THE GOULSTONIAN LECTURES
ON
THE LOCALISATION OF CEREBRAL DISEASE.

Delivered at the Royal College of Physicians of London.

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LECTURE III (*concluded*).—*March 22nd.*

Lesions of the Parieto-Temporal Region.—There remains, therefore, a region, situated between the motor area and the occipital lobes, in which it is natural to look for a central differentiation of these tracts. This region includes the supramarginal lobule and angular gyrus or inferior parietal lobe, the convolutions of the temporo-sphenoidal lobe on its external and internal aspect, viz., the superior, middle, and inferior temporo-sphenoidal convolutions, the occipito-temporal convolutions (lingual lobule, fusiform lobule), the uncinate gyrus, and hippocampus major or cornu Ammonis. (Fig. 1.) We may call the whole of this the parieto-temporal region. It has, I think, been shown conclusively that experimental lesions of the cortex in this region in the lower animals—a region in which I claim to have demonstrated the existence of individually differentiated centres of special sense—are capable of producing impairment or paralysis of sensation on the opposite side of the body.

This has been shown more particularly as regards vision (which seems specially to have been investigated), by the experiments of McKendrick on pigeons, and by those of Hitzig, Goltz, etc., on dogs. Without precisely defining the regions, lesion of which causes sensory disturbances, we may take it as firmly established that unilateral lesions of the cortex are capable of causing such effects in the lower animals. And here it will be convenient to consider the views advanced by Goltz, with respect to the effects of cortical lesions.*

According to Goltz, it is not so much the position as the extent of the injury on which the phenomena of cortical lesions depend. These he finds to be a conjunction of motor paralysis or paresis, tactile anæsthesia, and blindness or impairment of vision on the opposite side. I need scarcely say, from what I have already brought before you, that if that is the type of cortical lesions in the dog, then we must look upon canine and human pathology as having no resemblance to each other. But it requires very little examination of Goltz's facts to discover that his views are equally at variance with the facts themselves, as with those of clinical medicine and pathology. Instead of laying bare a distinct region in the brain, and accurately limiting his destructive lesion to the part the functions of which he is desirous to investigate, he merely trephines a hole or holes in the temporal region and destroys the cerebral substance by squirting it out with a strong stream of water. This method he adopts in order to avoid risk of hæmorrhage or subsequent meningitis; and therefore, to keep the animal alive as long as possible. While we may credit it with securing the latter object more or less, it is clearly impossible to say what extent of brain-substance may thus be rendered functionless; and that it produces profound derangement of the whole cerebro-spinal system, is evident from the frequently fatal consequences resulting from this procedure. The extent of grey matter destroyed or rendered functionless, Goltz himself admits, it is impossible to estimate, and he nowhere attempts it in the record he has given of his experiments.

These are fatal objections to Goltz's experiments, as bearing on the question of cerebral localisation. They are to be looked upon as experiments only on cerebral lesions. The explanation of his results is, I think, easily afforded by the facts to which I will presently call your attention, as well as by the abovementioned experiments of Veyssière as to the effect of lesion of the posterior part of the internal capsule. These latter, however, Goltz seems to have altogether ignored, as he makes no allusion to them.

The situation usually chosen by Goltz for his trephine openings and syringing operations is such as to, almost without fail, ensure damage of the sensory fibres of the internal capsule; and he has, in a rude way, practically produced the same results as Veyssière obtained by careful limitation of his experimental lesion.

While Goltz's description of the phenomena themselves resulting from this procedure may be accepted without question, his theory that the effects of cortical lesions depend more on their extent than on their position, must, I think, be unhesitatingly rejected.

I will now give you a brief *résumé* of the results of my experiments on the brain of monkeys, full details of which I have published elsewhere.* On these facts I take my stand, and hold that they establish the differentiation and localisation of special sensory centres in the cortex.

Angular Gyrus (fig. 15 [13] [13^1]).—Electrical irritation of the angular gyrus in the monkey causes movements of the eyeballs, pupils, and head, which are to be taken as reflex or associated signs of subjective visual sensation, for the reason that destruction of this region causes no motor paralysis whatever, whether of eyelids, ocular muscles, or pupils. But unilateral destruction has the effect of causing temporary blindness of the opposite eye, while bilateral destruction causes total and permanent blindness in both eyes. Hence it appears that each hemisphere is in relation with both eyes, and the destruction of this centre in one hemisphere is not necessarily followed by complete or permanent blindness. This conclusion is confirmed by Goltz's experiments on dogs, and is in harmony with the researches of Landolt in regard to the affection of both eyes in cerebral hemianæsthesia depending on lesion of the posterior third of the internal capsule.

Superior Temporo-sphenoidal Convolution (fig. 15 [14]).—Electrical irritation of this region causes twitching of the opposite ear and other reflex indications of excitation of subjective auditory sensation. Destruction causes no motor paralysis whatever; but, though it is certain that hearing is at least impaired on the opposite side, the difficulty of ascertaining the conditions of auditory perception in animals, when only one ear is affected, is such as to render it impossible to speak definitely as to the extent and duration of the affection of hearing; whereas, when these centres are destroyed bilaterally, there seems to be total loss of the sense of hearing; meaning by that, auditory discrimination as contradistinguished from mere auditory reaction.

