neuroma usually contains nerve fibres only, or in rare instances ganglion cells. Cases of ganglionic or medullary neuroma are extremely rare; some of them, as Lancereaux suggests, are undoubtedly instances of malformation of the brain substance. In other instances, as in the case which I reported,* the tumor is, in all probability, a glioma with cells closely resembling those of the central nervous system. The true fascicular neuroma occurs in the form of the small subcutaneous painful tumor—*tubercula dolorosa*—which is situated on the nerves of the skin about the joints, sometimes on the face or on the breast. It is not always made up of nerve fibres, but may be, as shown by Hoggan, an adenomatous growth of the sweat glands.

The true neuromata, as a rule, are not painful, and occasionally are found associated with the nerve fibres in various regions. Those which develop at the ends and along the course of the nerves of the stump after amputation consist of connective tissue and of medullated and non-medullated nerve fibres. The most remarkable form is the *plexiform neuroma*, in which the various nerve cords are occupied by many hundreds of tumors. The cases are usually congenital. The tumors occur in all the nerves of the body. One of the most remarkable is that described by Prudden, the specimens of which are in the medical museum of Columbia College, New York. There were over 1,182 distinct tumors distributed on the nerves of the body. Prudden † has collected forty-one cases from the literature, in a majority of which the peripheral nerves were affected.

Neuromata rarely cause symptoms, except the subcutaneous painful tumor or those in the amputation stump. Here they may be very painful and cause great distress. Motor symptoms are sometimes present, particularly a constant twitching. Epilepsy has sometimes been associated, and relief has followed removal of the growths.

The only available treatment is excision. The subcutaneous painful tumor does not return, and excision completely relieves the symptoms. On the other hand, the amputation neuromata may recur.

III. DISEASES OF THE CRANIAL NERVES.

I. Olfactory Nerve.

The functions of this nerve may be disturbed at its peripheral ending, at the bulb, in the course of the nerve, or at the central origin in the brain. The disturbances may be manifested in subjective sensations of smell, complete loss of the sense, and occasionally in hyperæsthesia.

(*a*) *Subjective Sensations; Parosmia.*—Hallucinations of this kind are found in the insane and in epilepsy. The aura may be represented by an

* Journal of Anatomy and Physiology, vol. xv.

† American Journal of the Medical Sciences, vol. lxxx.

unpleasant odor, described as resembling chloride of lime, burning rags, or feathers. In a few cases with these subjective sensations tumors have been found in the hippocampal lobules. In rare instances, after injury of the head the sense is perverted—odors of the most different character may be alike, or the odor may be changed, as in a patient noted by Morell Mackenzie, who for some time could not touch cooked meat, as it smelt to her exactly like stinking fish.

(*b*) *Increased sensitiveness*, or *hyperosmia*, occurs chiefly in nervous, hysterical women, in whom it may sometimes be developed so greatly that, like a dog, they can recognize the difference between individuals by the odor alone.

(*c*) *Anosmia; Loss of the Sense of Smell.*—This may be produced by: (1) Affections of the termination of the nerve in the mucous membrane, which is perhaps the most frequent cause. It is by no means uncommon in association with chronic nasal catarrh and polypi. In paralysis of the fifth nerve, the sense of smell may be lost on the affected side, owing to interference with the secretion.

It is doubtful whether the cases of loss of smell following the inhalations of very foul or strong odors should come under this or under the central division.

(2) The lesions of the bulb or of the nerves. In falls or blows, in caries of the bones, and in meningitis or tumor, the bulbs or the nerve trunks may be involved. After an injury to the head the loss of smell may be the only symptom. Mackenzie notes a case of a surgeon who was thrown from his gig and lighted on his head. The injury was slight, but the anosmia which followed was persistent. In locomotor ataxia the sense of smell may be lost, due possibly to atrophy of the nerves.

(3) Lesions of the olfactory centre. There are congenital cases in which the nerve structures have not been developed. Cases have been reported by Beevor, Hughlings Jackson, and others, in which this symptom has been associated with disease in the hemisphere. The centre for the sense of smell is placed by Ferrier in the uncinate gyrus.

