
*A Clinical Study of Cerebral Localization,
Illustrated by Seven Cases.*

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A CLINICAL STUDY OF CEREBRAL LOCALIZATION,
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DURING the past fifteen years great progress has been made in the study of cerebral localization, and it seems probable that in the immediate future the whole cerebral cortex will be mapped out into small areas; each being associated with a definite and distinct mode of mental action, depending on the peripheral connections of the nerve fibres which terminate in that particular area. A scientific basis for the theory of cerebral localization was obtained by Meynert from a study of the comparative and finer anatomy of the brain; and the wonderful advances made in our knowledge of cerebral anatomy by the researches of Meynert, Gudden, Flechsig, and others, have contributed largely to the perfection of this theory. More important, however, than anatomy in contributing facts in support of the theory of cerebral localization, have been the results obtained from experimental physiology, and from observations of disease in the human brain. Indeed, so large already is the accumulation of reported cases of cerebral disease which support the theory of cerebral localization, that the theory may in a general way be regarded as proved,¹ and it is desirable to publish only cases of such an unusual nature as will throw some light on the obscurity which still involves a great part of the subject. Such cases, it seems to me, are the following.

Of the seven cases here reported, one, Case II., was reported by Monakow in the *Archiv f. Psychiatrie*, the other cases are published now for the first time. I was present at the autopsies of Case I. and Case III., but not having seen the patients during their life, I was obliged to obtain the histories in part from the family of the patients, and in part

¹ For a most excellent article on the localization of cortical lesions of the brain, containing an abstract of all the American cases and references to similar collections of foreign cases, see M. Allen Starr, *AMERICAN JOURNAL OF THE MEDICAL SCIENCES*, vol. 87, pp. 65 and 366, and vol. 88, p. 114. Since Starr's article appeared a number of additional cases have been reported, notably a collection published by C. Günther in *Zeitschrift f. klin. Med.*, vol. 9, p. 1.

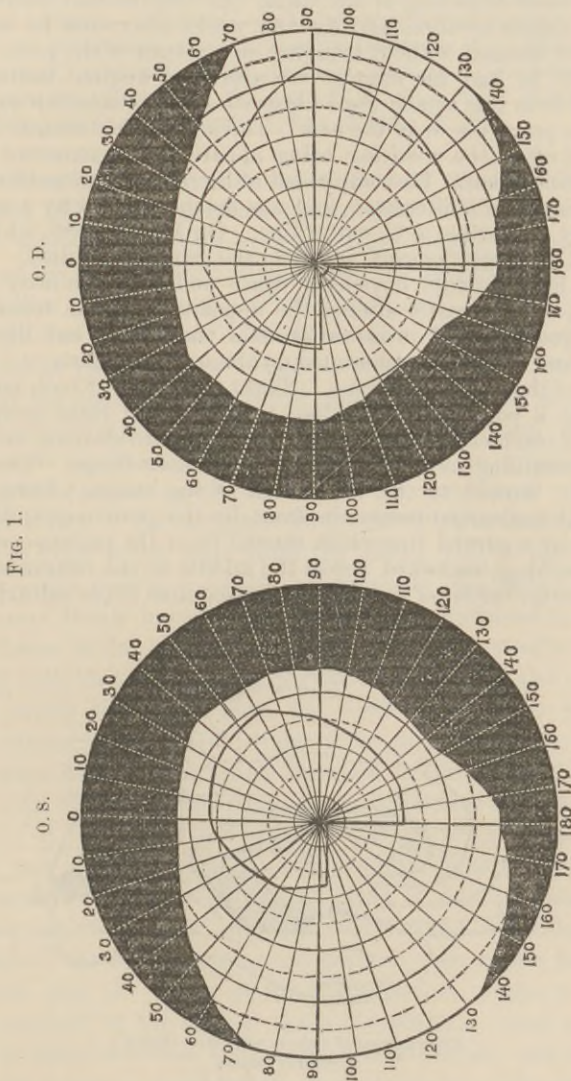
from their physicians, Drs. Boyd and Merrill in Case I., Drs. Curtis and Van der Veer in Case III., who have kindly permitted me to publish the cases.

CASE I. Defect in the fields of vision involving the lower left quadrant of each. Atrophy of the lower half of the right cuneus.

May 20, 1886. H. M., male, æt. fifty-seven, married, of extremely nervous temperament. In 1869 he had a severe attack of double pneumonia, and during the year following he had slight attacks of vertigo while walking, which were attributed to weakness; with this exception he never suffered from any severe sickness. From 1877 until his death, he was troubled by slight deafness and by more or less roaring in his ears, which was especially constant and severe during the last two months of his life. In 1882 he had a large carbuncle on his neck, and after that time he seemed less vigorous than before. In 1884 he began to notice that when hurrying or walking up hill he was frequently compelled to stand still on account of precordial pain, and on June 23d of that year he had a very severe attack of pain in the cardiac region, extending into the left arm, and accompanied by extreme pallor, profuse perspiration, and the conviction that he was dying. On June 29th he got out of bed early in the morning, and while stooping to place a basket of silver outside of the bedroom door he complained of vertigo, seemed bewildered, and repeated over and over again questions as to the time of day, where he was, etc. He could not find the bed although standing near it, and begged to be led to it. At eight o'clock he arose to dress, but could not remember which article of clothing he ought first to put on; he dared not cross the room as all seemed dark before him, and "he was on the edge of an abyss." Everything seemed changed to him, nothing natural, even his breakfast of clam broth when brought to him he called cucumbers. After breakfast he slept soundly for several hours. He awoke with pain over his right eye, and during that day and the next this pain continued and his face was flushed and hot, but he seemed to have little or no trouble with vision.

During the next week he went out on the street a little each day, and on July 5th he went to a large hotel in the country. The fatigue of the journey and the confusion of the hotel seemed to bring back the mental confusion or disturbance of vision. At times he seemed puzzled; he could not tell how he had entered the dining-room, nor remember his table in it, nor find his bedroom alone. He was timid and conscious of his trouble, and would not be left alone for a moment lest he should lose his way. This confusion of vision or of mind gradually passed away, but his family noticed that he did not see a dish or plate which the waiter offered him on his left side, and he often told of narrow escapes that he had from being run over on the street. In November, 1884, he became very dejected, morose, and melancholy, especially on religious subjects; at other times he became greatly excited. Early in December he was examined by Dr. Merrill, who found normal reaction of pupils, normal appearance of fundus, vision and color perception perfect, a defect in the fields of vision involving almost the whole of the left lower and the peripheral portion of the left upper quadrants in each field. The defect was somewhat more extensive, especially as regards the upper

quadrant, in the left field of vision than in the right. The fields of vision were carefully mapped out a number of times between December 3, 1884, and March, 1885, and always with the same result.

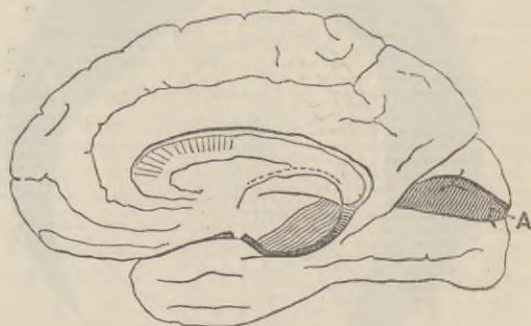


From this time the condition of the patient did not change materially. He continued to be very nervous and, at times, irritable and suspicious. He exhibited no paralysis of motion or sensation. He had little or no pain in the head, except that once after writing much, which always was a great mental strain for him, he complained of pain behind the left

ear, and said that he had with it strange hallucinations. His memory was weak in regard to names; he often called the same person by several names, in the course of a conversation; while in other respects, as in recognizing faces, it was excellent. He slept but little, and in his sleep there was much twitching of his limbs. In 1885 he had another severe attack of angina pectoris, and for two weeks afterward he could walk only a short distance without bringing on a return of the pain. In February, 1886, he had his most severe attack of angina, lasting several hours, and from this attack until his death he was scarcely a day without pain in the precordia or in the arm. During the last month of his life he vomited often, the vomiting being apparently unconnected with any gastric derangement. He complained of increased dulness of vision and of greater angle of obliteration, and was much troubled by a new building near by appearing to be out of line. On May 7, 1886, while quietly walking in the street he sank gently to the ground and died.

