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The frequency with which I have observed some sensory disturbance to be associated with Hemiplegia during a two years' residence in a Workhouse Hospital has led me to pay some attention to this subject, in the direction of estimating the numerical incidence of such phenomena, their detailed extent, and if possible something of their diagnostic importance.

So far as I can learn, little or, to be more accurate, no attention is paid by the majority of clinicians to such sensory manifestations as may exist in a case of Hemiplegia, possibly because they cannot always be demonstrated, possibly because they have not been considered of sufficient worth.

Among the pauper classes the causal factors of arterial degeneration are unfortunately all too common, and this somewhat undesirable state of affairs tends to increase even the relative frequency of cases of cerebral haemorrhage in a Workhouse. Besides such cases, however, there are met with many examples of the late effects of Hemiplegia from other and various causes, which, though sufficient to exonerate the patient from the necessity for self maintenance, yet have not proved incompatible with life. To such an institution many patients eventually drift who have gone the round of various general hospitals, and have served as subjects for many clinical demonstrations. As these unfortunates almost of necessity tread the downward path, the last step lands them perforce at the Workhouse, which for them is the end of all change, and constitutes their permanent residence for the remainder of their days.

Such are the cases on which for the most part I have made my observations which have been repeated again and again on the same patient so that confirmation of any particular

point has been assured.

Most of the motor phenomena in connection with cerebral disease have been thoroughly investigated, and the explanation of the sequence of motor symptoms following any cerebral lesion is now more or less generally accepted. Hitzig and Fritsch in 1870 were the first to prove by experiment that definite movements of the limbs followed electrical stimulation of certain regions of the cortex. Ferrier followed their lead in 1873, and since then, he, Charcot, Bastian, Dana, and numerous other observers have attempted to localize not only the motor but the sensory centres in the cerebrum.

But while the study of motor phenomena is comparatively a simple matter, the investigation of sensory lesions is beset with difficulties, and the variable, often contradictory results of the experimentalists leave much still to be done in the field of sensory cerebral localization. Here experimental evidence is not of the same value as in motor phenomena because of the many inaccuracies which must almost of necessity arise. Gross effects resulting from equally gross lesions may be easily demonstrable to the observer, and it is possible for an animal to show quite obvious signs of local analgesia after the removal of a particular part of the cortex. But in the finer shades of sensibility where the lesion is small and the effect slight, an animal has no power wherewith to express its appreciation or otherwise of a light touch, or to describe any distinction it may draw between different varieties of sensory impressions.

In human beings also, there are many fallacies to be avoided in the testing of sensation, and many difficulties arise which it may appear at first sight almost impossible to surmount. As a sine qua non consciousness must be pres-

-ent, and a patient profoundly comatose after a cerebral apoplexy is of no value as a demonstration of anaesthesia. The hemiplegic patient must have retained or recovered at least a moderate degree of intelligence sufficient to enable him to give some verbal expression to any mental response he may make to external stimuli. And even under such favourable conditions the human mental attitude is so much a variable quantity that it entirely precludes the possibility of fixing any definite standard whereby to judge of affections of sensation. Extreme sensitiveness to external impressions, unlimited capacity for all varieties of emotion, neurotic exaggeration, — these constitute a mental attitude capable of transforming the slightest tactile impression into the touch of a red hot iron. On the other hand, dulness or apathy, cerebral exhaustion, capacity for endurance — induced either by want of perceptive power or by a more becoming stoicism, — may render a patient apparently irresponsible to even a moderately severe stimulus. Again, a certain amount of power of concentration in the patient is necessary or at least acceptable to the observer, and still more so is his capability of accurate interpretation of various impressions. Then the condition of the periphery itself is of importance as regards its temperature, its hardness or softness, and its general sensitiveness.

Thus it happens that in the testing of sensation one must necessarily depend so much on the mental acuity of the patient that the difficulty of making trustworthy observations is materially increased.

But in spite of these several drawbacks, it is, I think, possible by minutely examining the same patient under varying conditions, from day to day, to gain a fairly accurate idea of the acuity of his general sensibility, and the extent to which it has been affected by any particular

lesion. This conviction led me primarily to approach the subject of sensation from a clinical point of view, and it is the results of these observations I now desire to advance and record.

Hemiplegias as they occur clinically are usually the result of a lesion either in the Internal capsule or in the so called "motor cortex." I propose to consider first the sensory phenomena following a lesion of the posterior division of the internal capsule, partly because this is the more common site for lesions productive of hemiplegia, and partly because it involves to a greater or less extent the nerves of special sense.

THE OLFACTORY NERVE.

Tracing this nerve back from the mucous membrane of the nose the fibres are seen to enter the Olfactory Bulb, which lies on the cribriform plate of the ethmoid bone. Thence the Olfactory Tract runs back in the Olfactory Sulcus on the under aspect of the internal frontal convolution. At the posterior extremity of the tract are seen the three roots, the outer running to the anterior end of the Hippocampal Gyrus of the same side, and the inner turning in and apparently joining the Callosal Convolution, while the middle or grey root lies between the others and is traversed by both.

The view was once held that the Olfactory Lobe was as much a separate and distinct division of the cerebrum as is the Parietal or Occipital lobe, and that it thus coincided with the cortical termination of the olfactory nerves. Now, however, the Olfactory Lobe is considered analagous to the other basal ganglia, merely fulfilling the function of a lower or secondary centre whence the fibres pass to their cor-

-tical destination. Some doubt still exists as to the exact locality of this destination, and still more as to the decussation or otherwise of the olfactory fibres on their way thither.

Broca considers the olfactory centre to be situated in the Hippocampal and posterior part of the Orbital lobule while other observers believe all the Limbic Lobe or the posterior part of the Hippocampal Lobe to be concerned in the perception of smell.⁽¹⁾ Ferrier,⁽²⁾ however, places the centre in the tip of the Uncinate Gyrus, and has proved by experiment that stimulation of this region causes muscular movements of the nostril, while removal results in anosmia of the same side in animals. And Collins (Op. Cit. p 50) mentions a case of progressive anosmia on the right side associated with erosion of the right uncinate convolution, and calls attention to two cases recorded by Hamilton and Jackson in which epileptiform seizures with an olfactory aura had resulted, one from softening and the other from tumour of the Temporal Lobe and adjacent uncinate gyrus. Turner⁽³⁾ also mentions several cases of lesions of the Uncinate Gyrus but he states that in the recorded cases of disease involving the cortical centres for smell the most prominent symptom has been a perversion rather than a diminution in the sense.

It is generally admitted that the Uncinate Gyrus represents the destiny of the olfactory nerves, but as regards the decussation of the fibres, clinical observations differ somewhat from anatomical and experimental evidence.

It is anatomically proved that the fibres of the outer root run to the cortex of the same side; but the inner fibres have, so far, not been definitely traced to their termination. Ferrier⁽⁴⁾ has found by experiment that destruction of one Uncinate Gyrus causes anosmia on the same side

in the monkey, and has stated his belief "that there is no anatomical basis of cross connection between the olfactory bulbs and their cerebral centres." But elsewhere⁽⁵⁾ he says that though the outer root of the olfactory nerve can be traced to the subicular region of the same side, yet the inner may pass to the other hemisphere, though so far it has not been traced further than the corpus striatum.

Hamilton,⁽⁶⁾ however, describes fibres running from one cortex across the corpus callosum to the opposite external capsule, and thence to the base of the brain in the region of the anterior perforated space. Here they become united with the olfactory tract forming one of its roots, so placing the olfactory nerve in connection with the opposite cortex. Bastian⁽⁷⁾ believes the anterior commissure to have important functions of some kind in connection with the sense of smell; and Testut⁽⁸⁾ describes "crossing fibres" passing from one olfactory bulb to the Temporal lobe of the opposite side. Turner^(op. cit.) thus epitomises what is known of the intracerebral course of the Olfactory fibres:-

1. A certain number cross, forming the "Pars Olfactoria" of the anterior commissure.
2. Many pass to the Uncinate Gyrus of the same side.
3. Some fibres are to be found in the neighbourhood of the internal capsule and optic thalamus.
4. A part of the anterior commissure forms a connecting strand between the Hippocampal lobes of the opposite sides.

So far as personal observation goes I believe clinical evidence to be quite in favour of at least a partial decussation of the olfactory fibres in the cerebrum, as lesions affecting the sensory division of the internal capsule cause

either complete or incomplete anosmia on the side opposite the lesion. Out of twenty-two cases of Hemiplegia, obviously the result of a capsular lesion, which I have had an opportunity of examining, eight were found to be suffering from complete anosmia while eleven showed diminished perception of smell on the same side as the hemiplegia. The remaining cases - with one exception - were patients in whom there was almost complete recovery, scarcely a trace of the hemiplegia remaining except a slight weakness in the arm. In no case was there any loss or obvious diminution of smell on the same side as the cerebral lesion; and in the numerous recorded instances of anosmia occurring in connection with hemiplegia this seems also to have been the condition observed.

Ferrier⁽⁹⁾ has attempted to explain the occurrence of the anosmia on the hemiplegic side by suggesting that it may be due to the loss of common sensibility of the Fifth nerve, in addition to the probable crossing of a few of the olfactory fibres through the anterior commissure. Bastian, however, (Op. Cit. p 380) states that an affection of the intracerebral roots of the fifth nerve probably does not affect the sense of smell at all. He suggests that if the olfactory fibres do decussate in the anterior commissure on their way to the tip of the Temporal lobe they must run near the outer border of the crusta, and thus would be close to the other afferent fibres in the posterior division of the internal capsule, and readily affected by any lesion occurring therein.

The question thus appears to be whether the number of olfactory fibres crossing in the anterior commissure is sufficiently great to explain the opposite anosmia in capsular lesions. Ferrier's explanation of the anosmia appears to me

quite inadequate inasmuch as the complete loss of smell so frequently met with clinically is often out of all proportion to the severity of the involvement of the Trigeminal.

In case No. 3 (see appendix), the nerves of general sensation are practically unaffected, while the olfactory sense is entirely abolished on the same side, and this without any discoverable defect in the external nasal apparatus. In various other cases (2, 4, 5, 7, 8) complete anosmia is associated with a general diminution, but no loss of common sensibility; and in one case only (Case 1.) is the fifth nerve affected to so great an extent as to give any degree of feasibility to such an explanation.

From a consideration of capsular lesions, therefore, it is probable that the cortical centre for smell is situated opposite to the peripheral distribution of the olfactory nerves. There appears to be forthcoming little or no satisfactory and convincing proof that an isolated lesion of one uncinate gyrus, apart from any involvement of the olfactory tract or bulb, will produce an anosmia of the same side rather than a perversion of the sense of smell. Anosmia pure and simple is evidently most commonly and most completely the result of a lesion in the sensory division of the internal capsule, thus indicating the existence therein of fibres with a definitely olfactory function. This conclusion is borne out by the fact that in hysterical hemianaesthesia, which is the result of a functional rather than of an organic change in the "sensory crossway," anosmia also is included in the phenomena observed.

It is sufficiently obvious that the olfactory, as well as the other afferent fibres in the posterior division of the internal capsule pass to the cortex opposite to their peripheral distribution. Thus it seems fair to conclude

that though there may, and probably does, exist a direct connection between the olfactory cortex and the periphery, nevertheless the crossed fibres play the most important part in connection with the conveyance of olfactory sensation.

T H E O P T I C N E R V E .

Tracing this nerve back from its ending in the retina the fibres are seen to run to the Optic Chiasma where they decussate in such a way that those from the nasal side of the right eye join those from the temporal side of the left eye to form the Left Optic Tract. The Tract, which is thus composed of two sets of fibres - one which has decussated and one which has not - runs backwards under the Temporo-sphenoidal lobe, and its fibres appear to terminate in the Anterior Corpus. Quadrigeminum, the External Geniculate Body, and the Pulvinar of the same side. Thence fresh fibres rise, some of which run through the posterior limb of the internal capsule, and spread out in the optic radiations towards the occipital lobe.

