IV. **Progressive Toxæmias.** Chronic Renal Disease.—Headache, vomiting and optic neuritis occur and the symptoms may closely simulate those of intracranial tumour. The late appearances of papillœdema closely simulate those of albuminuric retinitis. In such cases extensive focal softenings of the cerebrum may occur *gradually*, simulating the focal march of a cerebral tumour. High blood pressure is common in both conditions. In these cases it is often extremely difficult to make the diagnosis. A very high blood-urea reading is characteristic of renal disease.

Industrial Chemical Poisoning (*e.g.*, lead encephalopathy, § 553) may simulate intracranial tumour with its headache, vomiting and optic neuritis. Epileptiform fits may occur. The diagnosis is sometimes difficult. The poisonous substance may be demonstrated in abnormal concentration in the blood or excreta.

§ **830.** V. **Acquired Hydrocephalus** is usually the result of meningitis or neoplasm. When secondary to meningitis, there is usually much meningeal scarring and adhesions are present in the roof of the fourth ventricle and tentorium, round the veins of Galen. There is persistent headache, vomiting, mental impairment, convulsions and sometimes, papillœdema or optic atrophy. In children enlargement of the head is a striking feature, and in young children the sutures separate with bulging of the fontanelles. There is marked general wasting. Neoplasms, which cause hydrocephalus, are commonly situated in some part of the brain-stem from the third ventricle to the medulla. In such cases, X-rays may show enlargement of the pituitary fossa from distension of the infundibulum. Such a finding should not lead one to the diagnosis of pituitary tumour unless neighbourhood symptoms are present.

Hydrocephalus from stenosis of the Aqueduct of Sylvius may be *Congenital* and here the presenting symptom is enlargement of the cranium. Later mental failure, fits, ataxia, spastic weakness of the limbs, nystagmus and optic atrophy may occur. The *diagnosis* from other causes of enlargement of the head may be difficult. In most cases hydrocephalus is slowly progressive and uninfluenced by treatment. A few cases survive to adult life with their faculties more or less impaired.

GROUP XIII. THE CRANIAL NERVES AND SPECIAL SENSES

The investigation of the cranial nerves and special senses is of great importance, from the standpoint both of general medicine and neurology. A tabular statement of the cranial nerve-functions will be found in Table LIX. The applied physiological anatomy of the special senses and cranial nerves has been considered in §§ 675 to 678 and § 683, while the methods of examination have been described in § 703.

§ **831.** The **Olfactory Nerve** (§ 675) consists of fibres which arise in the upper nasal mucosa, penetrate the cribriform plate of the ethmoid bone, and terminate in the olfactory bulbs. The methods of examination are described in § 703. *Anosmia* is loss of smell. Bilateral anosmia may arise from local disease in the nose (§ 178) and is not necessarily of neurological significance. It not uncommonly follows cerebral

TABLE LIX.—CRANIAL NERVES AND THEIR FUNCTIONS

Cranial Nerves.	*Functions.*
I. Olfactory nerve.	Smell.
II. Optic nerve.	Sight. Afferent for pupillary light reflex.
III. Oculo-motor.	Supplies all the extrinsic muscles of the eyeball (except the superior oblique and external rectus) and the levator palpebræ superioris; also the sphincter pupillæ and ciliary muscle.
IV. Trochlear.	Supplies the superior oblique; turns the eye down and outwards.
VI. Abducens.	Supplies the external rectus; turns the eye outwards.
V. Trigeminal nerve. First division, Ophthalmic.	*Sensory* to forehead and part of vertex, anterior part of nose to tip, upper eyelid and temple, eyeball and lachrymal gland. Contains dilator pupillæ fibres from sympathetic.
Second division, Maxillary.	*Sensory* to cheek, lower eyelid, side of nose and upper lip; the upper teeth and gum; lining membrane of nose, roof of mouth, soft palate, tonsils and roof of pharynx. *Taste* of anterior two-thirds of tongue (through Meckel's ganglion by chorda tympani nerve). *Trophic* and vaso-motor fibres.
Third division, Mandibular.	*Sensory* to lower part of face, lower lip, side of head, ear, tongue lower teeth, gum, and inner side of cheek. *Motor* to masticatory muscles, temporal, masseter, pterygoids, anterior belly of digastric and mylo-hyoid, tensor tympani and? tensor palati. *Taste* of anterior two-thirds of tongue (by chorda tympani from lingual nerve); of posterior one-third of tongue through glosso-pharyngeal nerve, Jacobson's nerve, and otic ganglion.
VII. Facial nerve.	*Motor* to all muscles of face and scalp (excepting levator palpebræ superioris), platysma, posterior belly of digastric, and stapedius muscle. It is joined by the chorda tympani (conveying taste-fibres of anterior two-thirds of tongue from lingual branch of V. to Meckel's ganglion).
VIII. Auditory nerve.	Hearing and Equilibration.
IX. Glossopharyngeal nerve.	*Sensory* from pharynx. Collects taste fibres from posterior one-third of tongue, which ultimately join V. *Motor* to middle constrictor of pharynx and stylo-pharyngeus.
X. Vagus nerve.	*Motor* for soft palate (except tensor palati), pharynx and larynx (through accessory portion of XI). *Motor* (involuntary) and *sensory* for heart, respiratory passages and abdominal viscera.
XI. Spinal accessory nerve.	*Motor* to sterno-mastoid and trapezius. (Supplies vagus with motor fibres for larynx, pharynx, and palate).
XII. Hypoglossal nerve	*Motor* to tongue and depressors of hyoid bone.

concussion, from tearing of the olfactory filaments and is usually permanent. Both bilateral and unilateral anosmia may result from pressure of a prefrontal or pituitary tumour upon the olfactory bulbs. For hysterical anosmia, see § 888.

The olfactory filaments are also of importance, as they may constitute an axonic pathway of invasion of the nervous system in certain virus infections, *e.g.*, poliomyelitis.

§ 832. The **Eye** is innervated mainly by four cranial nerves, the *second*, *third*, *fourth*, and *sixth*. The *fifth* and the cervical *sympathetic* are also concerned in its innervation. Careful examination of the eye is of the greatest importance in many diseases.

The symptoms which reveal disease of the eye may be arranged under six headings: I. Pain, II. Superficial Alterations (§ 833), III. Defects of Vision (§ 834), IV. Condition of the Pupils (§ 838), V. Ocular Movements (§ 844), VI. Changes in the Fundi (§ 848). The reader should turn to the section dealing with the defect to which the patient's symptoms appear to belong.

The systematic examination of the eye consists of: Investigating pain if present; noting any superficial alterations; testing defects of vision, including visual fields, acuity and refraction; examining the pupils, the ocular movements, and the fundi.

I. **Pain in the Eyes** is not infrequently absent in ocular affections. Its commonest cause is some error of refraction (ametropia). Eye-strain may give rise to headache, eye-ache or neuralgia. Glaucoma is a serious cause of pain in the eye and neighbourhood. Dental disorders may not only give rise to reflex pain, but also to both functional and organic affections of the eyes. Among subjective sensations other than pain may be noted muscæ volitantes (black specks) and scintillating scotoma (zigzag lines). The former may be normal, but evident to the patient because of eye-strain or poor health, or they may be pathological and due to vitreous opacities. The latter occurs in association with mania.

ASTHENOPIA, with painful [illegible] of the eyes and lachrymation, is associated with some degree of photophobia [illegible] pharospasm. Often an expression of allergy, there may be a contributory [illegible]ctive error. When constant, dust and feathers, or otherwise dogs, cats and [illegible] are causal; when seasonal, pollens. (§ 179, III.)

§ 833. II. **Superficial Alterations** require examination in a good light, and with the help of a binocular loupe.

1. REDNESS of one or both eyes may be *generalised* when it is due to conjunctivitis, iritis or acute glaucoma. *Localised* redness suggests corneal ulceration, episcleritis or a blocked tear duct. (For further details a special text-book should be consulted.)

2. PROPTOSIS (exophthalmos) is an undue prominence of one or both eyeballs and may be detected by standing behind the patient and looking down over the forehead. Apparent exophthalmos may be seen in high myopia.

Unilateral proptosis occurs in (1) Exophthalmic goitre, (2) Exophthalmic ophthalmoplegia (Anterior pituitary excess), (3) Tumour or aneurysm in or invading the orbit, (4) Irritation of the Cervical Sympathetic in the neck or thorax, (5) Orbital cellulitis or periostitis, (6) Cavernous sinus thrombosis, (7) Facial asymmetry, and (8) Naso-pharyngeal tumour. Exophthalmos from goitre is usually bilateral: some of the others become so. *Recession* of the eyeballs (Enophthalmos) occurs in paralysis of the cervical sympathetic, the other symptoms of which are pseudo-ptosis, narrowing of the ocular fissure, contraction of the pupil, loss of the cilio-spinal reflex (reflex dilatation of the pupil when the skin of the neck is pinched) and absence of sweating over the face and the forequarter of the corresponding side.

3. THE EYELIDS.—The eyelids are puffy in renal disease, cardiac dropsy, angio-neurotic œdema, after violent coughing or vomiting, in eyestrain and arsenical poisoning, insect bites, frontal sinus suppuration, Graves' disease and mongolism. *Ptosis*, or drooping of the upper eyelid, may be partial or complete, and unilateral or bilateral. It may be due to (i.) paralysis of the unstriped muscle of Müller in the upper lid (cervical sympathetic palsy), (ii.) paralysis of the striped levator palpebræ superioris muscle when it is part of an Oculo-motor Nerve palsy due to tabes, leaking intracranial aneurysm, encephalitis lethargica, and mid-brain tumour. (iii.) Ophthalmoplegic migraine, (iv.) Myopathy affecting the face, (v.) Myasthenia Gravis, (vi.) Hysteria, or (vii.) as a Congenital condition. Ptosis is usually accompanied by a compensatory overaction of the frontalis muscle, except in myasthenia gravis (where

the frontalis is also paralysed) and hysteria. Blepharospasm is an *involuntary clonic twitching* of the eyelid. *Inability to close the eyelids*—Lagophthalmos—is due to weakness of the orbicularis oculi and is met with in Bell's (facial) palsy, myopathy and myasthenia gravis.

Lagging Behind of the Upper Eyelids when the patient looks down constitutes Von Graefe's sign in exophthalmic goitre (see § 186). In *Lid Retraction* a band of white sclerotic is seen between the upper lid margin and the iris. In *Proptosis* a white band of sclerotic is also visible between the iris and the margin of the lower lid.

The Slit Lamp is a refinement in the apparatus at our disposal for the minute examination of the living tissues of the eye. It consists of a binocular microscope carried on an arc and working in concert with a source of light shedding an intense beam on the part examined. By its means the minutest alterations in the anterior parts of the eye (including the anterior vitreous) can be examined, and the detailed progress of pathological phenomena noted.

§ 834. III. **Defects of Vision** may consist of (1) defective sense of form or acuteness of vision, (2) alteration in the field of vision, (3) defective sense of colour.

(1) Acuity of Vision implies the estimation of forms of objects. It may be roughly tested by asking the patient to count the number of fingers held up before him. The defect may be so great that he cannot perceive light from darkness. The eyes must be examined separately, as it is often found that defect of one eye has existed a long time without the patient being aware of it. If with defective acuity of vision the external parts of the eye are normal, the media transparent, and the ophthalmoscope reveals no disease, it is probable that the patient suffers from an error of refraction, tobacco amblyopia, or retrobulbar neuritis. Eye-strain is due to protracted overaction of the intrinsic muscles of the eye, and is manifested by headache, eye-ache, blepharitis and styes, blinking (in children), conjunctival hyperæmia or Vth nerve neuralgia.

Errors of Refraction.—For accurately testing the visual sense of form, the patient is asked to read Snellen's types at a given distance and the visual acuity is recorded as a fraction, the numerator being the distance at which the patient is placed from the types, usually 6 (metres), occasionally 20 (feet); and the denominator, the distance at which the healthy emmetrope could read the smallest line which the patient is able to see. This latter figure is printed in small type under each line of letters on the chart and it varies, according to the line, from 5 to 60 (metres). The error of refraction is ascertained (after paralysing the ciliary muscle and iris by homatropin) by placing various lenses in the trial frame before the eyes until it is found which of them completely corrects his error. Convex lenses are indicated by the sign +, concave by the sign —. The defect is measured by the focal length of the lens required to correct his error, and is expressed in diopters, indicated by the sign D. A lens of one diopter has a focal length of 1 metre. Thus, a + 3 D. lens indicates a convex lens with a focal length of $\frac{1}{3}$ metre, being three times as strong as a lens of + 1 D. Retinoscopy is a more accurate method of testing refractive errors (below). In *myopia* (or near sight) the image is formed in front of the retina, and the patient cannot see distant objects clearly. In *hypermetropia* (or far sight) the image is formed on a plane behind the retina, and the patient has to accommodate powerfully for near objects. Both may be due to defective shape of the globe. Concave lenses are used to correct myopia, and convex to correct hypermetropia. In *presbyopia* the rigidity of the lens renders it either difficult or impossible to accommodate for near objects; it generally shows itself at the age of 45. The far vision of presbyopes may be good, though they cannot read or see near objects distinctly without convex glasses. *Astigmatism* is a non-correspondence of the curve in the principal meridians of the cornea, the curvature being similar to that of the bowl of a spoon. In *simple* astigmatism one meridian is myopic or hypermetropic; in

compound astigmatism the error of the two meridians, though of the same kind, differs in degree; in *mixed* astigmatism there is a myopic error in one meridian, and a hypermetropic error in the other meridian; in *irregular astigmatism*, usually the result of scarring, the curves of the cornea vary even in the same meridian. Astigmatism is detected accurately by a skilled examination with retinoscopy, with the ophthalmometer or better still with a crossed cylinder.

In **Retinoscopy** a plane mirror is used. No details of the fundus are visible in this way, but an evenly red field is seen—the red reflex. On tilting the mirror up and down or from side to side the red reflex will be found to move in the direction of the tilting in Hypermetropia and against the tilting in Myopia. In Hypermetropia this movement will be neutralised by a plus sphere of a power one dioptre more than the degree of Hypermetropia present, this difference being due to the fact that one works at a distance of one metre. Therefore, a movement in a patient with a Hypermetropia of 2 dioptres will be neutralised by a + 3 D. lens and an Emmetrope will require a + 1 D. lens to neutralise the movement. The use of a lens of a higher power will produce an artificial Myopia and therefore reverse the movement. No movement and no lens represents a Myopia of 1 dioptre, and neutralisation of movement against will be brought about by a lens 1 dioptre less powerful than the measure of the Myopia present. Similarly, the use of a minus lens of a higher power would produce an artificial Hypermetropia with reversed movement.

In astigmatism the reflex tends to be rectangular in shape and is neutralised by different lenses at opposite axes; the difference in power of the lenses is the measure of the patient's astigmatism.

The movement of the red reflex is accompanied by the movement of a shadow, and the movement of this shadow may be observed instead of following the reflex, but most observers find the latter to be the more satisfactory method.

Opacities in the media may be detected as dark shadows upon the red field. The radiating streaks of commencing cataract or moving opacities in the vitreous may be thus detected, the former ceasing to move when the movement of the eyeball ceases.

(2) The FIELD OF VISION is the extent of the picture presented to the eye at any given moment. It may be roughly tested by instructing the patient to cover one eye and look fixedly at the opposite eye of the examiner at a distance of about 3 feet. You then hold up one finger on each side of you in turn midway between, and bring it gradually towards the centre, asking the patient to say "yes" the moment it comes into his view. By repeating this procedure in different directions you will roughly ascertain in what part of his field the vision is defective. The dimensions of the visual field can be tested accurately only by the perimeter (below). *Scotoma* is a word used to indicate a spot of blindness or imperfect vision within an otherwise healthy field—*e.g.*, a central scotoma is a blind spot in the middle of the visual field.

The Perimeter.—One eye is covered with a shade and the patient places his chin on the chin-rest. He must be educated to keep his eye steadily fixed on the spot opposite, while the operator, by turning a handle, moves the test object along the arc of the perimeter from periphery to centre. The position in which the patient can first see the test object (while looking fixedly all the time at the central spot) is then marked on the chart by an automatic pricker. With apathetic patients this is a tedious operation, and without due care erroneous results may easily be obtained. The perception of colours in the peripheral field varies normally in extent with the different colours. Thus, from without inwards they are seen in the following order: white, blue, yellow, red, green. For the purpose of detecting minute central and paracentral scotomata, such as are met with in retrobulbar neuritis and pituitary enlargements, the ordinary perimeter is not sufficiently accurate. The *Bjerrum's screen*, with small test objects in skilled and patient hands and with reliable patients, gives very satisfactory results, which enable the examiner to obtain valuable diagnostic indications otherwise unobtainable; it is of particular value in determining any increase in the size of the blind spot, or in demonstrating the arcuate scotomata of chronic glaucoma.

(3) COLOUR VISION may be tested by matching coloured wools, or by the Edridge Green lamp or by Ishihara test cards.

Colour Blindness (achromatopsia) is a symptom in some diseases of the retina and in optic atrophy. Red-green blindness as a congenital or familial deficiency is common: yellow-blue blindness is rare. In tobacco blindness and in some other forms of retrobulbar neuritis, anything from minute scotomata for red and green to complete colour blindness may occur (§ 836).

§ 835. The **Causes of Defective Vision** without very obvious ocular changes may be considered under 1. UNILATERAL BLINDNESS, 2. BILATERAL BLINDNESS, 3. DEFECTS OF THE VISUAL FIELDS, and 4. NIGHT BLINDNESS. The defective vision due to errors of refraction has already been dealt with.

AMBLYOPIA is diminished vision, AMAUROSIS total loss of vision, sometimes without discoverable changes in the fundi or error of refraction. It is obvious that amblyopia may be due either to some functional disturbance of the visual apparatus, or to some gross lesion of the brain or paths of vision.

1. UNILATERAL BLINDNESS may be sudden or gradual. (*a*) *Sudden unilateral blindness* occurs in (i.) retrobulbar neuritis, (ii.) thrombosis of the central retinal vein, or embolism or spasm of the central retinal artery, (iii.) sudden intra-ocular hæmorrhage, (iv.) detachment of the retina or (v.) hysteria. (vi.) A patient may suddenly discover blindness in one eye which has long been defective. See retrobulbar neuritis, § 851.

(*b*) *Gradual unilateral blindness* may result from (i.) Cataract, (ii.) Chronic glaucoma, (iii.) Retrobulbar neuritis, (iv.) Tumour of or pressure on the optic nerve, (v.) Optic atrophy in tabes dorsalis or disseminated sclerosis, (vi.) Local disease of the choroid or retina, *e.g.*, Choroido-retinitis, (vii.) Amblyopia ex anopsia (usually in history of squint).

§ 836. 2. BILATERAL BLINDNESS may be sudden or gradual.

(*a*) *Sudden bilateral blindness* may occur in: (i.) Exposure to blinding sunlight, snow, or electric light, (ii.) Hysteria, (iii.) Sudden and copious hæmorrhage from the stomach, bowels or uterus, (iv.) Uræmia, (v.) Diabetes, (vi.) Insulin hypoglycæmia, or from (vii.) Local Trauma, *e.g.*, gunshot wounds to the calcarine cortex and occipital poles.

