LITERATURE.

Notta. Recherches sur la perte de l'odorat. Arch. génér. de méd., April, 1870.
Ogle. Anosmia, or Cases illustrating the Physiology and Pathology of the Sense of Smell. Med.-chir. Transact., 1870, liii.
Mollière. Note pour servir à l'histoire du nerf olfactif. Lyon méd., 1871, No. 20.
Carbonieri. Zur Localisation des Centrum olfactorium. Riv. clin., xxiv, 9, p. 657, September, 1885.
Erben. Wien. med. Blätter, 1886, No. 43, 44 (kakosmia in tabes).
Moldenhauer. Die Krankheiten der Nasenhöhlen u. s. w. Leipzig, Vogel, 1886.
Gowers. Lectures on the Diagnosis of the Diseases of the Brain, delivered at University College Hospital, London. Churchill, 1885. Also Blakiston, Philadelphia, 1885.
Thudichum. On the Nature and Treatment of Hypertrophies and Tumors of the Nasal and Pharyngeal Cavities. The Lancet, August 27, 1887, p. 401.
Zuckerkandl. Ueber das Riechcentrum. Stuttgart, Enke, 1887.
Roth. Die Erkrankungen der Nasenschleimhaut, ihre Beziehungen zum übrigen Organismus und Behandlung derselben. Centralbl. für d. ges. Therapie, v, Heft x, October, 1887.
Zwaardemaker. Berliner Klinik. Reviewed in Wiener med. Presse, 1890, No. 39.
Zwaardemaker. Zur Methodik der klin. Olfactometrie. Neurol. Centralblatt, 1893, No. 21.

CHAPTER II.

DISEASES OF THE OPTIC NERVE.

THE optic nerves derive their fibers from the occipital lobes, the optic thalami, the outer and inner geniculate bodies, the anterior corpora quadrigemina, and the cerebellum (through the superior peduncle of the cerebellum).

What are known as the optic tracts before the chiasm is reached, after this point become the optic nerves. These are round hard cords, about four millimetres in diameter, which, passing in a diverging direction through the optic foramina, enter the orbits and reach the eyeballs after their passage through the orbital fat. Here they pass the sclerotic and choroid and spread themselves over the fibrous layer of the retina. The outer covering of the nerve, which is a process of the dura mater, is called the dural sheath; the process of the pia, the inner or pial sheath. The two are separated by a space which belongs to the lymphatic system, the so-called intervaginal or subvaginal space. The arteria centralis retinæ, a branch of the internal carotid, enters the optic nerve about fifteen or twenty millimetres from the eyeball and runs together with the vein of the same name in the substance of the nerve to the retina.

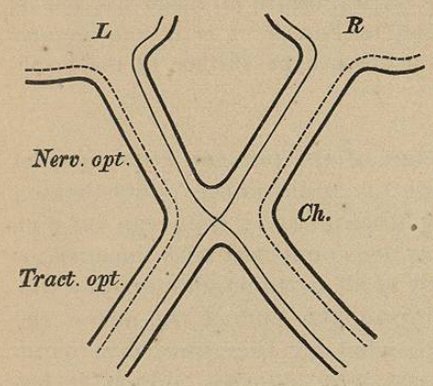

Fig. 2.—DIAGRAM SHOWING THE COURSE OF THE OPTIC FIBRES IN THE CHIASM.

The chiasm, which is formed by the union of the optic tracts, is a flattened four-sided body, in which the crossing of the optic fibers takes place. This crossing, as we now know with a fair amount of certainty, is, however, only partial, a semidecussation. The fibers from the outer half of the retina (represented by an interrupted line) pass to the center without decussating, while those of the inner half

cross over and pass to the centre of the opposite side (cf. Fig. 2). Each occipital lobe, therefore, receives fibres coming from the temporal as well as from the nasal half of the retina. Thus, for instance, the left receives fibres from the outer temporal half of the left and from the inner nasal half of the right retina. In diseases of this lobe, therefore, images falling upon the left half of the retina, or, in other words, those which lie in the right half of the field of vision, are no longer perceived—right-sided hemianopia.