Autopsy held thirteen days after death and after the body had been embalmed, which greatly altered the appearance of the tissues. Body was in a good state of preservation and the tissues cut like leather. Skull-cap and dura mater presented no abnormal appearance. Moderate atheroma of the cerebral arteries. Slight dilatation of both lateral ventricles. At a point on the median surface of the right occipital lobe there was a complete atrophy of the cerebral convolutions, only a trace of them remaining as a delicate gray gelatinous fringe. This atrophy was strictly limited to the lower half of the cuneus; being bounded below by the calcarine fissure, in front by the parieto-occipital fissure, and above by a curved line which started from the parieto-occipital fissure and arching backward across the middle of the cuneus terminated at the posterior border of the median surface close to the calcarine fissure.

FIG. 2.



Median aspect of right hemisphere. (Ecker.)

A, point of ~~atrophy~~

atrophy

The white matter underneath the point of atrophy was softened to a depth of about one-third of an inch. There was no deposit of pigment in the neighborhood. The corresponding point on the left occipital lobe showed no atrophy, nor did any of the other cerebral convolutions. Sections through the brain substance, the optic thalami, and the other

ganglia at the base revealed nothing abnormal. The optic nerves and tracts showed no macroscopic atrophy or degeneration. No microscopic examination was made.

The lesions found in the other organs were interstitial nephritis with cysts, moderate degree of atheroma of coronary arteries and of commencement of aorta. The wall of the left ventricle of the heart was very thin, without presenting any macroscopic increase of connective tissue, at a point near the middle of the posterior surface. No valvular lesion. General adhesive pleuritis. Patches of old cicatricial tissue, some of them calcified, were found at the apices of both lungs.

In this case there is a lesion which destroys the lower half of the right cuneus, and there is one constant symptom which is present during the whole course of the disease; a blindness limited to the lower left quadrant of the field of vision of each eye. In the absence of any other cerebral lesion the destruction of the lower half of the right cuneus must be regarded as the cause of the blindness in the lower left quadrant of each field of vision. Such a case as this, in which a homonymous *quadrant* of the fields of vision is lost and there is found as cause of it a limited cortical atrophy, has never been reported; and in order to understand its significance it is necessary to call to mind some well-established facts regarding the anatomy of the optic fibres.

That the cortex of the occipital lobes is the point of final termination of the nerve fibres which start from the retinae and is the perceptive centre for sight may be regarded as definitely established, since the experiments of Munk¹ have been, in part at least, confirmed by numerous cases of disease in the human brain. It is now universally considered that in the optic chiasm there is a partial decussation of the optic fibres of such a nature that fibres from the outer half of each retina pass directly backward to the optic tract of the same side, while the fibres from the inner half of each retina decussate in the chiasm and pass to the optic tract of the opposite side. Each optic tract is made up, therefore, in part of fibres from the retina of the eye of the same side and in greater part of fibres from the retina of the opposite eye; the ratio of the crossed to the uncrossed bundle being as 3 to 2. Consequently, while a destructive lesion of an optic nerve causes a blindness of one eye, a lesion of the optic fibres at any point in their course between the optic chiasm and the cortex of the occipital lobe causes a blindness limited to one-half of the field of vision of each eye. Such a condition of things is called a lateral homonymous hemianopsia,² and it may be produced by a lesion, either of one optic tract, or of the pulvinar of one optic thalamus, or of the posterior part of one internal capsule, or

¹ Ueber die Functionen der Grosshirnrinde, Berlin, 1881.

² Hemianopsia means a loss of one-half of the field of vision, hemiopia means a blindness of one-half of the retina; a right hemianopsia is, therefore, the same as a left hemiopia.

of the optic fasciculus as it passes backward from the internal capsule to the occipital lobe, or of the white matter of the occipital lobe, or of the cortex of the occipital lobe. A number of cases have been reported in which the lesion was confined more or less accurately to one or the other of these parts.¹ In all such cases when the lesion is situated on the left side of the brain the blindness is limited to the right half of each field of vision, and when the lesion is situated on the right side of the brain the blindness is limited to the left half of each field of vision. In many of the reported cases the hemianopsia is complicated by the presence of other nervous symptoms, which vary in character according to the situation of the lesion. When the lesion is confined either to the medullary substance or to the cortex of the occipital lobe, the symptom of hemianopsia is alone present and the reflex action of the pupil to light is normal. When the lesion involves the posterior part of the internal capsule the hemianopsia is associated with hemianæsthesia, and, if the lesion be extensive, with hemiplegia also. In this case also the reflex activity of the pupil to light is normal, whereas when the lesion is further down in the course of the optic fibres the reflex activity of the pupil to light is abolished or greatly impaired. When the lesion involves the pulvinar, the hemianopsia is frequently associated with hemianæsthesia and hemiplegia, because the lesion is usually not limited to the pulvinar but involves also the internal capsule. When the lesion involves the optic tract the hemianopsia is usually associated with hemiplegia and paralysis of some of the ocular muscles. Finally the hemianopsia sometimes occurs associated with aphasia, alexia, word blindness, and more or less complete hemiplegia and hemianæsthesia as a result of extensive occlusion of the cerebral arteries, especially of the left middle cerebral. These points and many others in regard to hemianopsia are well stated in an excellent paper in the *Journal of Nervous and Mental Diseases*, 1886, p. 1, and also in the *Archives de Neurologie*, 1886, i. p. 176, by Dr. E. C. Seguin, which is the most valuable article on the subject of hemianopsia with which I am acquainted.

Although in Case I. the blindness was, for the most part, limited to one-quarter of the field of vision, yet it must be classed for the present among the cases of hemianopsia or of hemiopic defect; because no class of tetartanopsia, to which it more properly belongs, is recognized. This case makes it probable that the fibres from the right upper quadrants of each retina terminate in the lower half of the right cuneus. It is true that not only the cortex of the lower half of the cuneus is completely atrophied, but also that the white matter immediately beneath it is the seat of softening. This focus of softening is, however, of little depth, and affects

¹ For a summary of the literature see Seguin, *Journal of Nervous and Mental Disease*, 1886, p. 1; Starr, *AMERICAN JOURNAL OF THE MEDICAL SCIENCES*, 1884, i. p. 65; Marchand, *Archiv für Ophthal.*, 1882, ii. p. 63.

principally the fibres running to the atrophied convolutions, and at most can involve only those fibres which run to the cortex in the immediate neighborhood of the atrophy. It may well be that this softening involving a few of the fibres running to the adjacent convolutions, causes the blindness in the peripheral portion of the left upper quadrant of each field of vision. So far then as this case shows anything in regard to the central termination of the optic fibres, it proves that the fibres from the upper right quadrant of each retina terminate in the lower half of the right cuneus.

Five cases have already been reported in which hemianopsia has been due to a lesion of the cuneus and of the adjacent part of the median occipito-temporal convolution;¹ so that it may now be considered definitely settled that in this portion of the cortex of the occipital lobe the fibres from homonymous halves of the retina have their final termination. Case I. not only supports this view but it carries the localization a step further, inasmuch as it shows that the fibres from the right upper quadrant of each retina terminate in the lower half of the right cuneus. The fibres from the right lower quadrant of each retina must, therefore, terminate either in the upper half of the right cuneus or in the right median occipito-temporal convolution. The cases of Seguin, Haab, and Heguinin can only be explained by this latter view; while the cases of Féré and Monakow can be interpreted either way. From an inspection of the plates in Seguin's article, which represent the cortical lesions that have been found in cases of hemianopsia, we are forced to the conclusion that *the fibres from the right upper quadrant of each retina have their final termination in the lower half of the right cuneus, and the fibres from the right lower quadrant of each retina terminate in the adjacent part of the right median occipito-temporal convolution.* Of course, the same relationship holds good between the left half of each retina and the left cuneus and left median occipito-temporal convolution.

If it be true that the optic fibres terminate in such a small portion of the occipital cortex, the question presents itself, What is the function of the remainder of the occipital cortex? That the cortex of the whole occipital lobe is the perceptive centre for sight, is shown not only by experiments on animals, but also by the fact that, in man, lesions of the occipital lobes, elsewhere than in the cuneus and median occipito-temporal convolution, cause disturbances of vision. It might be thought that, inasmuch as in most, if not in all, uncomplicated cases of hemian-

¹ Heguinin and Haab, *Klin. Monatsbl. f. Augenheilk.*, 1882, S. 141; Féré, *Archives de Neurologie*, 1885, I. 229; Monakow, *Archiv f. Psychiat. u. Nervenkrankh.*, 1885, S. 151; Seguin, *Journ. Nerv. and Ment. Disease*, 1856, p. 1. In a sixth case reported by Wilbrand (in Gräfe's *Archiv f. Ophthalm.*, Bd. 31, S. 119), the lesion included this area but also extended beyond it, and in two cases, the one reported by Curschmann, and the other by Westphal, *Archiv f. Psychiat. u. Nervenkrankh.*, Bd. 11, S. 822, the lesion involved this region, but also involved the white matter beneath it to a considerable depth.

opsia the central vision is intact, the very important fibres from the macula do not run to the cuneus or to the median occipito-temporal convolution, but have a wide connection with the rest of the occipital cortex. Such a supposition is immediately disproved by cases in which the lesion involved either an optic tract¹ or an entire occipital lobe,² and yet central vision was unimpaired; so that the fact that central vision is normal in cases of hemianopsia must be explained in some other way.³ The function, then, of the rest of the cortex of the occipital lobe must be sought in another direction, and in order to comprehend it, it is necessary to have clearly in mind the more important elements of a complete visual perception.