It was once supposed that some fibres ran straight from the tract through the posterior third of the capsule to the cortex, thus forming a "direct cerebral tract," but "there is increasing evidence that this view is an erroneous one. We may probably with great confidence assert that the connection of the cerebral hemispheres with the optic tract is not a direct but an indirect one."⁽⁸⁾

Though there is no longer any doubt concerning the localization of visual sensation in the occipito-angular region, it is still a disputed point as to which part of this region represents the highest visual centre. Munk showed first that complete unilateral destruction of the visual sphere caused, not blindness, but Lateral Homonymous Hemianopia; and as he obtained this result after section in

the line of the parieto-occipital fissure, he regarded the occipital lobe as the true visual field, and the Angular Gyrus as the "sensory sphere of the eye." Ferrier⁽¹¹⁾ however, denies this hypothesis, believing the Angular Gyrus to be the highest centre for the perception of sight, which conclusion he supports by numerous experiments. Here, again, however, experimental research is at variance with clinical and pathological observations, from which evidence it appears almost certain that the occipital lobe is the true visual centre. Among the many recorded cases illustrative of this is a particularly suggestive one mentioned by Dr. Delepine⁽¹²⁾, in which a lesion of the left cuneus was associated with right homonymous hemianopia. Swanzy⁽¹³⁾ and Percival⁽¹⁴⁾ also consider the highest visual centre to be situated in the cortex of the occipital lobe rather than in the Angular Gyrus, which in all probability is principally concerned with word memories: and Gowers⁽¹⁵⁾ believes that "Human Pathology has fully confirmed the correctness of Munk's conclusions."

It would further appear that each cortical centre must be related to the inner half of one eye and the outer half of the other, and also that certain parts of each cuneus must correspond to certain parts of each half visual field. Dr. Hun has recorded a case, quoted by Percival (Op. cit. p5) and by Swanzy (Op. cit. p453) from which he concludes that the fibres from the right upper quadrant of each retina terminate in the lower half of the right cuneus.

Again, Wilbrand has separated the visual centre into three, - one for light, one for form, and one for colour, - which he places in the occipital lobe either side by side or in strata. Verrey has recorded a case of colour blindness occurring alone which led him to conclude that the centre for colour sense lies "in the most inferior part of the

occipital lobe, and probably the posterior part of the lingual lobe;" while he places the light centre in the posterior part of the occipital lobe and the centre for form sense between the others.⁽¹⁵⁾ MacKay⁽¹⁶⁾ supports this view by recording a case of complete acquired colour blindness, in which the lesion consisted of atrophy of the posterior part of the temporo-occipital convolution of both sides.

The Left Angular Gyrus is generally believed to be the centre for word vision and its connection with speech; and Percival (Op. cit) quotes several cases of word blindness which demonstrate this. He further says there is reason to believe that both right and left visual fields are connected with this centre, but that the maculae luteae are evidently projected on to both cerebral hemispheres.

From a consideration of the relation of those various cortical centres to the retina the main symptom to be expected in a lesion affecting the cuneus, optic radiations, Pulvinar or Optic Tract, is Lateral Homonymous Hemianopia. Should the lesion be situated anterior to the Corpora Quadrigemina there may also be observed "Wernicke's Pupillary Inaction," which consists of non-reaction of the pupil to a light thrown on the hemiopic half of the retina. This phenomenon is explained by the fact that from the Anterior Corpora Quadrigemina fibres pass to the nucleus of the third nerve, and so give rise to the pupil reflex. Thus, any lesion occurring anterior to this region will necessarily affect the integrity of the reflex. Foster (Op. cit) believes this to be the main function of the corpora quadrigemina, which are thus not directly connected with vision, and one case which I have had an opportunity of observing illustrates this point to some extent.

The patient, a girl, was admitted with a history of headache and vomiting of five years' duration. Her sight

was not affected at all until three months before admission when optic neuritis followed by atrophy developed, and she became totally blind. Post Mortem, a fibrous tumour was found accurately confined to the four Corpora Quadrigemina, pressing on the veins of Galen and causing hydrocephalus. In this case the quadrigeminal tumour by itself appears to have had no effect upon vision until such time as the developing hydrocephalus became associated with optic neuritis and atrophy.

In lesions affecting the posterior third of the internal capsule and producing hemiplegia and hemianaesthesia, instead of Hemianopia as might be expected, I have occasionally found crossed amblyopia, consisting of contracted visual field, diminished visual acuity, and altered perception of colour. (see cases 1, 4, 6). Von Graefe formerly held the view that absolutely unilateral lesions of the cerebrum did not cause crossed amblyopia but lateral homonymous hemianopia. Charcot,⁽¹⁷⁾ however, insisted that "Lesions of the cerebral hemisphere producing Hemianaesthesia likewise determine crossed amblyopia," and Bastian (Op. cit. p 134) remarks that unilateral amblyopia has a distinct localizing value when it is associated with Hemianaesthesia, the lesion then being in the posterior third of the internal capsule.

Percival (Op. cit. p8) while admitting that crossed amblyopia may occur in connection with Hemianaesthesia attempts to explain the apparent anomaly in much the same way as Ferrier explains crossed anosmia, viz: by attributing most of the phenomena to the implication of the Fifth Nerve. He believes it possible, as Gowers suggests, that there is a communication between the centres of the Optic and Trigeminal nerves, so that any excitation of the centre of the fifth may alter the sensibility of that for vision. As an example of this he cites the amblyopia arising from dental

irritation of the Trigeminal.

But, so far as I can learn, no communication has ever been anatomically traced between the centres for the Optic and Trigeminal nerves. And here again, the affection of the fifth nerve in the cases I have examined is so slight as entirely to preclude any idea of its secondary influence on the optic nerve. And, in the example quoted - of amblyopia following dental irritation of the Trigeminal - the effect on the optic nerve is presumably an irritative one while that in Hemianaesthesia is paralytic, and it is difficult to imagine two such opposite lesions resulting in the same peripheral manifestation.

Swanzy (Op. cit.) says that "Lesions of the posterior third of the internal capsule are still believed to cause Hemianopia and Hemianaesthesia of the opposite side of the body; but analysis of clinical cases affords no support to this view, for there are no recorded cases which furnish any definite evidence in this respect. Yet anatomically, fibres have been traced from the occipital cortex through the optic radiations and internal capsule to the basal ganglia and from thence to the optic tracts. The fibres passing through the internal capsule from the External Geniculate body may perhaps be simply reflex fibres."

But it is quite an accepted fact that crossed Amblyopia does occasionally occur as a result of a lesion in the posterior part of the internal capsule; if that be so, then those fibres mentioned by Swanzy cannot be merely reflex in their function. It is much more probable that, as Foster (Op. cit. p 1176) suggests, the External Geniculate body is the most important of all the basal ganglia connected with vision, "and indeed there is evidence that this is specially connected with the maculae luteae". Turner (Op. cit. p 757) also believes the External Geniculate Body and

the radiation from it to the Calcarine fissure should alone be considered visual in function.

Gowers ⁽¹⁵⁾ suggests that the occurrence of crossed amblyopia in hysterical hemianaesthesia indicates the existence of a functional centre, capable of being inhibited, in which is represented the whole field of one eye and not the half fields of both eyes. But it is difficult to imagine a functional apart from an organic centre, and probably the one may serve as a guide to the other. Granting the site of the above lesion to be central, even though its character be functional, that is physiological, it is reasonable to argue that exactly similar peripheral evidences may be produced by pathological lesions affecting the same locality.

If, as Foster says, the External Geniculate Bodies are connected with the maculae luteae, and if, as Swanzy owns, fibres pass from these bodies to the internal capsule, these fibres are probably connected with the maculae. Now, it is generally believed that each macula is projected on to the opposite cuneus; and if this be the case, it is quite sufficient to explain the occasional occurrence of crossed amblyopia rather than Hemianopia in lesions of the posterior division of the internal capsule.

THE TRIGEMINAL and GLOSSOPHARYNGEAL NERVES.

In discussing the subject of taste in connection with cerebral lesions I propose to consider these nerves together rather than separately, opinions being still divided as to which nerve is responsible for the conduction of taste impressions to their cortical destination. The common sensory functions of the Trigeminal will be considered later along with affections of general sensibility resulting from capsular or cortical lesions.

The fibres of the 5th nerve reach the Gasserian Ganglion by the Ophthalmic, and the Superior and Inferior Maxillary nerves. Issuing from this, the trunk, consisting of a large, ganglionic or sensory part, and a small aganglionic or motor part, makes its way to the Pons, some of the transverse fibres of which separate the two roots. Here one division of the sensory root runs to a nucleus situated a little below the floor of the fourth ventricle, (the Upper Sensory Nucleus); while the other is connected with some cells in the posterior horn of grey matter near the Tubercle of Rolando in the Medulla (the ascending root.) This root has been supposed to rise from the Tubercle, but Quain⁽¹⁹⁾ does not believe this to be the case, as the fibres really have their origin in the Gasserian Ganglion (His.) From the upper sensory nucleus, and also from the cells of the gelatinous substance fibres run across the median raphe partly to end in the formatio reticularis, and partly to join the opposite mesial fillet. Turner,⁽²⁰⁾ however, believes that the sensory fibres of the cranial nerves lie mesial to the fillet in the pons and cerebral peduncle, as, he says, the fillet may be entirely schlerosed and the structures from which it arises destroyed without any impairment of sensation.

It is evident therefore that the course of the Trigeminal fibres after they leave the Medulla and Pons is somewhat obscure, but it is equally obvious, first that they pass to their cortical destination through the posterior limb of the internal capsule, and secondly that they decussate in their course.

The Glossopharyngeal Nerve is also supposed to be partly motor and partly sensory, and can be traced back from the pharynx etc. to the medulla where it lies close below the Facial Nerve in the groove between the Olivary and Res-

-tiform bodies. Thence the fibres run to a nucleus, situated beneath the inferior fovea in the floor of the fourth ventricle, but further than this they have not been anatomically traced. The Medulla and Pons thus contain the "basal ganglia" for both the Fifth and the Ninth nerves; and there is no doubt that the Glossopharyngeal also has a cortical destination, and that its fibres, after decussating, run in the posterior limb of the internal capsule.

Which particular part of the cerebrum is concerned with the perception of taste is not as yet quite certain. Ferrier⁽²¹⁾ states that he has found by experiment that if the lower extremity of the Temporal lobe is irritated the animal shows subjective sensations of smell and taste; while bilateral lesions of the anterior extremities of this lobe cause complete anosmia and ageusia. He quotes a case recorded by Dr. Anderson in which a tumour affecting the left Temporal lobe was found associated with loss of taste.

This seems to be the most generally accepted view, and is more or less confirmed not only by clinico - pathological observations on the Temporal lobe, but also more or less by a process of exclusion; for lesions occurring in most other cortical areas of the brain - without affecting the posterior limb, of the internal capsule - seldom if ever cause any diminution in the sense of taste.

There are various theories as to the route taken by the taste fibres on their way to the brain, and most authorities are divided in opinion as to whether the Fifth or the Ninth nerve is specially concerned with the conveyance of this sense. Gowers⁽²²⁾ believes the Trigeminal to be the true nerve of taste, and in support of this view he quotes several cases of isolated palsy of the Fifth nerve due to intracranial disease in which taste was lost not only anteriorly but also at the back of the tongue, and on the soft

palate and palatine arches. He thinks the taste fibres diverge from those of common sensibility in the Pons, and thus explains those cases of anaesthesia of the Fifth nerve without any affection of taste; and he quotes a case of a lesion which he says was certainly within the pons, which paralysed the motor part of the Fifth nerve, abolished taste all over that side of the tongue, but left unimpaired the other functions of the Fifth.

Gowers also believes the taste fibres in the posterior part of the tongue to be really part of the Trigeminal nerve, though distributed with the Glossopharyngeal, to which they must pass from the otic ganglion by means of the small superficial Petrosal nerve and Tympanic plexus. This he considers to be the explanation of the fact that taste may be lost on the back as well as on the front of the tongue in consequence of caries of the walls of the Tympanum. As Gowers confesses, this is a very circuitous route to be followed by a nerve of special sense; and the phenomena might be just as satisfactorily explained, if not more so, by assuming the Glossopharyngeal to be the special nerve of taste. In a lesion occurring in any particular locality, the nerve most closely connected with the affected part rather than one only in indirect communication therewith is admittedly that which demonstrates the peripheral manifestation of the lesion. Now it is obvious that the Ninth nerve is much more intimately associated with the Tympanic plexus than is the Fifth, and thus any affection of the walls of this region should be evidenced by some alteration in the function of the Glossopharyngeal. And, as this disturbance of function takes the form of ageusia the natural inference is that the fibres of this nerve are in some way concerned with the conveyance of impressions of taste.

