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A CONTRIBUTION TO THE PATHOLOGY OF HEMIANOPSIA OF CENTRAL ORIGIN (CORTEX-HEMIANOPSIA)

BY

E. C. SEGUIN, M.D.

CLINICAL PROFESSOR OF DISEASES OF THE MIND AND NERVOUS SYSTEM IN THE COLLEGE OF PHYSICIANS AND SURGEONS, NEW YORK; CORRESPONDING MEMBER OF THE VEREIN FÜR INNERE MEDICIN OF BERLIN, AND OF THE SOCIETÉ DE BIOLOGIE OF PARIS, ETC.

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A CONTRIBUTION TO THE PATHOLOGY OF HEMIANOPSIA OF CENTRAL ORIGIN (CORTEX-HEMIANOPSIA).

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THE importance of hemianopsia with reference to the recently developed doctrine of the localization of functions in the cerebral cortex is so great as to merit the closest study on the part of the physiologist and the practical neurologist. Few subjects of such apparently small intrinsic importance have attained to such a magnitude as this one, rendering quite impossible to treat of it fully in a paper for an ordinary Society meeting. Only one or two of its phases can be considered within the time allotted me, and I shall therefore limit my remarks to the relation of the symptom hemianopsia to certain central or cerebral lesions, and endeavor to show its value for purposes of diagnosis in actual practice, rather than develop its significance for the solution of physio-psychical problems.

My apology for presenting the subject is that during the past winter it was my fortune to observe a typical case of lateral hemianopsia, stationary till the patient's death many months afterward, and almost unaccompanied by other cerebral symptoms. The topographical diagnosis made during life was verified by the post-mortem examination, so that,

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apart from its scientific interest, I may venture to submit the case as an encouragement to the making of positive diagnoses *intra vitam*, in the light of the rapidly growing laws of so-called cerebral localization.

Before relating the case and presenting the specimen I should make a few remarks upon the scope of the paper, and briefly state certain data relative to parts of the subject which I cannot treat in full.

First, then, as to the scope and plan of the paper. I shall consider only the recorded cases of hemianopsia in which the autopsy revealed a lesion in some part of the brain inclusive of the optic thalami. Since the publication of Dr. Starr's valuable *résumé* of cases of hemianopsia in January, 1884, their number has somewhat increased, and I am able to tabulate forty. I should add that I have endeavored to obtain the original essay in each case, and have carefully prepared the abstracts myself: only one, No. 0, by Prévost of Geneva, has been impossible to obtain, and I quote it upon Westphal's authority, but exclude it from my tables. This labor I was induced to perform in order to avoid errors which otherwise easily occur in quoting cases, and also to be able to group these cases and more fully appreciate and present their pathological and diagnostic value. It is far from me to claim that my collection is absolutely perfect, but it is, I believe, almost complete and reliable in its critical arrangement. Let me repeat that I am anxious to present this relatively very rich and singularly harmonious collection of cases in such a way that it shall prove of most use to the practising physician for diagnostic purposes.

Second, as to the subject of hemianopsia in general.

The fact that a person might temporarily or permanently see only one half of objects placed directly in front of him has been known to physicians more than one hundred years. In 1723 Vater and Heinecke described three cases under the name of *visus dimidiatus*.

The same phenomenon was designated as hemiopia at the close of the last century, probably first by A. G. Richter, a term which prevailed and is still employed though in a dif-

ferent sense since the introduction of hemianopia by F. Monoyer in 1865, and of hemianopsia by J. Hirschberg in 1877. The latter is the preferable and preferred form.

As the terms are now accepted, hemiopia signifies loss of perceptive power in one lateral (or vertical) half of the retina, while hemianopsia means obscuration of one lateral (or vertical) half of the visual field. As rays of light cross within the eye before reaching the retina, it follows that, for example, right hemiopia is equivalent to left hemianopsia; or, in other words, that nasal hemiopia corresponds to and causes temporal hemianopsia.

In describing cases at the present time, it is customary and preferable to omit all mention of the retinal condition, or hemiopia, and to describe the hemianopsia, or the state of the visual field as determined by the perimeter or by ruder though sufficient tests.

Several varieties of hemianopsia are recognized.

1. *Horizontal*, superior, or inferior hemianopsia, almost always due to defects within the eye, and of relatively small interest to the neurologist.

2. *Vertical* hemianopsia, almost always due to lesions of the retro-ocular nervous visual tract, and hence of great importance in neuro-pathology. Quite a number of terms have been employed to designate the varieties of vertical hemianopsia. Of these we recognize and adopt:

(*a*) Temporal hemianopsia.

(*b*) Nasal hemianopsia.

(*c*) Lateral hemianopsia, often designated as homonymous hemianopsia.

The first two varieties are exclusively caused, as far as our present knowledge goes, by lesion of the optic chiasm, of its lateral, or of its frontal or caudal borders.

The last variety, lateral hemianopsia, is always produced, as far as our present knowledge goes, by lesions of one optic tract, or of the more central parts of the optic apparatus as far caudad as the cortical centre for vision in one hemisphere.

The object of this paper is to study the recorded cases of lateral hemianopsia, with autopsies, due to lesions situated

in the more caudal parts of the optic apparatus, its central portions, from the primary optic centres (lobi optici, corpora geniculata lateralia) to the cortical visual centres, of areas.

With reference to all three forms of hemianopsia, I shall assume the following propositions as established :

1. The fact of a semi-decussation in the optic chiasm or man has been proven chiefly by the researches of von Gudden. According to these recent views (which are in part a return to the ancient theoretical statements of Newton, Wollaston, Müller, Hannover, and von Graefe) the optic fasciculi are disposed as follows : The fibres of each tractus opticus at the chiasm divide into two parts : A larger one which decussates with its homologue and enters into the composition of the opposite optic nerve, supplying the nasal half of the retina. This is the fasciculus cruciatus. The other, smaller set of optic-tract fibres does not decussate but passes on directly to form a part of the optic nerve of the same side, supplying the temporal half of the retina. This is the fasciculus lateralis. Thus each retina receives nerve fibres from both optic tracts, or, in other words, each optic tract contains fibres destined for both retinæ.

The inter-retinal fasciculus of Hannover is purely imaginary ; there are no such fibres. The posterior loop of Hannover is now known, since the experiments of von Gudden, to be composed of non-optic fibres ; it is the inferior cerebral commissure.

2. The connection of the optic tracts with the corpora geniculata lateralia and the lobi optici (anterior corpora quadrigemina) is an intimate one, but probably (in man) more for trophic and reflex purposes than for vision. Whether mere perception of light (as an excitant) may take place in these bodies after removal of the hemispheres, is still an open question. Certainly sight, in the ordinary meaning of the term, is impossible under such conditions.

3. A total lesion of one tractus opticus fatally produces lateral hemianopsia of the fields opposite the lesion.

4. A lesion acting upon one side of a tractus opticus so as

to compress only some of its fibres will produce one-sided nasal hemianopsia.

5. A lesion acting simultaneously on the sides of the optic chiasm will, by injuring both fasciculi laterales, produce nasal hemianopsia in both eyes.

6. A lesion compressing the optic chiasm in its frontal or caudal borders will produce bilateral temporal hemianopsia by injury to both fasciculi cruciati.

7. All such lesions are apt to be accompanied by pupillary irregularity or immobility, by optic neuritis or atrophy; and their diagnosis is further facilitated by finding signs of paralysis of other basal nerves, or of crossed hemiplegia.

8. It must be borne in mind that a lesion of the hemisphere may be so situated as to press downward upon one tractus opticus, and thus produce hemianopsia of the peripheral type (see case of Hirschberg, No. 5).

9. Lesions of the lobi optici in man have been rarely observed, and when observed have been bilateral in their effects, so that nothing can be said at present of hemianopsia due to disease of these parts.

With this brief introduction I now pass on to the consideration of the clinical and pathological aspects of the subject of my paper as exemplified in forty cases with autopsies, and five traumatic cases without autopsies, which I have been able to collect.

After a careful analysis I have grouped these forty-five cases into six categories.

1. Cases which are indefinite or useless for the study of localization, four in number.

2. Cases of lesions of parts which we have good reason to believe unconnected with the central optic apparatus, and which produced hemianopsia by pressing upon the optic tracts or the chiasm, three in number.

3. Cases in which the hemianopsia was due to a lesion of the corpus geniculatum laterale or the thalamus opticus, or both, six in number.

4. Cases of hemianopsia due to lesion of the white substance of the occipital lobe, eleven in number.

5. Cases of traumatic hemianopsia, due to injuries of the

occipital region of the skull and lesion of the subjacent brain, five in number.

6. Cases of hemianopsia due to lesions of the cortex of the brain, cortex only, or also of the subjacent white substance, sixteen in number. In this class I have included my own case. Among these sixteen cases there are four (Nos. 28, 29, 41, 45) in which the lesion was circumscribed, and where it occupied so nearly the same spot in the cortex cerebri as to afford us, in my opinion, a solution of the problem of the location of the cortical visual centre in man.

In order to shorten this essay for publication I have tabulated the cases according to the above grouping of the cases.

The four conclusive cases I shall, however, offer in full abstract illustrated with diagrams in order to enable the reader to more fully appreciate their value. But first I shall give the details of a traumatic case which is of extreme interest, from the facts that hemianopsia has existed as the sole symptom for twenty-three years, and that the cicatrix in the head is so distinct as to allow of study at present.

Case 3.—Keen and Thomson. P. H., a soldier, aged twenty-three, was wounded in the head by a minié ball, during the battle of Antietam, in September, 1862. The missile entered the skull in the median line, 1¼ inches above the external occipital protuberance, and made its exit at a point 2 inches distant from the median line, and 3 inches distant from the point of entrance. There was no immediate loss of consciousness. In the next few days the patient complained of impaired vision. Ten days after the injury, loss of consciousness, with right-sided hemiplegia, occurred. Paralysis and imperfect memory lasted for two or three months. Apparently no aphasia.

