CHAPTER XIX

THE NERVOUS SYSTEM

The student will find the arrangement of this somewhat lengthy chapter conforming to the method pursued throughout the book. A brief account of the anatomy and physiology of the nervous system is followed by:

At the outset certain *Physiological Laws* applied to disease processes in the nervous system, and first formulated by Hughlings Jackson, may be stated:

(1) The nervous system may be visualised as consisting of a number of physiological levels, the functions of the lowest or spinal level (most automatic) being comparatively well-organised at birth, the highest or cortical (most voluntary) continually organising throughout life. The higher levels inhibit or control the lower levels.

(2) In disease the functions first acquired in development are the last to be destroyed. Thus when speech function is destroyed by a cortical lesion, gesture which is acquired chronologically before speech invariably remains. In acquired dementia the memory for recent events has gone, while the patient still remembers events of childhood.

(3) A destructive nervous lesion causes negative and positive symptoms. For example a capsular hæmorrhage will cause loss or impairment of voluntary movement dependent on destruction of the pyramidal tract—the *negative* symptom. We also observe new phenomena, not present before the onset of the lesion, muscular hypertonus and an extensor plantar response. These are *positive* symptoms due to release of intact mid-brain mechanisms which have escaped from pyramidal control.

(4) Chronic lesions of the nervous system at first irritate and later paralyse function. Thus a slowly growing meningeal tumour compressing the motor cortex will at first cause a focal Jacksonian convulsion (Irritative sign), later a monoplegia (Paralytic sign).

(5) Nerve-cells once destroyed never regenerate. Compensation occurs in cases of partial destruction, but this is never absolute.

(6) The more rapid the destruction the greater the dissolution. Acute lesions (*e.g.*, a blow on the head) produce at first widespread loss of function, complete loss of consciousness, flaccid paralysis and incontinence. These "shock phenomena" are usually transient.

APPLIED PHYSIOLOGICAL ANATOMY OF THE NERVOUS SYSTEM.

§ 667. The central nervous system consists of vast numbers of *Neurones.* A neurone is a nerve cell with its dendrites and axon. The *nerve-cells* are found in the grey matter of the cortex, basal ganglia and nuclei, the central grey matter of the spinal cord and posterior root ganglia. The *axons* are collected into bundles or tracts and run mostly in the white matter and peripheral nerves. The *nervous impulse* travels at different rates in different nerves, and may travel in both directions in a single nerve fibre. A *synapse* between a nerve-fibre and another nerve-cell will allow an impulse to pass in one direction only.

The activity of the cortical nerve cells can be studied with the Electro-encephalograph, by means of which cortical action currents are led off by electrodes placed on the intact scalp, amplified by wireless valves and recorded by a cathode-ray oscillograph. Waves of a frequency of 10 per second and 0·5 to 1·0 millivolt amplitude may be observed when the patient is at rest with the eyes closed. These waves are called alpha waves, and the ten-cycle rhythm is known as the Berger rhythm, after its discoverer (Fig. 174). Beta waves of a frequency of 25 to 50 per second are also obtained simultaneously from various parts of the cortex in normal persons. These waves can be inhibited if the individual concentrates on an arithmetical problem, or when he opens his eyes. Abnormal brain potentials accompany pathological cortical processes. In epileptics, sub-liminal discharges from cortical foci can sometimes be recorded in this way. In intracranial tumour slow waves can be picked up by the electrodes and the abnormal focus localised to a restricted area of cortex.

Neurones are extremely sensitive to oxygen-want (anoxæmia) and many nervous lesions produce their effects by alterations in the blood supply. Cerebral and spinal tumours produce their effects not so much by distortion or disruption of nerve-tracts as by *local anoxæmia.* In these cases, rapid recovery of function may follow simple decompression, even when it is impossible to remove the tumour. Certain inorganic poisons or organic toxins exert their effects selectively by picking out particular groups of neurones or muscles, *e.g.*, lead commonly affects the neurones proceeding from the sixth cervical segment, producing wrist-drop; diphtheria picks out the ciliary and the bulbar muscles. This *selective action* is also observed in infection with viruses, the virus of acute poliomyelitis affecting the anterior horn cells, that of epidemic encephalitis affecting the oculo-motor nuclei. An acute toxic lesion (*e.g.*, polyneuritis) may produce widespread recoverable paralysis without demonstrable structural changes in the neurones. Recovery, after actual destruction, is only possible in peripheral nerve lesions.

§ 668. The Cerebral Cortex.—The cortical MOTOR AREAS lie in the pre-central gyrus and the posterior parts of the frontal convolutions which are immediately anterior. *Here are represented not muscles but movements.* Topographically, the movements of the foot are represented at the upper end of the motor cortex; those of the leg, trunk, upper limb, hand, neck, face, lips and tongue, in that order from above downwards (Fig. 157). It will be noticed that the complicated movements of the face and hand have a relatively large cortical representation, a good example of adaptation of structure to function. Irritative lesions of these areas cause focal *Jacksonian fits.* Jacksonian fits commonly commence in one of three foci—(*a*) the thumb and index finger, (*b*) the angle of the mouth, or (*c*) the hallux, and are followed by the "paralytic sign," a transient monoplegia. Such a fit begins with clonic convulsions of one of these foci and (*a*) may remain local or, (*b*) more commonly, spreads in an orderly march in accordance with the cortical representation of the parts affected, so that face, arm and leg on one side of the body are involved and eventually the whole of the body musculature. So long as the convulsion is localised consciousness may be preserved, but when it is generalised consciousness is lost. At the posterior end of the second frontal gyrus is an area for the *conjugate movement of the head and eyes* to the contralateral side (oculogyric area). An area at the posterior end of the first

and second frontal gyri is connected with the *grasp reflex*. Stroking the palm of the contralateral hand or stretching the flexors of the fingers produces a tonic closure of the fist. Lesions extending deeply into the substance of the motor area produce a *monoplegia* or *hemiplegia*.

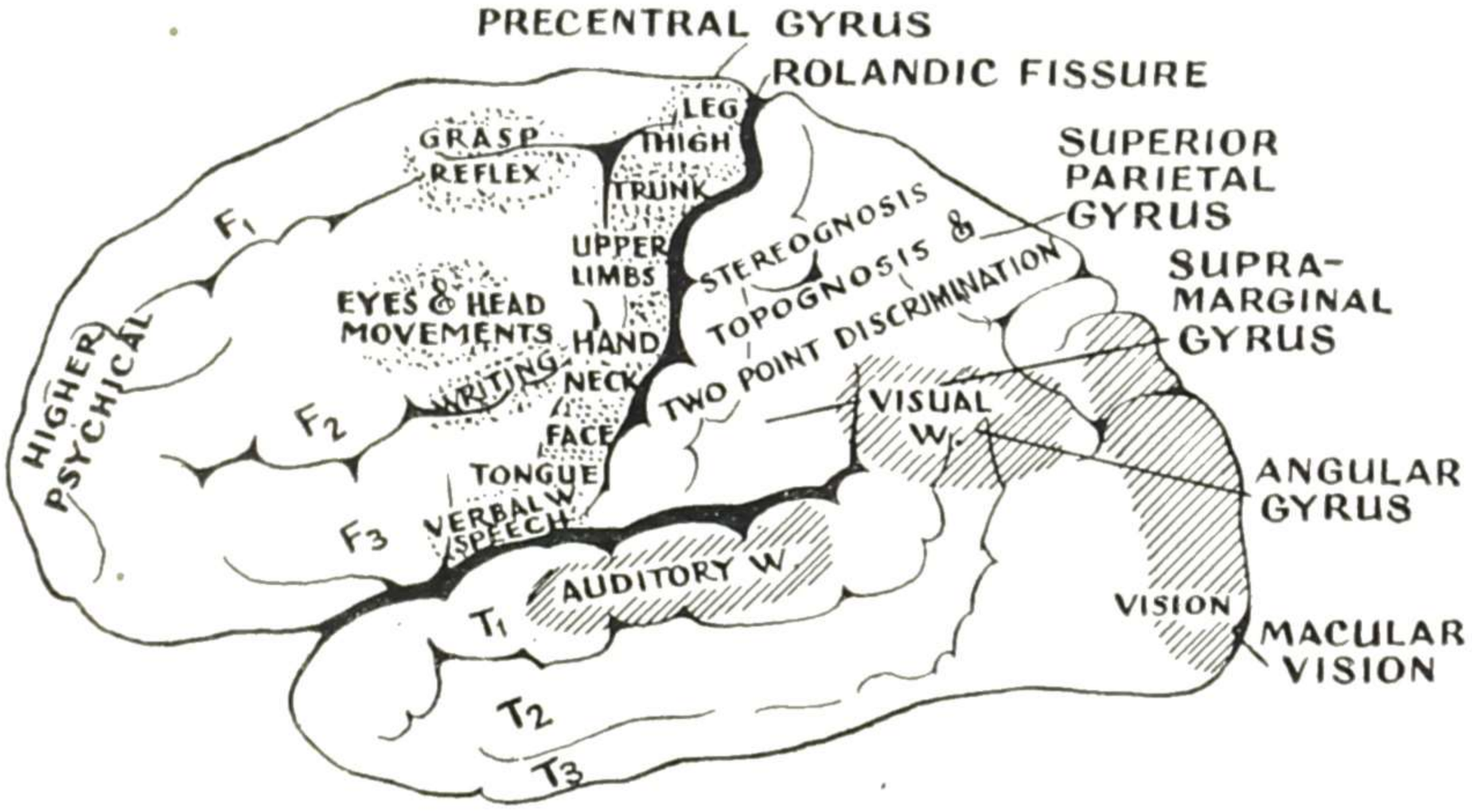


FIG. 157.—DIAGRAM OF THE CORTICAL AREAS (lateral aspect of hemisphere).
W. in diagram = Word area.

The SENSORY AREAS lie in the posterior lip of the Rolandic fissure and extend backwards to include the superior parietal, supra-marginal, and part of the angular gyri. Stimulation of these areas will produce, as the irritative sign, a sensory Jacksonian attack, while destruction causes: (1) Loss of ability to estimate the size, shape and consistency of objects held in the contralateral hand (Astereognosis), and (2) Impaired ability to localise tactile stimuli (Atopognosis), and to discriminate two simultaneous touches with compass points. Sensibility to pain and temperature is intact, but there is difficulty in appreciating the intensity of stimuli. The defect in spatial recognition leads to inco-ordination of the affected hand and digits.

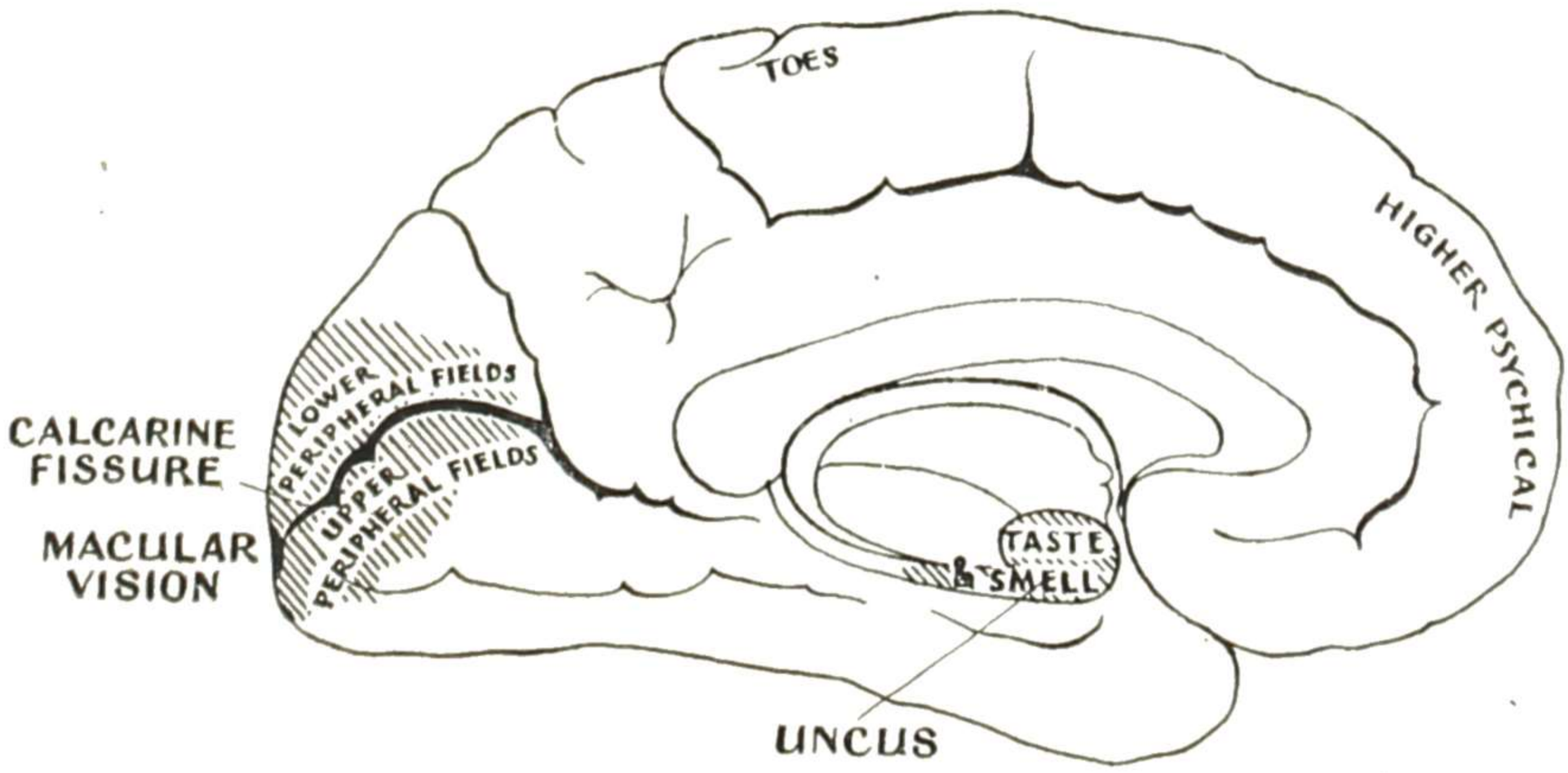


FIG. 158.—DIAGRAM OF THE CORTICAL AREAS (mesial aspect of hemisphere).

Further back is the VISUAL AREA (Fig. 158), represented in the upper and lower lips of the calcarine fissure on the mesial aspect of the occipital lobe; and extending, on the lateral aspect of the occipital lobe, into the angular gyrus. The right half of each visual field is represented in the left occipital cortex and vice versa.

Macular (*i.e.*, central) vision is represented at the tip of the occipital pole. The upper halves of both peripheral visual fields are represented in the lower lip of the calcarine fissure, the lower halves in the upper lip of this fissure.

Irritative lesions of the visual cortex cause visual Jacksonian attacks in the form of visual hallucinations of hemianopic distribution. In lesions far back on the occipital cortex these hallucinations take the form of moving lights, "sheets of

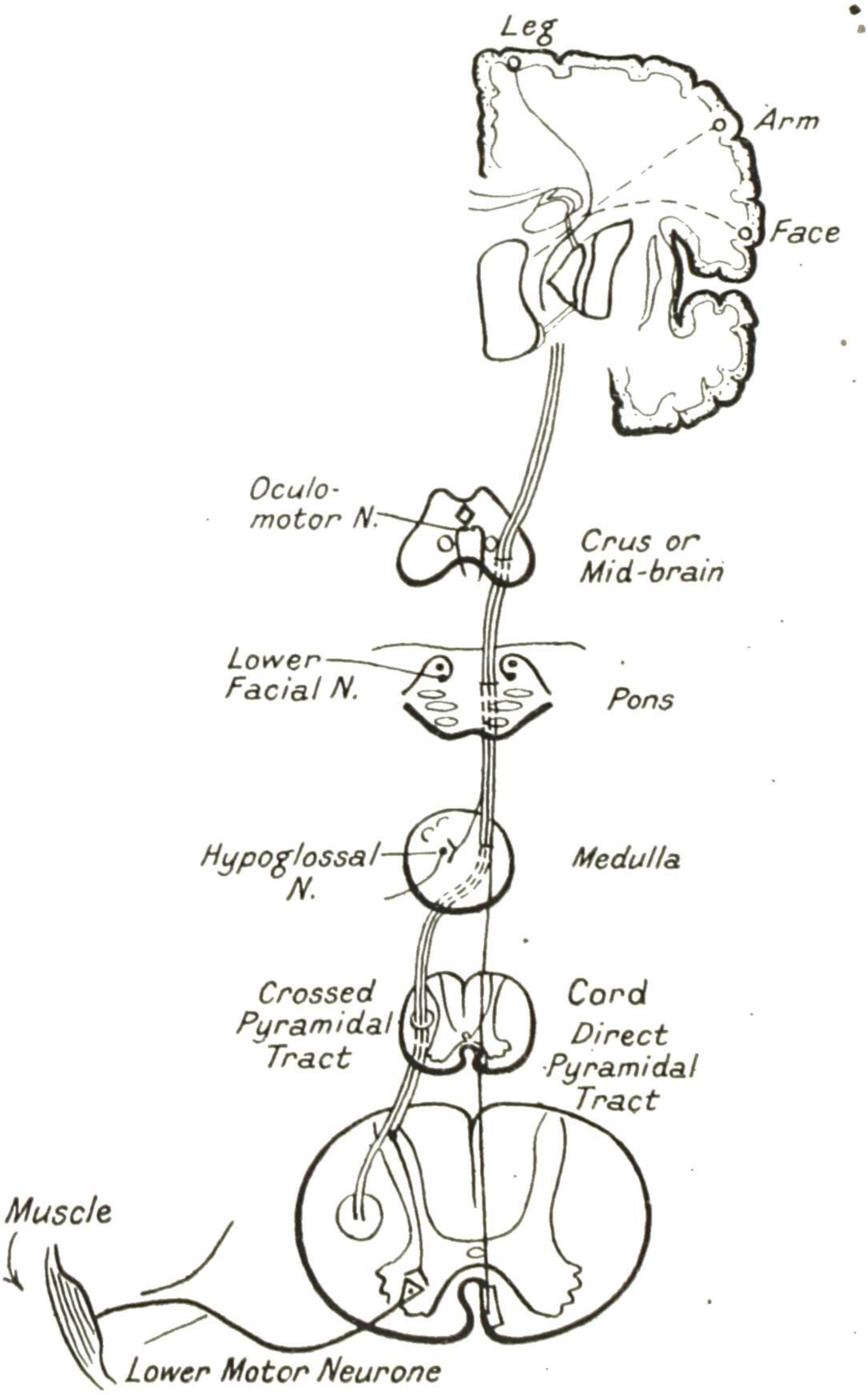


FIG. 159.—DIAGRAM OF THE PYRAMIDAL TRACTS, showing their course through the brain, brain-stem and cord.

flame," etc. With lesions involving the occipital and temporal cortex the visual hallucinations may be more complex, taking the form of scenes, "play-acting." Destructive lesions of the visual area will produce blindness of the crossed halves of each visual field (homonymous hemianopia), while partial lesions produce blindness of homonymous quadrants of the visual fields. Lesions of the angular gyrus cause impairment of stereoscopic vision and failure to recognise objects seen (visual agnosia).

The cortical AUDITORY AREAS are situated in the superior temporal gyrus. The cortical areas for TASTE and SMELL are in the uncinate gyrus on the mesial aspect of the temporal lobe. Irritative lesions of this area cause "uncinate fits," characterised by spitting and champing movements, associated with olfactory and gustatory hallucinations and a transient disturbance of consciousness or "dreamy state." The cortical areas for SPEECH are located in the left cerebral hemisphere, in right-handed individuals. They are situated in the posterior parts of the second and third frontal, the superior temporal, and in the angular gyri. Lesions of these areas cause various types of aphasia (see Fig. 175, § 743).

There are three great systems of projection fibres: the Motor, Afferent, and Visual Tracts. The Motor System consists of three groups of neurones: (1) Pyramidal Neurones, (2) Extra-Pyramidal Neurones, (3) Lower Motor Neurones.

§ 669. The Pathway for Voluntary Movements.—All impulses for voluntary movement are transmitted by the Pyramidal Tracts or Upper Motor Neurones. Damage to the pyramidal tract produces (1) *Impairment or loss of volitional movement* from interruption of the conduction of motor impulses. The resulting paralysis is termed a Monoplegia (paresis of one limb), Hemiplegia (paresis of face, arm and leg on one side of the body), Paraplegia (paresis of both lower limbs), or Diplegia (paresis of all four limbs). (2) *Release of extra-pyramidal motor phenomena*, viz., increase of tonus and exaggeration of the tendon reflexes, with the appearance of the extensor type of plantar response.

The Pyramidal Tract or Upper Motor Neurone (Fig. 159) extends from pyramidal cells of the pre-Rolandic cortex to the contralateral anterior horn cells of the spinal cord. From the cortical cells the fibres converge through a fan-shaped radiation, the corona radiata, to the internal capsule, which lies between the lenticular nucleus externally and the caudate nucleus and optic thalamus internally. The motor fibres occupy the genu and portion just anterior to this, and the anterior two-thirds of the posterior limb of the internal capsule. Here the fibres have undergone some rearrangement since leaving the cortex, for the order from before backwards is now face, shoulder, elbow, fingers, trunk, hip, knee and toes. Behind the motor fibres in the posterior limb of the internal capsule are the sensory and auditory fibres, and behind these the visual fibres of the optic radiations (Fig. 160).

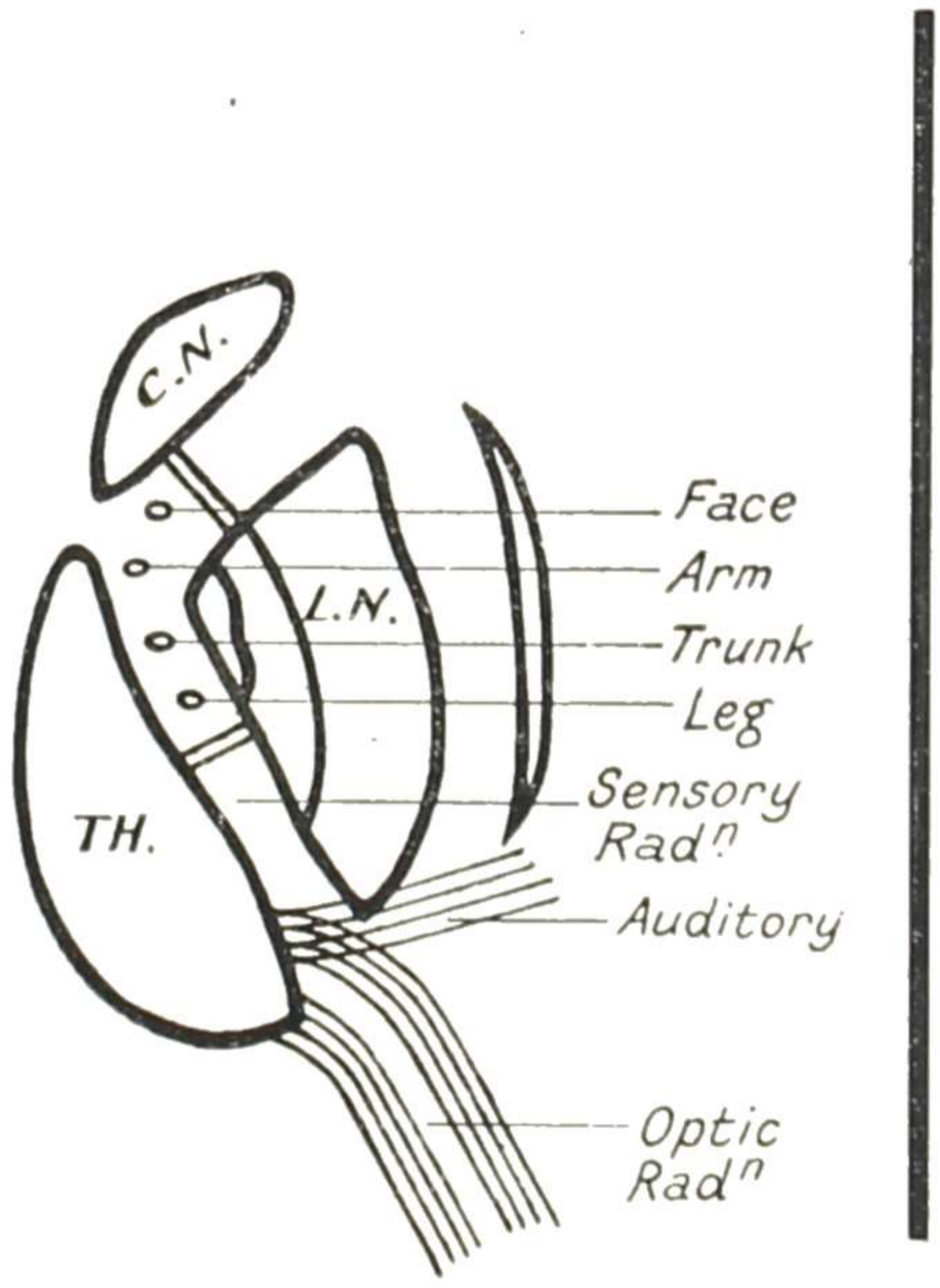


FIG. 160.—DIAGRAM OF INTERNAL CAPSULE.
(Horizontal section through the right internal capsule, showing the position of the different fibre tracts.)

From the internal capsule, the Pyramidal Tract descends through the ventral part of the crus cerebri (near the oculomotor nerve), spreading out a little in the ventrally situated formatio reticularis of the pons (the motor nucleus of the trigeminal is in the middle of the pons, and the abducens and facial nuclei in the lower part of the pons). On reaching the upper part of the medulla, the pyramidal tracts converge as the two ventrally and mesially placed pyramids. Below this, nearly all the fibres decussate to form the Crossed Pyramidal Tract which descends in the opposite lateral column of the cord. The pyramidal fibres do not end directly in the cells of the anterior horn but terminate in the region of the posterior horn,

whence short intermediary neurones connect to the anterior horn cells. A small proportion of pyramidal fibres are continued down the ipsilateral side of the cord as far as the mid-thoracic region lying near the anterior median fissure (the Direct Pyramidal Tract). These fibres eventually decussate to the opposite anterior horn cells.

All the motor nuclei of the cranial nerves receive fibres bilaterally from both pyramidal tracts, except the hypoglossal and that part of the facial nucleus connected with movements of the lower face. These receive fibres from the pyramidal tract of the contralateral side only. From the cells of the anterior horns and the cranial nerve motor nuclei the lower motor neurones arise (Fig. 159).

§ **670. Pyramidal lesions** may occur at various levels and produce the following clinical symptoms (Fig. 159):

(*a*) CORTICAL LEVEL: Owing to the extensive distribution of motor cells on the cortex a focal lesion will produce a monoplegia which is flaccid. If the lesion extends more deeply, involving many pyramidal fibres, the symptoms will be more widespread and spasticity will be present. Motor Jacksonian fits will occur as the irritative sign. A vertical lesion over both cortical leg areas will cause paraplegia, a paralysis of both lower limbs.

(*b*) INTERNAL CAPSULE: The convergence of the pyramidal fibres here is such that a relatively small lesion will produce a complete hemiplegia. In lesions of the genu the arm is more affected than the leg. In lesions farther back, there is hemianæsthesia and perhaps hemianopia, from involvement of the sensory fibres and optic radiations (Fig. 160).

In **Hemiplegia** (§ 752) there is unilateral loss of voluntary power in the affected limbs and the lower face. The tongue is protruded towards the paralysed side. The muscles of deglutition and mastication, which have bilateral pyramidal innervation from the cortex, usually escape, as do the trunk muscles. "Clasp-knife" rigidity (§ 704) with hypertonus appears in the affected limbs, in the flexors and adductors, so that the limb is held with the arm adducted at the shoulder, flexed at the elbow and wrist, with the forearm slightly pronated. The movements of the fingers and hand are more affected than the proximal movements. In the lower limbs the hypertonus appears in the extensors and adductors, while the movement most affected is dorsiflexion of the foot. The tendon reflexes become exaggerated and ankle- and rectus-clonus may develop, the plantar response is extensor in type and the abdominal reflexes on the corresponding side disappear, the lower abdominal reflexes disappearing before the upper ones. The gait is characteristic, the paralysed limb being dragged round in a semicircle, the toes scraping the floor.

(*c*) LEVEL OF CRUS: A "crossed paralysis" results, involving the oculomotor nerve on the same side as the lesion, and a contralateral hemiplegia (Weber's Syndrome) (Fig. 159).