Turner (Op. cit. p 972) says that though implication of the Seventh nerve in disease of the middle ear is associa-

-ted with ageusia, yet disease of the root of the Facial within the skull is not associated with any loss of taste. Bastian,⁽²³⁾ however, states that he has met with a well marked case of disease in the intracranial part of the Seventh nerve, in which taste was lost on the anterior part of the tongue on the same side. He believes the gustatory fibres run to the brain with the trunk of the Facial nerve as the Nerve of Wrisberg, which terminates in a part of the Glossopharyngeal nucleus; and he adds that the anatomical investigations of Duval support this view.

Turner (Op. cit. p 786) mentions a case recorded by Lehmann in which a basal fracture caused paralysis of the seventh, eighth, twelfth and tenth nerves, with loss of taste on the corresponding side of the tongue. And in a case recorded by Pope an aneurysm of the Left Vertebral artery pressed upon the roots of the ninth nerve, causing ageusia on one side of the tongue, though not completely in the anterior part. Turner therefore concludes that "the evidence regarding the ultimate destination of the taste fibres in the ninth nerve is conflicting, for the test case - complete isolated unilateral Glossopharyngeal root palsy - is not yet recorded, though Pope's case comes near it."

With regard to the occurrence of peripheral Trigeminal palsy unassociated with ageusia, Turner believes either that the implication of the nerve is not complete, or that the lesion is in the medulla, the anaesthesia being due to the implication of the ascending root. He quotes a case recorded by Ferguson in which there had existed loss of taste on the anterior two-thirds of the tongue while the posterior part was normal; and the post mortem revealed a small exostosis pressing on and dividing the left Vidian nerve.

But here, the same argument might be employed and with more reason, as was used in explaining crossed amblyopia

and anosmia, namely that the ageusia was the result of an implication of the nerves of common sensibility rather than those connected with taste, and was due partly to the dryness of the mucous membrane and partly to the anaesthesia induced by paralysis of the sensory division of the Trigeminal. In support of this view it must be remembered that Meckel's Ganglion has been excised on both sides, without any affection of taste; and even when in addition part of the Infraorbital nerve has been removed, taste remained quite unaffected.⁽²⁴⁾ Again, Dr. Tiffany⁽²⁵⁾ records four cases of removal of the Gasserian Ganglion in three of which persistent anaesthesia followed, while taste remained normal, and Lloyd⁽²⁶⁾ mentions a case in which a palsy of the Fifth nerve was followed by extreme anaesthesia of the face, and trophic disturbance, while taste was only blunted anteriorly - not abolished.

The evidence, therefore of the Fifth being the special nerve of taste is somewhat conflicting both as regards pathological and experimental observations. On account of the difficulty of satisfactorily testing the sense of taste, especially on the posterior part of the tongue, clinical observations lose something of their value. But in eight cases I have been able to demonstrate total ageusia on one side of the tongue, and in eleven a marked diminution. These were all cases of Hemiplegia in which there was also a varying amount of Hemianaesthesia, possibly sufficient in several instances to explain some slight affection of the sense of taste. But in no case was there a total anaesthesia of the head and face, such as would indicate an involvement of the Trigeminal fibres to so great an extent as to produce complete ageusia in addition to the other sensory manifestations. If we suppose the Trigeminal to be the special nerve of taste such ageusia may be explained by assuming the separation of the taste fibres from those of general sensation on their way to the cortex.

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But it appears improbable that a nerve with such definite functions as the Fifth should also perform the duty of a nerve of special sense. Taste is as much a special sense as is Smell, or Sight, or Hearing, and it is difficult to understand why each of those senses should be provided with a separate nerve to convey its impressions to the cerebrum, while taste should be carried promiscuously by various circuitous routes by a nerve which has already a definite function of its own. And as it is just as improbable that the same sense should be carried by two separate nerves it seems more reasonable to suppose the Glossopharyngeal to be the special nerve connected with taste. Thus taste would be analagous to the other senses, and the Glossopharyngeal to the other sensory cranial nerves. Supposing this to be the case, we must conclude that the taste fibres from the back of the tongue go directly to the cerebrum by the Ninth, while those anteriorly go by the Chorda Tympani in the Lingual to the Facial, whence they ascend by the Pars Intermedia to the medulla and nucleus of the Glossopharyngeal.

THE AUDITORY NERVE.

The fibres of the auditory nerve run backwards from the internal ear towards the medulla, where they separate into two roots. The upper runs through the lowest part of the pons to the Outer Auditory Nucleus beneath the floor of the Fourth ventricle, and from this some fibres are said to pass outwards to the cerebellum.

One division of the lower root passes backwards on the inner side of the Restiform body to the Inner Auditory Nucleus which lies in the upper part of the medulla, while other fibres run round the outer side of the Restiform body

to the floor of the Fourth Ventricle, where they form the Auditory Striae. From the Auditory nuclei fibres run across the pons - forming part of the Trapezium - to the Lateral Fillet, and thence by the posterior brachium to the Internal Geniculate Bodies and the Posterior Corpora Quadrigemina.

Turner^(6p. cit) believes that the posterior quadrigeminal bodies have no direct connection with the central auditory tract but are merely accessory to it; while the Internal Geniculate bodies stand in the same relation to the auditory nerve as do the External Geniculate to the visual fibres. Collins⁽²⁷⁾ on the other hand, says Weinland has shown disturbance of hearing to be a frequent result of tumours of the Corpora Quadrigemina. This result, Collins believes to be due to the close proximity of the lateral fillet, which may readily be involved in any lesion affecting these bodies; and Turner makes the same suggestion regarding Weinland's cases. Mynert states that "no extensive immediate connection of the auditory nerves with the cerebral lobes exists," and that the connection is brought about indirectly through the cerebellum; and Bastian says "If Mynert's view be correct, it would seem that the auditory fibres could only reach the posterior third of the internal capsule by way of the upper cerebellar peduncle." Thus lesions involving the upper part of the pons should not affect hearing, and this Bastian has observed in several cases.

By whichever route they travel, however, the auditory fibres ultimately reach the centre for hearing, which is supposed to be situated in the Superior Temporal Lobe. Ferrier (Op. cit.) quotes a case in which complete deafness followed bilateral softening of the First Temporal Gyrus; and Mills⁽²⁸⁾ also records a case in which marked atrophy of both Temporal lobes was found in a man who had been deaf for 30 years.

Hughes Bennett⁽²⁹⁾ mentions several instances of Subjective Auditory Sensations of Central origin followed by temporary loss of hearing on the opposite side; while Collins (Op. cit. p 55) suggests that destruction in the Superior Temporal lobe does not apparently cause complete crossed deafness, but a marked diminution of hearing on the opposite and a slight diminution on the same side as the lesion.

This statement appears to be borne out by a study of capsular lesions in which a large proportion of cases give evidence of impairment of auditory perception on both sides, though as a rule the defect is most perceptible on the side opposite the lesion. Out of a series of twenty-two cases of capsular hemiplegia I have found diminished sense of hearing in both ears in twelve. Of these, five showed complete deafness on the hemiplegic side, and the other seven a much more marked impairment of the sense than was evident on the sound side. The hearing in six of the remaining cases was apparently diminished on the hemiplegic side alone, while in four instances only did it appear entirely unaffected in either ear. This would appear to suggest a bilateral representation of auditory perception to some extent, the principal cortical centre being situated on the side opposite the peripheral distribution of the auditory nerve. It is clear that the auditory fibres travel in the posterior limb of the internal capsule, and the inference therefore is, either that their decussation is not complete, or that some strands of fibres cross twice on their way to the cortex.

The most common result of a lesion in the First Left Temporal convolution appears to be Word deafness. Mills (Op. cit.) records a case to illustrate this, while out of twenty five cases of word deafness analysed for Ferrier(*op.cit*) in 1890, ten were found to be due to a lesion of the Temporal lobe alone, while in seven the First Temporal Gyrus was speci-

-ally affected. In connection with this subject I may mention the following case which came under my care. J. J. Male, aet. 45, was admitted with a history of a kick on the right side of the head a week previously, up to which time his wife stated he had always been quite healthy and intelligent. On examination a large bruise was seen behind the right ear, and some clotted blood in both ears. The patient for a week showed no sign of intelligence, but lay almost comatose except for occasional fits of swearing. He then recovered slightly and was found to be suffering from quite distinct word deafness. He did not understand anything that was said to him until it was written down, when he replied rationally though with some hesitation. He remained in this condition about three weeks, when he gradually improved, though his manner remained somewhat strange. At no time was there any difference of hearing on the two sides, the tick of a watch being heard a foot distant from either ear.

The explanation of this case appears to be that the Left Superior Temporal Gyrus was injured by contre-coup, and its substance either lacerated by forcible contact with the skull, or pressed upon by the haemorrhage which would follow such an injury. That the lesion was caused by haemorrhage appears more probable considering the almost complete recovery made by the patient in a comparatively short space of time. The fact that perception of ordinary sound was not at any time affected lends probability to the view that there exists a centre for general acoustic perception apart from that for word hearing in the Superior Temporal Gyrus.

NERVES conveying IMPRESSIONS of GENERAL SENSIBILITY.

The consideration of the general sensory pathways and their cortical destinations will include such nerves as convey impressions of touch, pain, temperature and muscular sense to the cerebrum from the periphery.

Taking the sensory tracts as a whole, we find that they run in the spinal cord to the medulla, partly by a direct route in the posterior columns, and partly by a more general path in the antero-lateral part of the cord. Though anatomically each sensory path does not represent a particular variety of sensation, yet clinically it would appear that each form of cutaneous sensory impression is conveyed brainwards by its own special strand of fibres. It was once thought that tactile sensibility travelled by the posterior columns, but a lesion here affects all kinds of sensation equally, and in fact it has been shown by experiment on animals "that sensation as a whole is more interfered with by division of the lateral and especially of the antero-lateral columns of the cord than the division of any other part. Besides, both experiments on animals and clinical observations show that sensory impressions cross to the other side of the cord, and this corresponds with the anatomical feature of the general path, while the special tract by the posterior columns does not cross till the bulbar nuclei are passed." (Foster Op. Cit.)

On reaching the medulla the sensory fibres appear to end in the Nuclei Graciles and Cuneati from which fresh fibres rise, and after decussating run to the opposite median fillet. The fillet also receives those fibres which have already crossed in the cord, and being thus entirely composed of decussated fibres, it passes onwards by the Pons and crus Cerebri to the Optic Thalamus, where most of the

nerves end, though some are continued directly onwards to the cortex. Most observers believe the path for the transmission of cutaneous sensation to be situated in the fillet either alone or with the neighbouring part of the *Formatio Reticularis*; but Ferrier^(op.cit) found by experiment that after destruction of the nuclei *clavae* and *cuneati* the fillet degenerated, while sensation remained unaffected. He therefore believes the short fibre system of the *Tegmentum* or *Formatio Reticularis* to be the true path for the transmission of cutaneous sensibility.

But though the fillet degenerated after such an experiment without the occurrence of anaesthesia, that observation alone is scarcely sufficient to prove that sensation therefore does not travel by the fillet. It is well known that sometimes the removal of the *Pyramidal tract* in a dog is followed by no loss of voluntary motion, probably because impulses travelling normally by that path find their way to the periphery after its destruction by some other channel. And if this result may follow a motor lesion a similar sequence is possible in a sensory defect, sensation undoubtedly tending to recover more quickly than motion after a lesion. Thus, after destruction of the fillet, the sensory impulses normally travelling by this tract may find their way to the cortex by a more general path in the *Formatio Reticularis*. It appears at least probable that sensation travels principally by the fillet, which would thus constitute the direct route, corresponding with the posterior columns of the cord, while the short fibre system of the *tegmentum* may form a more general pathway, like that in the antero-lateral part of the cord, capable however of performing the duties of the whole in the case of a lesion affecting the fillet.

But however the sensory fibres travel they eventually reach the posterior limb of the internal capsule where

they are aggregated into a compact bundle, and so are readily implicated in any lesion affecting this part. The lesions most liable to involve this site are either haemorrhage by extension from the Lenticulo-striate branch, or embolism or haemorrhage of the Lenticulo-Optic branch, of the Middle Cerebral Artery. These lesions, if completely destroying the posterior limb of the internal capsule give rise to a typical cerebral hemianaesthesia on the opposite side, and four cases of this kind were described by Turck as early as 1859.⁽³¹⁾ This consists of insensibility to touch, pain and temperature with loss of muscular sense, and affection of the special senses, including as we have seen, anosmia, ageusia, and deafness on one side, with either lateral hemianopia or amblyopia.