Subicular Region.—Irritation of the lower extremity of the temporo-sphenoidal lobe, or region of the subiculum cornu Ammonis, causes movements of the nostril and head indicative of excitation of subjective olfactory sensation. Destruction causes no motor paralysis, but is followed by loss of smell on the same side; and, when the lesions invade not merely the subiculum but the neighbouring regions on one side, taste also is affected on the opposite side of the tongue. Bilateral lesions cause complete loss both of taste and smell.

Hippocampal Region.—On account of the concealed position of this region, it is impossible to cause localised irritation free from all complication; nor is it possible to destroy it, without injuring other parts of the hemisphere. I found, however, that only in cases in which this region was involved along with others, there occurred impairment or abolition of tactile sensation on the opposite side; and when the region of the hippocampus and uncinate gyrus was ploughed up in such a manner as to avoid the internal capsule and the medullary fibres of the other cortical regions (with the exception of part of the occipital lobe), tactile sensation was abolished on the opposite side, sight and hearing remaining unimpaired. A condition resembling motor paralysis was also induced; but in reality, a functional paralysis depending on the abolition of tactile and muscular sensation, such as occurs from division of sensory nerves.

Such being the indications furnished by experiments on monkeys, we may now proceed to consider the effects of disease of the corresponding regions in the human brain. The facts I am about to quote seem to point to a remarkable, and apparently irreconcilable, discrepancy between human pathology and experimental physiology. Cases are on record in which lesion or some form of degeneration has been found in one or other of all these so-called sensory areas, and in which no affection of sensation has been observed. Lesions here are usually said to be latent.

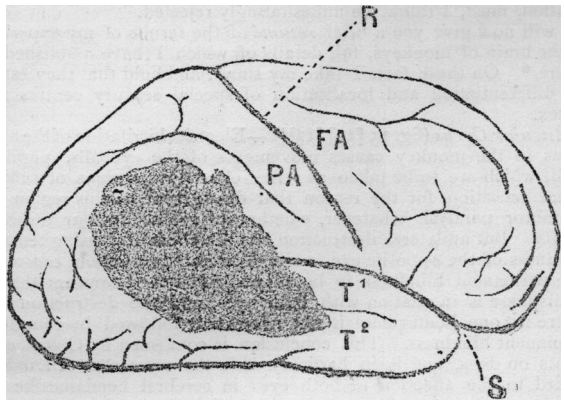
First, as to the cases: MM. Charcot and Pitres† report a case, latent as regards symptoms, in which there was found a yellow softening of the cortex of the right hemisphere, occupying the posterior half of the island of Reil, the posterior two-thirds of the inferior parietal lobule, comprising the angular gyrus, and the upper or posterior half of the second and third temporo-sphenoidal convolutions. (Fig. 25.) There was no secondary degeneration of the spinal cord.

M. Pitres‡ records a case of extensive hæmorrhage into the medullary substance of the left temporo-sphenoidal lobe, in which, though

* Pflüger's *Archiv für Physiologie*, Band xiii, Heft i; 1876.

* *Philosophical Transactions*, vol. ii, 1875; *Functions of the Brain*, chap. ix.
† *Revue Mensuelle*, No. 1, p. 10.
‡ *Lesions du Centre Ovale*, p. 54,

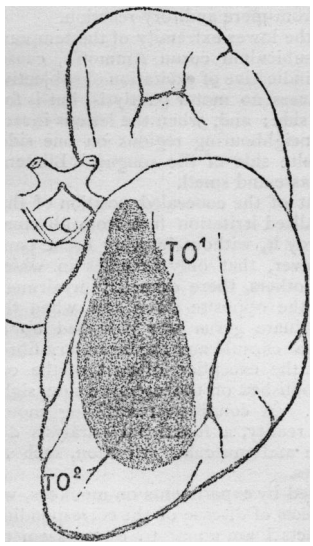
consciousness was deeply affected, there was no real paralysis and no lateral distortion or conjugate deviation of the eyes. He quotes a case reported by Thibault,* also latent as regards sensory or motor symptoms, in which, in addition to a layer of extravasation on the posterior three-quarters of the left hemisphere, there was found, in the



sphenoidal lobe, a large extravasation extending from its anterior extremity to within three *centimètres* of the posterior extremity of the hemisphere.

Sabourin† has recorded a case of extensive lesion of the sphenoidal and occipital convolutions in which there was no paralysis.

MM. Charcot and Pitres‡ also give a case in which the chief symptoms were a state of dementia and very marked itching of the chest and abdomen without apparent cause. After death, a yellow softening was found in the left hemisphere, occupying the first and second occipito-temporal convolutions, commencing about one *centimètre* behind the anterior extremity of the temporo-sphenoidal lobe, and extending backwards to within about three *centimètres* from the tip of the occipital lobe. (Fig. 26.)