When seen by the authors, in 1870, there was no paralysis, and the mental functions were unimpaired. The patient complained that the vision of his right eye was deficient. The pupils, ocular muscles, and fundus were normal. The left cornea bore an old opacity. Central vision on right side, = 1 ; on left side, ⅝. The fault complained of by the patient was found to be a complete right lateral hemianopsia, with a vertical division line.

Recently I traced this soldier, through the Pension Bureau at Washington and the office at Philadelphia, to his home in that city. He has consented to come here this evening, in order to make the report more exact. By the courtesy of Drs. Keen

TABLE I.

Cases of Hemianopsia in which the Relation between the Lesion and the Optic Defect was not Evident; Indefinite and Insufficiently Reported Cases.—Four in Number.

No.	Observer.	Sex.	Age.	Fundus and Pupils.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
12	Charcot and Pitres, 1877.	F.	52	(?)	Left lateral H. (De Wecker and Landolt.)	(?)	Localized epileptiform attacks in l. face, neck, and arm. Left hemiplegia with flaccid muscles.	Various old patches of softening in different gyri. Recent red softening of cerebellum.	State of occipital lobes, optic tracts, and chiasm not mentioned.
21	Linnell, 1881.	M.	63	At first exam. fundus normal; at second, right side of left disk pale. V. = $\frac{20}{30}$ or $\frac{20}{18}$.	Left lateral H.	Sclerosis (?) of right optic tract. Softening of corp. quad. et genic. (?)	Attacks of neuralgia in head and limbs for 8-9 years. For 4 years "paralysis agitans" right side, with numbness and tingling. Sudden blindness after severe headache: improvement in V. Blindness again, general tremor, halluc. of V. Death.	Large recent clot in right hemisphere ant. to its centre, and wholly in white substance. Left hemisphere normal. Tubercula quad. in state of white softening, as also the corp. genic. and ventral part of thalamus, more on left side. Right optic tract firmer than left, chiasm and nerves normal.	
25	Petrina, 1881.	M.	53	Normal fundus and papillæ. V. not measured: no mention of refraction or accommodation, or pupils.	No hemianopsia. Amblyopia of left eye: after using this eye alone a few minutes every thing becomes confused and gray.	Fissure in right lambdoid suture. Pachymeningitis, meningeal hemorrhage, and softening or degeneration of cortex of occipital gyri, especially the 2d and 3d, from sulcus temporalis sup. anteriorly to fissura calcarina behind. (?)	Fall backward on occiput with loss of C.; vomiting, headache, and vertigo. Failing sight. No motor or sensory symptoms. All special senses normal except sight. Died of pneumonia.		Apparently no oculist saw the case. If report is correct there was probably paralysis of accommodation in l. eye. The lesion may not have involved the cuneus.
40	Wiethe, 1884.	M.	54	Fundus normal. Pupils and V. not mentioned. Binocular V. preserved.	Superior lateral hemianopsia.	Lesion of left thalamus opticus. (?)	Fall upon occiput; unconscious, and bleeding from nose, mouth, and ears. Violent headaches, impaired mental action; no paralysis. In a few weeks apoplectic attack with left hemiplegia: recovery. Later, complete (?) blindness developed in 1½ hours: partial recovery. Death from hernia.	Atheromatous cerebral arteries. Old hemorrhagic foci in r. temporal lobe, lentiform nucleus, medullary subst. of frontal lobe and gyrus olfact. Also in left parietal lobe and left thalamus. Chronic pachymeningitis.	Extent of lesions not well given. No special mention of occip. lobe and optic tracts.

TABLE II.

Cases of Hemianopsia from Lesions Unrelated to the Cortical Centre for Sight; Cases from Pressure Transmitted to Optic Tracts, etc.—Three Cases.

No.	Observer.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
5	Hirschberg, 1875.	M.	40	Fundus normal. Central vision normal.	Right lateral H. with sharply defined vertical line passing close to point of fixation.	Left tractus opticus smaller than right.	For four years severe left-sided, intermittent headache. Imperfect V. to right. Right hemiplegia. Aphasia.	Glio-sarcoma, size of an apple, in left frontal lobe.	Tumor may have pressed on tractus. State of occipital lobe not mentioned.
8	Huguenin, 1876.	F.	46	(?)	Right lateral H. with a not well defined vertical line.	(?)	Attack of unconsciousness; right hemiplegia with partial anæsthesia, aphasia, alexia, and word-deafness.	Embolism of l. middle cerebral artery; softening of Broca's gyrus, gyrus precentralis, et postcentralis, in their lower parts; insula, external capsule, claustrum, and external division of N. lentiformis.	State of occipital lobe not mentioned.
16	Pflüger, 1878.	M.	62	(?)	Left lateral H.	Injury of inferior part of thalamus by clot, with pressure on tractus. (?)	Attack of cerebral hemorrhage.	In right lateral ventrical much semi-fluid blood. Hemorrhage in corpus striatum, and inferior part of thalamus.	State of occipital lobe not mentioned.

TABLE III.

Cases of Hemianopsia from Lesions of the Thalamus Opticus and Corpora Geniculata.—Six Cases.

No.	Observer.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
6	H. Jackson and W. R. Gowers, 1875.	M.	65	(?)	Left lateral H.	Softening of caudal half of right thalamus: pulvinar disintegrated.	Left hemiplegia and hemianæsthesia. Death from a non-cerebral disease.		
9	Pooley, 1877.	M.	55	V. normal when first seen. Later, choked disk in left eye, normal fundus in right eye.	Fields normal when first seen. Sudden right lateral H., passing away, but returning in a few days.	Complete softening of left thalamus and surrounding white substance.	For 6 years epileptiform seizures, halluc. of sight, maniacal attacks. Right hemiparesis, reduced sensibility, aphasic symptoms, weak memory.	In left occipital lobe there was a "gummy tumor" 12×30 mm., adherent to pia. Right lateral ventricle much dilated.	Syphilitic arteritis and tumors.
19	Dreschfeld, Case I., 1880	F.	41	(?)	Left lateral H.	A tuberculous tumor almost replaced the right thalamus, and extended laterad through int. capsule to nucleus lentiformis. Right tractus opticus reduced to a thin band.	Paroxysms of headache and weakness of left leg for 7 years. Apoplectic attack and left hemiplegia, convulsions, coma.	The tumor extended in a ventral direction almost to the surface of the brain.	
30	Dreschfeld, Case II., 1882.	M.	40	Fundus normal. Central V. good.	Left lateral H.	Cysto-sarcoma in place of lateral part of right thalamus; lobus opticus, internal capsule and part of nucleus lentiformis involved. Right optic tract flattened.	Headache, giddiness, diplopia, left-sided paresis and tremor for 18 mos. Three convulsions. Left hemianæsthesia, and hemiplegia.		No syphilis.
31	Dreschfeld, Case III., 1882.	F.	52	Pupils equal and react well. Central V. fairly good.	Left lateral H., with a vertical line not quite reaching point of fixation.	A clot, ovoid and 6×8 mm., in the upper and post. extremity of right thalamus (pulvinar). Optic tracts, lobus opticus, and corp. genic., normal.	Bright's disease and mitral stenosis. Apoplectic attack, followed by left hemiplegia and transient aphasia. Also diminution of sensibility on left side of body. Special senses normal except V.	Several clots within the r. brain: two corresponding to fasciculi from precentral gyrus, and one under the parietal gyri.	
36	Rosenbach, 1883.	F.	34	(?)	Right lateral H.	Softening of external part of left thalamus; tumors in post. segments of both lobi optici; atrophy of left tractus opticus and of right optic nerve.	Right hemiplegia, right amblyopia. Epileptoid convulsions. Amnesic aphasia. Impairment of mental activity. Symptoms of pressure.	Softening of corpus striatum and internal capsule.	

TABLE IV.

Cases of Hemianopsia from Lesions Situated Chiefly in the White Substance of the Occipital Lobe.—Eleven Cases.

No.	Observer.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
2	Levick, 1866.	M.	40	(?)	H., of which side not stated, when first seen, Feb'y 9, 1866.	Abscess in posterior lobe of right hemisphere, 1½ inches in diameter (Pepper).	In May and June, 1865, slight (?) injuries to head, by falling objects. In July and August, headaches and extreme drowsiness; vertigo, sense of feeling drunk. No convulsions or paralysis. Death in coma eight months after injury.	Opacities in arachnoid. Various structures at base of brain softened and discolored. Small abscess in anterior lobe of left hemisphere. Lateral ventricles lined by consistent whitish-yellow inflam. lymph.	
14	Hosch, 1878.	M.	54	V. R. $\frac{15}{20}$, V. L. $\frac{15}{30}$. Slight redness and veiling of papillæ, and a few streaks of hemorrhage in retinæ.	Left lateral H. with vertical line a little to l. of point of fixation.	Large hemorrhagic cyst, almost destroying right occipital lobe to cortex.	Slight apoplectic attack and weakness of left side of body, with darkening of l. fields of V. Complete left hemiplegia after a third apoplectic attack. In fourth attack, right side paralyzed; death.	Large pigmented cicatrix in right corpus striatum, extending into thalamus. N. caud. et lentif. atrophied. Last attack due to a large fresh hemorrhage in 3d vent., causing extensive laceration.	
13	Baumgarten, 1878.	M.	(?)	Central V. normal.	Left lateral H. suddenly developed and persistent.	In the substance of the right occipital lobe was an old hemorrhagic cyst as large as a walnut, and the various convol. of the occipital lobe were softened, though recognizable.	None (?). Death in several months, from paralysis of the heart.	In centre of right thalamus was a so-called apoplectic cicatrix, half size of a lentil. Tractus optici and optic nerves normal. Fatty heart and contracted kidneys.	
15	Dmitrowsky and Lebeden, 1879.	F.	22	Papillæ congested, obscure limits, veins enlarged.	Right lateral H.	In the left cerebral hemisphere, a clot which involved the greater part of the corona radiata, and penetrating the temporal lobe almost to the cortex.	Headache, drowsiness, and difficulty in speaking. Aphasia.	?	