(*d*) LEVEL OF LOWER PONS: A "crossed paralysis" results, involving the facial and abducens nerves on the same side as the lesion, and a contralateral hemiplegia (Millard-Gubler Syndrome).

(*e*) LEVEL OF SPINAL CORD: A spastic paresis of one or both lower limbs results. Lesions above the fifth cervical segment involve upper as well as lower limbs.

To summarise: **upper motor neurone lesions** produce, on the side affected: (1) Loss of voluntary power. (2) Spasticity of the "clasp-knife" type. (3) Increased tendon reflexes with ankle- and rectus-clonus. (4) Extensor plantar responses with absent abdominal reflexes. (5) Normal electrical reactions in the affected muscles. (6) No muscular wasting.

§ **671. Striatal Rigidity—Parkinsonism.**—The *corpus striatum* consists of the caudate and lenticular nuclei. From these important nuclei and from other masses of grey matter, notably the *Red Nucleus*, *Substantia Nigra*, and the *Sub-thalamic Body* (Corpus Luysii) of the mid-brain, and the *Vestibular* (*Deiter's*) *Nucleus* in the pons, the EXTRA-PYRAMIDAL MOTOR NEURONES arise and extend by one or more links to the anterior horn cells. The *Cerebellum* and its connections are really part of the

extra-pyramidal motor system, but for convenience these are considered later. It will be seen that the lower motor neurone is thus the "final common path" for both pyramidal and extra-pyramidal fibres (Figs. 159, 161).

The EXTRA-PYRAMIDAL MOTOR SYSTEM has no known connections with the cerebral cortex, and it probably represents a motor system phylogenetically older than the

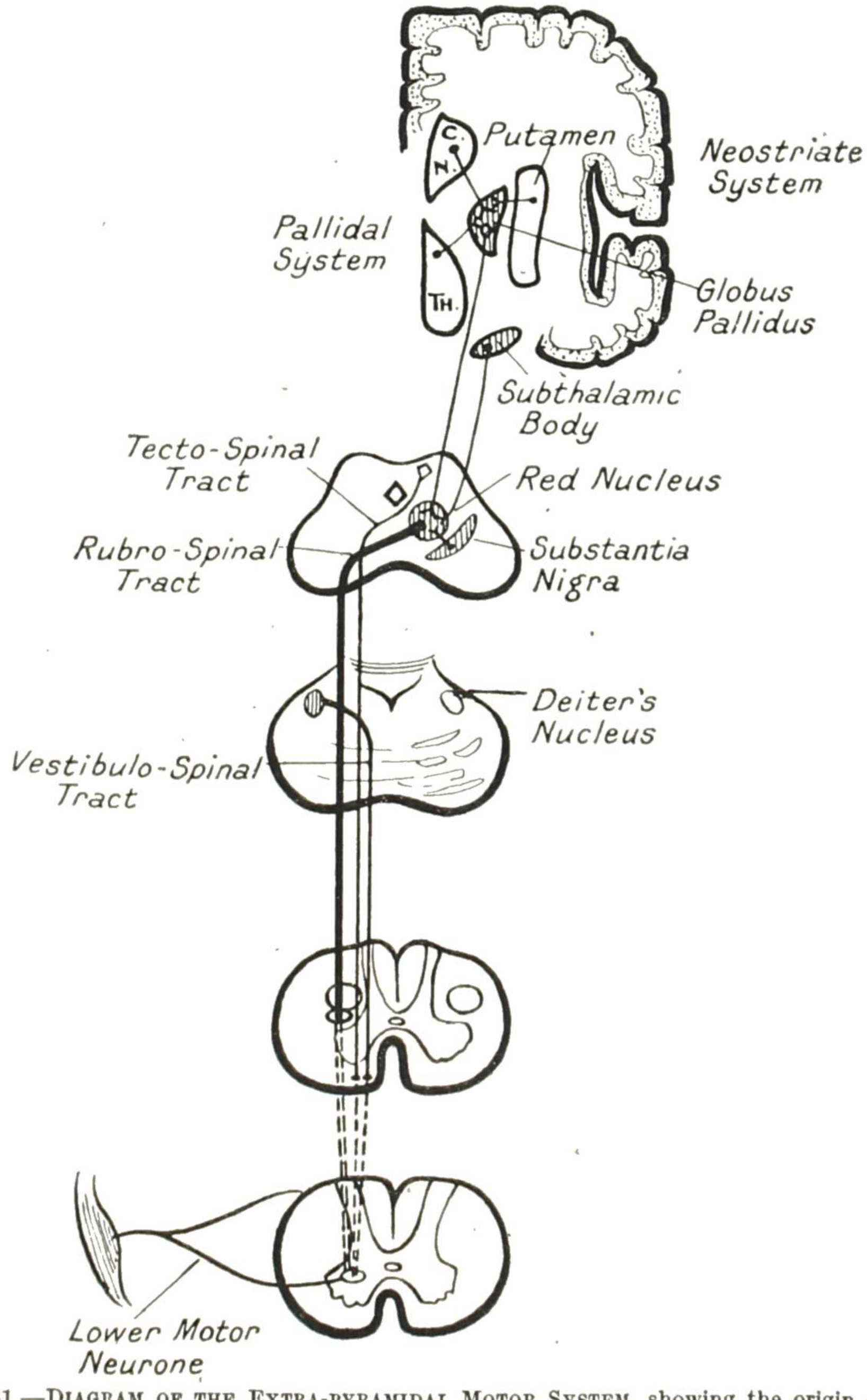


FIG. 161.—DIAGRAM OF THE EXTRA-PYRAMIDAL MOTOR SYSTEM, showing the origin of the extra-pyramidal motor tracts and their termination in the anterior horn cells.

pyramidal system. Lesions of the Extra-Pyramidal Pathways in man produce: (1) *Disturbance of muscle tone*, and (2) *Involuntary movements*, but *no true paralysis.* The reflexes are unaltered and there is no clonus, the plantar response remains of the flexor type, the abdominal reflexes are retained. *The symptom-complex most frequently encountered is Parkinsonism* or paralysis agitans, but hypotonia may also occur, with involuntary movements, athetosis, choreiform movements or tremor.

In **Parkinsonism** (§ 765) there is rigidity of both flexor and extensor muscles, the patient presenting a characteristic mask-like expression, with loss of the swinging movements of the arms on walking. On attempting to move the affected limbs passively, *e.g.*, at the wrist-joint, the examiner will encounter resistance like bending a piece of lead-pipe, or turning a cog-wheel, the so-called "lead-pipe" or "cog-wheel rigidity." The patient assumes an attitude of slight general flexion, the head and neck are bent forwards, the gait is shuffling or gliding, and the arms adducted and slightly flexed at the sides of the trunk (Fig. 4). The fingers are adducted in the "interosseal" attitude. All the movements are slow and restricted. Fine rhythmic tremor appears in the arm, leg, head or lower jaw. The reflexes are unaltered.

The anatomy of the Extra-Pyramidal Motor Neurones is complex (Fig. 161). The lenticular nucleus is subdivided into an external segment, the *putamen*, and a more important internal and smaller part, the *globus pallidus*. The globus pallidus consists of large pyramidal or multipolar cells like ventral horn cells, and receives short afferent axons from the optic thalamus, from the putamen and the caudate nucleus. It gives rise to efferent projection fibres ending in the substantia nigra and the red nucleus of the same side of the mid-brain. The whole corpus striatum is entirely unconnected with the cortex, and efferent fibres proceed only from the globus pallidus. These efferents do not pass directly to the ventral horn cells but link up with the red nucleus and vestibular and cerebellar tracts. There are three chief *Extra-Pyramidal Tracts*:

(1) *The Rubro-Spinal :* The fibres arise in the red nucleus, which is situated in the mid-brain, immediately decussate and pass down through the pons and medulla and the contra-lateral region of the cord to the anterior horn cells.

(2) *The Tecto-Spinal :* This tract arises in the mid-brain at the level of the superior corpora quadrigemina, decussates and passes down in the posterior longitudinal bundle to the anterior horn cells. It conveys impulses to the voluntary muscles as the result of stimuli from the retinæ, which have passed to the calcarine fissure, and thence to the superior corpora quadrigemina.

(3) *The Vestibulo-Spinal :* The fibres arise in the lateral vestibular nucleus of the VIII nerve (Deiter's nucleus) in the lower pons and pass, in the antero-lateral region of the cord, to the anterior horn cells of the same side. This tract is the efferent of mid-brain reflexes subserving muscular tonus.

The short axons, passing from the small cells of the caudate nucleus and putamen to the globus pallidus, are sometimes termed the *Small-Celled Neo-Striate System*. The large cells of the globus pallidus and their efferents are sometimes termed the *Large Celled Pallidal System*. The latter system is phylogenetically older, and lesions of its cells and efferents cause Parkinsonism.

§ 672. **Involuntary Movements—Tremor, Chorea, Athetosis.**—Three main types of involuntary movements occur in voluntary musculature. They are never present when the muscles are completely paralysed, *i.e.*, there must be relative integrity of the pyramidal tracts. They are: (1) *Tremor*—Involuntary, rhythmical oscillations of one or more parts of the body, resulting from the alternate contraction of muscle groups and their antagonists (§ 770). (2) *Chorea*—Irregular and spasmodic involuntary movements of groups of muscles, occurring during rest, and also superimposed upon voluntary movements, which they render inco-ordinate (§ 771). (3) *Athetosis*—Involuntary movements, of a writhing or (in the face) grimacing type, slower and more stereotyped than the movements of chorea. Between the periods of increased spasm the limbs are frequently hypotonic (§ 771).

The pathogenesis of these involuntary movements is obscure. By some, they are ascribed to lesions of the short axons passing from the small cells of the caudate nucleus and putamen to the globus pallidus (the small-celled neo-striate system). In congenital athetosis and chorea, the lesions are found chiefly in the caudate nucleus and putamen. Acute focal lesions of the *sub-thalamic body or corpus Luysii* (Fig. 161),

however, are known to produce hemichorea of the contralateral half of the body, and choreiform movements are known to follow lesions of the superior cerebellar peduncle and the optic thalamus. In this connection, it should be remembered that the cerebellum is really part of the extra-pyramidal motor system. The consensus of opinion is that these involuntary movements result from lesions of the extra-pyramidal paths when the pyramidal pathways are relatively intact.

§ **673. Flaccid Paralysis.**—*The Lower Motor Neurone* commences in the anterior horn cell, or motor cell in the brain stem, and ends in the muscle fibre. It is the final pathway for motor impulses, whether pyramidal or extra-pyramidal, and is an integral part of spinal reflex arcs subserving muscular tonus. When it is destroyed, therefore, muscular paralysis, flaccidity, and absence of tendon reflexes, ensue. The muscle, completely isolated from the central nervous system, atrophies, and contractures occur; its electrical excitability disappears (Reaction of Degeneration) (see § 709). When the damage to the lower motor neurone is short of actual destruction, these changes appear in proportionately slighter degree. To summarise: the characteristics of **lower motor neurone lesions,** are: (1) Muscular paralysis and wasting. (2) Flaccidity. (3) Loss of tendon reflexes. (4) Reaction of degeneration.

§ **674. Sensory Pathways.**—A mixed peripheral nerve contains fibres subserving every aspect of sensibility, cutaneous and deep. The *Cutaneous* sensory impulses are those of (1) Touch, (2) Pain, (3) Temperature. The *Deep* sensory impulses are (1) Vibration of a tuning-fork on bone, (2) Sense of passive movement of joints, (3) Sense of position, (4) Deep muscular sensibility, and tendon sensibility to deep pressure. All these sensory impulses, cutaneous and deep, enter the spinal cord through the posterior roots (Fig. 162).

The fibres for *Pain*, *Temperature* and *Touch* are freshly relayed in the posterior horn and cross over immediately on entering the cord in the *anterior commissure* and ascend directly in the spino-thalamic tract of the opposite side to the lateral nucleus of the thalamus. In the spinal cord the spino-thalamic tract lies in the lateral column, just ventral to the pyramidal tract (Fig. 163). The fibres for *Deep Sensibility* (viz., vibration, sense of passive movement and position, deep muscular sensibility, and tendon sensibility) together with some of the fibres for touch, ascend without relay in the posterior columns of Goll and Burdach of the same side, to the ipsilateral nucleus gracilis and nucleus cuneatus in the medulla. Here they are freshly relayed and ascend in the decussation of the *mesial fillet* (arcuate fibres) to the thalamus of the contralateral side. So that, eventually, all the sensory impulses, whether cutaneous or deep, undergo a decussation either immediately on entering the cord or later, in the fillet, and terminate in the contralateral thalamus. It will be noted that there are two pathways for touch. Tactile fibres, on entering the cord, ascend both in the contralateral spino-thalamic tract and in the ipsilateral posterior columns.

In the cord we have both crossed and uncrossed sensory pathways. In the spino-thalamic tracts and in the posterior columns, the longest posterior root fibres ascending from the coccygeal and sacral segments, lie nearer the mid-line. As fibres enter at higher segmental levels they are conveyed in a lamellar fashion, so that the fibres derived from lower segments are displaced inwards by those entering at higher levels. Furthermore, the fibres for touch, pain and temperature, entering the cord to cross to the spino-thalamic tract, decussate in the anterior commissure in a diagonal fashion; the fibres for pain and temperature crossing, say, in the mid-dorsal region in the space of one segment, those for touch crossing more slowly, the decussation occupying two segments. At higher segmental levels the crossing is more and more oblique.

The *spino-thalamic tract*, containing pain, temperature and touch fibres, ascends through the formatio reticularis of the medulla to join the crossed mesial fillet, in the pons, ultimately reaching the thalamus. The fibres for temperature and pain diverge in the medulla from the tactile fibres and pass to the outer side of the olive, subsequently converging to the mid-line and joining the fillet.

The *posterior columns of Burdach and Goll* are relayed in the nucleus cuneatus and nucleus gracilis, the latter more-mesial nucleus receiving fibres from the lower

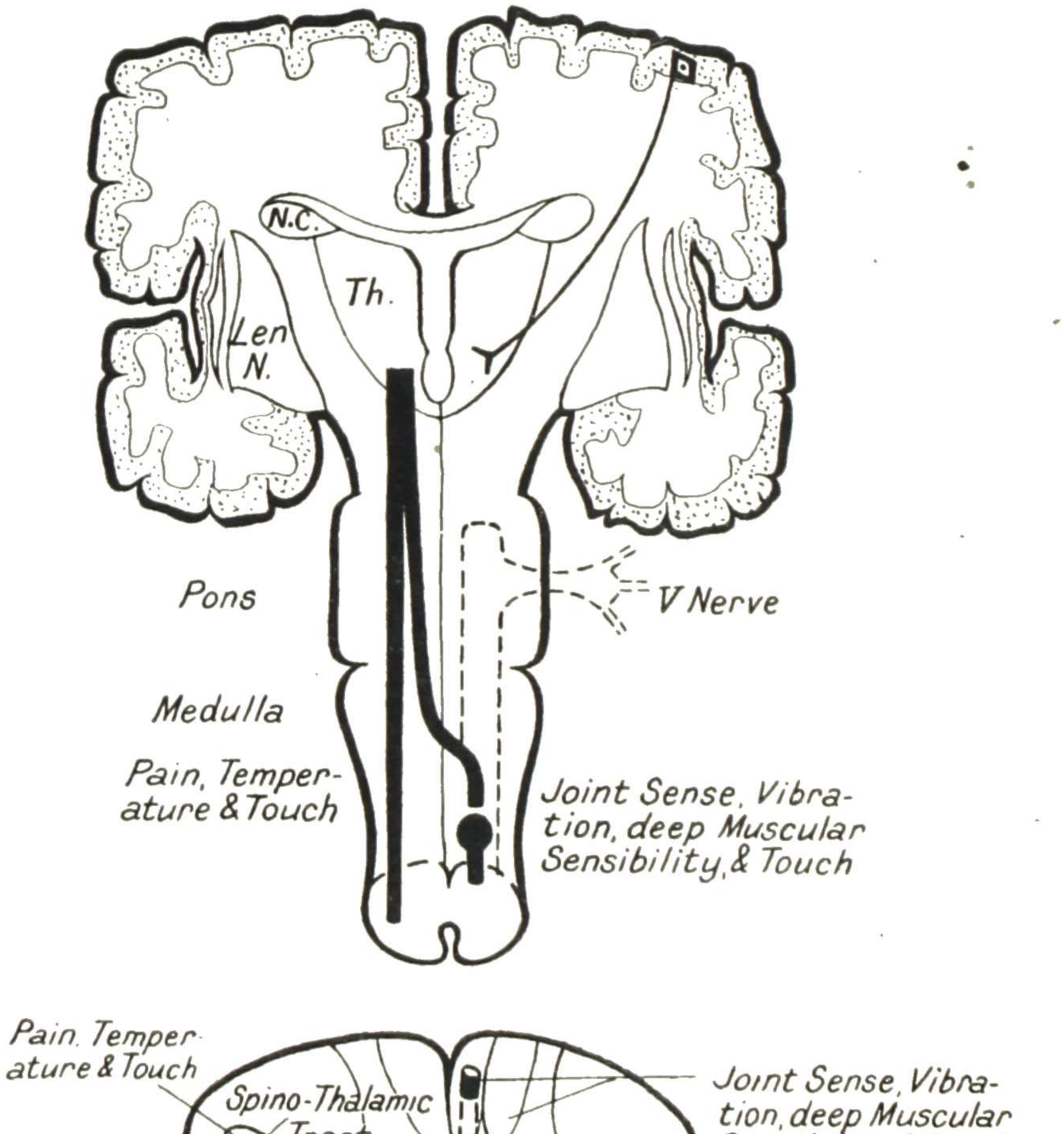


FIG. 162.—DIAGRAM OF THE SENSORY TRACTS. (After Head, Holmes, Walshe, Bing.)

limbs. Fibres concerned with deep sensibility pass upwards in these tracts and decussate, as the arcuate fibres in the medulla, to form the *mesial fillet*; this terminates in the lateral nucleus of the thalamus. The *fillet*, in the pons, passes along

the inner side of the sensory nucleus of the trigeminal nerve of the same side. The *thalamus*, situated in the lateral wall of the third ventricle, receives all the sensory impulses of the body, with the exception of the gustatory impulses, which have a direct connection with the cortex of the uncus. The thalamus registers the crude affective sensations of pain, heat, cold, producing emotions of pleasure or pain, and visceral sensations of hunger and thirst.

Inhibitory fibres run from the cortex to the thalamus, and, in certain circumstances, physical sensations are prevented from reaching the level of consciousness. For instance, we may be holding a book we are reading, quite unconscious of the pressure of the book on the hand which is holding it, until our attention is directed to this sensation. From the thalamus all sensory impulses are relayed to the *sensory cortex*, where sensation is discriminative.

Lesions of the *Sensory Cortex* do not affect crude sensations of temperature or pain. The characteristics are : (1) Loss of ability to localise tactile cutaneous stimuli (Atopognosis), (2) Defective appreciation of the size, shape and consistency of objects held in the hand (Astereognosis), (3) Light touches may be imperfectly felt, (4) there is impairment of Two-Point Discrimination (Compass-test), and (5) difficulty in appreciating the intensity of stimuli. The loss of these discriminative features of sensation may lead to inco-ordination of the affected limb. Sensory testing in cortical lesions gives great variety of response and threshold (Fig. 157).

Sub-cortical lesions produce a hemianæsthesia, affecting the contralateral face, upper and lower limbs. At the level of the sensory nucleus of the trigeminal nerve a lesion will produce a crossed hemianæsthesia, affecting the face on the same side as the lesion (from the proximity of the fillet to the ipsilateral sensory fifth nucleus) and the upper and lower limbs and trunk on the opposite side.

Lesions of the Thalamus release this structure from cortical control, with the production of characteristic symptoms of over-reaction to stimuli on the contralateral side—the "thalamic syndrome" of Déjérine and Roussy. This comprises (1) hemianæsthesia, (2) spontaneous pain on the affected side of the body, (3) over-reaction to painful or unpleasant sensory stimuli.

Central Cord Lesions (such as syringomyelia or intramedullary tumour) involve the fibres for pain and temperature, which cross in the anterior commissure, together with the touch fibres which cross in this region. Touch, as we have seen, has a double pathway ; some of the fibres ascend in the posterior columns and consequently escape. The resulting cutaneous anæsthesia was termed by Charcot "Dissociated Anæsthesia," *i.e.*, there is loss of sensation to pin-prick, hot and cold (pain and temperature) while touches with cotton-wool can still be felt.

In *Hemi-section of the Cord*, posterior column sensibility is lost on the same side as the lesion ; while pain and temperature are lost, and touch blunted on the opposite side. Lesions of the *posterior column* cause loss of joint sense, vibration and deep muscular sensibility. Sensitivity to light touches, as with cotton wool, is impaired. The loss of joint sense results in ataxia.

Lesions of the Posterior Roots or *Root-Entry Zone* of the cord (as met with in tabes) may produce impairment of all forms of sensation. From affection of the non-sensory afferents of spinal reflex-arcs subserving the tendon reflexes, these reflexes disappear, with loss of muscular tone.

These are also the pathways for certain **Non-Sensory Afferents** of reflex-arcs, situated at all levels in the spinal cord and brain-stem, conveying impulses which never reach consciousness, concerned with the *maintenance of muscular tonus*. They are, however, no less important than the sensory afferents and comprise :

(1) The long afferents of mid-brain reflex arcs concerned in the maintenance of tonus (see Muscular tonus, § 682).

(2) The dorsal and ventral cerebellar tracts which (with some collaterals from the posterior columns) carry to the cerebellum proprioceptive impulses for muscles and tendons important in the regulation of posture.

In a disease of the afferent system of projection fibres, such as tabes, besides sensory loss there are present also symptoms referable to destruction of these non-sensory reflex-arcs—*e.g.*, hypotonia, loss of tendon reflexes and inco-ordination.

Ascending and Descending Tracts in the Spinal Cord (Fig. 163).—The *Posterior Columns* are composed of the ascending *Tracts of Goll and Burdach*, carrying uncrossed fibres subserving Deep Sensibility (viz., Postural Sense, Sense of Passive Movement of Joints, Vibration Sense, Deep Muscular Sensibility) and the uncrossed fibres for Touch. These all pass to the nucleus gracilis and cuneatus, whence they are relayed in the mesial fillet to the contralateral thalamus.

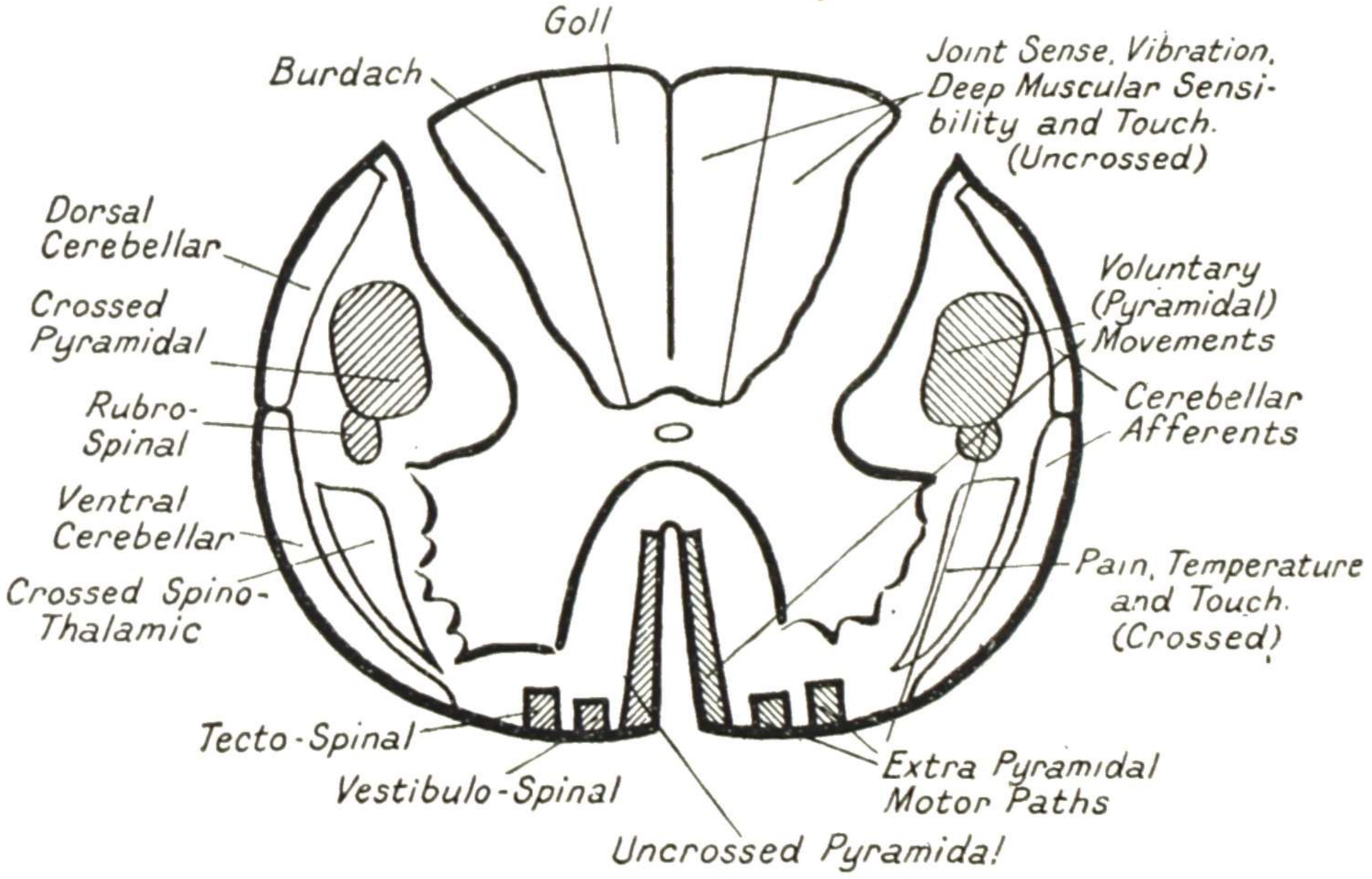


FIG. 163.—DIAGRAM OF ASCENDING AND DESCENDING TRACTS IN SPINAL CORD.

(Motor tracts shaded.) On the left half of the diagram are shown the names of the tracts, and on the right half of the diagram their functions.

The *Lateral Columns* contain the descending *Crossed Pyramidal Tract* and, just ventral to this, the *Rubro-spinal Tract*, the chief extra-pyramidal motor tract. These motor fibres are all relayed to the anterior horn cells. The periphery of the lateral columns is occupied by two ascending cerebellar tracts, the *Dorsal* (*Direct*) and *Ventral Cerebellar Tracts*, containing cerebellar afferents relayed from the cells of Clarke's Column.

The *Anterior Columns* contain the descending *Direct* (*Uncrossed*) *Pyramidal Tracts* and two extra-pyramidal motor tracts, the *Vestibulo-Spinal* and *Tecto-Spinal Tracts*. These descending motor tracts are all relayed to the anterior horn cells. The ascending *Spino-thalamic Tracts* lie in the antero-lateral region of the cord, carrying crossed fibres subserving Cutaneous Sensibility (viz., Pain, Temperature and most of those for Touch) to the fillet and ipsilateral thalamus.

Segmentation in the Spinal Cord. Each spinal segment or metamere has its anterior (motor) and posterior (sensory) roots. The *spinal nerves* are formed by fusion of one anterior and one posterior spinal root. The spinal nerve then divides into an *anterior* and a *posterior primary division*. Each of these divisions contains motor and sensory fibres, and ultimately supplies the cutaneous segment (Fig. 176) and the muscles (Table LII) developed in connection with its corresponding metamere.

§ 675. The Pathways for the Special Senses.—The peripheral organs for vision, hearing, taste and smell, are paired, and, in man, the sensory fibres proceeding from them to the cortex all exhibit a hemi-decussation, so that the cortex of one hemisphere subserves both organs in the case of each of these senses. In man, taste and smell have become intimately combined and have the same cortical representation, in the *uncus* and *hippocampal gyri*.

I. VISUAL SENSE.—For convenience the visual mechanisms are considered separately (§ 676).

II. AUDITORY SENSE.—The Eighth Cranial Nerve consists of a sensory or COCHLEAR division concerned with hearing, and a non-sensory or VESTIBULAR portion concerned with equilibrium. The *Cochlear nerve fibres* arise from cells in the SPIRAL GANGLION situated in the central pillar of the cochlea, their peripheral terminations ending in the hair cells of the organ of Corti. Centrally, the cochlear nerve passes to the brain through the internal auditory meatus and enters the lower border of the pons to terminate in the dorsally-placed *Cochlear nucleus* (tuberculum acusticum) which lies in the lower pons just external to the restiform body. From here, the fibres decussate as the *striæ acusticæ* and run in the *lateral fillet* to the Internal Geniculate Body and Inferior Corpus Quadrigeminum (*Primary Auditory Centres*). From the Primary Auditory Centres a fresh relay of fibres arises, which passes to the higher auditory areas in the superior temporal gyrus of the cortex.