(*b*) *Gradual bilateral blindness* may be due to (i.) Cataract, (ii.) Chronic glaucoma, (iii.) Tobacco Amblyopia, (iv.) Toxic Amblyopia, (v.) Pituitary Tumour or Suprasellar cyst, (vi.) Local disease of the Retina and Optic Nerve, *e.g.*, Primary Optic Atrophy as in tabes, or Consecutive Atrophy, following papillœdema or papillitis. (vii.) Leber's Hereditary Optic Atrophy (§ 852).

Tobacco Amblyopia occurs sometimes in hard smokers of over three or four ounces per week, or in debilitated persons and in women from a much smaller quantity. The patient first complains of defective vision in bright light; he sees better at dusk than at noon and mistakes silver for copper coins. The defect is slowly progressive, becoming most marked in the *central* field, and there is a central colour scotoma, especially for red and green. At first there may be no changes in the fundi, then the discs become slightly congested in the earlier stages, and pale and atrophied, especially on the temporal side, in the later. Defective vision is the earliest symptom to attract the patient's notice. It arises from a chronic retrobulbar neuritis. The pupil reaction, both in chronic and acute retrobulbar neuritis, is characteristic. The pupil contracts normally to light, but does not remain contracted under the same light stimulus and dilates slightly after a second or so. The local application of acetyl-choline by iontophoresis is recommended.

Toxic Amblyopia is exemplified in uræmia and diabetes. It may also be produced by large doses of quinine, bisulphide of carbon in india-rubber manufacture, iodoform, dinitrobenzol and arsenical preparations like tryparsamide. Little in the way of treatment can be done for such cases, unless they are seen early, when removal of the cause and functional rest to the structures involved may lead to recovery.

§ 837. 3. Defects in the Visual Fields.

Central Scotoma, or a blind patch in one or both visual fields, may occur from (1) Retrobulbar Neuritis (see § 851), (2) Toxic Amblyopia (see § 836), (3) Early Optic Atrophy (and Leber's Disease), (4) Retinal hæmorrhage, (5) Central opacities in the lens or cornea. As a temporary phenomenon it occurs in (6) Migraine.

Hemianopia has been considered in § 676, to which the reader is referred. The following types are recognised:

(*a*) *Homonymous Hemianopia* or loss of the corresponding halves of the visual fields, is a hemiplegia of the visual fields and results from a lesion in the contra-lateral visual part of the internal capsule or the occipital lobe. A right homonymous hemianopia means abolition of the right halves of the visual fields. Apart from its occurrence in migraine, it is due to a gross central lesion situated in some part of the

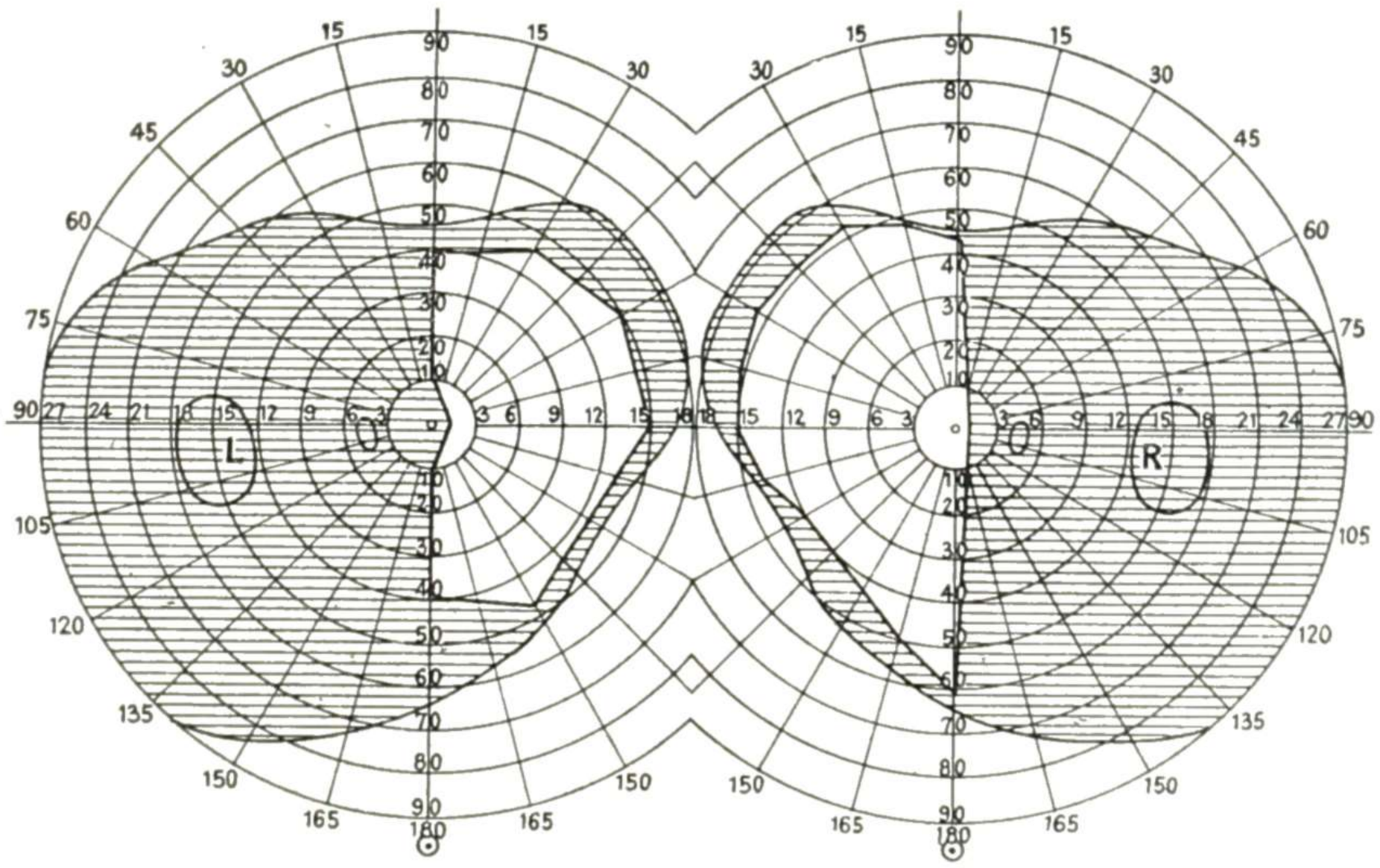


Fig. 189*a*.—Visual Fields showing Bitemporal Hemianopia. Case of Acromegaly due to pituitary adenoma in a woman aged 30 years; two years' duration.

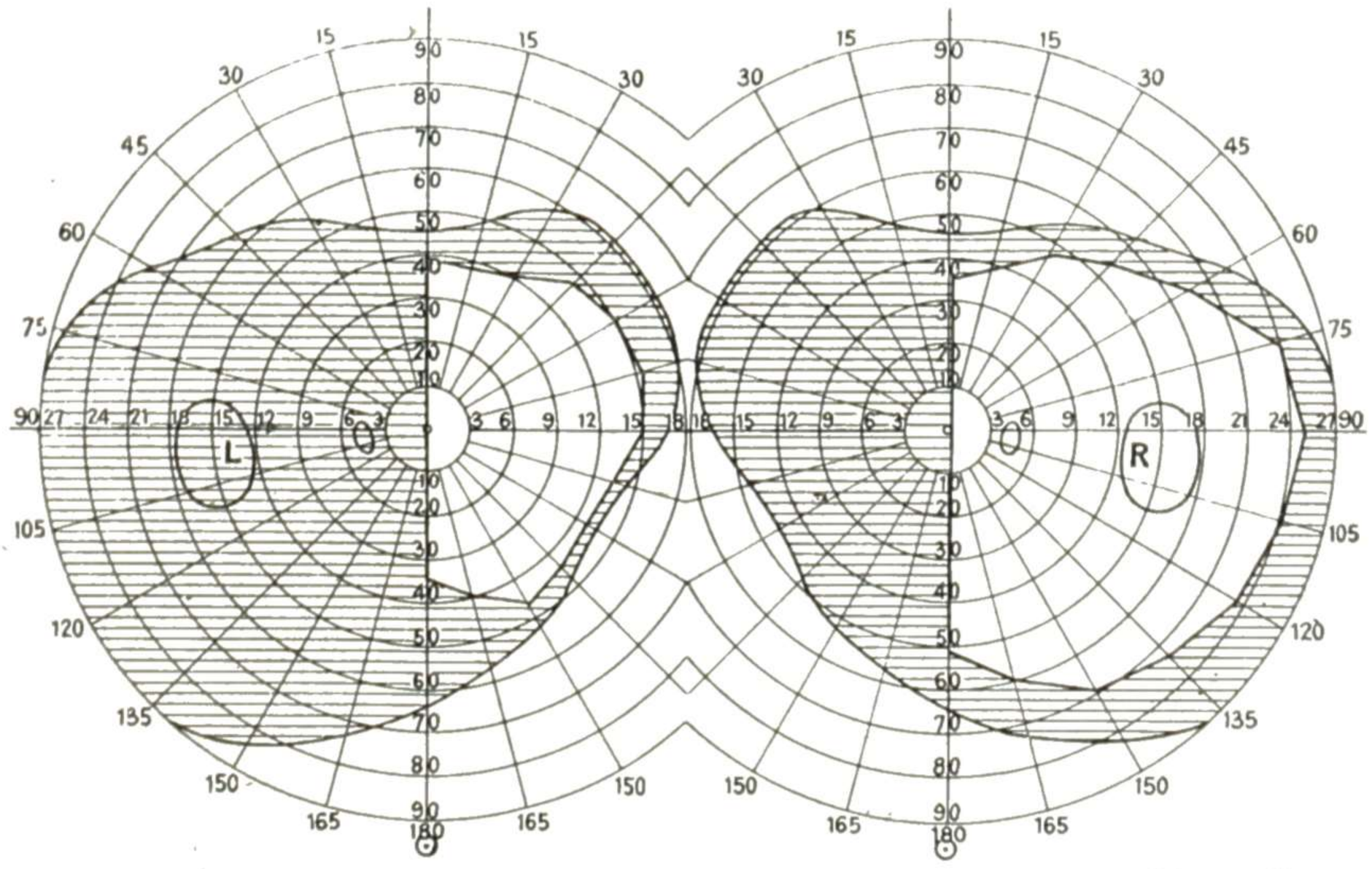


Fig. 189*b*.—Visual Fields showing left Homonymous Hemianopia. Case of large glioma of the right temporal lobe involving the optic radiation.

visual path behind the chiasma, (i.) in the optic tract, or (ii.) behind the corpora quadrigemina, *i.e.*, the hinder end of the internal capsule, (iii.) the optic radiation in the temporal or occipital lobe, or (iv.) in the calcarine cortex of the occipital lobe. By employing Wernicke's test (see below) the first may be excluded. For the rest, the precise position and character of the lesion can only be diagnosed by the accompanying symptoms. (*b*) *Quadrantic Hemianopia* is a loss of quadrants of corresponding sides of both visual fields. It results from (i.) a lesion deep in the temporal lobe, involving the "temporal knee" of the optic radiation, (ii.) a lesion of the calcarine cortex, (iii.) a lesion either above or below the optic chiasma. Lesions involving the *upper* fibres of the calcarine cortex, optic radiations or optic chiasma, produce blindness of the *inferior* quadrants of the visual fields and *vice versa*. (*c*) *Bitemporal Hemianopia* or loss of the temporal halves of both visual fields arises from a lesion in the neighbourhood of the optic chiasma, *e.g.*, Pituitary tumour in Acromegaly, distended infundibulum in Hydrocephalus or basal meningitis. (*d*) *Nasal Hemianopia* or loss of the nasal halves of both visual fields is of theoretical interest and would result from bilateral lesions on the outer margins of the optic chiasma or optic tracts, *e.g.*, pressure from aneurysmal dilatation of both internal carotid arteries. (*e*) *Altitudinal Hemianopia* or loss of the upper or lower halves of the visual fields might conceivably eventuate from (i.) optic neuritis, (ii.) a bilateral lesion involving the upper or lower lips of the calcarine cortex on the two sides, *e.g.*, from a tumour situated mesially between the occipital poles, (iii.) disseminated sclerosis, (iv.) a suprasellar cyst, or (v.) (temporarily) in migraine.

Causes of Hemianopia.—Hemianopia may result from : (1) Cerebral Tumour, (2) Pituitary Tumour, (3) Hydrocephalus, (4) Cerebral thrombosis, embolism or hæmorrhage, (5) Pyæmic Cerebral Abscess, (6) Syphilitic encephalitis or meningitis, (7) Tuberculomata, (8) Aneurysms.

Wernicke's hemianopic pupillary reflex helps you to determine the seat of a lesion in hemianopia. In hemianopia due to lesions in the optic radiations or occipital cortex, a beam of light thrown directly on the blind half of the retina by the concave mirror of the ophthalmoscope, produces contraction of the pupils, as the pupillary light reflex arc is intact (see Fig. 164). In hemianopia, due to lesions of the optic tract or anterior to this, pupillary contraction will fail to occur.

4. Night Blindness (*nyctalopia*) is defective vision in dim lights. It is a feature of (i.) retinitis pigmentosa, (ii.) syphilitic retinitis, or it may be without fundus changes, (iii.) as a congenital deficiency, or (iv.) as hysterical night-blindness. Concentric contraction of the visual field is met with and, finally, complete blindness. Acute night-blindness may attack those with defective nutrition who have been exposed to a very strong sun or artificial light, and in those cases the prognosis is good. Night vision is of particular importance for night-flying. Bishop Harman has elaborated a disc-spotting test for the dark adapted eye : improvement of function is brought about by breathing oxygen and in some cases by vitamin A administration.

§ 838. IV. **Condition of the Pupils.** The tonic dilator of the pupil is the cervical sympathetic (dilator iridis), the tonic constrictor of the pupil is the oculomotor nerve (ciliary muscle and sphincter iridis). Each of the three muscles may be paralysed separately : paralysis of the last two is known as *ophthalmoplegia interna*. The pupils must be tested with regard to their size, shape, equality, reaction to light, direct and consensual, and to accommodation (see Examination of the Nervous System, § 703).

Abnormal Dilatation of the Pupil (Midriasis) is met with in (1) Neurasthenia, Anxiety Neurosis and other states of Fear, (2) Hyperthyroidism, (3) Anæmia, or from : (4) Midriatic Drugs : Atropine, Homatropine, Belladonna, Cocaine. In nervous disease it may be the result of (5) paralysis of the sphincter pupillæ (oculomotor paralysis from post-diphtheritic, syphilitic or encephalitic paralysis), or (6) irritation of the dilator pupillæ (cervical sympathetic stimulation, *e.g.*, cervical tumour). It is met with in (7) Optic Atrophy, (8) Glaucoma, (9) in deep coma or collapse and in trauma (traumatic midriasis).

ABNORMAL CONTRACTION OF THE PUPIL (Miosis) is met with in (1) Pontine Hæmorrhage; (2) Tabes dorsalis and G.P.I.; (3) Disease of the Cervical portion of the spinal cord, *e.g.*, Syringomyelia; (4) Iritis; (5) Foreign body in the eye, or from: (6) Miotic Drugs: Eserine, Pilocarpine, Opium; (7) Congenital microcoria; (8) Old age.

SLIGHT INEQUALITY OF THE PUPILS may be observed in health, especially as the result of a refractive error. The above causes may operate.

IRREGULARITY OF OUTLINE OF THE PUPILS.—This may be due to (1) old iritic adhesions, or may result from (2) previous iridectomy or trauma. Deviations from the circular shape occur in (3) Neurosyphilis, *e.g.*, tabes, G.P.I., or (4) Glaucoma —in which the pupil may appear twisted. (5) Coloboma iridis is a congenital deficiency of the iris, usually in the lower part, generally on both sides. The defect may be continued backwards, involving the choroid below and even the optic nerve, while the vision is always more or less impaired.

EX-CENTRIC PUPILS (Ectopia pupillæ) may be (1) Congenital or observed after (2) iritis, or (3) in mid-brain lesions, *e.g.*, syphilis, encephalitis, neoplasms.

PUPILLARY REFLEX TO LIGHT.—In testing the light reflex, *both* eyes should be covered for half a minute, and each uncovered in turn, opposite a bright light which makes the pupils contract. The (*a*) direct and (*b*) consensual reactions should be tested (see § 703). In a good light the iris can sometimes be observed to contract and dilate rhythmically, a phenomenon known as *hippus*. Hippus may be seen in normal eyes or in some cases of chorea.

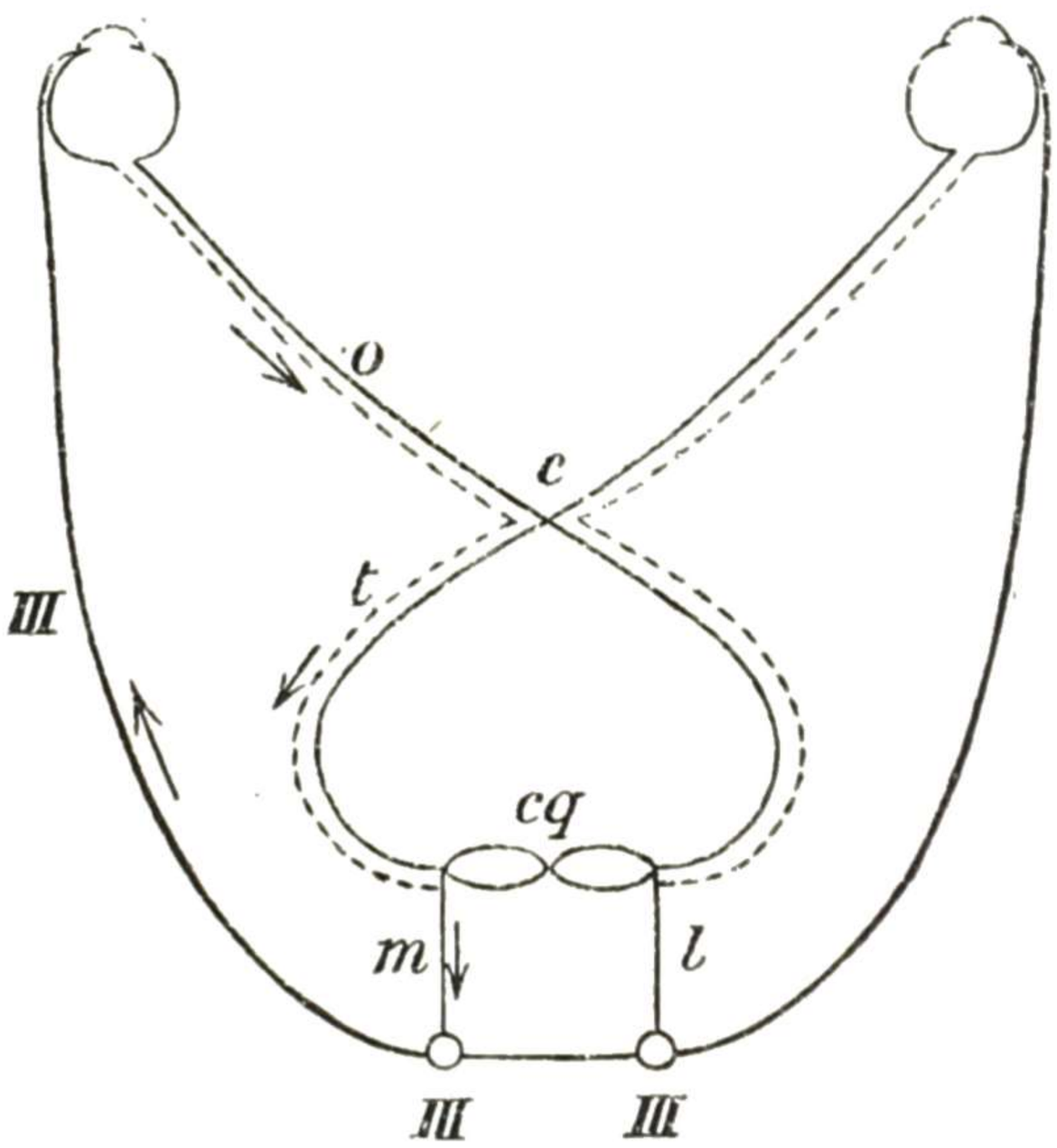


FIG. 190.—Diagram showing REFLEX ARCS concerned in the MOVEMENTS OF THE PUPIL (Horizontal Plane) —*o*, optic nerves; *c*, optic chiasma; *t*, optic tract; *cq*, corpora quadrigemina; *III*, third nerves and nuclei; *m* and *l*, Meynert's fibres communicating between the third nuclei and the corpora quadrigemina.