TABLE IV.—*Continued.*

No.	Observer.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesions.	Other Symptoms.	Other Lesions.	Remarks.
22	Westphal, Case I., 1881.	M.	42	Slight optic neuritis in l. eye.	Left lateral H. Vertical line passing through point of fixation.	Old focus of softening in white subst. of parietal and occip. lobes, as low as 2d temp. gyrus. Gyri of parietal and occip. lobes smaller and softer than those of left side. Volume of caudal end of right hem. much less than that of left hem.	Left-sided convulsions, followed by l. hemiplegia. Clonic spasms in paroxysms, lasting days or hours, often without loss of C. At end, some contracture of l. arm. Very slight anæsthesia; transient.	Basal optic apparatus and thalami normal.	
24	Senator, 1881.	M.	69	(?)	Left lateral H.	Clot occupying greater part of white subst. of right temporal lobe outside inf. horn of ventricle. Dorsally lesion destroys a large part of white subst. of inf. parietal lobule; mesad it extends to the lateral part of thalamus; complete interruption of post. part of internal capsule; occip. and temporal radiations cut through.	Left hemiparesis; contracture; conjugate deviation to right; speech normal. Sensibility to pain preserved. Later, movements of right arm ataxic; r. leg paralyzed.	Thalamus normal; right corp. genic. lat. flattened and yellowish-red. The clot also extended frontad through ext. capsule and outer segment of n. lent., as far as insula. Rest of brain normal.	
32	Stenger, Case VII., 1882.	M.	32	At close of life, for two months, blindness and halluc. of V.	Left lateral H. Verified by repeated tests.	In white substance bordering right post. horn, is a distinct, rather broad furrow, extending from post. border of thalamus caudo-laterad.	Symptoms of dementia paralytica; excitement, tremors, thick speech. Attack of l. hemispasm and hemiplegia. Later, attacks of left-sided spasm.	Decortication over left parietal lobe and whole of both occip. lobes, which are shortened and shrunken. Post. horns much dilated; the right more. Both thalami, especially in post. thirds, are collapsed and softer. Optic tracts equal and normal.	

TABLE IV.—*Continued.*

No.	Observer.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
34	Wernicke and Hahn, 1882.	M.	45	No lesion in fundus.	Right lateral H. Vertical line passing a little to right of point of fixation.	Abscess in white subst. of l. hemisphere, latero-dorsad of post. horn of V. At its frontal end the vent. was opened. The caudal end of abscess almost reached the apex of occip. lobe; mesad the abscess was limited by the ependyma. Destruction of white subst. of sup. and inf. parietal lobules.	Chronic phthisis. Pain in l. frontal and occipital regions; a cloud before right eye. Mind dull; right arm and leg awkward. Paresis of r. arm and leg; loss of muscular sense in r. arm. Later, complete paralysis. Trephining in upper post. quadrant of l. parietal bone; abscess in brain evacuated by deep incision. Relief to motor symptoms, etc. On 6th day, stupor, paralysis, and death.	Ventricle perforated and containing pus. No meningitis.	The diagnosis of abscess of occip. lobe was made *intra vitam* and confirmed. Surgically the case is most encouraging.
35	Jany, 1883.	F.	21	V. much reduced. R. + $\frac{1}{20}$, S. = $\frac{6}{20}$. L. fingers counted at 8'. Neuro-retinitis passing into atrophy, more in r. eye.	Right lateral H. In r. eye vertical line a little outside point of fixation. In l. eye outer field contracted.	Cysto-sarcoma occupying nearly whole of l. occip. lobe. Solid tumor lies at apex of occip. and mesad, as far as occipito-parietal sulcus.	Severe headache, most in l. occip. region. Vertigo. Paræsthesia and analgesia of r. hand and face. Stammering. Later, no objective symptoms, except in eyes; occip. pain, vomiting, convulsions; death.	Cystic part of tumor, of orange size, involves white subst. about post. horn of V., and under inferior parietal gyri.	
38	Richter, 1883, Case I.	M.	54	Pupils equal, normal in size and action. Optic nerves pale; vessels smaller.	Left lateral H. Vertical division line.	Clot of a certain age in r. occip. lobe in white subst., just laterad of post. horn of V., separated from it by ependyma. Cortex uninjured.	Hallucinatory paranoia. Left eye weak, and left hand numb. Left hemiplegia. Death in apoplectic attack.	Most ancient clot in r. temporal lobe. Third and fresh clot had disorganized the crura, filled third and fourth ventricles.	
39	Schmaltz. Cited by Vetter, 1883.	F.	69	Pupils and ocular muscles normal.	Right lateral H.	Yellow softening in left hemisphere, most destructive in occipital lobe. Caudal part of thalamus softened.	Apoplectic attack, followed by r. hemiplegia. Complete r. hemianæsthesia. Muscular sense lost. Left arm with choreiform movements. State of smell and taste uncertain.	Yellowish softening of various gyri in l. brain: pre- and post-central, parietal, and occip. gyri.	

46	Prevost, in Bulletin de la société méd. de la Suisse Romande, 1878.	M.	75	Fundus normal (Haltenhoff).	L. lateral H.	Yellow patch in right occipital lobe, from its mesal gyri inward into white substance along the post. horn of ventricle as far frontad as corp. genic. lat. and thalamus. Lobus opticus normal.	Attack sudden but without loss of c. Slight left hemiplegia. Partial left hemianæsthesia (hearing, taste, and smell uncertain because of stupidity).	Old thrombus of right posterior cerebral artery. Recent thrombosis of basilar. External gyri of brain and insula normal.	

TABLE V.

Cases of Hemianopsia due to Injuries to the Cranium and Brain; Lesion mostly Cortical.—Five Cases.

No.	Observer.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
3	W. W. Keen and W. H. Thomson, 1871.	M.	32	V. R. = 1. V. L. $\frac{2}{3}$. Opacities ant. cornea. Pupils, ocular muscles, and fundus normal.	Right lateral H. Vertical division.	In Sept., 1862, received a gunshot wound of post. end of cranium on left side. Entrance in median line 1¼ inches (31 mm.) above the external occipital protuberance. Exit 2 inches (50 mm.) from median line, and 3 in. (75 mm.) from entrance.	No immediate loss of consciousness. Impaired V. soon noticed. Ten days later loss of consciousness followed by r. hemiplegia. No aphasia. Paralysis and imperfect memory lasted for 2-3 months. No paralysis in 1870.		
Idem.	Observed by E. C. Seguin, in 1885.	M.	46	Pupils normal. Eye muscles normal. Fundus: blood-vessels normal, outer temporal quadrant of both disks whiter than normal; left a little whiter than right. V. R. = $\frac{20}{20}$ with — $\frac{1}{72}$; ax. 90. V. L. = $\frac{15}{15}$ with — $\frac{1}{72}$; ax. 90. R. reads No. 14 J. at 12″ with + $\frac{1}{12}$ S. No. 1 at 8″. L. reads No. 14 J. at 12″ with + $\frac{1}{12}$ S.; No. 6 at 8″. (Dr. Hale, House Surgeon, Manhattan Eye and Ear Hospital.)	Right lateral H. Division line not quite reaching point of fixation.	Entrance cicatrix barely noticeable. Exit is a large cup-shaped depression 5 × 6.5 cent. and 1.5 cent. deep. Bridge of bone between entrance and exit only 3 cent. Bottom of depression is firm but not osseous. Injury imitated on the cadaver shows an injury of parietal lobe, dorsad of angular gyrus. Occipital gyri uninjured. White substance deeply lacerated.	One epileptiform attack 6 years ago (1879). Right side a little weaker; more easily affected by alcohol. Uses left hand habitually. Mental action good.		Hemianopsia caused by injury to optic fasciculus. Occipital gyri uninjured.

TABLE V.—*Continued.*

No.	Observer.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
4	Hughes, 1873.	M.	38	Eyes normal except hemianopsia. (Prof. Wilson, Drs. Jacob and Swansy, of Dublin.)	Right lateral H. unchanged at time of discharge from hospital.	Injured by fall of a large iron vessel on back part of head. Compound depressed fracture of occipital and parietal bones. Several pieces of bone removed and coma relieved at once. Complete recovery in 3 mon. except impairment of V.	Coma relieved by operation.		
20	Schmidt-Rimpler, 1880.	M.	(?)	?	Left lateral H.	Vertical compound fracture of right occipital and parietal bones by a fall. A handful of bony splinters and some brain substance were extracted.	No paralysis. Recovered with complete deafness of r. ear, left lat. H., and occasional dizziness with loud tinnitus in r. ear.		
23	Heuse, 1881.	M.	(?)	Slight opacities of cornea, reducing V. and rendering ophthalmoscopic exam. unsatisfactory.	Right lateral H. Right temporal and left nasal half-fields were not absolutely dark but dim or very obscure.	Fall upon head causing a depressed fracture of cranium. Enormous cicatricial depression of the bones on the left side of the occipital end of the skull; a strong ridge-like depression extended from left parietal into the occipital bone.			
37	Nieden, 1883.	F.	22	During second paralytic seizure before operation: Eye muscles and pupils normal. Fields normal. Slight venous hyperæmia of papillæ and retinæ. Ninth day after operation V. L. = $\frac{12}{12}$. Field normal. V. R. = $\frac{1}{36}$, temporal field wanting to median line.	After operation monocular H. temporal field of right eye and in part of its superior inner (nasal) quadrant. Field of left eye limited in its upper and nasal areas.	Fall down steps, striking occiput repeatedly on succeeding steps. Unconsciousness, vomiting, pain in l. occipital region. At operation, 9 months later, no lesion found, but the dura mater (and brain) was injured by trephine to left of median line. After operation, complained of impaired vision.	Paralysis with partial anæsthesia of right side of body. Mental action normal. Great recovery in six months. Seven months after injury severe headache, vertigo, semi-unconsciousness. Right hemiplegia; face not involved. Also right hemianæsthesia. Attacks of headache with loss of C.; jerks of *left* side muscles, face, and limbs. Trephined in left upper quadrant of occip. bone. All symptoms passed away and, 13 months after original injury, she returned to her work.		This case was probably one of hysterical nature, and even the optic symptoms may have been of same sort.