Destruction of the Cochlea or the cochlear nerve produces *nerve-deafness*, irritative lesions produce *tinnitus*. Lesions of the cochlear nucleus in the pons, or the lateral fillets, will have a similar effect, but brain-stem deafness is rare. Lesions of the superior temporal gyrus do not destroy hearing because each ear has a bilateral cortical representation, but, in right-handed people, a lesion of the left superior temporal gyrus abolishes the comprehension of words and sounds heard (word-deafness).

III. GUSTATORY SENSE.—Smell and Taste are difficult to separate in man, in whom the old rhinencephalon or "smell-brain" has largely lost its function. Taste is concerned with cruder sensations of sweetness, sourness and bitterness, while smell is concerned with the appreciation of odours and flavours. Both taste and smell have the same cortical representation in the *gustatory area*, situated in the uncus and hippocampal gyri on the mesial aspect of the temporal lobe (Fig. 158).

(*a*) Taste.—The anterior two-thirds of the tongue is supplied by the lingual nerve. Fibres for taste leave the lingual nerve and enter the chorda tympani, having their cell-station in the geniculate ganglion in the aqueductus Fallopii. Thence they reach the nucleus of the nervus intermedius (dorsal nucleus of the seventh nerve, n. gustatorius) in the pons. From the posterior third of the tongue, taste-fibres pass along the glosso-pharyngeal nerve to their cell-station in the petrous ganglion, and thence they reach the dorsal nucleus of the glosso-pharyngeal nerve in the medulla. From the nuclei of the nervus intermedius and glosso-pharyngeal, fibres arise (fasciculus solitarius) and undergo a hemi-decussation passing to the uncus.

(*b*) Smell.—The olfactory nerves pass from the end-organs in the nasal mucosa through the cribriform plate of the ethmoids to the olfactory bulbs. Here fibres are relayed in the olfactory tracts. Each olfactory tract divides into a lateral and a mesial portion. The mesial portions decussate and join with the uncrossed lateral portions to terminate in the uncus.

Lesions of the olfactory bulbs or tracts produce *anosmia*. Irritative lesions of the uncus or hippocampal gyri produce *uncinate fits*—subjective sensations of taste and smell associated with champing or spitting and a transient dimming of consciousness, known as a "dreamy state." Anosmia does not occur from these cortical lesions. Loss of taste on the anterior two-thirds of the tongue occurs in lesions of the facial nerve in the aqueductus Fallopii, involving the geniculate ganglion.

§ 676. The Visual Mechanisms.—(*a*) **Visual Sense.**—The visual projection fibres are, clinically, of great importance. Each *Optic Nerve* passes backwards from

the globe into the cranial cavity and divides into a mesial and a lateral portion (Fig. 164). The mesial portions decussate at the *Optic Chiasma*, which lies just in front of the pituitary fossa, the uncrossed lateral divisions joining with the crossed mesial divisions to form the short *Optic Tracts*. The *Optic Tracts* bend round the lateral aspect of the mid-brain and terminate in the *External Geniculate Bodies or*

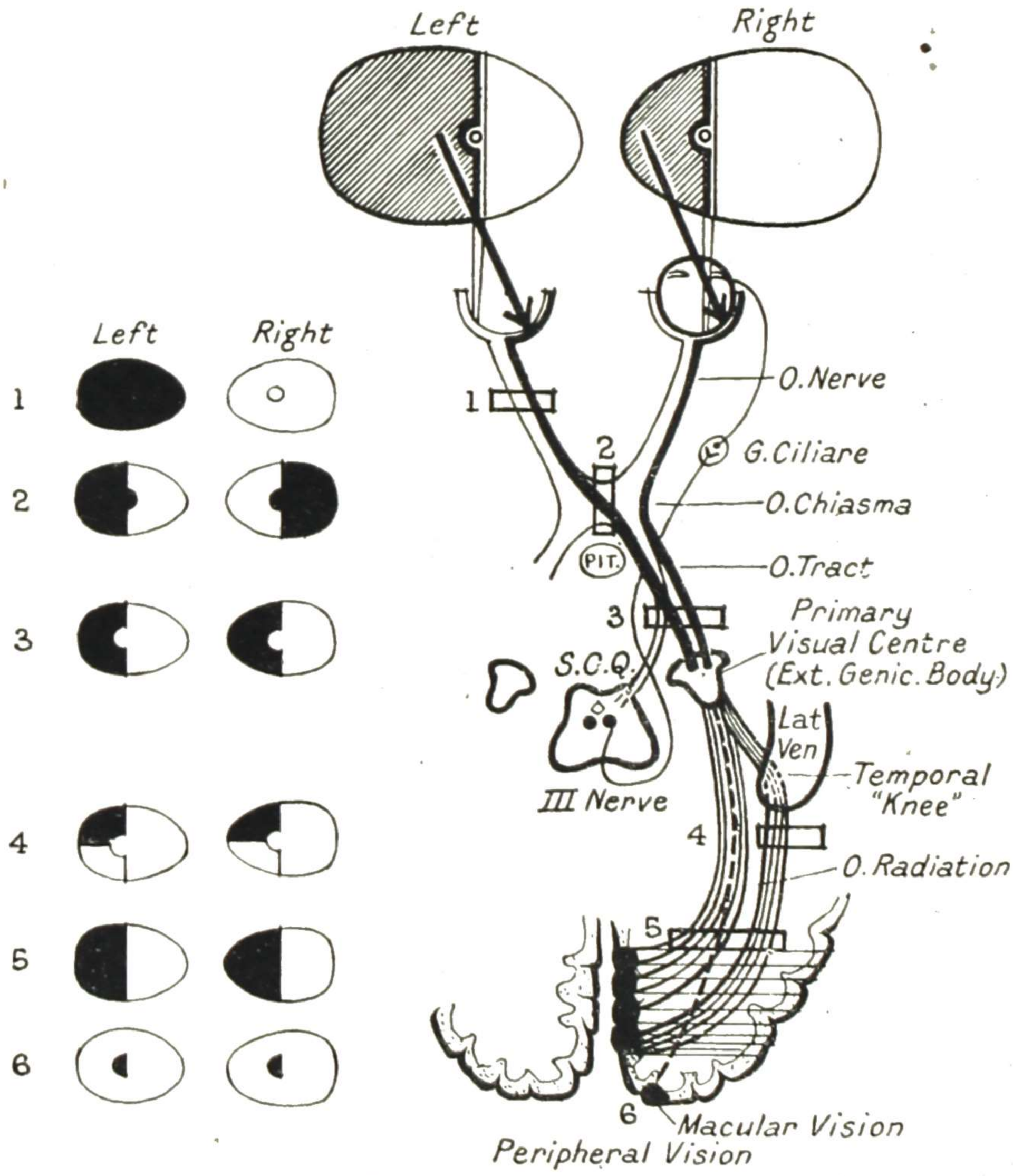


FIG. 164.—DIAGRAM OF THE VISUAL MECHANISMS.

Visual Field Defects resulting from Lesions at Different Points are represented on left of Diagram.

Lesion at 1 causes Left Optic Atrophy and Blindness in Left Eye.
" " 2 " Bitemporal Hemianopia with Involvement of Macular (Central) Vision.
" " 3 " Left Homonymous Hemianopia.
" " 4 " Left Upper Quadrantic Hemianopia from Involvement of Ventral Bundle (Temporal Knee) of Optic Radiations.
" " 5 " Complete Left Homonymous Hemianopia.
" " 6 " Left Homonymous Central Hemiscotoma.

The Path of the Pupillary Light Reflex is also shown. (Modified from Walshe.)

Primary Visual Centres. (N.B.—The Optic Thalamus has nothing to do with visual mechanisms.)

Some of the fibres in the Optic Nerves are non-visual in function and constitute afferent arcs of mid-brain reflexes concerned in the pupillary reactions to light and accommodation, and ocular movements. These non-visual fibres do not terminate in the External Geniculate Body but in the *Superior Corpus*

Quadrigeminum, which effects their connection with the ocular nuclei in the floor of the aqueduct.

From the Primary Visual Centres arise the *optic radiations*, which pass through the internal capsule behind the sensory fibres to the calcarine cortex. Each optic radiation contains fibres relayed from the corresponding halves of both retinæ. Furthermore, the dorsal bundle of the optic radiation, containing fibres relayed from the superior corresponding halves of both retinæ, passes directly backwards to the cuneus or upper lip of the fissure. The ventral bundle of the optic radiation, containing fibres from the inferior corresponding halves of both retinæ, runs first downwards into the uncus, then turning round the tip of the descending horn of the lateral ventricle ("temporal knee") runs backwards to reach the lower lip of the calcarine fissure.

The *macular fibres* also undergo a hemi-decussation in the chiasma, and are relayed from the external geniculate body to the tip of the occipital pole in the optic radiations. The tip of each occipital pole contains fibres relayed from the corresponding halves of both maculæ. The calcarine cortex subserves peripheral vision, while the cortex at the tip of each occipital lobe subserves central vision.

A lesion of one *Optic Nerve* will cause blindness in that eye. A lesion of the *Optic Chiasma* will abolish the functions of the nasal half of both retinæ and produce blindness of both temporal fields (bitemporal hemianopia) (Fig. 164). Lesions in this situation may also involve the uncrossed fibres, producing a complete blindness in one eye with a temporal hemianopia in the other. Lesions of the *Optic Tract* will abolish the functions of the corresponding halves of both retinæ, producing blindness of the contralateral halves of the visual fields (homonymous hemianopia). Lesions anterior to the superior corpus quadrigeminum and external genioulate body, causing blindness, are associated with loss of pupillary reaction to light when a pencil of light is thrown on one half of the retina, owing to the interruption of the non-visual afferents of the pupillary light reflex (Wernicke's hemianopic pupillary reaction) (Fig. 164). In blindness due to disease of the calcarine cortex, the pupils react when a beam of light is thrown on the retina.

Lesions of the *Optic Radiation* will produce homonymous hemianopia of the contralateral fields. Quadrantic hemianopia of the contralateral visual fields occurs when only a portion of the optic radiations are destroyed, the lower quadrants of the visual fields being the ones affected when the dorsal bundle is destroyed, the upper quadrants when the ventral bundle is destroyed.

Lesions of the *Visual Cortex*, when at the tip of one occipital pole, will produce a central hemiscotoma of the contralateral halves of both central visual fields. Central scotoma can also be produced by pressure lesions at the chiasma where the macular fibres lie ventrally and are delicate and vulnerable. Lesions of the upper lips of both calcarine fissures produce a horizontal hemianopia inferior, with blindness of the lower halves of both visual fields. Lesions of the left angular and supra-marginal gyri on the convex surface of the hemisphere, cause difficulty in recognising objects seen, without actual blindness, "visual agnosia" (word blindness) in right-handed people (Fig. 175).

§ 677. (*b*) **The Pupil and its Reactions.**—The cervical sympathetic is the *tonic dilator of the pupil*, the oculo-motor nerve is the *tonic constrictor of the pupil*. It has already been stated that the Optic Nerve and Optic Tract, besides containing visual fibres, also contain non-visual fibres which pass, not to the Primary Visual Centres but to the *Superior Corpus Quadrigeminum* (*Superior Colliculi*). These non-visual fibres form the afferent pathway for light impulses concerned with the *Pupillary Light Reflex* (Figs. 164 and 190). From the Superior Corpus Quadrigeminum in the mid-brain short *colliculo-ocular fibres* arise, and, surrounding the aqueduct and decussating below it, effect communication with the *Oculo-Motor Nuclei* in the floor of the aqueduct. This decussation puts each corpus quadrigeminum in connection with both oculo-motor nuclei and explains the *consensual light reaction*—when a beam of light

§ 679. Inco-ordination of Movement—Ataxia.—Any lesion of the spinal cord, brain-stem or cerebral hemispheres, which interrupts the afferent impulses subserving sense of position and the appreciation of movement (joint-sense), will produce *Sensory Ataxia*. It will be remembered that these impulses are conveyed in the posterior columns to the contralateral thalamus and are thence relayed to the cortex. Clinically, such ataxia is well exemplified in diseases affecting the posterior columns, such as tabes or subacute combined degeneration of the cord. It may also, however, result from peripheral nerve lesions (peripheral neuritis) or disease of the sensory cortex. This form of ataxia is increased when the patient's eyes are closed, as the eyes give useful information about the position of the limbs.

In lesions of the cerebellum or its connections, ataxia is also met with. It is usually attributed to the loss of tonus which is observed in cerebellar disease and it is termed *Cerebellar Ataxia*. It is well exemplified in the phenomenon of intention tremor, to demonstrate which the patient is asked to touch his nose with his forefinger. The finger oscillates about its objective just before it is reached, but the nose is touched correctly even if the eyes are shut.

§ 680. The Mechanism of Equilibrium. Equilibrium is dependent on perfect co-ordination of certain afferent impulses. These impulses are set up by : (1) movement of the limbs acting through the muscle-spindles, joints and tendons, (2) vision and (3) gravity, the latter acting through the vestibular apparatus.

The vestibular apparatus consists of: (*a*) The utricle and saccule containing sensitive hair-cells in contact with small crystals, the otoliths. The position of the otoliths with regard to the hair-cells varies with gravity, and impulses are set up in the saccules when the head is moved *vertically* or *forwards*. (*b*) The semicircular canals, also containing sensitive hair-cells which respond to movement of the endolymph. Impulses are set up in the semicircular canals by *angular* displacement of the head.

Impulses are constantly being received from the vestibular apparatus telling the position of the head in space. The cells of origin of the vestibular nerve are in Scarpa's ganglion in the internal auditory meatus. Fibres pass centrally to the vestibular (Deiter's) nucleus, which lies ventrally in the pons in the outer part of the floor of the fourth ventricle (Fig. 165). Efferent fibres connect this nucleus with the anterior horn cells (vestibulo-spinal tract) and the ocular nuclei (posterior longitudinal bundle). Deiter's nucleus receives afferents also from the cerebellum. Disturbances of the vestibular mechanism produce : (1) subjective vertigo, (2) hypotonia on the side of the lesion, (3) forced movements and falling, and (4) nystagmus.

In a normal subject certain disturbances (*e.g.*, sudden cessation of rotation, rocking) will cause maladjustment of postural reflexes and confusion. If a normal individual is rotated in a rotating chair which is suddenly checked and tilted backwards, the whole vestibular mechanism is so disturbed that the person may collapse or be flung out of the chair. For a time he will have no conception of how to orient himself in space. Ewald found that ablation of the vestibule causes hypotonia of the ipsilateral half of the body. A similar result will temporarily follow section of the vestibular nerve.

§ 681. The Cerebellar System (Brain of Posture) is an integral part of the extra-pyramidal motor mechanism, consisting of complex afferent and efferent systems of neurones. The *afferent* fibres come from the spinal cord (direct cerebellar tracts and fibres from the posterior columns) and the mid-brain (Deiter's vestibular nucleus and the oculomotor nuclei). The *efferent* fibres do not lead directly downward from the cerebellar cortex but are relayed from the central grey matter of the cerebellum (dentate and roof nuclei). These efferents run in three main groups : (1) *Cerebello-Rubro-Spinal :* to the opposite red nucleus and thence through the rubro-spinal tract, after recrossing to the anterior horn cells. Through these fibres the cerebellum is concerned with the tonus of the ipsilateral skeletal muscles. (2) *Cerebello-Vestibulo-spinal :* to the Vestibular (Deiter's) nucleus of the same side in the pons and thence through the vestibulo-spinal tract to the anterior horn cells. Through these fibres the cerebellum is concerned with correction for posture in voluntary movements. Through

the posterior longitudinal bundle these cerebellar efferents are brought in communication with the oculomotor nuclei in the floor of the iter, and the spinal portion of the spinal accessory nerve, controlling head movements. These connections explain disturbances of eye direction (skew deviation) and tonic neck reflexes ("cerebellar" attitude of head) met with clinically in cerebellar lesions. (3) *Cerebello-Cerebral;*

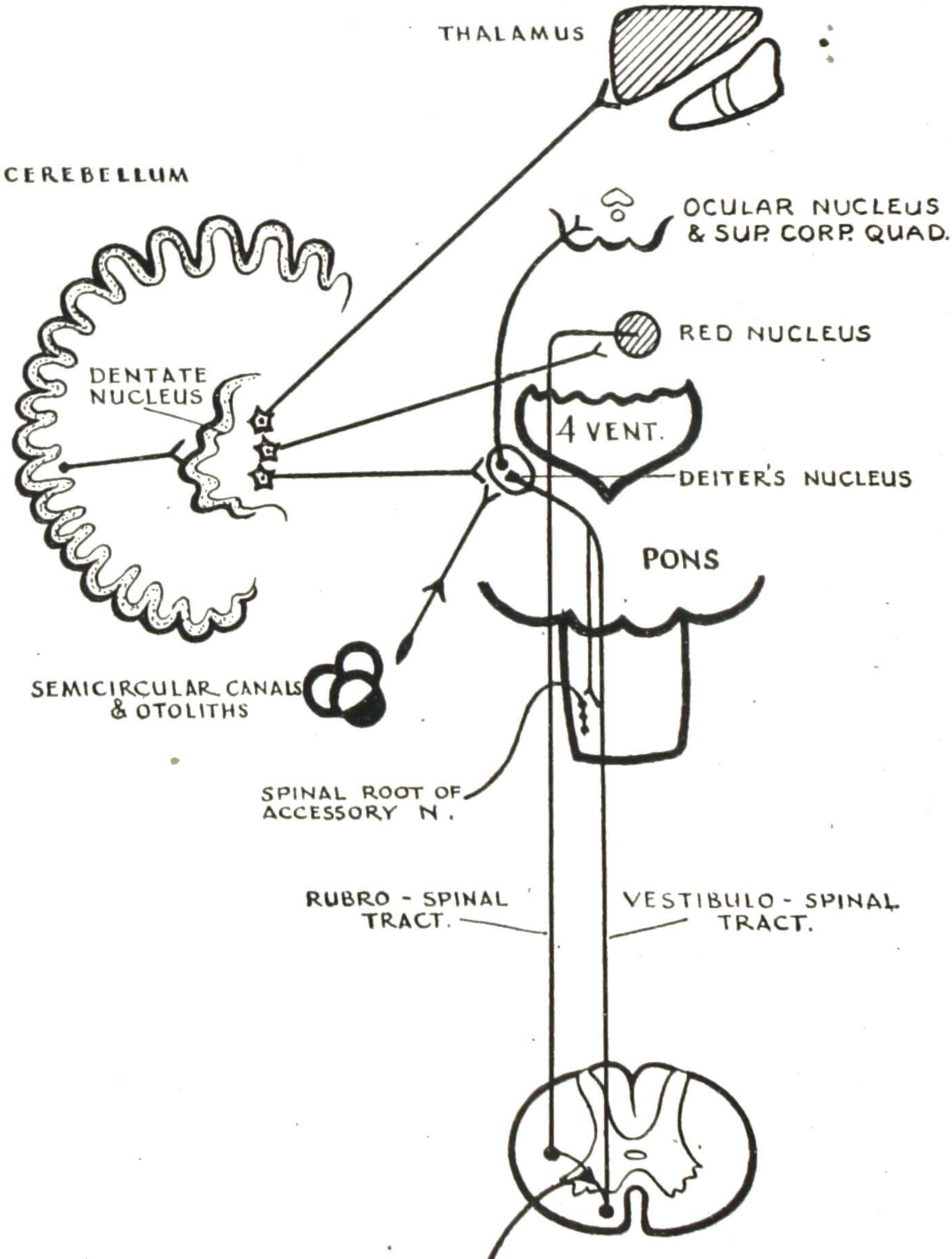


FIG. 165.—DIAGRAM OF THE CEREBELLAR AND VESTIBULAR EFFERENTS, showing the origin of these tracts and their termination in the ventral horn cells.

to connect with the opposite thalamus and the post-central and frontal cerebral cortex (Fig. 165).

The cerebellar afferents are concerned with the integration of muscle and joint and vestibular impulses. It is difficult to separate vestibular from cerebellar mechanisms as the two are intimately related.

In man, *irritative* lesions of the lateral lobes produce a vestibulo-ocular group of phenomena with notable hypotonia in the ipsilateral voluntary muscles, nystagmus, "cerebellar" attitude of the head and inco-ordination of voluntary movement. There is deviation from the straight line when the patient attempts to walk, the sound side, as it were, pushing him over to the side of the lesion. The patient pass-points to the side of the lesion. (See § 812.)

In *destructive* lesions of the cerebellum in man (*e.g.*, cerebellar atrophy) there is often absence of ocular phenomena and the only symptom may be a loss of the power of regulating pyramidal impulses (cerebellar ataxia). In patients who lost the whole of the cerebellum through war wounds, after a time there was only a general clumsiness of movement, intention tremor and slight slurring of speech. The inference is that if the cerebellum is destroyed the cerebral cortex can make itself independent of normal cerebellar adjustments.

Skew deviation of the eyes, a rare symptom, seems to depend on irritative lesions of the deep (dentate) nuclei of the cerebellum.

§ **682. Muscular Tonus.**—The tonus or continuously braced-up condition of voluntary muscle is due to a series of proprioceptive reflexes, muscular or vestibular. The proprioceptive reflexes arise in the tendons of the flexor muscles of the limbs, *e.g.*, in the legs, as the result of pressure of the sole of the foot on the ground, and in the labyrinths, as the result of alterations in the position of the head (tonic neck reflexes). The long afferents of the proprioceptive impulses, arising in tendons, pass up the spinal cord in the antero-lateral columns (not the posterior columns) to the mid-brain, where the reflex centres are probably Deiter's nucleus and the Red Nucleus. The *Mid-brain* may be thought of as the *Brain of Tonus.*

The main efferent tract of the proprioceptive reflex-arc is the vestibulo-spinal tract to the anterior horn cells. Any influence which cuts off pyramidal impulses below the mid-brain level (*e.g.*, spinal cord compression) allows of the release of this reflex system with resultant increased tonus below the level of the lesion—*extensor rigidity.* When the long mid-brain tracts themselves are disturbed, as in the late stages of compression paraplegia, simple segmental spinal reflexes are released subserving deep reflexes and reflex muscular movements and *flexor rigidity* is produced.

The *corpus striatum* also contributes to postural tone, but the explanation of the muscular rigidity of Parkinsonism and the hypotonia of chorea and athetosis is still obscure.

The cerebellum, through its connection with the thalamus, red nucleus and Deiter's nucleus, has an important influence on the maintenance of tonus.

§ **683. Anatomy of the Brain-Stem.**—The Brain-Stem comprises the Mid-Brain, Pons and Medulla. Here are grouped:

(1) The Sensory and Motor Pathways.

(2) The Cranial Nerve Nuclei.

(3) Important Reflex and Association Nuclei, *e.g.*, superior corpora quadrigemina, red nuclei, substantia nigra, Deiter's nucleus.

(4) Autonomic cells lying under the superior corpus quadrigeminum, connected by the *posterior longitudinal bundle* with the cervical sympathetic outflow from the antero-lateral columns of the spinal cord in the Th1 and 2 segments.

Any or all of these mechanisms may be involved in Brain-Stem lesions. Characteristically, one finds, as the result of a focal lesion, an ipsilateral cranial nerve palsy, with crossed hemiplegia or crossed hemianæsthesia, as described in § 670. For example, in lesions of the *mid-brain* there may be an oculo-motor palsy on the side of the lesion, with a contralateral hemiplegia from pyramidal involvement, signs of paralysis or irritation of the cervical sympathetic may be present (constricted pupil with enophthalmos, or the reverse of this), or, if the red nucleus and superior cerebellar peduncle are involved, contralateral involuntary movements and ataxia. Lesions of the dorsal part of the mid-brain will produce defective conjugate upward movement of the eyes. Lesions in the *pons* produce sixth or seventh nerve palsies, associated with pyramidal, sensory and cerebellar or vestibular signs.

Lesions in the *medulla* produce ninth, tenth, eleventh or twelfth nerve palsies in association with pyramidal and sensory signs, or are rapidly fatal.

(1) Motor and Sensory Pathways.—These have already been considered.

(2) Cranial Nerve Nuclei.—The student is advised to study the diagram (Fig. 166) which indicates the position of the various cranial nerve nuclei in the pons and medulla. The ocular nuclei have already been considered. The trigeminal nuclei are two, a motor and a sensory, and they are of great clinical importance, both from their extent and position. The ventral *motor nucleus* lies in the floor of the fourth ventricle and is concerned with the innervation of the muscles of mastication. The dorsal *sensory nucleus* is of wide extent, reaching from the third cervical segment of the spinal cord, extending through the tip of the posterior horns laterally in the

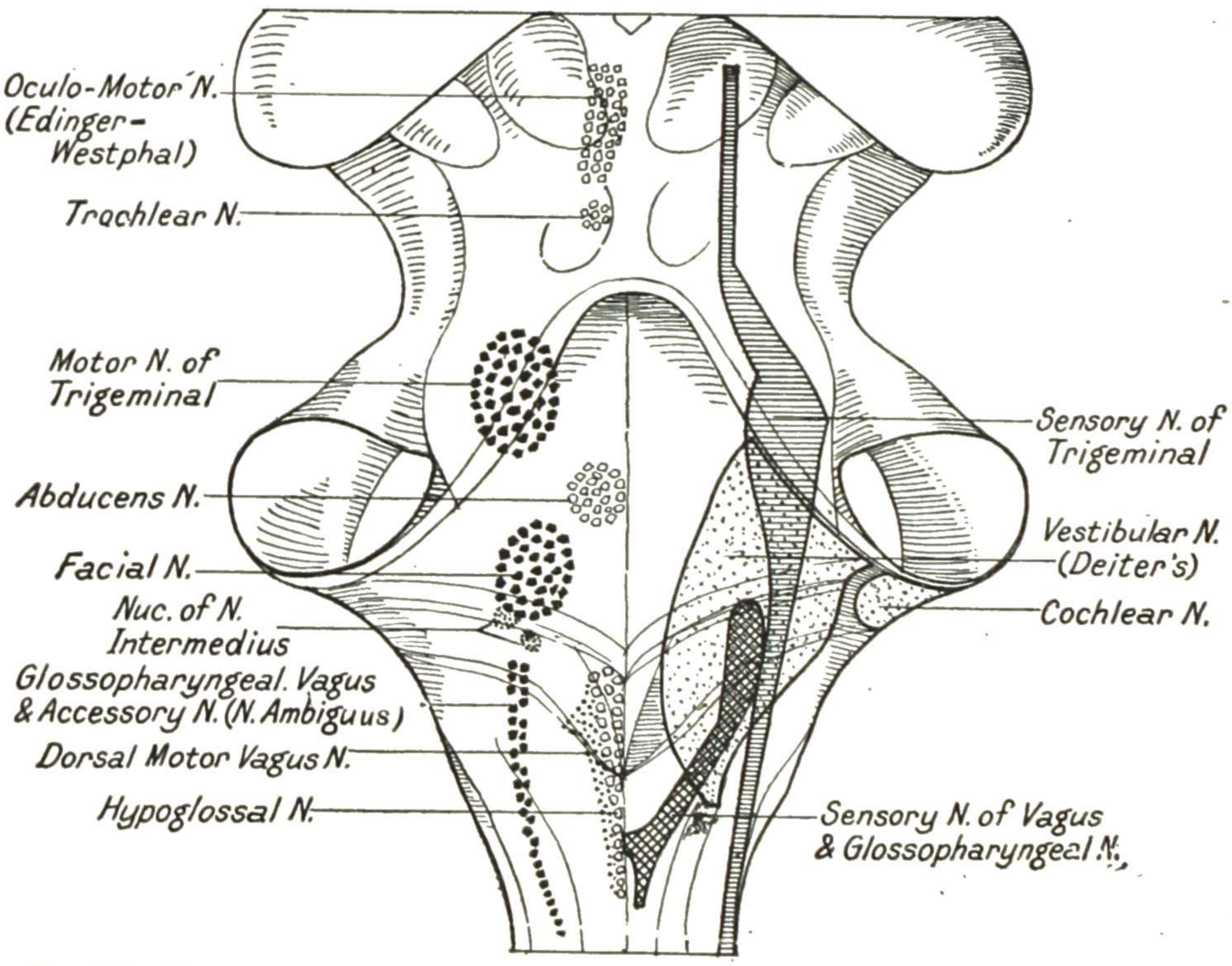


Fig. 166.—Diagram of the Cranial Nerve Nuclei (Motor Nuclei on Left, Sensory on Right). (Modified from Herrick.)

medulla, higher than the superior tip of the motor nucleus in the pons. In the pons it lies lateral to the fillet. This nucleus is peculiar in that its lower extremity is connected with sensation in the upper face and cornea, while its upper extremity is concerned with sensation over the distribution of the mandibular (third) division of the fifth nerve. The sensory nucleus is concerned with common sensation in the face, scalp, cornea and conjunctiva, tongue and nasal mucous membrane. Both roots emerge from the ventro-lateral aspect of the pons, and on the sensory root is the Gasserian ganglion.