The *pupillary light reflex* depends on the integrity of the retina and the following tracts (see Fig. 190): the optic nerve (*o*), the chiasma (*c*), the optic tract (*t*), the superior corpora quadrigemina (*cq*). These last-named nuclei (*cq*) are connected by means of the colliculo-ocular (Meynert's) fibres (*m*, *l*), with the nuclei of the third nerves (III) situated in the floor of the aqueduct of Sylvius. The fibres of the third nerve, through the long or short ciliary branches, conduct impulses to the sphincter iridis, causing pupillary contraction.

§ 839. Loss of the PUPILLARY REFLEX TO LIGHT (light iridoplegia) may be produced by a lesion situated anywhere in these afferent or efferent tracts. Loss of direct reflex to light, together with preservation of brisk pupillary contraction on convergence-accommodation constitutes the *Argyll-Robertson phenomenon*. Argyll-Robertson pupils may be large or small and the phenomenon may be unilateral or bilateral. In addition, such pupils may be (i.) ex-centric in position, (ii.) irregular in outline, (iii.) unequal in size.

Causes of Argyll-Robertson Pupils: Clinically: the Argyll-Robertson pupil is found in syphilis affecting the vicinity of the aqueductus Sylvii and the superior colliculi. Apart from the syphilitic lesion, Argyll-Robertson pupils have been observed and reported as a clinical rarity in the following conditions: non-specific encephalitis (*e.g.*, encephalitis lethargica), mid-brain tumours, syringobulbia, in traumatic lesions of the mid-brain, in alcoholic polyneuritis and diabetes mellitus.

The *anatomical* basis of the Argyll-Robertson phenomenon is obscure. The condition may be met with in: (1) lesions of both optic nerves, (2) lesions of both optic tracts, and (3) central lesions of the colliculo-ocular fibres which pass to the anterior end of the third nucleus in the mid-brain. (If one is to insist on small pupils as described by Argyll-Robertson the third situation is the only one possible.) The nerve centre for the pupillary light reflex is the oculomotor nucleus, but recent work has shown that there is no lesion of this nucleus in tabes.

Myotonic pupils are pupils which react only to very strong light, and on convergence-accommodation may contract slowly, remaining contracted for some seconds, before very slowly dilating again: in many cases only one eye is affected. The condition may be met with in association with absence of tendon reflexes, and may simulate tabes dorsalis, if the true condition of the pupils is not recognised (Adie).

Convergence—Accommodation Reaction (Near Reflex).—When looking at a near object the eyes are converged, the ciliary muscles contract (causing increased convexity of the lens) and the pupils narrow. These ciliary and pupillary reactions constitute accommodation. Loss of pupillary contraction on accommodation occurs in: (1) Post-Encephalitis Lethargica, (2) Diphtheria (with palatal paralysis or loss of tendon reflexes), (3) Poisoning with atropine or belladonna, (4) lesions of the oculomotor nerve, (5) Deficient convergence is commonly seen after head injuries, in association with defects of memory and of concentration.

§ **840. Iritis,** inflammation of the iris, is manifested by (1) immobility, change of colour, loss of pattern, and exudation; (2) pain (which may be absent in serous iritis) dimness of vision and watery discharge; (3) adhesion between the iris and anterior capsule, revealed under atropine; (4) circumcorneal injection, indicating hyperæmia of the ciliary vessels. Care must be taken not to mistake this disease for conjunctivitis or glaucoma, because the treatment suitable for either will make iritis worse. In conjunctivitis the injection is most marked away from the cornea, while in iritis the injection is most marked at the cornea, *i.e.*, pericorneally.

Causes of iritis: Focal sepsis, tuberculous, gonorrhœal, syphilitic, diabetic and other toxæmias may cause it. Syphilitic iritis is usually non-recurrent; the other forms are liable to relapse. Chill, bright light and injury may be determining causes.

Treatment consists in regular applications of atropine drops (1 per cent.), heat, and leeches to the temple. If attended by much pain, aspirin (gr. 15) generally gives complete relief in fifteen to twenty minutes. For chronic iritis dionin drops (4 per cent.) twice daily, along with atropine, may be tried. In recurring iritis, not associated with cyclitis (keratitis punctata), iridectomy in the quiescent stage is a good treatment. If complete annular synechiæ form, iridectomy should be performed, to prevent secondary glaucoma. The use of tuberculin should not be forgotten.

§ **841. Ophthalmia Neonatorum** has always been a frequent cause of blindness: though the number of notifications changes little, resultant blindness has been greatly reduced. The causal organism is the gonococcus in more than 60 per cent. of cases, and next in frequency is the streptococcus. *Treatment.* Penicillin drops (5,000 units per c.c.) frequently applied, have replaced all other forms of treatment, when the organism is penicillin sensitive. Otherwise the sulphonamides should be used in doses of $\frac{1}{8}$ G. six-hourly for five days.

§ **842. Cataract.**—The following points are of interest: *Lamellar cataract* is a post-natal condition associated with and due to rickets; the enamel of the margins of the following permanent teeth of both jaws

is defective—both incisors, the canines and the first molars, *i.e.*, the enamel which is being laid down between the sixth and twenty-fourth month of life. The malady is therefore preventable. It also occurs with parathyroid tetany. *Senile cataract* may also be preventable, but the cause is unknown. Sodium iodide drops or ointment are supposed to limit the rate of progress of cataract and even cause its disappearance in early cases. The urine should be examined for sugar and before operation all carious teeth should be removed, the tonsils and sinuses examined, and the lachrymal sac, if infected, should be removed.

§ **843. Cervical Sympathetic Lesions** may be due to (*a*) Paralytic, or (*b*) Irritative Lesions.

(*a*) *Paralytic lesions* produce (i.) small pupil, (ii.) loss of cilio-spinal reflex, (iii.) enophthalmos, (iv.) narrowing of the ocular fissure, with ptosis, and (v.) absence of sweating on the corresponding side of the face and forequarter.

(*b*) *Irritative lesions* produce (i.) dilated pupil, (ii.) brisk cilio-spinal reflex, (iii.) exophthalmos, (iv.) widening of the ocular fissure, with lid retraction, and (v.) increased sweating on the corresponding side of the face and forequarter.

The *Causes* of paralytic or irritative lesions of the Cervical Sympathetic are: (1) Intrathoracic aneurysm, Neoplasm or Enlarged Mediastinal glands (Lymphadenoma, etc.); (2) Apical pleural fibrosis in Pulmonary Tuberculosis, and Apical Pneumonia; (3) Tumours in the neck; (4) After operation for neck glands; (5) Cervical Rib; (6) High Brachial Plexus lesions, *e.g.*, trauma, secondary deposits; (7) Disease or injury of the cervical cord from implication of the bulbo-spinal sympathetic fibres, *e.g.*, syringomyelia, spinal tumours, fracture-dislocations; (8) Mid-brain lesions; (9) Myasthenia gravis; (10) Exophthalmic goitre; (11) Congenital Sympathetic lesions (see Fig. 173).

§ **844.** V. **Ocular Movements.**—The external muscles of the eyeball (as distinct from the internal or involuntary muscles of the eye) are six in number, and they are supplied by three cranial nerves: External rectus (VI. nerve); superior oblique (IV. nerve); internal, superior, and inferior recti and inferior oblique are supplied by the III. nerve (which also, it will be remembered, supplies the levator palpebræ, the sphincter fibres of the iris, and the ciliary muscle). It follows therefore that:

Complete paralysis of the third nerve (oculomotor) is attended by	Ptosis; external strabismus; pupil dilatation and immobility; loss of accommodation; inability to move eyeball inwards or upwards, and only imperfectly downwards; slight protrusion of the eyeball: crossed diplopia.
Paralysis of the sixth nerve (abducens) is attended by	Internal strabismus; inability to move eye outwards; homonymous diplopia.
Paralysis of the fourth nerve (trochlear) is attended by	Slight deviation of cornea upwards; homonymous diplopia on looking downwards.

Defects in the ocular muscles are revealed (1) by defective movements of the eyeball towards the side of the paralysed muscle; (2) by false orientation; (3) by squint in pronounced cases; (4) by diplopia (double vision); (5) by vertigo; and (6) by the head being tilted towards the side of the paralysed muscle.

The more recent the paralysis the more marked are the above symptoms, while in old paralyses the symptoms are more mixed.

Deficient movement of the eyeball—(Fig. 191)—*indicates paralysis of*	
outwards	external rectus—sixth nerve.
inwards	internal rectus—third nerve.
upwards	{ *superior* rectus / *inferior* oblique } third nerve.
downwards	{ *inferior* rectus—third nerve. / *superior* oblique—fourth nerve.

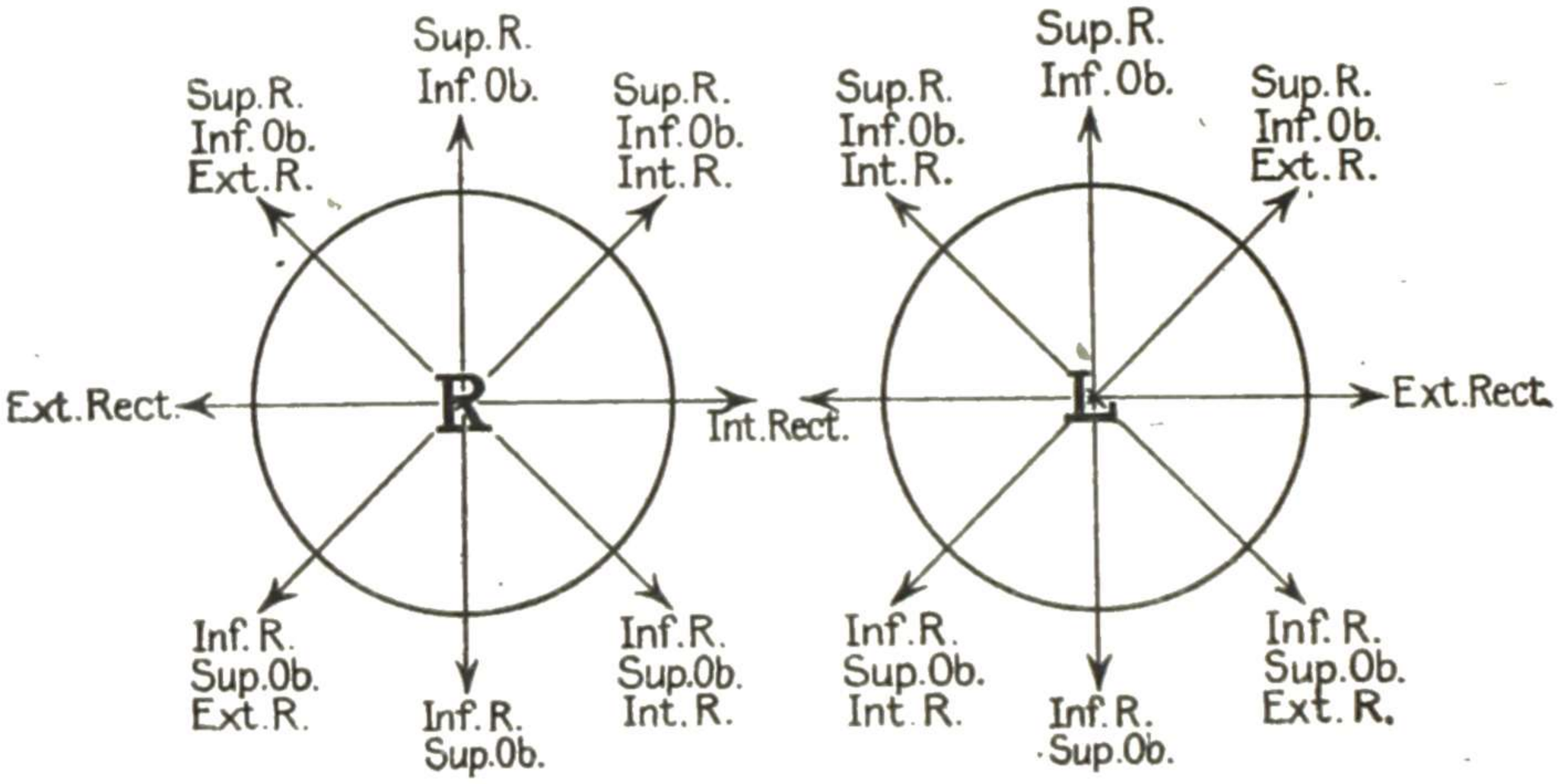


FIG. 191.—MACGILLIVRAY'S DIAGRAM showing muscles concerned in ocular movements. Lateral movements—one muscle. Vertical movements—two muscles. Oblique movements—three muscles; *e.g.*, Elevation and adduction—the resultant of the forces on each side, *i.e.* Elevation (Sup. Rect. and Inf. Ob.) and adduction (Int. Rect.).

Method of Detecting the Affected Eye and Paralysed Muscle.—Place a red glass before the patient's left eye, and hold a lighted candle before him in a dark room, on a level with his eyes at 2 yards distance. Suppose that it is found that the red image overlaps, or is a little to the left of the white image, and both images are on the same level, to determine which muscle is affected the candle must be moved to the right and to the left, and we must notice *in which direction the distance between the images becomes increased*. On moving the candle to the right the image approaches till only one candle is seen, and on moving to the left, the distance between the true and false images increases. Bearing in mind the rule that the weakened muscle is on the same side as the direction in which diplopia increases, it is evident that either the left external rectus or the right internal rectus (which turn the eyes to the left) is affected. Ask then on which side the red image appears. If on the left of the white image, homonymous diplopia is present; therefore the left *external* rectus is the paralysed muscle. If, however, the red image is to the right of the white image, crossed diplopia is present; therefore the right *internal* rectus is the paralysed muscle. Paralysis of the superior or inferior rectus, and of the superior or inferior oblique, gives rise to vertical diplopia. The former causes crossed diplopia, the latter homonymous diplopia. Loss of motion upwards is due to paralysis of the third nerve; loss of motion downwards may be due to paralysis of the inferior rectus (third nerve) or the superior oblique (fourth nerve). Werner's diagrams (Fig. 192) simplify the detection of the affected muscle in vertical diplopia. The black lines in the diagrams represent the true images, the dotted lines the false images. The dotted lines extend above and below the black lines, indicating that the false images are higher and lower than the true images. The names of the muscles in the upper and lower part of the diagrams indicate that the diplopia is caused by upward and downward movements respectively

of the eyes when these muscles are affected. Thus, for example, in paralysis of the right inferior rectus an analysis of the diagram shows that (1) the diplopia occurs with downward movements of the eyes; (2) the diplopia is crossed, the false being to the left of the true image; (3) the false image has its upper part inclined towards the

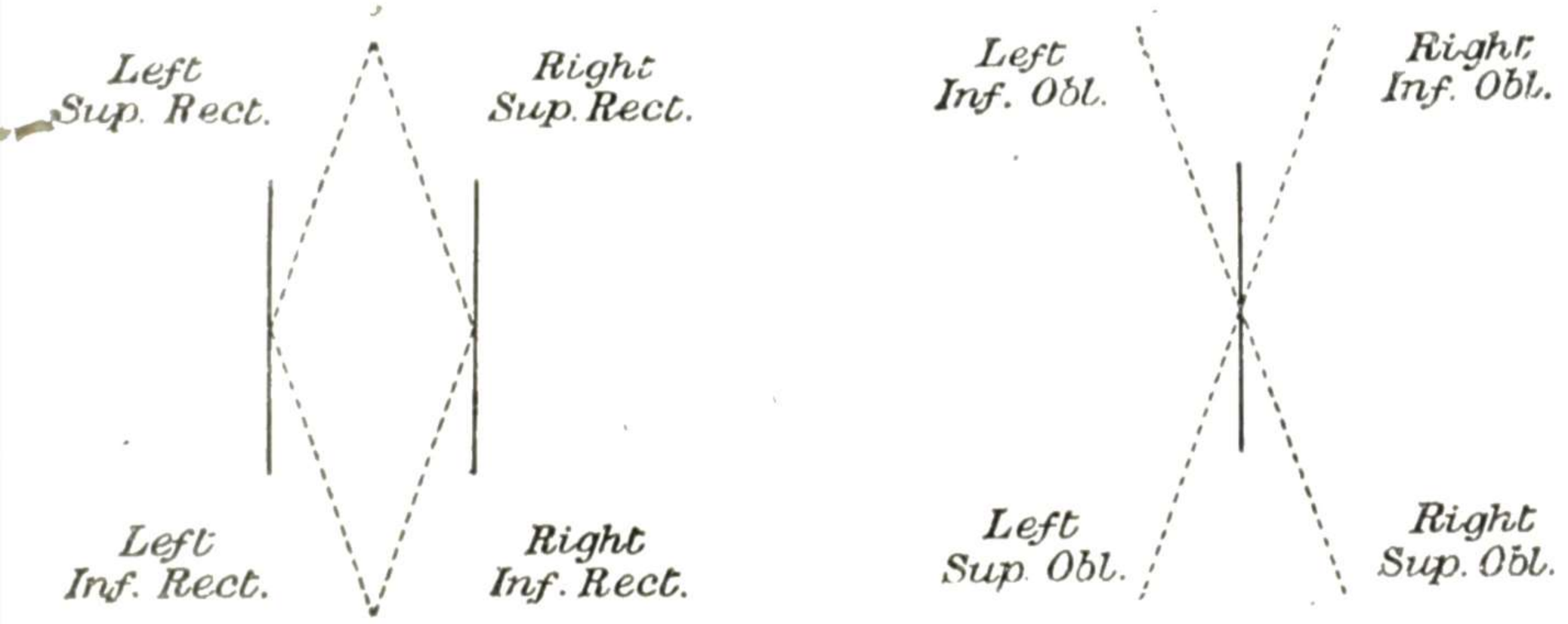


FIG. 192.—WERNER'S DIAGRAMS for detecting which is the affected muscle in cases of diplopia.

true image; and (4) the false image lies lower than the true one. With the oblique muscles it must be remembered that the superior oblique moves the eye downwards, and therefore the false image due to the paralysis of the superior oblique appears on moving the eye downwards. By remembering the diagrams it is comparatively easy to diagnose the paralysed muscle causing a diplopia.