When the image of an external object is thrown upon the retina, nervous impulses are conducted along the optic fibres to their point of termination on the median surface of the occipital lobes, and give rise to a visual sensation and a very simple representation of the external object. The perception of the object is something different from this, and depends not only on nervous sensations originating in the retina, but also on feelings of innervation of the ocular muscles which are either taking place at the time of the perception, or by means of which in past time each point of the retina has become associated with a relative position in space. Another element in a complete visual perception is the binocular effect produced by a combination of the nervous impulses coming from each retina. The visual perception of an object is, therefore, distinct from, and much more complicated than, the visual sensation which is derived merely from the nervous impulses originating in the retina unassociated by education with other sensations. Such simple visual sensations occur alone only in babies or in blind persons to whom sight is suddenly restored; ordinarily the visual sensation is merged into and lost in the visual perception. Furthermore, every visual perception produces a permanent change in the occipital cortex, in consequence of which thereafter a memory of this perception is easily brought into consciousness. If the same object is seen again after a varying interval, the perception calls up more or less easily the memory of it, and the individual remembers that he has seen the same object before—he recognizes it. Every act of perception is almost inseparably connected with an act of recognition. It is very probable that the simple act of visual

¹ Hirschberg, Virchow's Archiv, Bd. 65, S. 116.

² Baumgarten, Centralbl. f. d. med. Wiss., 1878, S. 369.

³ In order to explain the normal central vision found in cases of uncomplicated hemianopsia, Schweigger (Gräfe's Archiv f. Ophthal., Bd. 22, S. 276) thought it possible that fibres from each macula lutea might be connected with both cerebral hemispheres. Mauthner (Vorträge a. d. gesammte Gebiet d. Augenhellk., Bd. 1, S. 360) states, however, that since in cases of hemianopsia one half of the macula lutea is intact, "upon this half of the macula images can be projected which are as sharply defined as ever, and, therefore, the persistence of normal central vision is not remarkable." Certainly, in such cases it requires but a very slight lateral movement of both eyes to throw the image on the healthy half of the macula.

sensation may be associated with cellular activity in a different part of the occipital cortex from that accompanying the complicated acts of perception and recognition, and it is, therefore, quite possible that a lesion of the median surface of the occipital lobe destroying the point of termination of the optic fibres might cause complete blindness in the corresponding halves of the retinae, and that a lesion of the convex surface of the occipital lobe might prevent full visual perception and might destroy all the memories of things that had been seen while simple visual sensation might be preserved.

In support of this view, and more especially on account of its great intrinsic interest, a very brief report is here introduced of a case of Monokow,¹ in which the symptoms were carefully observed, and in which the central nervous organs were examined with extreme care and exactness.

CASE II.—J. B., *æt.* seventy, an active, amiable, married man, with no hereditary taint. In 1878, he had several severe epistaxes, and soon afterward an apoplectic attack followed by a transient left hemiparesis, slight aphasia, hallucinations of sight and weakness of sight. These symptoms slowly disappeared and there remained only slight unsteadiness in walking, slight disturbance of sight, slight mental weakness and melancholia. In 1879, he had an epileptic attack, the convulsive movements being limited to the left side of the body, followed by a transient slight left hemiparesis, and the existing disturbance of vision (probably hemianopsia?) became more decided. He remained fairly well till January, 1882, when he complained that his sight was rapidly failing, although an ophthalmoscopic examination revealed nothing abnormal. In February, 1882, he had an apoplectic attack with transient left hemiparesis, blindness, hallucinations of sight, and disturbance of speech. Motility returned in a few days, but the disturbance of speech and an almost complete blindness remained. The patient, however, was no longer conscious of his blindness and did not even speak of dimness of vision, of which he complained in January. He often said that he was stupid, old, etc., but never that he was blind, although he thought that he was in a cellar and cried for light and fire. At other times he thought he was outside the house when he was really in it. During the last half year of his life the condition of the patient was substantially as follows: There was no disturbance of sensibility. The left hemiparesis was very slight. There was nothing abnormal about his speech except its irrelevancy due to his inability to understand spoken words. He heard noises and connected the proper associations with them. When, for example, the door was opened he asked who had entered. When he was spoken to he was conscious of it, listened attentively, and made answer, but his answer showed that he had not at all understood what was said to him. He understood only two words, "father" and "adieu." The patient had complete left hemianopsia. On the other hand, he saw things on his right side; he walked about the room without stumbling over the furniture; he found the door without difficulty and walked from one room into another; he found his bed and sat on it: but only when all these things were on his right side; to all things on his left side he was entirely blind. Although he saw things on his right side, more or less distinctly, yet he did not recognize them. He could not eat his food, though it was before him, and he was complaining of hunger; it was only when the food was placed in his hand and he felt it that he ate it greedily. Rapid and threatening movements of the fist toward his eye did not move him in the least. He could not recognize his wife except by the sense of touch. Often he called for his wife when she was by his side,

¹ Archiv f. Psychiat. u. Nervenkrankheiten, Bd. 16, S. 166.

and she was able to quiet him only by caresses and petting, and not by her voice, nor by standing in front of him, nor on his right side. He took interest in things about him, had a good memory, and correct judgment. He had hallucinations of sight and hearing but had no delusions, and was cleanly in his habits. In November he became drowsy, the drowsiness deepened into coma, and he died on December 17, 1882. From the very accurate and minute report of the examination of the brain I mention only those lesions which interest us in this connection. In the right hemisphere was found an almost complete atrophy of the cuneus, the median occipito-temporal, and the descending occipital convolutions; and associated with this atrophy was a degeneration and atrophy of the optic fasciculus, the pulvinar, the external geniculate body, and the optic tract, all of the right side and of both optic nerves. In the left hemisphere was found a great atrophy of the superior and middle temporal convolutions, a slight atrophy of the middle and inferior occipital convolutions, and a softening of the white matter beneath all of these convolutions. The lesion of the right hemisphere was of older date than that of the left.

In this case the lesion of the right occipital lobe caused a left-sided hemianopsia, and it is one of the cases which prove that the optic fibres from the right half of each retina terminate in the right cuneus and right median occipito-temporal convolution. The descending degeneration connecting these atrophied convolutions with each optic nerve also speaks strongly in favor of this anatomical connection. The lesion of the left occipital lobe did not involve these convolutions, and the patient saw things with the left half of each retina—*i. e.*, objects on his right side. Although, to a certain extent, he had visual sensation of objects situated on his right side, yet he did not perceive them fully or recognize them; not even recognizing his food or his relatives. This failure of complete intelligent perception must have been due to the lesion on the convex surface of the left occipital lobe, and, therefore, the cortex of the convex surface of the occipital lobe seems to be essential to the complete perception and recognition of things seen, just as the cortex of the temporal lobe is essential for the recognition of spoken words.

From this case, then, it seems probable that the cuneus and the median occipito-temporal convolution is the portion of the occipital lobe where the optic fibres terminate, and is the point for simple visual sensation; while the cortex of the convex surface of the occipital lobe is the point where the visual perceptions are completely elaborated and are fully recognized. That atrophy of the cortex of the occipital lobe will produce a condition of consciousness in which things are seen to a certain extent, but are not completely recognized and understood, appears probable from those not very rare cases of progressive paralytic dementia in which, especially after apoplectiform attacks, the patient sees but does not understand what he sees.¹ Such cases have also been reported as occurring in sane persons.² Very few cases have been reported in which

¹ Fürstner, *Archiv für Psychiatrie und Nervenkrankheiten*, Bd. 8, S. 162, und Bd. 9, S. 90. Stenger, *Archiv für Psychiatrie und Nervenkrankheiten*, Bd. 13, S. 218.

² Wilbrand, *Ueber Hemianopsie*.

the lesion has been strictly limited to the cortex on the convex surface of the occipital lobes, so that it is impossible yet to be certain whether such lesions cause only a loss of full perception and recognition while a certain amount of simple visual sensation remains, as in the case of Monokow; or whether there is a complete hemianopsia, as in a case (which I have not had an opportunity to read in the original) reported by Westphal,¹ but the former view seems the more probable. Whether, as is the case in both motor and sensory aphasia, the left occipital lobe is more important than the right as a centre for full visual perception cannot as yet be decided.