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M. Sabourin communicated to MM. Charcot and Pitres a case exactly similar to the above, and another in which a yellow softening existed in the *cuneus* and posterior two-thirds of the quadrilateral lobule of the left hemisphere, likewise latent as regards symptoms.

A case is reported by Humbert§ of abscess in the anterior and inferior part of the right temporo-sphenoidal lobe, resulting from suppuration of the ear, in which there were no special symptoms indicative of such a grave cerebral lesion.

Of similar affections of this part of the brain in connection with otitis, and with nothing but the general symptoms of cerebral abscess, I might quote a multitude. References are given by Pitres to cases of this kind related by Ormerod, Sir W. Gull, Blondeau, Haslewood, Homolle, and Renault.

Of lesions specially confined to the hippocampus, I have not been able to find any on record, except those of Bouchet, Meynert, etc., in respect to degeneration or sclerosis of the hippocampus in chronic epileptics. Bouchet,‖ in twelve out of forty-three cases which he examined, noted the existence of sclerosis in one or both hippocampi, but he did not attach any special importance to this; as induration of the brain in chronic epilepsy he looked upon as a general affection, of which this was only a local manifestation. This condition of the hippocampus has been observed also by Cazauvielh, Foville, Lelut, Delasiauve, Bourneville, in epileptics. In 1868, Meynert* called special attention to this degeneration of the hippocampus in epileptics, giving nineteen cases in which one or other hippocampus was indurated or atrophied. Meynert, without looking upon this as *the* cause of epilepsy, thought that there was some special relation between this degeneration and the lesion on which epilepsy depended. In a recent paper, Hemkes† states that he has seen atrophy of the hippocampus in only six out of thirty-four cases. Beyond the fact of the existence of such degeneration in epileptics, we have no record of the exact symptoms in the cases in which it was found. Meynert‡ believed that the optic tracts had special relations with this region; and he gives four cases of disease situated in or near the hippocampus and fusiform lobule, in which disorders of vision were observed. The cases, however, are altogether deficient as regards ophthalmoscopic appearances, on which it would be necessary to have some information before coming to a conclusion as to whether the disorders of vision were the direct or indirect results of the cerebral disease.

I have quoted a number of cases of unilateral lesion of the sensory regions, mostly of a chronic form, in which no special symptoms were noted. It may be said that the absence of symptoms in all these cases may be accounted for by functional compensation by the same or the opposite hemisphere. Yet there are on record cases of traumatic lesion, also apparently latent, which would militate against this idea, supposing them in every respect carefully investigated.

M. Herpin§ has recorded a case of fracture of the skull and injury of the brain in the region of the squamous portion of the temporal and greater wing of the sphenoid. The man did not lose consciousness, and, from the time of the accident till death, four days afterwards, nothing was observable, either as regards motility or sensibility. After death, a contusion of the third degree was found in the inferior aspect of the temporo-sphenoidal lobe (which side not stated), extending five *centimètres* in an antero-posterior direction, three *centimètres* in breadth, and affecting more particularly the middle and external (inferior) temporo-sphenoidal convolutions; a situation corresponding exactly to the cranial injury. A clot, of the size of a bean, existed at the anterior extremity of the lesion. A case in some respects similar has been put on record by Alcock.‖ This was a case of cranial injury, followed by restlessness and sleeplessness; and only on the third day after the accident did the patient seem to hear when spoken to. He gradually got worse, and died on the thirty-third day after the accident, without being affected by paralysis. Wilfulness and obstinacy were his most prominent mental symptoms. On the right hemisphere, there was a patch of ecchymosis, of the size of a florin, in the pia mater, over the upper extremity of the superior temporo-sphenoidal convolution, but the brain-substance underneath was not injured. On the left side, "the portion of brain corresponding to the lower part of the squamous portion of the temporal bone was soft and pulpy, being easily washed away by a stream of water, leaving a cavity with ragged walls, the area of which equalled that of half-a-crown, and about a quarter of an inch in depth. The ventricles contained an excess of serum". The only indication as regards sensory affection in this case was the apparent want of auditory perception at first; but whether this was a part merely of the general dazed condition of the man, or as the result of affection of the auditory centres, it is impossible to say definitely, though the position of the lesions might be taken in favour of the latter.

I have not been able to find any cases of bilateral lesions of the hemispheres in corresponding parts of the sensory area without such profound mental disturbance as to render the determination of the existence or absence of sensory impairment a matter of impossibility. This is greatly to be regretted, as on these the question of special sensory localisation in man to a large extent depends.

But if we confine our attention to the cases of unilateral lesion of the sensory area which I have mentioned, and compare the negative results as regards sensation, whether with chronic or sudden lesions of the sensory regions in man, with the affections of sensation, carefully observed and confirmed by many physiologists, which result from similar cortical lesions in the lower animals, we cannot but be struck by the discrepancies which exist. To account for these, we must adopt one or other of two suppositions: either, taking the facts as equally well established, that the parallel which has hitherto been shown to exist between the brain of man and that of the monkey and the lower

* *Bulletin de la Société Anatomique*, 1844, p. 93.
† *Bulletin de la Société Anatomique*, October 21st, 1876.
‡ *Op. cit.*, p. 11.
§ *Bulletin de la Société Anatomique*, 1870, p. 367.
‖ "Sur l'Epilepsie", *Annales Médico-Psycholog.*, 1853, t. v, p. 209. Quoted by Lepine, *op. cit.*, p. 130.