To test the sense of smell the pungent bodies, such as ammonia, which act upon the fifth nerve, should not be used, but such substances as cloves, peppermint, and musk. This sense is readily tested as a routine matter in brain cases by having two or three bottles containing the essential oils. In all instances a rhinoscopical examination should be made, as the condition may be due to local, not central causes. The *treatment* is unsatisfactory even in the cases due to local lesions in the nostrils.

II. Optic Nerve and Tract.

(1) *Lesions of the Retina.*

These are of importance to the physician, and information of the greatest value may be obtained by a systematic examination of the eye-

grounds. Only a brief reference can here be made to the more important of the appearances.

(*a*) **Retinitis.**—This occurs in certain general affections, more particularly in Bright's disease, syphilis, leukæmia, and anæmia. The common feature in all these states is the occurrence of hæmorrhage and the development of opacities. There may also be a diffuse cloudiness due to effusion of serum. The hæmorrhages are in the layer of nerve fibres. They vary greatly in size and form, but often follow the course of vessels. When recent the color is bright red, but they gradually change and old hæmorrhages are almost black. The white spots are due either to fibrinous exudate or to fatty degeneration of the retinal elements, and occasionally to accumulation of leucocytes or to a localized sclerosis of the retinal elements. The more important of the forms of retinitis to be recognized are:

Albuminuric retinitis, which occurs in chronic nephritis, particularly in the interstitial or contracted form. The percentage of cases affected is from fifteen to twenty-five. There are instances in which these retinal changes are associated with the granular kidney at a stage when the amount of albumen may be slight or transient; but in all such instances it will be found that there is a marked arterio-sclerosis. Gowers recognizes a degenerative form (most common), in which, with the retinal changes, there may be scarcely any alteration in the disk; a hæmorrhagic form, with many hæmorrhages and but slight signs of inflammation; and an inflammatory form, in which there is much swelling of the retina and obscuration of the disk. It is noteworthy that in some instances the inflammation of the optic nerve predominates over the other retinal changes and one may be in doubt for a time whether the condition is really associated with the renal changes or dependent upon intracranial disease.

Syphilitic Retinitis.—In the acquired form this is less common than choroiditis. In inherited syphilis *retinitis pigmentosa* is sometimes met with.

Retinitis in Anæmia.—It has long been known that a patient may become blind after a large hæmorrhage, either suddenly or within two or three days, and in one or both eyes. Occasionally the loss may be permanent and complete. In some of these instances a neuro-retinitis has been found, probably sufficient to account for the symptoms. In the more chronic anæmias, particularly in the pernicious form, retinitis is common, as determined first by Quincke.

In *malaria* retinitis or neuro-retinitis may be present, as noted by Stephen Mackenzie. It is seen only in the chronic cases with anæmia, and in my experience is not nearly so common proportionately as in pernicious anæmia. Of many instances which have come under my observation of severe malarial anæmia, particularly at the Philadelphia Hospital, there were only two with retinal hæmorrhages.

Leukæmic Retinitis.—In this affection the retinal veins are large

and distended; there is also a peculiar retinitis, as described by Liebreich. It is not very common. Of the seventeen cases of leukæmia which have come under my observation, retinitis existed in only three of the ten in which the eye-grounds were examined. There are numerous hæmorrhages and white or yellow areas, which may be large and prominent. In one of my cases the retina post mortem was occupied by many small, opaque, white spots, looking like little tumors, the larger of which had a diameter of nearly two millimetres. In Case 13 of my series the leukæmia was diagnosed by Norris and De Schweinitz, at whose clinic the patient had applied on account of failing vision, from the condition of the eye-grounds alone.

Retinitis is also found occasionally in diabetes, in purpura, in chronic lead poisoning, and sometimes as an idiopathic affection.

(*b*) **Functional Disturbances of the Retina.** (1) *Toxic Amaurosis.*—This occurs in uræmia and may follow convulsions or come on independently. Thus, a patient who had become suddenly blind the previous day, was led into one of my wards at the Montreal General Hospital. He had had no special symptoms, but examination showed extensive cardio-vascular changes. The urine was albuminous. The ophthalmoscopic examination was negative. The condition, as a rule, persists only for a day or two. This form of amaurosis occurs in poisoning by lead and occasionally by quinine. It seems more probable that the poisons act on the centres and not on the retina.

