution certainly makes part of the auditory sphere in the dog, but demonstrate also that this sphere irradiates from its central point in the temporal lobe, upwards towards the parietal region, forwards towards the frontal, backwards toward the hippocampal, inward towards the cornu ammonis." He thus includes in his auditory sphere a large portion of the cortex which is concerned with other functions. In reference, however, to this and other sensory centres, Luciani appears to contend for a form of localisation which is no localisation at all, as every centre seems to perform to some extent the functions of every other centre; results which I regard as absolutely opposed by the facts of strictly localised lesions.

It will be observed, in reference to the position of the auditory area in the dog, that it does not occupy the same relation to the fissure of Sylvius that the superior temporal gyrus does in the monkey, but is separated from it by the posterior limb of the

Sylvian convolution. It has been assumed by Meynert that the posterior division of the Sylvian convolution is the homologue of the superior temporo-sphenoidal gyrus.¹¹ I, however, have ventured to suggest that the resemblance between the posterior division of the Sylvian convolution and the superior temporal gyrus is only superficial, and is conditioned by the shallowness of the fissure of Sylvius. The investigations of Sir William Turner¹² have led him to the conclusion that the Sylvian convolution in the dog is in reality the homologue of the island of Reil, visible on the surface owing to the shallowness of the Sylvian fissure. In this case the posterior division of the supra-Sylvian convolution would correspond accurately with that of the superior temporal gyrus.

The occurrence of deafness from cerebral disease in man is unusual, owing to the extreme rarity of bilateral lesions affecting simultaneously both superior temporal gyri. There are, however, on record two important cases in which this double lesion occurred.

Fig. 19.

Fig. 20.

Shaw¹³ has recorded the case of a woman, aged 34, who, two months before her admission into his asylum, lost power in the right arm, and soon after had a sudden apoplectic seizure, resulting in loss of speech and deafness. The loss of power in the right hand speedily passed off. She became excited, incoherent, and subject to delusions. On admission, she was found, after repeated testing, to be perfectly deaf and blind. Tactile sensibility and smell were unimpaired. She had occasional fits and ultimately died of pneumonia a year after her admission

¹¹ Die Windungen der convexen Oberfläche des Vorder-Hirns, *Archiv f. Psychiatrie*, Bd. vii, 1877.

¹² Report on the Seals, *Challenger Expedition*, Part LXVIII, p. 124.

¹³ *Archiv. of Medicine*, February, 1882.

⁹ *Sui Centri Psico-Sensori Corticali*, 1879.

On the Sensorial Localisations in the Cortex Cerebri, *Brain*, 1885, p. 151.

Post-mortem examination showed complete atrophy of the angular gyri and superior temporo-sphenoidal convolutions of both hemispheres. (See Figs. 19 and 20). The grey matter of the atrophied regions had entirely disappeared, leaving the outer layer attached to the pia mater, with a cavity underneath formed at the expense of the grey matter. The other cranial nerves were normal in appearance, but the optic nerves showed increase of the connective tissue septa, atrophy of the nerve fibres, and spaces filled with a colloid-like material. Whether the blindness was due to the lesion of the angular gyri alone or to degenerative changes in the optic nerves is a question, but the sudden onset of deafness in this case, coincidently with symptoms of cerebral lesion and the condition of the brain *post mortem*, point to the destruction of the superior temporal convolutions as its cause. A similar case has been reported by Wernicke and Friedländer:¹⁴ "A woman, aged 43, who had never suffered from deafness or affection of vision,

Fig. 21.

Fig. 22.

was attacked on June 22nd, 1880, with right hemiplegia and aphasia. She remained in the hospital until August 4th, when she was discharged. At this time the patient could speak, but she spoke unintelligibly, and was sometimes believed to be intoxicated. She not only could not make herself understood, but she could not understand what was said to her. She was received into the hospital again on September 10th, with slight paresis of the left arm. The right hemiplegia had entirely disappeared. The patient was looked upon as insane. She was absolutely deaf, so that she could not be communicated with. She died of an attack of hæmatemesis on October 21st. An extensive lesion was found in each temporal lobe, invading the superior temporal convolution on both sides. (See Figs. 21 and 22). The rest of the brain ex-

hibited no abnormality, nor were there any conditions leading to increase of the intracranial pressure or secondary affection of the cranial nerves.