TABLE VI.

Cases in which Lateral Hemianopsia was due to Lesions of the Cortex of the Brain and Subjacent White Matter (mostly Cortical Lesions).—Sixteen Cases (Arranged in the Order of their Value for Studying the Localization of the Visual Centre).

No.	Author.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
1	Chaillou, 1863.	F.	69	(?)	Lateral H. Side not stated; most probably on left side, corresponding with hemianæsthesia observed	Atrophy of gyri at the end of left (?) fiss. of Sylvius and of inner face of occipital lobe; white matter of occip. lobe much atrophied.	Disorder of speech followed by coma. Recovery with imperfect V. and weakness of r. arm. Previous to admission patient had had a similar "congestive" attack, leaving left hemianæsthesia. Hemianopsia older still. Death in a third attack.	Small patches of softening in left (?) thalamus. Inferior part of left corpus striatum contained a cavity 2 x 5 cent. Right hem. of cerebellum in inf. aspect contained a cavity (old patch of softening) 3 x 1 cent. Oldest lesion was that in occip. lobe.	Sides of brain probably reversed in autopsy. Lesion of occip. lobe and thalamus must have been in *right* hemisphere.
26	Westphal, Case II., 1882.	M.	38	Central V. normal. R. pupil trifle wider. Eye-muscles not stated. No lesion to ophthalmoscope.	R. Lateral H. discovered June, 1880. Nearly vertical line passes a little to left (?) of point of fixation.	Softening of gyrus angularis, of occipital lobe (much adhesion over cuneus and precuneus). Optic nerves and chiasm normal.	In June, 1879, alcoholic (?) convulsions, followed by delirium and imperfect speech. In august, awoke with r. hemiplegia (arm most) and complete loss of speech; word-deafness and amnesic aphasia. Later, repeated convulsions (clonic) on r. side. Slight r. sided anæsthesia and paresis; great loss of muscular sense.	Left hemisphere showed an extensive cortical softening, involving post-central as far as supra-marg. gyrus, the whole of parietal lobe, gyrus angularis, and nearly the whole of occipital lobe. Slight softening of cortex at junction of 2d and 3d temp. gyri. No central lesion.	
32	Stenger, Case VIII., 1882.	M.	52	Special senses normal on admission.	L. lateral hemianopsia after left hemiparesis. Persistent.	Softening of white subst. of r. par. lobe interrupting fasciculus of Gratiolet.	Admitted with dementia paralytica. Shortly after, convulsions in left side of body, followed by l. hemiparesis with numbness of l. hand. Muscular sense much impaired in l. hand. Varying l. hemiparesis. Death in general convulsions.	Int. and ext. hydrocephalus. Cortical softening over right parietal and occipital lobes; subjacent white subst., yellowish, and much atrophied. Right thalamus smaller and softer than left. Int. aspect of occip. lobe not mentioned. Right post. horn much distended.	Degeneration from visual cortex along optic fasciculus to thalamus.

TABLE VI.—*Continued.*

No.	Author.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
7	Förster and Wernicke, 1876.	M.	—	(?)	At first, temp. field of right eye obscured vertically almost to point of fixation; the nasal field of left was darkened also almost to point of fixation. The left temp. field was also slightly contracted. Later, a degree of left lateral hemianopsia was found, but the original limits of the right hemianopsia remained unchanged.	The area of softening caudad of the fissure of Sylvius, including the inf. parietal lobule and gyrus angularis, penetrated to the lateral ventricle, probably interrupting the optic fasciculus.	Repeated paralytic attacks on right side; aphasia.	Embolus in left Sylvian artery; softened patch in cortex, including inf. parietal lobule, gyrus angularis, and frontal part of occip. lobe; numerous small necrotic foci in l. nucleus lenticularis, nucleus caudatus, thalamus, and external capsule. Insula, chiasma, and optic nerves normal.	This case has been sometimes cited twice, as separate accounts were published by W. and F.
10	Jastrowitz, Case I., 1877.	M.	(?)	Transient paralysis of R. N. VI. Papillæ normal; V. relatively good; fixation possible.	Right lateral hemianopsia (Hirschberg).	Soft sarcoma of left occipital lobe, involving occip. gyri and precuneus. It extended in conical form toward the post. horn of lateral ventricle as far laterad as Gratiolet's fasciculus which presented a slight discoloration and punctate hemorrhages as far as the thalamus.	Vertigo; loss of memory, of energy; general weakness; amnesic and ataxic aphasia, alexia, and agraphia. Right hemiplegia appeared later; paralysis of variable intensity.	Thalami, lobi optici, tractus, chiasma, and nervi optici presented no alterations.	
18	Curschmann, 1879.	M.	50	(?)	Left lateral H., which persisted from 10th to 16th day (death).	Large focus of softening in right occipital lobe, extending to the surface, especially on its caudal and mesal parts.	Drank sulphuric acid with usual local effects. There occurred, on 10th day, embolism of right brachial artery. A few days later, patient complained that he could not see well with his left eye. No other symptom of local disease in brain. Death, of inanition, on 16th day.	Usual lesions of æsophagus, etc. An extension of inflam. to inner coat of aorta. Complete embolism of right brachial artery.	

TABLE VI.—*Continued.*

No.	Author.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
11	Jastrowitz, Case II., 1877.	(?)	(?)	(?)	Limitation of visual fields to the right.	Large patch of softening in left occipital lobe.	Aphasia.	Partial embolism of left internal carotid artery.	
17	Nothnagel, 1879.	M.	51	(?)	Partial right lateral H.; shortly before death, total blindness.	In left hemisphere (besides several small lesions) there was a yellowish-red softening of the entire occipital lobe. Chiasma, optic tracts, and nerves normal.	Patient awoke with paralysis of left arm and obscurity of vision. There was monoplegia of left arm; no anæsthesia. Death by inanition.	Carcinoma of pancreas, with various secondary deposits. Endocarditis aortica verrucosa. Numerous spots of embolic softening in brain. In r. hem. a patch in middle of precentral and postcentral gyri, softening of caudal extremity of sup. parietal lobule, with extension into interparietal fissure, and into white substance to ventricle. Small patches in r. occipital lobe.	
27	Marchand, 1882.	M.	72	(?)	Left lateral H. — "complete." (Dr. Pepmüller)	Patch of softening in right occipital lobe; pia adherent; apex of occip. lobe occupied by a necrotic patch as large as a hazel-nut, separated from deeper parts of brain by a softened yellowish zone about .5 cent. thick.	Sudden left hemiplegia. Death in a few months. No details as to motor and sensory symptoms.	Gyri adjacent to occipital in same condition of yellowish softening. Arteries tortuous and thickened.	
42	Richter, Case II., 1885.	M.	70	After H. there occurred conjunctivitis, keratitis, cataract; phthisis of left eye.	Complete left lateral H. Defect persisted in right eye.	Patch of softening in right occipital lobe.	Senile dementia.		
43	Richter Case III., 1885.	M.	48	L. pupil reacts well; right very slightly. Left optic nerve normal; right, atrophied.	Left Lateral H.	Patch of softening in right occipital lobe.	Syphilis in 1865. Since 1880, repeated apoplectic attacks, accompanied by temporary r. hemiplegia and aphasia. In 1882, l. hemiplegia and hemianopsia. Dementia.	Small psammoma in r. optic nerve. Patch of softening in l. island of Reil.	

TABLE VI.—*Continued.*

No.	Author.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
44	Richter, Case IV., 1885.	M.	40	Pupils react well. Fundus normal. Muscles normal. Shortly before death, no ophthalmoscopic changes.	Left lateral H. suddenly developed in hospital. Limit to left of fixation point.	Firm adhesion of pia to occipital gyri, also over cuneus, and lower temporal gyri. Tip of occip. lobe least injured. Granular bodies found in white substance of occip. lobe, from cuneus and gyrus hippocampi, and from inferior temporal gyri, along outer wall of ventricle, in a tract which can be followed to atrophied pulvinar. No atrophy of tractus or optic nerves.	Syphilis in 1861. Epileptiform attack, mental failure, staggering gait, weak legs, tongue tremulous, speech thick. Hallucination of V. Death from epileptoid seizures.	Basal nerves normal. In various places, opacity and thickening of pia.	
45	Seguin, 1885.	M.	46	Pupils and fundus normal. Central vision good with glasses for presbyopia (Dr. C. R. Agnew).	Sudden attack of left lateral H. persisting till death. Vertical line passing a little to left of point of fixation. Was able to read and write. Always complained that his left eye alone was affected.	Patch of old softening involving almost the whole of the right cuneus, not quite reaching apex of lobe, including a part of the gyrus hippocampi, the fifth temporal, and encroaching on the fourth temporal gyrus. No other lesion. (?)	Mitral regurgitation, enlargement of the heart, various peripheral embolisms, pseudo-intermittent fever. No hemiplegia or anæsthesia. Slight ataxia of left hand. Death six months after hemianopsia appeared.	Verrucose disease of mitral valve, tissue containing balls of micrococci. Large infarcts in kidneys and spleen. No other important superficial lesions on surface of brain. Chiasm, tracts, and nerves normal.	The absence of hemiplegia, distinct anæsthesia, and convulsions makes it almost certain that there were no gross lesions within the brain. Location of lesion correctly diagnosticated during life.
28	Haab, 1882.	M.	68	Central vision = 1. Optic nerves presented "a senile grayish color."	Left lateral H. Complaint of left eye only. Vertical line reaching quite (?) up to point of fixation. H. persisted till death.	Caudal end of right hemisphere .5 cent. shorter than its fellow. Patch of softening (cavity and debris) mostly on mesal aspect of occip. lobe, including apex. It involves the cuneus in its inferior half, the fifth temporal gyrus, and fissura hippocampi. White substance destroyed as far as ventricle.	Endo- and peri-carditis, sudden attack of left hemiparesis rapidly passing away almost wholly. No hemianæsthesia. Death in two years.	No other cerebral lesion. Optic nerves, chiasm, and tracts normal (microscopic examination).	Diagnosis during life; embolism of artery supplying hinder part of right thalamus.