From the study of hemianopsia thus far made, the following deductions may be drawn:

1st. The optic fibres from the right upper quadrant of each retina terminate in the lower half of the right cuneus.

2d. The optic fibres from the right lower quadrant of each retina terminate in the adjacent part of the right median occipito-temporal convolution.

3d. The lower half of the cuneus and the adjacent part of the median occipito-temporal convolution is the point of termination of the optic fibres from the homonymous halves of the retina; the right half of each retina being represented in the right occipital lobe, and the left half in the left lobe.

4th. On the median surface of the occipital lobe take place those actions which are associated with simple visual sensations.

5th. On the convex surface of the (left?) occipital lobe take place those actions which are associated with complete visual perception and recognition.

Before dismissing Case I. from our consideration, there remain one or two points worthy of mention. The mental disturbance due to the failure of the patient to recognize familiar objects, which characterized the commencement of the attack, was due probably not so much to the lesion of the cuneus as to the disturbance in the circulation in the adjoining portions of the brain, especially in the rest of the cortex of the right occipital lobe, and to a less degree in the left occipital lobe also. The recurrence, seven or eight days later, of this failure to recognize objects may be explained either by his being placed in strange surroundings, or, more probably, by the circulatory disturbances taking place in the occipital lobe consequent upon the reactive inflammation surrounding the point of softening. The attacks of angina pectoris were probably due to the atrophy of the ventricular wall at or about the middle of its posterior surface, which atrophy, very likely, resembled the cortical atrophy in that it depended on embolism, or thrombosis.

¹ Charité Annalen, 1882, p. 466.

Case II., besides being of great value in connection with the subject of hemianopsia, is also of interest in that it shows that an atrophy of the superior and middle temporal convolutions on the left side of the brain causes an inability to understand spoken words, although sounds are still heard. As a consequence of this inability to understand spoken words, the symptoms of sensory aphasia occur—*i. e.*, the patient, not understanding what is said to him, makes irrelevant remarks, and if by any chance in speaking he uses the wrong word, he cannot correct himself by the sense of hearing. Many cases have now been reported in which the inability to understand spoken words and the consequent sensory aphasia have depended on a lesion of the superior, or of the superior and the middle temporal convolutions on the left side;¹ so that we may justly conclude that the memory of spoken words depends upon the integrity of the left superior temporal convolution, and that the full perception and recognition of spoken words are associated with actions taking place in that portion of the cortex. In what portion of the temporal lobe the fibres of the auditory nerve actually terminate is not known, but that they must do so at some point other than the superior and middle convolutions is evident from the fact that when the whole temporal lobe, or the bundle of fibres which runs from the internal capsule to the temporal lobe is destroyed, there results not merely inability to understand spoken words, but complete deafness.²

CASE III. *Impairment of motion and sensation in right arm and leg; agraphia, alexia, and aphasia. Atrophy of lower three-quarters of posterior central convolution, and of the inferior parietal lobule of the left cerebral hemisphere.*

March 11, 1886. R. B., male, set. sixty-six, married, lumber merchant. With the exception of an attack of pleurisy, when quite a young man, he has always enjoyed good health. He has been active and has devoted himself to business. On September 9, 1876, he was taken sick with typhoid fever, which lasted nearly six weeks, and was of moderate severity, his temperature never rising above 103° F. In the third week of this fever his family noticed one afternoon that he had suddenly lost his power of rational speech and could not make himself understood. On the arrival of the attending physician it was found that the patient had a right hemiplegia and aphasia. For some time after this the patient was slightly delirious and complained of some extra legs in bed with him, etc. After the fever ceased he remained much prostrated for a long time, and went down stairs for the first time in February, 1877. The paralysis of the right side of the body, especially of the arm, continued, and he had well-marked aphasia. Frequently at table he asked for a chicken when he wanted an egg, etc. He always knew in such cases that he had used the wrong word and recognized the right word as soon as he heard it.

¹ Seppilli, *Revisit. Speriment. d. freniatrie, etc.*, 1884, x, p. 94. Amidon, *New York Med. Journal*, vol. 41, pp. 113, 181.

² Wernicke und Friedländer, *Fortschritte d. Med.*, 1883, No. 6; also the case of Hutin cited in Wernicke's *Lehrbuch der Gehirnkrankheiten*, Bd. 2, S. 180.

On July 6, 1877, he was examined by Dr. Clymer, of New York, who found: "Right hemiplegia and aphasia. In right lower limb motor power fair, sensibility impaired and perverted. In right upper extremity motor power fair in arm and forearm, but motility of hand impaired; sensibility impaired and perverted, particularly in hand; sensations retained for some minutes after impact. There is a decided tendency toward contracture in the muscles of the right hand." Dr. Clymer also states in this letter that "there is probably an irritative process going on in the brain from the symptoms complained of." On October 25, 1877, Dr. Clymer examined him again and wrote: "I found him no worse and, I think, in some respects better. He drops fewer words and uses his words more correctly. He has, he tells me, less confusion. He says he does not get strong, and there is no improvement in his right arm. His mouth, too, is badly cankered." On December 7, 1877, Dr. Clymer wrote: Mr. B. has gained in the five weeks between his visits to me very much."

In the year 1878, the patient improved steadily. He walked without limping. He was able to use his right hand in cutting his food, in eating, etc., although it was awkward. He was able to dress himself and to take care of himself, but whenever he could he used the left hand in preference to the right. But the difficulty with the right hand seemed to be awkwardness rather than weakness. He complained that his hand felt numb, and he kept rubbing and working it almost constantly. The aphasia almost entirely disappeared, so that he resumed business and travelled about a good deal and had very little difficulty in expressing himself, especially when he was interested in what he was saying and was a little excited. His principal trouble, and the one which did not improve at all, was that he could not spell, read, or write. He could not spell the simplest word, such as "cat," from memory. There was a sign opposite his house on which was the name John Kingsbury. Many times a day he would look at the sign and spell out the name, J-o-h-n K-i-n-g-s-b-u-r-y. The John he almost always got correctly, but it was rather the exception when he read all the letters of Kingsbury without a mistake. After having spelled the name John by reading it many times, he would shut his eyes and try in vain to spell from memory the name John which he had just spelled by reading it. He was entirely unable to read. He might read one, or perhaps two words correctly, but could go no further. He understood perfectly whatever was said to him. He never learned to write with his left hand. He could sign his name with his right hand if he went at it with a rush, and everything went smoothly, but if by any chance he was stopped in the middle of the name he could not finish it, not knowing what letter came next. If the next letter were told him, he might, perhaps, finish the signature. He was able to write a little if some one told him the letters of most of the words. The following are fac-similes of parts of about the only two letters that he wrote during his sickness. He frequently complained of a "dull, bad feeling in his head." At times his mind seemed clear, but at other times he seemed dull and could not clearly comprehend some simple business transaction. At one time for several months he was very despondent, and thought that he had no money and must go to the poor-house. Before his sickness he was subject to sick headaches, but they did not trouble him much during his sickness. Toward the end of the year 1878, he had a severe attack of

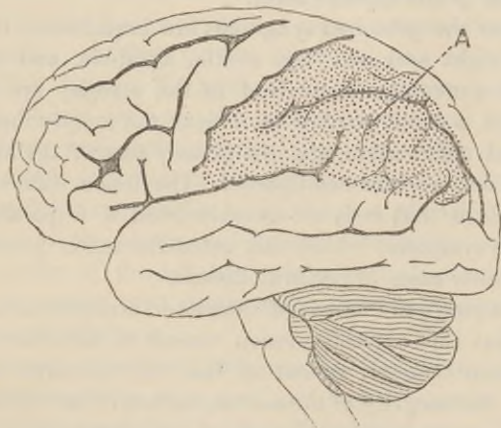
I was to your
 office this afternoon
 this afternoon
 to see you to
 wish me to go
 to Blueken Tabin
~~to~~ Hunting and
 Stoving as it is
 good weather
 for you

I cannot think
 how to make
 letters will
 I ever be able
 to understand
 or ever will
 you will see
 that I cannot
 Compose well

gall-stones which confined him to bed for several months; gall-stones being found in the feces. Subsequently he had several milder attacks. He remained pretty well till 1881, when his son died; after that time he was somewhat worse. In the summer of 1884, one very hot day, he probably had a slight sunstroke, and after that he was decidedly worse and steadily failed. His leg dragged in walking and his arm became almost useless. His mind became a little dull. He found great difficulty in expressing himself, and would work a whole day trying to frame a sentence before he could get it right and express his meaning. His articulation was distinct and he rarely showed any mental irritability.