The optic tract, the superficial fibres of which can be traced into the white covering of the pulvinar (the so-called stratum zonale thalami), originates by two roots—an outer, much stouter, the end ganglia of which are the anterior corpus quadrigeminum, the outer geniculate body, and the pulvinar, and by an inner root which can be easily followed to the inner geniculate body (Wernicke). These end ganglia of the optic tracts form at the same time the terminal points of certain fibres of the corona radiata, which run in a sagittal direction forward from the occipital lobe, and are connected with the pulvinar, the brachium anterius of the quadrigeminal body, and the outer geniculate body. This bundle of fibres is the sagittal medullary tract of the occipital lobe, or what is called the optic radiation, and is designated in the diagram by *s* (*vide* Fig. 3).

The exact localization of the cortical centre of vision has not as yet been established. According to Ferrier, it is in the angular gyrus; according to Munk, it is in the convex surface of the occipital lobe.

It would be beyond the scope of the present work to treat *in extenso* of those diseases of the optic nerve which belong strictly to the domain of ophthalmology; they can be considered here only so far as they are connected with the nervous system. To these belong, first of all, certain inflammatory conditions which act upon the intraocular end of the nerve, the papilla (disk), and give rise to what we therefore term papillitis (choked disk). The name optic neuritis, which is frequently used as a synonym for papillitis, is inexact, because it may imply an affection of the whole nerve trunk.

The papillitis, choked disk (*Stauungspapille*, as the Germans call it, after von Graefe, 1859), is frequently, although not always, met with in cases of intracranial tumors, and is (according to von Graefe) to be attributed to a high grade of venous engorgement, produced by an impediment to the reflux of the venous blood into the skull cavity. Later, when Schwalbe had discovered that there was a communication between the fluid

contents of the skull and the intervaginal space of the optic nerve, it was shown that the subdural space was distended with a serous inflammatory fluid, and that the optic nerve at its passage through the lamina cribrosa of the sclerotic becomes compressed (Schmidt-Rimpler). Finally, Deutschmann (cf. lit.)

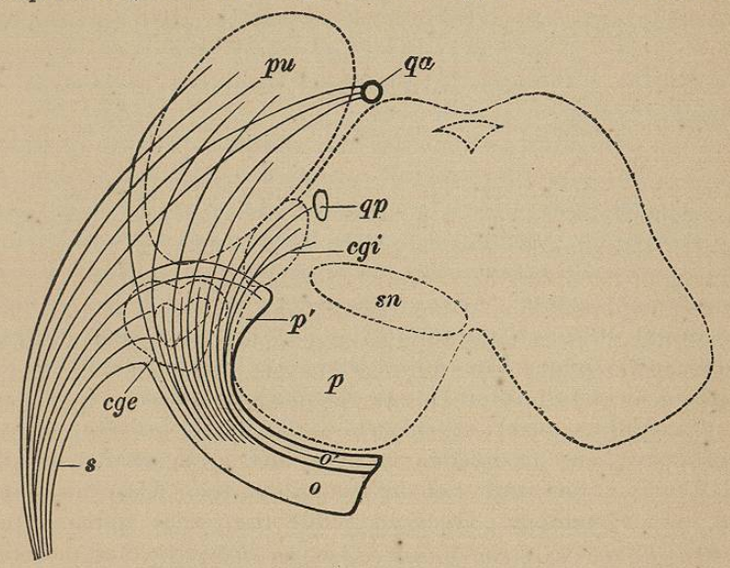

Fig. 3.—Diagram showing the Origin of the Optic Nerve. (After Wernicke.) *p*, crusta of the crus cerebri; *sn*, substantia nigra; *cgi*, inner, *cge*, outer geniculate body; *qp*, brach. post. corp. quadr.; *qa*, brach. ant. corp. quad.; *pu*, pulvinar; *s*, optic radiation.

has put forth the view that papillitis is not caused by mechanical influences, but that it is due to the action of pathogenic organisms which enter from outside. How far this view is correct further experience will show. Besides the pure papillitis there is also found a papillo-retinitis, the ophthalmoscopic picture of which differs from that of the former affection, and which is to be referred to a meningitis, which has advanced along the sheath of the optic nerve.