It was proved that the patient had previously enjoyed excellent hearing. Her total deafness occurred suddenly in connection with the other indications of cerebral disease. In order to exclude possible local affection of the ears these organs were submitted to examination by Professor Lucae. The result of this examination was practically entirely negative, only a slight dry catarrh being found, and nothing locally to account for deafness. These authors conclude "that the auditory nerves ultimately end in the temporal lobe, and that double or bilateral lesion in these lobes causes complete deafness. It may therefore be stated with all certainty that the temporal lobes are the cerebral centres of hearing."

Though the lesions were not confined to the superior temporal gyri, yet these were involved both as to the grey matter and medullary fibres. The case, therefore, confirms, if it does not of itself suffice to indicate, the position I have assigned to the auditory centre in these gyri.

The affections of hearing with which we are most familiar in connection with cerebral disease are the various forms of what is termed "word-deafness"—a condition in which auditory ideation is impaired more particularly as regards the association of articulate sounds with acts of articulation and things signified. The word-deaf is not devoid of auditory sensation, for he can hear the ticking of a watch, and can recognise and hum an air, but articulate sounds, except, perhaps, his own name or some simple combination of words, have no meaning, and cannot be repeated. Word-deafness has been found associated with affection of the superior temporal gyrus in the left hemisphere. Seppili¹⁵ finds that of seventeen cases in which a *post-mortem* examination was made in every one there was lesion of the superior temporo-sphenoidal convolution, and twelve in which also the second or middle convolution was involved.

Of twenty-five cases of word-deafness of which I have notes, collected by Dr. Ewens, ten were from lesions of the temporal lobe alone. In seven of these the first temporal gyrus was particularly affected; in the remaining three, the exact limits of the lesion were not stated. Eight were from lesions implicating the angular as well as the upper temporal gyrus; six were from lesions invading the superior temporal gyrus and adjoining portions of the occipital or parietal lobes, and one was stated to have been due to lesion of the angular gyrus alone. In this case there appears to have been both word-blindness and word-deafness. In all, therefore, except one, there was obvious lesion of the superior temporal convolution.

Further confirmation of the localisation of the auditory centre in this convolution is afforded by cases of auditory discharges or subjective auditory sensations in connection with irritative lesions implicating this gyrus. Gowers has reported two cases of this nature.¹⁶ In the one a tumour, 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 tumour affecting the superior temporal gyrus caused unilateral convulsions, preceded by a loud noise as of machinery. And Hughes Bennett¹⁷ has reported several cases of auditory sensory discharges followed by 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 the more so. The superior gyrus has also been found atrophied in cases of long-standing deafness or congenital deaf-mutism. Mills¹⁸ records the case of a man deaf for thirty years: the brain, otherwise normal, showed extensive atrophy of both superior temporal gyri, more particularly on the left side. Broadbent¹⁹ describes the brain of a deaf and dumb woman in which, in addition to some defect of the unconnected convolutions and supramarginal lobule, there was atrophy of both superior temporal gyri more marked on the left side. The facts of human pathology, therefore, undoubtedly support the view that the sense of hearing is localised in the temporal lobe, and more especially in the superior temporal gyrus of this lobe.

¹⁵ *Revist. Speriment. di Frenist*, vol. x, 1884.

¹⁶ *Diseases of the Nervous System*, vol. ii, p. 21.

¹⁷ *Sensory Cortical Discharges*, *Lancet*, 1889.

¹⁸ *University Medical Magazine*, November, 1889.

¹⁹ *Journal of Anatomy*, 1870.

¹⁴ *Fortschritte der Medicin*, Bd. 1, No. 6, March 18th, 1883; *Brain*, April, 1883, p. 19.