Autopsy held forty-one hours after death. The skull-cap was rather abnormally thick and the dura mater, which presented a slightly yellowish tint at points but was otherwise normal, was excessively adherent to it. The sinuses and vessels of the pia mater contained only a small quantity of blood. The pia mater presented numerous patches of opacity following the course of the vessels, and over the whole surface of the left hemisphere there was an increased amount of sub-arachnoid fluid. This effusion was especially marked at a point a little posterior to and above the posterior termination of the fissure of Sylvius where it formed a large fluctuating bag which resembled a mass of jelly. There was a slight atrophy of all the convolutions of the left hemisphere, and beneath the point of greatest effusion mentioned above there was almost complete atrophy of the convolutions, which were represented by a thin gray fringe resembling a fold of pia mater. This atrophy involved the whole of the posterior central convolution, except a small portion of it near to the superior longitudinal fissure, the adjoining part of the superior

FIG. 3.



Outer aspect of left hemisphere. (Ecker.)

A, point of atrophy.

N. B.—It is to be remembered that these charts of Ecker are schematic. In this case, although the lesion is mapped out according to the convolutions affected, yet the lesion appears decidedly larger as represented on the chart than the actual lesion was on the surface of the brain.

parietal lobule, and the whole of the inferior parietal lobule, comprising the supramarginal convolution and almost the whole of the angular con-

volution. The fissure of Sylvius thus seemed to extend directly backward to the occipital lobe. The area of this atrophy corresponded with the area of distribution of the third (ascending parietal) and fourth (parieto-sphenoidal) branches of the middle cerebral or Sylvian artery. The white matter beneath the atrophied convolutions was neither softened nor atrophied.

The convolutions of the island of Reil, the left inferior frontal convolution, and the white matter immediately beneath it, were entirely normal, as were, also, the convolutions of the right hemisphere. The arteries at the base of the brain, and even some of the smaller cerebral arteries, presented an extreme degree of atheromatous degeneration. The posterior communicating arteries were obliterated and resembled threads. The ventricles of the brain were normal, and no abnormal appearance was found on sections through the cerebral substance and the ganglia at the base of the brain. No descending degeneration could be discovered on macroscopic examination. No microscopic examination of the nervous tissue was made.

The other lesions found were hypertrophy and fatty degeneration of heart, thickening of mitral and aortic valves, atheroma of aorta, old double adhesive pleuritis, firm adhesions of colon to under surface of liver enclosing the gall-bladder, which presented the cicatrix of a healed perforation into the colon, and a moderate degree of interstitial nephritis in both kidneys.

The atrophy of the convolutions in this case and the attendant symptoms were evidently due to an embolism or thrombosis of the terminal portion of the left middle cerebral artery, which occurred in the third week of the typhoid fever.¹

In this case the principal symptoms are impairment of motion and sensation in right arm and leg, alexia, agraphia, and aphasia; and the principal convolutions involved in the atrophy are the posterior central, except a small portion of it near the longitudinal fissure, the supramarginal convolution, and the greater part of the angular convolution on the left cerebral hemisphere. The lesion differs greatly from that in Case I. in that it is so extensive, and it is possible to explain the numerous symptoms which this extensive lesion produced only in the light of other cases of cerebral disease.

In the first place, the case is of interest in a negative way inasmuch as it shows that this extensive lesion caused no disturbance of sight or hearing. From Case II. we learned that the memories of things seen depended on the integrity of the convex surface of the left occipital lobe, and that the memories of spoken words depended on the integrity of the left superior temporal convolution. Case III. shows that these centres cannot encroach to any extent on the parietal lobe, as it is sometimes claimed that they do. It is, also, one of the already some-

¹ Cases of recovery from probable occlusion of the middle cerebral artery occurring in the course of typhoid fever have been reported by Vulpian in the *Revue de Médecine*, 1884, p. 162; and Kühn in *Deutsches Archiv f. klin. Med.*, Bd. 34, S. 56.

what numerous cases which show that the cortical centre for sight is not situated in the angular convolution, as Ferrier concluded it was from his experiments on apes.

The most striking and persistent symptom in this case is the inability of the patient to read or write, or the alexia and agraphia, as these disturbances are respectively called. Neither the alexia nor the agraphia was complete. The patient could read single letters and even short words with difficulty, but he could not spell the word with his eyes shut immediately after having read it. He could not store up the memory of the word that he had just seen, although he could store up the memory of the same word when he heard it spoken. He could not remember the appearance of words. Both the alexia and agraphia of the patient can be easily explained on the supposition that he had lost the memory of the appearance of words. When he saw a word in reading he had no already existing memory with which to compare it and he was unable, therefore, to recognize it. When he tried to write he had no memory of the appearance of the letter or word which he wished to write, and, hence, he was unable to reproduce it on paper; as he said in one of the only two letters which he wrote during his illness, "I cannot think how to make letters." The agraphia did not depend on the motor disturbance of the right hand, for in his letter of March 20 the letters were written legibly enough, and subsequently the awkwardness of his arm became much less.

When we consider the act of reading, and especially when we consider how a child first learns to read, it seems natural that the cortical centre for reading, or that portion of the cortex on the functional activity of which depends the memory of the appearance of written or printed words, should lie near the auditory centre and especially near the visual centre; and such is indeed the case. For in the cases of alexia and agraphia in which an autopsy has been made, the lesion has involved the angular convolution.¹ In this case, therefore, we must consider the cause of the alexia and agraphia to be the atrophy of the angular convolution of the left hemisphere, and since this atrophy did not involve the entire convolution, the alexia and agraphia were not complete although nearly so.

Another constant symptom in this case is a disturbance in the motility or an awkwardness of the right arm, and, to a less extent, of the right leg. The lesion corresponding to this disturbance must lie in the so-called motor zone, which is the portion of the cerebral cortex that has been studied with special care. As a result of extended experimental and clinical study, it may be considered established that the two central

¹ M. ALLEN STURT, AMERICAN JOURNAL OF THE MEDICAL SCIENCES, vol. 87, p. 389. A. B. BALL, Archives of Medicine, 1881, vol. 5, p. 136.

convolutions and the adjacent part of the cortex are the cortical centres for movements of the opposite side of the body. The centre for the leg includes the paracentral lobule, the upper third of the two central convolutions, and the greater part of the superior parietal lobule. The centre for the arm includes the posterior part of the superior frontal convolution, the middle third of the two central convolutions, and the anterior part of the inferior parietal lobule. The centre for the face includes the lower part of the central convolutions, especially the anterior one. These different centres are not sharply defined from each other; the one merges into the other; and it is rare to find a cortical lesion so situated and so small that it produces a paresis or paralysis of only one extremity or of only one-half of the face. In Case III. the lesion involves a large part of the centre for the arm, and a small part of the centre for the leg, and consequently there is much impairment of the motility of the arm and a slight impairment of that of the leg. There is no distinct statement concerning the face made in the history, although the lesion seems to involve a portion of the centre for the face.

When we examine the motor disturbance of the arm more closely, we find that it consisted in awkwardness rather than weakness, and that it was confined more particularly to the hand and fingers, or to that part of the arm with which those fine and delicate movements are executed which are the result of much education. Complicated movements which he had formerly executed easily, the patient executed with difficulty. When he tried to write he said: "I cannot think how to make the letters," and in the same way, when he tried to use his hand in other acts, he probably could not think how to make the movement. He had lost his skill in using his hand. He had forgotten the necessary feelings of muscular innervation; the memory of former innervation feelings was destroyed. In order to understand this clearly it must be remembered that one of the most important functions of the cerebral cortex is the reproduction of former perceptions in the form of memories. The cortex of the so-called motor centres is essential for the reproduction of memories of muscular innervation for movements which have previously been performed, and which can be performed again easily and readily only by the aid of the corresponding innervation memories. These memories of muscular innervation have been originally acquired, in part at least, through reflex acts.¹

The lesion in Case III. involved only the posterior half of the centre for the arm, and the symptoms produced were those of awkwardness rather than weakness. From a consideration of the reported cases of lesions of the motor centres, it seems that lesions situated in front of the

¹ Meynert, *Psychiatry*, transl. by Sachs, vol. 1, p. 160.

fissure of Rolando cause a more complete paralysis,¹ and are more often associated with descending degeneration, than lesions situated behind the fissure of Rolando, which produce rather a paresis or awkwardness.² It seems probable, then, that the nerve fibres for the arm and leg have their origin in that part of their respective centres which is situated in front of the fissure of Rolando, (in the case of the arm the fibres probably start from the base of the superior frontal convolution), while that portion of the centre situated behind the fissure of Rolando is essential to the production of the memories of innervation feelings.

