is rigidity of the muscles, with paralysis. The duration is very chronic, and paralysis agitans is essentially a disease of the old, but sometimes (rarely) it affects the young. Pathological changes have been found in the spinal cord and medulla, of the nature of congestion in the grey matter, increase of interstitial tissue, and atrophy of cells.

The **treatment** is not satisfactory. Prolonged rest of the limbs during the early stages should be tried. Chloride of barium (in one grain doses) is recommended; and arsenic sometimes produces improvement.*

* It may be of use to the reader, and an aid to the memory in the examination of a case of spinal cord disease, to note the order and classification of the foregoing diseases. The "paraplegic" group comprises *myelitis, compression paraplegia, secondary degeneration, the sclerosis* group, *functional paraplegias*, and the group *neurasthenia*; then follow three *special* diseases, viz.:—*Landry's acute ascending paralysis, locomotor ataxia*, and *meningitis*; the group associated with *muscular atrophy*; and lastly, three diseases associated with *trembling* of the muscles.

CHAPTER XI.

DISEASES OF THE NERVOUS SYSTEM.—Section II.

Contents. — Cerebral hæmorrhage; Apoplexy; Occlusion of the cerebral vessels—*i.e., thrombosis, embolism*, and *syphilitic disease*; Hemiplegia; Aphasia—Intra-cranial tumours—Cerebral abscess—Meningitis; acute, chronic, and tubercular; Pachymeningitis—Cerebral congestion and anæmia—Sunstroke—Chronic hydrocephalus—Epilepsy—Hysteria; Catalepsy; and Hypochondriasis—Chorea—Writer's cramp; Athetosis; Singultus or hiccough—Spasmodic tics—Meniere's disease—Migraine—Tetanus—Hydrophobia—**The differential diagnosis of brain diseases.**

Cerebral Hæmorrhage; Apoplexy; Occlusion of the Cerebral Vessels—Thrombosis, Embolism, and Syphilitic Disease. — These diseases may with clinical advantage be considered together, as they form a group in which the symptoms are very similar, and the points of difference may afterwards be stated. In *cerebral hæmorrhage* there is rupture of a blood-vessel in the brain—the most frequent site being the internal capsule, corpus striatum, lenticular nucleus, or optic thalamus. More rarely, it occurs in the cerebellum, pons, or medulla. The symptoms arise from the damage done to these, and the neighbouring parts. The clot may vary in size, from a pea to a large walnut, but in extensive ruptures the fluid blood often diffuses into the ventricles. The clot undergoes changes, and there may be secondary inflammation of the surrounding cerebral tissue. Ultimately a spongy connective tissue is developed, which encloses the clot—in those cases which survive. Small clots may become absorbed, and large clots—instead of becoming enclosed and thus promoting a favourable termination—often light up general inflammation. In chronic cases when the tissues have been much broken up, "atrophic degenerative" changes take place in the descending fibres of the nerve tissue, through the *crus* and *pons* to the spinal cord (*secondary degeneration*).

The commonest *cause* of cerebral hæmorrhage is disease of the cerebral blood-vessels — *atheroma* and *miliary aneurisms; fatty degeneration of vessel-walls*, as in pernicious anæmia, scurvy, purpura and post-infective diseases—and any sudden increase of the blood pressure may produce the rupture. The latter may arise in hypertrophy of the heart and gout; cirrhosis of the kidney; muscular effort; the use of stimulants; hot and cold baths; and violent emotions. The tendency to *atheroma* is often inherited; and it rarely occurs before the age of forty years.

In *thrombosis* and *embolism*, there is occlusion of a blood-vessel by the formation of a clot, in the former disease—while in the latter, a small clot, concretion, or particle of fibrin (embolus) is carried up in the circulation and becomes impacted. Emboli may be derived

from thrombi developed elsewhere in the circulation, from atheromatous disease, generally; and most commonly they are derived from diseased valves of the heart. The left carotid, from its position is more liable than the right to receive these emboli. The embolus generally passes up to the left Sylvian artery, in which it becomes