The experiments of Baginsky,²⁰ as well as the microscopical investigation of Flechsig and Bechterew,²¹ indicate that the auditory nerve is in relation with the auditory centre of the cortex through the lower fillet of the opposite side, and thence by means of the posterior tubercle of the corpora quadrigemina and corpus geniculatum internum with the medullary fibres of the cortex. Baginsky's experiments consisted in destroying the labyrinth in rabbits, and then tracing the course of the paths of degeneration thereby caused. He found a marked disappearance of the fibres of the lower fillet in the opposite side, and some degree of atrophy also in the posterior tubercle and brachium and corpus geniculatum internum. Von Monakow also states that, after extirpation of the temporal lobe in newborn rabbits, atrophy occurs in the corresponding medullary fibres of the internal capsule and in the corpus geniculatum internum of the same side; thus furnishing confirmation of the views of Baginsky and Flechsig.

We have thus grounds for believing that the central fibres of the auditory nerves do not, as Meynert has stated, all pass through the cerebellum on their way to the cerebral hemispheres—a hypothesis which is otherwise inconsistent with the results of destruction of the cerebellum itself. Some of the fibres of the eighth nerve undoubtedly pass into the cerebellum, but these appear to be the vestibular fibres from the semicircular canals, and not the cochlear or true nerve of hearing.

CENTRES OF TACTILE SENSIBILITY.

I will now proceed to consider the locality of the centres of common and tactile sensibility.

Many uncertainties still exist as to the paths and centres of the various forms of common sensation. It is universally admitted, since the classical experiments of Brown-Séquard, that, with the exception, perhaps, of the so-called muscular sense, the paths of all the other forms of sensibility are conveyed upwards on the opposite side of the spinal cord. But neither experimental, nor pathological, nor microscopical investigation has accurately determined in which particular part of the opposite side of the spinal cord the sensory tracts ascend to the brain. The experiments of Ludwig and Woroschiloff²² appear to show that sensory impressions may be conveyed upwards, without any apparent disturbance of the normal relations, when the whole of the anterior and posterior columns, as well as the grey matter, has been severed; when, therefore, only the lateral columns of the cord remain intact. They found that, when only one lateral column remained, movements of the arms and anterior part of the body could be readily excited by irritation of the opposite leg behind the section, but only with difficulty by irritation of the leg on the same side. In order that impressions on the opposite leg should readily excite movements in the anterior part of the body, they found that that portion of the lateral column must remain intact which lies in the area bounded by the prolongation outwards of the anterior and posterior commissures, that is, the middle third. Ludwig and Woroschiloff were not able to differentiate the sensory from the motor tracts of the lateral columns, and concluded that both were more or less mingled together; but the facts of human and experimental pathology indicate that they are, to a large extent at least, distinctly separable from each other, and doubt may be entertained as to how far their experiments indicate the paths of true sensation as distinct from those of mere reflex reaction, more or less general. It does not appear that the paths of sensation proper degenerate upwards to any great extent after complete severance of the cord at any part. Of the paths which degenerate upwards the principal are the posterior median columns, or columns of Goll, which degenerate upwards, in some measure at least, as far as the nuclei graciles or post-pyramidal nuclei of the medulla oblongata. The posterior external columns, or columns of Burdach, degenerate upwards at most for a distance of only a few roots. In addition, upward degeneration occurs in the direct cerebellar tracts, which can be traced continuously to the restiform bodies, and thence into the superior vermiciform process of the cerebellum. Anterior to the direct cerebellar tract, but more or less distinct from it, there is another tract in which ascending degeneration is frequently observed, as was first pointed out by Gowers, a tract which, as Bechterew has shown,²³ differs in its period of develop-

ment from the other tracts of the cord. This constitutes the antero-lateral column. None of these tracts, however, have been conclusively shown to be the path of any form of sensation proper, using this term to distinguish the paths of conscious from those of mere afferent or centripetal impressions.