* *Vierteljahrsch. für Psychiatrie*, p. 381.
† *Allgemeine Zeitsch. für Psychiatrie*, Band xxxiv, Heft. vi.
‡ *Op. cit.*, p. 400.
§ *Bulletin de la Société Anatomique*, May 1876.
‖ *Lancet*, Marc 10th, 1877.

animals now suddenly ceases to hold, and that, in respect to sensory localisation, the brain of man is constituted on a totally different type from that of the lower animals; or, if this be regarded as improbable, that the latency which is said to characterise lesions of the sensory area in man is a latency not so much of actual symptoms as of observation.

M. Pitres* is of opinion that the sensory fibres which are gathered together in the posterior third of the internal capsule, instead of distributing themselves like the motor fibres to individually differentiated areas in the cortex, spread themselves indifferently over the whole occipito-sphenoidal region. But this does not remove the difficulty; for we should then expect that extensive lesions of this region should cause general impairment of all the sensory powers on the opposite side together; a hypothesis which the clinical records no more support than that of special localisation. And it would seem strange if there should be a distinct differentiation of centres of motion, and a general *confusion* in the centres of organs so highly specialised as the organs of sense. This is a supposition which I cannot entertain, and for which I see no substantial grounds. That the organs of sense may be more bilaterally represented in each hemisphere in man than in the lower animals, is not impossible. That bilateral representation does exist to a large extent, and particularly as regards sight and hearing, is in accordance with the facts of experiment, and is sufficient to account for the absence of any very obvious impairment of these faculties in cases of unilateral lesion of a slowly progressive character. But that there is no impairment at all of sensory perception or discrimination in *sudden* unilateral lesions, or even in *chronic* lesions, is a fact which I do not admit as proved; and I adopt the alternative supposition, that the latency has been in observation rather than in symptoms.

This may seem a sweeping charge, and a very summary method of disposing of difficulties; but I cannot help expressing the frequent disappointment I have experienced in reading through multitudes of cases of cerebral lesion, which might have served to throw light on this subject, and finding no indication of any attempt having been made to investigate the conditions of special sense. Considering the perfunctory manner in which this is so commonly carried out, if investigation be made at all, and the frequent omissions in this respect which are to be found in the records of even our most accurate and competent clinical observers, I cannot take mere absence of remark as proof of negation of symptoms, unless there be clear evidence that the various points had been fully and fairly investigated, and the negation of symptoms positively established. For the clinical facts are not all of the same negative order as those I have brought before you, and many of them are, in my opinion, capable of satisfactory explanation only on the views I have advanced.

Let us take first the question of affections of sight directly dependent on cerebral lesion. Here, of course, we must eliminate all those cases in which impairment or abolition of vision is caused by changes in the optic nerve and retina, secondary to intracranial disease wherever situated. Hence most of the records of cerebral disease, before the invention of the ophthalmoscope, and, I may add, very many since, are for the most part worthless in this relation. But the following case, which is recorded by Abercrombie,† and which I give in his own words, has a special value. "The effect of superficial inflammation of the brain or its membranes is well illustrated by another case related by Dr. Anderson, in which the disease took place under his own eye. A boy suffered from an injury of the head, the depression of a considerable portion of the *right parietal bone*, the depressed portion being forced through the dura mater and driven inwards upon the brain. *He had paralysis of the left side, and the left eye was insensible.* The depressed portion being removed, the paralysis was greatly diminished, and the eye recovered a considerable degree of vision. On the third day after the operation, the wound in the dura mater was inflamed, with considerable tumefaction; and immediately the left leg and arm became paralysed, the paralysis being accompanied by convulsion; and *the left eye also became again insensible.* He had frequent convulsion of these parts for several days, the right side not being in the least affected, when, suppuration having taken place, all the symptoms subsided" (p. 121-2). Now, though recovery took place, and therefore the case is incomplete in an anatomical point of view, it is clearly a case of cortical lesion, and possesses all the typical features of such; and that the affection of the opposite eye, which proceeded *pari passu* with the motor symptoms, had a similar cause—viz., lesion of the cortex—is, I think, unquestionable. Though the exact extent and position of the depressed fracture is not stated, yet, as it was in the parietal region, we may conclude that the lesion involved not only the cortical *motor* area, but also the *visual* centre, which is in close proximity to it under the parietal eminence (fig. 3). This case, in my opinion, distinctly confirms the sensory localisation which I have arrived at by experiment, or at least is explicable only in this way.

The same author also quotes another case related by John Bell, in which, from injury to the head, extravasation of blood occurred on the surface of the brain, for which the patient was repeatedly trephined. Local inflammatory attacks with suppuration occurred from time to time on the left hemisphere after the trephining. These local attacks, when they occurred towards the anterior part, were accompanied by double vision; but, "when they were towards the posterior part, there was not double vision, but a state of vision in which a candle was seen with a halo round it" (p. 122).