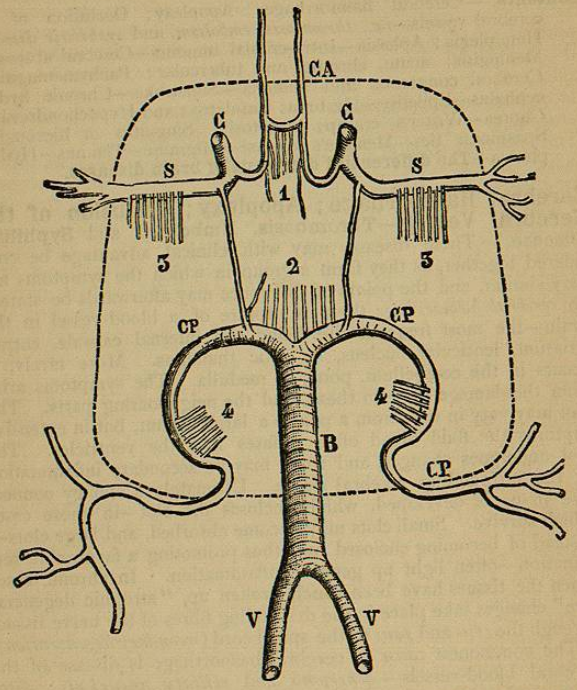

Fig. 38.—Arteries of the base of the brain, or circle of Willis.—C, C, Internal carotids; CA, anterior cerebral; S. S, Sylvian arteries; V, V, vertebrals; B, basilar; CP, posterior cerebrals; 1, 2, 3, 3, 4, 4, groups of nutrient arteries. The dotted line shows the limit of the ganglionic area. (From *Charcot*.)

impacted and hence the greater frequency of right hemiplegia. The vertebral arteries are rarely entered by emboli. The effect of the clots is to cut off the blood supply to the brain in the regions corresponding to the distribution of the blood-vessels. It is important to note that the brain is supplied by *two sets*, viz.:—(1) the blood-vessels to the *cortex* which have a free anastomosis; and (2) the

blood-vessels to the *centres* which are terminal. The plugging of the latter produces a complete anæmia, while in the former the circulation may be soon restored by the collateral branches.

The *antero-median* group (1) supplies the anterior part of the head of the caudate nucleus. The *postero-median* group (2) supplies the internal surfaces of the optic thalami and the walls of the third ventricle. The *antero-lateral* groups (3, 3) supply the corpora striata, the anterior parts of the optic thalami, and the internal capsules. (One of these nutrient arteries is larger than the others, and is the "lenticulo-striate artery" or "artery of cerebral hæmorrhage." It terminates in the anterior part of the caudate nucleus after crossing the upper part of the internal capsule). The *postero-lateral* (4, 4) supply large parts of the optic thalami.

The *anterior cerebral* curves round the corpus callosum, and supplies the gyrus rectus and the supraorbital lobe, the first and second frontal convolutions, the upper part of the ascending frontal, and the inner surface of the hemisphere as far as the quadrate lobe. The *posterior cerebral* goes to the region of the occipital lobe and the inferior aspect of the temporal lobe. The *middle cerebral*, or *Sylvian* artery, divides into four branches, which go to the posterior parts of the frontal lobes, the ascending frontal, and to all the parietal lobes,—*i.e.*, chiefly to the motor areas, the angular gyrus, and to the first temporo-sphenoidal lobule. Thus the *anterior cerebral* supplies the prefrontal area, and a small part of the motor area—viz., that for the leg centre in the paracentral lobule and upper end of the ascending frontal (and perhaps that for the trunk). The *posterior cerebral* supplies the centre for vision, and that connected with the course of the posterior part of the optic expansion, and also the sensory part of the internal capsule. The *middle cerebral* supplies the motor areas of the cortex (except part of the leg centre), and the basal ganglia (in part), the auditory centre, and that for speech.

Occlusion of a *terminal* blood-vessel results in *softening* of the brain tissue supplied by the vessel. "White or yellow softening" results from a complete block of the vessel by coagulation of the blood beyond the embolus, and the consequent prevention of a backward flow from the capillaries. The tissues affected become yellow from fatty deposition in the nerve cells. "Red softening" occurs when there is no such coagulation. A hyperæmia is then produced, and the tissues are coloured by the "diapedesis of the red blood corpuscles." Minute extravasations often occur from rupture of the capillaries. In a few weeks the "red softening" becomes "yellow softening," from the transformation of the hæmoglobin, and fatty degeneration of the nerve elements. When death occurs rapidly the brain tissue may appear quite normal and there is no softening.