As the direct cerebellar tract undoubtedly ends in the cerebellum, and, as the observations of Tooth²⁴ would seem to show, is mainly derived from the posterior roots of the upper dorsal and cervical regions, and not from those of the lower extremities, we may eliminate these from the paths of sensation proper. The antero-lateral tract in which Gowers is disposed to place the path of painful impressions, appears, from the researches of Tooth, to consist largely of very fine fibres, and to arise from the cells of Clarke's column. It ascends to the nucleus lateralis, which is the upward continuation of the lateral horn of the cervical cord, also called the visceral horn. The further course of this tract is uncertain though its larger fibres are supposed by Tooth to ultimately reach the cerebellum. Gowers reports a case of unilateral lesion of the spinal cord²⁵ which would appear to support his hypothesis, and Bechterew states that analgesia follows transverse section of the anterior half of the cord. In neither of these observations, however, as Tooth remarks, can we eliminate affection of the grey matter itself.

I have lately performed an experiment on a monkey in which I divided the convex or outer half of the lateral column in the middle of the dorsal region. (Fig. 23.) Though a slight amount of paralysis occurred on the limb of the same side there was no impairment of tactile or painful sensibility on the opposite leg the day after the lesion. The slightest touch on either limb immediately attracted the animal's attention. In another, in which I divided the greater portion of one half of the cord, excluding the posterior column, part of the anterior column, and that portion of the lateral column lying in the angle formed by the anterior and posterior cornua (Fig. 24), there was almost complete motor paralysis in the leg of the same side, but sensibility was not abolished on the opposite side. Whether sensation was at all impaired could not be determined with certainty, but the sense of pain was undoubtedly retained. These experiments are, therefore, opposed to the hypothesis that the antero-lateral tract is the path either of tactile or painful sensation.

In another experiment which I performed on a monkey, the particulars of which I have elsewhere recorded,²⁶ I divided the

Fig. 23.

Fig. 24.

Fig. 25.

whole of the left side of the cord with the exception of the anterior and posterior median column. Though the greater portion of the left posterior median column and the whole of the right posterior median column, as well as the grey matter on the right side, and that surrounding the central canal on the left side were intact, there was complete anæsthesia and analgesia on the opposite side of the body. This experiment, therefore, negatives the hypothesis that the posterior columns are the paths of tactile sensibility. It also is opposed to the hypothesis that the posterior median column is the path of the so-called muscular sense on the same side. For the behaviour of this animal indicated, so far at least as can be judged from the observation of the lower animals, that it had entirely lost muscular sense on the opposite limb; for while it was able to move its right leg volitionally in all directions, and for all purposes, without any appearance of uncertainty or ataxy, and could grip strongly with its foot, this was only when vision was free. When, however, the eyes were blindfolded the animal was utterly unable to extricate its leg from any opposition to its intended movements.

In another experiment I endeavoured accurately to divide the posterior median columns in the mid-dorsal region. The lesion was effected by plunging a triangular keratome into the posterior median fissure to such a depth—as I had calculated by repeated

²⁰ Sitzungsber. Akad. d. Wissenschaften zu Berlin, 1886, 12.

²¹ Neurolog. Centralblatt, December, 1886.

²² Der Verlauf motorischen und sensiblen Bahnen durch das Lendenmark des Kaninchens, 1874.

²³ Neurolog. Centralblatt, 1885.

²⁴ Secondary Degeneration of the Spinal Cord, 1889.

²⁵ Clinical Society's Transactions, vol. xi, 1877.

²⁶ Hemisection of the Spinal Cord, Brain, vol. vii, p. 1.

experiments on the dead spinal cord—as would divide the posterior median columns down to the posterior commissure. Though it seemed as if the whole of the columns should have been severed, I could not after death verify greater destruction than of the portions immediately adjoining the median fissure. (Fig. 25.) Though the animal was for a few hours somewhat weak or awkward in its hinder extremities next day, not the slightest impairment of tactile or muscular sense could be discovered. It ran about without any sign of ataxy, climbed with its customary freedom, clambered down the bars of its cage, planted its feet with precision and without the aid of vision, and was at once aware when its feet reached the ground. The slightest touch on either foot, or on any portion of the lower part of its body, at once attracted its attention.