A pure papillitis, as we have said, is chiefly found in intracranial tumors. Patients in whom a brain tumor is suspected ought to be examined for choked disk even if they do not complain of any subjective symptoms pointing to it, because sight may, even if the disk is markedly swollen, remain normal for a long time. Only when the nerve or the chiasm is strongly compressed does amblyopia or amaurosis occur in the early stages.

The seat of the tumor has nothing to do with the occurrence of papillitis. Basal neoplasms can, through direct pressure upon the optic nerve, cause a simple atrophy of the same. Nor does the nature of the tumor play any part here. Gummata, tubercles, entozoa (cysticerci, echinococci), carcinomata, gliomata—any one of these may produce a papillitis, which is usually bilateral (in ninety-three per cent of the cases, Annuske and Reich), although the processes need not necessarily be equally developed in both eyes.

Of practical importance are the sudden spells of blindness which occur sometimes in the course of a papillitis, termed by H. Jackson epileptiform amaurosis. They are probably due to a temporary swelling of certain tumors and the consequent compression of certain areas of the brain or the vessels (Leber) distributed to them. These attacks may last for hours or days, and either disappear completely or leave a permanent increase in the amblyopia. The ophthalmoscopic examination does not teach us anything about this periodical blindness.

A papillitis rarely ever gets well; in by far the greater number of cases a papillitic atrophy and total amaurosis take place, first in one and then in the other eye. Cases in which one eye is seriously damaged while the other remains perfectly well are extremely rare. I have, however, had occasion to observe an instance of this with Magnus. More frequently both eyes become diseased, one soon after the other. Dropsy of the ventricles may give rise to a simultaneous amaurosis of both eyes.

Papillo-retinitis is not very rare in tubercular basilar meningitis; in epidemic cerebro-spinal meningitis it is exceptional. Chronic cerebral affections of children often lead to it, the amaurosis in these instances usually developing quickly, while the general symptoms become intensified.

Inflammations of the optic-nerve trunk occurring alone may be caused by cold, febrile diseases, syphilis, disturbances in menstruation, and hereditary influences. On ophthalmoscopic examination either nothing remarkable or only a slight blurring of the disk is recognizable, because the inflammation affects more especially that part of the nerve which is behind the eyeball (retrobulbar neuritis of von Graefe). The disturbance of vision usually begins gradually, and is confined either to the periphery of the field of vision or it consists of a central amblyopia or a circumscribed central amaurosis. It does not terminate in complete blindness; frequently only marked disturbance of color vision remains.

To the neurologist the cases of optic neuritis in patients with a neuropathic family history are of extreme interest. Such persons usually suffer even in early youth from migraine, nervous palpitation of the heart, vertigo, sometimes also from epileptiform attacks. Between the ages of twenty and thirty they begin to complain of trouble with their sight, either of subjective light or color sensations or else that objects appear to them enveloped in a dense mist; within from four to six weeks they may become completely blind, but their blindness as a rule does not persist, but gives place to a central amblyopia with normal sight at the periphery of the field of vision. The prognosis differs markedly in different families. It is of interest to note that as a rule only the male members of the family are wont to be affected by the disease.

In the second place we will consider atrophy of the optic nerve. It consists in a wasting of the nerve elements, and may be either primary (genuine) or inflammatory, the consequence of a previous neuritis. It may also affect the trunk of the nerve as well as the intraocular end of it. If the nerve, besides the wasting of its pulp, also undergoes a diminution in its volume, so that it appears like a gelatinous grayish-yellow cord, the atrophy is known as gray degeneration.

Tumors and inflammatory exudates, as well as splinters of bone, may by pressure, by shutting off the blood supply (as, for instance, in embolism of the arteria centralis retinæ), and through interference with the nutrition lead to atrophy.

The progressive atrophy, or, as it is better termed, progressive gray degeneration, which may be of cerebral or spinal origin, is characterized clinically by a diminution in the acuteness of the central vision, a contraction of the whole visual field, and disturbance of the color sense. In the ophthalmoscopic examination the bluish-white discoloration of the disk and the atrophic excavation of the nerve (due to wasting of the substance of the disk) are very apparent. The acuteness of vision, grows gradually but progressively less, and months and years may pass before complete amaurosis is developed. On the other hand, the whole process may run its course in two or three weeks. The contraction of the field of vision is rarely concentric; usually the defects are in one direction only, and are often sectorial (Leber). Enormous contraction of both fields of

vision, with at the same time normal acuteness of sight in the center, which was eventually followed by blindness, has been observed by Schweigger. The disturbance in color vision is at first limited to the perception of green, which is confused with white or gray, the perception of blue and yellow being relatively longest retained. The atrophy develops bilaterally, although one eye alone may at first be affected, and the other eye remain intact for years.