§ 845. Squint or strabismus is a want of parallelism between the visual axes when looking at a distant object. It is called *convergent* when one eyeball looks inwards and *divergent* when one eyeball looks outwards. In *children* it is chiefly associated with some error of refraction—hypermetropia (internal strabismus, the commonest in children), or myopia (external strabismus). In many cases the patient or a parent is left-handed: the refractive error may be small and a psychological factor may be of great importance. In adults or children, squint of recent acute origin is due to definite paralysis of an ocular nerve.

Diplopia or double vision is, in many cases, due to weakness of one or more ocular muscles, but may be due to any condition altering the visual axes, *e.g.*, unequal proptosis, fracture of the orbit. Erroneous projection—*i.e.*, error in judging the position of objects—and vertigo (due to the same cause) are invariably associated with diplopia.

Clinical Investigation of Squint.—There are three steps. (1) Diagnosis of the Type of Squint, (2) Diagnosis of the Affected Muscle, (3) Diagnosis of the Position of the Lesion and its cause.

(1) DIAGNOSIS OF THE TYPE OF SQUINT.—Squints are of three kinds: (A) Concomitant, (B) Paralytic, and (C) Alternating.

(A) *Concomitant Squint* is met with most frequently in children. (i.) It comes on slowly. (ii.) Each eye, when the other is covered, moves perfectly in all directions, there being no paralysis, but when examined together, the squint is present in all positions of the eyeball. (iii.) Spontaneous diplopia is absent. (iv.) The affected eye follows the sound eye

with equal defect in all directions (hence the name "concomitant"), so that the defect of parallelism is the same in all directions. It is due, in about 90 per cent. of cases, to hypermetropia or other error of refraction, or to a defect in the fusion faculty, and when these are remedied, particularly with the aid of orthoptic exercises, in childhood the squint may disappear.

(B) *Paralytic Squint* is met with in children or adults. (i.) It usuall appears suddenly. (ii.) It is always accompanied by double vision. (iii.) There is limitation of movement of the globe corresponding to the direction of traction of the paralysed muscle. Paralytic squints are due to ocular paralyses resulting from intracranial or other serious mischief.

(*c*) *Alternating Squints* are nearly always associated with left-handedness in the patient or family, and sometimes with stammering.

(2) DIAGNOSIS OF THE AFFECTED MUSCLE.—In *Paralytic Squint* the affected muscles *can usually be recognised by simply testing the external ocular movements as described* in § 703.

In cases of slighter weakness, and in *Concomitant Squint*, tell the patient to look at an object straight in front of him, that being the normal position of the eyes at rest, and fix some object. The eye with which he fixes is the normal eye. The deviation of the affected eye from the middle line is known as the "primary deviation." Now partially cover the sound eye and let him fix with the affected eye. The sound eye will now be found to deviate ("secondary deviation"). In concomitant squint the primary and secondary deviations are equal, but in paralytic squint, the secondary exceeds the primary. The patient unconsciously turns his face *towards the side of the weak or paralysed muscle*. To detect which is the affected muscle, hold a pencil vertically in front of the patient and move it rapidly to the right, to the left, and in various directions, and ask him whether he can see two pencils in any of these directions. *The weakened muscle is on the same side as the direction in which the double vision appears*. Diplopia may be homonymous or crossed. In simple or homonymous diplopia the false image lies on the same side as the affected eye; in crossed diplopia the false image lies on the side opposite to the affected eye. Paralysis of the external rectus, or obliques, causes homonymous diplopia; paralysis of the internal rectus, or superior or inferior recti, causes crossed diplopia.

MONOCULAR DIPLOPIA is recognised by the persistence of diplopia when one eye is closed. It is met with in (i.) Hysteria and in (ii.) Malingering. (iii.) It may arise from defects of the media.

Treatment—Orthoptic training, special exercises for promoting fusion and increasing amplitude, are becoming routine treatment.

(3) DIAGNOSIS OF THE POSITION OF THE LESION AND ITS CAUSE.—This is of importance in PARALYTIC SQUINT.

(*a*) *There is Diplopia, squint, and defective movement of* ONE EYE *towards the direction of traction of the affected muscle*. The lesion is in the PERIPHERAL NERVE (III, IV or VI).

The *Causes* of this type of Diplopia are:

I. Rheumatism.
II. Arterio-sclerosis.
III. Syphilis.
IV. Meningitis.
V. Trauma.
VI. Cerebral Tumour or Aneurysm.
VII. Subarachnoid Hæmorrhage.
VIII. Polyneuritis.
IX. Herpes Ophthalmicus.
X. Gradinego's Syndrome.
XI. Cavernous Sinus Thrombosis.
XII. Syndrome of the Sphenoidal Fissure.

I. Rheumatism produces a sudden oculomotor paralysis, usually of the external rectus muscle. There is severe pain in the face and in the eye itself with paralysis of one or more ocular muscles. The condition is analogous to Bell's palsy.

II. Arterio-sclerosis.—Isolated external rectus palsy in the aged may be due to pressure on the sixth nerve in its long intracranial course by a distorted atheromatous artery.

III. Syphilis.—A chronic syphilitic lepto-meningitis may cause isolated ocular alsies in tabes and other forms of neuro-syphilis.

IV. Meningitis of tuberculous or other origin (see § 726) may cause diplopia.

V. Trauma.—Blows without fracture, or fractures involving the anterior or middle fossa of the skull may cause damage to the oculomotor nerves and cause diplopia. In other cases, the displacement of the orbit or damage to its muscles resulting from the fracture is the cause of the diplopia. Diplopia may follow spinal anæsthesia and frontal sinus operation.

VI. Intracranial Tumour or Aneurysm.—Tumours of the base of the skull, pituitary growths, or internal carotid aneurysm, may cause oculomotor paralysis. Small berry aneurysms on the Circle of Willis may cause unilateral headache and oculo-motor palsy (ophthalmoplegic migraine). In increased intracranial pressure from any cause whatsoever (*e.g.*, Hydrocephalus) the sixth nerve may be damaged in its long intracranial course by pressure from above, with resultant external rectus palsy.

VII. Subarachnoid Hæmorrhage may be massive with unconsciousness, and later the patient may develop a paralytic squint, with hæmorrhage near the optic disc, and field defects.

VIII. Polyneuritis.—The polyneuritis of alcoholism, diphtheria, lead, etc. (see § 793), may be accompanied by external ocular palsies.

IX. Herpes Ophthalmicus may be followed by external ocular palsies, pupillary abnormalities, or even by optic atrophy.

X. Gradinego's Syndrome.—In association with acute suppurative mastoiditis in children, an external ocular palsy develops (usually the external rectus muscle) with pain referred to the distribution of the trigeminal nerve on the face. Other cranial nerves, *e.g.*, facial, hypoglossal, may be affected on the same side. The syndrome is believed to be due to localised meningitis or granulations at the tip of the petrous temporal bone. The symptoms clear up completely some weeks after surgical treatment of the mastoid infection.

XI. Cavernous Sinus Thrombosis (see § 738).—In pyæmic conditions with rigors and high fever, sudden unilateral proptosis develops with œdema, external ocular paralysis of the nerves running in the cavernous sinus (see Fig. 169), blindness and retinal hæmorrhage. The symptoms usually become bilateral within a few hours or days.

XII. Syndrome of the Sphenoidal Fissure.—There is acute supra-orbital pain, and pain in the eye, followed by signs of paralysis of the oculomotor, trochlear or abducens nerves. There may be sensory impairment over the supra-orbital nerve. The condition is supposed to be due to periostitis of the bones of the sphenoidal fissure and clears up in some months, after treatment with iodides and salicylate. Syphilis or orbital neoplasm may cause similar signs.

(*b*) *There is Diplopia, squint, and defective conjugate movement of* BOTH EYES *to the right, left, upwards or downwards*, or *in all directions* (ophthalmoplegia externa). The lesion is IN THE MID-BRAIN and is either NUCLEAR or SUPRA-NUCLEAR.

There may be associated pupillary abnormalities. The functions of the oculomotor nuclei in the mid-brain from before backwards are : (1) Pupillary reflex to light, (2) Convergence-Accommodation mechanism, (3) Upward, (4) Downward, and (5) Lateral movement of both eyes. The

symptoms of "crossed paralysis" resulting from lesions in the mid-brain are described in § 670.

The Causes of *Conjugate Ocular Paralyses* are:

I. Mid-brain Tumours.
II. Mid-brain Vascular Lesions.
III. Myasthenia Gravis.
IV. Encephalitis Lethargica.
V. Neurosyphilis.
VI. Diphtheritic Polyneuritis.
VII. Chronic Bulbar Palsy (motor neurone disease).
VIII. Botulism.

I. MID-BRAIN TUMOURS.—Tumour of the crus, pons or pineal body, produces loss of conjugate upward movement of the eyes with pupillary abnormalities, and progressive headache and vomiting. The symptoms are slowly progressive.

II. MID-BRAIN VASCULAR LESIONS.—Thrombosis, embolism, or hæmorrhage, may produce conjugate ocular paralysis of sudden onset usually with hemiplegia.

III. MYASTHENIA GRAVIS shows diplopia, ptosis, and loss of conjugate movement of the eyes. The paralyses may appear only in fatigue or may be permanent; their variability is characteristic. Other signs of myasthenia (see § 808) may be present. The pupils usually escape, but the light reflex may be lost.

IV. ENCEPHALITIS LETHARGICA.—The eye-signs of encephalitis lethargica are described in § 698. Diplopia is common at the onset of the disease, internal and external ophthalmoplegias may be present in an acute attack or afterwards. Residual paralysis of convergence-accommodation is characteristic of post-encephalitis.

OCULO-GYRIC CRISES (tonic eye-fits) occur in patients with post-encephalitis-lethargica. The patients' eyes suddenly become fixed in a position of conjugate deviation (usually upwards). They can be brought to the resting position by an effort of will, but only for a few seconds. They remain tonically deviated for minutes or hours until the spasm relaxes.

V. NEUROSYPHILIS, especially tabes dorsalis, may cause conjugate ocular palsies.

VI. DIPHTHERITIC POLYNEURITIS occasionally produces diplopia. The paralysis of convergence-accommodation is more characteristic.

VII. CHRONIC BULBAR PALSY (see § 747) is rarely associated with conjugate ocular paralysis.

§ 846. VIII. **Botulism** is a very rare severe disease following the consumption of artificially preserved food infected with the *B. botulinus*. It occurs in small localised epidemics. The first symptoms, headache and giddiness, occur twelve to twenty-four hours after infection. Then come double vision, failure of accommodation and ptosis, with external and internal ophthalmoplegias. Signs of gastro-intestinal irritation are usually absent and the temperature is subnormal. Later, there is dysphagia and dysarthria and general severe muscular asthenia, but sensory changes are absent and the mind is clear. The disease is fatal in over 50 per cent. of cases; death may occur from respiratory failure.

Diagnosis.—The disease bears a superficial resemblance to acute encephalitis lethargica; the history of infection in other consumers of the same tinned food, the absence of lethargy and the subnormal temperature, make the diagnosis clear.

Treatment.—Wash out the stomach and colon repeatedly and keep the bowels open with saline aperients. Alcohol should be given early. An anti-toxic serum is available and may prevent extension of the disease if given before all the exotoxin is fixed in the nervous system.

(*c*) *There is* CONJUGATE DEVIATION OF THE HEAD AND EYES. *The lesion is in the* INTERNAL CAPSULE *or above this.*

Following a cerebral thrombosis, embolism, or hæmorrhage, the patient loses the power of turning his eyes to the contralateral side and the eyes are pulled over to the side of the lesion by the unopposed action of the

antagonists. Irritative lesions of the second frontal convolution cause conjugate deviation of the head and eyes to the contralateral side.

Treatment of Squint.—In concomitant squint the visual refractive error is corrected, and afterwards attempts are made to develop binocular vision. In paralytic squint, to relieve diplopia, the false image should be excluded by covering the affected eye; but the cause must always be sought for and treated.

Skew-deviation of the eyes is a transient state of loss of parallelism of the visual axes, so that they cross, one eye looking down and in, and the other up and out. It occurs in acute lesions of the cerebellum or pons.

§ 847. **Nystagmus** is a rapid involuntary oscillation of the eyeballs, usually from side to side (lateral nystagmus), occasionally in a vertical direction (vertical nystagmus), or in a circular direction (rotatory nystagmus). Both eyes are usually involved, though each eye should be separately examined. The movements may be constantly present, but slighter degrees can only be brought out by causing the patient to follow your finger or a bright object to the extreme left or right. Very slight nystagmus can be discovered by direct ophthalmoscopic examination, when the image of the fundus, becoming magnified about fifteen diameters, shows the slightest movements of the eyeball. The symptom is notably present in labyrinthine disorders, disseminated sclerosis, cerebellar tumour, and Friedreich's disease, and in tumour involving the corpora quadrigemina, or one side of the pons. So-called CONGENITAL NYSTAGMUS is generally produced by any condition, such as ophthalmia neonatorum, which prevents the child using his eyes during the first few weeks of infancy when the co-ordination of the extrinsic muscles must be acquired; or in albinos, where, for want of pigmentation, the retina never receives definite images and cannot therefore acquire the power of fixation and muscular co-ordination. OCCUPATIONAL NYSTAGMUS is met with in miners, compositors, iron-founders, and those who work at close quarters, or in conditions of strain with deficient light.

Miner's Nystagmus.—Four stages are described: Latent, subacute, acute and neurasthenic. The latent form is found after leaving work and is elicited on rotation. The subacute is found at work; there are headache, dazzling, giddiness, eye movements (nystagmus), and a defect in visual acuity by day and poor vision at night (night blindness). In the acute stage all the above symptoms are aggravated and complicated by photophobia and spasm of the lids; the fourth stage is similar to the acute but general nervous symptoms develop. Bad illumination and poor ventilation have been blamed, but the causes are largely psychological, *i.e.*, hysterical symptoms superimposed on congenital or acquired instabilities of the ocular mechanisms.

§ 848. VI. **Changes in the Fundi.**—The reflecting **ophthalmoscope** is now rarely seen, having been replaced by the electric ophthalmoscope. To examine the eye, commence with a + 12 lens in the aperture and examine with its aid the cornea, aqueous and lens; by rotating the battery of lenses the power is gradually reduced, any opacities in the vitreous can be noted and when the fundus comes into focus the number of the lens in the aperture should be observed. This gives a rough measure of the refraction of the eye. Estimation of visual acuity without a knowledge of the patient's refraction is bound to mislead.

The *optic disc* should be examined as to its shape, its borders, its colour, its vessels, and its level. Normally the disc is circular or slightly oval, with a clearly defined

border, especially at the outer edge. It appears oval in astigmatic eyes. The *colour* of the disc is a rosy vermilion, but paler than the rest of the fundus. The vessels curve from the centre, and then lie flat. Arteries and veins go together, but the arteries are narrower (two-thirds) than the veins, a trifle paler, and have a broader, more continuous light stripe running along the centre. Normally the arteries do not pulsate, but the veins may do so. Pulsation in the arteries may indicate (i.) increased intraocular tension or (ii.) aortic regurgitation. The *level* of the disc is important, but a little difficult to gauge. If when using the ophthalmoscope, the surrounding *reti* can be seen clearly without the aid of any lens placed in the mirror hole, but the *disc* cannot be seen clearly without the aid of the lens, it must be at a different level. If a weak — lens is necessary to see the disc under these circumstances, then the disc must evidently be behind the retinal level (cupping). If, on the other hand, a weak + lens is necessary, then the disc is on a level anterior to the retina (swelling). One can gauge the amount of swelling or cupping in this way, for roughly each 3 D. = 1 mm. of swelling or cupping. Thus, supposing it is necessary to use 1 D. to focus the retinal vessels precisely, and 4 D. to focus the disc, then there must be 3 D. or 1 mm. swelling. This is an accurate method of measuring, provided the observer is able to *relax his own accommodation thoroughly*.

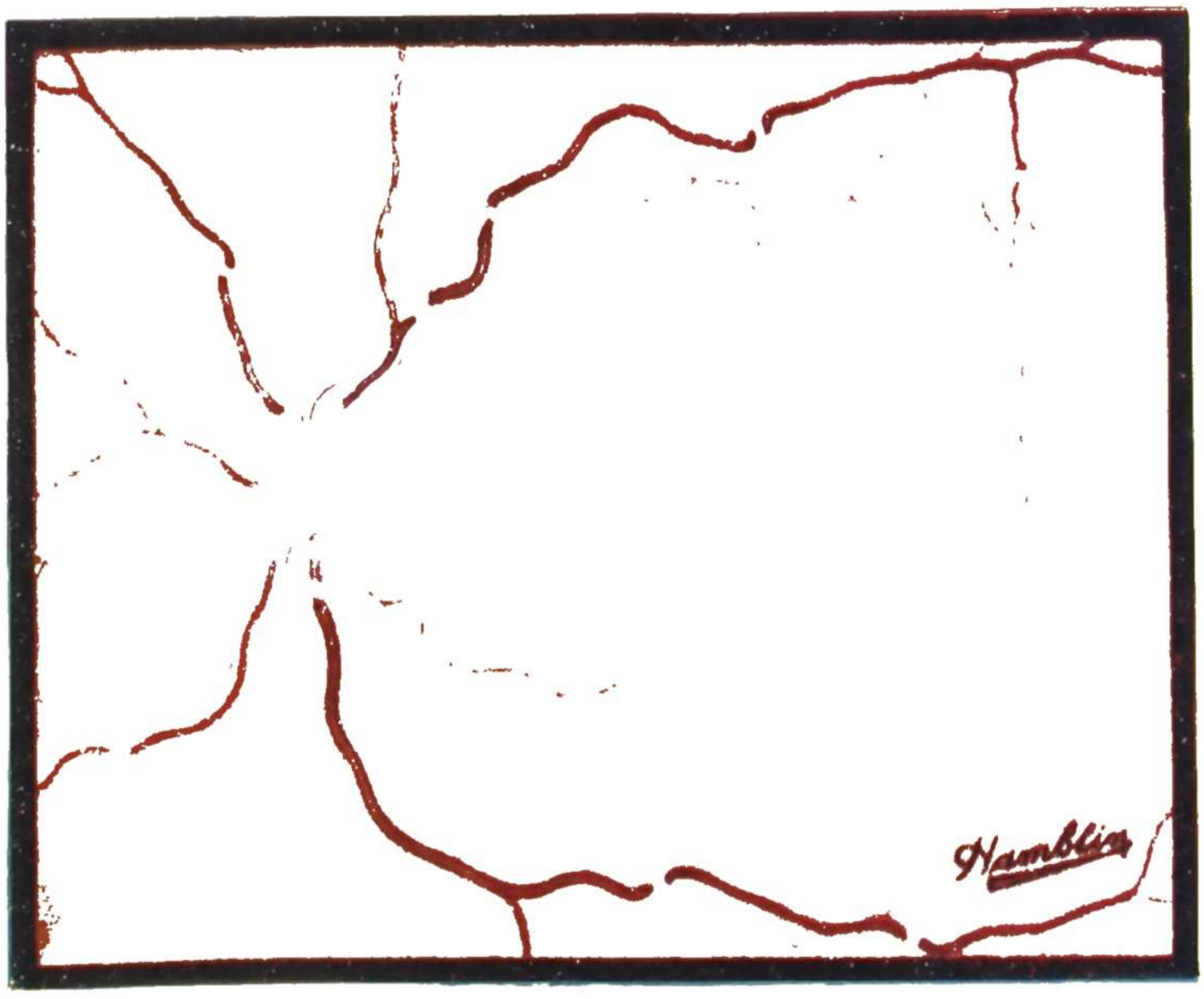
§ **849. Papillœdema** is œdema of the optic nerve at its entrance into the globe, and is evidenced, in a typically marked form, by blurring of the edges, swelling, and increased vascularity of the disc (Plate VI.4). The arteries become narrower, and the veins are enlarged and tortuous, the vessels curving over the œdematous edge. The vessels, moreover, may appear broken here and there, as they are hidden by the œdema. In the early stage the disc has simply a fluffy look, and then the upper and lower edges only are blurred. These œdematous changes may gradually subside, or go on to "consecutive" atrophy (Plate VII.6). It should be remembered that the *acuity of vision may be undisturbed*, even when there is considerable papillœdema, though the visual field is usually diminished in some degree. Disturbance of vision is generally more marked as the acute stage subsides. In papillœdema with preservation of vision the pupils react to light. *Bilateral papillœdema* is very strongly suggestive of intracranial disease, and especially (i.) intracranial tumour, in which it is present at some time in about 85 per cent. of the cases. It is especially common in cerebellar tumour. It is rare in cerebral hæmorrhage and embolism. (ii.) Increase of pressure from other causes—*e.g.*, subarachnoid hæmorrhage or hypertensive encephalopathy. It is uncommon in acute meningitis. Syphilis may produce papillœdema. *Unilateral papillœdema* may indicate disease in the orbit or behind the orbital fissure, *e.g.*, from orbital tumour, exophthalmic ophthalmoplegia, aneurysm of the internal carotid, infections.