Bechterew has also found²⁷ that section of the posterior columns in the cervical region in dogs causes no loss of tactile or muscular sensibility, though the operations appear to be followed by disorders of equilibration. These, however, tended to disappear with the lapse of time. Bechterew's experiments, therefore, are opposed to the view that the posterior columns are the paths of any of the forms of sensation proper.

It has been stated by Brown-Séquard, and generally accepted by physiologists and pathologists, that the paths of the muscular sense do not cross with the other sensory tracts, but ascend in the cord on the same side; so that in hemisection or unilateral disease of the spinal cord, the muscular sense is impaired or lost on the side of the lesion and retained on the opposite and otherwise anæsthetic limb. The facts of hemisection of the spinal cord in the monkey are, in my opinion, opposed to this hypothesis, but I am ready to admit that experiments on the lower animals, in which we can only infer as to the conditions of consciousness, are not so satisfactory in this relation as accurate observations on human beings. On investigation, however, of the cases which have been adduced in support of Brown-Séquard's contention, I find that the evidence of the retention of muscular sense on the otherwise anæsthetic limb, and its impairment or loss on the side of lesion, is far from satisfactory.

Out of 43 cases²⁸ of apparently unilateral disease of the spinal cord in which, however, there were only two *post-mortem* examinations, in only 24 was there any mention of the condition of the muscular sense in the original papers. In 6 the only evidence given of the muscular sense being retained was the ability to perceive strong pressure or the power of directing movements with precision; a condition, however, which has been proved to be possible in the entire absence of any sense of movement. In 4 the method of testing the muscular sense is not stated. In 1 muscular sense appeared to be normal on both sides, no further details being given. In another the only note was that the paralysed limb did not judge as exactly as the other side difference in weights. In 3 the muscular sense²⁹ was present on the otherwise anæsthetic side, but in these three tactile sensibility was not lost. In one case³⁰ muscular sense appeared to be lost on the paralysed leg; but in this case there was some anæsthesia of the arm on the same side, and probably, therefore, also on the leg. In one no investigation seems to have been made of the muscular sense while the limb was paralysed. Afterwards it was said to have a certain degree of inco-ordination and loss of motion of position. In another³¹ the only note was that the patient retained the power of estimating weights and the consistency of bodies on the anæsthetic limb. The state of the other was not mentioned, and no exact details are given as to the condition of the various forms of sensibility. Of the remaining cases, in one muscular sense was lost on the anæsthetic side.³² The condition of the other is not recorded. In another³³ the conditions varied according to the progress of the disease, but the muscular sense was always retained on the anæsthetic limb, so long as tactile sensibility and the power of localisation were unaffected. In the third³⁴ muscular sense was retained on the paralysed limb, though its condition on the otherwise anæsthetic limb was not mentioned. This was the only case which was followed by a *post-mortem* examination. In another, Köb-

ner's case,³⁵ it was found, on applying proper tests to the paralysed limb, that the patient had a perfect knowledge of any movement passively communicated to it; and a similar case has been recorded by Jaccoud.³⁶ Though, therefore, there appear to be some facts in favour of Brown-Séquard's theory, others are absolutely opposed to it. So that clinical observation hitherto cannot be said to give unqualified support to the theory that the muscular sense remains unaffected when all the other forms of sensibility have been lost. Such being the state of the question, the particulars of the following case, which I have recently had under my observation, are of some importance.