The movements of the face and tongue may be mentioned here. The *facial nucleus* lies in the lateral aspect of the pons, its nerve-fibres turning round the abducens nucleus under the floor of the fourth ventricle before emerging to supply the facial muscles. The *hypoglossal nucleus* is purely motor and concerned with the movements of the tongue. It lies lowest of all the cranial nuclei in the floor of the fourth ventricle near the mid-line. All the cranial nerve nuclei receive supranuclear pyramidal fibres from the motor cortex of both hemispheres *except* the part of the facial nucleus supply-

ing the lower facial muscles, and the hypoglossal nucleus, which receive fibres from one hemisphere only, the contralateral. In hemiplegia, due to a focal lesion in the internal capsule, all the cranial nerve nuclei escape paralysis owing to their bilateral innervation, except these two nuclei. We thus observe, in hemiplegia, a weakness of the lower face (the upper face is spared) and deviation of the tongue to the hemiplegic side when it is protruded.

The sensory *cochlear nucleus* is situated on the dorsal and outer aspect of the pons. The motor *glossopharyngeal-vagus-accessorius* nucleus (n. ambiguus) lies in the medulla and is concerned with the innervation of the muscles of the pharynx, larynx, palate, sterno-mastoid and trapezius. The motor nucleus of the spinal accessory nerve reaches as low as the third cervical segment. The *dorsal motor nucleus of the vagus* supplies the unstriped muscle of the alimentary tract and air passages. There is also a *sensory glossopharyngeal-vagus* nucleus in the medulla, concerned with common sensation from the ear, mouth, pharynx and afferents from the abdominal and thoracic viscera.

(3) Reflex and Association Nuclei of the Brain-Stem.

Superior Corpus Quadrigeminum.—This reflex centre in the mid-brain receives afferents from the optic tracts and from the visual cortex, and through its efferents the oculo-motor nerves and the tecto-spinal tracts, effects pupillary alterations, ocular movements, protective blinking and turning away from sudden visual stimuli. Lesions produce loss of upward movement of the eyes, with disorder of pupillary reactions.

Red Nucleus.—This nucleus and the substantia nigra, both in the mid-brain, subserve reflex tonus in the voluntary muscles. It is a head ganglion in a series of proprioceptive reflexes whose afferents come from (1) the cerebellum, controlling through the rubro-spinal tract the co-ordination of the limbs, and (2) from the globus pallidus, governing the performance of automatic association movements, *e.g.*, swinging the arms when walking. Lesions of the red nucleus may produce involuntary movements and ataxia. In animals, section of the mid-brain through the red nucleus, produces the phenomena of "decerebrate rigidity," due to the destruction of pyramidal control and the releasing of the extra-pyramidal mid-brain centres for reflex tonus in the voluntary muscles (see § 764).

Deiter's Nucleus. This nucleus, lying ventrally in the pons, receives fibres from Scarpa's ganglion (vestibular nerve), proprioceptive afferents from the antero-lateral columns of the cord and from the cerebellum, and transmits, through the vestibulo-spinal tract to the anterior horn cells, impulses regulating contractile tonus and equilibrium. Lesions of this nucleus produce hypotonia and loss of balance. From this nucleus also the posterior longitudinal bundle passes upwards to the ocular nuclei regulating the tonus of the external ocular muscles.

(4) Autonomic Cells and Fibres.—Disturbance of the sympathetic innervation of the eye, with irritative signs (midriasis, exophthalmos or lid-retraction) or paralytic signs (miosis, enophthalmos and pseudo-ptosis) follow upon lesions of the posterior longitudinal bundle in the mid-brain, pons, or medulla.

§ 684. The Vascular Supply of the Brain and Spinal Cord.—The Vertebral Arteries, before entering the foramen magnum, give off the anterior spinal and two posterior spinal arteries. These run downwards the entire length of the cord, forming, with their anastomoses, a vascular chain reinforced by branches of the intercostal and lumbar arteries, entering the vertebral column through the intervertebral foramina, and running along the anterior and posterior nerve roots. On entering the foramen magnum, the vertebral arteries give off the *posterior cerebellar arteries* and unite at the lower border of the pons to form the *basilar artery* (Fig. 167).

The *posterior inferior cerebellar* artery supplies the lateral aspect of the medulla, including the superior cerebellar peduncle, the lower part of the long sensory nucleus of the trigeminal, the fillet, the decussating arcuate fibres of the fillet, and the glosso-pharyngeal-vagus nucleus. Lesions of this artery produce "Cerebellar Apoplexy" with acute vertigo and hemiataxy, paralysis of the pharynx, soft palate and vocal

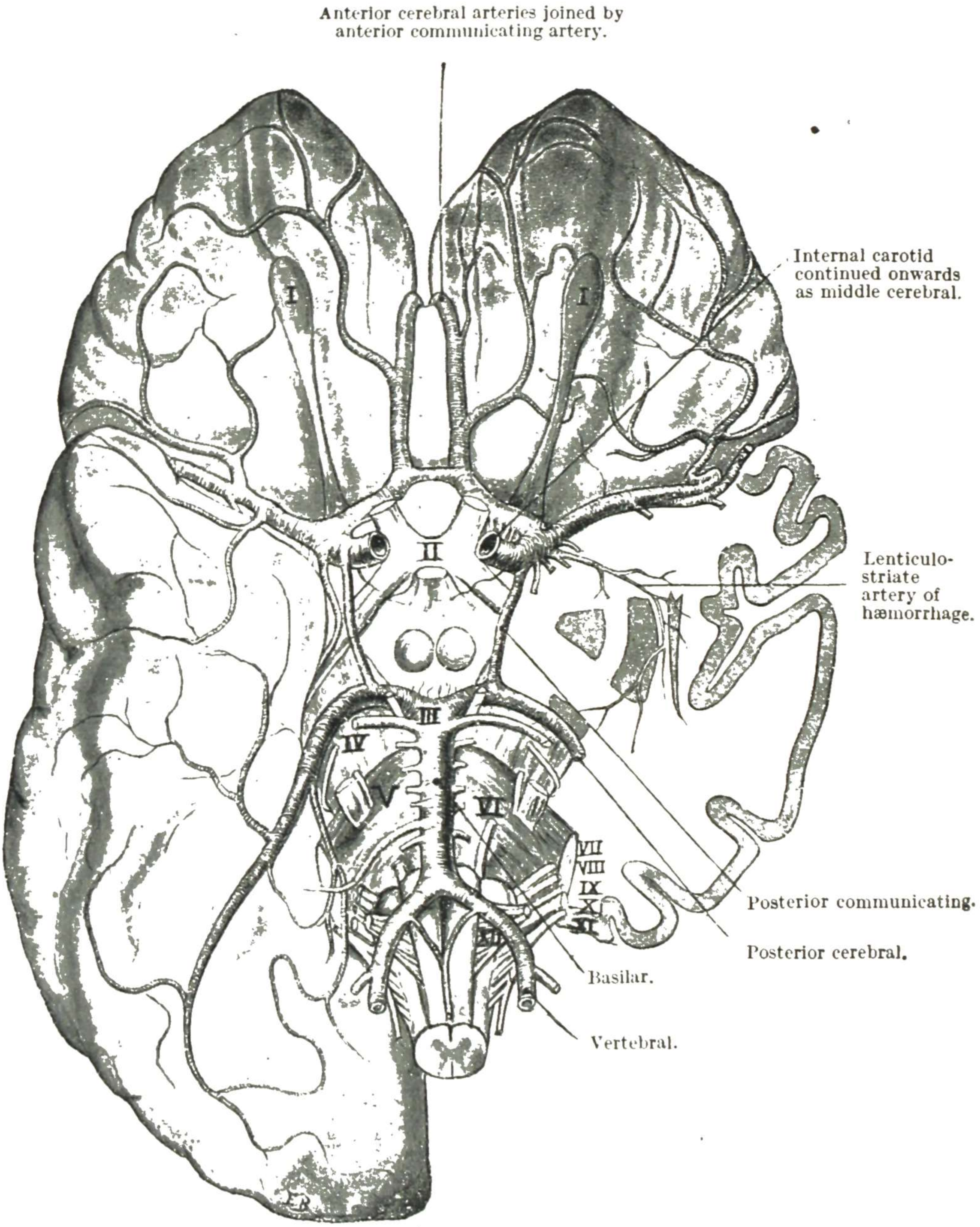


FIG. 167.—THE BASE OF THE BRAIN, showing the arterial distribution and the cranial nerves.—In the oblique section of the left hemisphere are seen from without inwards—grey matter of the island of Reil; claustrum (grey); external capsule (white); lenticular nucleus (grey); internal capsule (white) with artery of hæmorrhage; and caudate nucleus (grey). I., Olfactory lobe; II., optic chiasma; III., bifurcation of basilar artery between the third nerves; IV. (on right crus cerebri), beside fourth nerve; V. (on pons Varolii), beside fifth nerve; VI., sixth nerve (abducens); VII., facial nerve; VIII., auditory nerve; IX., glossopharyngeal nerve; X., vagus or pneumogastric; XI., spinal accessory; XII., hypoglossal nerve.

cord, with sympathetic oculo-pupillary signs, all on the side of the lesion, with a crossed dissociated hemianæsthesia of the body and sensory loss on the ipsilateral side of the face (*unilateral bulbar syndrome*).

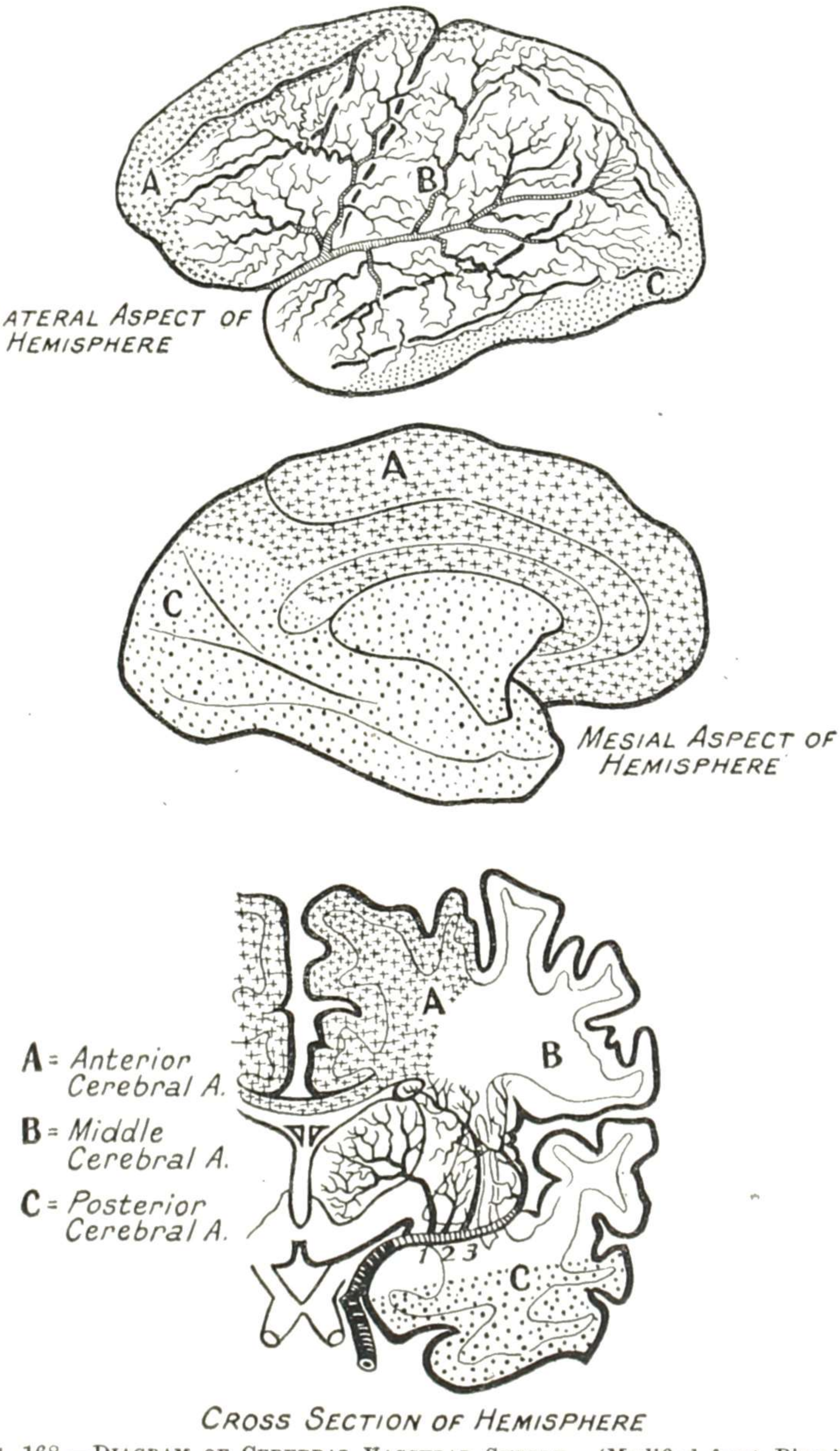


FIG. 168.—DIAGRAM OF CEREBRAL VASCULAR SUPPLY. (Modified from Bing.)

In the lowest figure:
1. Lenticulo-Optic Branch of Middle Cerebral.
2 and 3. Lenticulo-Striate Branches of Middle Cerebral.

The *basilar artery* supplies branches to the pons, and two large branches, the *superior cerebellar arteries* to the upper cerebellum, and divides at the level of the crura of the mid-brain to form the two *posterior cerebral arteries*.

The *internal carotid arteries* enter the skull just laterally to the posterior clinoid processes of the pituitary fossa and give off the ophthalmic arteries supplying the

globe and orbit. Obstruction of one internal carotid will produce a "*Carotid Hemiplegia*"—blindness, with optic atrophy on the side of the lesion, with a transient contralateral hemiplegia, the circulation through the middle cerebral being rapidly established through the Circle of Willis. The internal carotid arteries then divide into *middle* and *anterior cerebral arteries*, supplying, roughly, the anterior two-thirds of the cerebral hemispheres (Fig. 168).

The anterior, middle, and posterior cerebral arteries are connected to form the *Circle of Willis* in the subarachnoid space at the base of the brain. This circle is occasionally incompletely developed (Fig. 167).

The *anterior cerebral artery* passes anteriorly into the great median fissure, and, curving round the genu of the corpus callosum, runs backwards upon the superior aspect of this structure, supplying its anterior seven-eighths and giving off branches to supply the mesial surfaces of the hemispheres as far back as the parieto-occipital fissure, and supplying, in this territory, the cortical areas for the foot and leg at the top of the pre-central gyrus. The anterior cerebral artery sends perforating branches inwards towards the caudate nucleus. Obstruction of an anterior cerebral artery gives rise to a crural monoplegia or hemiplegia, with sensory impairment, most marked in the lower limb, with apraxia of the left upper limb from involvement of the corpus callosum, § 745 (Fig. 168).

The *middle cerebral artery* supplies most of the convexity of the cerebral hemisphere, including the motor, sensory, and speech areas of the cortex. Leaving the Circle of Willis, it gives off several small but important perforating arteries, which pierce the anterior perforated spot to supply the corpus striatum, optic thalamus, and region of the internal capsule. These are terminal arteries, without anastomoses, and they are termed the *lenticulo-striate* and *lenticulo-optic* arteries (Fig. 168). The middle cerebral artery then runs along the Sylvian fissure to supply the whole of the convexity of the cortex, with the exception of the area supplied by the anterior cerebral artery, and at the occipital pole of the hemisphere anastomoses over the cortical area for central vision with the posterior cerebral artery. The middle cerebral artery also supplies, by penetrating branches, the major part of the centrum ovale, including the temporal knee of the optic radiations and external capsule.

The *posterior cerebral artery* winds round the crus, supplying the mid-brain nuclei (corpora quadrigemina, red nucleus, etc.), and supplies the inferior mesial surfaces of the temporal and occipital lobes, the uncus, and posterior seventh of the corpus callosum, anastomosing, with the middle cerebral artery, over the convexity of the occipital pole (area for central vision). It gives off an important branch, the *calcarine artery*, to supply the cuneus and lingual gyri above and below the calcarine fissure, respectively. In obstructive lesions of the posterior cerebral artery, whilst peripheral vision is destroyed from destruction of the calcarine cortex, the anastomosis with the middle cerebral artery over the occipital pole ensures a blood supply to the area of central vision which, therefore, escapes.

The *choroid plexuses* of the lateral ventricles, which have to do with the formation of spinal fluid, are supplied by the choroid branches of the internal carotid and middle cerebral. The choroid veins drain backwards to join the great vein of Galen, draining into the anterior extremity of the straight sinus in the tentorium cerebelli.

§ 685. The Cranial Venous Sinuses.—The walls of the venous sinuses consist of dura mater. They drain blood from the brain and meninges, and cerebro-spinal fluid passes into these sinuses through the Pacchionian bodies. They empty into the internal jugular vein and communicate with the veins of the head and neck through the orbital vein and various emissary veins, the most important of which is the large mastoid emissary vein. The chief sinuses are the Superior Longitudinal, the Lateral, the Straight and the Cavernous. The basal sinuses, *e.g.*, Lateral and Cavernous, communicate freely, but the *Superior Longitudinal Sinus*, which drains the cortical veins, opens only into the Torcular Herophili, and obstruction of this sinus leads to widespread bilateral cortical necrosis, characterised, clinically, by a paraplegia from destruction of the cortical motor leg areas.

The *Straight Sinus* runs medially between the two halves of the tentorium cerebelli, draining the choroidal veins and the great vein of Galen.

The *Lateral Sinus* drains the veins of the posterior fossa and runs from the external occipital protuberance, in an arched fashion, forwards and downwards, to open into the internal jugular vein through the jugular foramen. It occupies a groove in the mastoid part of the temporal bone and communicates with the mastoid emissary vein. Obstruction causes œdema over the mastoid process.

The reticulated *Cavernous Sinuses* lie on either side of the sphenoidal air cells, draining the veins at the base of the brain and the orbital veins. They communicate with one another by means of the circular sinus, and communicate posteriorly through the superior and inferior petrosal sinuses with the lateral sinuses. Thrombosis of the lateral sinuses may thus spread into first one cavernous sinus and then the other. On the medial wall of the cavernous sinus lies the internal carotid artery, with the abducens nerve. The oculo-motor, trochlear, and ophthalmic divisions of the trigeminal nerve pass forwards, on the lateral wall of the sinus, to enter the orbit through the sphenoidal fissure. These structures are separated from the blood in the sinus only by its lining membrane (Fig 169). Occlusion or compression of this sinus causes proptosis (protrusion of the eyeball) with orbital œdema, ocular palsies, and pain and sensory loss over the first and second divisions of the trigeminal nerve.

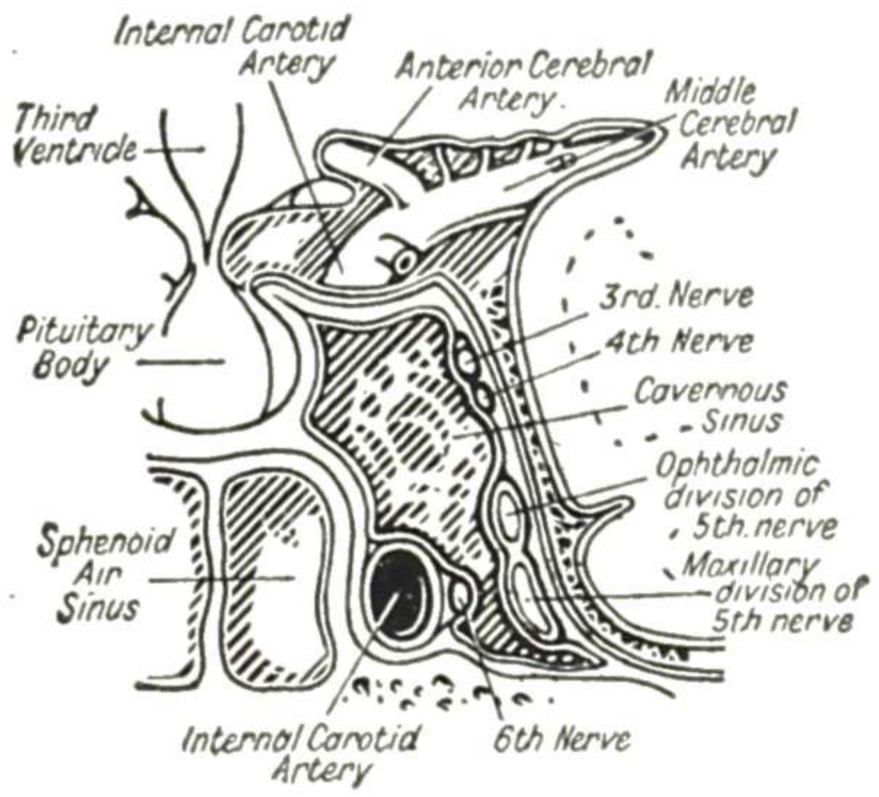


FIG. 169.—DIAGRAM OF THE CAVERNOUS SINUS.

§ 686. The Cerebral Ventricles.—In each cerebral hemisphere lies a *lateral ventricle* having three horns meeting in the parietal region. The anterior horn is deeply indented on its lateral surface by the head of the caudate nucleus. The temporal or descending horn is smaller and extends into the temporal lobe. The occipital or posterior horn is very inconstant in size and length. The anterior horns in their posterior two-thirds are separated only by the thin septum lucidum. The occipital horns are somewhat widely separated, while the temporal horns diverge more markedly from one another. The lateral ventricles communicate through small apertures called *interventricular foramina* (of Monro), with a single median cavity, the *third ventricle*, which lies between the optic thalami. The third ventricle communicates anteriorly with the infundibulum or pituitary stalk, and posteriorly by the long narrow iter (aqueduct of Sylvius) through the mid-brain with the *fourth ventricle* which lies between the bulb and cerebellum. The fourth ventricle opens into the subarachnoid space by three foramina, one in the median dorsal line (foramen of Magendie) and two laterally near the flocculi of the cerebellum (foramina of Luschka).

§ 687. The Cerebro-Spinal Fluid.—The central nervous system is enclosed in the bony case of the skull and vertebral column, and is suspended in a water-cushion of cerebro-spinal fluid. This cerebro-spinal fluid is formed by the epithelial-covered choroid plexuses of the lateral, third and fourth ventricles, by a process of dialysis. The total amount of fluid normally present within the cranio-spinal dura mater is 120–150 cubic centimetres. From the lateral ventricles the fluid passes through the foramina of Monro into the third ventricle and thence by the aqueduct of Sylvius to the fourth. It leaves the ventricular system by the medial foramen of Magendie in the roof of the fourth ventricle and the bilateral foramina of Luschka, one in each lateral recess of the fourth ventricle.

Within the subarachnoid space, the cerebro-spinal fluid bathes the whole surface of the brain and spinal cord. The fluid circulates forward through the basal cisterns, passes through the opening between the tentorium and the brain-stem and upwards over the surface of the hemispheres, to be absorbed directly into the cranial venous

sinuses by way of the arachnoid villi. These villi are invaginations of the subarachnoid space through the fibrous dural wall of the sinuses and project into their lumen. It is possible that some absorption may take place through perivascular spaces, into the cerebral capillaries, but this is of subordinate importance.

The subarachnoid space, in which the cerebro-spinal fluid circulates, is traversed by numerous delicate trabeculæ stretching from the arachnoid on the outer side to the pia on the inner. Within this space lie the cerebral and spinal blood vessels, and it is crossed by the cranial and spinal nerves. All these structures, like the walls of the subarachnoid space, are covered by flattened mesothelial cells.

Certain deep expansions of the subarachnoid space, called cisterns, exist at the base of the brain. Of these the most important are (1) the *cisterna magna*, situated between the inferior vermis and the medulla, and extending outwards on each side beneath the cerebellar hemispheres; and (2) the *cisterna basalis*, in the neighbourhood of the interpeduncular space, in which lie the Circle of Willis and the third nerves. The subarachnoid space sends important funnel-shaped prolongations along the spinal nerves as far as their foramina of exit from the dura mater, and, within the cranium, there are important prolongations (*a*) along the optic nerve as far as the exit of the nerve from the globe and (*b*) along the trigeminal nerve; so that the Gasserian ganglion is enclosed in a tiny pool of cerebro-spinal fluid, the cave of Meckel. The perilymph of the internal ear communicates through the internal auditory meatus with the cerebro-spinal fluid in the subarachnoid space.

The subarachnoid space dips into the sulci and is continued as sleeve-like channels surrounding the pial vessels into the brain substance. These perivascular spaces were first described by Virchow and Robin, and they are called *Virchow-Robin Spaces*. They subdivide with the blood vessels, and eventually communicate within the cerebral substance with pericellular spaces about the nerve-cells. The Virchow-Robin Spaces normally empty into the subarachnoid space, and, in inflammatory conditions affecting the central nervous system, they are packed with cells. Seen on a cross-section, they present the so-called "perivascular cuffing" appearance.

It is possible that the spinal fluid, when secreted, contains neither cells nor protein, these being added to it in the course of its circulation, from the perivascular spaces. It is possible also that the lymph from the peripheral nerves may pass into the subarachnoid space by way of the nerve-roots.

Normal cerebro-spinal fluid is clear and colourless. Whereas the ventricular fluid is almost non-albuminous and cell-free, the fluid obtained normally from the dependent spinal theca by lumbar puncture contains not more than 4 lymphocytes per cubic millimetre, 0·025 to 0·03 per cent. protein, 0·05 to 0·08 per cent. glucose, and 0·725 per cent. chlorides. The chloride in the spinal fluid is normally higher than that in blood plasma (0·6 per cent.); glucose, calcium, cholesterol and uric acid, are present in lesser amounts in the cerebro-spinal fluid than in the blood plasma. Spinal fluid, obtained by lumbar puncture, is normally under a pressure of 60 to 150 millimetres of water measured with a manometer (§ 919).

§ 688. Surface Markings.—The *Rolandic Fissure* is identified by taking a point midway between the nasion (root of the nose) and the external occipital protuberance, and drawing a line from a point 1 cm. behind this, downwards and forwards, at an angle of 67½ degrees, *i.e.*, *three-quarters* of a right angle (easily obtained by folding the square edge of a card appropriately) with the mid-line.

The *Spinal Cord* terminates in the conus medullaris, which is opposite to the 1st lumbar spinous process, while the *spinal theca* reaches as low as the level of the 2nd sacral spinous process.

The Hypothalamus and Autonomic Nervous System

§ **689**. The **Pituitary** gland with its hollow infundibulum is intimately connected with the Hypothalamus. It is the most vascular gland in the body; it weighs 0·5 gram, and it lies in the bony sella turcica (Fig. 187A). The anterior lobe (pars

glandulosa) is made up of eosinophil and basophil secretory cells, and of their precursor chromophobe cells, which are non-secretory. The intermediate lobe consists of vesicles filled with colloid. The posterior lobe (pars nervosa) consists of nerve cells and neuroglia, and it produces a special internal secretion.