(2) *Hysterical Amaurosis.*—More frequently this is loss of acuteness of vision—amblyopia—but the loss of sight in one or both eyes may apparently be complete. The condition will be mentioned subsequently under hysteria.

(3) *Tobacco Amblyopia.*—The loss of sight is usually gradual, equal in both eyes, and affects particularly the centre of the field of vision. The eye-grounds may be normal, but occasionally there is congestion of the disks. On testing the color fields a central scotoma for red and green is found in all cases. Ultimately, if the use of tobacco is continued, organic changes may develop with atrophy of the disk.

(4) *Night-blindness—nyctalopia*—the condition in which objects are clearly seen during the day or by strong artificial light, but become invisible in the shade or in twilight, and *hemeralopia*, in which objects cannot be clearly seen without distress in daylight or in a strong artificial light, but are readily seen in a deep shade or in twilight, are functional anomalies of the retina which rarely come under the notice of the physician.

(5) *Retinal hyperæsthesia* is sometimes seen in hysterical women, but is not found frequently in actual retinitis. I have seen it once, however, in albuminuric retinitis and once, in a marked degree, in a patient with aortic insufficiency, in whose retinæ there were no signs other than the throbbing arteries.

(2) *Lesions of the Optic Nerve.*

(*a*) **Optic Neuritis** (*Papillitis*; *Choked Disk*).—In the first stage there is congestion of the disk and the edges are blurred and striated. In the second stage, the congestion is more marked, the swelling increases, the striation also is more visible. The physiological cupping disappears and hæmorrhages are not uncommon. The arteries present little change, the veins are dilated, and the disk may swell greatly. In slight grades of inflammation the swelling gradually subsides and occasionally the nerve recovers completely. In instances in which the swelling and exudate are very great, the subsidence is slow, and when it finally disappears there is complete atrophy of the nerve. The retina not infrequently participates in the inflammation, which is then a neuro-retinitis.

This condition is of the greatest importance in diagnosis. It may exist in its early stages without any disturbance of vision, and even with extensive papillitis the sight may for a time be good.

Optic neuritis is seen occasionally in anæmia and lead poisoning, more commonly in Bright's disease as neuro-retinitis. It occurs occasionally as a primary idiopathic affection. The frequent connection with intracranial disease, particularly tumor, makes its presence of great value to practitioners. The nature of the growth is without influence. In over ninety per cent of such instances the papillitis is bilateral. It is also found in meningitis, either the tuberculous or the simple form. In meningitis it is easy to see how the inflammation may extend down the nerve sheaths. In the case of tumor it was thought at first that a choked disk resulted from increased pressure within the skull. It is now more commonly regarded, however, as a descending neuritis.

(*b*) **Optic Atrophy.**—This may be: (1) A primary affection. Some of the cases have been hereditary and have come on in all the males of a family shortly after puberty. A large number of the cases of primary atrophy are associated with spinal disease, particularly locomotor ataxia. Other causes which have been assigned for the primary atrophy are cold, sexual excesses, diabetes, the specific fevers, alcohol, and lead.

(2) Secondary atrophy results from cortical lesions, pressure on the chiasma or on the nerves, or, most commonly of all, is a sequence of papillitis.

The ophthalmoscopic appearances are different in the cases of primary and secondary atrophy. In the former, the disk has a gray tint, the edges are well defined, and the arteries look almost normal; whereas in the consecutive atrophy the disk has a staring opaque-white aspect, with irregular outlines, and the arteries are very small.

The symptom of optic atrophy is loss of sight, proportionate to the damage in the nerve. The change is in three directions: "(1) Diminished acuity of vision; (2) alteration in the field of vision; and (3) altered perception of color." (Gowers.) The outlook in primary atrophy is bad; the

majority of cases go on to complete blindness. In the consecutive form there is greater chance of retention of slight vision.