TABLE VI.—*Continued.*

No.	Author.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
41	Féré, 1885.	F.	52	Ophthalmoscope showed no lesions. Pupils not mentioned.	Right lateral H. Vertical line passing through point of fixation.	Yellow patch destroying the greater part of the left cuneus, and encroaching somewhat on fifth temporal gyrus. No secondary degeneration.	Nov. 2, 1883, slight and transient attack of right hemiplegia. On admission to Salpétrière, no motor symptoms, but slight r. hemianæsthesia to pain and cold. Hearing, taste, and smell normal.	No other cerebral lesions.	Lobi optici, tractus, chiasm, and nerves normal.
29	Huguenin, 1882.	F.	8	At first visit, April 16th, vision and hearing good. Optic nerves normal. On 27th, slight optic neuritis.	Left lateral H. discovered on May 20th.	A caseous tumor. 3 x 3 x 2.5 cent. lay on the mesal aspect of the right occipital lobe; partly embedded in the brain, firmly adherent to pia. It was over the fissura hippocampi, extending above and below it, and into the cuneus.	Pertussis followed by slow mental action and ill-health; a few months later, headache in paroxysms, vomiting; recurring convulsions; increasing dementia. Death of broncho-pneumonia; never localized paralysis, or anæsthesia.	A small tumor on the the apex of the right frontal lobe. Ependyma of ventricles granular; pia over chiasm, and in both fossæ Sylvii, slightly thickened.	

and Thomson, I have the additional information that a few days ago the hemianopsia was found unchanged, twenty-three years after the reception of the injury. I made an examination of the patient, Hughes, this morning, with the following results :

He presents no distinct paralysis or anæsthesia, or aphasic symptoms. His tongue deviates a trifle to the right, and the grasp of the right hand is a little less than that of the left. Dynamometer test : L, 38, 34 ; R, 35, 34. The knee-jerk is abnormally great on both sides, but equally so.

Tests reveal no anæsthesia, but the patient thinks that his tactile sensibility is very slightly dull on the right side of head and in right hand. The muscular sense, tested by knowledge of passive movements of fingers, and by different weights laid in both hands, while the patient's eyes are closed, is normal.

He habitually uses the left hand more than the right, but this is on account of his loss of vision on the right.

He adds that when under the influence of liquor his right leg and arm feel the effects first and most.

Only one epileptiform seizure is known to have occurred, viz.: an attack in the night, about six years ago. He claims that his memory is now good. It was formerly weak, but he never, apparently, had amnesia of words.

The scalp presents two cicatrices ; that of entrance very small, that of exit of the ball enormous and greatly depressed. The following are some topographic measurements with the head placed so as to have the skull resting on the alveolo-condyloid plane of Broca.

The entrance wound is in the median line, about 3.5 cent. above the external occipital protuberance. From the bregma along the median line to this cicatrix is 15.5 cent.

The exit scar is a large cup-shaped depression situated dorso-laterad of the other, near the parietal eminence. Its frontal edge is 6.5 cent. from the bregma ; its mesal edge nearly at the median line (.5 cent. distant) ; and its fronto-lateral edge is 12.75 cent. from the left tragus. Its transverse diameter is 5 cent., its longitudinal diameter 6.5 cent. Its depth is 1.5 cent. The bridge of bone between the two scars is only 3 cent. broad.

The bottom of the exit scar is very firm though not bony, and the patient is not affected by reasonable manipulation.

A rough test with a small white object at 18 inches shows right lateral hemianopsia, with line passing outside of point of fixation ; there is besides a darkened area in the left upper temporal quadrant.

Dr. G. W. Hale, House Surgeon of the Manhattan Eye and Ear Hospital, has very kindly made an examination of H.'s eyes, and made diagrams of his visual fields. He finds the following:

R = $\frac{20}{60}$: $\frac{20}{40}$ w. — $\frac{1}{12}$; ax. 90° :
L = $\frac{16}{200}$: $\frac{16}{10}$ w. — $\frac{1}{12}$; ax. 90° :
R reads No. 14 J at 12″ w. + $\frac{1}{12}$s No. 1 J at 8″ :
L reads No. 14 J at 12″ w. + $\frac{1}{12}$ No. 6 J at 8″ :

Pupillary reaction, normal.
Eye muscles : no insufficiency either at 20′ or 1′.
Fundus : blood-vessels of normal size ; outer temporal quadrant of either disk whiter than normal, left a little whiter than right. No other lesions.

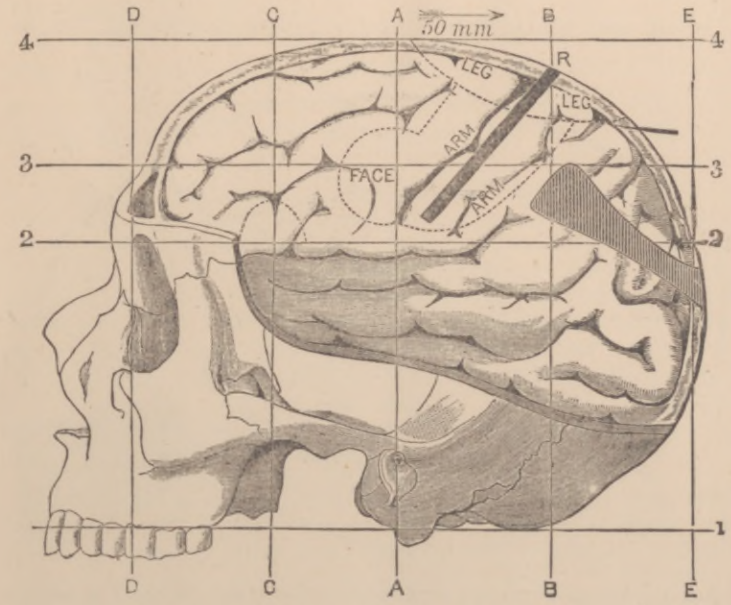

FIG. 1.—The probable course of the minié ball through the brain in case 3 (Keen and Thomson) is indicated by the club-shaped shaded figure in the occipital part of the head, extending across lines 2 and B.

By the kindness of Drs. Peabody and Ferguson I have had the opportunity of repeating the injury upon the cadaver at the New York Hospital. Trephine openings were made in the cranium of a male subject at points corresponding with the author's measurement of H.'s cicatrices, and an iron rod pushed through, followed by a large seton of jute. The hemisphere was placed in alcohol for harden-

ing. It was then found that the track of the ball was entirely dorsad of the occipital, through the parietal lobe almost to the confines of the postcentral gyrus. Its penetration was such, however, that it must have injured the optic fasciculus on its way to the cuneus. See fig. 1.

CASE 28.—Haab: male, æt sixty-eight years. In Feb., 1878, while under treatment for endo- and peri-carditis, experienced an attack of paresis of the left extremities. This rapidly passed away, leaving a certain degree of disability, for after working with the left hand patient experienced pain in the left arm, and palpitation.

When seen by Haab in July the patient complained that he

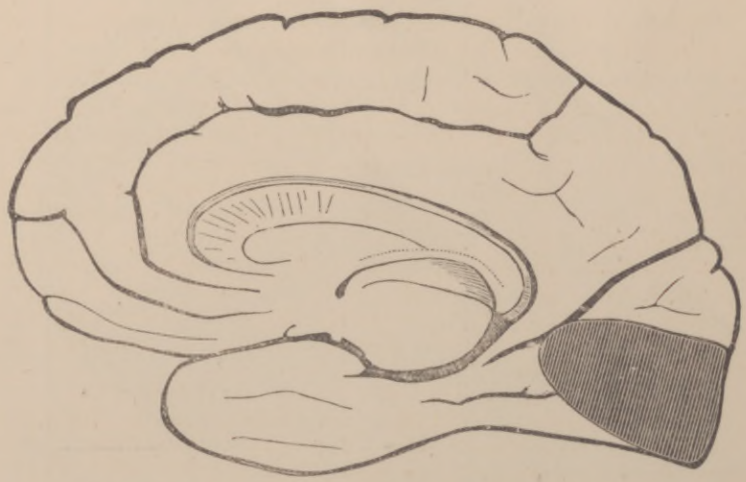

FIG. 2.—Mesal aspect of right hemisphere (Ecker) showing patch of softening in case 28 (Haab).

could not see to his left with his left eye—thought his right eye was normal. Examination showed only a trace of paresis or awkwardness in left extremities; no anæsthesia. Intelligence normal. Hearing good. Central vision = 1 (H. 2). There was left homonymous hemianopsia, the limit reaching quite (?) up to the fixation point. In right fields color-perception good.

Optic nerves present a "senile grayish color."

During the year several re-examinations gave the same results. The patient insisted that there was a veil or cloud over the *left* eye. Death in July, 1879.

Autopsy.—The caudal end of the right hemisphere was .5 mm. shorter than its fellow. There was a depression in right occipital lobe, the pia hanging loosely over a cavity containing clear fluid. The patch was mostly upon the mesal aspect of the hemisphere (including apex). It occupied the site of the fissura hippocampi, and extended beyond it above and below. The frontal end of the cyst was at six cent. from the apex of the lobe. The white substance was but slightly injured, and there was no communication between the cyst and the posterior horn of the ventricle. The vertical height of the patch was 2–3 cent.