The *causes* of thrombosis are chronic endarteritis; pressure by tumours, &c.; and weakening of the blood current, such as occurs in the aged, and in those suffering from chronic wasting disease. The sources of emboli are endocarditis, infections, and blood diseases; clots within the auricle, from wasting disease; fatty heart; and aortic

aneurisms. *Syphilitic* disease of the cerebral arteries produces its symptoms from occlusion of the blood-vessels by the gradual thickening of their coats.

The **symptoms** common to this group of cerebral diseases are the symptoms which are associated with **apoplexy, hemiplegia,** and **aphasia.** Any one or all of these conditions may occur as a result of cerebral hæmorrhage, thrombosis, embolism, or syphilitic disease of the cerebral arteries; but it is better to describe them separately, as they often require (clinically) to be regarded as substantive affections, the actual causes of which can only be inferred.

Apoplexy is not always sudden; but the "warnings" are often very indefinite. Epistaxis, and hæmorrhages into the conjunctivæ, sometimes occur several months before a "shock." The temper becomes irritable, and the speech and memory are frequently defective for some time previously, and many complain of slight paresis, numbness, or tingling in an arm or leg. Giddiness, diplopia, and headaches come somewhat nearer to the actual shock, and often are the immediate forerunners; while the other prodromata may be supposed to be due to diseased blood-vessels or to very small ruptures. In a typical apoplectic seizure there is generally violent pain in the head, and in a few minutes the patient becomes comatose. Sometimes the headache is complained of for a longer period, but the insensibility is rarely delayed beyond a few hours, if it be about to supervene at all. Sometimes, again, there is no warning, and the patient falls with a loud cry; or there may only be nausea and vomiting, and the unconsciousness comes on very slowly and after a few hours have elapsed. The face may be flushed or pale. The breathing is at first stertorous, with puffing out of the cheeks, and soon frothy saliva appears at the lips. The conjunctivæ are insensible, and the pupils may be contracted, dilated, or unequal. When the lesion is situated in the pons, the pupils are often extremely contracted (pin-points), and the respirations are very slow. The eyes and head are often turned to the side on which the lesion is situated in the brain (*conjugate deviation*). This symptom is probably due to the lesion exciting "rotatory movements," such as occur in animals when one side of the brain is injured. The pulse is often full and tense, and sometimes very slow. The forehead and body are generally bathed in perspiration. Sometimes apoplexy begins with convulsions, and the amount of insensibility varies much. If incomplete, pinching the limbs may reveal that only one is drawn away; or, on raising the arms, one may fall helplessly back, while the other remains rigid. Sometimes, however, there is no hemiplegia. In apoplexy, the temperature at first falls, and it remains low in rapidly fatal cases; but in a few hours it rises two or three degrees, and keeps up when inflammatory changes supervene. It may reach 107° Fahr. shortly before death. If recovery be about to take place the temperature falls again within three days. A pulse below sixty is a bad sign, and so also is a very rapid or irregular pulse. The breathing may be of the Cheyne-Stokes character, and just before death it becomes extremely slow,

with long intervals. The *duration* of a case varies from a few minutes to two, eight, and even fourteen days or longer. Consciousness may return partially, and may be followed by drowsiness or delirium; but these cases may still terminate fatally. Sometimes consciousness is completely regained; and if this should not occur within forty-eight hours, and be accompanied by other favourable symptoms—death may be expected within the periods above-mentioned. Death may result from pneumonia or œdema of the lungs.