W. S., aged 25, admitted as an out-patient at the National Hospital for the Paralysed and the Epileptic on March 21st, 1890. Previous health had been good until three years ago, when he contracted syphilis. At Christmas, 1888, he observed that he had difficulty in emptying his bladder. Soon after this he had temporary paralysis of the sphincters, both of the bladder and rectum. In April, 1889, he complained of weakness of the left leg, which passed off after a few months, at the end of which time the right leg became affected and has continued so, becoming more and more rigid up to the present date. On examination, there is tenderness to percussion from the tenth to the twelfth dorsal vertebra, and to a lesser extent for some distance above this point. The patient complains of constriction round the abdomen, just above the umbilicus. There are no abdominal or cremasteric reflexes on the left side, but both are present and well marked on the right. Condition of the limbs: The right leg is parietic and rigid. The knee-jerk is greatly exaggerated, and there is well marked ankle clonus. In this leg all varieties of sensation are normal. When his eyes are closed, he can indicate with perfect accuracy every movement that is communicated to his limb. There is weakness of the left leg, and the knee-jerk is increased. There is no complete anæsthesia, but there is absolute tactile anæsthesia from the foot up to the knee, and dulled from this to the level of the umbilicus. Sensibility to heat and cold is very much impaired. With his eyes closed he is absolutely unable to say in what position his limb or any part of his limb is placed, but he is able to direct its movements with fair precision. In another case, the patient suddenly became paralysed on the left leg, and anæsthetic on the right. This fact he discovered by the insensibility of the leg to hot water which he could not bear with the right. At the date of my examination, a month after the attack, he had recovered from the motor paralysis of the left leg, but he was still absolutely insensible to thermal and painful stimuli on the right leg. Tactile sensibility was, however, equally good on both sides, and with it the muscular sense was also perfect in both. To my mind the evidence, from the various facts mentioned, is in favour of the view that the whole of the sensory paths pass up the opposite side of the spinal cord, and that they are not contained either in the posterior median column, or in the direct cerebellar tract, or in the antero-lateral tract; and, as the pyramidal tract may be entirely sclerosed without any affection of sensation, we are led by a process of exclusion to suppose that the sensory tracts ascend in immediate relation with the central grey matter. If the sensory tracts retain constant relation with the grey matter, this would account for their non-degeneration upwards like the other continuous afferent tracts of the cord.

²⁷ *Archiv f. klin. Med.*, 1877, p. 208.

²⁸ *Léçons de Clin. Méd.*, 1887, p. 451.

HEALTH OF THE SPANISH ARMY.—A report recently published by Dr. Pacheco, Subinspector of the Spanish Army of the First Class, shows that in 1886 the strength of the Spanish Army was 100,434, of whom 1,355 died. Of these deaths, 2.12 per mille were due to typhoid fever, 2.66 to tuberculosis, 1.42 to pneumonia, 1 to small-pox, and 12.21 to yellow fever (in Cuba). The sickness from malaria was 29.94, and that from venereal diseases 35.69 per mille. In Cuba the sickness from the last named cause amounted to 58.6, while in the Philippine Islands it was only 4.81 per mille.

PUBLIC HEALTH ADMINISTRATION IN ITALY.—On the initiative of Professor Carlo Ruata, of the University of Perugia, a society of Italian medical men, bearing the title of "Italian Association for the Triumph of State Medicine," has recently been formed for the purpose of "freeing society from preventable diseases and death by means of wise medico-social arrangements." The Association proposes to take part in public affairs with the object of making sanitary administration autonomous, by placing the direction of everything relating to the public health in the hands of Government officials entirely independent of local authorities.

²⁹ *Neurolog. Centralblatt*, February 1st, 1890.

²⁸ Most of these cases have been quoted by Brown-Séquard in the *Archives de Physiologie*, vol. 1 and 2.

²⁹ Perroud, *Journ. de Méd. de Lyon*, vol. ix, 1868; Gilbert, *Archives de Neurologie*, tome 3, p. 275; Bayne, *Lancet* ii, 1865, p. 117.

³⁰ Brown-Séquard, *Lancet*, vol. ii, 1868, p. 689, Case 2.

³¹ Dundas, *Edin. Med. Journ.*, 1825, p. 304.

³² Sir Charles Bell's case, *Nervous System*, p. 245.

³³ McKenzie's case, *Lancet*, 1883, vol. i, p. 995.

³⁴ Charcot and Gombault, *Archives de Physiologie*, vol. v, p. 144.