No other cerebral lesion. The optic nerves, chiasm, and tractus were normal to a microscopic examination. Haab's diagnosis during life was embolism of an artery supplying the hinder part of right thalamus opticus.

Case 29.—Huguenin. A girl, æt. eight years. In autumn of 1878 whooping-cough, followed by ill-health and sluggish mental action. In January, 1879, headache in paroxysms ; later, frequent vomiting, sleep broken ; no motor or ocular symptoms. At end of March severe convulsions, which have frequently recurred, constituting the principal phenomenon. Increasing dementia. Seen by Huguenin, 16th April, 1879. Child demented ; understands what is said, and, according to parents, replies well ; memory feeble ; general muscular weakness, but no localized paralysis. Vision and hearing good. Seems sensitive to pinching, etc. Optic nerves normal.

Temporary improvement under Ki. and syr. ferri iodidi.

April 27th, second ophthal. exam. Slight neuritis with some swelling (no "stauung") Headaches. In middle of May it was noticed that patient held her head obliquely to the left. Exam. on 20th revealed left homonymous hemianopsia. This symptom was the only one indicating a focal lesion of the brain, and it persisted. Death in June, of broncho-pneumonia.

Autopsy.—Two tumors were found in the brain ; one at the apex of the left frontal lobe, the other near the apex of the right occipital lobe. Ependyma of ventricles granular ; slight thickening of pia over chiasma and in both fossæ Sylvii.

The second tumor lay in the mesal aspect of the right occipital lobe, projecting a few mm. above the level of the brain, firmly adherent to the pia and only slightly to the

dura. Its length was 3 cent., height 3 cent., thickness 2.5 cent.—mostly buried in brain substance. It lay directly over the sulcus hippocampi, extending to either side of it. Basis of occip. lobe not involved. Tumors caseous.

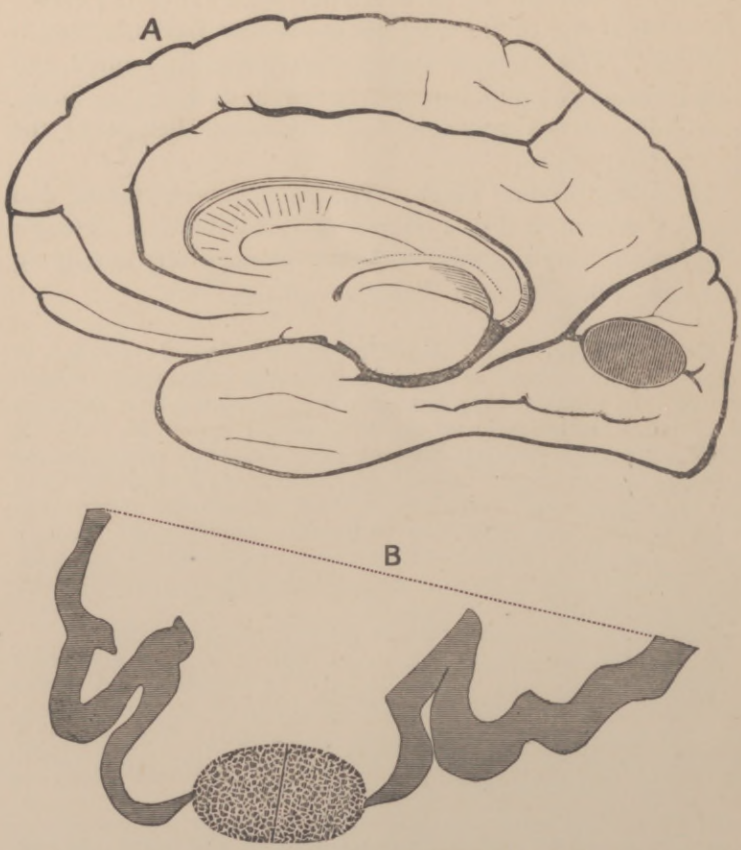

FIG. 3.—A. Tumor in mesal aspect of right occipital lobe : left lateral hemianopsia. B. Diagram of horizontal section, showing the slight penetration of the tumor. Case 29 (Huguenin).

CASE 41.—Féré. Female, æt. fifty-two. In November, 1883, sudden apoplectic attack followed by transient right hemiplegia. On admission to the Salpétrière, no motor symptoms. Partial and slight right hemianæsthesia to cold and pain. Hearing, taste, and smell normal. Typical right lateral hemianopsia, vertical line

passing through point of fixation. No ophthalmoscopic lesions; state of pupils not mentioned.

Death Dec. 24, 1884. Autopsy showed only a yellow patch destroying the greater part of the left cuneus and encroaching somewhat on the adjacent second temporal gyrus (gyr. temp. 5 of Ecker). No secondary degeneration. Corpora geniculata, lobi optici, tractus, chiasm, and optic nerves normal. Gray commissure of 3d V. absent.

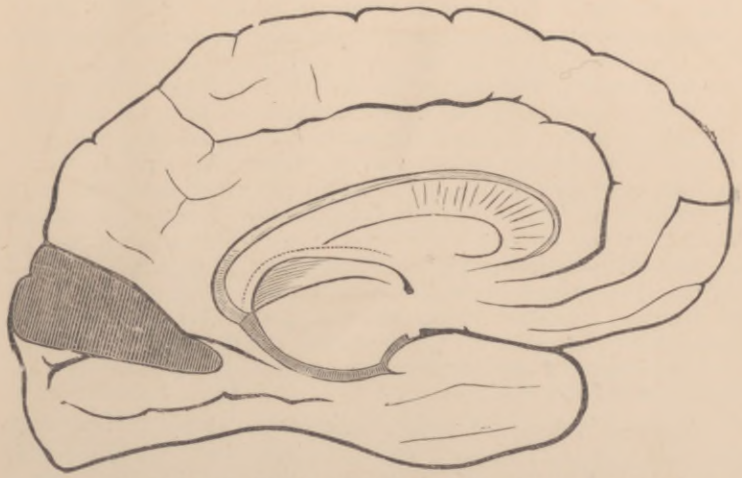

FIG. 4.—Mesal aspect of left hemisphere (Ecker). Patch of softening causing right lateral hemianopsia. Case 41 (Féré).

CASE 45.—Seguin. Mr. J. W. D., æt. 46, consulted me on January 18, 1884, for insomnia and dyspepsia. Wakefulness was most marked in early morning. Has grown paler, weaker, and thinner of late. Denies dyspnœa. Examination showed general anæmia, feeble, slow pulse (63 to 66 beats per minute). Heart feeble, with a distinct, harsh, mitral regurgitant murmur. There were pulsations in the external jugular veins. Urine normal, though of high specific gravity.

Under digitalis, cannabis indica, nux vomica, and arsenic, in various combinations, and a much more nutritious diet, with a glass of rich claret at his meals, a good recovery was obtained in about six weeks. Sleep was sound; the patient had regained weight and color.

November 26, 1884, I was sent for to visit him at his home. I learned that in the spring he had travelled south as far as Havana, and returned in excellent health, to all appearances. He was then, and for the succeeding three or four months, much overworked, and especially worried about his business, which was far from

prosperous. He had given up his claret, and, most unfortunately, purchased and used quite actively a rather strong home-gymnasium. His house was situated at the top of one of our hilliest streets, and this he climbed rapidly every day.

I found him suffering apparently from regular intermittent fever; severe chills followed by high fever and sweating. He had been severely purged, and was quite weak. His heart was larger than when first seen, and the mitral murmur was much louder and more diffused. He was given quinine and nutritious food.

A few days thereafter, about December 5th, I was sent for in haste, because of an attack of a nervous nature. I found Mr. D. considerably alarmed, but rational, and free from serious symptoms. He complained of numbness in the whole left side, cheek, arm, leg, and trunk; most marked in the hand and foot. There was no distinct hemiplegia, and no anæsthesia to ordinary tests; he thought, however, that tactile sensibility, as tested by passing his fingers over objects, was somewhat duller. He was most concerned, however, about another symptom, which he stated as a "blindness of the left eye." He could not, he said, see objects on his left without turning his head and eyes. Testing by means of a small bright object in the usual way, revealed typical left lateral hemianopsia, with a vertical division line not including the points of fixation. Central vision was as good as ever, as tested by newspaper type. Dr. C. R. Agnew was asked to see the patient the next day, and the following is a copy of his report of the condition of the patient's eyes:

"My dear Doctor: I have examined Mr. D.'s eyes. He has left hemiopia, as you say. He has opaque nerve fibres in nasal half of left optic disk, extending off a little distance into the fundus, which is physiological. He has a few punctate changes in the pigment layer of retina in *both* eyes, chiefly the left. I do not think that these things have any thing to do with the eye trouble—*that is central*, as you say. I agree with you in all you say, and have nothing to suggest in the way of topical treatment.

"Yours faithfully, C. R. Agnew."

My diagnosis at the time was embolism of a branch of the posterior central artery supplying the meso-caudal part of the right occipital lobe.

Mr. D.'s illness lasted, with most remarkable symptoms and extraordinary remissions, until May 17, 1885, when he died.

The chief features of this long sickness may be summarized as follows:

In December he had a violent attack of acute hallucinatory mania (both aural and visual hallucinations), due probably to cerebral anæmia. Under large doses of chloral, digitalis, and most persistent feeding with large quantities of milk and eggs, this subsided.

In February Mr. D. was able to go to Nassau, N. P. While there the severe chills, high fever, and sweats returned, and

proved rebellious to large doses of quinine. These chills followed no distinct type of periodicity ; they occurred twice a day, every second day or daily.

He returned to New York April 5th, and to the last, recurring febrile paroxysms usually clearly intermittent, were prominent features of the case. His general condition was better, but the heart was larger and presented an extremely loud and diffused mitral regurgitant murmur. During the month several attacks of visceral and peripheral embolism occurred, characterized by hæmaturia, splenic pain, and enlargement, a few discolored patches under the skin.