I mention this case chiefly because it harmonises with the observations of Hughlings Jackson, already referred to, in respect to the frequent association of optical illusions, coloured vision, etc., with disease of the posterior lobes. These spectra are the counterpart of the motor discharges caused by irritative lesions of the motor centres. That they should occur more particularly with lesions situated towards the posterior aspect of the hemispheres, is quite in accordance with the localisation of the visual centre in the angular gyrus. These sensory discharges in connection with epilepsy of cortical origin, whether in the domain of sight, hearing, smell, taste, or tactile sensation, are without doubt to be looked upon as indications of irritative lesion of the sensory centres, though we have not yet sufficient material to enable us, from a purely clinical point of view, to connect any particular form of sensory discharge with a specially localisable lesion, unless we regard it as established in respect to optical illusions. Not unfrequently the sensory centres are discharged together, as in a general unilateral convulsion, and there is no clear discrimination of one form of sensation from another. This was well exemplified and graphically described to me by a highly intelligent patient, who told me that his epileptic attacks (*petit mal*) were ushered in by a "horrible smell of green thunder", or by some equally strange compound of smells, colours, and sounds, inextricably intermingled.

Reverting to the impairment of sight in connection with destructive lesions, it has been remarked by Dr. Bastian that not unfrequently, in cases of thrombosis of the posterior cerebral artery, vision is impaired on the side of motor paralysis.* This he attributes to affection of the opposite optic tract, or to the opposite side of the corpora quadrigemina. But, as lesion of the optic tract would seem to be associated rather with bilateral hemiopia than with unilateral amblyopia, and as lesion of the corpora quadrigemina is generally accompanied by more complex symptoms than mere motor hemiplegia, it seems to me that the impairment of vision may be attributed to sudden interference with the visual centre. I advance this only as a suggestion, without pretending to pronounce definitively on the subject.

Apart from the evidence of auditory discharges and subjective auditory spectra of various kinds in connection with epilepsy and other cerebral affections, I cannot find any altogether satisfactory evidence of impairment or abolition of hearing in connection with destructive lesions of the cortex. Hughlings Jackson repeatedly emphasises the statement, that he has never met with deafness as the result of disease of the cerebral hemispheres directly.

But, though we may admit, in accordance with the results of experimental physiology, that unilateral destruction of the centres of hearing and sight need not cause actual blindness or deafness of a complete or enduring character, there are certain facts which tend to show that unilateral lesions of these centres may produce what we may call *subjective* deafness and blindness. Such conditions are not unfrequently classed with aphasia, and may occur with it; but they may occur *without* true aphasia or speechlessness. They have been termed by Kussmaul "word-blindness" and "word-deafness" (*cæcitas et surditas verbalis*). These two conditions may occur separately or in association. In the one case, though a man may be able to speak and write, he cannot translate written symbols into ideas, though he may understand articulate sounds; in the other, he may be able to read, though he cannot understand spoken words, or he may be unable to do either. In neither case is there actual objective blindness or deafness. In a case of word-deafness of this kind reported by Wernicke,† there was, besides a general atrophy of the convolutions, a thrombotic softening of the *first and a large portion of the second temporo-sphenoidal convolution of the left* hemisphere. The auditory centre was thus destroyed. A very interesting case of subjective or word-blindness has been recorded by Dr. Broadbent.‡ The essential points are thus

* *Lesions du Centre Ovale*, p. 53.
† *Diseases of the Brain and Spinal Cord*, second edition, 1829.

* *Paralysis from Brain-Disease*, p. 113.
† *Der Aphatische Symptomen-Complex*, 1874, Case II.
‡ "Cerebral Mechanism of Thought and Speech", *Med.-Chir. Trans.*, vol. lv, 1872.

summed up by him. "After an acute cerebral attack, absolute inability to read printed or written words (except own name), while the patient wrote correctly from dictation, and composed and wrote letters with a little prompting. Inability to recall the name of the most familiar object presented to his sight, while he conversed intelligently, employing an extensive and varied vocabulary, making few mistakes, but occasionally forgetting names of streets, persons, and objects. Death from apoplexy; extensive atheroma of cerebral vessels; old clots in substance of left hemisphere, with softening of adjacent substance to outer side of lateral ventricle, at junction of descending cornu. Recent hæmorrhage in same situation." The primary lesions on which the softening and subsequent fatal hæmorrhage appeared to depend were two old clots. The first, of the size and shape of an almond, was loosely imbedded in the inframarginal gyrus or superior temporo-sphenoidal convolution, about opposite the junction of the upper third with the lower two-thirds of the descending cornu. The other, which Dr. Broadbent regards as the more important and the cause of the softening which led to the fatal hæmorrhage, was a clot of the size of a bean, surrounded by softening, situated at the upper extremity of the fissure of Sylvius externally, and at the junction of the descending cornu with the body of the ventricle internally. This, it will be seen by reference to fig. 16, is in the region of the angular gyrus and supra-marginal lobule, the homologue of the visual centre in the monkey. These cases I take to be in harmony with the views I have elsewhere expressed, that the sensory centres are also the substrata of corresponding sensory memory and sensory ideation. In the one of these cases (Wernicke's) in which the *auditory* centre was the seat of lesion, there was paralysis of auditory ideation; in the other (Broadbent's), in which the *visual* centre was the seat of disease, there was paralysis of visual ideation, more particularly in connection with articulate symbols or their visible equivalents.