But clinically it is comparatively rare for a patient to present an entirely typical hemianaesthesia after such a lesion. In some cases, one or other of the special senses may be partially affected in addition to complete loss of general sensibility, or the various forms of general sensation may not be equally involved. In a series which I have observed of twenty two cases of diminished sensibility, varying in degree, but always readily demonstrated, entire general and special sensation was affected in sixteen cases only. Of the remainder three were cases in which, with practically no impairment of general sensation the special senses were obviously diminished in acuity, smell and taste being abolished and hearing and vision diminished in one, while all senses were about equally affected in the others. The remaining three cases showed diminished general sensation without any impairment of the special senses.

The variable degree to which general sensibility was involved in the above series was approximately as follows:-

The Sense of Temperature presented some variety in

its impairment that for heat being evidently much more readily affected than that for cold in lesions implicating the internal capsule. In nineteen of my patients in whom this sense was defective, ten were cases in which the cold tube was almost invariably recognised while the hot test was not appreciated at all, even when water warm enough to be almost painful was used. Generally speaking, it appeared that if the hot test were correctly appreciated by the patient, the cold tube presented no difficulty, and thus the sense of temperature as a whole was but little affected, though both kinds of thermal sense might be less acutely felt than on the sound side.

Tactile Sensation appeared to be impaired to the greatest extent, being more or less implicated in the entire series of cases, though sometimes to a very slight degree. In such instances only the lightest touches were unrecognized, and the sense of localization was perfect, though transmission of impressions was delayed. In the more marked cases where an ordinary touch was unappreciated the sense of localization was not so accurate, and such patients generally gave evidence also of some impairment of their Muscular sense. This appeared to be implicated in direct ratio to the severity of the tactile anaesthesia, being entirely unaffected in the milder cases and markedly involved in the more severe types. Painful Sensation appeared more or less impaired in all those patients who gave evidence of tactile anaesthesia. The analgesia, however, was less obvious than the affection of touch, being practically nil in the less severe cases, and but seldom complete even in definitely anaesthetic areas. In no instance did I find any impairment of the sense of pain apart from that of touch, though there are recorded various cases illustrating such a condition. ⁽³²⁾

Paget ⁽³³⁾ believes the senses of pain and temperature

to be the most readily and the most permanently affected in any lesion of the brain or cord, and he suggests that the paths for these senses lie close together entirely apart from the track conveying impressions of touch. This, however, I have not observed, but rather that tactile and muscular senses are most intimately associated, that painful sensation to a less degree is connected with touch, and that temperature apparently is distinct from either and may be affected independently of both. It is more than probable that each form of general sensation travels by a pathway of its own in the sensory crossway; but clinically it appears that the tracts conveying impressions of touch and of pain lie near one another; and thus a capsular lesion large enough to result in analgesia almost certainly produces in addition a marked degree of tactile anaesthesia.

Again, the manner in which the thermal sense is affected in capsular lesions suggests the existence of a separate set of fibres for the conveyance of each variety of this sensation. Supposing impressions of heat and cold to travel to the cortex by the same bundle of fibres, and this to be entirely destroyed by a lesion occurring in the internal capsule, the patient should then be quite unable to appreciate either heat or cold applied to the periphery of the body. Or were the destructive lesion only partial, the result would be impaired sensibility to both heat and cold. But there is no variety of lesion capable of producing diminished sensitiveness to heat impressions while allowing impulses of cold to pass onwards to the cortex by the fibres which are presumably implicated in the loss of thermal sense.

In most cases of Hemiplegia, the lesion in the internal capsule has consisted of a haemorrhage from the Lenticulo-striate artery, which though it supplies only the anterior two thirds of the posterior limb, still may involve the pos-

-terior third by the extravasation of blood backwards. This fact may serve as a diagnostic point between haemorrhage and embolism of the Lenticulo-striate artery, the latter lesion causing no impairment of sensation. Two cases which I have seen illustrate this in different ways. One was that of a woman, aged 30, suffering from mitral stenosis, who suddenly developed a complete left hemiplegia, without any impairment of sensation, the lesion obviously being an embolism of the Lenticulo-striate artery. The second case illustrates also the diagnostic importance of a correct estimate of the impairment of sensation. The subject was a man with a large aneurysm of the aorta, in whom there suddenly occurred a right hemiplegia and motor aphasia. After several days he gradually developed sensory aphasia (and hemianaesthesia?) The question was then raised as to what constituted the lesion. In view of the existence of the aneurysm an embolism of the Lenticulo-striate artery appeared at first the most probable explanation, but if such were the case, whence came the sensory symptoms? After various more or less ingenious suggestions had been offered, the death of the patient helped to solve the problem. Post mortem, an embolus was indeed found in the Lenticulo-striate artery, and extending backwards from this to the circle of Willis, and involving the Lenticulo-Optic artery in passing, was a distinct thrombus, this accounting for the gradual onset of the sensory symptoms after the sudden occurrence of the motor phenomena.

It seems to be a very generally accepted theory that sensation is only temporarily affected in lesions of the internal capsule: and Osler⁽³⁴⁾ says, in Hemiplegia "Disturbance of the special senses is not common," and again,⁽³⁵⁾ that "Hemianaesthesia is rare in Hemiplegia. Slight numbness or tingling may be present, or there is loss of sensation after a

day or two, which gradually passes off." Dana⁽³⁶⁾ says "In a good many cases there is a slight amount of Hemianaesthesia during the early stage of cerebral apoplexies, but this almost invariably disappears in a few days or weeks, and it is rare that any anaesthesia of cutaneous or muscular sense is observed." And Bastian⁽³⁷⁾ says that haemorrhage from the Lenticulo-striate artery may cause a minor amount of pressure on the sensory segment of the capsule "so as to produce mere numbness or some slight temporary loss of sensibility." The clinical observations which I have made do not bear out these statements, as in the first place I have found the special senses more often affected than entirely unimpaired, and secondly, diminution of general sensibility has almost invariably been observed to some extent even in lesions of many years' duration. Nothnagel⁽³⁷⁾ says "From my own experience I am inclined to believe that incomplete restoration (of sensation in hemiplegia) is oftener met with than is generally believed:" and with this opinion my observations entirely coincide. It is well known that sensory fibres resist pressure better than do motor, and thus perception of sensation appears to return after a lesion more quickly and more completely than the power of voluntary motion. But in capsular lesions, where the posterior third of the posterior limb has been involved to any extent it does not seem possible for sensation to be entirely regained by the patient. In explanation of its partial recovery it is a feasible supposition that such fibres as are stretched or compressed may recover as the compressing blood clot absorbs, while such as are lacerated give rise to permanent impairment of sensation, and it must be seldom indeed that all escape.

C O R T I C A L D E S T I N A T I O N .

The cortical destination of those nerves of general sensibility passing through the posterior limb of the internal capsule constitutes a vexed question in the subject of cerebral localization. Ferrier⁽²⁸⁾ for many years has upheld the theory that sensation is represented chiefly in the Hippocampal lobe, and is not in any way directly connected with the motor cortex. This conclusion he has based for the most part on observations made on animals after experimental destruction of the Hippocampal and Rolandic areas. The result of these experiments, briefly, appears to have been, first, that sensation is profoundly impaired after removal of the Hippocampal lobe, but returns in a few weeks; and second, that no permanent anaesthesia follows destruction of the motor cortex. The recovery of sensation after the removal of the Hippocampus Ferrier suggests may be due to either an incomplete destruction of the lobe, or to a bilateral representation of sensory impressions. If such an explanation be accepted, then it is inferred that the entire cutaneous sensation from one side of the body may be represented in the small portion of the Hippocampal lobe remaining after the removal of the greater part. This however does not coincide with clinical observations on Hemianaesthesia generally, which demonstrate that impairment of sensibility lasts longest in the limb most paralysed as to motion. And if we suppose sensation to be represented bilaterally to such an extent as to allow of complete recovery in several weeks after the destruction of the sensory cortex of one side, then a capsular lesion should give rise to one of three conditions

1. No affection of sensation on either side
2. Impairment of sensation on both sides of the body.

3. A minor implication of the sensibility of the trunk, supposing Broadbent's hypothesis to apply also to sensory phenomena.

Instead of this, however, an opposite Hemianaesthesia more or less complete, and certainly permanent, follows a lesion of the sensory division of the internal capsule, and this fact suggests that normally one side of the cortex receives general sensorial impressions from the opposite periphery. And Ferrier himself says:- "In respect of tactile sensation there is less bilateral representation in each hemisphere than as regards the other forms of sensibility." Yet tactile sensation is that which is most impaired in the majority of cerebral lesions, and especially in those occurring in the Hippocampal lobe. Again, Ferrier remarks that "practically the whole of the Rolandic area can be removed without permanent loss of sensation," but, from his own experiments it seems that practically the whole of the Hippocampal lobe can be removed without permanent loss of sensation. And if "It requires a lesion of considerable extent in the Hippocampus to produce any noteworthy defect of sensation," a lesion of the same extent in the Rolandic area would probably have as much effect on general sensibility. As Ferrier owns, anaesthesia may occur after a lesion in the motor cortex, and if as he believes this necessitates the involvement of the Hippocampal lobe, then the existence of a second lesion is implied. Beevor, it is true, states that the Hippocampus and the motor cortex are in intimate connection by the fibres of the Cingulum, and Ferrier thinks that by these or similar paths sensory impulses travel from the Rolandic area to the Falciform lobe, thus indirectly affecting sensation in an essentially motor lesion. Were this the case, however, we should expect, in some instances at least, to find degenerated fibres running from the motor

cortex to the Falciform lobe. But instead of this, it rather appears that the ganglion cells of the Cingulum (i.e. the cells from which impulses arise) are situated in the Hippocampal lobe, while only their dendritic terminations are connected with the Rolandic Zone. Now, no lesion, irritative or destructive, of the motor cortex is capable of sending impulses along fibres whose terminations only are represented in that region. Unless it can be demonstrated that there exist ganglion cells in the Rolandic area, the processes of which run to the Hippocampal lobe, - after the manner of the cells associated with reflex action in the spinal cord - we must assume that any impressions passing between these two cortical centres travel from the Hippocampus to the motor cortex.

That the motor area is also the chief centre for the perception of cutaneous sensation is generally believed among American and German writers (except Meynert). In fact Munk⁽³⁹⁾ and Schiff suggested that the so-called motor centres were not motor at all, but that in this region were stored memories of sensation associated with certain movements which are reflexly produced when the centre is stimulated. This corresponds to some extent to the views expressed by Bastian^(op. cit.) who considers the Rolandic area to be the Kinaesthetic centre - that is, the centre for "unconscious impressions from muscles which seem to be so intimately connected with the production of movements." But, without going so far as to consider the so-called motor centre to be entirely sensory, observations based on the different branches of medical science render it reasonable to suppose that sensation to some extent at least is represented here.

Anatomically, it has been demonstrated that some of the fibres of the median fillet run to the motor cortex.

Broadbent⁽⁴⁰⁾ says "I have found that the fibres of the Corpus Striatum (motor) and the optic Thalamus (sensory) run, not one set to the anterior and the other to the posterior end of the hemisphere, but to the very same convolutions." And Foster (Op. cit.) says:- "To this region [the Rolandic zone] passes the direct cerebral continuation of the fillet." Now if the fillet be concerned with the conveyance of sensation - and this seems established almost beyond doubt - then of necessity sensation must be represented to some extent in the area which receives the terminations of its fibres.

Embryologically, Flechsig has demonstrated that the fibres of the fillet receive their myelin at an earlier period than those of the Pyramids;⁽⁴¹⁾ and Dr. Allen Starr⁽⁴²⁾ gives a detailed account of the examination of the brain of a microcephalic infant, in which all the sensory tracts were present, while the motor were absent. In these cases the fibres from the posterior limb of the internal capsule were found to run towards the Rolandic area, distinct from, and developed before, those of voluntary motion, and were presumably connected with the transmission of sensory impulses to the cortical area in which they terminated.

Experimentally, In man Dana⁽⁴³⁾ and Laycock⁽⁴⁴⁾ report cases in which electrical stimulation of definite parts of the motor cortex caused disturbance of sensation. In animals, Horsley⁽⁴⁵⁾, Vulpian⁽⁴⁶⁾, Fripiet⁽⁴⁷⁾, Tripier⁽⁴⁸⁾ and others give details of experiments on dogs and monkeys to show that sensory defects follow motor cortical lesions: and Mott⁽⁴⁹⁾ says he found by experiment that every case of excision of the motor cortex sufficiently extensive to cause monoplegia resulted also in anaesthesia to all stimuli.