(3) *Affections of the Chiasma and Tract.*

At the chiasma the optic nerves undergo partial decussation. Each optic tract, as it joins the chiasma, contains nerve fibres which supply half of the retina of either eye. Thus, of the fibres of the right tract, part pass the chiasma without decussating and supply the temporal half of the right retina, the other and larger portion of the fibres of the tract decussate in the chiasma and join the left optic nerve, supplying the nasal half of the retina on the other side. The fibres which cross are in the middle portion of the chiasma, while the direct fibres are on each side. The following are the most important changes which ensue in lesions of the tract and of the chiasma:

(*a*) *Unilateral Affection of Tract* (Fig. 1 *B*).—If right this produces loss of function in the temporal half of the retina on the right side, and on the nasal half of the retina on the left side, so that there is only half vision, and the patient is blind to objects on the left side. This is termed homonymous hemianopia or lateral hemianopia. The fibres passing to the right half of each retina being involved, necessarily the left half of each visual field is blind. The hemianopia may be partial and only a portion of the half field may be lost. The affected visual fields may have the normal extent, but in some instances there is considerable reduction. The color vision is, as a rule, lost in the half field—hemiachromatopia—but the half vision for color may be lost in central disease without any change in the field for white. When the left half of one field and the right half of the other, or vice versa, are blind, the condition is known as heteronymous hemianopia.

(*b*) *Disease of the Chiasma.*—(1) A lesion involves, as a rule, chiefly the central portion, in which the decussating fibres pass which supply the inner or nasal halves of the retinæ, producing in consequence loss of vision in the outer half of each field, or what is known as temporal hemianopia (Fig. 1 *H*).

(2) If the lesion is more extensive it may involve not only the central portion, but also the direct fibres on one side of the commissure, in which case there would be total blindness in one eye and temporal hemianopia in the other.

(3) Still more extensive disease is not infrequent from pressure of tumors in this region, the whole chiasma is involved, and total blindness results. The different stages in the process may often be traced in a single case from temporal hemianopia, then complete blindness in one eye with temporal hemianopia in the other, and finally complete blindness.

(4) A limited lesion of the outer part of the chiasma involves only the direct fibres passing to the temporal halves of the retinæ and inducing

blindness in the nasal field, or, as it is called, nasal hemianopia. This, of course, is extremely rare.

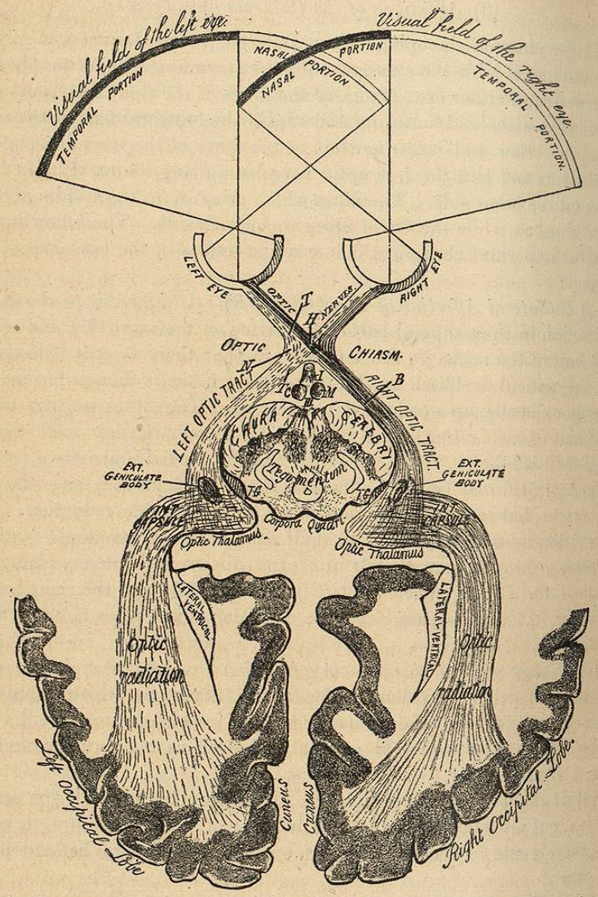

FIG. 1.—THE OPTIC AND VISUAL TRACTS (Starr). *N*, Lesion causing nasal hemianopia. *T*, Lesions causing temporal hemianopia. *H*, Lesion causing bilateral heteronymous hemianopia. *B*, Lesion of tract causing homonymous hemianopia.