The paralysis of visual and auditory ideation in special reference to words in these cases is accounted for by the fact, that in both the disease was situated in the visual and auditory centres of the left hemisphere, between which and the speech-centre we may reasonably suppose there exists a more intimate organic or functional connection than between this and the sensory centres of the right hemisphere. But, as regards sensory discrimination and sensory ideation in general, we have not the same grounds for regarding the right hemisphere as subordinate to the left, as is the rule in respect to voluntary movements and motor ideation; for, with equally acute sensibility on both sides, we find that, for delicate sensory discrimination, some invariably use one eye or one ear in preference to the other, and therefore the opposite cerebral hemisphere. Thus the same individual will use his *right* eye for microscopic work, and his *left* ear for auscultation; which we may take to mean that his left visual and right auditory centres are more especially cultivated and developed.

Hence we may conclude that unilateral lesions of the sensory centres will vary considerably in respect to their effects on sensory ideation, according as the lesion is on the side of the more or less developed centre. It is not impossible, therefore, that what Hughlings Jackson terms "defective perception" may be more common with lesions of the sensory regions of the right hemisphere, if these be more commonly cultivated and developed.

Before passing from this subject, I would refer to an interesting case related by Dr. Banks* of Dublin, in which, though unfortunately no *post mortem* examination could be made, there are certain facts bearing on the question as to whether actual deafness may occur from cerebral disease. In this case, after a sudden cerebral seizure, but without coma or paralysis, the patient was found to be incapable of understanding either speech or writing, though he could both speak and write. He was found to be completely deaf, taking no notice of what was said to him, or even of the loudest noises; and, indeed, he used to allude to his deafness himself. One day, he said he could neither hear nor read; "only a little could read the words, but not take in the meaning". This patient died ultimately of coma and right hemiplegia; but no *post mortem* examination was allowed. Unless we suppose, in this case, that the patient had a separate lesion in both auditory nerves or both ears, occurring simultaneously with his cerebral lesion, we may take it as a case of deafness depending directly on cerebral disease; but whether the lesion was unilateral or bilateral, the absence of a *post mortem* examination unfortunately renders it impossible to decide.

Affections of smell and of taste, we have seen, occur with affections of the other senses in cerebral hemianæsthesia; but affections of smell alone, or of taste and smell combined, may occur without other sensory impairment in connection with certain forms of cerebral lesion.

* *Dublin Quarterly Journal*, Feb. 1865, vol. xxxix, p. 62.

As regards smell, there seems to be some discrepancy between my localisation of the olfactory centre and the facts of cerebral hemianæsthesia. I find that destruction of the subicular region causes loss of smell on the *same* side; while in hemianæsthesia the impairment of smell is on the side *opposite* the cerebral lesion. I have endeavoured to account for this by the fact, discovered by Magendie, that abolition of the *common* sensibility of the nostril by section of the sensory branches of the fifth nerve causes loss of smell; and, as in hemianæsthesia the sensibility of the mucous membrane of the nostril is lost, so we may consider this to be a sufficient cause of the unilateral anosmia. I see no reason to doubt the validity of this explanation; but I would supplement it by another consideration. Though the outer root of the olfactory tract can be directly traced to the subiculum of the same side, it is not unlikely that the inner root passes on to the opposite hemisphere with the other sensory tracts; and hence each hemisphere may maintain a bilateral relation with the organ of smell. If this were so, then the partial impairment of smell, which would result from lesion of the special sensory paths of the opposite hemisphere, would be rendered more complete by the simultaneous abolition of common sensation in the nostril. I cannot give anatomical evidence of this arrangement, for the inner root of the olfactory tract has not been traced by Meynert beyond the corpus striatum; but that it ends here is, I think, more than improbable.

Unilateral anosmia has been observed in many cases of cerebral lesion, and on the same side as the lesion; but, without a necropsy, it is of course difficult to decide whether this was due to direct lesion of the olfactory tract, or of its centre. Several such cases have been reported in connection with aphasia, the anosmia being on the left side.* A good many cases are now on record of loss of smell, or combined loss of smell and taste, as the result of blows on the head, more particularly of the vertex or occiput.†