Hemiplegia is the result of unilateral disease of the brain. The leg, side of body, arm, and face, are more or less paralysed upon the opposite side from the lesion in the brain. The face often escapes, and there is never the complete paralysis which occurs in peripheral lesions of the seventh nerve. The eye can be closed voluntarily—and this cannot be done in a case of Bell's paralysis. Sometimes, however, he cannot wink with the affected eye (paralysis of the *orbicularis palpebrarum*). The mouth may be only drawn slightly to the sound side. The tongue sometimes cannot be protruded beyond the teeth; but when this is possible, the tip generally points to the paralysed side. The speech is defective, even when the intelligence is not much impaired. Almost always both leg and arm are paralysed; but sometimes the arm alone may be affected. The chest muscles of the damaged side do not act so well. Some amount of anæsthesia and hyperalgesia is believed to be present in the early stages of hemiplegia, but, if so, it quickly disappears. As a rule, the mental condition during the onset is not favourable to the testing of the sensory nerves. In a recent hemiplegia the limbs are usually redder, and they may be raised in temperature (1° Fahr.). The damaged limbs are liable to sweat and to become œdematous. Sometimes bed-sores form. The electrical re-actions vary, and they are often normal. "Early rigidity" may be present, with flexion of the fingers, hand, and elbow. This condition is believed to be due to laceration and irritation of the brain tissues, or to *inhibition*. In the later stages, the muscles contract, and there is much deformity. The hand and arm is flexed, and the nails may even pierce the skin of the palm. It often requires much force, and it gives much pain, to straighten them. This "late rigidity" is due to the secondary degenerations in the lateral tracts of the spinal cord irritating the sound nerve fibres which are connected with the sclerosed columns. The reflexes are increased. Spasms and tremors are common in the paralysed limbs; and "post-hemiplegic chorea" is a less frequent after-effect. The facial nerve of the opposite (sound) side is sometimes paralysed (crossed hemiplegia), and this occurs when the pons is the seat of the lesion. If the upper part of one lateral half of the pons be the seat of a lesion destroying the facial nucleus, there is also a crossed paralysis, and probably, then, the adjacent nucleus of the sixth nerve also suffers, and there will be a paralysis of the external rectus as well. Extreme disease of the pons may cause a paralysis of both facial nerves, with rigidity in both legs. The pupils are often contracted (pin-points). If there be paralysis of the third nerve along with the facial, the lesion is in the *crus*.

Charcot states that when a lesion is confined to the grey nuclei of the corpus striatum the paralysis is transitory and incomplete; when it involves the internal capsule, it is complete and permanent. The anterior two-thirds of the internal capsule contain the motor fibres, and those to the leg lie nearer the median line. Probably, this is why the leg recovers first, as these fibres are more likely to escape complete destruction. The posterior third of the internal capsule contains the sensory fibres, and lesions affecting this part give rise to anæsthesia, hyperalgesia, and loss of tactile sensibility. Sight, hearing, and sense of smell, may also be affected, and Jackson says there is often lateral hemianopsia.

Hemiplegia usually follows apoplexy, although not always. In some cases there is no antecedent apoplexy, but merely a confusion of mind followed by paralysis. Transitory attacks sometimes usher in the severe form, and lesions of the cortex sometimes give rise to temporary paralysis, which disappears with the establishment of the collateral circulation in the part affected. This cannot occur when terminal vessels to the basal ganglia are plugged. An embolus may plug the central artery to the retina and cause blindness; and coarse lesions may light up *double optic neuritis*. The mental functions in chronic cases of hemplegia are much impaired, as a rule.*

Aphasia.—Under this head, are grouped several affections concerned with the loss of speech and memory. *Aphasia* means the inability to use spoken language, and it is "the outgoing language and motor processes that are interfered with." If it be due to a *loss of memory* for words, it is said to be *amnesic aphasia*. *Alalia* means inability to articulate—as in bulbar paralysis, &c. *Agraphia*—another form, in which there is inability to recognise, or form the written characters—may co-exist with the aphasia, and sometimes it is present alone. The power of expression by signs may also be lost, sometimes. The memory for written language is often present, and aphasic patients read the papers, sometimes over and over again. A good test of his understanding is to read a book aloud with him, and ask him to turn over at the proper place. The *third left frontal*, or *Broca's convolution*, including a part of the *second left frontal convolution*—is the seat of the memory and language centres. This area is supplied by the middle cerebral artery, which is so very often the seat of obstruction by an embolus, and hence the frequent association of right hemiplegia with aphasia. Aphasia may be the result of embolism, thrombosis, hæmorrhage, inflammation and abscess, and tumour; or it may be due to mental conditions. *Embolism* is the commonest cause. To explain the absence of aphasia when the internal capsule and descending pathways are much injured, Dr. Broadbent thinks there must be two routes for language, and

*A *meningeal hæmorrhage* is usually the result of an injury, and the symptoms are discussed in surgical works. It does sometimes occur (chiefly in asylums) idiopathically. The symptoms of hemiplegia, then, are not so marked, and are more transient. In *pial* apoplexy the hemiplegia is incomplete. *Cerebellar* apoplexy is rare and invariably fatal.

suggests that the commissural fibres probably connect Broca's convolution with the corresponding parts on the other side, and hence the impulses travel to the pons and medulla by this route. In left-handed persons, aphasia has been known to occur from a lesion of the *right* third frontal convolution.