(4) *Affections of the Tract and Centres.*

The optic tract crosses the crus to the hinder part of the optic thalamus and divides into two portions, one of which goes to the thalamus and external geniculate bodies and to the anterior quadrigeminal bodies. From

these parts fibres pass into the posterior part of the internal capsule and enter the occipital lobe, forming the fibres of the optic radiation (Fig. 1), which terminate in and about the cuneus, the region of the visual perceptive centre. The fibres of the other division of the tract pass to the internal geniculate bodies and to the posterior quadrigeminal body. It is still held by some physiologists that the cortical visual centre is not confined to the occipital lobe alone, but embraces the occipito-angular region.

A lesion of the fibres of the optic tract anywhere between the cortical centre and the chiasma will produce lateral hemianopia. The lesion may be situated: (*a*) In the tract itself. (*b*) In the region of the thalamus and the corpora quadrigemina, into which the larger part of each tract enters. (*c*) A lesion of the fibres passing from the corpora quadrigemina to the occipital lobe. This may be either in the hinder part of the internal capsule or the white fibres of the optic radiation. (*d*) Lesion of the cuneus. Bilateral disease of the cuneus may result in total blindness. (*e*) There is clinical evidence to show that lesion of the angular gyrus may be associated with visual defect, not so often hemianopia as crossed amblyopia, dimness of vision in the opposite eye, and great contraction in the field of vision. Lesions in this region are associated with mind blindness, a condition in which there is failure to recognize the nature of objects.

The effects of lesions in the optic nerve in different situations from the retinal expansion to the brain cortex are as follows: (1) Of the optic nerve —total blindness of the corresponding eye; (2) of the optic chiasma, either temporal hemianopia, if the central part alone is involved, or nasal hemianopia, if the lateral region of each chiasma is involved; (3) lesion of the optic tract between the chiasma and the geniculate bodies, produces lateral hemianopia; (4) lesion of the central fibres of the nerve between the geniculate bodies and the cerebral cortex produces lateral hemianopia; (5) lesion of the cuneus causes lateral hemianopia; and (6) lesion of the angular gyrus may be associated with hemianopia, sometimes crossed amblyopia, and the condition known as mind blindness.

Diagnosis.—The student or practitioner must have a clear idea of the physiology of the nerve centres before he can appreciate the symptoms or undertake the diagnosis of lesions of the optic nerve. Having determined the presence of hemianopia, the question arises as to the situation of the lesion, whether in the tract between the chiasma and the geniculate bodies or in the central portion of the fibres between these bodies and the visual centres. This can be determined in some cases by the test known as Wernicke's *hemiopic pupillary inaction*. The pupil reflex depends on the integrity of the retina or receiving membrane, on the fibres of the optic nerve and tract which transmit the impulse, and the nerve centre in the geniculate bodies which receives the impression and transmits it to the third nerve along which the motor impulses pass to the iris. If a bright

light is thrown into the eye and the pupil reacts, the integrity of this reflex arc is demonstrated. It is possible in cases of lateral hemianopia so to throw the light into the eye that it falls upon the blind half of the retina. If when this is done the pupil contracts, the indication is that the reflex arc above referred to is perfect, by which we mean that the optic nerve fibres from the retinal expansion to the centre, the centre itself, and the third nerve are uninvolved. In such a case the conclusion would be justified that the cause of the hemianopia was central; that is, situated behind the geniculate bodies, either in the fibres of the optic radiation or in the visual cortical centres. If, on the other hand, when the light is carefully thrown on the hemiopic half of the retina, the pupil remains inactive, the conclusion is justifiable that there is interruption in the path between the retina and the geniculate bodies, and that the hemianopia is not central, but dependent upon a lesion situated in the tract. This test of Wernicke's is sometimes difficult to obtain. It is best performed as follows: "The patient being in a dark or nearly dark room with the lamp or gas-light behind his head in the usual position, I bid him look over to the other side of the room, so as to exclude accommodative iris movements (which are not necessarily associated with the reflex). Then I throw a faint light from a plane mirror or from a large concave mirror held well out of focus upon the eye and note the size of the pupil. With my other hand I now throw a beam of light, focussed from the lamp by an ophthalmoscopic mirror, directly into the optical centre of the eye; then laterally in various positions, and also from above and below the equator of the eye, noting the reaction at all angles of incidence of the ray of light." (Seguin.)