Pathologically, Alexander⁽⁵⁰⁾, Dana⁽⁵¹⁾, Starr⁽⁵²⁾, Dreschfeld⁽⁵³⁾, Donald Fraser⁽⁵⁴⁾ and others have described cases in which a monoplegia - resulting from a motor cortical lesion

demonstrated post mortem - had been associated with an enduring anaesthesia; and Petrina⁽⁵⁷⁾ is reported to have described six cases of small lesions in the Rolandic area associated with very marked sensory symptoms. On the other hand Dr. Delepine⁽⁵⁸⁾ records a case of a lesion of all the left motor area and the gyrus fornicatus without any effect of sensation; and Hale White⁽⁵⁷⁾ mentions an instance in which a clot in the frontal lobe and partly in the gyrus fornicatus was unassociated with any motor or sensory symptoms at all. But Saville⁽⁵⁸⁾ records a case of Hemianaesthesia associated with a lesion in the opposite Gyrus Fornicatus, and Dr. Seymour Sharkey⁽⁵⁹⁾ mentions one of Charcot's cases where a lesion of the motor cortex caused no defect of sensation.

But though there may be few isolated instances of lesions in or about the Gyrus Fornicatus associated with anaesthesia, or some more frequently recorded cases of motor cortical lesions without impairment of sensibility, - in the vast majority of cases lesions situated in the Rolandic area implicate cutaneous sensibility to a degree varying with the severity and extent of the lesion. In this connection I may mention a case which came under my own care:-

B. T. Female, aet. 58, was admitted with what was found later to be a depressed fracture of the left parietal bone. A few days after admission she complained of numbness of the right leg, but on examination no defect of sensation could be made out. She gradually lost the power of voluntary movement in this leg, and twitching began in the right arm, while her speech became indistinct so that she called 'leg', "edge." On examination of sensation on the affected side I found it diminished in all its forms, but especially with regard to tactile sensibility, an ordinary touch being quite unappreciated and localization defective. In testing thermal sense, the patient several times failed

to recognise either heat or cold, but occasionally she appreciated the cold tube, though never the warm. Muscular sense in the arm was difficult to test as the patient had also a Colles' fracture, but in the leg it was impaired, though not absent. Pain sensation was present, but dulled. These sensory defects were most marked in the leg, to a less degree on the right side of the trunk, while sensation appeared unaffected in the face. The special senses were unaffected. On the following day the skull was trephined, and a spicule of bone was removed, which was found pressing down into the motor area. The condition of cutaneous sensation appeared practically unaltered after the operation, and the patient died six days later. On examination of the site of the injury septic softening was found in the ascending frontal and ascending parietal convolutions, and apparently confined to this area of the cortex. This case is of interest inasmuch as there existed a definite affection of cutaneous sensibility resulting from a quite evident motor cortical lesion without suspicion of any involvement of the Hippocampal lobe. Sensation was diminished to some extent in all its forms, but the impairment was most marked in the appreciation of tactile impressions. This is so also in lesions affecting the posterior third of the internal capsule, and suggests that the same fibres are involved in both cases. And that the anaesthesia following such an injury is less profound than that which would result from a capsular lesion of the same recent date, is explained by the fact that in the cortical lesion only the diffuse terminations of the sensory nerves are affected, while in the capsule the lesion involves the aggregation of fibres which go to form the sensory crossway.

Clinically. In cases of unilateral convulsions, presumably from irritation in the opposite cortex, the spasm

is usually preceded by a peculiar tingling sensation and followed by temporary hemiplegia and hemianaesthesia. One case of this kind which I have observed was that of a lead worker who was suddenly seized with left sided convulsions affecting the face, arm and leg. On examination I found tactile sensation markedly diminished, and localization defective, all down the left side, but most evident in the arm. Sensation to pain was affected to a less extent. The patient recognised the cold tube always, but heat only occasionally, and the muscular sense appeared little impaired. The special senses were not involved except for a left lateral hemianopia.

(See also Appendix... Case 23. .)

Wilson,⁽⁶⁰⁾ Broadbent,⁽⁶¹⁾ Jackson,⁽⁶²⁾ Knapp,⁽⁶³⁾ Starr,⁽⁶⁴⁾ Beevor and Ballance,⁽⁶⁵⁾ Parker⁽⁶⁶⁾ and others, record cases illustrating the occurrence of anaesthesia following a presumably motor cortical lesion; and Gowers⁽⁶⁷⁾ says "Disease of the motor cortex often causes impairment of tactile sensibility, and when convulsions are caused by disease of this part they often begin with a sensory aura. Thus clinical evidence as well as the facts of anatomy point to the outer part of the hemisphere in the middle region as being the part concerned in sensation." But whether the entire representation is situated in the Rolandic area, or whether the Hippocampal lobe also plays some part in the perception of sensory impulses, is I think still doubtful. For it has been found that entire extirpation of the Rolandic area does not cause complete and enduring hemianaesthesia, and it is also certain that at least temporary hemianaesthesia does follow extirpation of the Hippocampus; and that lesions affecting this area result in some disturbance of sensation. The vast majority of clinical and pathological observations however point to the cortical motor area as being the chief sensory

centre. And until these observations are found to coincide with experimental evidence, the motor cortex for all practical purposes, as far as concerns the clinician, must be considered the principal area in the cerebrum for the perception of sensory impressions.

C O N C L U S I O N .

On reviewing the subject of sensory phenomena in Hemiplegia it seems justifiable to conclude that general and special sensation is more often impaired after a capsular lesion than is usually supposed, even when the lesion is of old standing.

Many Hemiplegias, whether of capsular or of cortical origin, present such definite and unmistakable signs of motor paralysis that the more subjective sensory phenomena are apt to be overlooked. Yet these sensory manifestations are of interest and importance, not only because of their diagnostic value - which as we have seen is undoubted - but also in view of the dubiety still existing as to the locality of the sensory cortex.

In numerous recorded cases of Hemiplegia the condition of sensation is absolutely ignored, or is described generally as "unaffected," leaving the reader in some legitimate doubt as to the care with which the examination of the patient had been conducted. Occasionally, it is true, the impairment of cutaneous sensibility after a capsular lesion is so slight that the patient can feel and localise the gentlest touch. But he cannot tell the difference between the head and the point of a pin and his appreciation of each form of sensation is less acute than on the sound side. Such slight peripheral evidence of a cortical sensory lesion may indeed seem scarcely worth recording, yet if accuracy is to be de-

-sired in the examination of a patient the smallest deviation from the normal should be of as great importance as any well marked defect. A diagnosis founded on the examination of motor phenomena alone without due consideration of any existing sensory impairment is but half the truth and is an omission of valuable and helpful evidence to both the locality and extent of the lesion.

On the other hand, an opinion based on a conscientious study of such motor and sensory phenomena as are presented by the patient, may be confirmed by post-mortem examination, and so lead by the accumulation of evidence to the definite determination of the paths and destiny of the various sensory nerves. Or, - as happens occasionally in the experience of all - should the autopsy reveal a condition entirely at variance with the ante mortem diagnosis, the fact that sensation has been definitely affected in such a case and the degree and nature of the impairment correctly recorded, may be of no small value in the localisation of the sensory pathways.

The testing of sensation is a task which necessitates the expenditure of much time and infinite patience, yet such expenditure appears to be fully justified when we consider its aim to be the elucidation of one of the most puzzling problems in the intricate subject of Cerebral localization.

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C A S E I.

M. J. Female. Aet. 49 years.

HISTORY. The patient had a "stroke" twenty years ago, the history of such being that three days after her first confinement, while scrubbing a floor, she fell down unconscious. On recovering her senses she found her left arm and leg paralysed and numb, her face being drawn to the right and her speech mumbling. Speech recovered first, and then the leg, and though eventually able to raise her arm she could not open her hand.

PERSONAL AND FAMILY HISTORY unimportant.

CONDITION on EXAMINATION. Intelligence average.

FACE:- Slight arcus senilis present in both eyes. No obvious facial paralysis. Speech unaffected.

SENSATION:- Over the left side of the face, there is complete loss of tactile sense and both thermal and pain sense are markedly diminished.

SPECIAL SENSES :-

SIGHT. No Hemianopia. Diminished visual acuity and contracted field of vision in left eye.

HEARING:- Watch faintly heard when laid against left ear. Heard 4 inches from right ear.

TASTE:- Totally lost on left side of tongue, the patient being tested with salt, sugar, quinine and citric acid.

SMELL totally lost in left nostril, when tested with cloves, peppermint, lavender and asafoetida.

UPPER EXTREMITY:- Lies by the side almost quite flaccid with the elbow flexed and the hand closed, secondary rigidity being evidenced only by resistance and pain on attempted extension of the elbow and fingers, and by exaggerated Radial and Deltoid reflexes: there is no power of grasp in the fingers.

SENSATION:- Tactile sense is quite gone over the finger and hand. It is present to a slight extent in the arm, but transmission is delayed and localization inaccurate. Thermal sense is absent over the hand, but the cold tube is occasionally recognised in the arm. Pain sense is also absent over the hand and diminished in acuity in the arm. Muscular sense is gone.

TRUNK. Tactile and thermal senses are diminished over the side of the chest and abdomen. Pain sense though present is somewhat dulled.

LOWER EXTREMITY. Gait typically "paralytic," the left leg being rigid and spastic and the toes scraping the ground with each step. Power of both flexion and extension is diminished on the left side. Reflexes; knee jerks are well marked on both sides, but especially on the left. Plantar reflex is exaggerated and Babinski's toe phenomenon elicited on that side only.

SENSATION. Tactile, thermal and painful senses are lost over the foot, and impaired over the leg to about the same degree as in the arm. Muscular sense is gone, the patient recognizing no position of the limb or movement of the various toes.

DIAGNOSIS: Left Hemiplegia resulting from a lesion involving all the posterior limb of the internal capsule. The history points to embolism of the Lenticulo-striate artery, but this would scarcely account for the definite affection of sensation or for the very fair motor recovery. A haemorrhage therefore is a more feasible supposition, though the cause for such is obscure.

HISTORY: About 15 months ago the patient found on attempting to rise in the morning that he could not move his right leg and arm. He was conscious of a numb sensation all down his right side, but does not think his face was affected, and his speech was all right. He staid in bed for a week, and has never been able to walk properly since, though he can move his arm fairly well.

PERSONAL AND FAMILY HISTORY unimportant.

EXAMINATION: Intelligence rather below the average.

FACE: No facial paralysis. Commencing cataracts in both eyes.

SENSATION... appears perfect to all stimuli.

SPECIAL SENSES: Sight cannot be tested.

HEARING: Quite lost on right side. Watch heard when laid against left ear.

TASTE: Very markedly diminished on right side.

SMELL: Gone on right side.

UPPER EXTREMITY is not obviously affected, not being held in any fixed position, and retaining full range of movement. There is no rigidity, but the grasp is inferior to that of the left hand.

SENSATION: Is diminished slightly to all forms of stimuli, tho' localization is perfect. The patient occasionally does not know which finger is moved and sometimes fails to appreciate a very light touch. This is most marked in the hand and forearm: Above that sensation is almost perfect.

TRUNK: Sensation somewhat dulled all over right side.

LOWER EXTREMITY: The leg lies extended in bed, but can be drawn up and the knee flexed, tho' the ankle and toes cannot be voluntarily moved. The leg feels rigid, ankle clonus is easily obtained, and the knee jerks are exaggerated.

SENSATION Tactile sensation is much diminished especially over the outer side of the leg and the dorsum of the foot.

Thermal sense is also deficient as regards the cold tube, though the hot was almost invariably recognised. Painful sensation was somewhat dulled, though not to the same extent as the others. Muscular sense was affected in so far as the patient did not know which toe was moved and could not differentiate between different weights laid on his foot and leg.

DIAGNOSIS: Left capsular haemorrhage involving both motor and sensory paths in the capsule. Secondary descending degeneration.

General sensation is impaired and special senses are also affected.

HISTORY. Five years ago the patient had a sudden attack of headache and vomiting followed by paralysis of the right arm and leg. Her speech was mumbling, but there was no aphasia. She kept her bed for about a week and then the leg recovered sufficiently to enable her to get about. The arm gradually developed athetoid movements which have persisted ever since.

EXAMINATION: Intelligence good.

FACE: No paralysis. Sensation appears perfect.

SPECIAL SENSES: Sight. No Hemianopia or amblyopia, but visual acuity is defective.

Hearing Diminished in right ear.

TASTE. Gone on right side.

Smell. Gone on right side.