The intelligence in aphasic patients may be unaffected, although, most frequently, there is mental weakness, especially when the aphasia is associated with hemiplegia. The sense of smell is sometimes lost. The patient is often unconscious of his mistakes; but sometimes the half-amused and half-annoyed expression which follows the use of a wrong word, shows that the intelligence is not impaired. Aphasia sometimes disappears, or is cured, before the associated hemiplegia; or, again, it remains after the patient has recovered the use of his limbs. When due to syphilis a cure may be expected; and in young patients, *training* and education may produce good results. The longer aphasia exists, the less is the prospect of recovery.

The **differential diagnosis** of each member of this group may now be considered—leaving the general diagnosis of apoplexy, &c., from other diseases, until the end of the section. *Thrombosis* is somewhat rare; and it occurs in the aged and very feeble, and the onset is *gradual*. To decide between cerebral hæmorrhage as a cause of *apoplexy*, and embolism and syphilis—the age is an important point. Apoplexy, occurring in a patient over fifty years, is probably due to hæmorrhage; while the younger the patient the greater is the probability of its being due to embolism or syphilis. To differentiate the latter two, a careful examination must be made for evidences of syphilis; and the heart must be examined for endocarditis, and the liver, spleen, and kidneys for enlargements (infarctions). Heart disease is the commonest source of emboli. It should be noted that embolism and cerebral hæmorrhage may co-exist.

In a case of *hemiplegia* without loss of consciousness, the probability is that it is due to obstruction of blood-vessels—embolism, syphilis, or atheromatous disease. The attack may be gradual or sudden, generally the latter. The association of hemiplegia with aphasia points to obstruction of the Sylvian artery, and it may be due to any one of the three causes just stated. The same careful examination as before, is here necessary. In syphilis the symptoms are apt to be irregular, but atheroma of the blood-vessels may also be general. In syphilis a peculiar somnolent condition, and a "sulky disposition" are described. He resents examination. The cranial nerves are often affected, and the paralysis often leaves one limb and attacks another. Such cases may have paralysis at one time and not at another. Sometimes, there is only a drowsy condition, or headache, loss of memory, vomiting, &c., with no hemiplegia. Hemianæsthesia—when the posterior third of the internal capsule is involved—is generally due to hæmorrhage; but it may be due to extensive atheromatous disease. In the history of a case of hemiplegia it is important to know if it began with apoplexy. The more marked the coma, and the longer the duration and severity of the

initial state, the more likely is it to have been due to rupture of a blood-vessel—especially if occurring after fifty years of age.

The **treatment** of cerebral apoplexy—whether due to hæmorrhage or to obstruction of the blood-vessels—is quite the same. Should there be any warnings of a seizure, venesection may be practised, or leeches may be applied to the mastoid processes. An active purge may be administered. When the shock has occurred the head should be raised, and an ice-cap worn; while mustard should be applied to the stomach or lower extremities. The room should be darkened, and the utmost quiet maintained. Two drops of croton oil may be given, and it is the most convenient purgative for such cases, as it may be dropped upon a small piece of sugar, and placed far back upon the tongue and swallowed unconsciously. Tincture of aconite may be given in one minim doses, every ten minutes, to reduce arterial tension. Bromide of ammonium or potassium, should be prescribed in ten or twenty grain doses, when the patient is very restless. Should the patient recover consciousness, the bromides should be continued for some time, and then ℞ 55 may be ordered. In the later stages, galvanism and faradic electricity with massage, &c., should be commenced. The diet should be very light and digestible. Tonics may be given. Stimulants must never be used in the early stages.

(Cerebral palsies in children, see *Handbook of Children's Diseases*, Elder and Fowler.)

Intra-Cranial Tumours.—These include aneurisms, sarcomata and gliomata, &c., hydatids, and cancerous, tubercular, and syphilitic tumours.

The symptoms. Headache is a prominent symptom, and the seat of the pain may sometimes indicate the situation of the tumour. The pain is often paroxysmal, and it is worse upon coughing, and upon taking a deep inspiration. The scalp may be tender at certain points. The headache may at times be so agonising as to cause delirium. Vertigo is very commonly present, and especially when the cerebellum is the seat of the tumour. Vomiting and constipation are present in most cases, and if the vomiting be persistent there is loss of flesh and emaciation in extreme cases. Epileptiform convulsions are common to all tumours of the brain; but Reynolds believes convulsions to be more frequently present in tumours affecting the posterior lobes, or the cerebellum. The pulse is often slow. The sight is often affected, and as the vision may be good at the centre, yet defective at the periphery, the visual field should be tested in all directions. It varies in amount from a mere mistiness to actual blindness; but the blindness often comes on suddenly, even although the ophthalmoscope has for some time revealed the changes mentioned below. These changes consist of the conditions known as "choked disc," optic neuritis, and atrophy of the disc. The "choked disc" probably arises when "the fluid, which is always present in small quantity in the arachnoid cavity, is driven into the optic nerve . . . . whenever the intra-cranial pressure is from any cause increased." Optic neuritis may be