Foci of softening in the brain, progressive paralysis of the insane, sometimes also epilepsy, are the cerebral diseases in which the affection is not rarely observed. It is besides also noted in multiple sclerosis, although in this disease it never leads to total amaurosis, a fact which Charcot was in the habit of emphasizing in his lectures.

More important is the fact that in locomotor ataxia optic atrophy is comparatively frequent. Wharton Jones (British Medical Journal, July 24, 1869) makes the sympathetic responsible for this, assuming that the paralysis of the vaso-motor nerves, producing first hyperæmia, leads finally to atrophy of the optic nerve. This explanation, however, is at once overthrown by the fact that in the optic atrophy of tabes there are at no time any traces of hyperæmia.

Congenital optic atrophy can sometimes be traced to hereditary influences, or to consanguinity of the parents; several cases have been known to occur in the same family without apparent cause (Nicolai, Nederl. Weekbl., 1890, i, 5); sometimes it is due to hydrocephalus. Injury to the skull in consequence of instrumental interference at birth very rarely has anything to do with it.

The diseases of the chiasm and optic tract may be considered together, since they possess one symptom in common which is of special interest to the neurologist, viz., hemianopia. It is the only form of visual disturbance where one can with certainty diagnosticate a central affection of the optic nerve. It is likely to be of cortical origin if the hemianopia occurs suddenly as the only symptom, there being no change to be found on ophthalmoscopic examination; whereas if other symptoms accompany it—aphasia, hemiplegia, etc.—this idea of a cortical lesion must be given up. By hemianopia in general we mean a loss of one half (the right or the left) of the field of vision, so that patients affected with right-sided hemianopia see the objects which are in the left half of their visual field, whereas

those to the right are not perceived. If the disturbance affects the halves on the same side of both eyes—that is, the nasal on the one, the temporal on the other—we call it a homonymous hemianopia. If in both fields the temporal halves are lost, this constitutes what is known as temporal hemianopia, which is of rarer occurrence; the absence of both nasal halves of the field of vision does not seem to occur, and the superior and inferior hemianopia, where the line of division is not vertical but horizontal, seems to be extremely rare.

The explanation of the hemianopia in lesions of the cortical center for sight is quite evident if we accept, as is now generally done, the existence of the above-described semidecussation of the fibres in the chiasm. The path from the optic tract to the cortex of the occipital lobe may be divided into the following segments (Wernicke): The first includes the optic radiation in the occipital lobe, the lesions of which give rise to homonymous hemianopia without any other focal symptoms, lesions of the right occipital lobe causing left-sided, those of the left right-sided, hemianopia; the second will include the place where the fibres of the optic radiation enter the internal capsule, and the ganglia of origin of the optic tract, the pulvinar, and the outer geniculate body—hemianopia and hemianæsthesia; the third will include the optic tract in its course at the base of the brain—hemianopia with hemiplegia. If in the region of the visual center or the optic radiation a bilateral focal lesion occurs, then we may have complete blindness setting in with an apoplectiform attack. This is in reality a bilateral hemianopia, and is designated cortical blindness. The function in the two halves of both eyes need not be totally lost; atrophy of the optic nerve does not take place. Weir Mitchell has shown that a lesion of the chiasm may produce bilateral hemianopia; his case was one in which an aneurism pressed upon the chiasm (Journal of Nervous and Mental Diseases, January, 1889).

Of diagnostic value in these cases is sometimes the so-called hemianopic pupillary reaction (Heddaeus, Wernicke), or hemianopic inactivity of the pupil (Leyden). With the mirror of the ophthalmoscope we reflect the light first upon the left, then upon the right half of the retina, and observe the pupillary reaction. If the reflex occurs normally, the optic tract must be intact, and the disturbance must be due to a bilateral lesion of the optic radiation in the occipital lobe, or in the cortical cen-