The aphasia which was present in the early part of this case, but which later almost completely disappeared, was not due to any lesion of the left inferior frontal convolution, which is the portion of the cortex now generally regarded as essential to the production of memories of the innervation processes necessary to the pronunciation of words and hence of speech. This is one of the not very rare cases of aphasia due to a lesion which causes an interruption in the conduction along the association fibres which connect the left inferior frontal convolution either with the temporal lobe or with the angular convolution—*i. e.*, with the cortical centres for the memory of either spoken or written words. A number of cases of aphasia dependent on lesion of the supramarginal convolution or of the white matter just below it have been published.³ Since in this case the aphasia was temporary, it was probably due to some temporary cause, as, for instance, anæmia of the conducting fibres in the white matter beneath the supramarginal convolution, which anæmia gradually disappeared as the collateral circulation became more or less perfect. It is quite possible, too, that the aphasia was in part due to the loss of memory of the appearance of words. In the case of Cornil cited by Wernicke⁴ a small lesion of the supramarginal convolution caused not only aphasia, but also hemianæsthesia; so that it might be inferred that the impairment of sensibility noted by Dr. Clymer in Case III. was due to the lesion of the supramarginal convolution. Such an inference, however, is unnecessary, for it is now well known that lesion of the so-called motor zone causes loss of sensibility, although it is not yet settled whether the cortical centre for cutaneous and muscular sensibility is limited to the motor zone or whether it includes also the parietal lobe.

From the above considerations it is evident that although in Case III.

¹ Mickle, *Journal of Mental Sciences*, April, 1885; Hirschfelder, *Pacific Med. and Surg. Journ.*, Oct. 1881; Spitz, *Deutsche med. Wochenschr.*, April, 1882; Prévost and Cotard, *Obs. 15 in Études physiol. et pathol. sur le ramolis. Cérébrale*, Paris, 1886.

² J. Hughlings Jackson, *Brain*, Oct. 1882; H. B. Sands, *Med. News*, April, 1883; Wm. Carson, *Am. Practitioner*, vol. xv. p. 217; Vetter, *Deutsches Archiv f. klin. Med.*, Bd. 22, p. 421.

³ Broadbent, *Medico-Chirurgical Trans.*, vols. lv. and lxi; S. West, *Brit. Med. Journ.*, 1885, p. 1242; Cornil, *Gaz. Méd.*, 1864, p. 534.

⁴ *Lehrbuch der Gehirnkrankheiten*, vol. ii. p. 176.

the lesion is very extensive, yet it confirms the following deductions which can be drawn from reported cases in which the lesion was less extensive:

1st. In the anterior part of the cortical centre for the arm originate the nerve fibres for the arm, and lesions of this part cause absolute paralysis.

2d. In the posterior part of the cortical centre for the arm take place those cellular actions which are essential to the production of memories of muscular innervation, and lesions of this part cause awkward, uneducated movements.

3d. The cortical centre for muscular and cutaneous sensibility extends over the motor zone and probably over the parietal lobe also.

4th. The left angular convolution is essential for the memory of the appearance of words and lesions of it cause alexia and agraphia.

The subject of aphasia will be considered later.

In Case III. the lesion affected principally the cortical centre for the arm, while in the following case the cortical centre for the leg was alone affected.

CASE IV. *Left hemiparesis. Convulsions limited to the left side of the body always commencing in the left leg. Endothelionia pressing on right superior parietal lobule.*

M. C., female, æt. forty-two, married, a servant, entered St. Peters Hospital June 7, 1883. Family history unknown. Patient has never been very strong, but has worked hard. Four and a half years ago she slipped and fell on the sidewalk, striking on the back of her head. About three and a half years ago she had the first of a series of attacks of which she has had about a half dozen in all. These fully developed attacks commence with a tingling and twitching of the left foot which gradually extend up the leg and then commence in the left arm; next the tingling sensation creeps up the back to the left side of the head, and the head commences to twitch; after the head has continued twitching for from ten to fifteen minutes and while the whole left side of the body is twitching she loses consciousness and remains unconscious for a quarter of an hour or longer; while unconscious she frequently has involuntary micturition. The right side of the body has never taken part in these convulsions. She has never bitten her tongue, and she has never had a convulsion which did not commence in the left foot. Besides these half dozen fully developed attacks she has had more frequently attacks in which she feels a pain in the left heel, the left great toe works up and down for a long time, and sometimes the muscles of the leg twitch, but the twitching goes no further and the attack passes off. Patient also complains that the left arm and leg are much weaker than the right and that she has much darting pain in the left leg, arm, face, and left side of head. She often feels as if a strap were drawn firmly about portions of the left arm and leg, and at times it feels as if her toes had fallen off; while it feels at other times as if her teeth had fallen out. These latter sensations are most marked immediately after an attack of convulsions. She says that her eyes are so weak that

she cannot read and that she has flashes of light before her eyes which make her dizzy. She complains of fulness and pain in vertex of head, and thinks that at times she is not in her right mind.

On entrance into the hospital patient seems weak and anæmic. An examination of the thorax and abdomen reveals nothing abnormal. Sensibility intact over both sides of face, both arms, and right leg. Sensibility in left foot and leg is somewhat blunted to tactile impressions but not to painful ones. Left leg is much weaker than right, so that she is unable to walk without a cane. Left arm is weaker than right. Grasp of right hand 50, of left 40, as registered on the outer circle of Mathieu dynamometer. Ankle clonus and exaggerated knee-jerk on both sides, especially on the left. No exaggeration of tendon reflexes in arms. Pupils equal and react to light. An ophthalmoscopic examination by Dr. Merrill reveals a typical specimen of choked disk. In both eyes the veins are tortuous, the disk reddened and œdematous. In the left eye there is rather less œdema, but there is a patch of exudation in the disk and in the adjoining part of the retina. No tenderness on percussion over any part of the skull.

December 1, 1883. Since entrance into the hospital, the condition of the patient has not changed materially. She had ten fully developed unilateral convulsions between time of entrance and August 21, 1883, since which time she has had no convulsion, although she has had some abortive attacks. The unilateral convulsions which she had in the hospital have all commenced in the left foot and gradually extend up over the whole left side of the body, the right never being affected. The convulsive movements are clonic contractions of the muscles of left leg, left arm, left side of neck, left side of face, left side of tongue, and left eyelid and eyeball. Consciousness persists during the earlier part of the attack, but soon becomes more or less completely lost. Each attack can be promptly arrested by the inhalation of ether, and when so arrested does not return when the ether is withdrawn. After these attacks it has been noticed that the left side of the body is decidedly weaker than before and the strength returns slowly. Besides these attacks she has attacks of convulsions limited to the left leg. In addition to these convulsive attacks she has numerous attacks of a curious nature in which all mental action seems to be abolished. She will sit as if in a trance, perfectly motionless, unable to answer any question or to do anything that she is told, for a space varying from a few minutes to an hour or more, when she will suddenly come to herself and be entirely natural again. When she comes to herself she sometimes has no memory of anything that has happened, while at other times she has a pretty clear idea of all that took place in the room while she was in the fit. At other times, while in such a fit and incapable of answering questions, etc., she keeps repeating over and over the phrase "Lord, have mercy on me!" and in all her fits she never uses any other phrase than "Lord, have mercy on me!" She has had much pain at times in left side, especially in left side of skull, and has had several severe attacks of nausea and vomiting.

August 1, 1884. Has been more comfortable during the past eight months. Has not had more than half a dozen unilateral convulsions, and has had less pain in left side of head. Feels a little stronger and can walk a little better with the aid of a cane. No decided change is found on physical examination except that there is now at times decided

tenderness on percussing the skull over the upper posterior part of the right parietal bone, and that there is slight but evident left facial paresis which is especially well marked after a convulsive attack. The remedies which seem to benefit her the most are small doses of bromide of potassium or of chloral.

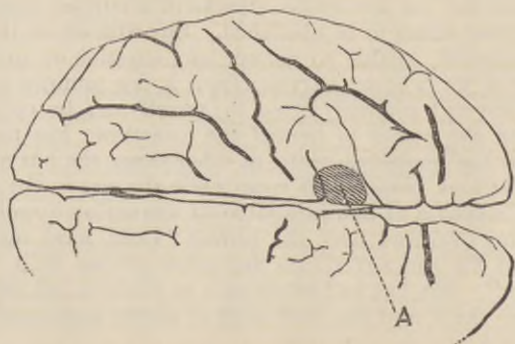
October 30. Slowly failing during past few days. To-day lies in a semi-comatose state, with the head, but not the eyes, turned toward the right. She answers questions in monosyllables. Tongue protruded fairly straight. She cannot move either left arm or leg, which are flexed and moderately rigid.