§ **850. Optic Neuritis** or **Papillitis** may develop at any part of the optic nerve and is usually associated with loss of acuity of vision and central field defects. It is visible ophthalmoscopically only when the optic papilla is involved. Such cases are called intra-ocular neuritis, or because of changes in the papilla, papillitis. Distinguish from these the cases in which the inflammation is located in the optic nerve farther back (retrobulbar neuritis). Since in retrobulbar neuritis the focus of inflammation cannot be seen, its diagnosis is inferred from other symptoms. Various toxic conditions of the blood may cause Papillitis, chief among which is renal disease, giving rise to a special form (see § 853 and Plate VI.3). Papillitis occurs in intracranial inflammations, *e.g.*, syphilis, abscess. Plumbism, anæmia and disseminated sclerosis are occasional causes, as are toxic encephalomyelitis and the encephalomyelitis of acute specific fevers.

§ **851. Retrobulbar Neuritis** is an inflammation of the optic nerve behind the nerve head. The symptoms are: (1) *Sudden* loss of vision in one eye, with (2) aching in the orbit and tenderness of the eyeball, (3) central scotoma, and (4) defective vision for red and green. (5) The pupil tends to be dilated and reacts poorly to direct light, but reacts normally to consensual illumination; it reacts normally on

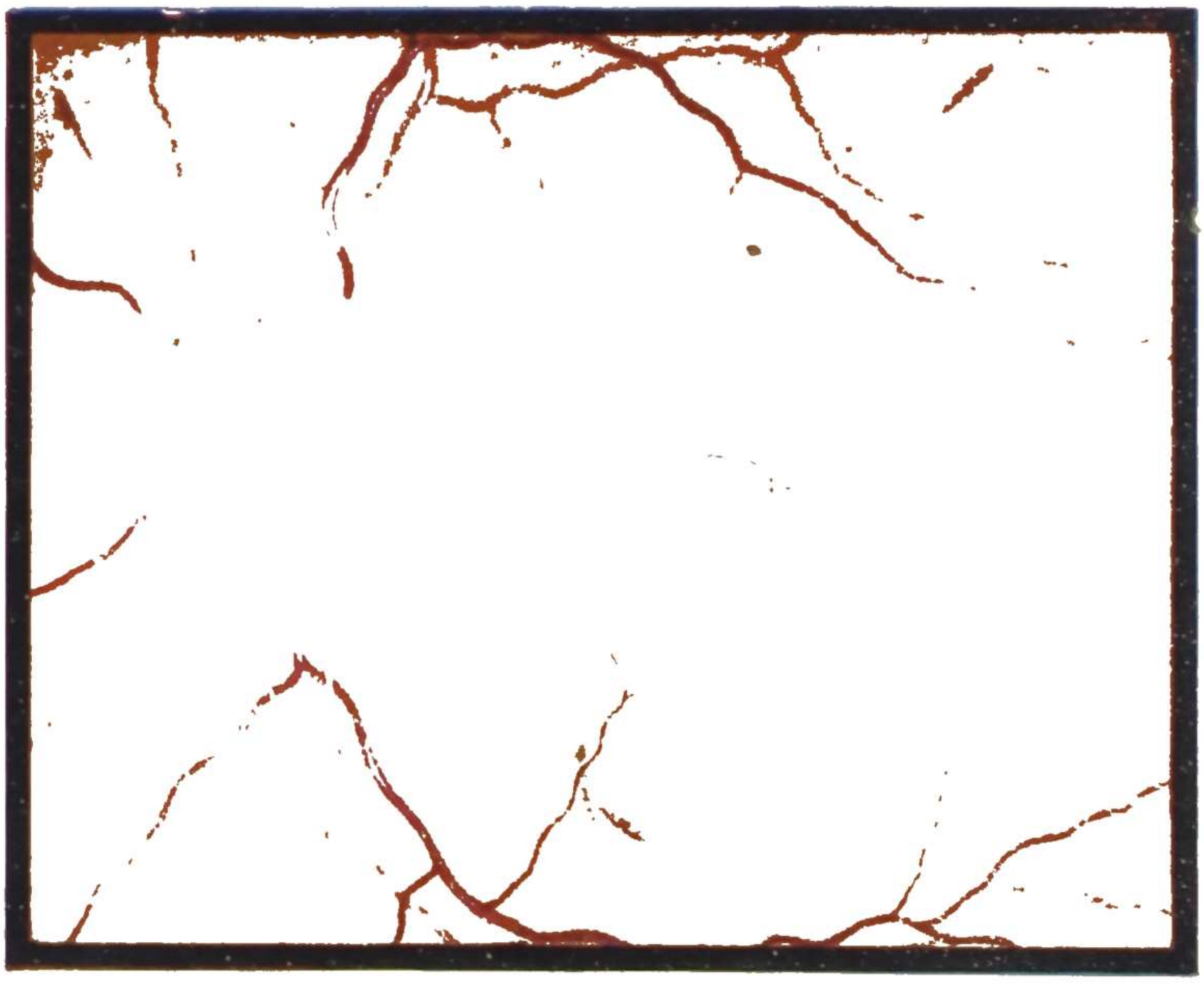
PLATE V.

1. NORMAL.



2. ARTERIO-SCLEROSIS.

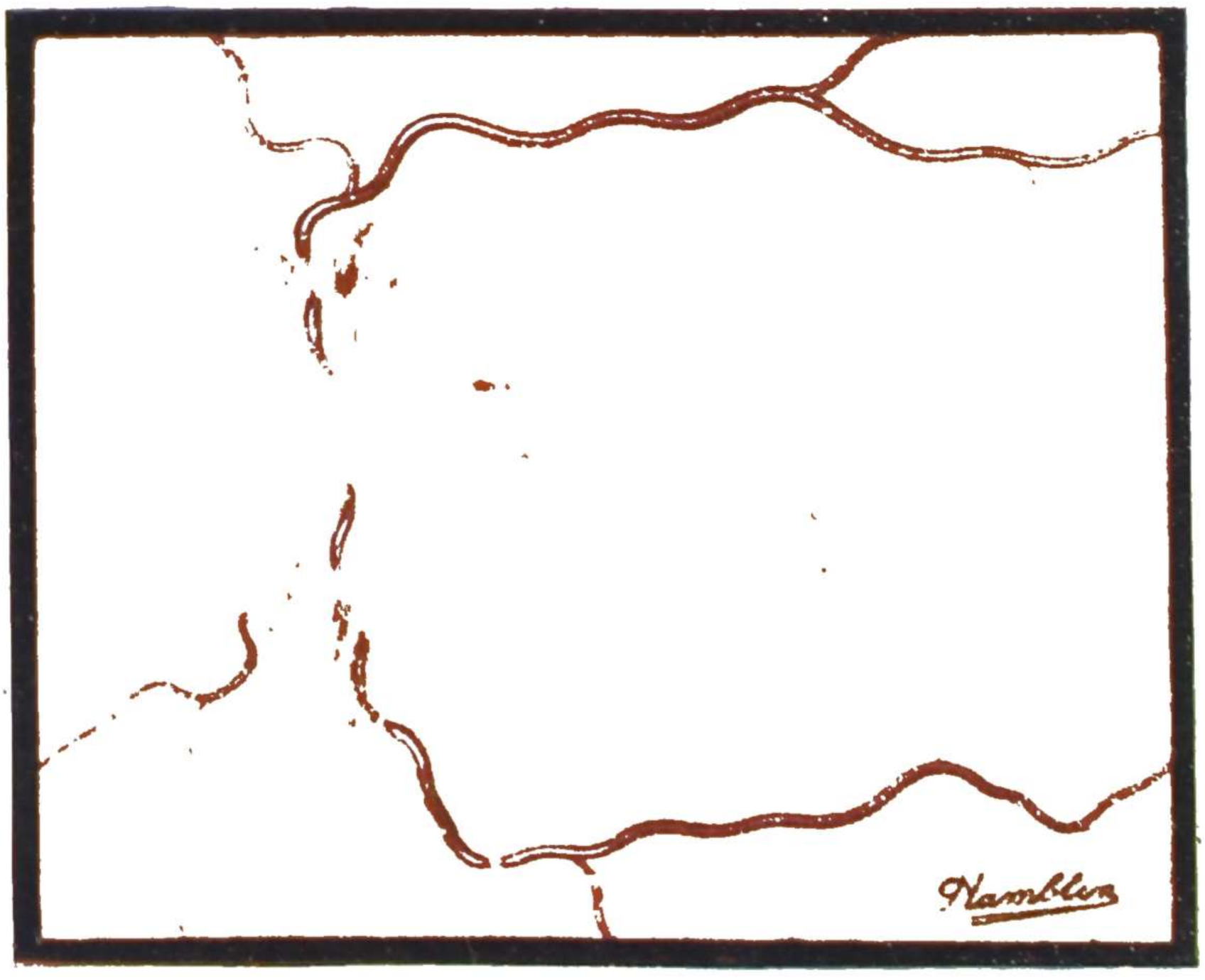
Arteries tortuous and irregularly narrowed, with light reflex marked, and loss of transparency. Veins kinked and their course deviated where crossed by arteries.



3. CHRONIC NEPHRITIS.

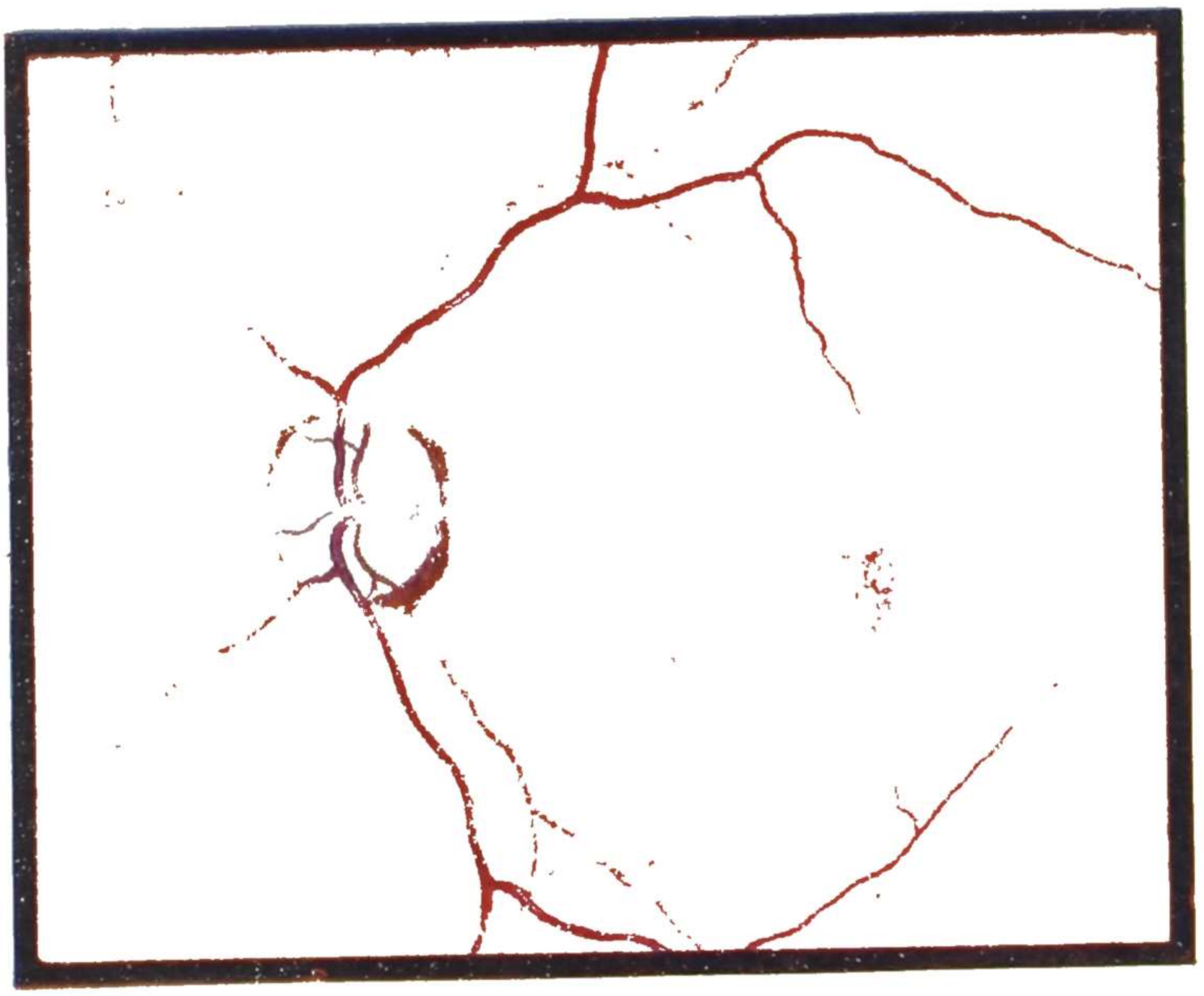
Later stage.

Disc—Swollen and margin blurred. Macula—Radiating lines of white dots resembling the Star of India. Fundus —Flame-shaped hæmorrhages disposed radially to the disc; White woolly patches.



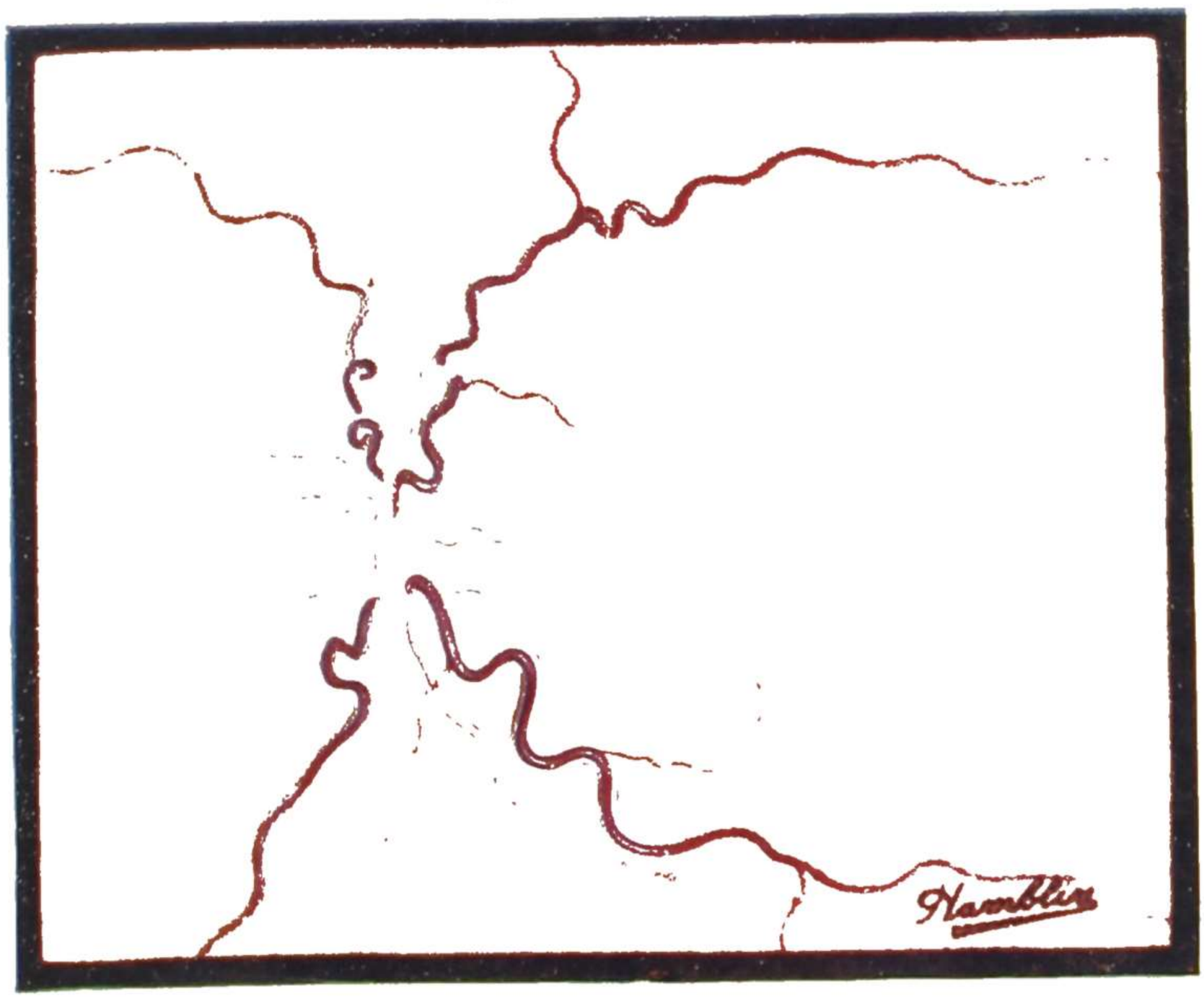
4. PAPILLŒDEMA.