UPPER EXTREMITY. Hangs at the side with the third and fourth fingers extended while the others and the thumb are flexed on the palm. Athetoid movements are marked. Voluntary movement is good. Radial reflex present.

SENSATION appears unaffected, except for a dullness in the appreciation of tactile impressions.

TRUNK: Sensation slightly impaired.

LOWER EXTREMITY. Seems to have recovered very completely, and there is very little evidence of the paralytic gait. Knee jerk is well marked and Babinski's toe phenomenon is present.

SENSATION appears very little affected.

DIAGNOSIS.

Cerebral Haemorrhage involving incompletely motor and sensory part of internal capsule and contiguous optic thalamus. Here the interesting point is the involvement of the special senses to so marked a degree, while general sensation is practically unaffected.

HISTORY. About 15 months ago the patient became troubled with numbness of the right arm and leg, and a feeling of coldness down the right side. This lasted about six weeks, and then one evening on trying to rise from her chair she found she was unable to do so and had to remain all night where she was as she could not move her right side. She was never unconscious, but was troubled with forgetfulness of the names of things. She has never recovered the use of her limbs to any extent.

PREVIOUS HISTORY About twelve years ago she had evidently an attack of acute nephritis, coming on with sickness, diminished micturition and swelling of the face and legs. Urine contains moderate amount of albumin.

FAMILY HISTORY. Grandfather and grandmother both died of apoplexy.

EXAMINATION: Intelligence very good.

FACE: No obvious facial paralysis

SENSATION: Is defective all over the right side.

SPECIAL SENSES: Sight Amblyopia distinctly present in the right eye, evidenced by diminished visual acuity, contracted field of vision and altered perception of colour (bright red is seen as a dirty brownish colour.)

Hearing Entirely gone in right ear:
Watch heard at one inch from left ear.

Taste: Absent over right side of tongue.

Smell: Absent on right side.

UPPER EXTREMITY. Has no voluntary movement except a slight action of the deltoid. Rigidity is very marked and radial reflex is much exaggerated.

SENSATION. Tactile and thermal senses are lost over hand and defective over arm. Painful sensation is present but impaired over arm and especially over hand. Muscular sense is gone.

TRUNK: sensation is affected to about the same extent as over arm.

LOWER EXTREMITY. Is also very rigid: knee jerks are well marked and a slight ^{ankle} clonus is present on both sides.

SENSATION entirely gone to all stimuli over foot.
Much diminished over leg and thigh.

DIAGNOSIS Capsular haemorrhage involving extensively both motor and sensory paths: probably secondary to high arterial tension and arterial degeneration following nephritis.

Both general and special sensations are markedly involved. The absence of aphasia is remarkable.

C A S E 5. G. P. Female. Aet. 67.

HISTORY. In April 1900 the patient awoke one morning and found she could not move her right arm or leg. Her face and speech were not affected and she did not lose consciousness. Her leg improved first and she was able to get about, but could not use her arm much.

PERSONAL AND FAMILY HISTORY. Unimportant.

EXAMINATION. Intelligence good.

FACE: No facial paralysis. Commencing cataracts in both eyes.

SPECIAL SENSES. Sight not tested

HEARING. Watch heard only when pressed against right ear. Heard two inches from left.

TASTE. Gone on right side.

SMELL. Gone on right side.

UPPER EXTREMITY. has free voluntary movement. Radial and deltoid reflexes are well marked.

SENSATION is dulled to all stimuli, especially over the hand, but muscular sense appears perfect. Cold is appreciated more than heat.

TRUNK. Sensation affected less than over arm.

LOWER EXTREMITY has also free voluntary movement.

Knee jerk is well marked but there is no ankle clonus.

SENSATION is affected to about the same extent as in arm and muscular sense is perfect.

DIAGNOSIS.

Left capsular haemorrhage involving sensory as well as motor path.

The special senses here appear to be impaired to a greater extent than general sensation.

C A S E 6. W. P. Male. Aet. 39.

HISTORY. About two years ago the patient drank very heavily for several months and suffered from an attack of delirium tremens. On recovery he found his legs were weak, but he managed to walk with the aid of a stick. Gradually however he noticed a loss of power in his left arm and leg and this was associated with a peculiar numb sensation all down the left side and with occasional twitching of the fingers and toes, which on these occasions became quite blue and cold. He has also suffered occasionally from vertigo and sees objects as if they were swinging in the air.

EXAMINATION. Patient is a very intelligent man, but is evidently nervous, and has an anxious expression.

FACE: No paralysis.

Sensation of touch, pain and temperature are all markedly impaired, this being not quite limited by the middle line but extending about half an inch on the right side of the forehead and chin.

SPECIAL SENSES. Sight. Distinct amblyopia in left eye, consisting of contracted visual field, diminished visual acuity and altered perception of colour.

HEARING: Watch heard only when pressed against left ear. Heard two inches from right ear.

TASTE. Gone on left side of tongue.

SMELL. Gone on left side.

UPPER EXTREMITY. Movements quite free and no obvious rigidity: Grasp is evidently diminished and hand and arm are blue and cold.

SENSATION. Tactile, thermal and painful sense is diminished all over, but especially on hand. Muscular sense appears perfect.

TRUNK. Sensation diminished as on arm, the anaesthesia extending one inch beyond the middle line back and front.

LOWER EXTREMITY appears somewhat stiff and there is a slight drag in walking, though voluntary movement is good. Knee jerk is well marked but there is no ankle clonus.

SENSATION. diminished to touch, pain and temperature all over, especially in foot: muscular sense appears perfect.

DIAGNOSIS Thrombosis of middle cerebral artery? Specific?

HISTORY. When aged nine years the patient had a "stroke" while working. He suddenly lost consciousness and on recovering his senses found his left side paralysed. He was removed to St. George's Hospital (London) and remained there till he had regained the use of his limbs to some extent. Ever since then he has been troubled with "fits" which come on about once a month, but occur more seldom now.

EXAMINATION. Intelligence good. No. cardiac murmur. No albuminuria.

FACE. External strabismus due to blindness in right eye, with which the patient can see only very little.

SENSATION. Generally, in diminished over the left side of the face.

SPECIAL SENSES. Sight.. diminished visual acuity in left eye.

HEARING: Watch heard only when pressed against the left ear. Heard three inches from right.

TASTE: Lost on left side.

SMELL: Ditto.

UPPER EXTREMITY. Movements free. No rigidity, but the arm is weaker than the right. Tactile, thermal and painful sensations all obviously diminished. Muscular sense defective when the fingers were moved.

TRUNK: Sensation diminished all down left side.

LOWER EXTREMITY. Has a slight drag in walking. There is no obvious rigidity.

SENSATION affected much to the same extent as in the arm, but muscular sense appears more impaired in the lower extremity.

DIAGNOSIS : Embolism with softening? involving both distribution of Lenticulo-striate and Lenticulo-optic?

Cannot have been cortical lesion on account of extensive involvement of special senses.

HISTORY. About 3 years ago the patient suddenly fell down while crossing the room, and found she could not use her right side. She gradually became unconscious and on recovery was evidently aphasic. She has never been able to use her right arm or leg since then but the speech has improved.

FAMILY HISTORY. Grandfather, grandmother and uncle also suffered from hemiplegia.

EXAMINATION. Intelligence good.

FACE. Slight flattening of right side of face. Sensation is diminished to slight extent over right side.

SPECIAL SENSES. Sight... distinct hemianopia.

HEARING: Gone on right side. Watch heard three inches from left ear.

TASTE: Gone on right side.

SMELL. Gone on right side.

UPPER EXTREMITY. Movements very limited. The arm feels soft and there is a kind of athetoid movement on voluntary exertion.

SENSATION is only slightly diminished to all forms of stimuli, touch, pain and temperature all being appreciated but less distinctly than on the left side. Muscular sense appears perfect.

TRUNK. Sensation is affected here to the same extent as in arm.

LOWER EXTREMITY. Is very rigid and cannot be flexed either at the hip or knee. No knee jerk can be elicited (probably on account of extreme rigidity)

SENSATION. is affected to the same extent as in arm over the thigh and leg, but is rather more defective over the foot. Muscular sense is difficult to test but appears unaffected.

DIAGNOSIS. Left capsular haemorrhage involving motor and sensory paths. Athetosis indicating involvement of contiguous optic thalamus. Hemianopia.

HISTORY. Four years ago the patient suddenly observed during dinner that he could not speak and on attempting to move he found his left side was powerless and numb. He was never unconscious. He partially recovered his speech in several hours, though it was some months before he could speak distinctly. His leg improved quickly and in a week's time he was able to walk with the help of a stick and could move his arm to some extent.

CONDITION on EXAMINATION. Intelligence fair.

FACE. No facial paralysis. Speech unaffected.

SENSATION. Tactile, thermal and painful sensations all diminished to about the same extent over the left side.

SPECIAL SENSES Sight... visual acuity is diminished in the left eye. No Hemianopia or contracted field of vision.

HEARING is entirely lost on the left side, a watch placed against the right ear is heard.

TASTE. Diminished on left side.

SMELL. Diminished on left side. The patient could smell both lavender and asafoetida but could not distinguish between them except when applied to the right nostril.

UPPER EXTREMITY. The left arm is held flexed at the elbow with the fingers closed on the palm. A fair amount of movement is present, the patient being able to extend the elbow joint to an obtuse angle. He can also open his fingers, but the movements are very slow. The grasp is good, but the fingers are with difficulty unlocked. The Radial reflex is slightly exaggerated. The deltoid is not obtained.

SENSATION. Tactile, thermal and painful sensations are all perceived by the patient, and his power of localization is perfect, but the sensation is much more dull than on the right side and he cannot tell the difference between the head and the point of a pin. Muscular sense is difficult to test as the patient can tell by pain when the arm is moved. But he cannot appreciate different weights laid on his left hand and arm.

TRUNK. Tactile, thermal and painful sensations are affected as in arm over the left side.

LOWER EXTREMITY. Spasticity very marked, the patient having some difficulty in moving the leg and the movements being very slow. He cannot bend the ankle nor flex the toes. Reflexes. Knee jerks very well marked on both sides, but especially on the left. On attempting to lift either leg there is a clonic spasm. Planter reflex is exaggerated, but Babinski's toe phenomenon cannot be obtained as the toes are already hyperextended.

SENSATION. Appears affected as in arm, but the sensations are more dull and occasionally the cold tube is not recognised when testing thermal sense. Muscular sense cannot be tested.

D I A G N O S I S. Haemorrhage from the Lenticulo-striate branch of the right middle cerebral artery.

The haemorrhage in this case has evidently extended into and partially destroyed the sensory fibres in the posterior third of the posterior limb of the internal capsule. The special senses are definitely involved.

HISTORY. Five years ago the patient was confined with her seventh child, and twenty four hours after delivery she was suddenly seized with a feeling of numbness and great weight in the left leg which gradually extended to the arm. She became unconscious and remained so for several hours. On recovery she found she could not move her left side and her mouth was drawn to the right. Her speech was not much affected. The leg did not recover so well as the arm, which ^{she} has been able to move freely for some time, while she can walk only with the help of a stick.

CONDITION on EXAMINATION. Intelligence good.

FACE. No obvious facial paralysis.

SENSATION. Diminished to all forms of stimuli on left side, and transmission is delayed.

SPECIAL SENSES.

Sight Distinct lateral homonymous hemianopia towards right side.

HEARING. Appears equal on both sides:
Watch heard at a distance of 8 inches.

TASTE. Diminished on left side.

SMELL. Diminished on left side.

UPPER EXTREMITY. Though the arm is held flexed, the patient can move it quite freely, and there does not appear to be any secondary rigidity. Radial and deltoid reflexes are not exaggerated, but the power of grasp in the fingers is less than on the right side.

SENSATION. Tactile, thermal, and painful sensations are perceptible all over but transmission is delayed and the sensation is dull. Muscular

sense appears perfect.

TRUNK. Sensation appears perfectly perceived, tho' somewhat dull.

LOWER EXTREMITY. "Paralytic" gait. Left leg is rather rigid, and there is markedly diminished power of movement. Reflexes. Knee jerk well marked on left side and plantar reflex exaggerated. Slight ankle clonus.

SENSATION: Is affected more than in the arm, though the patient can still feel each variety of sensation. Localization of tactile sensibility occasionally defective especially over the foot and here also the hot test tube is occasionally not recognised, tho' she appreciates the cold test. Muscular sense appears perfect.

DIAGNOSIS Left Hemiplegia resulting from haemorrhage from lenticulo striate which had involved also the posterior third of the Internal capsule, and the greater part of which had been absorbed. The maximum destruction had evidently been posteriorly.