November 1. This evening the pupil of the right eye was contracted and did not respond to light, while the pupil of the left eye was dilated and did respond to light. Temperature $106\frac{1}{2}^{\circ}$ F. All these symptoms disappeared in a few hours and did not recur.

3d. Entirely comatose. Left side of body completely relaxed. Some resistance offered to passive motion on right side. Eyes examined by Dr. Merrill, with the following results: Outlines of nerves are indistinct, arteries small; both arteries and veins are very tortuous. Retinæ in the neighborhood of the nerves and extending well out toward the periphery are œdematous, and present an unusually opaque appearance. The appearance of both eyes is much alike, except that in the left eye the arteries and veins are much smaller and more tortuous.

4th. Patient died to-day. *Autopsy* held five hours after death. Skull cap unusually dense and not symmetrical, being unusually hollowed out at a point near the right parietal eminence. Dura mater not so adherent to skull cap as usual; inner surface of dura mater normal. Pia mater normal except for much congestion of both the larger and smaller veins. Base of brain normal. Cerebral convolutions are generally flattened. Lying close against the superior longitudinal sinus and just posterior to the upper extremity of the posterior central convolution on the right side is a hard ovoid tumor, measuring one and one-half inches in its longer, and one and one-quarter inches in its shorter

FIG. 4.



Superior surface of right hemisphere, after Ecker.

A, depression caused by tumor.

diameter. The tumor pressed on the cerebral substance, causing a rather deep concavity in the most anterior part of the right superior parietal

lobule on the very edge of the superior longitudinal fissure. The tumor is firmly adherent to the dura mater and around its periphery the dura mater, the pia mater, and the cerebral cortex are all firmly adherent to the tumor and to each other. Just beneath the tumor the cerebral substance is slightly softened. The left lateral ventricle is dilated in all its parts, while the right lateral ventricle is rather smaller than normal, and its posterior horn is almost completely obliterated. Sections through the brain and spinal cord show no other lesions.

On microscopical examination the tumor was found to be an endothelioma. It presented a well-marked alveolar structure, and the cells were flat and had a concentric arrangement. No descending degeneration could be found either in the spinal cord or in the pyramidal tract at the base of the brain.

In the first three cases the lesion was a cerebral atrophy which simply put certain portions of the brain out of action. In Case IV., however, the lesion is a tumor which, in its growth, pressed upon and irritated the cerebral cortex and the cerebral meninges, and consequently in this case we get for the first time symptoms of cerebral irritation—*i. e.*, nausea and vomiting, and pain and convulsions limited to the opposite side of the body. In this case the tumor never actually destroyed the cerebral cortex, and, therefore, the symptoms of irritation were never completely replaced by those of paralysis, although toward the end of the patient's life the convulsions became rarer and the hemiparesis became more decided.

The principal symptoms in the case were the pain, the convulsive attacks, and the attacks in which the patient was unable to answer questions or to perform any other voluntary action. It has long been known that the nearer a tumor is to the surface of the cerebral hemispheres the greater is the pain that it causes, and this tumor growing in the meninges caused paroxysmal attacks of intense pain throughout the left side of the body, and was especially constant and severe over the whole left side of the head. The distribution of the pain is difficult of explanation. The most reasonable supposition is that it was due to irritation of the cortical termination of the sensory nerves of the left side of the body, especially of the fifth nerve; and yet, according to our present views of cerebral localization, the fibres from the fifth nerve terminate in the lower rather than in the upper part of the central convolutions. It was only toward the end of life that there were pain and tenderness over the seat of the tumor.

Unilateral convulsions have been very frequently noticed in cases of tumors and other irritative lesions of the cerebral cortex. Such convulsions are sometimes called cortical epilepsy or Jacksonian epilepsy, after J. Hughlings Jackson, who has long given them especial attention and study, and who insists on the importance of the temporary paresis following the convulsion and limited to the muscles taking part in the

convulsion, which temporary paresis was well marked in Case IV. It is customary in these cases for the convulsion to commence in that part of the body which is connected with that portion of the cortex in which the lesion is situated, and, accordingly, in this case the convulsive movements always began in the left foot; the tumor lying in the cortical centre for the left leg. In many cases the convulsion, although commencing on one side, soon becomes general and is associated with loss of consciousness, thus closely simulating epilepsy. It is such cases which have given much support to the theory of the cortical origin of epilepsy, which has now replaced the vasomotor theory of epilepsy; although true epilepsy is entirely distinct from such epileptiform convulsions dependent on gross disease of the brain. In Case IV. the convulsions were never general, and the loss of consciousness was often incomplete.

Besides the unilateral convulsions, the patient had another form of attack which is very interesting, and which has never to my knowledge been noticed in cases of cerebral tumor. In the attacks of this kind consciousness, although obscured, was rarely or never completely lost. The patient was in a kind of trance; she knew what was going on about her and tried to answer questions and to do what she was told, but there seemed to be an inhibition put upon all action. The most she could do in the milder attacks was to say "Lord, have mercy on me." These attacks in many respects resembled the attacks of petit mal of epileptics. We know that from the motor area of the cortex two kinds of impulses originate and pass down along the pyramidal tract to the sub-cortical centres. One kind of these impulses sets the sub-cortical centres in activity and causes movements; the other kind stops the activity of the sub-cortical centres and inhibits movement. Destructive lesions, either of the motor area in the cortex or of the pyramidal tract, destroy voluntary movements, and, at the same time, by destroying inhibition, increase reflex movements. In Case IV. it seems that the irritation of the tumor caused at one time a discharge of motor impulses which set the sub-cortical centres into activity and produced the convulsive movements, and at another time caused a discharge of inhibitory impulses which repressed all activity of the sub-cortical centres and prevented movement. It seems then as though the two contradictory forms of activity which this portion of the cortex manifests in health, and which cause respectively movement and inhibition, were both reproduced in an abnormal discharging manner by the irritation of the growing tumor.

Besides these symptoms, there was in this case a left hemiparesis which was always more decided immediately after a convulsion, and which was due in part, perhaps, to the left-sided convulsions, but certainly in greater part to the direct pressure of the tumor upon the cortex. Although the action of the tumor was confined to the cortical centre for the leg, yet the paresis was not confined to the leg, but affected

also the arm and face to a less degree. This would indicate that in each cortical motor centre the face, arm, and leg are all represented to a varying degree, and that there is no sharp line to be drawn between the centres. This and many other cases can be better explained in this way than by the supposition of transmitted pressure. Certainly it is very rare to find a cortical lesion so small that it causes disturbance in only one member.

In all the cases so far reported in this article the lesion has been confined to the cortex of the posterior part of the brain and has in no instance extended in front of the fissure of Rolando. I have not had any case in which the cortex of the frontal lobe was involved, but I will report briefly three cases in which the lesion involved the white matter of the frontal lobe.

CASE V. *No symptoms, coma; tumor of frontal lobe.*—W. Mc. P., male, æt. forty-four, widower. Always healthy. Infected with syphilis eight years ago. During the past year and a half he has been troubled by a varicose vein in his leg which has ruptured four times, causing a great loss of blood each time. Otherwise he has felt well except that he has felt weak of late, on which account he entered the hospital. On entrance he exhibited anæmia and general weakness, which were attributed to the excessive loss of blood. No paralysis of motion or sensation and no aphasia could be discovered. Eyes were not examined. Four days later he became comatose and the next day he died. At the autopsy a tumor as large as a small hen's egg was found in the lower part of the right frontal lobe near its middle, surrounded by a narrow zone of yellow softening. Tumor was dense, mahogany-colored on section, subdivided by indistinct septa and contained several distinct hemorrhages.

CASE VI. *Convulsions, aphasia, somnolence, coma, death; abscess just anterior to left corpus striatum.*—H. T., male, æt. forty-three, married. Enjoyed good health until one day when, without any warning, he had a severe convulsion followed by unconsciousness for about an hour, when he had a second convulsion which was also followed by unconsciousness for an hour, after which he recognized persons about him and appeared well. During the next fortnight he went out, but seemed averse to bodily or mental exertion. He grew more and more listless and dispirited. He complained of paroxysmal pain in his left eye and darting through his temples. There was no paralysis of any sort, but there was a slight degree of aphasia. His pulse did not vary much from 80. He steadily grew more and more drowsy, became comatose, and died just one month after his first convulsion. At the autopsy two encapsulated abscesses, each the size of a small hen's egg, and communicating with each other, were found a little in front of the left corpus striatum; the top of the upper abscess being on a level with the floor of the lateral ventricle. The cerebral substance in the immediate neighborhood of the abscess was the seat of a softening which involved the anterior part of the corpus striatum and the convolutions of the island of Reil.