[In the preceding November, shortly after the hemianopsia, he one day complained of pain and swelling of the right palm, followed by a turgid condition of the whole hand for several days ; probably embolism of a part of the palmar arch.]

These embolisms were all recognized at the time as dependent upon the mitral disease, and it finally occurred to me that the intractable, irregular intermittent fever might also be of cardiac origin, each attack due to the detachment of microscopic particles of the diseased valves.

Dr. William H. Draper was called in consultation May 8th, and made the formal diagnosis of ulcerative or malignant endocarditis.

Previous to death, for a period of about a fortnight, the patient's speech was sometimes difficult to understand ; his articulation was defective, partly from extreme general debility, but also from some want of power in the buccal muscles. The hands both showed disorders of movements, choreiform tremors, and in the left hand slight ataxia in larger motions.

Often Mr. D. complained of numbness and coldness of the left hand.

At no time was there distinct hemiplegia or monoplegia, and repeated tests of sensibility showed it to be nearly if not quite normal, so that I was of the opinion that no emboli of any size had reached the brain since the attack in November. Several tests were made of the hemianopsia, one a few days before death. It persisted to the last unchanged, as judged by rough measurement, and vision remained good. The patient always insisted that his left eye was weak (a statement made by other hemianopsic patients). He was able to read and write easily until a few weeks before death, when increasing debility confined him to his bed.

The autopsy was made with the assistance of Dr. W. R. Birdsall, on the evening of the fatal issue, May 17th. The spleen and kidneys contained several infarcts of various ages, some very large, and looking like hemorrhagic foci.

The heart was much enlarged ; the mitral valves deformed and bearing enormous rough vegetations, one almost polypoid. Sections through some of these vegetations, stained by Gram's method, showed under the microscope globular nests of micrococci and separate colonies of bacteria. The aortic valves and aorta were normal.

The brain was generally anæmic. The basal vessels and middle cerebral arteries free from emboli and thrombi. The basal nerves, the optic tracts, and the chiasm were most carefully examined and found normal. On the right lateral aspect of the pons, caudad of the IVth nerve, a small branch of the basilar artery contained a firm thrombus of dark red color about 4 mm. in length; the vessel supplied the velum medullare anterius. The left hemisphere presented a small area of extreme congestion and ecchymosis over the folds of the second frontal gyrus; there was another patch at the foot of this gyrus extending toward the orbital gyri.

The right hemisphere had a similar superficial recent lesion (ecchymosis) at the vertex, extending over the dorsal extremity of the fissure of Rolando.

FIG. 5.—Mesal aspect of right hemisphere (Ecker). Patch of softening causing left lateral hemianopsia. Case 45 (Seguin).

Viewing the brain from above, the occipital extremity of the right hemisphere appears thinner than its homologue. This is found to be due to the destruction of the mesal surface of the right occipital lobe by a large focus of yellow softening, evidently an old patch. The lesion involves the basal part of the cuneus, the fourth and fifth temporal gyri (Ecker), and a part of the gyrus hippocampi. The destruction does not quite attain the tip of the occipital lobe.

The remaining gyri of both hemispheres were normal.

I would add that the above records of the appearance of the brain were made at the time of examination by our President, Dr. Birdsall, and me.

Most unfortunately, the brain was not cut at once. The left hemisphere was separated, leaving the "stamm" attached to the right hemisphere, and these were placed in bichromate of potassium, with the intention of making a series of sections after complete hardening.

Through mishaps the process of hardening was not very successful, and the right hemisphere particularly suffered from too prolonged pressure upon its temporal lobe, which disintegrated. The result is that I can only show you to-night the occipital half of the right hemisphere with the patch, which I consider the essential and truly causal lesion of the hemianopsia. The destruction of tissue extends only a few mm. into the subjacent white substance. The state of the internal capsule, thalami, etc., remains unknown, owing to the misfortune in preserving the specimens. From the history of the case, however, judging from the absence of hemiplegia and marked anæsthesia, it may be safely assumed, in the light of our present pathological knowledge, that there were no lesions, or at least no tangible lesions, in the central parts of the brain.

That the destruction of the right cuneus and fifth temporal gyrus was the cause of the left lateral hemianopsia during life, I have not a shadow of a doubt.

The softening was produced by embolism of the third branch of the posterior cerebral artery, the occipital artery of Duret.

The objections which may be presented against the value of my case, in consequence of its imperfect anatomical investigation, are greatly reduced in force by the consideration that the case is one in harmony with many others. Were it a contradictory or anomalous case, it would certainly possess much less value.

Now, gentlemen, what conclusion may reasonably be drawn from all these cases?

1. That lesions in the mesal aspect of the temporal lobes, or even in other basal districts of the hemispheres, may give rise to hemianopsia indirectly by pressing upon the primary optic centres or upon the tractus optici and chiasm.

2. That lesions of the corpus geniculatum laterale, pulvinar, and latero-caudal parts of the thalamus may cause hemianopsia; usually in association with hemianæsthesia and hemiplegia, or hemianæsthesia alone.

3. That a lesion of the white substance of the occipital lobe, in the caudal radiations of the internal capsule, may cause hemianopsia alone, or with hemianæsthesia.

4. That lesions of the supra-marginal gyrus, angular gyrus, and inferior parietal lobule with the subjacent white substance may cause hemianopsia—with or without other symptoms (hemiplegia, loss of muscular sense, word-deafness, etc.).

5. That a lesion of greater extent, involving the speech centre, the motor convolutions, and the parts enumerated above (4), due usually to embolism or thrombosis of the entire Sylvian artery, will, when existing on the left side, produce aphasia, alexia, hemianopsia, and hemiplegia.

6. That lesions of the occipital lobe, cortex, and subjacent white matter produce blindness when bilateral, and hemianopsia when unilateral. This conclusion is in accord with Exner's (1881).

7. That a lesion of the cuneus and adjacent 5th temporal gyrus (Ecker) on one side produces lateral hemianopsia of the opposite side.

In support of this last conclusion I would again invite your attention to the cases 28, 29, 41, and 45.

I have endeavored to fuse the diagrams of the sixteen cases with occipital lesions (exclusive of the traumatic ones) on one chart, by the successive application of layers of India ink. The larger lesions were first indicated on the outline diagram, and the most limited lesions washed last. I was somewhat hindered by the "running" of the black in the lines indicating gyri, yet I think that the maximum color due to the superposition of the greatest number of layers is over the cuneus, and next to the occipital apex, as a whole. This is a simplification of one of Exner's methods, and I think may, with some improvements, be made serviceable for clinical teaching. [The diagram was shown at the time of reading the paper.]

Let us now turn to the physiological and theoretical aspect of our subject. My time is limited, and I can only treat this highly important matter in a most summary way.

What do the most modern physiological researches teach us upon this question of the location of the cortical visual centre and its white connecting fasciculi?

The views of Munk and Ferrier are the authoritative ones.

The former physiologist has persistently taught that the visual areas, or centres for psychic vision, are in the occipital lobes, and that each visual area has connections with both retinæ. He invariably produced hemianopsia in dogs by destruction of one occipital lobe. These experimental results have been verified by Ganser, von Gudden's assistant, upon kittens.

Ferrier's theory, supported, as he believed, by experiments upon monkeys, has received an apparent verification at the hands of Prof. John C. Dalton.[1] Ferrier thought that the visual centre was in the angular gyrus. The following are his most recent conclusions, as presented to the Royal Society, and published in its Proceedings, xxxv., p. 229, and abstracted in *Brain*, April, 1884.

1. Lesions of the occipito-angular region (occipital lobes and angular gyrus) cause affections of vision without affection of the other sensory faculties or motor powers.

2. The only lesion which causes complete and permanent loss of vision in both eyes is total destruction of the occipital lobes and angular gyri on both sides.

3. Complete extirpation of both angular gyri causes for a time total blindness, succeeded by lasting visual defect in both eyes.

4. Unilateral destruction of the cortex of the angular gyrus causes temporary abolition or impairment of vision in the opposite eye—not of a hemiopic character.

5. Deep incisions may be made in both occipital lobes at the same time, or the greater portion of one or both occipital lobes at the same time may be removed without any appreciable impairment of vision.

6. Destruction of the occipital lobe and angular gyrus on one side causes temporary amblyopia of the opposite eye and homonymous hemianopia of both eyes toward the side opposite the lesion.

7. As in none of the cases recorded, either of partial unilateral or bilateral destruction of the occipito-angular region, were the amblyopic or hemianopic symptoms permanent, it is concluded that vision is possible with both eyes if only portions of the visual centres remain intact on both sides.

[1] John C. Dalton, in *New York Medical Record*, Oct. 26, 1881.

It will be seen that the results of our pathological analysis are seemingly favorable to both the theories of Munk and of Ferrier. But, on the one hand, the most conclusive cases, *i. e.*, those with the most limited cortical lesions, are wholly opposed to Ferrier's views and in favor of Munk's; and, on the other hand, a peculiarity in the anatomy of the occipital extremity of the brain goes to explain Ferrier's results without assuming the existence of a cortical visual centre in the angular gyrus. It is this: that the optic fasciculus of Gratiolet and Wernicke, on its way from the caudo-lateral aspects of the thalamus, in the internal capsule, passing out caudad, lies latero-dorsad of the posterior horn of the lateral venticle, and close under the inferior parietal lobule and the angular gyrus, on its way to the occipital lobe (cuneus chiefly). A lesion of the angular gyrus, the supra-marginal gyrus, and even of the inferior parietal lobule, is almost certain to involve this optic fasciculus, and thus cut the communication between the visual centre and the eyes.

I pass around a specimen in which, on a horizontal longitudinal section of the human brain hardened in bichromate of potassium, the optic fasciculus is plainly visible as a homogeneous whitish band. It is evident that lesions in the angular gyrus and supra-marginal gyrus could easily penetrate deeply enough to injure this fasciculus.