Mushroom-like swellings of disc with blurring of margins due to lymphatic stasis engorgements of veins. Vessels bent by swelling and partly covered up. Hæmorrhages variable. No loss of visual acuity in early stages.



5. PRIMARY OPTIC ATROPHY.

Clear cut blue white disc with fine vessels.



6. SECONDARY OPTIC ATROPHY.

White disc blurred by deposition of fibrous tissue which may extend along the vessels, giving them a fibrous sheath. Tortuosity of vessels near disc.

accommodation. The symptoms may be transient and last only a few weeks, although a permanent scotoma and atrophy may follow. The commonest *cause* is (1) disseminated sclerosis. The condition may rarely occur as a complication of (2) suppuration in the sphenoidal or ethmoidal air sinuses. Retrobulbar Neuritis, coming on gradually, may be due to (3) neuromyelitis optica, (4) tobacco or alcoholic poisoning (especially wood alcohol), (5) syphilis, (6) diabetes and nutritional disorders, (7) Leber's hereditary optic atrophy (§ 852).

§ **852. Optic Atrophy** (Plate VII).—*Primary* Atrophy is characterised by porcelain-like-pallor of the disc and sharply defined outline. The best example is tabetic atrophy, but atrophy in disseminated sclerosis and that following various forms of retrobulbar neuritis are included, though the atrophy is not strictly primary. Optic atrophy may be secondary to retinal disease such as retinitis pigmentosa or other forms of extensive choroido-retinal disease. In these cases the disc is a waxy yellow colour. When atrophy follows papillœdema from any cause, or papillitis, it is termed *secondary* or *consecutive;* the disc is white, opaque, the edges are blurred and the vessels sheathed in fibrous tissue to an extent varying with the severity of the primary lesion.

Leber's Hereditary Optic Atrophy is a hereditary type of optic atrophy affecting males and females but transmitted through the females alone. It commences as a retrobulbar neuritis, soon after puberty, with central scotoma and slight swelling of the disc. The atrophy and visual failure are rarely complete; often useful vision is retained. The malady is probably analogous to Cerebro-macular Degeneration (see § 763).

§ **853. Retinitis.** Most affections of the retina are described as retinitis, though few of them are true inflammations. Œdema of the retina, whatever the cause, involves loss of transparency as is seen *par excellence* in embolism of the central artery; it is also a marked feature of severe albuminuric retinitis. Other important features of retinal disease are hæmorrhages, exudates of various kinds, aggregations of pigment (superficial to the retinal vessels) and changes in the vessels themselves. Retinitis is symptomatic of disease, and nephritis is the most important cause. The retina is essentially a vascular structure and presents direct evidence of high blood pressure and arterio-sclerosis; this leads eventually to arterio-sclerotic retinitis. It also shows numerous manifestations of general arterio-vascular disease and its complications, as for example, thrombosis of a retinal vein. Retinitis occurs also in syphilis, diabetes, leukæmia and in septic states of the teeth, tonsils and other parts. The picture depends on the stage at which the examination takes place, on the age of the patient and on the condition of the blood vessels at the time of onset of the retinal disease. Retinitis causes no pain but a patient may complain of entoptic flashes or intolerance of light.

Detachment of Retina.—In practically all cases a hole is found in the retina. The method of treatment involves the production of an adhesive choroiditis around the hole by means of coagulation or perforating diathermy, electrolysis or a combination of these coupled with evacuation of the subretinal fluid. Detachment complicating inflammatory disease is not suitable for such treatment: solid detachment due to an underlying new growth may be accompanied by a fluid detachment.

Albuminuric Retinitis (Plate VI) is really a neuro-retinitis, consisting of three elements. (i.) Papillitis (see above); (ii.) hæmorrhages into the retina, usually flame-shaped and most plentiful towards the disc; and (iii.) fine white spots near the macula, and large woolly patches on the retina. One or other of these is sometimes wanting, but in its typical form this kind of retinitis is sufficiently distinctive to diagnose renal disease without examining the urine. It may occur in any form of renal affection, but is frequently associated with chronic nephritis. Albuminuric retinitis is of very grave significance, the patient seldom surviving more than six months after the diagnosis is made; albuminuric retinitis of pregnancy is an exception to this, complete recovery often following parturition, normal or induced. **Hæmorrhages** into the retina and choroid are met with as dark red patches. They accompany any severe retinitis papillitis or papillœdema, particularly albuminuric retinitis, diabetes

and hypertension with arterial degeneration: they are also met with in pernicious anæmia, leukæmia, pyæmia, malaria, scurvy, and other purpuric and toxic conditions. **Embolism of the Central Artery of the Retina** occurs most frequently in the course of cardiac disease, with vascular sclerosis, especially in disease of the aortic valves, in auricular fibrillation and ulcerative endocarditis and in pregnancy where the embolus may be a fragment of a chorionic villus. An embolus may be arrested in a branch of the central artery or in the main trunk, causing immediate loss of part or the whole of a visual field in one eye. On examination, the retinal vessels are found empty, the retina opaque, and a peculiar round, cherry-red spot is seen in the macular region. The disc is pale. Thrombosis of the central vein similarly causes sudden but incomplete blindness. **Retinitis Pigmentosa** is characterised by narrowing of the lumen of the retinal and choroidal vessels, secondary atrophy of the optic nerve with a waxy looking disc and the deposition of aggregations of pigment, particularly in the periphery, which resemble bone-corpuscles. Night-blindness is the chief symptom; the disease begins in childhood.

§ **854. Choroiditis Disseminata** (usually bilateral, though sometimes limited to one eye) is frequently an evidence of acquired syphilis (in which case it arises three months to three years after infection): or of congenital syphilis. On examination, discrete, white, atrophic patches, with irregular black edges, are found scattered over the fundus, *most marked at the periphery*. This condition has to be differentiated from the slow type of choroido-retinitis. **Acute tuberculous lesions** may be found as yellowish ill defined spots in the choroid in tuberculous meningitis. **Atrophic Patches in the Choroid,** with retinal involvement, occur in the secondary changes of progressive myopia: the macular region is apt to suffer.

§ **855. Glaucoma** is a term applied to many conditions of the eyeball having one common feature, increased tension. There are three main varieties: (1) primary, (2) secondary, (3) infantile. (1) For *primary glaucoma* there are many theories but none generally accepted: whatever the cause, secretion of aqueous into the eye exceeds excretion from it, there is a tendency for dilatation of the pupil and, in the later stages, the filtration angle is more or less closed by adhesion of the peripheral part of the iris to it. The old operation of iridectomy is replaced by the modern operation of trephining the corneo-scleral junction with iridectomy: some prefer iridenclesis, iridodialysis or a flap sclerotomy. The alternative to operation, which should always be given a trial in mild cases, is the instillation of pilocarpine or eserine drops, the latter being stronger, in the lowest concentration compatible with the attainment of normal tension and stationary fields of vision.

Chronic glaucoma is characterised by reduction of the field, particularly a nasal "bite" and enlargement of the blind spot with cupping, increasing pallor and, finally, atrophy of the disc, the atrophy being an ascending one from the moribund ganglion cells. Emotional disturbances may play a part in the etiology.

Acute primary glaucoma may come on suddenly or complicate a chronic case: extreme pain, rapid loss of sight and vomiting are found, and the eye may be lost in twenty-four hours. Owing to the abdominal symptoms, such cases have been operated on as appendicitis. Treatment is extremely urgent—eserine repeatedly instilled, fomentations, leeches and purgation. Operation—a large iridectomy—should be performed as soon as possible.

(2) *Secondary glaucoma* sometimes complicates iritis and cyclitis, and is due either to complete posterior ring synechiæ damming up the fluid in the posterior chamber with iris bombé and an idle excreting mechanism, or to the increased protein content and viscosity of the aqueous fluid interfering with the proper functioning of the filtration apparatus in the Spaces of Fontana and Canal of Schlemm. Operation in these cases, where necessary, is iridectomy or multiple perforations of the bombé iris with a Grafe knife. Secondary glaucoma may follow herpes ophthalmicus and thrombosis of the central veins.

(3) *Infantile glaucoma* or *buphthalmus*, fortunately rare, is due to faulty development of the angle of the anterior chamber with poor excretion and increased tension:

the globe enlarges as the pressure increases. Trephining offers the only hope, but the prognosis is bad.

§ 856. The Trigeminal Nerve.—The *motor* and *sensory* nuclei of this, the largest of the cranial nerves, have been described in § 683. The motor and sensory roots leave the ventro-lateral surface of the pons in a sheath of dura mater called the cavum Meckelii. In this cave, on the tip of the petrous portion of the temporal bone, lies the trilobed Gasserian ganglion. The ganglion divides into three nerves—(1) The Ophthalmic division, which enters the orbit; (2) The Maxillary division, which leaves the skull by the foramen rotundum; and (3) The Mandibular division, which is joined by the motor root and leaves the skull by the foramen ovale. The first two divisions are purely sensory, the third is a mixed nerve.

(1) *The Ophthalmic division* supplies the eyeball and conjunctiva (corneal reflex), the skin of the forehead and scalp up to the centre of the vertex, a median cutaneous strip on the nose, the meninges and the mucous membrane of the upper nasal cavity and lachrymal glands (Fig. 186).

(2) *The Maxillary division* supplies the skin of the face on the lateral aspect of the nose, on the cheek, from the upper lip to the lower eyelid inclusive and, laterally, as far as the pinna, the upper jaw and its teeth, pharynx and tonsil, and the lower part of the nasal cavity.

(3) *The Mandibular division* supplies the skin of the posterior aspect of the temple, the upper part of the pinna and side of the face (not the angle of the jaw, C1), the tongue, lower cheek and gums and the Eustachian tube. The motor fibres supply the masseter, temporal, pterygoids, mylohyoid, anterior belly of digastric, tensor palati and tensor tympani muscles.

The methods of examination are described in § 703.

Symptoms.—Lesions of the fifth nerve produce sensory loss, diminution or loss of corneal and conjunctival reflexes on the side of the lesion, and paræsthesiæ over the anatomical distribution of the nerve and, if the motor root is involved, paralysis of the muscles of mastication. Slowly progressive lesions are painless and produce, as their earliest sign, diminution of the corneal reflex. Wasting of the masseter and hollowing of the temporal fossa often precede loss of power in the muscles affected.

Etiology.—In its intracranial course the nerve may be involved by (1) Extra-cerebellar tumours, (2) Secondary growths at the base of the skull, (3) Meningitis at the tip of the petrous bone in chronic suppurative middle-ear disease, or (4) Basal Syphilitic Meningitis. In Tabes Dorsalis a "butterfly" zone of analgesia is common over the nose and cheeks (masque tabétique), while in Syringomyelia, involvement of the spinal root leads to a zone of analgesia on the periphery of the face.

Herpes Ophthalmicus is an inflammation of the Gasserian ganglion, due to the virus of herpes zoster (§ 826). It is a disease of maturity, but may occur in young adults. Vesiculation and pain occur over the distribution of the ophthalmic division only. Keratitis and corneal ulceration may be present, and vesicles occur also in the upper nasal cavity. The vesiculation in the forehead may be followed by permanent scarring and often by intractable supra-orbital neuralgia and sensory impairment. The trophic corneal lesions may cause blindness. External ocular palsies, pupillary abnormalities, even optic atrophy, may accompany the condition and there may be associated iritis.

§ 857. Progressive Facial Hemiatrophy.—This rare condition is characterised by atrophy of the skin and its appendages, subcutaneous tissues, muscles, bones and cartilage, within the territory of the trigeminal nerve. The eye may be involved.

The malady comes on in early life and is commoner in females. There is no sensory loss or paralysis.

TRIGEMINAL NEURALGIA (see § 822).

§ 858. The Facial Nerve.—Of all the nerves of the body the facial is the most frequently affected by paralysis. It is peculiar in having a long tortuous course through a bony canal, the aqueductus Fallopii. During the onset of slow paralysis, or during slow recovery, clonic or fibrillary twitches may be observed in the paralysed muscles: these are seen in the case of no other nerve in the body.

Motor and *sensory* roots are described. The *motor root* arises from a nucleus in the lower part of the pons and the fibres bend round the sixth nucleus before emerging from the brain-stem at the junction of pons and medulla. The nerve passes forwards

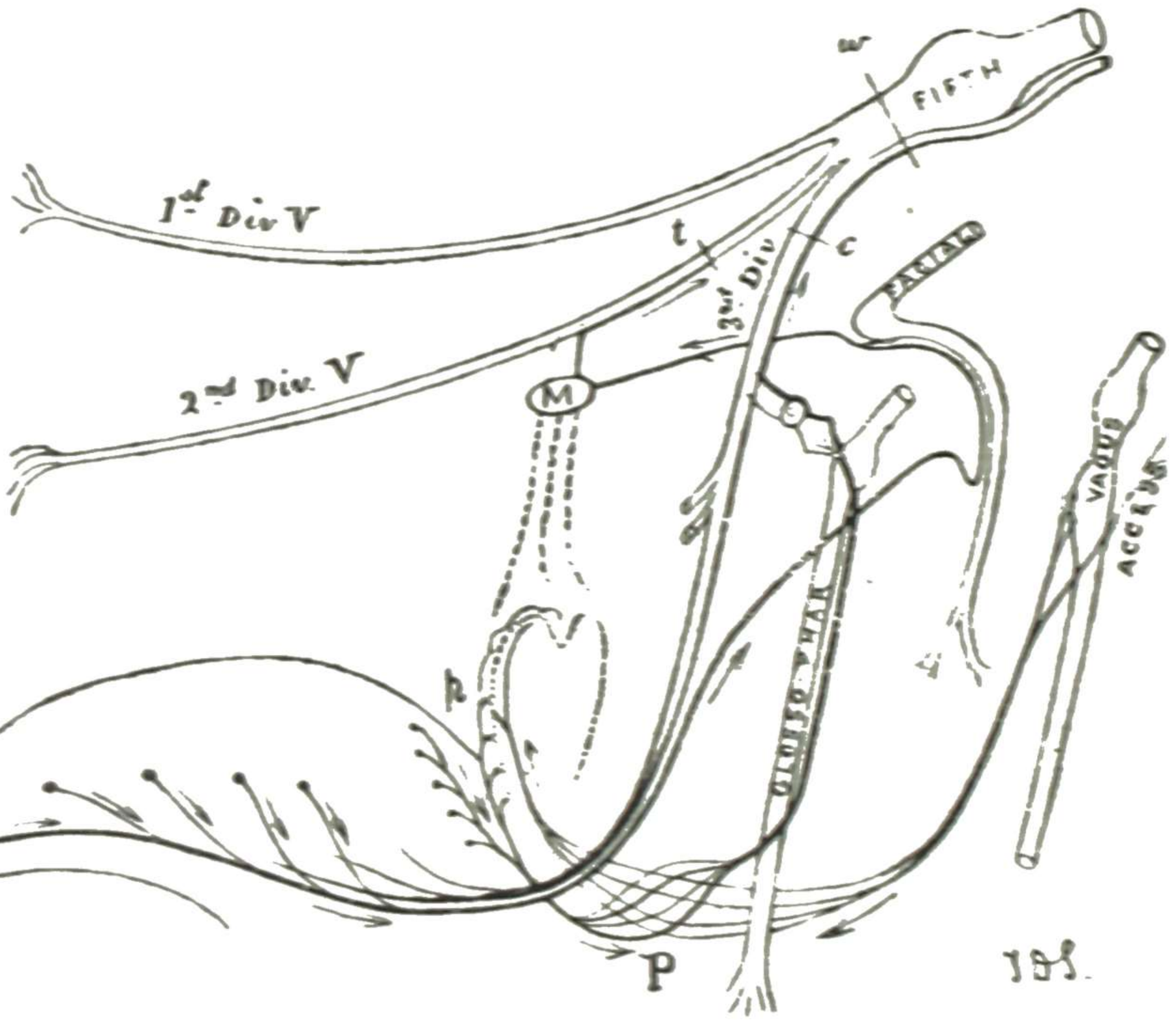


FIG. 193.—SENSE OF TASTE and NERVE-SUPPLY OF PALATE.—Diagram to show how taste impressions reach the fifth nerve ⟶, and the motor-supply of the palate ⟵. T, branches of the chorda tympani conveying taste from the tip and sides of the tongue, and running with the lingual branch of the fifth, then through the facial to Meckel's ganglion (M), and thence to the second division of the fifth. *p*, soft palate from which, and from the dorsum of the tongue, taste fibres pass through the pharyngeal plexus (P) to join the glosso-pharyngeal, and thence through the otic ganglion (*o*) to the third division of the fifth. The palate, it will be seen, is supplied by the accessory portion of the spinal accessory through the vagus.

and outwards with the auditory nerve to enter the internal auditory meatus, where it is joined by the sensory root (n. intermedius). In the aqueduct of Fallopius, the nerve pursues a curved course. To it is attached the geniculate ganglion of the n. intermedius. The nerve within the petrous temporal bone gives off a nerve to the stapedius muscle and runs down behind the tympanum. A quarter of an inch above its emergence from the stylo-mastoid foramen, it gives off the chorda tympani to join the lingual. On emerging from the stylo-mastoid foramen the nerve passes forwards in the substance of the parotid gland to supply all the facial muscles of expression and the platysma. The *sensory root* (n. intermedius of Wrisberg) is really a separate nerve with its own ganglion (geniculate ganglion) functionally distinct from the facial. From the geniculate ganglion, fibres run centrally to a nucleus

in the pons (n. gustatorius) and peripherally with the facial, then into the chorda tympani with which they are distributed to the anterior two-thirds of the tongue, supplying this with taste fibres (Fig. 193).

The METHODS OF EXAMINATION are described in § 703.

Symptoms.—Supranuclear affections of the facial nerve produce weakness of the lower face only (see § 683). These are described under Hemiplegia (§ 752). The facial nerve may be affected in four situations:

(1) *After its exit from the stylo-mastoid foramen*, lesions produce paralysis of voluntary and emotional movements of the facial muscles, with loss of tone and diminished reaction to faradic stimulation. This may occur from toxic or infective neuritis, injuries, or tumours of the parotid.

(2) *Within the aqueductus Fallopii* a lesion produces similar effects, but accompanied by loss of taste on the anterior two-thirds of the tongue on the affected side, from involvement of the taste fibres in the geniculate ganglion. This may follow toxic or infective neuritis, injury, meningitis from caries of the petrous bone in suppurative otitis media, or pressure from bony or cholesteatomatous growths in the middle ear. Such palsies are often slow in onset and accompanied by facial hemispasm.

(3) *Between the pons and the internal auditory meatus* lesions cause purely motor symptoms, and from the proximity of the auditory nerve tinnitus or deafness. The common lesion is an extra-cerebellar acoustic neurofibroma with occipital pain (§ 814).

(4) *Within the pons* lesions again cause purely motor symptoms. There is usually an associated diplopia and ipsilateral external rectus palsy, from paralysis of the neighbouring sixth nucleus, and extensor plantar responses may be present from pyramidal involvement.