secondary to a choked disc, or it may arise from extension of inflammation downwards. Atrophy of the disc follows these pathological changes, but sometimes it occurs as a chronic change without antecedent neuritis or choked disc. (See the diagrams of these conditions in Gower's or Liebreich's atlas, and compare with the normal.) The mental state often becomes changed, and there is loss of memory, irritability, depression, and stupor. Many cases become maniacal, and require to be removed to an asylum.

The foregoing symptoms are common to all intra-cranial tumours; but they are often accompanied by other signs which enable them to be more exactly localised.

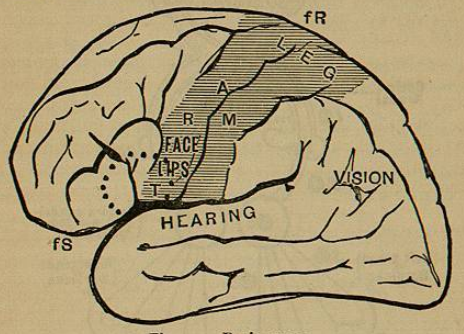

Fig. 39.—Brain areas.

VISION = *Word vision centre.*
HEARING = *Word hearing centre.*

(1) Tumours affecting the cortex may produce spasms and convulsive movements (Jacksonian epilepsy) in the hand, arm, or leg of the opposite side, and if situated in the posterior lobes there may be disorders of sensation. One would expect homonymous hemianopsia in disease of the occipital lobe, *word blindness* in disease of the left angular gyrus, and *word deafness* in disease of the left upper temporal lobe; and loss of taste and smell when the under surface of the temporal lobe is affected.

(2) Tumours affecting the motor-tract, corpus striatum, optic thalamus, &c., will produce spasms and contractions in the limbs of the opposite side when the lesion is *irritative*, but paralysis when the lesion destroys the part without irritation.

(3) Aphasia may result from a tumour affecting Broca's convolution and the immediate neighbourhood.

(4) Tumours of the medulla affect the speech, deglutition, and respiration, &c.

(5) Tumours of the cerebellum cause loss of co-ordination, and there is a reeling, staggering gait. The vertigo is generally

marked, and convulsions, nystagmus, and headache, optic neuritis, and blindness are common symptoms.

(6) The *olfactory nerve*. When irritated, there is often a complaint of strange smells; when destroyed, smell is lost.

(7) The *optic nerves*. A reference to the accompanying diagram* (which is not, however, complete in *anatomical* details)