CASE VII. *Convulsions, aphasia, paralysis of leg, somnolence, death; tumor involving the whole of the medullary substance of the left frontal*

lobe.—M. D., female, æt. thirty-nine, married, always enjoyed good health till one day when, without known cause, she was seized with three epileptoid convulsions in succession, after which she remained comatose for a few hours, and then seemed to be perfectly well again. These attacks occurred on the same day that her menstrual discharge ceased. One month later, on the last day of her menstruation, she had another set of altogether similar convulsions, and still again, one month later, the attacks recurred. Just before the termination of the next menstruation twenty leeches were applied to the inside of the thighs, and a brisk cathartic was administered with the result that the convulsions did not appear till two weeks later. After this they continued to appear at irregular intervals which were seldom less than four weeks. Except for the convulsions she continued in fair health for five months, when she began to complain of paroxysmal pain in both temples, especially in the left one, and became listless and dispirited, and had also severe attacks of vomiting. Three months later the right leg became so weak that she could not walk, and her eyesight began to fail. Eyes were not examined ophthalmoscopically. She became more and more listless and developed well-marked aphasia. Ten months after her first convulsion she had a severe convulsion, after which she remained unconscious, and vomited constantly. The right side remained nearly motionless, while the left arm and leg were in continual motion. On the morning of the next day she died. At the autopsy a gelatinous tumor was found occupying the place of the whole of the medullary substance of the left anterior lobe. The tumor cut tough and gave the sensation as though the scalpel was drawn over wool. It reached to within half an inch of the surface of the hemisphere. The cerebral substance around it was softened; the corpus striatum being involved in the softening.

Case V. shows that a large tumor may exist in the frontal lobe without giving rise to any symptom until it causes coma and death. Numerous cases are on record which prove the same thing, and it is now generally accepted that lesions of the frontal lobe may cause no characteristic symptoms (except perhaps an ill-defined change in the character and disposition of the patient) unless they are so far back that they involve the motor centres, or unless they involve the posterior part of the left inferior frontal convolution, in which case they produce aphasia. It is well known that in the left inferior frontal convolution take place those cellular actions which are essential to the production of the innervation feelings necessary for speech, and lesions of this convolution produce motor aphasia—*i. e.*, the more or less complete inability to speak, just as lesions of the left superior temporal convolution causes sensory aphasia—*i. e.*, the inability to understand spoken words, and hence irrelevant speech. In Case VI. the softening about the abscess involved either the left inferior frontal convolution or the fibres coming from it, and produced a slight degree of aphasia; while the extensive lesion in Case VII. involved not only the fibres from the inferior frontal convolution but also some of those from the central convolutions, and produced not only well-marked aphasia but also paralysis of the right leg.

Cases VI. and VII. are good examples of general convulsions depend-

ing on tumors of the anterior lobes, just as Case IV. was a good example of localized convulsions dependent on a tumor located in the motor region of the cortex. The fact that the cessation of menstruation acted as an exciting cause for the convulsion in Case VII. and that a free bleeding at that time delayed the appearance of the convulsion is very interesting.

From this study of cerebral localization, the following conclusions may be drawn :

1st. The greater part of the cerebral cortex can be divided into small areas, each of which is functionally associated with a definite mode of mental action, and is consequently called the cortical centre for that action.

2d. The cortical centres connected with the sensory nerves are situated in the posterior half of the cerebral cortex including the temporal lobe; and the cortical centres connected with the motor nerves are situated in the middle portion of the cerebral cortex and in the posterior part of the cortex of the anterior lobe.

3d. Each sensory cortical centre probably consists of two parts; a smaller one in which the peripheral nerve has its final termination and in which take place those molecular changes which correspond to simple sensation; and a larger one in which take place those molecular changes which correspond to the mental processes of memory, judgment, and comparison which together constitute complete perception and recognition.

4th. Each motor cortical centre probably consists of two parts: a smaller one in which the peripheral motor nerve has its origin, and in which take place those molecular changes which correspond to the action of the will in originating voluntary movements; and a larger part in which take place those molecular changes which correspond to the memories of coördinated muscular innervation which are factors in the production of voluntary movements.

5th. The optic fibres from the right upper quadrant of each retina terminate in the lower half of the right cuneus. (See Case I.)

6th. The optic fibres from the right lower quadrant of each retina terminate in the adjacent part of the right median occipito-temporal convolution. (See remarks on Case I.)

7th. The lower half of the cuneus and the adjacent part of the median occipito-temporal convolution is the point of termination of the optic fibres from homonymous halves of the retinae: the right half of each retina being represented in the right occipital lobe and the left half in the left lobe. (See remarks on Case I.)

8th. Functional activity of the cortex of the median surface of the occipital lobe is necessary for simple visual sensation. (See remarks on Case II.)

9th. Functional activity of the cortex of the convex surface of the left

occipital lobe is necessary for full visual perception and recognition and for the production of visual memories. (See remarks on Case II.)

10th. The temporal lobe is the cortical centre for hearing and complete destruction of a temporal lobe or of the auditory fibres running to it causes complete deafness of the opposite ear. (See remarks on Case II.)

11th. Functional activity of the cortex of the left superior temporal convolution is necessary for the perception and recognition of spoken words and for the production of the memory of these words; lesions of this part causing inability to understand spoken words and sensory aphasia. (See remarks on Case II.)

12th. Functional activity of the cortex of the left angular convolution is necessary for the production of memories of the appearance of written or printed words; lesions of it causing alexia and agraphia. (See Case III.)

13th. Only in virtue of the fact that on its functional activity depends the production of the memories of the appearance of written or printed words can the angular convolution be considered as forming part of the visual centre. It does not constitute the visual centre, as Ferrier claims. (See remarks on Case III.)

14th. The cortical centre for the leg includes the paracentral lobule, the upper third of the two central convolutions, and the greater part of the superior parietal lobule. (See Case IV. and remarks on Case III.)

15th. The cortical centre for the arm includes the posterior part of the superior frontal convolution, the middle third of the two central convolutions, and the anterior part of the inferior parietal lobule. (See Case III. and the remarks on it.)

16th. The cortical centre for the face includes the lower third of the two central convolutions, especially the anterior one. (See remarks on Case III.)

17th. In the anterior part of the cortical centre for the arm originate the nerve fibres for the arm, and lesions of this part cause absolute paralysis of the arm. The same thing is probably true in the case of the cortical centres for the leg and face. (See remarks on Case III.)

18th. In the posterior part of the cortical centre for the arm take place those molecular changes which are necessary for the production of memories of coördinated muscular innervation. The same thing is probably true in the case of the cortical centres for the leg and face. (See Case III. and the remarks on it.)

19th. No sharp line can be drawn between the motor centres of the leg, arm, and face of the same side, and it is very possible that in each centre all three parts may be more or less completely represented. (See remarks on Case IV.)

20th. The cortical centres for muscular and cutaneous sensibility are

the same as those for motility, and probably extend backward beyond the latter over the parietal lobe also. (See remarks on Case III.)

21st. The faculty of speech cannot be located in any one portion of the cortex, and aphasia can be produced by a lesion situated in various parts of the left cerebral hemisphere; the right hemisphere apparently not being concerned in the production of speech except in the case of left-handed persons. The memories of the muscular innervation feelings necessary to produce spoken words depend on the functional activity of the cortex of the left inferior frontal convolution. The memories of the sound of spoken words depend on the functional activity of the cortex of the left superior temporal convolution. The memories of the appearance of written or printed words depend on the functional activity of the left angular convolution. These centres are all connected together by means of association fibres. The faculty of speech in its completeness depends on the integrity of all these parts, except, perhaps, that of the angular convolution. According as one or the other of these parts is affected, the symptoms of one or the other of the principal varieties of aphasia are produced. Motor aphasia is due to a lesion of the left inferior frontal convolution or of the white matter immediately beneath it. (See Cases VI. and VII.) Sensory aphasia is due to a lesion of the left superior temporal convolution (see Case II.), perhaps also to a lesion of the left angular convolution (see Case III.). Conduction aphasia is due to a lesion of the association fibres mentioned above. (See remarks on Case III.)

22d. Tumors or other irritative lesions situated in the non-motor region of the cerebral hemispheres can cause general convulsions associated with loss of consciousness. (See Cases VI. and VII.)

23d. Tumors or other irritative lesions situated in the cortical centres for the leg, arm, or face, may cause convulsions commencing in the leg, arm, or face, respectively, and which may become general, though they more frequently remain unilateral, and which are sometimes associated with loss of consciousness and sometimes not. (See Case IV.)

24th. Tumors situated in the cortical centre for the leg may produce a trance-like condition, or conditions resembling the attacks of petit mal of epileptics. (See Case IV.)