It seems to me that with this anatomical knowledge the discrepancies between Ferrier and Munk's results disappear in part, and that some of the cases of my sixth category (cases 26, 32) are reconciled with the others.

Next, as to the various purely hypothetical or clinical theories of the course of the optic paths. Of these the best known is that of Prof. Charcot. His well-known diagram of the course of the optic fibres from the retina to the visual centres represents a second decussation of the fasciculi laterales through the corpora quadrigemina (lobi optici) on their way to the internal capsule, so that finally each internal capsule contains all the fibres for the opposite eye. This diagram was made to explain and support Charcot's theory of the production of amblyopia of one eye by lesion of the occipital lobe and the internal capsule of the opposite side. He

thought that he had observed that amblyopia of one eye, and not hemianopsia, was the companion of hemianæsthesia produced by lesion of the internal capsule.

I regret to say that my illustrious master's theory has not been supported by either clinical observations or by post-mortem results. I know of but one case with a post-mortem examination which is in favor of Charcot's view,[1] while the sixteen cases I have read to you speak emphatically against it. Indeed, there is reason to believe that Prof. Charcot has never attached much value to his diagram, and I understand that he has already abandoned it, yielding, as he is ever ready to do, any theoretical views of his own to opposing pathological facts.

Grasset has recently (1883) offered a modification of Charcot's diagram, which is extravagant. He would have still a third decussation (counting the chiasmic as the first) somewhere in the callosal fibres, so that after the fibres for one whole retina, according to Charcot's schema, have passed a certain distance in the internal capsule, the fibres of the fasciculus lateralis again cross the median line, so that the visual centre receives fibres from both retinæ. This far-fetched attempt to reconcile Charcot's opinion as to the effect of lesion of one internal capsule in its caudal division, with the well-established results of lesions of the occipital lobes, is hardly deserving of serious criticism ; but it may be as well to state that more recent (1884)[2] experiments by W. Bechterew show that in dogs at least section of the posterior part of the internal capsule produces lateral hemianopsia—a result in full harmony with some of our human cases.

From his latest pathological observations von Monakow[3] draws the following conclusions as to the course of the central optic fasciculi in man :

" The collective optical bundle forms a solid tractus in the sagittal white substance of the occipital part of the brain, which passes alongside of the corpus callosum fibres or tapetum, and ends in the cortex of the occipital gyri,

[1] Petrina, in *Prager Zeitsch. f. Heilk.*, II., p. 595, case viii. *Vide* Table I.

[2] W. Bechterew : Ueber die nach Durchschneidung der Sehnervenfasern im innere der Grosshirnhemisphären, etc. *Neurol. Centralbl.*, 1884, No. 1.

[3] *Westphal's Archiv f. Psychiatrie*, xvi., 352.

more especially in that of the cuneus, lobus lingualis, and gyrus descendens."

The diagram of optic paths which I offer you is, I believe, in agreement with Munk's view of the physiology of the visual centre, with what we know of the anatomy of the optic tracts by dissection and by secondary degeneration (Monakow), and lastly, best of all, with the results of now numerous post-mortem examinations.

From the above data, pathological, anatomical, and experimental, are we now in a position to induce diagnostic laws with reference to the symptom hemianopsia? I think we are, and I would propose the following as a preliminary set of rules.

1. Lateral hemianopsia always indicates an intra-cranial lesion on the opposite side from the dark fields.

2. Lateral hemianopsia with pupillary immobility, optic neuritis or atrophy, especially if joined with symptoms of basal disease, is due to lesion of one optic tract, or of the primary optic centres on one side.

This diagnosis may be further strengthened and rendered quite certain by seeking for and finding one-sided pupillary reaction, as recently suggested by Wernicke.[1] He ingeniously predicts that only one lateral half of each iris will be found to contract by the reflex effect of light when one optic tract has been interrupted. He designates this as "hemiopic pupillary reaction."

3. Lateral hemianopsia, or sector-like defects of the same geometric order, with hemianæsthesia and choreiform or ataxic movements of one half of the body without marked hemiplegia, is probably due to lesion of the caudo-lateral part of the thalamus, or of the caudal division of the internal capsule.

4. Lateral hemianopsia, with complete hemiplegia (spastic after a few weeks) and hemianæsthesia, is probably caused by an extensive lesion of the internal capsule in its knee and caudal part.

5. Lateral hemianopsia, with typical hemiplegia (spastic

[1] Wernicke: Ueber hemiopische Pupillenreaction. *Fortschritte der Medicin*, 1883, i., 49–53.

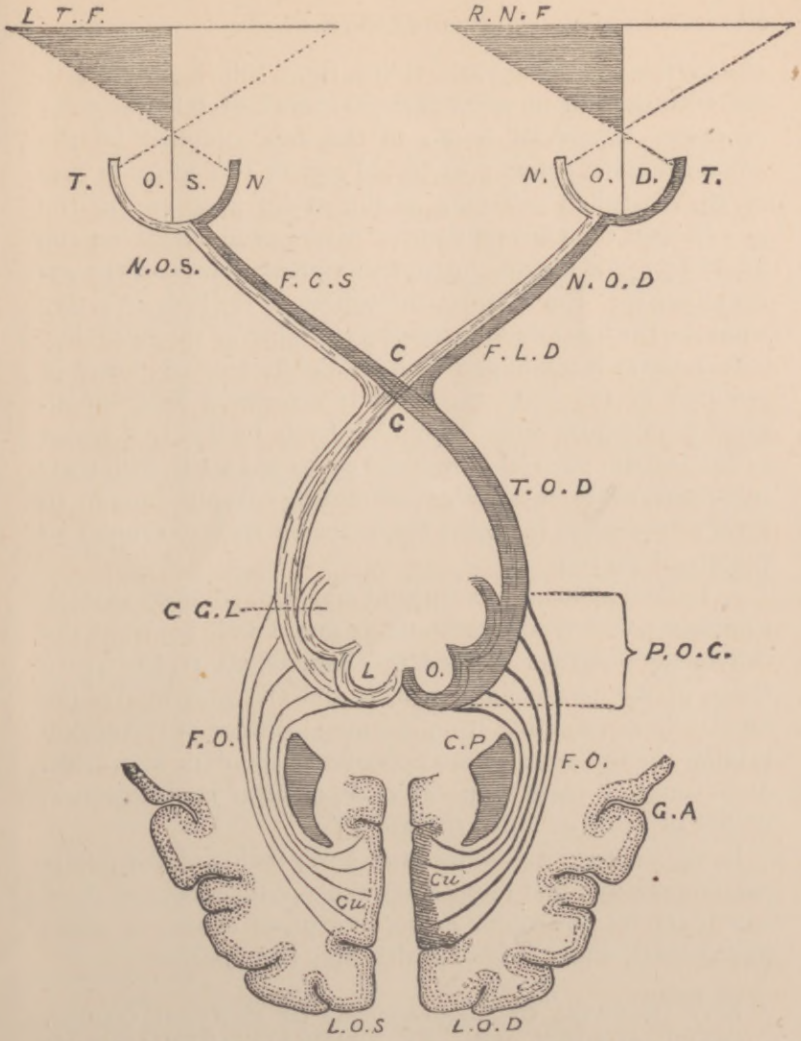

FIG. 6.—Diagram of Visual Paths ; designed to illustrate specially Left Lateral Hemianopsia from any lesion. L. T. F., left temporal half-field. R. N. F., right nasal half-field. O. S., oculus sin. O. D., oculus dexter. N. T., nasal and temporal halves of retinæ. N. O. S., nervus opticus sin. N. O. D., nervus opticus dext. F. C. S., fasciculus cruciatus sin. F. L. D., fasciculus lateralis dext. C., chiasma, or decussation of fasciculi cruciati. T. O. D., tractus opticus dext. C. G. L., corpus geniculatum laterale. L. O., lobi optici (corpus quad.). P. O. C., primary optic centres, including lobus opticus corp. genic. lat., and pulvinar of one side. F. O., fasciculus opticus (Gratiolet) in the internal capsule. C. P., cornu posterior. G. A., region of gyrus angularis. L. O. S., lobus occip. sin. L. O. D., lobus occip. dext. Cu., cuneus and subjacent gyri constituting the cortical visual centre in man. *The heavy or shaded lines represent parts connected with the right halves of both retinæ. The reader may place the lesion as he pleases.*

after a few weeks), aphasia if the right side be paralyzed, and with little or no anæsthesia, is quite certainly due to an extensive superficial lesion in the area supplied by the middle cerebral artery; we would expect to find (as in case 26, by Westphal) softening of the motor zone and of the gyri lying at the extremity of the fissure of Sylvius, viz.: the inferior parietal lobule, the supra-marginal gyrus, and the gyrus angularis. Embolism or thrombosis of the Sylvian artery would be the most likely pathological cause of the softening.

6. Lateral hemianopsia with moderate loss of power in one half of the body, especially if associated with impairment of muscular sense, would probably be due to a lesion of the inferior parietal lobule and gyrus angularis, with their subjacent white substance, penetrating deeply enough to sever or compress the optic fasciculus on its way caudad to the visual centre.

7. Lateral hemianopsia without motor, or common sensory symptoms, this symptom alone, is due, I believe, from the convincing evidence afforded by Cases 28, 29, 41, and 45, to lesion of the cuneus only, or of it and the gray matter immediately surrounding it on the mesal surface of the occipital lobe, in the hemisphere opposite to the dark half-fields. Most surgical cases come at once, or after convalescence, within this rule or in No 6 (Case 3.)

In all cases coming under rules 3 to 7 inclusive, the pupils react normally; and rarely does the ophthalmoscope show any lesion of the optic nerve, except, of course, in some tumor cases, when neuro-retinitis may be expected.

A LIST OF CASES OF HEMIANOPSIA OF CENTRAL ORIGIN, WITH AUTOPSY, AND OF TRAUMATIC HEMIANOPSIA, TO OCTOBER 30, 1885, ARRANGED IN CHRONOLOGICAL ORDER.

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