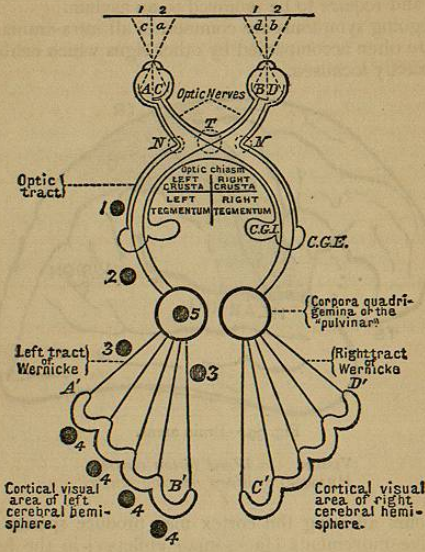

Fig. 40.—A diagram of the optic tract, to explain hemianopsia. The rays, *a b* and *c d*, from the objects in front of the eyes (1, 2), fall upon the temporal and nasal halves of the retina, as indicated, at the capital letters A B and C D. A and D to A′ and D′ indicate the course of the fibres from the temporal halves of the retina; while B and C to B′ and C′ show the course of the nasal fibres—the important difference being the decussation of the latter at the optic chiasma. From this point backwards the fibres (both temporal and nasal) pursue the same course, passing through the optic tract, external geniculate body, corpora quadrigemina or "pulvinar" of the optic thalamus, and the internal capsule to the occipital lobes. A and B to A′ and B′ indicate the fibres associated with the left cerebral hemisphere; while C and D to C′ and D′ indicate the fibres of the right. A lesion, involving the tract at 1, 2, 3, 4, or 5, produces blindness of the temporal half of the left eye (A), and the nasal half of the right (B)—*i.e.*, *right lateral hemianopsia* (*homonymous*). A lesion at T produces blindness of the nasal halves of both eyes—*i.e.*, *bi-temporal hemianopsia*; while two lesions at N N (very rare) will produce blindness of the temporal halves—*i.e.*, *double nasal hemianopsia*. The lesions at T and N N are *heteronymous*. [From *Ranney* (modified).]

*Fibres are now known to pass to the optic thalamus, external geniculate body, corp. quad., and cortex direct; but they do not affect the clinical significance of the diagram as given above.

will explain the various defects of vision possible by pressure upon the chiasma, or part of the optic tract.

(8) The *third nerve*. A tumour of the *crus*, when *irritative*, gives rise to nystagmus in the eyeball of the same side, and spasm or rigidity in the muscles of the opposite side of the body. When the lesion is *destructive* there is ptosis, convergent strabismus, and dilated pupil, in the eye of the same side, and paralysis or paresis of the muscles of the opposite side of the body. The eye symptoms occur very frequently in syphilitic lesions. Lesions of the corpora quadrigemina also affect the movements of the eyes; and there may be double optic neuritis, vertigo, blindness, and occasionally deafness, and sometimes paralysis of the muscles of the opposite side of the body.

(9) The *fourth*, *fifth*, *and sixth nerves*, when implicated, point to a lesion of the *pons*, and there may be disorders—motor and sensory—of the opposite side of the body. An *irritative* lesion involving the *fifth* nerve will give rise to *tic-douloureux*, while a *destructive* lesion will produce anæsthesia of the face.

In tumours at the base of the brain, *retinitis* occurs earlier; and pressure upon the cavernous sinus will produce a fulness about the orbit, swelling of the eyelids, and often epistaxis.

The *course* of intra-cranial tumours is very indefinite. They may take months to develop, and the early symptoms are often obscure. They often terminate in convulsions and coma, cerebral hæmorrhage, or meningitis. An aneurismal tumour may be relieved, and syphilitic tumours are certainly curable under proper treatment.

The **diagnosis** of the different forms of tumours can only be inferred; and often it is quite impossible to know their nature. The history and constitution may suggest a correct diagnosis. In syphilitic tumours, the headache is worse at night, and the pain is increased by tapping the head. There is often a state of somnolence present, and the paretic or paralytic symptoms which may be present are liable to disappear and return, affecting often different limbs. The **treatment** should consist of the administration of large doses of iodide of potassium (and bromides) in *all* cases.

Cerebral Abscess.—This disease is seldom, if ever, primary; but it follows injuries, diseases of the ear (especially chronic suppuration of the tympanum, &c.), chronic disease of the nose, pyæmia, and infections from other organs—*e.g.*, empyema, tuberculous inflammation of the lungs, and infectious fevers. When secondary to disease of the ear and nose, or the result of an injury, the abscess is generally single and large. They vary in size from a pea to a hen's egg.

The **symptoms** vary much. In pyæmia the symptoms of blood poisoning generally mask the symptoms of suppuration in the brain. In other forms, severe pain in the head, noises in the ears, nausea, vomiting, and epileptiform seizures are present. The temperature rises to 103° Fahr. The pulse is sometimes exceedingly slow. The

pupils sometimes act sluggishly. Rigors are not always present; and the fever is intermittent in type. When the abscess is developed, the symptoms of compression, and the means of localisation, are the same as in intra-cranial tumours. Death may be ushered in by delirium, epileptiform fits, stupor and coma. There may be a prodromal stage lasting a few weeks or months. When acute inflammation begins the case terminates in about a week. Optic neuritis is often present. Phlebitis of the superior petrosal and lateral sinuses, and meningitis, are common complications. If the primary disease is in the mastoid the abscess is usually cerebellar. A *hæmorrhagic encephalitis* is described, in which there is congestion, hæmorrhages, and inflammation without suppuration. The causes are influenza and other infectious fevers, malignant endocarditis, sunstroke and alcoholism. The **diagnosis** generally rests upon the association of one or other of the causes with the above symptoms; and the **treatment** is the same as in meningitis. Surgical procedures may save life in some cases.