Though this case must have been due to cerebral haemorrhage the cause for such haemorrhage could not be definitely ascertained. Embolism would have suggested itself as an explanation of the onset of hemiplegia in so young a patient, but the combination of sensory with motor symptoms renders this improbable.

HISTORY. About a year ago while sitting by the fire the patient felt a peculiar sensation in his left leg, and on attempting to rise found he could scarcely move the leg. He however managed, with some assistance, to walk upstairs, dragging his leg after him. His arm and his face, he says, were not affected at this time. He Staid in bed ten days and then managed to walk with the help of a stick. In November 1899 he again suddenly experienced a similar numb sensation in the left leg, but this time it affected the arm also, and his whole side was powerless, though his face was not affected. He was never unconscious.

FAMILY HISTORY. One brother died of "apoplexy," and one sister of rheumatism.

EXAMINATION. Intelligence good.

FACE. Slight flattening on left side, speech unaffected, sensation unaffected.

SPECIAL SENSES. Sight, diminished visual acuity on left side, but no hemianopia or amblyopia.

HEARING. A watch is heard ticking at a distance of two and a half inches on both sides.

TASTE. Diminished on left side but present.

SMELL. Ditto. ditto.

UPPER EXTREMITY can be freely moved, but cannot be fully extended, the patient complaining of pain at the elbow when this is attempted. Radial and deltoid reflexes are exaggerated. The grasp is fair, though much inferior to that on the right side.

SENSATION. Tactile and pain sense are

almost perfect over the arm and hand, though somewhat more dull than on right side. Muscular sense appears unaffected. Thermal sense is lost over almost all the arm and hands. Several times the cold tube was recognised when applied to the outer surface of the forearm, but when the hot tube was employed the patient invariably said either that it was cold or that he did not know.

TRUNK. Tactile sensation fair all over side, though occasionally not perceived on the left side of the back. Thermal sense diminished, especially with regard to the cold tube. Painful sense not much affected.

LOWER EXTREMITY can be flexed fully at hip and knee but not at ankle. The leg appears slightly rigid. Knee jerk is well marked and plantar reflex is exaggerated, but there is no ankle clonus.

SENSATION. Tactile sense is present over thigh and legs, though occasionally a touch on the dorsum of the foot was referred to "the shin bone." Thermal sense is diminished as in arm, the anaesthesia being most marked on the dorsum of the foot. Painful sensation is only slightly affected, but more dull than on the right side. Muscular sense appears perfect.

DIAGNOSIS Two separate haemorrhages into right internal capsule, at an interval of a few months: the first involving the leg only, - that part of motor capsule immediately contiguous to sensory path: the second, more extensive, implicating sensory as well as motor fibres.

HISTORY. After being "out of sorts" for about a week and complaining of sickness and dragging sensation in the head, the patient suddenly, one day three years ago, dropped her fork during dinner and on trying to pick it up she "fainted." On recovering consciousness she found she could not use her left arm or leg and that they felt numb, and that she could not articulate very distinctly. Her face was not affected. She never regained sufficient power to walk again and has never since been able to use her arm.

PERSONAL HISTORY. History of syphilis, in sore throat, falling out of hair and intractable ulcer of left leg.

EXAMINATION. Intelligence average. Patient is very emotional

FACE. No obvious paralysis

No loss of sensation.

SPECIAL SENSES:- Sight, diminished visual acuity on the left side, but no hemianopia.

HEARING. Equal on both sides, a watch being heard at a distance of six inches.

TASTE: Diminished to some extent on the left side.

SMELL. Ditto ditto ditto

UPPER EXTREMITY Lies across the chest pronated with the fingers bent on the palm. The deltoid action is the only voluntary movement possible. No grasp. Radial and deltoid reflexes are exaggerated.

SENSATION: Tactile sense is present all over upper arm, though transmission is delayed. It is lost over the forearm and hand where a light touch is not felt except once or twice over the extensor surface of the wrist. Thermal sense is diminished all over. Painful sensation is dulled all over. Muscular sense is also impaired, different movements not being recognised.

TRUNK. Sensation to all stimuli slightly impaired.

LOWER EXTREMITY is very rigid and the lower third on the external surface is almost covered by an ulcer which has lasted five years. There are also several pale round scars on the upper part. Movements are very slow and imperfect: knee jerk is difficult to obtain and plantar reflex is present but not exaggerated.

SENSATION: Is very difficult to test because of the scarred and ulcerated con-

-dition of the leg. But all kinds
of sensation appear to be diminished
to some extent.

D I A G N O S I S. Right capsular haemorrhage involving sensory
as well as motor path: secondary descending degeneration:
Cause... Specific arteritis?

Here general sensation appear impaired to a greater extent
than the special senses.

LOWER EXTREMITY. Movements are free. Knee jerk

exaggerated. Ankle clonus, knee clonus and Babinskis toe phenomenon present on the right side.

SENSATION. Tactile and thermal absent over foot, and painful sense is diminished. All senses are present but impaired over leg and thigh. Muscular sense is diminished.

On the 25th March, the skull was trephined over the motor area and a needle inserted in various directions. No surface tumour was detected.

On examination on the 28th, sensation was found affected almost to the same extent as before in the face and arm but was more defective in the leg and foot, which area in the motor cortex had been irritated with the needle.

P.M. There was found a large tumour - glio-sarcoma - occupying all the left internal capsule, corpus striatum and optic thalamus. It extended to the middle line pressing on the anterior corpora quadrigemina and veins of Galen. The Ventricles were found much dilated.

DIAGNOSIS: The complete hemiplegia with descending degeneration indicates involvement of whole motor path: The paraesthesia over the same distribution indicates involvement of whole sensory path. The Athetoid movements indicate implication of optic thalamus; and the slight elevation of temperature on the same side of the body points to the lesion involving also the corpus striatum. These phenomena, together with headache, vomiting and double optic neuritis, - evidences of a general increase of intracranial tension, - make the diagnosis of cerebral

tumour quite clear and also indicate its locality as above described.

The nystagmus is probably explained by the secondary hydrocephalus.

HISTORY. On waking one morning 12 years ago the patient found she was unable to use her left side, and on trying to get out of bed she lost consciousness. At first, on recovery she could not speak but this improved, though her articulation has since been somewhat defective.

FAMILY HISTORY. Father died after a "stroke."

EXAMINATION. Intelligence fair.

FACE. No obvious facial paralysis. Internal strabismus.

Sensation is not affected over the face.

SPECIAL SENSES. Sight cannot be tested as patient has been blind in the left eye for many years.

HEARING slightly more diminished on left than on right side.

TASTE. Diminished on left side.

SMELL. Diminished on left side.

UPPER EXTREMITY is held at right angles, with the fingers flexed on the palm. The arm appears very rigid and the radial and deltoid reflexes are exaggerated.

SENSATION is diminished over the arm and hand, in all its forms, though the cold tube is sometimes appreciated in the testing of thermal sense.

TRUNK. Sensation appears almost perfect.

LOWER EXTREMITY Rigidity is well marked. Knee jerks are well marked and ankle clonus present on both sides, but Babinski's sign is elicited only on left side.

SENSATION is diminished, but only slightly, to all stimuli over thigh and leg and more markedly over the foot.

D I A G N O S I S. Right capsular haemorrhage involving both motor and sensory paths: affecting motor speech (aphemia); secondary descending degeneration.

C A S E 15. S. D. Female. Aet. 50.

HISTORY. On waking one morning two years ago the patient found her left side paralysed and her mouth twisted and her speech gone. She was not unconscious and gradually recovered, till she was able to use her limbs fairly well.

EXAMINATION. Intelligence fair. Heart sounds pure. No albumin in urine.

FACE. Slight flattening on left side.

SENSATION appears perfect all over.

SPECIAL SENSES Sight is equal and good on both sides.

HEARING Entirely gone on left side but apparently unaffected on right.

TASTE Diminished on left side

SMELL Diminished on left side.

UPPER EXTREMITY. Very good voluntary movement. Radial and deltoid reflexes exaggerated.

SENSATION appears practically unaffected

TRUNK " " " "

LOWER EXTREMITY is somewhat rigid. Knee jerk exaggerated and ankle clonus well marked on left side.

Sensation appears almost perfect.

D I A G N O S I S. Left hemiplegia: right capsular haemorrhage involving posterior (sensory) third to slight extent: motor speech (aphemia) recovered.

Here the special senses are more or less impaired, hearing being lost on the affected side, but normal on the sound side.

C A S E 16. A. H. Female. Aet. 56.

HISTORY. About 3 years ago the patient began to be aware of a numbness on her right side, and one day when walking across the floor she fell. She did not lose consciousness but found she could not speak and that her right side was powerless. Her speech gradually improved, and she recovered almost completely the use of her limbs.

EXAMINATION. Intelligence good. Urine contains albumin.

FACE. No facial paralysis.

Sensation perfect

SPECIAL SENSES Sight equal and good in both eyes.

Hearing. Equal and good on both sides.

Smell Diminished on right side.

Taste Diminished on right side.

UPPER EXTREMITY. Voluntary movements perfect. No obvious rigidity save in exaggerated radial and deltoid reflexes. Grasp inferior to left.

Sensation appears very little affected.

TRUNK. Sensation appears very little affected.

LOWER EXTREMITY has a slight drag in walking. Knee jerk is well marked.

Sensation appears almost perfect.

D I A G N O S I S. Left capsular haemorrhage: involvement of sensation evidenced only by partial anosmia and diminished taste.

C A S E 17. M. M. Female. Aet 60.

HISTORY. Four years ago on coming home after a day's work the patient suddenly perceived she could not use her left side though she did not lose consciousness. She lay up for 3 months and then recovered so far as to walk about, but she could never use her arm again.

FAMILY HISTORY. Brother suffered from hemiplegia.

EXAMINATION. Intelligence fair. Arteries very tortuous. No albumin in urine.

FACE. No facial paralysis.

SENSATION slightly impaired to touch, pain, and temperature.

SPECIAL SENSES. Sight diminished visual acuity in the left eye

HEARING. Watch heard only when pressed against left ear. Heard two inches off right.

TASTE. Diminished on left side.

SMELL. Diminished on left side.

UPPER EXTREMITY very rigid. No voluntary movement. Tactile, thermal and painful sensation all slightly diminished especially over the hand Muscular sense is much impaired.

TRUNK. All sensations diminished slightly

LOWER EXTREMITY is also rigid, and knee jerk is well marked, very little voluntary movement.

SENSATION is diminished to all stimuli and muscular sense is much impaired.

DIAGNOSIS. Right capsular haemorrhage involving both motor and sensory paths: permanent rigidity of arm and leg.

C A S E 18. M. B. Female. Aet. 41.

HISTORY: About 12 months ago the patient fell down in the street, and though she was not unconscious she could not rise owing to loss of power on the right side. She was assisted home, remained in bed for ten days and then gradually recovered. Her speech was defective at first, but evidently rather in the direction of difficulty in articulation than from aphasia.

EXAMINATION. Intelligence fair. Speech somewhat slurred and hesitating.

FACE. No paralysis.

Sensation slightly diminished on right side.

SPECIAL SENSES: Sight appears equal in both eyes.

Hearing: Diminished on right side.

TASTE: Lost on right side of tongue

SMELL. Diminished but present on right side

UPPER EXTREMITY is slightly rigid and radial and deltoid reflexes are well marked. Grasp is inferior to that of left hand.

SENSATION. Tactile sensation absent over hand and diminished over arm. Painful sensation is present all over but impaired. Thermal sense is affected in that the patient feels both hot and cold tubes as cold. Muscular sense is defective.

TRUNK. All sensations are definitely impaired.

LOWER EXTREMITY is rather rigid and knee jerk is well marked. Distinct drag in walking.

SENSATION is affected as in arm but to a greater extent. Tactile, thermal and painful senses are all absent over the foot and much diminished over the thigh and leg. Muscular sense is diminished.

D I A G N O S I S: Left capsular haemorrhage involving sensory as well as motor path: partial recovery, - descending degeneration.

HISTORY. About 15 months ago the patient had her right arm amputated for "blood poisoning," and six weeks after the operation she suddenly lost consciousness while lying in bed. On recovery she found she could not move her right leg, that her whole right side felt numb and that she could not express herself in speech. She has never been able to use the leg since, though her speech has improved.

EXAMINATION. Intelligence fair.

FACE. Slight right paralysis evident.

Sensation is impaired to all stimuli but present.