The significance of hemianopia varies. There is a functional hemianopia associated with migraine and hysteria. In a considerable proportion of all cases there are signs of organic brain-disease. Hemiplegia is common and the loss of power and blindness are on the same side. Thus, a lesion in the left hemisphere involving the motor tract produces right hemiplegia, and when the fibres of the optic radiation are involved in the internal capsule, there is also left lateral hemianopia, so that objects in the field of vision to the right are not perceived. Hemianæsthesia is not uncommon, owing to the close association of the sensory and visual tracts at the posterior part of the internal capsule. Certain forms of aphasia also occur in many of the cases.

III. Motor Nerves of the Eyeball.

Third Nerve.—Arising from the floor of the aqueduct of Sylvius, the nerve passes through the crus at the side of which it emerges. Passing along the wall of the cavernous sinus, it enters the orbit through the sphenoidal fissure and supplies, by its superior branch, the levator palpebræ superioris and the superior rectus, and by its inferior branch the internal and inferior recti muscles and the inferior oblique. It also supplies the ciliary muscle and the constrictor of the iris. Lesions may affect the centre or the nerve in its course and cause either paralysis or spasm.

Paralysis.—A nuclear lesion is usually associated with the disease of the centres for the other eye muscles, producing a condition of general ophthalmoplegia. More commonly the nerve itself is involved in its course, either by meningitis, gummata, or aneurism, or is attacked by neuritis, as in diphtheria and locomotor ataxia. Complete paralysis of the third nerve is accompanied by the following symptoms:

Paralysis of all the muscles, except the superior oblique and external rectus, by which the eye can be moved outward and a little downward and inward. There is divergent strabismus. There is ptosis or drooping of the upper eyelid, owing to paralysis of the levator palpebræ. The pupil is of medium size. It does not contract to light, and the power of accommodation is lost. The most striking features of this paralysis are the external strabismus, with diplopia or double vision, and the ptosis. In very many cases the affection of the third nerve is partial. Thus the levator palpebræ and the superior rectus may be involved together, or the ciliary muscles and the iris may be affected and the external muscles may escape.

There is a remarkable form of recurring oculo-motor paralysis affecting chiefly women, and involving all the branches of the nerve. In some cases the attacks have come on at intervals of a month; in others a much longer period has elapsed. The attacks may persist throughout life. They are sometimes associated with pain in the head and sometimes with migraine. Mary Sherwood has collected from the literature twenty-three cases.

Ptosis is a common and important symptom in nervous affections. We may here briefly refer to the conditions under which it may occur: (*a*) A congenital, incurable form, which is frequently seen; (*b*) the form associated with definite lesion of the third nerve, either in its course or at its nucleus. This may come on with paralysis of the superior rectus alone or with paralysis of the internal and inferior recti as well. (*c*) There are instances of complete or partial ptosis associated with cerebral lesions without any other branch of the third nerve being paralyzed. The position of the cortical centre is as yet unknown. (*d*) Hysterical ptosis, which is double and occurs with other hysterical symptoms. (*e*) Sympathetic or pseudo-ptosis is associated with symptoms of vaso-motor palsy, such as elevation of the temperature on the affected side with redness and œdema of the skin. Contraction of the pupil exists on the same side and the eyeball appears rather to have shrunk into the orbit. (*f*) In idiopathic muscular atrophy, when the face muscles are involved, there may be marked bilateral ptosis. And, lastly, in weak, deli-