As regards the anosmia, the mode of causation suggested by Ogle,‡ viz., injury by counterstroke to the olfactory nerves, bulbs, or tracts, seems in every way satisfactory. To the loss of smell, Ogle further ascribes such affections of taste as may be combined with it, viz, the impairment or abolition of the perception of flavours, which are a compound of smell and taste. And, indeed, in many of the so-called cases of loss of taste and smell, taste proper does not appear to have been affected. Hence they may be accounted for in the manner indicated by Ogle. But even when there is absolute loss of smell, we find cases in which taste is but little interfered with. A patient of mine who had suffered from complete anosmia for six years, dating from a fall on his head which had rendered him temporarily unconscious, made no complaint as regarded his power of taste, as he could distinguish all the ordinary articles of food from each other, and could clearly perceive the flavour of onions. Yet, though there was no obstruction of the nasal passages, anteriorly or posteriorly, he could recognise no smell in assafœtida or musk; acetic acid, he said, caused some sensation about two-thirds up the nostril, but no real odour. I have no doubt that in this case there had been rupture of the olfactory nerves or tracts; but the mere loss of smell is incapable of accounting for the symptoms in another case which I have seen. This patient had lost both smell and taste in consequence of a fall on his head into the street six years before. I was not aware until lately that my colleague Dr. Burney Yeo had already brought the particulars of the same case before the Clinical Society,§ and, therefore, the subsequent history of the patient will be all the more interesting. This man had not merely total loss of smell, but also total loss of taste proper, such as for bitter, sweet, salt, sour, etc. One day, in fact, when suffering from pain in the stomach, he swallowed a glass of what he took to be brandy, and was not aware it was vinegar until the aggravation of his pain made him ask his wife what was in the bottle. While under Dr. Yeo's care, and taking iodide of potassium, he recovered taste to some extent, but he did not, as he told me, recover smell, though he once or twice had something like a subjective sensation of camphor or burnt wood. When he left off the iodide, he became as bad as before. This was in 1872. In 1875, when he came under my care for another affection, he had absolute loss of taste and smell, and had given up all thoughts of recovery, and had tried to accommodate himself to circumstances. Again, on the administration of iodide of potassium, taste returned to some extent, but there was no improvement as regards smell, with the exception of an occasional subjective sensation;

* Ogle, *Med.-Chir. Trans.*, 1870; Fletcher and Ransome, BRIT. MED. JOURNAL, April 1864; Hughlings Jackson, *London Hospital Reports*, vol. i, 1864, Cases II, V, XV, XXII.

† See "Collected Cases" by Knight, *Boston Medical and Surgical Journal*, September 13th, 1877.

‡ *Medico-Chirurgical Transactions*, 1870.

§ BRITISH MEDICAL JOURNAL, May 25th, 1872.

and a relapse again occurred on leaving off the medicine. I lost sight of him till the end of 1876, and found him in his original condition. In January, 1877 I began to treat him with the constant current (ten cells gradually increasing) directed transversely through the head in the zygomatic fossæ, varied occasionally by the application of one pole here, and the other on the bridge of the nose. After one or two applications, while he felt somewhat giddy, various subjective smells were experienced during the passage of the current, which he described as "gassy", "rank", etc. At the end of a week of daily treatment with the current, he began to smell strong odours; subjective sensations also occurred at intervals, and taste became more acute. He gradually and steadily improved, and, after a few weeks' treatment, the power of smell returned, so that he could recognise such things as assafœtida, musk, coffee, tobacco. He could readily distinguish between one smell and another, but continued to have some difficulty as to identification of the substance. There has been no relapse, and now (Feb. 28th, 1878), at the end of a year, he continues to enjoy perfect taste, and his powers of smell, which were never very acute, he thinks are as good as ever.

I will not attempt to decide what was the exact *modus operandi* of the galvanic current, whether it acted by stimulation of the olfactory nerve direct, or by stimulation of the cerebral centres of taste and smell; but, as a therapeutic experiment, it may be regarded as worthy of repetition in similar cases. But, as regards the pathology of this case, I think it is evident that both smell and taste were abolished independently of each other, and that we cannot account for the loss of taste by the loss of smell; nor can we say that the olfactory nerves were ruptured. It is also in the highest degree improbable that the loss of taste and smell could have resulted from simultaneous affection of the various nerves concerned in these functions; situated as they are so widely apart from each other, and bound up more or less with others not conjointly affected. But it might well happen that such a blow on the vertex as this man received would cause such injury to the subicular regions, by what is usually termed counterstroke, or by what Duret terms the *cône de soulèvement*, as to cause impairment or temporary abolition of the functional activity of the cerebral centres of taste and smell, which, as experiments on monkeys indicate, are here localised. Hence I would take this, and similar instances in which smell and taste proper are abolished by cranial injuries, as clinical corroboration of physiological experiment.

In respect to tactile sensation, though this form of sensibility is more frequently affected than any of the others by cerebral disease, it is extremely difficult, from a clinical standpoint only, to establish a distinct relation between this and certain cortical lesions, or localise the centres of tactile sensation. Motor paralysis and tactile anæsthesia are frequently associated with each other. But that the cerebral centres of motion and tactile sensation are distinct from each other, is evident from the fact that we may have the most complete motor paralysis without impairment of tactile sensation, as is the case with cortical lesions. And though motion is more or less impaired by the abolition of tactile sensation (by which motion is mainly guided), yet we have many instances in which the power of voluntary motion is retained notwithstanding the complete annihilation of tactile sensation, cutaneous or deep. There is, therefore, no organic fusion of the motor and tactile centres with each other, seeing that each may be affected independently of the other, and the two do not vary quantitatively with each other when they are conjointly affected.