SPECIAL SENSES: Sight: Diminished visual acuity in right eye.

Hearing: a watch is heard only when pressed against the right ear. Heard ten inches distant from left.

Taste)
) Diminished on right side.
Smell)

TRUNK. Sensation impaired all over right side.

LOWER EXTREMITY. Is rather rigid: knee jerk on the right is well marked and ankle clonus is present. Very little voluntary movement is possible.

SENSATION is diminished to all stimuli all over especially on the dorsum of the foot, and muscular sense is impaired.

D I A G N O S I S. Haemorrhage from the left Lenticulo striate artery, affecting also the sensory division of the capsule. Hearing is apparently unaffected on the sound side.

HISTORY. About six years ago as the patient was scrubbing a floor she suddenly found she could not rise. After several attempts she got up and proceeded to bathe her left hand as it felt numb and tingling. While doing so she fell down and remained on the floor until lifted by a neighbour. She was never unconscious and her face does not seem to have been affected, nor was her speech: but she could not move her left arm or leg. She has been bedridden ever since, as her leg never recovered sufficiently for her to move about.

FAMILY HISTORY. An uncle, a brother, and a sister have all suffered from "apoplexy."

EXAMINATION. Intelligence good.

FACE. No facial paralysis. No arcus senilis.

Sensation appears perfect

SPECIAL SENSES: Sight does not appear affected, being equal and good on both sides.

Hearing: equal on both sides, but diminished in power. A watch is heard only when placed against each ear.

Taste appears perfect

Smell appears perfect.

UPPER EXTREMITY lies at the side, supinated, with the fingers flexed on the palm. Voluntary movement is very defective, the upper arm only being movable to a slight extent. The arm can be almost fully extended on passive movement, but feels very rigid. Grasp is fair. Radial reflex exaggerated, but deltoid is not obtained.

SENSATION : Tactile sense appears to be present to some extent all over the arm and

hand, though in the latter, localization is defective, the patient invariably referring a touch on the fingers or hand to the forearm. Pain sense is also diminished, a pin prick being appreciated only when applied with some force. Thermal sense is very defective all over, the cold tube being occasionally recognised but the warm almost never. Muscular sense also deficient, the patient recognizing that a finger is moved but never which finger. She does not appreciate a change in position of the arm, nor are different weights perceived.

TRUNK. Sensation is rather less impaired than in the arm but localisation is defective, and occasionally a touch on the side was referred to the arm. Thermal and painful sensations were also defective

LOWER EXTREMITY is very rigid and there is practically no voluntary movement. Knee jerk well marked on left side and slight ankle clonus is obtained. Plantar reflex is exaggerated and Babinski's toe phenomenon is elicited.

SENSATION: Tactile present to some extent all over thigh and leg but a touch on the dorsum of the foot was invariably referred to the lower third of the leg, and one on the sole was referred to the dorsum of the foot. Thermal sense not much affected over the thigh but the hot tube was never recognised over the leg and foot and the cold but seldom. Painful sense was affected as in arm. Muscular sense is defective in that patient can't tell which toe is moved and has no power to appreciate different weights laid on the leg or foot.

D I A G N O S I S. Right capsular haemorrhage involving both sensory and motor paths: secondary descending degeneration. The sensory paths being obviously involved, the very slight impairment of the special senses is remarkable.

HISTORY. About a year ago the patient woke one morning and found she could not move her right arm or leg and that her whole side felt numb. She could only mumble but could say what she wanted. Her face was not affected and she was never unconscious. She improved in a short time but has never quite regained the power of her right side.

EXAMINATION. Intelligence fair.

FACE. No facial paralysis

SENSATION appears unaffected.

SPECIAL SENSES: Sight equal and good in both eyes

Hearing Diminished on right side: good left.

Taste)
) Equal and good on both sides.
Smell)

UPPER EXTREMITY has free voluntary movement, though it cannot be quite fully extended. The grasp is inferior to that on the left side and the radial and deltoid reflexes are much exaggerated.

Sensation. Tactile, thermal and painful sensation diminished all over hand and arm and localization is defective. The cold tube is occasionally recognised. Muscular sense appears perfect.

TRUNK. Sensation appears almost perfect.

LOWER EXTREMITY can be freely moved. Knee jerks are exaggerated and ankle clonus obtained on both sides Babinski's toe phenomenon present in right foot

Sensation affected to about the same extent as in the arm: as regards perception of touch pain and temperature. Muscular sense appears perfect.

D I A G N O S I S. Right hemiplegia partially recovered, with secondary descending degeneration. Lesion has involved sensory as well as motor fibres in Internal capsule.

Here again the special senses are unaffected in a lesion which has obviously implicated the nerves of general sensation.

HISTORY. Two months ago the patient suddenly fell down while walking across the room. He lost consciousness and on recovery found he could not move his right side nor say what he wanted. The aphasia gradually improved and he has also since regained the power of his limbs to a great extent.

EXAMINATION. Intelligence fair.

FACE. Slight right paralysis.

Sensation to all stimuli rather dulled on right side.

SPECIAL SENSES do not appear to be affected, taste and smell being normal, while the patient can see and hear equally well on both sides.

UPPER EXTREMITY Fair amount of voluntary movement; no obvious rigidity. Grasp less than left.

Sensation to all stimuli is diminished over the arm, and tactile sense appears absent on the hand. Muscular sense is impaired inasmuch as the movements of the fingers are concerned

TRUNK: Sensation slightly impaired all over.

LOWER EXTREMITY is more rigid than arm, knee jerk is exaggerated and ankle clonus present on both sides

SENSATION is diminished all over as in arm and lost to all stimuli over foot. Muscular sense appears gone.

D I A G N O S I S Haemorrhage from the left lenticulo striate artery, involving principally the leg area and the sensory division of the capsule, though the nerves of special sense appear to have escaped.

HISTORY. About 2 years ago the patient first noticed a tingling sensation in her left leg. This lasted about a month and was accomplished by a feeling of weakness in the limb. The leg then began to twitch, the knee and the ankle flexing and extending, but the toes not moving. She gradually lost all power in the leg, but has since regained some degree of voluntary movement. About a year ago the left arm became affected with a similar numb sensation and subsequent twitching: and weakness. This was accompanied by a peculiar "creeping" sensation in the face, especially on the left side and most marked near the mouth. About two months ago her tongue and mouth began to feel numb and she could not take milk into her mouth without spilling it. At this time she could scarcely make herself understood as she mumbled in her speech. This condition has since improved, but her eyesight has failed gradually so that she is now totally blind. Headache and vertigo have been marked since the onset of her illness, and occasional sickness has occurred.

EXAMINATION. Intelligence good.

Face. Slight left facial paralysis. Sensation very little affected.

SPECIAL SENSES. Patient is totally blind from post-neuritic optic atrophy: other senses unaffected.

UPPER EXTREMITY. Fair amount of voluntary movement. The fingers are hyperextended at the first phalangeal and flexed at the terminal joint. There is practically no grasp. Radial and Deltoid reflex present.

SENSATION. Tactile sensation is appreciated all over the left arm and hand and localization is perfect: but the feeling is more dull than on the right side. Pain and thermal senses are also diminished, but present. Muscular sense is defective. The patient occasionally knows which finger is moved, but has no idea on which position the arm is placed.

TRUNK. Sensation affected as in arm.

LOWER EXTREMITY. Voluntary movement is good but there is little strength in the limb. The leg gives an occasional twitch consisting of a flexion of the knee, especially if suddenly touched on the calf.

SENSATION. Is affected in all forms to about the same extent as in the arm.

OPERATION on MAY 30TH:-

A piece of bone two and a half inches in diameter was removed from the right parietal region and the dura was found thickened and the brain bulged through the wound. A tumour was found covered with a thin layer of cortex. The lower angle of the wound was penetrated by the finger to find the lower edge of the tumour which was diffuse and could not be excised. The specimen removed was found to be a gliosarcoma.

On EXAMINATION OF THE PATIENT on June 4th.

There is much more distinct facial paralysis than before: and sensation if anything is rather more dull.

The Upper Extremity is much more markedly paralysed as to voluntary motion, the patient being unable to raise it from the bed.

Sensation as a whole is much more impaired. A light touch is not felt at all over the arm and hand and localisation is often inaccurate. Pain and thermal senses are also affected and muscular sense is gone.

THE LOWER EXTREMITY does not appear to have been so much affected by the operation, the condition being practically as at the previous examination.

D I A G N O S I S. Subcortical gliosarcoma in right Rolandic area, involving radiating fibres.

T A B L E O F C A S E S.

Case	Sex	Age	Hemi- plegia	Extent of Impairment of general sensation.				Extent of Involvement of special Senses				Lesion
				Face	arm	trunk	leg	Sight	Smell	Taste	Hearing	
1	F.	49	Left	Dulled	Hand lost, arm im- paired.	Dulled	Foot lost, leg im- paired.	Amblyopia	Lost	Lost	Diminished	Capsular.
2	M.	60	Right	Perfect	Dulled slightly.	Dulled	Much im- paired.	Cataracts	Lost	Diminished	Diminished	Capsular.
3	F.	67	Right	Perfect	Almost perfect.	Almost perfect.	Almost perfect.	Acuity diminished	Lost	Lost	Diminished	Capsular & Optic Thal- amus.
4	F.	68	Right	Dulled.	Hand lost, arm im- paired.	Dulled	Foot lost, leg im- paired.	Amblyopia	Lost	Lost	Lost	Capsular.
5	F.	67	Right	Slightly dulled.	Impair- ed slight- ly.	Dulled	Slightly dulled.	Cataracts	Lost	Lost	Much dim- inished.	Capsular.
6	M.	39	Left	Dulled	Dulled	Dulled	Dulled	Amblyopia	Lost	Lost	Diminished	Capsular.
7	M.	21	Left	Dulled	Dulled	Dulled	Dulled	Blindness	Lost	Lost	Much dim- ished	Capsular.
8	F.	63	Right	Dulled	Dulled	Dulled	Dulled	Hemianopia	Lost	Lost	Lost	Capsular & Optic Thal- amus.
9	M.	75	Left	Slightly dulled.	Dulled	Dulled slightly.	Dulled	Acuity diminished.	Diminished	Diminished	Lost	Capsular.
10	F.	33	Left	Slightly dulled.	Dulled	Dulled	Dulled	Hemianopia	Diminished	Diminished	Equal and good.	Capsular.
11	M.	64	Left	Slightly dull	Dulled	Dulled	Dulled	Acuity diminished.	Diminished	Diminished	Diminished	Capsular.
12	F.	56	Left	Perfect	Hand lost, arm im- paired.	Dulled	Much im- paired.	Acuity diminished.	Diminished	Diminished	Equal and good.	Capsular.
13	M.	11	Right	Perfect	Impaired	Impaired	Foot lost, leg im- paired.	Optic neuritis.	Diminished	Diminished	Diminished	Capsular; Optic Thal- amus; corpus striatum.
14	F.	64	Left	Perfect	Dulled	Dulled	Dulled	Blindness	Diminished	Diminished	Diminished	Capsular.
15	F.	50	Left	Perfect	Almost perfect.	Almost perfect.	Almost perfect.	Equal and good.	Diminished	Diminished	Diminished	Capsular.
16	F.	56	Right	Perfect	Almost perfect.	Almost perfect.	Almost perfect.	Equal and good.	Much dim- inished.	Diminished	Equal and good.	Capsular.
17	F.	60	Left	Dulled	Dulled	Dulled	Dulled	Acuity diminished.	Diminished	Diminished	Much dim- inished.	Capsular.
18	F.	41	Right	Dulled	Much im- paired.	Dulled	Much im- paired.	Equal and good.	Diminished	Lost	Slightly diminished.	Capsular.
19	F.	58	Right	Dulled	Dulled	Dulled	Dulled	Acuity diminished.	Diminished	Diminished	Much dim- inished.	Capsular.
20	F.	60	Left	Perfect	Much im- paired;	Dulled	Much im- paired.	Equal and good.	Equal and good.	Equal and good.	Much dim- inished.	Capsular.
21	F.	55	Right	Perfect	Dulled	Dulled	Dulled	Equal and good.	Equal and good.	Equal and good.	Diminished	Capsular.
22	M.	43	Right	Dulled	Dulled	Dulled	Dulled	Equal and good.	Equal and good.	Equal and good.	Equal and good.	Capsular.
23	F.	17	Left	Slightly dulled.	Impaired	Impaired	Impaired	Blindness	Equal and good.	Equal and good.	Equal and good.	Cortical.