Meningitis.—Tubercular: Acute and Chronic Meningitis.—Pachymeningitis.—Tubercular meningitis is an inflammation of the membranes of the brain produced by the development of tubercular granulations. The tubercles vary in size, from very minute points to a pin's head, and they are grey-white in colour. They are more numerous around the arteries at the base, but they are also distributed along the course of the arteries of the convexity of the brain. A sero-purulent effusion accompanies the tubercles. The membranes themselves are thickened and opaque, and there is increased effusion into the ventricles, and often œdema of the cortex.

The causes of tubercular meningitis are favoured by bad hygienic conditions. The disease occurs in those who have inherited the tubercular *diathesis;* and it is common between the ages of two and six years. The *type* of child apt to be affected is described as "the pale, thin-skinned, blue-eyed child of pale, flabby, and delicate parents." The bacillus tuberculosis is derived from a diseased focus in other organs.

The symptoms are sometimes divided into stages. In the *prodromal* stage, which may last for three months, there is irritability, loss of appetite and sleep, and emaciation. The child grinds his teeth, or cries out in the night. There is often a frown upon the brows. Headache and vertigo is complained of, and there is often vomiting without apparent cause. The bowels are irregular, and the abdomen is apt to be swollen. *Double vision* may be an early sign.

The stage of *excitation* is characterised by fever (102° or 103° F.), which is remittent in type. The pulse is irregular and subject to great variations in frequency. It often becomes very slow. The headache, vomiting, and constipation is now marked. The headache is subject to exacerbations, and it is increased by exposure to strong light. There is increased fretfulness, and all movements cause pain. The head and neck are generally held rigid, and other

muscles are in a state of spasm. Convulsions are common. The *hydrocephalic cry*, or shriek, is often heard at this stage. The period of excitation lasts one or two weeks. *Tache cérébrale* is the name given by Trousseau to a symptom of tubercular meningitis. It consists of a bright red line produced by gently scratching the skin. It appears quicker, remains longer, and is more marked than a similar line produced in a healthy child. Before the actual development of the stage of depression, there is a period in which excitation and depression exist alternately. This stage may last from one to three weeks. There are paroxysms of pain and convulsions, alternating with periods of somnolence and torpor. Strabismus, double vision, inequality of the pupils, and retinal changes —are prominent symptoms at this period. The ophthalmoscopic examination reveals the *choked disc* and *atrophy*. Tubercles are often detected in the choroid.

Sometimes there is now an improvement; but it is almost invariably followed by the stage of *depression*. In this, there is delirium, convulsions, and gradually increasing unconsciousness. The pupils dilate, and there is often nystagmus. The coma deepens, and the respirations become more and more shallow. The breathing may be of the Cheyne-Stokes type. The pulse—which has been irregular and slow—becomes rapid and feeble before death. The stage of depression may last one or two weeks. A tubercular meningitis, arising as a secondary disease (as in pulmonary tuberculosis) has no prodromal symptoms. There is intense headache and delirium, but no convulsions (in adults).

In **simple acute meningitis** there is intense hyperæmia, followed by a purulent and fibrinous exudation, over the base and convexity of the brain. The ventricles may be distended with fluid. It is due to an acute infective process, and various micro-organisms are found. Injuries and alcoholism predispose, but the chief sources of infection are extension of disease from the bones, as in ear disease; infectious diseases; pneumonia; erysipelas, and pyæmia. It sometimes occurs during the course of Bright's disease, acute rheumatism, or puerperal fever. The **symptoms** begin with a chill, the temperature rising to 103° or 104° Fahr. The face is flushed, and the eyes are injected. Headache and vertigo, nausea and vomiting, are prominent symptoms. There is delirium, with illusions and hallucinations. In the earlier stages there is hyperæsthesia of the skin, and spasms of the muscles of the neck and the extremities. Irritation of the cranial nerves accounts for the eye disturbances. Later, the symptoms of depression supervene, and there is somnolence, coma, and paralysis. The ophthalmoscopic changes are the same as in the tubercular form; but no tubercles are found in the choroid. The duration is about one to eight weeks—the condition during that period being very variable. It usually terminates fatally, but sometimes there is recovery. The **diagnosis** in relation to other diseases is considered later. The differentiation from tubercular meningitis requires the consideration of the history of the onset, the presence or absence of tubercles in the choroid or other organs, the age, the causes, and the *diathesis*.