The facts of cerebral disease in general, and of cerebral hemianæsthesia in particular, would seem to show that in respect to tactile sensation there is less bilateral representation in each hemisphere than as regards the other forms of sensibility. For, in central hemianæsthesia, tactile sensation is always most deeply affected, and may still remain greatly impaired after all the other forms of sensory impairment have disappeared. Hence, in the slighter forms of affection of the posterior third of the internal capsule, tactile sensation only may be impaired. Hence, also, with motor paralysis due to lesion of the anterior division of the internal capsule, we frequently get partial or temporary impairment of tactile sensation, owing to pressure on, or slight organic or functional derangement of the posterior or sensory fibres.

If, therefore, tactile sensibility be more unilaterally represented in each hemisphere—and this we might conclude from the remarkable power we possess of localising the seat of tactile impressions on any part of the body—we should naturally expect to find that lesions of the cortical centres of tactile sensation should be accompanied by symptoms of impairment or abolition of this sense. These centres, as I have already indicated, are situated in the hippocampal region. Lesions of this region are not, however, common; and I have not been able to find any cases of localised lesion of the hippocampus, except those to which I have already alluded in connection with chronic epilepsy and insanity. But, as we have no record of the facts relating to the condition of tactile sensation in these cases, I must leave this question to be settled by future clinical investigation.

There are, however, some facts which would seem to indicate that lesions in the neighbourhood of the hippocampus do cause affection of tactile sensation, though doubt may be entertained as to whether the phenomena are dependent on direct affection of the hippocampus, or affection of the posterior front of the internal capsule, directly or indirectly.

Mr. Jonathan Hutchinson* concludes, from his observations on cranial injuries, that contusion of the sphenoidal lobe more particularly, causes, along with partial motor paralysis, paralysis of tactile sensation on the opposite side of the body. As I have said, these effects may be attributed to injury of the sensory fibres of the internal capsule; but contusion of the sphenoidal lobe might also be interpreted as injury of the hippocampal region; and if the impairment of sensation in the cases described by Hutchinson were to be proved absolutely restricted to *tactile* sensation, we should have good grounds for considering the phenomena dependent on lesion of the cortical centres here situated. The definitive settlement, however, of these various points must be left to future research.

I have now brought under your notice a considerable number of facts, both positive and negative, in reference to the localisation of special sensory regions in the human brain; and though the positive clinical evidence is as yet comparatively scanty, and leaves much to be desired, I entertain the hope and belief that it will not long remain so. And I trust that those who rely more on the evidence of human pathology and the phenomena of disease than on the facts of experiment, even on the most human of the lower animals, and do not, therefore, share my own very decided convictions as to the localisation of special sensory regions, will take the facts I have adduced into careful consideration, and, when opportunities occur, investigate the conditions as to sensation in cerebral disease, with the accuracy and thoroughness which are much needed in order to clear up the doubts and obscurities which still surround this important question.

CROUP: DIPHTHERIA: TRACHEOTOMY.†

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I HOPE I shall be excused by this society for so persistently bringing before it the well worn subjects of the heading of my paper. But, although these subjects have now for some years past anxiously engaged the attention of our profession, it does not appear, from the long and interesting correspondence in the *Lancet* during the year 1875, and the discussion at the Royal Medical and Chirurgical Society in the latter part of that year, that any decided opinion has been arrived at; still, the tendency of opinion (led chiefly by the authority of Bretonneau, Trousseau, Sir William Jenner, Dr. George Johnson, Dr. Semple, and others) is clearly in the direction of admitting that membranous croup is always diphtheria, *i.e.*, dependent only upon the specific contagium of diphtheria. Dr. West, whose excellent article on croup has always appeared to me to express the truth, in holding the opposite opinion, confesses that he is wavering; and there are many others in the same position as himself.

To no members of our profession is this matter of so much importance as to us who live in the provinces; especially to those of us who are in lone country districts. Croup is of more frequent occurrence in the country; and as many of us are compelled to act upon our own responsibility and act promptly, it is most desirable that we should be well supported by the authority of the leaders of our profession in any views we may feel ourselves compelled to adopt. Suppose, for example, one of us has lost a case of croup in a large country house—in the house of our best patient—that we had looked upon the case as catarrhal and inflammatory, and had treated it with tartar emetic or calomel, the family goes to town and is there told that "such theories are exploded, that the case was certainly one of diphtheria and ought to have been treated by wine, tincture of iron, or such like remedies, and that it was no wonder the child died". The happiness of the family is gone, and the reputation of the doctor with it. It well behoves us, therefore, to give anxious consideration to this question, and to think for ourselves; we have, I think, more opportunities of making accurate and complete investigations into the origin of our cases, than

* *Medical Times and Gazette*, 1875, p. 165.

† Read before the East Kent District of the South-Eastern Branch of the British Medical Association.