The *pituitary hormones* are exceedingly complex. Those formed by the eosinophil cells of the anterior lobe are probably concerned with bodily growth. The secretions

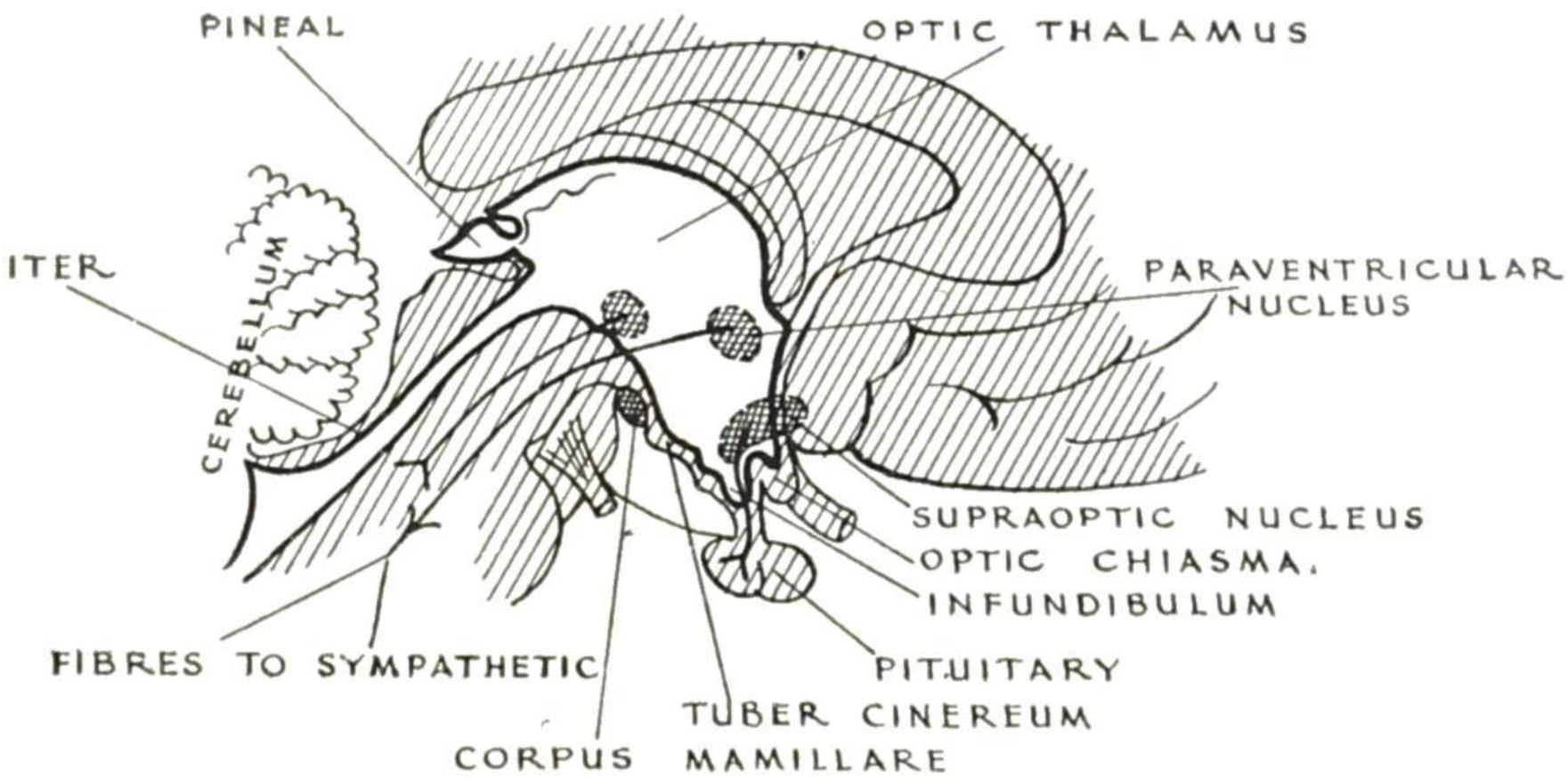


FIG. 170.—MEDIAN SURFACE OF THE HYPOTHALAMUS; shows the more important nuclei and the tracts proceeding from them.

of the basophil cells of the anterior lobe have to do with the development and activity of the gonads and mammæ. They are also concerned with the activity of the thyroid, and with carbohydrate metabolism. The posterior lobe secretion consists of two substances (*a*) vasopressin, with an " anti-diuretic " hormone regulating the constancy of the osmotic pressure of the plasma by causing water retention; and (*b*) oxytocin, which has a specific constricting effect on uterine muscle.

The **Hypothalamus** (Fig. 170) is that region of the brain forming the floor and lateral walls of the third ventricle. Various cell groups can be defined in the grey matter here, giving rise to three main groups of efferent fibres: (1) Fibres arising in the supra-optic group of cells going to the posterior lobe of the pituitary and pars intermedia. (2) A second group passing to the brain stem, and (3) Efferents from the posterior (paraventricular) group of cells controlling the autonomic nervous system.

Not only does the hypothalamus regulate pituitary activity but it is the head ganglion of the autonomic nervous system; sympathetic and parasympathetic components having their representation there. Hypothalamic stimulation causes autonomic and other effects. Regulation of sleep mechanism and control of body-temperature are functions of the hypothalamus. Clinically, lesions of the hypothalamus cause (1) Diabetes insipidus, and sometimes glycosuria; (2) Genital dystrophy and amenorrhœa; (3) Infantilism, obesity, and other disorders of growth; (4) Disorders of sleep rhythm.

The *pathological causes* of these syndromes may be: Cranio-pharyngeal cysts, third ventricle and basal neoplasms, fractures of the base of the skull, vascular lesions, syphilitic and tuberculous basal meningitis and encephalitis lethargica.

Through the posterior longitudinal bundle and the vestibulo-spinal tract the hypothalamus connects with the Edinger-Westphal (oculomotor) nuclei, the salivary nuclei and the dorsal nucleus of the vagus, in the brain-stem.

§ **690**. **The Autonomic Nervous System** consists of (i.) Sympathetic and (ii.) Parasympathetic Divisions, which are anatomically and physiologically separate.

The confusion of nomenclature of different writers is troublesome to the student and the following scheme may be found helpful.

Vegetative = AUTONOMIC = Involuntary

Sympathetic or Thoracolumbar (from Th1–12 and L1 and 2).	Parasympathetic or Craniosacral (from oculomotor, facial, glosso-pharyngeal, vagus and S2, 3 and 4).

The Autonomic Nervous System innervates bodily structures which are not under direct voluntary control. These include *smooth muscle* organs, the iris, and *tubular viscera* such as the bronchi, gastro-intestinal and genito-urinary tracts, the lachrymal, sweat and digestive *glands*, and the *heart* and *blood vessels*. Each of these structures has a dual innervation from the sympathetic and parasympathetic divisions, which are physiologically in a state of balanced opposition. When one division is excited, the other is inhibited. Sympathetic stimulation causes a diffuse reaction in several organs, but the result of parasympathetic stimulation is more specific and local.

I. The SYMPATHETIC Division consists of (*a*) *Ganglion cells* situated in the lateral

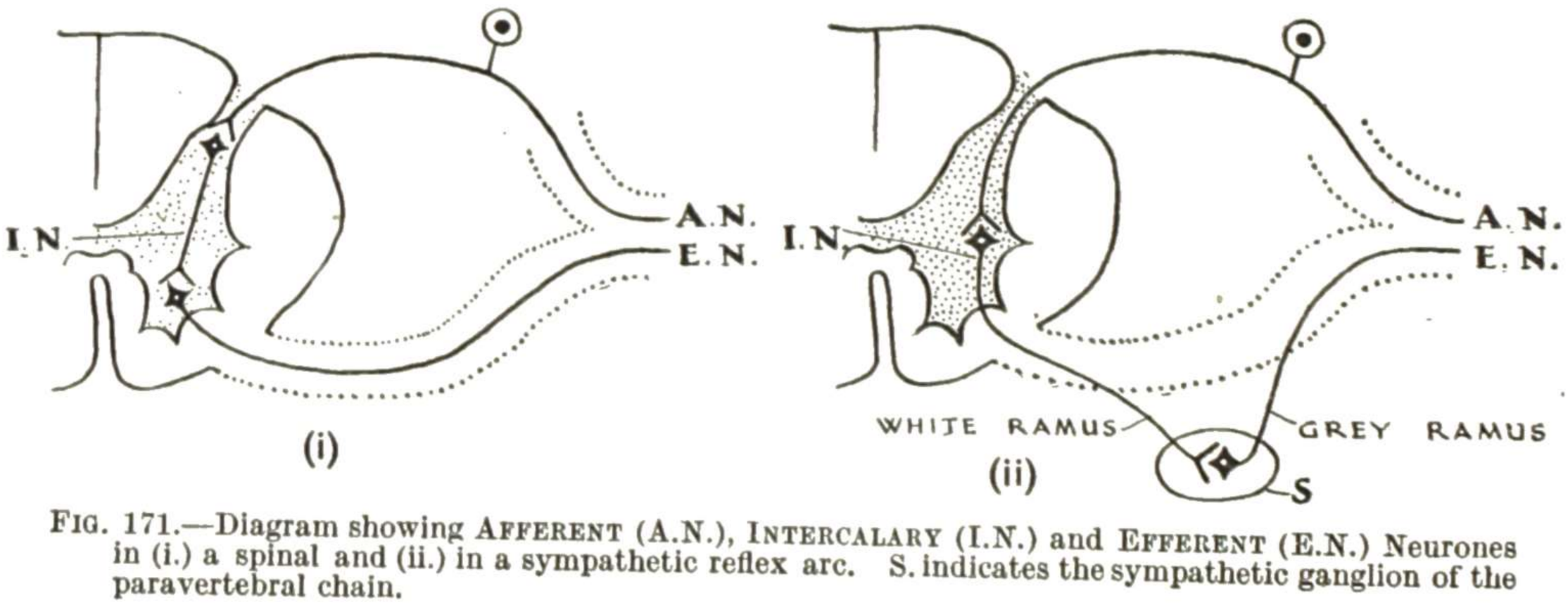


FIG. 171.—Diagram showing AFFERENT (A.N.), INTERCALARY (I.N.) and EFFERENT (E.N.) Neurones in (i.) a spinal and (ii.) in a sympathetic reflex arc. S. indicates the sympathetic ganglion of the paravertebral chain.

horn of the grey matter of the spinal cord extending from the Th1 to L2 level (lateral column); (*b*) The paravertebral *Sympathetic Chains* lying close to the vertebral column on the two sides, and (*c*) Peripheral *Sympathetic Nerves* (greater, middle and lesser splanchnic nerves) and *Collateral Plexuses* (cardiac, cœliac and hypogastric plexuses). The sympathetic chains and the peripheral plexuses are all that may be seen naked-eye by the casual dissector. In the thoracic region each ganglion of the chain is connected to its anterior spinal nerve root by two little *rami communicantes*.

Histologically, it has been shown that the sympathetic is made up of a number of reflex arcs with afferent, intercalary and efferent neurones, as in a spinal reflex arc (Fig. 171). The *afferent neurone* has its cell in the posterior root ganglion and enters the spinal cord to terminate in cells of the lateral column. From this column the *intercalary neurone* arises, and is projected out of the cord, within the anterior nerve root, which it leaves to enter the corresponding ganglion of the sympathetic chain. The *efferent neurone* arises in the ganglion and joins the anterior nerve root to be distributed with it peripherally. The sympathetic intercalary neurone is called the *white* (myelinated) ramus; the efferent neurone is called the *grey* (unmyelinated) ramus. The student will find it easy to remember that the grey ramus passes *away* from the ganglion. To the naked eye the grey and white rami are indistinguishable: the difference is histological.

The *sympathetic chains* consist of 24 ganglia: superior, middle and inferior cervical (stellate) ganglia, 12 ganglia in the thoracic, 4 in the lumbar and 5 in the sacral region. The chains fuse below in front of the coccyx (ganglion impar) and above they break

up over the internal carotid arteries. The outflow of intercalary neurones (white rami) from the spinal cord is between the Th1 and L2 segments only. These intercalary neurones, when they enter the sympathetic chain ganglia, behave in one of three ways (Fig. 172) :—(1) They may terminate here and anastomose with an efferent neurone (*e.g.*, fibres to sweat glands or blood vessels). (2) They may pass upwards or downwards in the sympathetic chain to terminate in a ganglion above or below (*e.g.*, fibres from Th1 and 2 passing to the cervical ganglia, and from L1 and 2 passing to the lumbar and sacral ganglia). (3) They may branch in the ganglion of the chain and

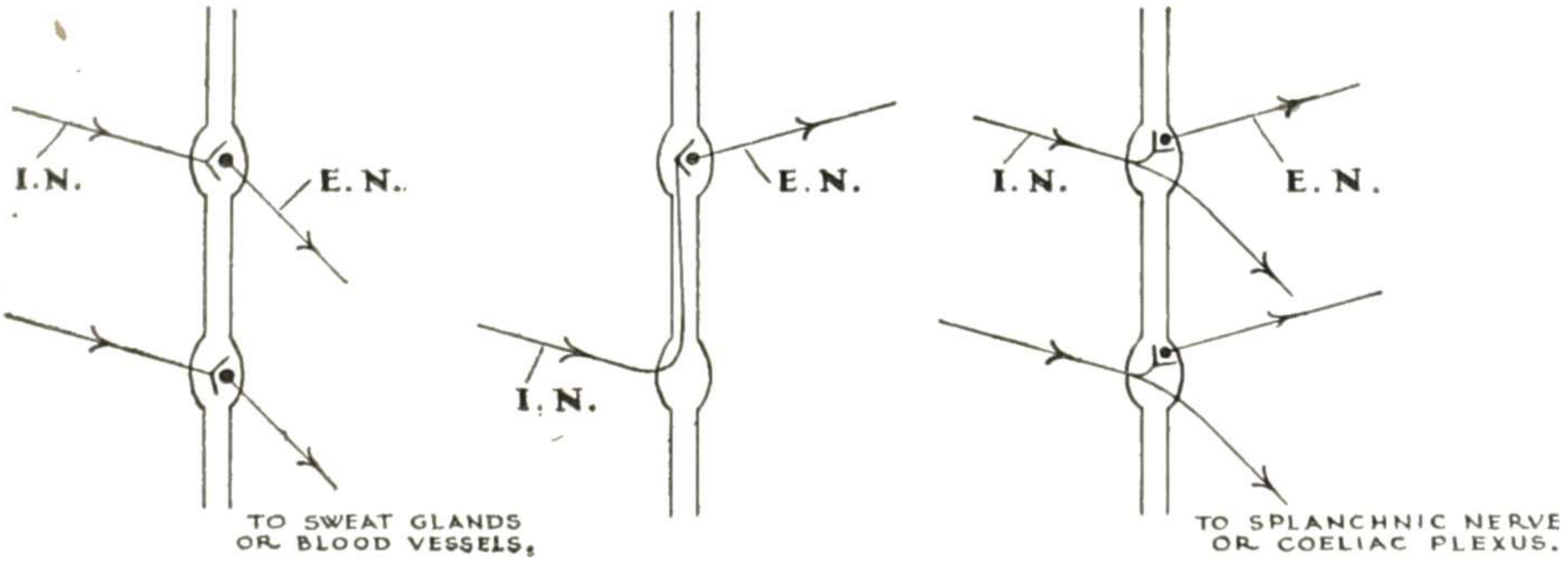


FIG. 172.—Diagram to show the three possible arrangements of intercalary neurones on entering the paravertebral sympathetic chain.

pass through it to form peripheral sympathetic nerves (*e.g.*, splanchnic) and plexuses (*e.g.*, cœliac).

It is characteristic of the sympathetic that there is always a ganglion between the cord and the viscus, blood vessel, or gland supplied by the efferent neurone. The intercalary neurone entering the ganglion is sometimes called the *pre-ganglionic* fibre, the efferent neurone the *post-ganglionic* fibre.

The cells of the lateral column are brought into relationship with the *hypothalamus* through the posterior longitudinal bundle and the vestibulo-spinal tract.

The *cervical sympathetic* fibres pass from the cord at the Th1 and 2 level and ascend in the cervical sympathetic chain to the superior cervical ganglion. Here efferent neurones arise and ascend in the carotid plexus to the ophthalmic division of the fifth nerve and run *via* the naso-ciliary nerves to the eyeball. The cervical sympathetic is the tonic dilator of the pupil. The middle cervical ganglion gives fibres to the thyroid gland and the stellate and upper thoracic ganglia furnish accelerator fibres to the heart and vaso-constrictor fibres to the upper limbs.

Destruction of the cervical sympathetic causes *Horner's syndrome*—a small pupil, narrowing of the ocular fissure from paralysis of the unstriped muscle of the levator palpebræ superioris, and apparent but not real enophthalmos. Irritative lesions of the cervical symphathetic cause opposite effects. Excision of the *stellate ganglion* will cause in addition vaso-dilatation in the upper limbs and the corresponding side of the face, with absence of sweating and gooseflesh and insignificant slowing of the heart.

Sympathetic stimulation raises blood pressure, inhibits alimentary peristalsis, causes erection of the hairs and sweating, and excites secretion of adrenalin, raising the blood sugar.

II. The PARASYMPATHETIC Division has two main outflows from the central system : (*a*) The Cranial Outflow and (*b*) The Sacral Outflow.

(*a*) The Cranial Outflow occurs through the oculomotor, facial, glossopharyngeal and vagus nerves, the ganglion cells being situated in the mid-brain in the oculomotor (Edinger-Westphal) nucleus, and in the medulla in the inferior and superior salivary nuclei and the dorsal nucleus of the vagus. Every bodily structure innervated by the sympathetic has also a parasympathetic innervation. It is through the vast ramifications of the vagus nerve that the parasympathetic is brought into communication with most of the viscera.

(*b*) The Sacral Outflow leaves the spinal cord with S2, 3 and 4 nerves in the cauda equina. They leave the spinal nerves, do not pass through the sacral sympathetic chains, but forming the nervi erigentes, run directly into the hypogastric ganglia and thence to the walls of the pelvic viscera. As with the sympathetic, there is always a ganglion between the cord and the viscus supplied. In the case of the parasympathetic, however, the ganglia lie in the walls of the viscera, so that the preganglionic fibres are long and the postganglionic fibres very short.

Stimulation of the parasympathetic causes effects antagonistic to those caused by sympathetic stimulation. The pupil is constricted, the heart retarded, the bronchioles constricted and peristalsis promoted; secretion of insulin and lowering of blood sugar occurs.

Pharmacologically, various drugs and hormones act on the autonomic nervous system. Depending on the dosage, sometimes a sympathetic, sometimes a parasympathetic effect is obtained with any one substance, but in the main the following table is true:—

	Sympathetic	*Parasympathetic*
Stimulating . . .	Adrenalin, Ephedrine	Physostigmine, Acetylcholine
Depressing	Nicotine	Atropine, Nicotine

Sir Henry Dale and his co-workers have recently established that a portion of the cerebrospinal system and the whole of the autonomic nervous system achieve their effect through chemical mediators formed at the nerve endings. These substances, normally found in the body, are acetyl-choline and adrenalin (sympathin). Thus there are two kinds of nerves, the *cholinergic* and *adrenergic*. In the case of the cerebrospinal system the motor fibres to striate muscles are cholinergic. In the case of the autonomic nervous system the parasympathetic fibres, both pre- and post-ganglionic, are cholinergic. The sympathetic fibres to the sweat glands are cholinergic but most of the sympathetic post-ganglionic fibres are adrenergic. Orbelli, using an excised skeletal muscle preparation, has shown that sympathetic stimulation diminishes fatigue, the maximum effect being reached some time after sympathetic stimulation has ceased.

Innervation of the Bladder. (1) The parasympathetic, by stimulating the detrusor muscle and relaxing the sphincter, empties the bladder of urine. Tonic activity of the sympathetic causes the opposite effect—retention of urine. The integrity of the parasympathetic reflex arc is essential for normal emptying of the bladder. The stimulus of bladder distension sets up impulses which pass in parasympathetic afferents to the conus and sacral region of the cord. The intercalary neurones emerge from the cord with the sacral outflow (S2 and 3) as the *nervi erigentes*, which anastomose with the efferent fibres in a plexus on the bladder walls (Fig. 173). Disturbances of this reflex arc, such as occur in tabes dorsalis (degeneration of the afferent fibres), or in lesions of the conus, result in retention from unopposed sympathetic activity.

Micturition can be initiated voluntarily in response to a cerebral stimulus, capable of activating the parasympathetic reflex arc. The cord pathways of this mechanism are not known. The Compressor urethræ and Bulbocavernosus muscles are under voluntary control and are innervated by the spinal pudendal nerve (S1 and 2). Incomplete spinal cord lesions cause precipitancy or retention. Complete transverse cord lesions cause reflex incontinence.

(2) The sympathetic fibres to the bladder leave the cord at the L1 and 2 segments and pass to the superior hypogastric plexus (presacral nerve, Fig. 173, G. Inf. Mes.) to anastomose with efferent fibres to the bladder. The tonic activity of the sympathetic retains urine in the bladder. Lesions of L1 or 2 roots may cause dribbling incontinence.

Innervation of Blood Vessels. The sympathetic vaso-constrictor fibres for the vessels of the upper limbs come from the stellate and upper two thoracic ganglia. Those for the lower limbs come from the 2nd, 3rd and 4th lumbar ganglia. They run in the spinal nerves and are distributed to the blood vessels chiefly in the periphery of the limbs where the arterial bed is richest (Woollard). Resection of these ganglia is carried out for Raynaud's disease, erythrocyanosis and circulatory disturbances

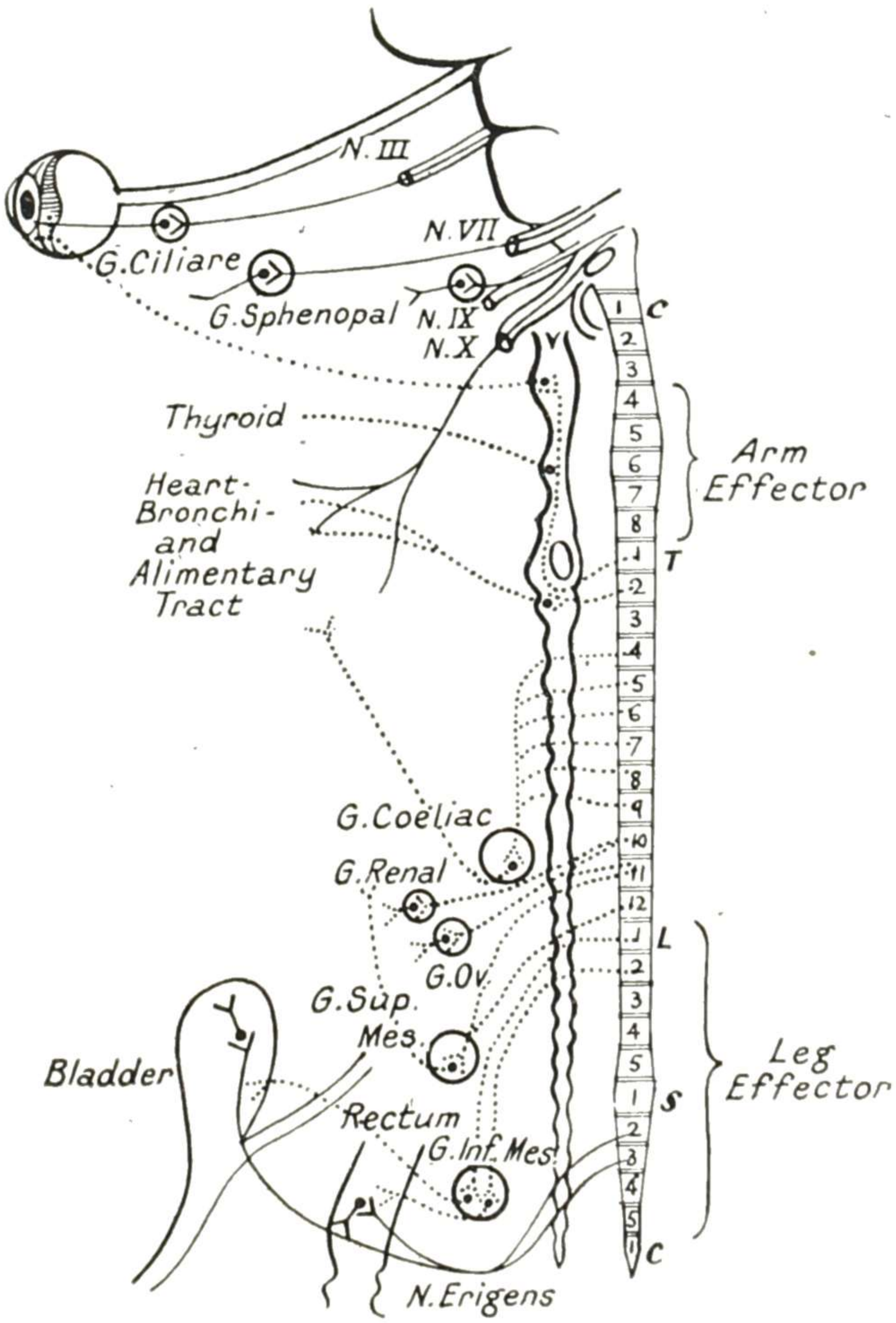


Fig. 173.—Diagram of the Autonomic Nervous System.

(Sympathetic represented by dotted lines and the parasympathetic by continuous lines.)

following poliomyelitis. Dorso-lumbar splanchnectomy is practised to produce lowering of high blood-pressure.

The autonomic nervous system and the hypothalamus may be thought of as an emergency protective mechanism which may be aroused by physical or psychological factors. The physical factors are pain, extremes of temperature, hæmorrhage, or infection. The sympathetic response is by vaso-constriction or dilatation, change in the rate of the heart beat or respiration, release of glycogen and adrenalin, etc. Emotional factors can cause similar physical effects, visceral reactions to psychological stimuli.

PART A. SYMPTOMATOLOGY

§ 691. The symptoms of disease of the nervous system may be *subjective* or *objective*. The *subjective symptoms* are those of which the patient complains to you, *e.g.*, headache, pain, giddiness, sleeplessness, numbness and tingling, a useless feeling in a limb, or double vision. The *objective symptoms*, or physical signs, are those signs noted by the patient's doctor on examination, *e.g.*, papillœdema, pupillary abnormalities, external ocular palsies, dysarthria, inco-ordination, muscular wasting, spastic or flaccid paralysis, loss of tendon reflexes, etc. Subjective symptoms occur in organic as well as in functional disease, and may or may not be accompanied by physical signs. It is impossible, without a careful physical examination, to exclude the presence of organic disease on the patient's statements alone. General lassitude, such as is met with in the neuroses, may be the first symptom complained of in an organic disease such as disseminated sclerosis, and the diagnosis is made only by finding an extensor plantar response, absent abdominal reflexes, pallor of the temporal halves of the optic discs and diminished vibration sense over the tibial malleoli. Again, a patient with an organic disease may suffer from an associated neurosis.

The chief subjective symptoms of which patients complain are:

I. Nervousness and Exhaustion. Tremulousness.
II. Giddiness.
III. Pain, Numbness and Tingling.
IV. Headache.
V. Disorders of Sleep.
VI. Loss of power or inability to control one or more limbs.

I. **Nervousness and Exhaustion. Tremulousness.**—Patients presenting these symptoms should on no account be diagnosed as "neurotic" until after careful and painstaking physical examination. The finding of a persistent tachycardia, an enlarged thyroid and lid-retraction with raised basal metabolic rate may reveal *Hyperthyroidism*. Such symptoms may also be complained of in individuals addicted to *Alcohol* or *Drugs*, while toxic absorption from a *septic focus*, *B. coli* infection or early *pulmonary tuberculosis* may manifest themselves first by these symptoms.

TREMULOUSNESS is a common finding in *Striatal Disease*, notably Parkinsonism, following Encephalitis Lethargica, or in Idiopathic Paralysis Agitans. The general picture of rigidity and slowness of movement may escape the doctor's notice. The tremor, in the early stages of Parkinsonism, is monoplegic or hemiplegic in distribution and eye signs may be present (see § 765). The mistake is most frequently made in post-encephalitic Parkinsonism, where one meets with a "neurasthenic" type of sequela which has an organic basis. General tremulousness is a common finding in the early stages of *General Paralysis of the Insane* (an Argyll-Robertson pupil, or tremor of the face and tongue and tremulous articulation, in such cases, are often missed) (§ 902). The early symptoms of *cerebral arterio-*

sclerosis may resemble those of functional disease, but the retinal arteries show arterio-sclerotic changes and other signs of arterial degeneration are present.

§ 692. II. **Giddiness. Vertigo.**—Vertigo may be defined as the consciousness of disordered orientation of the body in space. True vertigo implies a subjective sensation of rotation or oscillation, either of self (subjective vertigo), or of surrounding objects (objective vertigo). In many cases the postures and movements of the limbs, especially the lower limbs in standing and walking, are ill-adjusted and unsteady. The mechanism of equilibrium is described in § 680.

Examination of Cases with Vertigo.—Enquire if the patient experiences true subjective or objective sensations of rotation. Patients use the word "giddiness" to cover a variety of vague sensations of loss of confidence, viz. nausea, sinking feelings and even losses of consciousness, which are not true vertigo. Are the symptoms constant or paroxysmal? Enquire for a history of head injury or taking of sedative drugs. Is tinnitus present? Test the hearing, look at the ear-drums and fundi, and test the corneal reflexes. Examine for nystagmus and note its character. Test the limbs for ataxia and past-pointing, and test the tonus of the limbs. Examine the reflexes. Examine the heart, and take the blood pressure, and test the urine for albumen. Look for septic foci; take blood for the Wassermann Reaction, and have the Eustachian tubes inflated.