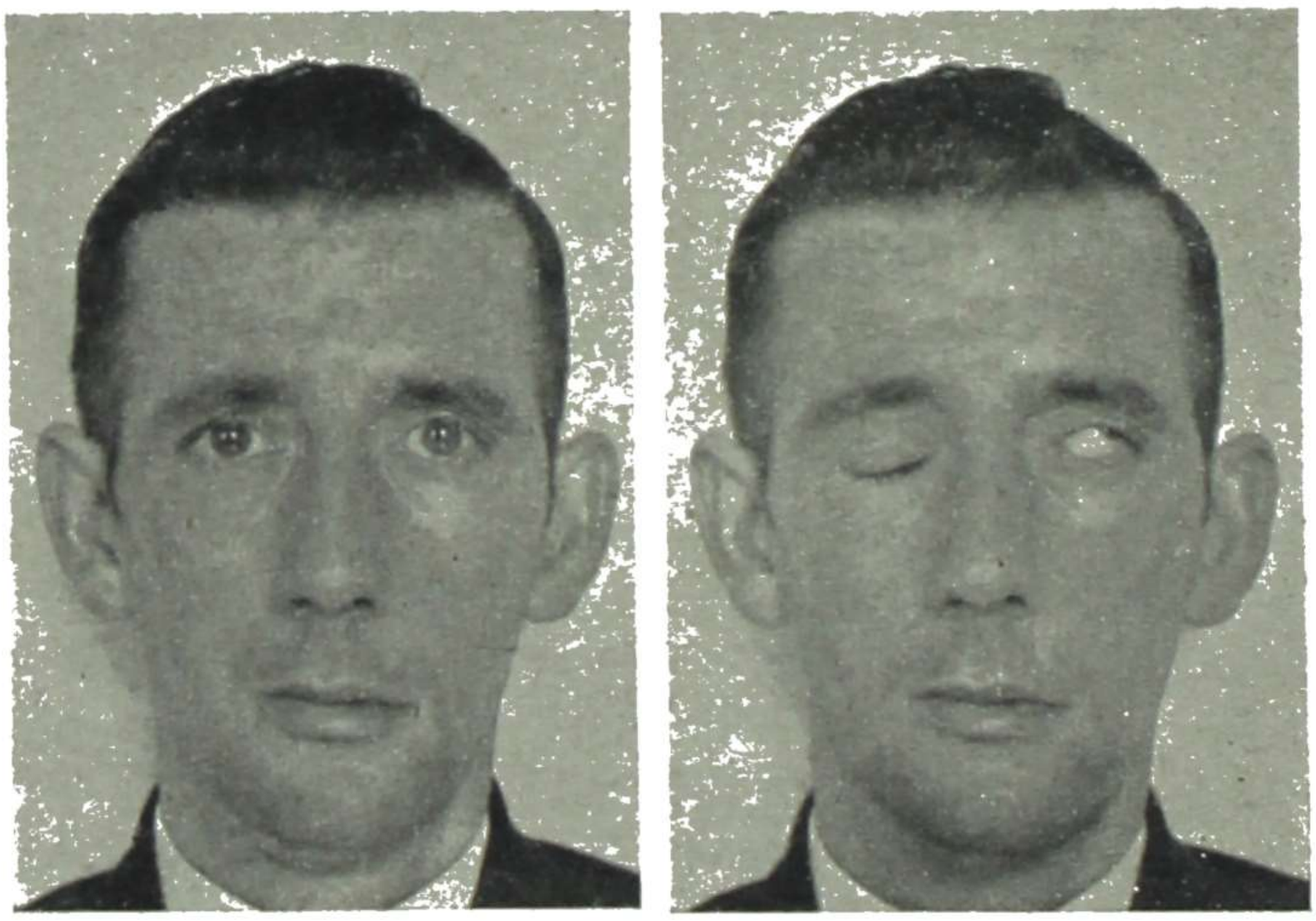
§ 859. Bell's Palsy.—The onset is rapid and there is commonly a history of exposure to cold. Pain and muscular tenderness below and behind the ear, and even swelling in the neighbourhood of the parotid, may precede the palsy and persist for three or four days. The patient notices stiffness of the affected side of his face, he cannot close his eye properly, and tears trickle from the flaccid lower lid. Articulation is indistinct at first and fluids are spilled in drinking. In severe cases, there is complete immobility of the upper and lower face on the affected side and no voluntary or emotional movement is possible. The muscles are toneless, and, especially in old people, epiphora occurs from paralysis of the tensor tarsi. In severe cases, there is nearly always loss of taste on the lateral edge of the anterior two-thirds of the tongue. When the patient attempts to close his eyes, the globe on the affected side turns upwards and outwards more than on the normal side (Negro's sign).

Secondary Contracture occurs in the paralysed muscles, after some months, in severe cases. The unsightly distortion thus produced leads to narrowing of the ocular fissure and exaggeration of the naso-labial fold, and drawing up of the angle of the mouth, so that the healthy side appears the weaker. In other cases *distortion of facial expression* occurs from misdirection of the regenerating fibres so that corresponding nerve bundles

do not supply the same muscles on the two sides of the face. Both defects are permanent.

Diagnosis.—Peripheral facial paralysis may occur in poliomyelitis, diphtheria, tetanus and encephalitis lethargica. When the paralysis is due to petrous temporal disease, deafness, otorrhœa and perforation of the tympanum will co-exist. Lesions within the skull (acoustic neurofibroma) are accompanied by deafness, cerebellar ataxia and diminished corneal reflex from trigeminal involvement. Lesions in the pons cause associated diplopia, hemiplegia, hemianæsthesia or hemiataxia.

Prognosis.—Most cases clear up in a few weeks or a few months. The *electrical reactions* of the paralysed muscles should not be tested until a fortnight has elapsed from the onset of the paralysis: by this time all



(*a*) (*b*)

FIG. 194.—CASE OF LEFT-SIDED FACIAL PALSY.—(*a*) At rest. Observe the facial asymmetry with widened ocular fissure and drooping of the angle of the mouth on the left side. (*b*) On attempting to shut the eyes the patient merely rolls the eyeball upwards and outwards on the affected side so that the cornea passes under cover of the upper lid.

degenerating fibres will be destroyed and the results will give valuable information. If at the end of four weeks the muscles do not react to faradism, recovery is likely to be incomplete and long delayed (§ 709). In almost all cases, however, recovery occurs, and this is true also in traumatic cases and obstetric cases resulting from the pressure of forceps during delivery. Where taste is intact, recovery may be expected within three months. Profound paralysis and flaccidity of muscles at the onset is of bad prognostic origin. If after three to six months there is little recovery, secondary contracture or distortion of facial expression are likely to occur in varying degree.

Etiology.—Bell's palsy is believed to be due to an infection allied to that of herpes zoster. Other cases of facial palsy are due to otitic and other infections or to new growths.

Treatment.—The patient should be given a peroxide mouth-wash to

use after meals to get rid of food particles which collect between the cheek and the gum. When the lids cannot be closed, in order to prevent exposure conjunctivitis, the eye is bathed night and morning with boric lotion. The patient should be instructed to wipe his eye from below upwards, so as to lessen the danger of epiphora. Voluntary re-educative exercises should be practised for ten minutes by the clock, night and morning, in front of a mirror. The patient attempts to screw up his eyes, wrinkle his forehead, show his teeth, blow out his cheeks, whistle, etc. A piece of copper wire, covered with rubber tubing, can be bent over one ear to hook up the angle of the mouth on the flaccid drooping side. This should be worn at night. Facial massage is of use, radiant heat in skilled hands is of value. Iodides or salicylates may be given internally. Cases due to mastoid or middle ear disease rarely show any recovery and, after a year, an attempt may be made to anastomose the peripheral cut end of the facial to the proximal cut end of the hypoglossal or spinal accessory nerve. Duel and Ballance suggest decompression of the nerve in the aqueduct. In intractable cases the deformity may be considerably lessened by a plastic operation in which an attempt is made to support the paralysed muscles by fascial slings or strips inserted subcutaneously.

Bilateral Facial Palsy.—It may occur in Bell's palsy, with basal gummata or secondary deposits, in diphtheritic and alcoholic polyneuritis and in *uveoparotitic paralysis* (see § 9). Bilateral facial weakness may occur in Myasthenia gravis and in the Myopathies.

Facial Hemispasm (see § 774).

Facial Tics (see § 772).

Geniculate Herpes.—Herpes Zoster (§ 826) may attack the geniculate ganglion, producing vesiculation in the external auditory meatus, hard palate or tonsillar fossa, with severe pain in the ear, and associated facial paralysis. Such cases may be followed by tinnitus or vertigo, which is transient, like the facial paralysis.

The **Auditory** and **Vestibular Nerves.**

§ 860. The Eighth Nerve consists of two separate nerves which are distinct peripherally and have separate central connections. (*a*) The *Auditory* (*Cochlear*) *Nerve*, concerned with hearing, and (*b*) the *Vestibular Nerve*, concerned with equilibrium.

(*a*) *The Auditory* (*Cochlear*) *Nerve* is described in § 675. It is concerned solely with hearing. Lesions of the auditory nerve produce deafness and tinnitus.

(*b*) *The Vestibular Nerve* originates in the ganglion of Scarpa, which lies at the outer end of the internal auditory canal. Peripherally this ganglion sends filaments to (1) the semicircular canals, and (2) the otolith organs (utricle and saccule). The semicircular canals are stimulated by movements, the otolith organs by alteration in position of the head. The central connections are described in § 680. This nerve is concerned with the mechanism of equilibrium. Lesions of the vestibular nerve or semicircular canals produce Vertigo (see § 692).

Clinical Investigation.—*Hearing* can be tested by the voice, the watch, and the tuning-fork—the two former by air-conduction, the last by bone-conduction and air-conduction; with the audiometer more exact testing and the recording of hearing is now possible. Wax in the external meatus must be first removed, and if necessary the ear should be syringed with warm water, after the wax has been softened by warm oil, or bicarbonate of soda solution (2 teaspoonfuls to the pint). To test

the *acuteness* of *hearing* by *air conduction*, stand behind your patient, close one of his ears with one of your hands, and place a watch in the other hand outside the range of his hearing, then approximate it slowly, asking the patient to say directly he hears the tick, and then estimate the distance. Examine the other ear in the same way. Ascertain on yourself what is the normal distance at which that particular watch should be heard, and supposing this is 60 inches, and the patient hears with the left ear at a distance of 5 inches, and with the right at 60 inches, then the acuteness of his hearing in the left ear is represented by the fraction $\frac{5}{60}$. The speaking and the

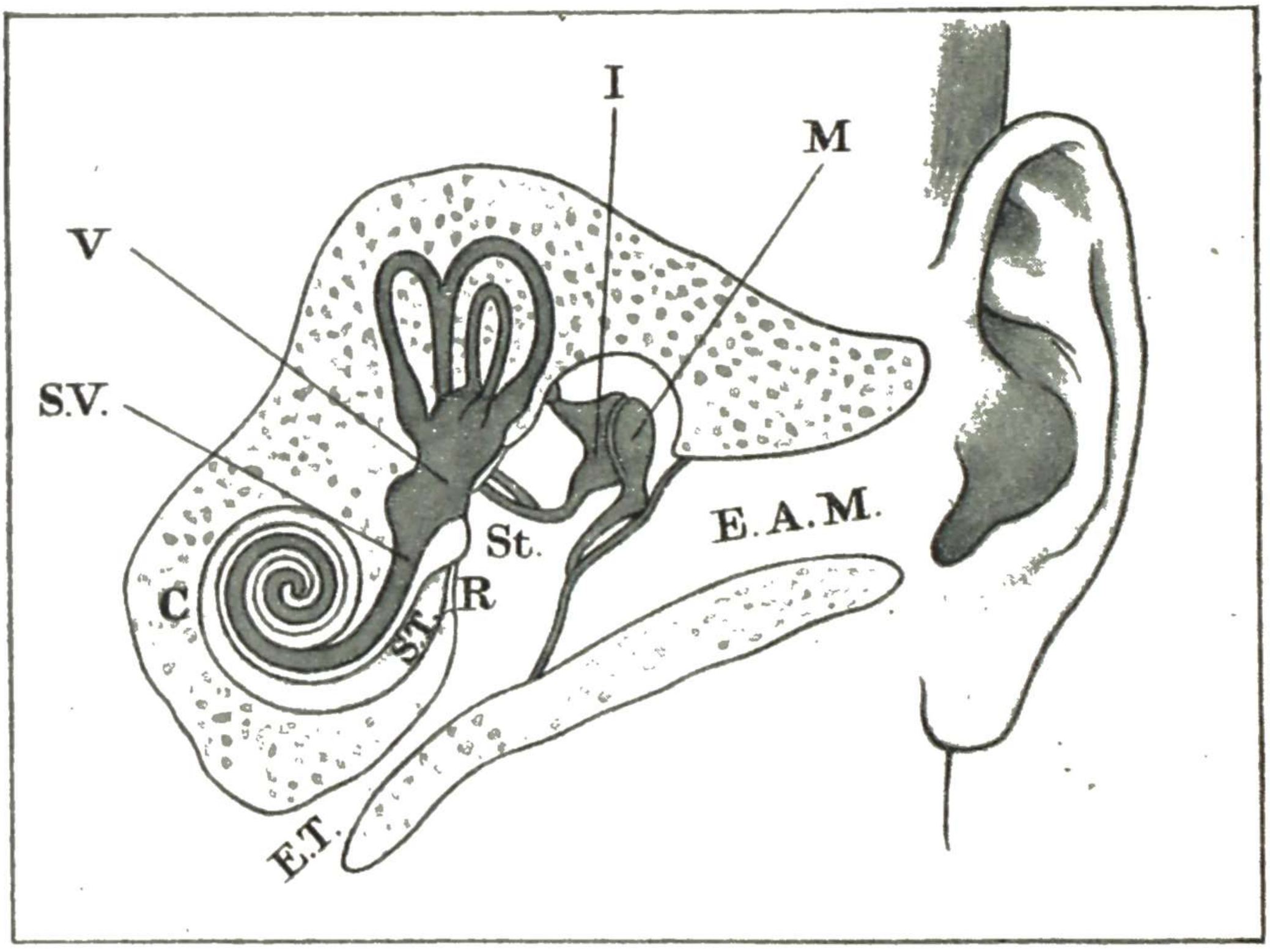


FIG. 195.—AUDITORY APPARATUS (diagrammatic representation) of the left side seen from the front, the internal parts being magnified two-fold.

E.A.M., External auditory meatus, separated by tympanic membrane from tympanum in which R. is situated.
M., on head of Malleus.
I., Incus fixed to wall by its short process, and articulating with the stapes by its long process.
St., Stapes, the foot of which fits into the fenestra ovalis.
V., Vestibule consisting of saccule (below) and utricle (above). Into the latter open the three semi-circular canals, superior, posterior, and external (or horizontal). The vestibule leads on to the scala vestibuli (S.V.) of the cochlea (C.).
R., Fenestra rotunda leading from the scala tympani (S.T.) to tympanum.
E.T., Eustachian tube.

whispering voice are very useful for testing hearing. Each ear is tested separately, the other for the time being closed by the patient's finger. Various words are used; the observer gradually approaches until the patient can repeat them without mistakes, and the distance is noted. To test by *bone-conduction* (*Schwabach's test*), a tuning-fork in vibration is placed with its stem on the patient's mastoid process, and when he ceases to hear it, it is applied to the observer's mastoid. If the observer still hears the sound, there is said to be diminution of bone-conduction. On the other hand conduction may be lengthened. A vibrating tuning-fork is held on the observer's mastoid till it ceases to be heard; it is then transferred to the patient. If the patient still hears the sound, bone-conduction is longer than normal (assuming that the

observer's hearing is normal). A watch applied to the mastoid may be used in the same way to give an idea of bone-conduction.

Weber's Test.—To ascertain whether an impairment of hearing is due to *nerve deafness* or to *obstructive deafness*, test the per-osseus hearing by placing a watch or a vibrating tuning-fork on the centre of the patient's forehead. If the deafness is due to disease of the auditory nerve it will be heard not in the affected ear, but in the good ear. This indicates nerve deafness. If the deafness is due to obstructive ear disease the sound will be heard *on the defective side.*

Rinne's Test.—In a normal person a tuning-fork (256 double vibrations per second) placed on the mastoid bone until no longer heard in that situation, can still be heard by him if removed and held opposite the meatus (Rinne's test positive), hearing by air conduction being more prolonged than by bone-conduction ; it indicates an absence of any considerable middle-ear disease. When the middle ear or conducting apparatus is definitely diseased the tuning-fork cannot be heard opposite the meatus after it has ceased to be heard when held on the mastoid (Rinne's test negative). If Rinne's test is positive in a deaf ear there is probably nerve deafness present.

Galton's Whistle and the *monochord* are used for testing the upward limit of audition of a patient. Diminution of audition for highly-pitched notes occurs in old age and in nerve deafness. The introduction of the *audiometer* enables hearing to be accurately tested and recorded. *Paracusis Willisii*, "hearing best in a noise," is a characteristic of bilateral middle-ear disease, especially oto-sclerosis, and is usually associated with fixation of the stapes. Such patients can hear conversation better in a train or omnibus than otherwise. In boiler-makers' and some other forms of nerve deafness the converse is true.

(*b*) *Vestibular Nerve.*—The condition of the vestibular system can be ascertained by endeavouring to produce nystagmus by syringing with cold water and hot water, or by rotating in a rotating chair. Normally, syringing with cold water (30° C.) induces a nystagmus to the opposite side in about forty seconds ; while hot water (42° C.) produces an opposite effect. Cold air may be blown in by Dundas Grant's apparatus. Rotation (ten times in twenty seconds) produces a nystagmus towards the side from which the patient is rotated if the semicircular canals and vestibular nerve are healthy.

INSPECTION OF THE EAR.—Note should be made of any discharge and its character (see below), any pain or tenderness over the mastoid (see below), any eczema of the meatus, etc. To examine the *meatus* (which should be done first without a speculum) the auricle should be pulled gently upwards and backwards, the tragus being pulled forwards by making traction with the thumb on the skin in front of it. If a speculum is to be used, the auricle should be held between the middle and ring fingers of the left hand (for the patient's right ear), the speculum being inserted with the right hand inwards and slightly downwards and forwards. The speculum can then be held between the thumb and forefinger of the left hand. It facilitates examination to have a mirror on one's forehead as in laryngoscopy (§ 164) to reflect the light from a lamp at the side of the patient's head. Cerumen is of dark colour and soft consistence. The membrana tympani may present indrawing (due to blocking of the Eustachian tube), congestion, thickening, or loss of lustre, atrophic areas, or perforations.

The NOSE, THROAT and NASO-PHARYNX should be next examined. Note (1) the activity of the palatal muscles, (2) the Eustachian tubes and back of the nose by posterior rhinoscopy, and (3) the patency of each nostril, (4) the presence of pus or any other abnormality in the nose, (5) the condition of the tonsils.

The PATENCY OF THE EUSTACHIAN TUBE is usually tested by inflation of the middle ear by *Politzer's* method. The nozzle of the rubber bag is inserted into one nostril, and both nostrils are then held close between the thumb and finger of the operator. The patient is then directed to swallow or to say "hic," and at the same moment the air from the rubber bag is forced into the nose. Deglutition raises the palate and opens the Eustachian tube, and the air, having no other outlet, is forced into it. A

tube connecting the ear of the patient with that of the operator will enable the latter to hear an audible " pick " if the middle ear is inflated, and this will reveal the patency of the Eustachian tube. A second point to note is the effect which inflation has upon the symptoms—deafness and tinnitus. The hearing is temporarily improved in middle-ear or Eustachian disease, and is unaltered in otosclerosis and nerve deafness. In *Valsalva's* method of inflation the patient pinches his nostrils firmly, and makes an expiration as if to blow his nose, but without allowing the air to issue. The *Eustachian catheter* is also used to inflate the ear for diagnosis or treatment. It is not a difficult operation, but requires a little practice. Pass it tip downward very gently along the floor of the nose to the edge of the hard palate, the patient being directed to breathe through the nose so that the soft palate may droop. Immediately the tip of the catheter has reached the edge of the hard palate turn it upwards and outwards, and it will enter the Eustachian orifice. It may be aided by the patient swallowing at the same time. The nozzle of Politzer's bag may now be carefully introduced into the catheter, and inflation performed as before.

§ 861. **Causes of Deafness.**—Two kinds of deafness are recognisable: **nerve deafness,** due to lesions of the auditory nerve or internal ear, and **obstructive deafness,** due to some disease in the middle ear or auditory passages.

DIAGNOSIS OF NERVE DEAFNESS AND OBSTRUCTIVE DEAFNESS.

Nerve Deafness.	*Obstructive Deafness.*
Diminution of air and bone-conduction. Positive Rinne; Weber to good ear.	Loss of air-conduction only, with negative Rinne, and Weber to deaf ear.
Loss of hearing for very high-pitched tones (except in hysterical deafness).	Better hearing for high than for low tones.
Decreased hearing in midst of noise.	Better hearing in midst of noise.
Hearing for conversation relatively better than for watch.	Hearing for conversation relatively worse than for watch.

I. **Nerve Deafness** may be of (*a*) Sudden or (*b*) Gradual onset.

(*a*) Nerve Deafness of *sudden onset* may be due to:

I. Ménière's Disease.
II. Acute Labyrinthitis.
III. Syphilis.
IV. Concussion of the Labyrinth.
V. Hysterical Deafness.
VI. Lesions of the Central Nervous System.
VII. Small Hæmorrhages or Thromboses in the Labyrinth.

I. MÉNIÈRE'S DISEASE (see § 692).

II. ACUTE LABYRINTHITIS (see § 692) may result from acute or chronic otitis media and is usually suppurative. In mumps, influenza and meningococcal meningitis as a rule no suppuration occurs. Although the above are the commonest causes, the labyrinth may be destroyed and nerve deafness result from any acute infection.

III. SYPHILIS, ACQUIRED or CONGENITAL.—A syphilitic neuritis of the cochlear nerve, or syphilitic neuro-labyrinthitis may occur within a few weeks after infection. This form may be bilateral (Table XXXIX) and is more sudden in its onset than nerve deafness due to syphilitic lepto- or pachymeningitis, which may be unilateral or bilateral.

IV. CONCUSSION OF THE LABYRINTH may result from head injuries, explosions and loud noises, forcible syringing of the ear or fracture of the petrous temporal bone. In fractures of the skull the auditory nerve may be torn or bruised. The facial nerve is often paralysed at the same time.

V. HYSTERICAL DEAFNESS may be sudden in onset, following an emotional shock. (i.) Blinking of the eyes occurs if sudden loud noises are made near the patient, and (ii.) even if he appears stone-deaf it may be possible to waken him from sleep by simply calling his name. (iii.) Galvanic currents to the mastoid produce vertigo.

VI. Lesions of the CENTRAL NERVOUS SYSTEM may (rarely) cause deafness.

VII. Sudden complete nerve deafness sometimes occurs presumably from some VASCULAR CHANGE (thrombosis or hæmorrhage). In leukæmia hæmorrhage or *leukæmic deposits* may be responsible (§ 543).

(*b*) Nerve deafness of *gradual onset* may be due to:

I. Extra-Cerebellar tumour.
II. Syphilitic Pachymeningitis.
III. Congenital Syphilis.
IV. Meningitis.
V. Occupational Deafness.
VI. Drugs or tobacco.
VII. Toxic Causes.
VIII. Senile Changes.

I. An ACOUSTIC NEURINOMA (Acoustic Neurofibroma, § 829) is an important cause of chronic deafness in middle life. Other symptoms gradually appear: giddiness, tinnitus, headache, vomiting, etc., and the corneal reflex on the same side as the deafness may be found to be diminished, or absent, or there is slight facial weakness.

II. SYPHILITIC PACHYMENINGITIS in the lateral region of the posterior crania fossa causes gradual nerve deafness.

III. CONGENITAL SYPHILIS is an important cause of chronic nerve deafness (§ 552).

IV. MENINGITIS.—Local meningeal changes in the lateral recess may cause gradual nerve deafness and occur (1) in connection with suppurative middle ear disease (circumscribed serous meningitis), or (2) after cerebro-spinal meningitis (see § 503).

V. OCCUPATIONAL DEAFNESS may occur from repeated loud noises of machinery or guns.

VI. POISONING with Quinine, Salicylates, Paraphenylenediamine or Tobacco, may cause transient nerve deafness.

VII. TOXIC NERVE DEAFNESS may arise from circulating bacterial toxins, (i.) dental sepsis; (ii.) intestinal sepsis; (iii.) nasal, sinus, tonsillar or other infection.

VIII. In OLD AGE a certain amount of nerve deafness is very common.

II. **Obstructive Deafness** or deafness due to disease of the middle ear or auditory passages may be (*a*) acute or (*b*) chronic.

(*a*) ACUTE OBSTRUCTIVE DEAFNESS may be due to:

I. Impaction of wax.
II. Impaction of a foreign body in the meatus.
III. Acute Eustachian catarrh.
IV. Acute otitis media with inflammation of the tympanum.

(*b*) CHRONIC OBSTRUCTIVE DEAFNESS OF SOME STANDING:

(*a*) *Without a History of Previous Discharge.*

1. If the deafness dates from an acute naso-pharyngeal catarrh, tinnitus is not constant, and on inspection, the tympanic membrane is indrawn, opaque, and thickened, and inflation by Politzer's bag gives some relief; the disease is due to *Eustachian obstruction* and the presence of adhesions in the middle ear.

2. If the deafness has an insidious onset, tinnitus is a prominent symptom, inflation gives no relief, and on inspection the tympanic membrane is practically normal, with a pink sheen in its posterior part; the disease is probably *otosclerosis*. Hearing by bone conduction is longer than normal and the Rinne test negative.

(*b*) *With a History of Previous Purulent Discharge.*—There are probably perforations or cicatrices resulting from suppurative inflammation of the middle ear, and inspection of the drum may confirm this.

Combined Obstructive and **Nerve Deafness.**—With this combination it is sometimes difficult to make out the exact state of matters. Such cases may be grouped into those with and those without discharge.

(*a*) If there is a *history or presence of discharge*, a suppurative otitis media spreading to the labyrinth is probably in operation. In these circumstances we get signs of nerve deafness gradually supervening on those of obstructive deafness.

(*b*) If there is *no discharge, past or present*, the most usual conditions are: 1. Disease

of the cochlea supervening on an old chronic catarrh or sclerosis of the middle ear. 2. If the history of nerve deafness precedes the obstructive deafness, the middle-ear catarrh has supervened on the nerve deafness. 3. In advanced cases of otosclerosis there is usually marked loss of hearing for high notes as well as for low notes—in fact, the deafness is mixed middle ear and nerve deafness.

Pain in the Ear may be due to: 1. Otalgia, when there is no sign of local disease or defective hearing, and a reflex cause such as a carious tooth or diseased tonsil is present. 2. Disease of the external meatus, such as furuncle or eczema. 3. Disease of the middle ear, when there is deafness, some pyrexia, and examination reveals congestion and bulging of the membrane.

PAIN IN THE MASTOID REGION may be due to: 1. Mastoiditis; this is accompanied by redness, swelling and tenderness, deep throbbing and constitutional disturbance. It may follow acute or chronic suppuration. 2. A furuncle on the posterior wall of the meatus may give rise to a swelling behind the auricle. 3. A tender post-auricular gland may be found with local sepsis or pediculi capitis. 4. Mastoid neuralgia, which sometimes follows old mastoid disease.

Pain more or less GENERALISED OVER THE HEAD, accompanied by PYREXIA, may be associated with the following diseases of the ear:

(*a*) *Acute Diseases.*—1. Acute middle-ear suppuration, which is relieved by outlet of pus; 2, acute meningitis. 3. If the temperature is continuously high, it may be due to retention of pus, extradural abscess, or meningitis. 4. If the temperature oscillates, there may be sinus thrombosis and pyæmia (§§ 738, 515). 5. If the temperature after an initial rise is normal or subnormal, and there are headache, slow pulse, and delayed cerebration, suspect abscess of the temporo-sphenoidal lobe or cerebellum (§ 737). 6. Labyrinthitis (nystagmus, giddiness, vomiting and pyrexia).

Discharge from the Ear.—A STICKY OOZING may be due to eczema of the meatus, boils or condylomata. A HÆMORRHAGIC discharge may be due to vascular granulations or erosion of blood-vessels occurring with middle-ear disease. In acute otitis media due to the hæmolytic streptococcus there are often hæmorrhagic blebs on the drum or meatal wall and the discharge at first may be blood-stained. An offensive SANIOUS discharge, with fungating granulations, acute radiating neuralgia, and enlargement of the neighbouring glands, is characteristic of malignant disease of the ear.

A PURULENT discharge (*a*) *which is or has been copious*, and associated with deafness from the beginning of the symptoms, is due to acute or chronic suppuration of the middle ear. When associated with chronic suppuration, its chronicity may be due to the presence of polypus, granulations, or cholesteatoma, caries of the malleus, incus, or temporal bone, disease of the mastoid, antrum or naso-pharynx, or to constitutional causes, such as diabetes mellitus, tubercle, or syphilis.

(*b*) A PURULENT discharge *which is not, and never has been, copious*, and deafness, which, if present, did not supervene till some time after the onset of symptoms, may be due to external disease of the ear, acute or chronic. On the other hand, with chronic mastoiditis, there is not necessarily a copious discharge and deafness may be slight.

The *Prognosis* and *Treatment* of these various symptoms depend mainly on the cause in operation. To deal with them individually would be beyond the scope of this work. Nerve deafness is not very amenable to treatment. Any toxic or other cause in operation should, if possible, be removed. Obstructive deafness is, in a large proportion of cases, due to Eustachian catarrh, and treatment should relieve the deafness. A certain amount of good may be done in chronic catarrh by regular inflations, which the patient can be taught to do himself, and regular inhalation of various remedies such as tr. benzoin co. The nose and throat must be searched for abnormalities or infections, and they must be cleared up. Acute middle-ear disease requires prompt measures. Hot fomentations and incision of the tympanic membrane may be necessary. Warm drops of glycerine with 5 to 10 per cent. carbolic acid should be used. The sulphonamides by mouth and penicillin injections are most useful. Mastoiditis and other intracranial symptoms demand surgical interference. It is found that if the discharge of acute otitis media does not stop in about four weeks

with adequate treatment (if adenoids, nasal sepsis, etc., have been attended to), the mastoid is usually infected. X-rays are useful in demonstrating this. Operation is therefore necessary in these cases. It is most important to prevent the case becoming chronic. In chronic suppurative otitis media treatment should be by drops such as spirit or argyrol 10 per cent. Zinc ionisation and insufflation of boracic acid powder containing iodine 1 per cent. may be used. In many cases, however, a conservative or radical mastoid operation will be necessary. In cases of deafness an aid to hearing may be prescribed; lip reading is of great help to the very deaf. This short outline will be found useful in many cases to indicate the direction in which further investigations should follow.

§ **862. Tinnitus,** or subjective noises in the ear, is an extremely common symptom. The noise may be described as hissing, whistling, singing, buzzing "like a bell," roaring, and may be pulsating, continuous or intermittent. If the noise is referred by the patient definitely to the ear, the condition is true tinnitus and is due to disease of the (1) external auditory canal, (2) middle ear, (3) cochlear apparatus, or (4) cochlear nerve. If the noise is not definitely referred to the ear the condition is "head noises" and the cause probably psychical. Hallucinations of sound, the hearing of imaginary voices, is usually associated with mental illness.

The commonest *causes* of true tinnitus are: (1) Otosclerosis, (2) Nerve deafness, (3) Temporary Eustachian obstruction in coryza, (4) Anæmia (relieved by lying down), (5) Wax in the ear, (6) Drugs, *e.g.*, salicylates, quinine, (7) Prolonged loud noise affecting boiler-makers and those who work with electric drills.

Pulsating tinnitus (1) checked by compression of the carotid artery is due to arteriolar dilatation within the middle or external ear; (2) checked by compression of the vertebral arteries in the suboccipital triangle—a similar dilatation within the internal ear; (3) audible on auscultating the head—probably intracranial aneurysm.

Treatment.—The cause must be determined by a systematic examination of the patient generally and the auditory apparatus. "Head noises" are common in the silent watches of the night in neurasthenia and anxiety states, when the patient fears they will lead to insanity. True tinnitus is usually an intractable symptom, but it may be relieved by administering potassium iodide, hydrobromic acid or luminal.

Vertigo and its causes are dealt with in § 692.

§ **863.** The **Glossopharyngeal-Vagus-Accessorius Nerves,** see § 683.

The methods of examination are described in § 703.

Lesions of the **glossopharyngeal nerve** produce (1) sensory loss over the upper part of the pharynx, (2) loss of taste on the posterior third of the tongue. Lesions of the **vagus nerve** produce (1) unilateral paralysis of the soft palate and pharynx, with a "curtain-movement" of the posterior pharyngeal wall to the sound side on swallowing, (2) laryngeal palsies. Lesions of the **spinal portion of the accessory nerve** produce sterno-mastoid and trapezius paralysis. The winging of the scapula produced is greater than that seen in serratus magnus paralysis, and the scapula lies further from the vertebral spines and is more rotated. This nerve may be affected by toxic neuritis or by injuries in its course in the posterior triangle. The three nerves are commonly affected together, as they arise together in the medulla and leave the skull together through the jugular foramen.

Medullary Lesions of the Glossopharyngeal-Vagus-Accessorius are due to focal thrombosis, syringobulbia, diphtheria, or chronic bulbar paralysis (motor neurone disease). The symptoms produced are unilateral paralysis of the palate, pharyngeal muscles and larynx (Avellis' Syndrome). In Syringobulbia and vascular lesions, crossed anæsthesia of the dissociated type is commonly present (Fig. 162).

Lesions at the Base of the Skull are due to secondary malignant deposits, fractures or syphilitic meningitis. Unilateral paralysis of pharynx, palate, larynx, sterno-mastoid and trapezius and hypoglossal (Hughlings Jackson's Syndrome) may result.

Lesions in the Neck result from injuries, tuberculous, lymphadenomatous, or malignant glands and aneurysms. Unilateral paralysis of the vocal cord and palate results.

The palate and pharynx escape if the lesion is below the point of origin of the pharyngeal branches. Transient palsies of the soft palate and laryngeal muscles occur in Myasthenia Gravis (§ 808). Paralysis and wasting of the sterno-mastoids is often marked in Myotonia Atrophica. Wasting of the trapezii and winging of the scapulæ may occur in Myopathy.

§ 864. The **Hypoglossal Nerve** leaves the skull through the anterior condylar foramen and runs forwards above the hyoid bone and resting upon the hypoglossus muscle, to the under surface of the tongue. It is a purely motor nerve and supplies all the muscles of the tongue, intrinsic and extrinsic. The methods of examination are described in § 703.

The *symptoms* of a unilateral hypoglossal palsy are atrophic paralysis of one side of the tongue with loss of faradic excitability. The affected side of the organ shrinks and the tongue cannot be pushed into the contralateral cheek. There is little defect of articulation except at first, but the patient soon learns to overcome it.

Etiology.—Bilateral nuclear or supranuclear lesions occur in Chronic Bulbar Palsy (§§ 746, 747) with spasticity, or atrophy and fibrillation of the tongue. Spastic paralysis of the tongue also occurs in double hemiplegia (§ 746). Transient paralysis of the tongue occurs in Myasthenia Gravis. Unilateral hypoglossal palsies occur from motor neurone disease, medullary tumours, syringobulbia and gummatous meningitis. The nerve may be injured in the neck or under the tongue during operations upon the neck or throat.

CHAPTER XX

PSYCHOLOGICAL DISORDERS

THE forms of illness considered in this section are related essentially to the behaviour of the individual. They represent failure to make a satisfactory adaptation to life, environment and the other members of his social group. All students in general medicine have been impressed by the incidence of psychological factors in physical illnesses and how they influence their duration. Here we are concerned only with those conditions in which the psychological symptoms predominate. These can be studied in common with all other forms of illness, and failure to do so results, inevitably, in a lack of understanding of the factors involved. The major forms of mental illness have acquired for the student a sense of mystery, aggravated by the conflicting views and theories of the various schools of thought. The less severe forms include neurotic, emotional and personality disorders: individuals vary in their types of reaction to various forms of stress. Therefore many factors, constitutional, environmental, physical and psycho-biological may be involved; the significance of each can be evaluated only in relation to the other factors.

§ 875. Psychopathology. In order to understand the nature of psychological symptoms one must have some knowledge of *mental mechanisms*. The following account is a brief summary, involving the minimum of theory. Mental health depends upon the maintenance of a state of equilibrium between the conscious and the unconscious. Normally, these work in harmony and the individual adapts himself to his environment. The experience and stress of everyday life, with its struggles between desires and their gratification, within limits that allow a satisfactory adjustment of the personality, inevitably give rise to a state of mental *conflict*. Conflict is then a condition of normal mentation which is maintained by the formulation of a satisfactory solution. Difference of opinion has arisen regarding the nature of the conflicting forces. Freud originally considered they had a sexual basis, but it is obvious that this limitation is erroneous. Conflicts are accompanied by emotional tension and are the cause of much waste of mental energy. Most are solved successfully on the conscious level, are finally disposed of and give rise to no symptoms. Unfortunately, for various reasons, inherent or acquired, many patients are unable to deal with their conflicts in this manner, and devise various methods to maintain the state of equilibrium above-mentioned. These methods are common, invariably uniform in pattern, and are described as *mental mechanisms*.

Suppression and *repression* are the most common methods. Suppression is a conscious effort to forget what is unpleasant and is always accompanied by a focusing of the attention on something else. Repression is an attempt to expel from consciousness the factors that offend; they are transferred to the level of the unconscious. There they continue to exist and seek expression. Each repressed thought with its emotional component constitutes what we speak of as a *complex*. Invariably the personality elaborates methods on the conscious level to prevent expression of the repressed ideas; consequently exaggerated personality traits become prominent. Prudishness, for instance, results from the repression of normal sexual impulses; prejudices are developed by the individual to prevent the expression of repressed ideas. The establishment of such mechanisms need not disturb the mental equilibrium,