A. *The Vertigo is* Paroxysmal *and* Deafness *is present.*

Ménière's Syndrome. This disease affects persons in middle life and late middle age, men more frequently than women. A previously healthy individual is suddenly seized with intense giddiness causing uncertain gait, reeling or even falling, with nausea, vomiting, and syncope. Consciousness may be lost in severe attacks, or there may be transient loss of vision or diplopia. After the attack the patient is left with (1) diminution in hearing in one ear (nerve deafness), which is progressive, and (2) continuous tinnitus. Attacks of vertigo last minutes or hours, and occur every few days or weeks. As the deafness progresses, the attacks of giddiness diminish, and finally disappear, when loss of hearing is complete. Deafness may not be present after the first few attacks, but appears later.

The condition has been ascribed to a toxic affection of the labyrinth. Cairns and Hallpike have described gross dilatation of the endolymph system in two cases examined histologically. Ménière's original case was due to a hæmorrhage into the labyrinth, but this is a rare cause. A metabolic disturbance of fluid balance has been blamed for the condition, and some cases are relieved by dehydration treatment. Syphilis is a rare cause.

Diagnosis.—The residual deafness and tinnitus distinguish the condition from *epilepsy* and from *migraine.*

Treatment.—Sources of focal sepsis must be eradicated. Over-smoking does harm. Some patients are improved by restriction of fluids to $1\frac{1}{2}$ pints daily. Blistering the mastoid may help others: a small fly blister $\frac{3}{4}$ inch square is applied for twenty-four hours. If syphilis is present it must be treated. Inflation of the Eustachian tubes should be practised in all doubtful cases. Phenobarbitone (luminal) in $\frac{1}{2}$-grain doses twice or three

times daily helps many cases and should be taken with religious regularity over a period of months. The patient should not be allowed to swim, drive a car or ride a bicycle until free of attacks for six months. When the attacks are rendering the patient's life a misery, destruction of the affected labyrinth by alcohol injection into the external semicircular canal may be undertaken by an expert, or surgical division of the vestibular nerve. Both these operations usually destroy hearing.

Aural vertigo. Patients with chronic nasal infection and Eustachian obstruction may suffer from paroxysms of vertigo. The nasal infection should be treated and the Eustachian tubes inflated until the obstruction is relieved. The giddiness disappears when this end is achieved. In *Otosclerosis* the attacks of giddiness are treated as in the Ménière syndrome.

B. *The Vertigo is* PAROXYSMAL *and* DEAFNESS IS ABSENT.

The vestibular reflexes may be affected by alterations in the cardio-vascular system, causing a vertigo which occurs when rising from the horizontal or sitting posture, or bending down, or on prolonged standing. In *cerebral arterio-sclerosis*, the retinal vessels may show disease and the blood pressure may be raised; in *aortic regurgitation*, signs of aortic disease will be present, and in *Stokes-Adams'* syndrome heart-block will be present. In the vasomotor instability of the *menopause*, of *convalescence* and after *hæmorrhage*, *e.g.*, from a duodenal ulcer, vertigo of this type may occur. Cases of *emphysema* may suffer in this way after a bout of coughing. Paroxysmal vertigo may occur in *migraine*, and may be associated with teichopsia and a family history of the affection. A *petit mal* attack may manifest itself by a bout of giddiness. After *head injuries*, giddiness on alteration of posture, increased by coughing or sneezing, is sometimes a severe and persistent symptom. In many of these cases there has been contusion or laceration of the labyrinth itself. Various *toxæmias* and severe *anæmias* cause occasional vertigo.

C. *The Vertigo is* CONTINUOUS.

(1) *Suppurative ear disease is present.* Suspect ACUTE LABYRINTHITIS and look for signs of CEREBELLAR ABSCESS, or INTRACRANIAL SUPPURATION.

Acute Labyrinthitis is an extension of inflammation of the middle ear into the cochlea and labyrinth. The symptoms are rapidly progressive, with pyrexia, vertigo, deafness, and tinnitus. Coarse nystagmus on looking to the side of the lesion, hemiataxia and hypotonia together with past-pointing, are present in the ipsi-lateral limbs. In progressive cases a radical mastoid operation is often necessary. Cholesteatoma in the middle ear may be associated with cerebellar abscess.

(2) PARALYSIS OF AN OCULAR MUSCLE *is present.* Diplopia resulting from squint may cause continuous vertigo from visual disorientation.

(3) *Signs of* NEUROLOGICAL DISEASE *are present.*

If the corneal reflex is diminished or absent on one side, with unilateral deafness and tinnitus, an *acoustic neurofibroma* should be suspected. Facial weakness and blunting of trigeminal sensation may be present on the side

of the lesion and hemiataxia may be present with an extensor plantar response. Irrigation of the external auditory meatus with hot and cold water will reveal impairment of labyrinthine functions. If the patient is a young adult and the vertigo is associated with intention tremor, absent abdominal reflexes, extensor plantar responses, and diminished vibration sense over the tibial malleoli, the condition is likely to be *disseminated sclerosis*, with acute lesions in the pons.

If the patient is elderly, and sudden vertigo is associated with ipsilateral hypotonia and hemiataxia, with loss of pain and temperature sensibility over the corresponding half of the face and opposite side of the trunk and limbs, the condition is a *unilateral bulbar syndrome* from thrombosis of the posterior inferior cerebellar artery. In these cases there may be unilateral paralysis of the palate, pharynx, vocal cord and tongue, together with miosis on the side of the lesion. Signs of arterial disease will be present.

Vertigo occurs with nerve deafness, tinnitus and facial paralysis, in cases of *herpes zoster* affecting the external auditory meatus, when the lesion is in the geniculate ganglion.

(4) In PSYCHONEUROSES, feelings described by the patient as "giddiness" are frequent. This is rarely a true vertigo, but rather a feeling of insecurity or lack of confidence. The feeling may be accompanied by sensations of unreality, either of the individual or her surroundings. Such patients tend to be asthenic, with vasomotor instability, visceroptosis and low intra-abdominal pressure. The anxieties will diminish with explanation and reassurance, and the giddiness disappears as the patient acquires more confidence. An abdominal belt will help very thin patients with poor abdominal muscles.

III. Pain, Numbness, Tingling.

§ 693. Pain.—A careful observer will always investigate four important features of a pain. (1) The exact *distribution and sites of radiation.* (2) Its *character and degree*—whether throbbing, burning, stabbing, aching, etc. (3) The *factors which increase the pain*, *e.g.*, coughing, sneezing, deep breathing, or movement in a particular direction. (4) Its *constancy*, *i.e.*, whether persistent or paroxysmal. Local examination of the painful part should never be omitted. A patient's own diagnosis of "rheumatism" should not be accepted without question. The lightning pains of tabes dorsalis, which commonly precede all the other symptoms of that disease by years, may be wrongly diagnosed by the doctor as rheumatic, on insufficient examination. Painful neurological conditions are dealt with in § 816.

SECONDARY CARCINOMA OF THE VERTEBRÆ gives rise to severe and persistent root pains, without marked objective neurological signs or local abnormality in the spine. Two years or more after an operation for cancer (commonly mammary carcinoma), the patient, while making some physical effort, suffers sudden intense pain in the back. These "alarm pains" become increasingly frequent and persistent, and, with increasing cachexia

and sleeplessness, may be the only symptoms present. Radiograms of the vertebræ often appear normal. Such deposits occur mostly in the lumbar vertebræ, but signs of compression of the cauda equina (see § 798) are late, and the patient may die of secondary deposits in other organs before they appear.

Dysæsthesiæ or **Paræsthesiæ** are subjective sensations of tingling, "pins and needles," crawling sensations under the skin, numbness, burning, etc. Their distribution and constancy should always be carefully noted and whether they are accompanied by local vasomotor changes (Raynaud's Disease, § 579). Such symptoms are early and transient phenomena in a limb in *disseminated sclerosis*. The "girdle sensation" of *tabes dorsalis* is a feeling as if a tight cord were tied round the waist. Pins and needles occur bilaterally in the hands, feet and legs, in cases of *subacute combined degeneration of the spinal cord* and *polyneuritis*, accompanied by sensory and reflex changes. Where nerve trunks are compressed (*e.g.*, in *the rib-pressure syndrome*), paræsthesiæ may occur before the onset of the pain. Paræsthesiæ of peripheral nerve distribution occur in *peripheral nerve lesions* of varied etiology. Pins and needles in the hands and fingers occur in *severe anæmia* and in *myxœdema*.

§ **694. Acroparæsthesia** is a condition of numbness with tingling, pins and needles, and often disagreeable burning sensations in the hands and fingers. The fingers may be clumsy but they do not alter in colour and there is no loss of tendon reflexes, true paresis, wasting, or gross sensory loss. The intrinsic muscles of the hands may be tender and there may be slight blunting of cutaneous sensibility. The patients are commonly middle-aged women and there may be a history of endocrine disorder or debility, or harder manual work than usual. The condition does not respond immediately to treatment but eventually clears up entirely. *Treatment.* Rest in bed for 2 to 3 weeks with the elbows supported on pillows will help many cases. Patients should be told to keep the hands as dry as possible. Mild sedatives and hypnotics help some. Massage and faradism to the shoulder-girdle muscles are sometimes advised.

§ **695.** IV. **Headache.**—This is a name applied to any feeling of discomfort in the head, not necessarily pain. An exact description of the type of discomfort should always be obtained. Headache is productive of great incapacity, owing to inability to concentrate, intolerance of light and noise, irritability and anxiety occasioned by the pain. The brain itself, the pia arachnoid and ependyma are insensitive, and the pain of headache arises either in the dura mater or around the venous sinuses or in the neighbourhood of the intracranial arteries. Pain in the head may arise from irritation of the roots of the fifth, ninth and tenth cranial nerves, and of the sensory roots of the upper three cervical nerves. The pain may be referred to the cutaneous distribution of the nerves of the cervical plexus, over the scalp and bones of the head (see Fig. 186). Headache is often a manifestation of a generalised increased sensitiveness of the nervous system referred to a single nerve (*e.g.*, the supraorbital) or group of nerves. To some doctors, headaches spell only constipation or eye-strain, but these are infrequent causes of all but

episodic headaches. *Anxiety* is probably the most important cause of continued headaches.

Investigation of a Case of Headache.—In investigating a case of headache it is important to obtain a detailed history, especially with regard to the character of the discomfort, its site and distribution, the time of onset of the headache, its periodicity, and any accompanying signs and symptoms, *e.g.*, visual spectra, paræsthesiæ in the limbs, swelling of the cheek or puffiness of the orbit during the pain. A family history of headache should always be sought for and the importance of searching for a local cause, *e.g.*, rheumatic deposits in the occipital muscles, new growths of the skull, increased intraocular tension in glaucoma, etc., cannot be sufficiently emphasised. The cause is usually revealed, not by the stethoscope, but by special examinations—the examination of the fundi and nervous system, taking the blood pressure, testing the urine, and by X-raying the skull and accessory nasal sinuses.

Headaches are due to (A) Neurological and Local Causes, (B) General Causes, (C) Reflex Causes.

A. *Neurological and Local Causes of Headache.*

These headaches have characteristic clinical distinguishing features. They are:

(I) Anxiety Headaches.
(II) Migraine.
(III) Neuralgia.
(IV) Sinus Headaches.
(V) Meningeal Headaches (including Subarachnoid Hæmorrhage.
(VI) Syphilitic Headaches.
(VII) Traumatic Headaches.
(VIII) Headaches due to Increasing or Decreasing Intracranial Pressure (including Pituitary Tumour).

(I.) Anxiety Headaches.—The abnormal sensation complained of is rarely pain; usually it is described as something worse and less bearable than pain. It may be a feeling of weight or pressure, or aching tightness of the head. Other symptoms are fatigue, listlessness, restlessness, inattention, sleeplessness. Such are the headaches most commonly met with in practice.

§ 696. (II.) Migraine (Synonyms: Hemicrania, megrim, sick headache, bilious headache).—This is an affection characterised by recurring paroxysms of intense headache with, or without, visual and sensory phenomena, and nausea with, or without, vomiting.

Symptoms.—On the day before an attack the patient may feel vague malaise and know that one is impending. The following phenomena may be present in an attack: (1) Slow visual auræ of several kinds. A black spot at the side of the visual field, hemianopia, the appearance of golden X's or Z's, or a shimmering spot which opens out into a curved zig-zag "fortification" spectrum. (2) Slow sensory auræ of tingling in one hand, spreading up the arm into the lips and tongue, or down the trunk into the leg, sometimes accompanied by weakness of the affected limbs and aphasia. These auræ are characterised by the *slowness* of their spread;

they last 10 to 20 minutes and are replaced by the characteristic headache. (3) The headache is often unilateral and temporal, later becoming bilateral. When the aura is absent the headache commonly commences in the early morning and is described as a burning, throbbing pain, which increases in intensity, accompanied by (*a*) photophobia and (*b*) pain on attempting to move the eyes. (4) Nausea or even vomiting, and sometimes abdominal pain due to tonic spasm of the colon. Cases have been described of transient unilateral blindness at the height of the headache, due to spasm of the central retinal artery, seen ophthalmoscopically. Recurring external ophthalmoplegia in association with migraine headache, so called *migraine ophthalmoplégique*, has been described, but many such cases have been found, at autopsy, to have suffered from leaking "berry aneurysms" of the circle of Willis or from intracranial tumour.

The attack lasts an hour or two, or all day. The following day the patient generally feels prostrated or exhausted. Attacks recur at varying intervals, once or twice a week in severe cases, or every few months. Recurring intense headache may be the only symptom, or the slow sensory auræ or visual phenomena may recur alone, without being followed by headache.

Trigeminal Migraine is a type of the malady in which there is radiation of the pain to the face or supraorbital region. This should never be mistaken for tic douloureux, for the pain never runs along the lips and tongue, the pain is never started off by eating, talking, or washing the face, and "trigger zones" are absent.

Ciliary Migraine is a type of migraine in which the pain is referred to the eyeball or to the retro-orbital region. Great care should be taken in these cases to exclude glaucoma or orbital growths. Injection of the supraorbital and infraorbital nerves with alcohol, as advised by Wilfred Harris, is a useful adjunct to the usual treatment for migraine in this type of case.

Diagnosis.—Migraine-like headaches occur in tumours of the occipital lobe and in chronic nephritis with uræmia. The diagnosis of migraine is made on the recurring paroxysms of headache, the unilateral character of the headache, the accompanying phenomena, and the absence of objective signs of disease. The slowness of the aura in migraine is of great value in the diagnosis from Jacksonian or other epilepsy, as the duration of an epileptic aura is but a few seconds.

Etiology.—The disease runs markedly in families; a direct inheritance can, in most cases, be traced. In some cases a history of epilepsy in the family can be ascertained, and migraine and epilepsy may occur in the same subject. The disease may begin in childhood, but commonly shows itself just before puberty, and it continues through life until past middle age. It is commoner in women than in men, and in women there is often an exacerbation in the severity and frequency of the headaches at the menopause; after this they tend to disappear, but do not always do so. The disease is probably analogous to epilepsy. In cases of recurrent unilateral migraine, small berry aneurysms ("congenital" aneurysms) are sometimes found and these may cause subarachnoid hæmorrhage by rupturing. The disease has been ascribed, without much foundation, to a recurring unilateral hydrocephalus. Amongst the factors which excite

attacks are (1) Eye-strain, (2) Dietetic indiscretions, (3) Menstruation, (4) Exposure to cold or (5) Emotional stress.

Treatment.—The nature of the malady should be clearly explained to the patient, who may fancy that he has a cerebral tumour. He should be told at the outset that it is unlikely that his headaches will disappear completely as the result of treatment, although it is possible to mitigate the severity of the attacks and lengthen the intervals between them. These patients should avoid overstrain, cold, and dietetic indiscretions. A morning dose of magnesium sulphate gr. 60, and the administration of glucose ½ oz. in orangeade, thrice daily between the attacks, and light diet, help some cases. Refractive errors should be corrected and a dentist consulted regularly. Some cases are helped by psychotherapy. The most useful measure in treatment is the continuous administration of a drug, such as phenobarbitone, gr. i., at night, and the patient should be advised to take this regularly until he has been free from headaches for a year. Later, the dose may be reduced. Other remedies are sometimes used: (1) ℞ sod. bromide gr. x., liq. trinitrin ♏ 1, liq. strychnini ♏ 5, ac. hydrochlor. dil. ♏ 10, tinct. gelsemii ♏ 5, aq. chloroformi ad fl. oz. ½; t.i.d., p.c., and (2) Thyroid extract in gr. ½ doses, once or twice a day. A cup of strong coffee or a walk in the fresh air may stave off an attack.

In the Attacks.—Once the attack has commenced little can be done to prevent its inevitable progress. Two drugs given hypodermically are sometimes successful in aborting an attack. These are: (*a*) adrenaline hydrochloride 1/1000 solution, in 3 to 5 minim doses, and (*b*) ergotamine tartrate (femergin): 0·5 c.c. (*i.e.*, 0·25 mgm.) intramuscularly, may be repeated in 2 hours, or 1 tablet (1 mgm.) of femergin may be given orally and repeated in an hour. It should not be given to pregnant women; in the non-pregnant female it may cause uterine colic if given during the menstrual periods. Alternatively, the patient should lie down in a darkened room and take (1) cibalgin gr. 5, in tablet form, every half-hour until three doses have been taken, or (2) phenacetin gr. 15, caffeine citrate gr. 5, to be repeated at half-hourly intervals for three doses, if necessary.

(III.) NEURALGIAS.—Persistent pain over the distribution of the supraorbital nerve without objective sensory impairment, *Supraorbital Neuralgia*, is a common symptom of anxiety neurosis, although it may follow injuries to the supraorbital nerve. A severe form of recurring *Frontal Neuralgia* is met with coming on in middle life, mostly in men. In many cases it does not appear to have a psychological basis, and may yield to injection of the supraorbital nerve with alcohol. Severe frontal neuralgia may also follow ophthalmic herpes (see § 856). *Occipital neuralgia* is commonly due to rheumatic infiltration in the posterior cervical muscles, but secondary carcinoma, Pott's Disease and spondylitis, may cause similar pain. In the rheumatic cases the pain may yield to massage and applications of antiphlogistine or a mustard plaster. In severe cases the nodules should be felt for and injected with 5 to 7 minims of 90 per cent. alcohol, using a hypodermic needle and syringe.

(IV.) SINUS HEADACHES.—Disease of the nasal accessory sinuses is an important cause of pain referred to the supraorbital region or to the teeth. These headaches are intermittent, and are characteristically accompanied by œdema of the orbital tissues (in frontal sinusitis) or of the face (in antral suppuration) and are relieved by a gush of pus from the nose (§ 179). Local tenderness is often present on palpating or percuss-

ing the wall of the sinus, or, in the case of the frontal sinus, on upward pressure from the orbit on the floor of the sinus. Fœtor may be present. The sinus affected does not transilluminate clearly. Suppuration in the ethmoidal sinuses is characterised by pain over the nasal bridge, behind the eyes or over the temples. Palliative treatment consists of putting the patient to bed and applying hot fomentations to the face or forehead, and an oily solution of ephedrine to the nasal mucous membrane. A brisk purge, *e.g.*, calomel gr. 3, is administered, and aspirin gr. 10 four hourly, may be given for the pain. Relief may be obtained by inhaling the vapour of ten drops of a 25 per cent. solution of menthol in spt. vini rect. in a pint of boiling water. Surgical treatment is sometimes necessary, however.

(V.) MENINGEAL HEADACHES.—The pain in meningeal irritation is intense and severe in the occipital region and in the spine, and is accompanied by rigidity of the posterior cervical muscles and spine. There is characteristic irritability and drowsiness, passing on to coma in severe cases. The occurrence of this type of headache is sufficient indication to perform a diagnostic spinal puncture. Meningeal headaches arise from a variety of causes of meningeal irritation, *e.g.*, *Meningitis*, *Meningism* (see §§ 726, 731), the leakage of blood or pus into the subarachnoid space, or irritation of the meninges by vascular tumours, especially tumours of the vermis of the cerebellum, the fourth ventricle or base of the skull. *Sunstroke* produces sudden, severe meningeal headache (see § 508). *Subarachnoid Hæmorrhage* (see § 717).

(VI.) SYPHILITIC HEADACHES.—Headaches due to syphilitic lepto- or pachymeningitis are often nocturnal, increasing in intensity. They may be accompanied by alteration in the character of the patient, and cranial nerve palsies, *e.g.*, Argyll-Robertson pupil may be found. The characteristic changes in the spinal fluid of lymphocytosis with positive W.R. confirm the diagnosis.

(VII.) TRAUMATIC HEADACHES.—Cases of UNRESOLVED CONTUSION OF THE BRAIN, after head injuries, are followed by characteristic (1) localised headaches intensified by exertion, noise or alteration in posture, (2) giddiness, (3) inability to concentrate. There may be accompanying localised tenderness of the scalp and slight facial weakness, pupillary abnormalities and alterations in the reflexes may be present. The symptoms are due to cerebral œdema persisting around the contused area of the cortex. Owing to the lack of elasticity of the dura the reactionary swelling does not resolve, as in a superficial contusion, say of the skin, but may persist for many months, producing a disordered state of the cerebral circulation. Contusion headaches may be prevented by keeping patients who have had moderate or severe head injuries in bed, with complete physical and mental rest, from four to six weeks, even in the absence of signs of focal organic lesions. When present, the headaches are treated by resting the patient in bed, propped into the position which affords him greatest comfort, giving bromides gr. 10 thrice daily, well diluted, and a morning saline aperient (see also § 716).

Headache, following trauma, is one of the characteristic symptoms of *Subdural Hæmatoma* (see § 827).

Patients who have suffered head injuries may develop *compensation hysteria*, especially if there is litigation for compensation (§ 889). These cases have accompanying tachycardia, loss of weight and sleeplessness, with anxiety dreams, symptoms not present in pure Cerebral Contusion.

(VIII.) HEADACHES DUE TO ALTERED INTRACRANIAL PRESSURE.—The headache of Cerebral Tumour, Abscess or Chronic Subdural Hæmatoma, is paroxysmal, especially evident on waking and relieved by lying down. It is accentuated by coughing, vomiting or stooping. Vomiting may be present with the headaches, papillœdema and signs of a slowly progressive local cerebral lesion, *e.g.*, a gradually increasing hemiparesis. In tumours of the posterior fossa the headache may be produced when the patient turns his head. The cause of such headaches is uncertain, but may be attributed to local pressure on the dura or blood vessels, or to direct pressure of the brain-tissue or the growth upon the trigeminal nerve or Gasserian ganglion. Where an intracranial growth is suspected, local tenderness of the scalp or skull should always

be sought for. *Pituitary Tumour* is associated with bitemporal headache and a feeling of bursting pressure behind the eyes. Other symptoms and signs will be present (see Group XII. § 829). *Lumbar puncture* headache associated with *low* intracranial pressure may be cured by intrathecal instillation of 20 c.c. warm sterile saline.

B. *General Causes of Headaches.*

Amongst the *General* or *Constitutional Causes* of Headache are: (1) *Uræmia* in any form of nephritis. It may be suspected where there is abnormality of the urine, nocturnal frequency of micturition, general œdema, high blood pressure with recurrent epistaxis, retinal hæmorrhages or albuminuric retinopathy. (2) *Arterio-sclerosis* with high blood pressure and (3) *chronic lead poisoning* (lead encephalopathy) may be attended by severe headache. Headaches arise from the toxæmias of (4) *acute specific fevers*, (5) *malaria*, (6) *gout*, (7) *diabetes*, and (8) *alcoholism*. (9) *Constipation* is an important cause of episodic headaches. (10) The headache of *anæmia* is probably mainly toxic in origin.

C. *Reflex Causes of Headaches.*

(1) *Disease of the Eye*—glaucoma; iritis; refractive errors, such as hypermetropia and astigmatism, frequently combined with emotional strain; defective convergence. Prolonged use of the eyes, *e.g.*, for moving pictures, television, or in picture galleries, "eye-strain," produces episodic headaches. (2) *Disease of the Nose, Teeth or Ears.* (3) *Ovarian Uterine, Gastric or Cardiac Disease.* (4) *Wearing tight hats.*

Headaches in Children.—In young children headaches may be due to digestive disturbances or to rheumatism. The character of the stools should be enquired into, and specimens of fæces examined. A history of growing pains or chorea or tonsillitis should be sought for and the subcutaneous tissues of the elbows and shins palpated for rheumatic nodules. Eye-strain is a frequent cause of headache in school-children. Urinary infections may cause headaches in the young. A malady analogous to migraine is "cyclical vomiting" (§ 271), and until this is treated by restriction of fats, administration of glucose and alkalies, the headache and vomiting may persist. In obese children of the Fröhlich type, persistent and intractable headaches may be present, apart from intracranial tumour. Highly-strung children may suffer from anxiety headaches just as do adults, the cause lying in some factor of the child's environment, either at home or at school.

Treatment of Headaches.—Treatment consists in determining and treating the cause of the headache. The prescribing of anodyne drugs before the cause has been elucidated cannot be too strongly deprecated.

§ **697.** V. **Disorders of Sleep.**—The *amount* of sleep required in healthy individuals varies with the age of the person and the individual peculiarity. In general, it may be said that infants require about sixteen hours, adolescents ten hours, the middle-aged eight, and the aged five hours. Some families and individuals are notoriously poor sleepers and do not seem to suffer in health or comfort on this account. Man can exist without sleep for about the same time that he can do without food, viz., three to four weeks, but he cannot live without it.

In *normal sleep* the power to make voluntary movements is first lost, then the use of the special senses disappears, hearing being the last to go and the easiest to evoke on arousing the patient. General muscular relaxation, with ptosis, develops, the eyeballs turning upward and becoming slightly divergent. Respiration becomes slower and noisier, and tends to periodicity in the very young and the very aged. The pulse frequency lessens, the blood pressure falls with cerebral anæmia, and the general body temperature falls. Temporarily, the knee-jerks are abolished and the plantar responses become extensor. Two important features of normal sleep are: (1) Its fixed periodicity in the rhythm of sleeping and wakefulness. (2) The sleeper can be roused from sleep to normal activity, unlike the comatose or stuporose patient.

We have no precise knowledge of the *physiology of sleep*. Pavlov believes it to be a state of *active inhibition* of the cortical mechanism. Its function is to protect the nerve-cells so that they can recuperate from fatigue and recover their normal functions. In the region of the *hypothalamus* and *the grey matter of the floor of the third ventricle*, exists a nervous mechanism intimately connected with sleep. Damage to this area, by tumours or other structural disease, usually results in excessive drowsiness.

Sleep may be (A) *Diminished in Quantity* (insomnia), (B) *Defective in Quality* (disturbed sleep), (C) *Increased in Quantity* (protracted sleep). (D) *Sleep Rhythm* may be Inverted or Disturbed.

A. **Sleeplessness (Insomnia).**—The chief causes of sleeplessness are: (1) *Psychical*, but there are many other causes. Sleeplessness may arise (2) as the result of *pain* anywhere in the body, or *discomfort*, such as is caused by flatulence in the stomach or intestine, or by dyspnœa in cardiac disease or dropsy. (3) In *febrile conditions*. (4) In *organic brain disease*. In *Encephalitis Lethargica* absolute sleeplessness, lasting several days, may be met with, especially in children. Sleeplessness is an early symptom of *General Paralysis of the Insane*, when the patient is restless or excited (later, drowsiness and apathy are common). In *cerebral arterio-sclerosis*, especially when the blood pressure is high, the patient may fall off to sleep, but wakens in the early morning unable to sleep again. (5) In many forms of *chronic toxæmia*, *e.g.*, *uræmia* and *alcoholism*, sleeplessness may be present in the early stages.

The *Psychical Causes*, owing to their relatively great importance, must be considered in detail. They may be divided into three groups: (1) The patient is unable to sleep because of *anxiety* (by far the largest group). (2) The patient is unable to sleep because of some *bad habit of thought*, anxious preoccupation with affairs of the past, present or future, visualising of scenes, rehearsing of conversations, etc. (3) The patient, usually a bad sleeper, is *obsessed* with the idea of sleeplessness, or "insomnia" as he calls it. (See Anxiety Neurosis, §886.) Persistent insomnia may be the prelude to the development of a psychosis.

Treatment of Insomnia.—(1) The *factors underlying the neurosis* should be elucidated. Bad sleepers are nearly always apprehensive that the

TABLE XLV.—HYPNOTICS.

Indications.	Hypnotic.	Dosage and Method of Administration.	Untoward Effects.
(1) Sleeplessness due to mental perturbation	Aspirin	Two 5-gr. tablets crushed and taken with warm milk at bed-time	Sweating.
	Sodium Seconal	¾–1½ gr. by mouth	Fairly short action.
	Butobarbitone (Soneryl)	3 gr. by mouth	
	Sodium Barbitone (Medinal)	3 to 15 gr., in cachets	In large doses may produce confusion on waking. Possibility of habit formation.
	Allonal	2½ to 5 gr. by mouth (tab.)	
	Dial	1½ gr., in tablet form	More powerful in its effect than medinal.
	Sodium Amytal Pentobarbitone (Nembutal)	1–3 gr. by mouth 3 gr., in capsules.	More powerful than those above.
(2) Sleeplessness due to pain	Aspirin, Allonal	See above	
	Pyramidon	10 gr., in tablet form, by mouth	Continued administration of Pyramidon may cause agranulocytosis.
Pain of secondary deposits in bone	In severe cases A.P.H. Powder	A.P.H. Powder = Aspirin 10 gr., Pyramidon 7 gr., Heroin $\frac{1}{12}$ gr.	May cause confusion on waking. Possibility of habit formation.
	Veramon	6 gr. by mouth	
	Morphine	$\frac{1}{6}$–$\frac{1}{3}$ gr. hypodermically	
	Pethidine	50–100 mgm. by mouth or injection	
	Physeptone	5–10 mgm. by mouth or injection	Very effective.
(3) Sleeplessness due to motor excitement, *e.g.*, early psychosis, delirium tremens (for latter never give morphine)	Paraldehyde	60–480 ℥ in equal amount of olive oil, per rectum, or 30–240 ℥ by mouth or 4 c.c. intramusc.	
	Phenobarbitone	¼ to 3 grains dissolved in hot water by mouth, or by intramuscular injection	Confusion on waking. Erythematous rash.
	Sodium Amytal	1–3 gr. Can be repeated.	
	Somnifaine	20–40 drops (8–16 ℥) by mouth, or 2 c.c. intramuscularly.	A powerful sedative.
(4) Sleeplessness in cardiac disease.	Chloralamide	25 gr. in 1 fl. oz. of brandy	
(*a*) Without dyspnœa	Potassium bromide Chloral hydrate	20 gr. 15 gr. In a mixture	Furring of tongue. Digestive upset. Rarely confusion.
(*b*) With dyspnœa	Morphine	1/6 to ¼ gr., hypodermically	Constipation.
(5) Sleeplessness, with dyspnœa, in acute pneumonia	Morphine Atropine	1/6 gr. 1/100 gr. Hypodermically	
(6) Sleeplessness in febrile states	Morphine Hyoscine	¼ gr. 1/100 gr. Hypodermically	Excitement and confusion.
(7) Senile and arteriosclerotic insomnia	Alcohol	Given as whisky or cognac with the last meal, or before retiring	
	Blue Pill	½ gr., at night	

lack of sleep will cause insanity, or that if they take hypnotics they will become drug-addicts. Such patients should be reassured. (2) In slight

cases *physical treatment* is prescribed—comfort, quiet, warmth, change of surroundings, hot baths followed by massage, hot packs, hot drinks, last thing at night, may suffice to restore the lost sleep habit. Heavy meals and over-fatigue, emotional or intellectual strain should be avoided late in the evening. Some sleep better at the seaside, some at a higher altitude. A period of rest in bed, away from home and its distractions and worries, helps the poorly-nourished and tired-out patient. A dry biscuit and a thermos-jug of hot milk by the bedside will sometimes help those who waken in the night to fall asleep again. (3) *Hypnotics.* (Table XLV.) In more severe cases, where the continued loss of sleep is rendering the patient panicky and less and less capable of dealing with his anxieties, the insomnia should be immediately relieved by hypnotics. The *rules for prescribing a hypnotic* are: (1) It should first be explained to the patient that he is not being drugged, and that it is better for him to obtain sleep with a mild sedative than to go on having sleepless and wearying nights without it. (2) It should be prescribed nightly *for a stated initial period, e.g.*, three nights, a week, a fortnight. (3) The *hour at which it is to be taken* must be definitely stated. The additional anxiety of having to decide when to take his hypnotic is bad for the patient. (4) If possible, the patient should not know what hypnotic he is having. (5) It must be given in adequate dosage to produce sleep. (6) If the patient says that it is losing its effect, the same dose should be given in divided quantities, every quarter of an hour, before it is decided to increase the dose or change the hypnotic. *Medinal*, in doses of 5, 7½, or 10 gr., is the best of the hypnotics for use in severe cases. *Chloral hydrate*, 3–20 grains, is a useful hypnotic, and can be given in 3–5 grain doses to children. *Bromide* is not very satisfactory as a hypnotic, as in large doses it causes confusion on waking, gastro-intestinal upset and furring of the tongue. It should be given, well diluted, combined with chloral hydrate, in 10–15 grain doses. Chloral is useful in cases of cerebral arterio-sclerosis, and should be combined with a nightly dose of blue pill, ½ grain, and a morning saline aperient. In severe cases of sleeplessness the number of hours of sleep should be charted by the nurse. Patients who sleep badly, notoriously exaggerate this symptom, and it is often hard for the physician to obtain accurate statements.

B. **Defective Sleep.**—(1) *Nightmares* or *Night-Terrors* may be due to physical or psychical causes. In *children* the cause is commonly some unhappiness in the child's domestic or school environment, an over-anxious, bullying or quarrelsome parent, or an emotional trauma. Physical causes also operate, *e.g.*, thread-worms, gastro-intestinal fermentation, adenoids severe enough to impede respiration. In *adults*, night-terrors are commonly the result of a psychoneurosis; disturbed sleep and frequent waking up may also occur in advanced cardiac disease, chronic uræmia and other toxæmias. (2) *Somnambulism* or sleep-walking is a condition in which the sleeper rises apparently asleep and behaves automatically. It is commoner in children than in adults (see Hysteria, § 888).

C. **Protracted Sleep.**—Pathological drowsiness is met with in (1) Lesions affecting the hypothalamic region and the grey matter of the floor of the third ventricle, *e.g.*, Tumours of the *pituitary stalk*, *encephalitis lethargica* and *trypanosomiasis* (African sleeping sickness). (2) Increasing intracranial pressure, from any progressive intracranial lesion, *e.g.*, cerebral tumour. After a *head injury* the patient may sleep for several hours. (3) Chronic toxæmias, such as uræmia and diabetic ketosis, lead to drowsiness and, later, coma. (4) Obesity, such as the obesity of *myxœdema* and *pituitary disease*, is often accompanied by sleepiness. (5) After an *epileptic* fit the patient may sleep for many hours.

In trance states of psychopathic origin the patient appears to sleep but resists if one attempts to open the eyelids. Mutism is common. The condition may be acute or subacute in onset and may last for weeks or months. It is met with in schizophrenia, in confusional states and other psychoses, and in hysteria. Recurrences are frequent.

§ **698. Encephalitis Lethargica** (Synonyms: Epidemic Encephalitis, "Sléepy Sickness") is an infection of the nervous system characterised by acute and chronic phases. The acute stages of the illness are often marked by drowsiness, ptosis, squint, diplopia and a mild febrile reaction; absolute sleeplessness or inversion of the sleep rhythm is also met with. Since the first recognition of the disease in England, in 1918, the clinical picture has varied widely in different outbreaks. In the great majority of cases seen at the present time, the initial phases pass unnoticed by the patient; there is no history of an " acute attack," and the case comes to you because of some late manifestation, such as Parkinsonism (§ 765).

Symptomatology.—After an incubation period of a few days, during which the patient feels malaise, headache, pains in the limbs, coryza or constipation, the patient is conscious of persistent double vision and increasing drowsiness whilst he goes about his work. By the time he is seen by the doctor, some hours later, lethargy has set in. The patient lies in bed for days, intensely drowsy and heedless of his surroundings. There is usually mild fever. The *lethargy* is profound in the daytime. The patient can be roused by shaking to talk intelligently or to take food, if commanded, but often falls off to sleep again with unswallowed food in his mouth. At night the drowsiness gives place to muttering occupational delirium, and hallucinations may be acted by the patients.

At this stage ocular disturbances are common, or facial palsy, of the upper or lower motor neurone type, which later clears up. The *ocular changes* are varied: (1) Pupillary disturbances of all kinds, *e.g.*, irregularity and inequality, impairment of light reaction and the reaction on accommodation. The reverse of the Argyll-Robertson phenomenon may be found, viz., loss of reaction on accommodation, with preservation of reaction to light. The pupils may be strongly contracted or dilated. (2) Ptosis is common, usually bilateral; it is often missed, as it is thought to be due to the sleepiness. (3) External ophthalmoplegias of all types, either paralysis of individual muscles or paralysis of conjugate movement.

(4) Papillœdema is rare; when present, the swelling rarely measures more than one dioptre. (5) Nystagmus and photophobia.

As the long pyramidal and sensory tracts are unaffected, there are no sensory phenomena nor paralysis of the limbs. Sometimes, however, an extensor plantar response or an absent abdominal reflex is found; very rarely a hemiplegia. Widespread muscular asthenia is common, and, owing to the profound stupor, the bed is soiled with the excreta, or retention of urine may occur. The *cerebro-spinal fluid* is clear and without clot. In a third of the cases it is normal serologically; in the others it is under increased pressure, shows a few extra lymphocytes or rarely up to 100 cells per cu.mm. (always a *pure mononuclear* increase) and normal chloride and glucose content. In a few cases the fluid is hæmorrhagic, and in these clot and polymorphonuclear leucocytes may be present owing to the contained blood. The Lange curve may be of the luetic type.

Diagnosis is made on the clinical picture and spinal fluid findings. In *neoplasm of the hypothalamic region* signs of increasing intracranial pressure will show themselves, and papillœdema is usually more marked than in encephalitis lethargica.

Rarer Clinical Types of the Malady.—(1) *Type with Insomnia.*—Despite its name, lethargic encephalitis may manifest itself by succeeding nights of insomnia or by inversion of the sleep rhythm. (2) *Myoclonic Type.*—Sudden, shock-like contractions occur in individual muscles, usually the abdominal muscles or anterior thigh muscles. This may be associated with persistent hiccough. In some cases of this type severe root pains occur (§ 775). (3) *Choreiform Type.*—Choreiform movements appear in the limbs and face, like those of rheumatic chorea. Characteristic pupillary disturbances may be present. Lethargy is absent in such cases as a rule; sleeplessness and delirium are present. (4) *Akinetic Type.*—The onset is usually subacute and the temperature only slightly elevated. Features of Parkinsonism, with poverty of movement, rigidity and tremor, gradually appear. (5) Sudden Hemiplegic cases (acute subarachnoid hæmorrhage or cerebral hæmorrhage). (6) Polyneuritic cases and Cerebellar cases are described, but they are excessively rare. (7) Epidemic Hiccough is described in § 273.

Prognosis.—The lethargy usually clears up in one to three weeks. In 40 per cent. of cases the disease is fatal. The outlook is always very serious, owing to the liability to (1) residual symptoms or (2) sequelæ (Post-Encephalitis). In the report of Parsons, published by the Ministry of Health (1928),[1] one-third of the cases died, and one-third were so seriously disabled as to be unable to continue their ordinary work. The post-encephalitic phenomena may appear as long as nine years after the original signs of the disease. The commoner manifestations of Post-Encephalitis are discussed in other sections, but in order of frequency they are: (1) Parkinsonism, with its attendant bizarre symptoms (§ 765); (2) mental symptoms, especially in children (see § 907); (3) sleep disturbance, either lethargy or insomnia; (4) oculo-gyric crises and respiratory tics; (5) adiposity, polyuria and polydipsia, hyperthyroidism, extreme cachexia, excessive salivation and sebaceous secretion

[1] "Reports on Public Health and Medical Subjects," No. 49, London, 1928.

are all met with, due to hypothalamic involvement. The ocular symptoms responsible for diplopia are usually evanescent, but the pupillary abnormalities and slight ptosis are often permanent, and, in the later stages, afford valuable diagnostic marks of the disease. The spontaneous movements of chorea or myoclonus usually clear up after months, but tremor tends to stay.

Etiology.—No *age* is exempt. The virus of epidemic encephalitis is a filter-passing organism, but so far it has not been observed or cultivated.

Treatment of Acute Symptoms.—A $2\frac{1}{2}$ per cent. solution of sodium salicylate in normal saline has been injected intravenously in doses of 30–50 c.c., twice or once daily, for a week or ten days. Drugs, however, are not specific and their value is unproved. The mouth, nasopharynx and nose should be swabbed or sprayed four-hourly with 1 : 1000 potassium permanganate solution. Lethargy, if profound, is treated by *lumbar puncture* daily, or by 20–30 c.c. of 15 per cent. hypertonic saline solution intravenously, injected daily. Constipation should be relieved by enemata, and bowel wash-outs may be given on alternate days, with improvement in the general condition. The patient should be kept in bed for at least two weeks after the disappearance of constitutional symptoms or focal nervous symptoms, and should not be allowed to return to his work for a further period of six months.

D. **Disturbances of Sleep Rhythm.**—In *Encephalitis Lethargica*, especially in children, the patient falls into a heavy sleep during the day but at night-time becomes wakeful and restless, often destructive, *e.g.*, tearing the bedclothes into shreds.

§ **699.** In **Narcolepsy** the patient is periodically overcome by an irresistible desire to sleep. He can be roused, but, if left undisturbed, may slumber for half an hour or longer, waking to normal consciousness. Dreams may occur. The attacks may occur several times a day and the depth of sleep varies, as in normal sleep. There is often abnormal slowness in waking up after normal sleep. Associated with this is a phenomenon known as *Cataplexy*. Whenever the patient feels a strong emotion, after hearty laughter, after anger, or when intensely interested in something, a sudden weakness overcomes him and he sinks helpless to the ground, retaining full consciousness but unable to utter a sound. The whole attack lasts only a second or two, and, during this, the knee-jerks are abolished and the plantars are extensor in type. Narcolepsy occurs as an idiopathic disease; it may occur as a sequel of encephalitis lethargica, in cases of idiopathic epilepsy, after head injuries and in cerebral tumours, especially in the hypothalamic region. Patients with obesity and genital atrophy may suffer from narcoleptic attacks.

Treatment.—The patient should be advised not to drive any vehicle, swim or ride, or work at a height from the ground. Ephedrine hydrochloride (or sulphate) in $\frac{1}{4}$ to $\frac{1}{2}$ grain doses twice daily has been recommended in the treatment of this condition. Caffeine citrate 5 grains may be prescribed twice daily to enhance the effect of ephedrine. Amphetamine sulphate, 10 mgm. given on waking or at midday, will in many cases avert the sleep attacks. Continuous daily administration of this substance over long periods is not advised. All these drugs should be given as tablets on rising and at midday. If taken in the evening they are likely to interfere with the patient's normal sleeping.

VI. Subjective feelings of **Uselessness, Loss of Power** or **Inability to control** one or more limbs are met with in the slighter degrees of paresis, or when there is loss of sense of position. Such feelings, together with

other subjective sensations, *e.g.*, diplopia, precipitancy of micturition, are characteristic of organic neurological disease.

PART B. CLINICAL INVESTIGATION

§ 700. The acquisition of a proficient technique in examining patients, and routine in applying it, is absolutely essential for solving neurological problems. The examination must be thorough and accurate, otherwise the deductions of the examiner will be erroneous. Experience in neurological disease never absolves one from the necessity of careful examination, for the case history, without the physical signs, may be entirely misleading. A sign such as hemianopia may be carelessly missed by even an experienced observer.

General Considerations.—Ambulatory patients should be examined seated on a stool in a good light. In eliciting reflexes it is advisable to have good muscular relaxation, and the patient should lie on a couch during this part of the examination. When seen in bed the patient should be asked to walk, whenever possible, on the ward or bedroom floor for inspection of the gait.

In writing case-notes on neurological cases remember the following points: (1) *Put down your observations categorically* and systematically under the various headings. (2) *Never omit to record negative as well as positive findings:* if the pupils are equal, central, circular, and react to light directly and consensually, say so. It may be as important in the final diagnosis as the finding of a fixed pupil. (3) Only certain abbreviations are permitted. These are SJ, BJ, TJ, KJ, AJ, for supinator, biceps, triceps, knee-jerk and ankle-jerk, respectively. AC and RC may be used for ankle- and rectus-clonus. + signifies a reflex elicited, + + an exaggeration of a reflex. O an absent reflex. The signs > greater than, < less than, are also permissible (*e.g.*, K.J.'s $\underline{R} > \underline{L}$). In sensory testing C.W., P.P., V.S., J.S., and T° are used for cotton-wool, pin-prick, vibration sense, joint-sense, and temperature respectively. $\underline{R}$ and $\underline{L}$ underlined, are used to indicate Right and Left; thus $\underline{R}$.V. = 6/6, the visual acuity of the Right eye is six-sixths.

The patient's surname should be printed in block capitals at the head of the case-sheet; the address, age and occupation should be recorded, and whether married, single or widowed. No neurological examination is complete without a full examination of other systems, including the urine. Progress notes should always be made. In the majority of cases a spinal puncture with examination of the cerebro-spinal fluid will be necessary.

The ordinary methods of clinical examination—palpation, percussion, are not available in examining the nervous system. Special methods are used, requiring special tools, and the observer should provide himself with one of the heavier types of reflex-hammer, a tuning-fork (C 256) for testing vibration sense, and a good electrical ophthalmoscope. A skin pencil for marking out areas of diminished sensation is useful.

The History.—The *mode of onset* of the symptoms is of the greatest importance in diagnosing the cause of the lesion. Thus, a hemiplegia coming on in a few seconds is commonly due to a cerebral hæmorrhage or embolism, one coming on in a few minutes or hours, to a cerebral thrombosis, whilst a hemiplegia developing gradually over weeks or months is commonly caused by a slowly-forming tumour or abscess. In a chronic malady like disseminated sclerosis, the first symptom may have occurred in the patient's youth, and neurological histories often cover the major

part of the patient's lifetime. Events should be set down chronologically, using a fresh paragraph for recording the occurrence of each fresh symptom in the story. Experience is necessary to avoid inclusion of irrelevant matter. Important points may be missed if leading questions are not asked. The examiner should never omit to ask for a history of double vision, precipitancy of micturition, frequency or incontinence, since through shyness a patient may suppress important facts. A history of double vision, elicited in response to a leading question, should not be accepted unless the patient can recall what was the first object seen as a double image. It is often necessary to obtain additional history from *relatives* or *friends*, to supplement, corroborate, or amend the patient's story. In cases of epilepsy, we should, whenever possible, obtain a description of the attack from an eye-witness.

Previous History.—A history of syphilis or venereal disease—"a sore on the penis," should be inquired for in male patients. In women tact is necessary, and you have often to rely on a story of stillbirths, or the absence of pregnancies over a long period of married life. Rheumatism or other specific fevers, "influenza," aural discharge, tuberculosis, surgical operations, *e.g.*, for malignant disease, and accidents or wounds, especially to the head or spine, may all have neurological sequelæ and should always be inquired into.

Family History is particularly important in neurological disease, as some neurological diseases, *e.g.*, Friedreich's ataxia, are heredo-familial, whilst others, *e.g.*, Amyotonia Congenita, are familial. The patient should be asked regarding the occurrence of any nervous or mental trouble in his brothers or sisters, parents, uncles, aunts, cousins, etc. The health of his wife and of his family should be ascertained.

Habits and Occupation.—Inquiry should be made as to past residence abroad. If alcoholism is suspected, the patient's own statement must be accepted with "philosophic doubt." Tobacco addiction may cause a retrobulbar neuritis. The patient's occupation often has a close bearing on his illness.

THE EXAMINATION

§ 701. SCHEME FOR ROUTINE NEUROLOGICAL EXAMINATION. Build and general appearance. Temperature. Pulse rate. Body weight.

PSYCHICAL FUNCTIONS: (*and see Chapter XX*).

Intelligence, Attentiveness, Memory, Orientation, Emotional—Phobiæ, Hallucinations, Delusions—Sleep, Delirium, Coma.

SPEECH AND ARTICULATION.

Is the patient right- or left-handed? Aphasia, Apraxia, Articulation.

CRANIAL NERVES.

Smell—Visual acuity; Fields of Vision; Optic Discs and Fundi—Pupils; External ocular movements; Nystagmus; Oculo-pupillary sympathetic phenomena—Corneal reflexes; Sensation over face; Masseters, Temporals—Facial movements and symmetry of face—Hearing—Palate; Tongue; Sterno-mastoids and Trapezii.

MOTOR FUNCTIONS.

(1) Power; (2) Co-ordination; (3) Tonus; (4) Wasting and Fibrillation, Hypertrophy of muscles; (5) Involuntary movements and Fits.

EXAMINATION OF GAIT.

SENSORY FUNCTIONS.

Cutaneous Sensibility—(1) Touch (cotton-wool); (2) Pain (pin-prick); (3) Temperature.

Deep Sensibility—(4) Joint Sense; (5) Vibration; (6) Sensibility of Muscles and Tendons to Deep Pressure.

Stereognosis, Tactile Localisation, Compass Tests.

REFLEX FUNCTIONS.

Tendon Reflexes—Biceps, Triceps and Supinator Jerks.
Knee-Jerks and Ankle-Jerks. Presence of Clonus.

Cutaneous Reflexes—Epigastric Reflexes, Upper and Lower Abdominal Reflexes. Plantar Reflexes.

Visceral Reflexes—Micturition and Defæcation.

Tonic Reflexes—Kernig's Sign.

SPINE AND CRANIUM.

Deformities or Tenderness.

TROPHIC CHANGES.

Skin—Bed-sores, perforating ulcers.

Bones and Joints—Arthropathies, pes cavus.

EXAMINATIONS OF OTHER SYSTEMS.

SPECIAL EXAMINATIONS.

(1) Cerebro-spinal Fluid—Cells, Total Protein, Globulin, Wassermann Reaction and Lange Gold Curve.
(2) Blood—Wassermann Reaction.
(3) Radiological Examinations.
(4) Electrical Reactions of Muscles.

Psychical Functions.—You should note the patient's intelligence and attentiveness, his memory for recent and remote events, and whether he is orientated in space and time. In some cases it is necessary to test the reasoning power and to record delusions (*i.e.*, erroneous beliefs impervious to reason) or hallucinations, phobiæ (*e.g.*, fear of traffic), or obsessions. The *intelligence* and *attentiveness* of the patient can be gauged roughly when taking the history from the patient. For more accurate testing, especially in the case of children, Intelligence Tests, verbal and performance, are used.[1] Memory for recent and remote events may be tested by obtaining confirmation of the patient's history from a reliable relative. *Memory span* is tested by asking the patient to repeat six digits, *e.g.*, 4—7—1—9—5—2, or to repeat a sentence of 28 syllables, *e.g.*, "Walter likes very much to go on visits to his grandmother, because she always tells him many funny stories." *Orientation* in space is tested by asking the patient, "What place is this?" "How did you come here?" etc., and in time, by asking, "What month (or year) is this?" "How long have you been here?" Aphasia or apraxia due to organic brain lesions should never be mistaken for mental disease. (See §§ 743, 745.)

§ 702. Speech and Articulation.—(*a*) *Aphasia and Apraxia.*—The tests for these are complicated and are described in §§ 743, 745. In certain cortical lesions the

[1] The *Progressive Matrices* test (Raven) requires no vocabulary, and will be found useful for older children and adults: vide *Progressive Matrices and the Mill Hill Vocabulary Scale*, by J. C. Raven (H. K. Lewis & Co.): and *Performance Tests of Intelligence*, by J. Driver and M. Collins (Oliver and Boyd Ltd.).

patient has difficulty in translating his thoughts into words, either spoken or written, or difficulty in comprehending spoken or written speech. This is aphasia. An apraxic patient recognises objects, *e.g.*, a key or a pipe, but cannot demonstrate how to use them, although he is aware of their proper use and may not be paralysed.

(*b*) *Articulation.*—This has to do with the peripheral speech mechanism. Dysarthric patients have difficulty "in getting their tongues round words." This is tested by getting the patient to repeat certain catch-phrases, *e.g.*, "Mutual eligibility," "West Register Street," "Baby Hippopotamus."

(*c*) The occurrence of speech areas in the left cerebral cortex in right-handed people, and in the right hemisphere in left-handed people, renders it necessary that you should know whether the patient is right or left-handed.

§ **703. Cranial Nerves.**—I. Olfactory.—You ask the patient to close his eyes. *Each nostril* is tested separately, by applying a smelling-bottle of peppermint or lavender to the nostril tested and closing the other with your finger. It is sufficient if the patient can recognise differences in odour. Odours need not be named specifically. Ammonia, or substances containing it, such as smelling-salts or sal volatile should not be used, because they stimulate the fibres of the trigeminal in the nasal mucosa—(common sensation). Ask the patient "Do you smell anything?" "Of what does this smell?" "Is it different from this?"

II. Optic.—You must make three examinations:

(*a*) Visual acuity.

(*b*) Fields of vision—peripheral and central.

(*c*) Ophthalmoscopic examination.

(*a*) *Visual Acuity.*—This is tested with Snellen's test types. The patient is placed 6 metres from the card and asked to read the letters from the top as far down as he can. *Each eye is tested separately* (see § 834).

(*b*) *Fields of Vision.*—You sit exactly opposite the patient, so that your eye is at a distance of about one metre from that of the patient. In examining the right field the patient's left eye is covered. You then say, "I want you to look directly at my left eye," pointing to your left eye and closing your right eye. Now, fixing the patient's pupil with a steady gaze so that you can note any deviation of the eye you are examining, hold your clenched hands almost at arms' length at a place midway between yourself and the patient. Now tell the patient, "I want you to point to anything you see moving." Move the thumb of one hand rapidly once, and if the movement is seen, the patient will point to the moving object. The moving thumbs are in this way brought from the periphery of the visual field towards the centre, testing all four quadrants of the visual field. You can thus compare the patient's field with your own; if there is a defect he does not see the moving finger at a time when you yourself see it, provided your own visual field is full. If any defect is suspected a careful perimetric chart should be made with a reliable perimeter. *Central vision* is tested with a 5 mm. white or red object, on a black rod held in the centre of the visual field, midway between one's own and the patient's eye. For convenience, a fragment of blotting-paper stuck in the nib of a pen is often used. If a central blind spot or *scotoma* is present the patient will not see the object until it is moved radially outwards. If a field defect exists it should be charted accurately by Mechanical Perimetry or Bjerrum's Screen (§ 834).

(*c*) *Ophthalmoscopic Examination.*—The optic discs and retinæ must be examined in every case of nervous disease. Every physician must know how to use an ophthalmoscope, and to recognise papillœdema, optic atrophy, and the commoner pathological appearances in the retina and its vessels (see § 848).

III, IV, VI. Oculomotor, Trochlear, Abducens.

These nerves supplying the internal and external ocular muscles are conveniently examined together. The *Oculomotor* supplies the superior, inferior and internal recti and inferior oblique, the striped muscle of the levator palpebræ superioris, and contains efferent autonomic fibres supplying the tonic constrictor fibres of the sphincter

pupillæ and ciliary muscle. The *trochlear* supplies the superior oblique muscle and the *abducens* the external rectus alone. The *Cervical Sympathetic* supplies the tonic dilator fibres of the pupil, the unstriped part of the levator palpebræ superioris and unstriped muscle at the back of the orbit.

(*a*) *Pupils.*—You must note if the pupils are equal, central, circular, oval, or irregular in outline. Observe their reactions to light, tested directly, consensually and on accommodation. Examine the external ocular movements for paresis and diplopia, look for nystagmus and note any inequality in the size of the ocular fissures, proptosis or enophthalmos (sinking of the eye into the socket). *The normal pupil* dilates to shade, and contracts briskly when light falls on the same eye (direct reflex) or on the opposite eye (consensual reflex). The *light reflex* is best tested by covering and unshading first one eye of the patient and then the other as he looks at the light. Each eye must be observed separately. Watch the effect on the pupil of shading and uncovering the opposite eye. Another way of testing the pupillary reflex to light is: turn the patient's face away from the light and (observing one pupil at a time) throw a beam of light from an electric torch first into the one eye and then into the other. Loss of direct reflex to light with preservation of pupillary contraction to accommodation constitutes the *Argyll-Robertson phenomenon*. Test the *accommodation-convergence reaction* (near reflex) of the pupils by asking the patient to look first at a distant object in the room or out of the window, and then suddenly to look at your forefinger which is held about a foot from his eyes. The normal pupil contracts briskly as the eyes converge.

Tonic pupils (Saenger) contract very slowly on accommodation. The contraction is long sustained and may last for half a minute or more. *Fixed pupils* react neither on accommodation nor to light stimuli.

(*b*) *External Ocular Movements.*—Steady the patient's chin with your left hand, in order to fix the head. Then raise the forefinger of your right hand at least one metre from the patient's eyes and say, "I want you to follow my finger closely with your eyes." Move the finger to the extreme right, and pause in order to see if there is lack of movement in one or other eye, or the presence of nystagmus. Test both eyes together, making the patient follow the finger to the extreme left, pausing again, and so also upwards and downwards. Ask in each case if the vision is clear or blurred.

(*c*) *Nystagmus* is an involuntary rhythmic oscillation of the eyeballs (§ 847) usually appearing when the gaze is directed to a fixed point, *e.g.*, the examiner's finger, at a distance from the rest-point of the eyes. This is "fixation" nystagmus, in contrast to the much rarer nystagmus which occurs when the eyes are directed straight forwards. The movement may be quicker in one direction than in the other. The *quick phase* is taken to indicate the direction of the nystagmus. Nystagmus may be *coarse* or *rapid*, *horizontal*, *vertical* or *rotatory*. You should learn to recognise the infrequent blinking component of the "Parkinsonian Mask."

V. Trigeminal.—This nerve supplies sensory fibres to the anterior part of the scalp, eyes, face, nose, mouth, and parts of the ear and tongue, as well as the dura mater (see § 856). The motor root supplies the muscles of mastication, masseter, temporals, pterygoids, mylohyoid, anterior belly of digastric, tensor palati and tensor tympani muscles.

You must make three examinations: (*a*) corneal reflex, (*b*) sensation over face, (*c*) the muscles of mastication.

(*a*) The *corneal reflex* is tested by lightly blowing on the cornea and watching for the bilateral reflex blinking, or by asking the patient to look upwards and touching the cornea from below with a long pointed strand of cotton-wool. The reflexes should be compared on the two sides. Care should be taken to touch the cornea, not the conjunctiva. (*b*) *Sensation over the face* is tested with cotton-wool, pin-pricks and tubes of hot and cold water. Where there is diminution or loss of sensation, this is mapped out with a skin pencil, working from the dull to the sensitive area. (*c*) *The muscles of mastication* are tested by asking the patient to clench his jaws, when the masseters and temporals can be palpated as they contract on the two sides. In

unilateral lesions, when the patient opens his mouth, the jaw deviates to the side of the lesion, being pushed over by the unantagonised external pterygoid muscle of the opposite side. Wasting of the masseters or temporals should be looked for.

VII. Facial.—Test the *voluntary movements* of both the upper and the lower face and the reflex *emotional movements*, *e.g.*, facial movements on smiling. Taste on the anterior two-thirds of the tongue is conveniently tested with this nerve, as taste fibres for this part of the tongue are distributed with the chorda tympani (Fig. 193).

Voluntary movements of the *upper face* are tested by asking the patient to wrinkle up his eyebrows and screw up his eyes. Slight degrees of weakness can be observed by the difference in burying of the eyelashes on the two sides, or by comparing the effort needed to open with your thumb the screwed-up eyelids. Voluntary movements of the *lower face* are tested by asking the patient to show his teeth, blow out his cheeks, or whistle. Slight degrees of facial weakness are shown by widening of the ocular fissure on the affected side, and flattening of the nasolabial fold. In upper motor neurone lesions only the lower face is affected.

Emotional movements are tested by asking the patient to smile and noting the difference in the angles of the mouth, or they may be observed during the general examination. *Taste* is best tested with a weak galvanic current; the wire electrode on the taste-sensitive areas produces a metallic taste. Or the patient is asked to protrude his tongue and to keep it out, and to nod if he tastes anything. Powdered sugar, salt, citric acid, are then rubbed on the tongue with a clean glass rod. If the patient tastes it he nods and is allowed to withdraw the tongue and describe the taste.

VIII. Auditory.—The sensory or cochlear component subserves *hearing*, the vestibular non-sensory component subserves reflexes concerned with equilibrium. (*a*) Before testing *hearing* examine the external auditory meatus to make sure that it is not blocked with wax. Whispered voice sounds are used, *e.g.*, "Charing Cross," "Waterloo," at a distance of one metre. Test the ears separately, the opposite ear being rapidly opened and closed by intermittently pressing the tragus into the meatus with your forefinger. Objectively a lesion of the nerve (nerve-deafness) may be differentiated from middle-ear deafness by the following tests: *Weber's Test:* You place the base of a vibrating tuning-fork (C 256) on the patient's forehead. In nerve deafness the sound is best heard by the patient on his normal side, while in middle-ear deafness, he hears it best on his affected side. The *Rinne Test:* Air-conduction is compared with bone-conduction. Normally, a vibrating tuning-fork is heard better when it is held slightly away from the meatus than when its base is placed on the mastoid of the ear tested. In nerve deafness both air and bone-conduction are reduced; in middle-ear deafness the fork is heard better when it is placed on the mastoid of the deaf ear.

(*b*) Testing the vestibular part of the nerve is described in § 860.

IX, X, XI. Glossopharyngeal, Vagus, and Spinal Accessory.

These nerves are intimately related in their central connections; they leave the skull by the jugular foramen and are conveniently tested together. (*a*) *Sensory tests:* Tickling the soft palate or posterior pharyngeal wall with cotton-wool on the end of a probe will normally produce reflex movements (Glossopharyngeal). Fibres of the glossopharyngeal nerve supply taste to the posterior third of the tongue. (*b*) *Motor Tests:* (1) *Palate:* The patient is asked to open his mouth and say, "A-Ah." When the patient phonates the soft palate will rise in the mid-line; if one side is paralysed the palate will be deviated to the sound side. Bilateral palatal palsy produces a characteristic nasal intonation, with regurgitation of fluids through the nose, on swallowing. (2) *Pharynx:* In unilateral paralysis, the posterior pharyngeal wall moves like a curtain pulled over to the sound side ("curtain movement") when the patient phonates. (3) *Larynx* (see § 164). (4) *Sterno-mastoids and Trapezii:* Examine the sterno-mastoids by asking the patient to turn his head forcibly to the right and then to the left, or by asking him to push his forehead downwards against

the resistance of the palm of your right hand. The trapezii are tested by asking the patient to shrug his shoulders up to his ears against the resistance of your hands placed lightly on his shoulders. In all cases the muscles should be inspected and palpated for wasting.

The visceral functions of this group of nerves are described in the examination of the other systems.

XII. Hypoglossal.—Ask the patient to protrude his tongue and to push it first into his right cheek and then his left. The protruded tongue is examined for spasticity, fibrillation, atrophy, or wrinkling. If one side of the tongue is paralysed and the patient attempts to protrude the organ, the tip is pushed round to the paralysed side, in a sickle-shaped curve, by the healthy side.

§ 704. Motor Functions.—It is advisable to have the patient in pyjamas or stripped in a dressing-gown. First, you examine the upper limbs, then the trunk, and then the lower limbs, and the results are recorded in that order. You should note especially if the limbs are steady and strong; also examine their tonus, and look for wasting and fibrillation or hypertrophy of muscles.

(1) Power.—The hand-grips are conveniently tested by crossing one's forearms and giving your three middle fingers, shaped in the form of a cone, to the patient to squeeze as hard as he can. The flexors of the fingers can be tested by asking the patient to hook the flexed fingers of his right hand round the flexed fingers of your right hand and then you attempt to extend his fingers against resistance. The dorsiflexion of the feet is similarly tested against resistance by asking the patient, when recumbent, to cock his feet upwards. You then attempt to plantar-flex them whilst he resists. In these tests the power is compared on the two sides. Each joint and each movement should be tested separately, fixing the proximal part of the limb and instructing the patient to perform various movements—flexion, extension, rotation outwards, inwards, etc., separately. If the weakness is marked, support the limb in the optimum position for action of the particular muscle or group of muscles you are investigating. Inability to relax the handgrip for some seconds is characteristic of *tonic innervation* or *myotonia*.

(2) Co-ordination.—In the "finger-nose" test the patient, keeping his eyes open, moves his forefinger alternately to his nose and then to the examiner's finger which is held about one metre in front of him. He is asked to repeat this movement several times. When "intention tremor" is present the movement is jerkily performed and a coarse oscillation of the forearm and hand appears just before the objective is reached. When ataxia is present the finger misses the nose by a greater or smaller interval. You should notice if the unsteadiness is increased when the patient shuts his eyes. Co-ordination of the fine finger movements is tested with the "thumb-finger" test, the patient being asked to touch the tip of each finger in rapid succession (beginning with the fourth) to the tip of the thumb of his same hand. Other useful tests are picking up a pin, fastening the buttons of a coat, etc. In the "heel-knee" test the patient is placed in the recumbent position and is asked to place the heel of one foot on the opposite knee and to slide the heel slowly down the front of his shin. Here, too, you should notice if ataxia is increased on shutting the eyes. Other tests in the lower limbs are made with examination of the *gait*. Special cerebellar tests are described in § 812.

(3) Tonus.—*Hypotonia* is tested by shaking the relaxed limbs like a flail and comparing the resistance of the two sides, or attempting to fling the patient's hand on his chest. In another test the hands are passively dorsiflexed and the angle made with the forearm on the two sides compared; the knee-joints are passively hyperextended, etc. *Hypertonus* or rigidity is of three main types: (*a*) "Clasp-knife" rigidity; (*b*) "Cog-wheel" or "Lead-Pipe" rigidity; (*c*) Hysterical Spasm.

(*a*) "Clasp-knife" rigidity is seen typically in a residual hemiplegia. The limb is flexed at the elbow and pronated. When you attempt to undo the flexion, resistance is encountered until the elbow is almost extended, when the resistance suddenly "gives" as in the opening of a clasp-knife. This is characteristic of pyramidal disease. (*b*) "Lead-pipe" or "Cog-wheel" rigidity is characteristic of extra-pyramidal disease and is seen typically in Parkinsonism (§ 765). When you attempt to extend the elbow-joint, resistance is felt like the bending of a piece of lead-piping. Or on attempting to flex and extend the patient's wrist-joint, a feeling like turning a cogged wheel is met with. (*c*) In Hysterical Spasm the prime-movers and antagonists are simultaneously innervated by the patient so that little or no movement results—*e.g.*, when he attempts to use his hamstrings, the quadriceps, instead of relaxing reciprocally, tighten up. The spasm increases with the amount of force used to overcome it.

(4) Wasting and Fibrillation. Hypertrophy.—By inspection and palpation you observe if the muscles are increased in bulk (hypertrophy) or wasted (atrophy). Sometimes hypertrophied muscles are feeble in their performance (pseudo-hypertrophy). The names of the affected muscles are recorded. You should carefully inspect all aspects of the limb and the back as well as the front of the patient. Whenever wasting is present you should look for *fibrillation* in the atrophied muscles. This is a flickering or quivering of muscle-fibres or bundles, best seen when the muscles are relaxed. Direct percussion of a muscle with a reflex hammer may show in certain pathological conditions the appearance of a dimple, which is persistent for some seconds—*myotonia on percussion.*

(5) Involuntary Movements.—*Tremor* is best seen when the arms and fingers are extended. It may be coarse or fine. Tremor never exists in flaccid limbs; it usually ceases during sleep, and can often be controlled by an effort of will. *Choreic movements* are irregular, non-rhythmic, spontaneous but purposeless movements of groups of muscles. In organic disease they are more marked on one side than the other, and in the facial muscles they occur bilaterally. *Athetoid Movements* (athetosis) are slow, irregular, writhing movements, seen in hemiplegic limbs, more often in children than in adults (Fig. 177). The limbs are never completely paralysed: the movements are more marked peripherally and usually greater on one side than the other, and are bilaterally distributed in the face. The movements are intensified by emotion and by voluntary movements, and, between the accesses of mobile spasm, the limb is generally hypotonic. *Myoclonus* is a sudden shock-like contraction occurring regularly or irregularly in various muscles. *Tics* are sudden clonic jerks of a stereotyped character occurring in people of neuropathic constitution, increased by emotion.

(6) Electrical Reactions of Muscles.—These are described in § 708.

§ 705. Examination of the Gait.

Except in patients confined rigidly to bed the gait should always be examined. It is frequently forgotten by students. The patient is asked to walk away from you to a given point, to turn round and then come back. It is advisable to pull or pin up the clothing or pyjama-trousers so that as much as possible of the legs can be seen. The patient walks on a strip of carpet with bare feet. You must look for dragging of the lower limbs, reeling or tottering, especially in turning, whether the patient deviates from the straight line and, if so, to what side he deviates, and whether he swings both arms as he walks. Where cerebellar or posterior column disease is suspected you ask the patient to heel-and-toe a straight line, to stand or hop, first on one leg and then on the other. Where myopathy is suspected the patient (usually a child) is laid flat on the floor and asked to stand upright. The various motions performed in

accomplishing this are characteristic. The following gaits can be recognised clinically :

(1) In *Spastic Gait* the toes are dragged on the ground. This occurs in pyramidal disease, *e.g.*, disseminated sclerosis, residual hemiplegia. Where extensor rigidity is combined with adductor rigidity, a "cross-legged" or "scissor-gait" is met with, the patient walking on the toes, *e.g.*, in cerebral diplegia in children.

(2) *Spastic Ataxic Gait* is met with in combined disease of the pyramidal tracts and posterior columns, *e.g.*, disseminated sclerosis, subacute combined degeneration, spinal tumour.

(3) *Ataxic or Reeling Gait* occurs in cerebellar, vestibular, or posterior column disease, *e.g.*, cerebellar tumour, Friedreich's hereditary ataxy, Ménière's disease, or tabes dorsalis.

(4) *A Festinant or Shuffling Gait*, in which the patient glides forward with little running or shuffling paces, occurs in striatal disease, *e.g.*, Parkinsonism. In this condition the normal swing of the arms is lost and the patient turns *en bloc*. It is seen in the general muscular rigidity of cerebral arterio-sclerosis.

(5) *Waddling Gait* is met with in conditions of weakness of the pelvic muscles, *e.g.*, myopathy ; or from deformities, *e.g.*, congenital dislocation of the hip, or dwarfism.

(6) *High-stepping Gait* occurs in conditions of foot-drop, *e.g.*, polyneuritis ; or from loss of joint sense, as in tabes, in which the feet are lifted high and the heels banged on the ground.

(7) *Jaunty Gait* is met with in chorea.

(8) *Limping Gait* may result from poliomyelitis or any injury or joint affection confined to one side.

(9) *Bizarre Gaits*, in which the patient walks with bent knees or trunk, or in zig-zag fashion, are seen in hysteria. A gingerly insecure gait, where the patient seeks support from the walls, furniture or the observers, is common in that disease.

§ 706. Sensory Functions.—In order to make your sensory testing accurate it is desirable to gain the intelligent co-operation of the patient, and each test should be briefly explained before it is carried out. The room should be as quiet as possible and the patient's eyes closed in order to shut out extraneous stimuli.

The following rules should be observed : (1) Test each variety of sensation separately over the whole body before proceeding to the next ; (2) Always compare the sensibility over corresponding areas on the two halves of the body ; (3) Whenever possible chart the findings on an outline diagram ; (4) Avoid suggesting the presence of a sensory change to the patient by the manner in which commands or questions are put. The following are useful approaches which suggest little to the patient : "Shut your eyes and say 'Yes' every time I touch you" (C.W.). "What does this feel like ?" (P.P.). "Is there any difference in the feeling here, and here ?" (P.P.).

CUTANEOUS SENSIBILITY.—(1) *Touch* is tested with a wisp of long-fibred cotton-wool. In mapping out anæsthetic areas, proceed from the area of impaired sensibility towards normal skin and from below upwards on the trunk. The point at which sensation becomes normal is recorded on the skin by a single dot made with a skin pencil. A number of these dots are made, and they can subsequently be connected, as in a graph, to outline the area. An anæsthetic area so mapped out should be recorded on the chart and shaded. Vertical hatching is used conventionally in diagrams for recording impairment of touch.

(2) *Pain* is tested with the prick of a sharp pin. In order to obtain a uniform stimulus the pin should be held with the point just projecting between the pads of the thumb and middle finger. *Dragged pin* is used to map out areas of hyperæsthesia, the pin being lightly dragged across the skin from the less to the more sensitive area. Loss of pain sensation is termed *analgesia* and is represented in outline diagrams conventionally by horizontal hatching; *hyperæsthesia* or exalted sensibility to pain is represented by a series of small crosses. The distinction should be made between "impaired sensibility" and "total analgesia." In mapping out sensory levels on the trunk remember that the segmental distribution of the spinal nerve roots runs downwards anteriorly, and upwards posteriorly, towards the mid-line. Horizontal upper levels of sensory loss are found only in hysteria and are produced by suggestion. The hysterical nature of a totally anæsthetic area may sometimes be demonstrated by Janet's "Yes-No" test. The patient is instructed to close his eyes and say "Yes" every time he feels a pin-prick and "No" every time he does not feel it. In hysteria the patient will say "No" every time he is touched over the apparently totally anæsthetic area, thus demonstrating that he is really able to feel but does not comprehend that he feels.

(3) *Temperature* is tested with tightly-corked test-tubes of cold and warm water (60° C.). The results are recorded on outline diagrams using conventional oblique hatching (Rt. to Lt. = Loss to Hot; Lt. to Rt. = Loss to Cold). Loss of sensation to temperature is called Therm-anæsthesia. In centrally situated diseases of the brain-stem and cord, such as Syringomyelia, the patient may feel the lightest touches with cotton-wool but cannot appreciate painful or thermal stimuli—this was called by Charcot "*dissociated anæsthesia*."

DEEP SENSIBILITY.—(4) *Joint sense* comprises the sense of passive movement of the joint surfaces on one another, and the sense of position. In testing, the patient closes his eyes, or they are covered up. In order to avoid sensations of pressure, you grasp the digit you are testing, *laterally*, with your thumb and forefinger. *When testing sense of passive movement* wait a few moments, then move the joint gently, having previously asked the patient to say "Yes" when he feels any movement. In testing *sense of position* ask the patient to say "Up" or "Down" every time you move the joint. You then passively move the joint in either direction, waiting for the patient's reply after each movement. Corresponding fingers and toes are tested on the two sides.

(5) *Vibration* is tested by placing a low-pitched tuning-fork on the radial styloids and tibial malleoli. Other bony prominences may be used. Vibration sensibility may be diminished or lost very early in disease of the posterior columns or posterior roots.

(6) *Sensibility of Muscles and Tendons to Deep Pressure.*—An increased sensitiveness of the calf muscles to deep pressure of your thumb is commonly met with in polyneuritis—*i.e.*, the *deep muscular sensibility* is increased. In tabes dorsalis, as an early sign, the normal sensibility of the tendo Achillis to pressure may be diminished or lost (Abadie's sign).

Stereognosis is tested by placing various objects—*e.g.*, key, coin, rubber, in the hand or against the sole of the patient, whose eyes are closed, asking him to describe the shape, size and consistency of the object used. For true astereognosis to be present, cutaneous sensibility in the hand or foot tested should not be impaired. *Tactile localisation* (topognosis) is tested by asking the patient, who has his eyes closed, to point to where he has been touched with a pin. Normally the localisation

is exact to a fraction of an inch. *Compass Tests* are performed with small blunt-pointed calipers. The patient, who has his eyes closed, states whether he has been touched with one or two points, while the distance between the limbs of the calipers is gradually narrowed.

§ 707. Reflex Functions.

In routine examinations you must test the TENDON REFLEXES, certain CUTANEOUS REFLEXES, including the PLANTAR REFLEXES, and you must note the condition of certain VISCERAL REFLEXES, *e.g.*, micturition, defæcation. TONIC or POSTURAL REFLEXES, *e.g.*, Kernig's Sign, are also used in clinical diagnosis.

1. **Tendon Reflexes.**—These are elicited by percussing the tendons of insertion of certain muscles.

	Method of Eliciting.	*Response.*	*Segmental Distribution.*
Biceps-Jerk.	Tapping biceps tendon.	Biceps contracts.	C5–6.
Triceps-Jerk.	Tapping triceps tendon.	Triceps contracts.	C6–7.
Supinator-Jerk.	Tapping above radial styloid.	Supinator longus contracts.	C6–7.
Knee-Jerk.	Tapping patellar tendon.	Vastus internus etc. contract.	L2–4.
Ankle-Jerk.	Tapping tendo Achillis.	Calf muscles contract.	S1–2.

In UPPER MOTOR NEURONE disease these reflexes are exaggerated and may be accompanied by sustained *clonus*, a rhythmic series of involuntary muscular contractions, produced by the sudden stretching of the tendon. In LOWER MOTOR NEURONE disease and in the MYOPATHIES, the deep reflexes are diminished or abolished in the affected muscles.

The *Biceps-Jerk* (C5–6) is elicited by supporting the patient's forearm with his elbow loosely flexed. Your thumb is placed over the biceps tendon and the thumb percussed with a hammer. The resultant jerk of the biceps is both felt and seen. The *Triceps-Jerk* (C6–7) is then investigated by abducting the patient's arm loosely and percussing the triceps tendon. The *Supinator-Jerk* (C6–7) is elicited by tapping just above the radial styloid, the patient's forearm supported in a semi-supinated position, the elbow loosely bent to a right angle. The resultant contraction of the supinator longus and flexors of the elbow is looked for. In testing the knee-jerks and ankle-jerks it is best to have the patient in the recumbent position to ensure muscular relaxation. To elicit the *Knee-Jerks* (L2–4) the recumbent patient's knees are slightly flexed, resting loosely on your arm, while you percuss the patellar tendons on the two sides and look for the resulting contraction of the vastus internus. To elicit the *Ankle-Jerk* (S1–2) the lower limb is then rotated outwards at the hip and the knee-joint slightly flexed, the sole is grasped and slightly dorsiflexed to stretch the tendo Achillis and the tendon percussed. If difficulty is encountered in eliciting the ankle-jerks in this way the patient should be asked to kneel on a *padded* chair with the calf muscles relaxed and the feet dangling over the edge. When the tendo Achillis is percussed a brisk contraction of the calf muscles results, which can be felt as well as seen if you are slightly dorsiflexing the foot with your left hand to stretch the tendon. In eliciting deep reflexes the responses on the two sides should be compared with the greatest care and noted. Always use the *minimal stimulus*, especially when testing knee-jerks. Never use much force, otherwise no accurate comparison of

the reflexes on the two sides can be obtained. In testing sluggish knee- and ankle-jerks it may be necessary to use *reinforcement*. This is achieved by asking the patient to look to the roof and clench his hands tightly, or he is asked to attempt to pull apart the interlocked fingers of the two hands. The presence of sluggish deep reflexes may thus be evident.

RECTUS-CLONUS is elicited in spastic limbs by sudden downward traction on the patella, with the knee extended. ANKLE-CLONUS is elicited with the knee passively flexed; the ankle is then suddenly dorsiflexed by light upward pressure on the sole. True clonus is always sustained and accompanied by an extensor type of plantar response.

2. **Cutaneous Reflexes.**—These are elicited by stimulating certain areas of skin or mucous membrane. The cutaneous reflexes of greatest practical importance are the Plantar Reflexes and the Abdominal Reflexes. These must be tested in every case. In upper motor neurone lesions these cutaneous reflexes disappear. This is most strikingly seen in the disappearance of the abdominal reflexes, first the lower and then the upper, in pyramidal disease.

The EPIGASTRIC (Th6–8) and UPPER (Th8–10) and LOWER (Th11–12) ABDOMINAL REFLEXES are elicited by stroking the lower anterior chest wall and the upper and lower quadrants of the abdominal wall respectively on the two sides. These reflexes may not be elicited when the abdominal wall is obese or flaccid. In the abdomen of a healthy young adult they are constantly present. In pyramidal disease these reflexes are diminished, tire easily or are lost on the affected side. The lower reflexes go before the upper. An absent abdominal reflex is an important early sign of disseminated sclerosis. In suspected disease of the cauda equina or lowest segments of the cord you have to test three other cutaneous reflexes: the Cremasteric (L1–2), Bulbo-cavernosus (S3–4), and Superficial Anal (S4–5).

The CREMASTERIC REFLEX (L1–2) is the reflex drawing up of the testis, on downward stroking or firm pressure, applied on the inner side of the thigh. The BULBO-CAVERNOSUS REFLEX (S3–4) gives valuable information about lesions of the third sacral segment. It is elicited by placing one finger on the perinæum and pricking the dorsum of the glans penis. Normally, the bulbous urethra can be felt to contract briskly. The SUPERFICIAL ANAL REFLEX (S4–5) is obtained by watching for the contraction of the external sphincter when the skin of the perinæum is pricked.

A GRASP-REFLEX is elicited in the contralateral palm in lesions of the posterior ends of the first and second frontal gyri. To obtain this you draw your fingers across the patient's palm near the thenar eminence. The patient's fingers contract reflexly, and, when you attempt to withdraw your hand, the involuntary tonic contraction of the fingers increases, and may take seconds to relax.

The PLANTAR REFLEXES are of the utmost importance in clinical neurology, and the student should be thoroughly conversant with the correct technique of eliciting the responses. The patient should be in the recumbent position with the lower limb slightly rotated outwards at the hip and the knee slightly flexed. The feet should be comfortably warm. Normally, firm stroking along the *outer* border of the sole of the foot with a key or the end of a penholder produces flexion of the great toe—*flexor plantar response*. In pyramidal disease, however, when we stroke the outer margin of the sole there results a dorsiflexion of the great toe, associated with dorsiflexion and fanning of the other toes—*extensor plantar response* (Babinski). This extensor movement is part of a general withdrawal reflex of the whole lower limb from a painful stimulus and is always accompanied by an associated contraction of the hamstring muscles. When, therefore, the plantar responses are equivocal, the hamstrings should be palpated for contraction, while the sole of the foot is being stimulated.

	Method of Eliciting.	*Response.*	*Segmental Distribution.*
Epigastric.	Stroking lower anterior chest wall.	Epigastrium dimples.	Th6–8.
Upper Abdominal.	Stroking below costal margin.	Rectus abdominis contracts.	Th8–10.
Lower Abdominal.	Stroking above Poupart's ligament.	Obliquus abdominis contracts.	Th11–12.
Cremasteric.	Stroking inner side of thigh.	Testis is drawn up.	L1–2.
Bulbo-cavernosus.	Pricking dorsum of glans penis.	Bulbo-cavernosus contracts.	S3–4.
Superficial Anal.	Pricking skin of perinæum.	External anal sphincter contracts.	S4–5.
Plantar.	Stroking outer border of sole of foot.	Dorsiflexion of hallux with fanning of other toes, etc.	L5–S2.

The *flexor* plantar response is associated with plantigrade functions associated with standing and walking. It is a cortical reflex. In infants, who have not learned to walk, the normal plantar response is of the extensor type. This extensor reflex is of spinal origin, and, in later life when the infant learns to walk, is normally inhibited by cortical control. When the cortical control is removed by pyramidal disease the more primitive spinal extensor response is re-established. In pyramidal disease the receptive field for this reflex spreads over the skin of the whole limb, and the reflex can be obtained by pinching the skin almost anywhere on the lower limbs. An *extensor* response may be observed during sleep or deep coma from any cause, and after epileptic fits.

3. The **Visceral Reflexes** of clinical importance are those concerned with micturition and defæcation.

MICTURITION.—(*a*) Precipitancy of micturition is a frequent early symptom of cord lesions, *e.g.*, disseminated sclerosis, slow cord compression, etc. (*b*) Difficulty in commencing micturition and dribbling incontinence of urine, especially at nights, occurs in tabes dorsalis when the bladder is anæsthetic and distended. (*c*) Retention, with overflow dribbling, occurs in coma, or during the initial three weeks of spinal shock following an acute transverse cord lesion, *e.g.*, myelitis, fracture-dislocation; and in the later stages of total transverse cord lesions. (*d*) Involuntary periodic micturition may occur, in which the bladder empties reflexly but never completely. (See § 690 : innervation of the bladder.)

DEFÆCATION.—Incontinence of fæces is commonly due to anæsthesia of the rectum from paralysis of the afferent nerves from the rectum, *e.g.*, in tabes or lesions of the cauda equina. In such cases, the internal anal sphincter, if felt by rectal examination, is flaccid. In spinal cord lesions, above the spinal centre in the conus, the internal sphincter retains its tone, and there is intermittent rectal incontinence, the patient being aware of the passage of fæces. Mentally confused patients are commonly incontinent.

4. Certain **Tonic** or **Postural Reflexes** are used clinically. In cases of suspected meningitis or meningeal irritation, you test for reflex tonic

contraction of the hamstrings (Kernig's Sign) and posterior cervical muscles (Brudzinski's Sign). *Kernig's Sign* is a reflex tonic contraction of the hamstring muscles, made evident by passively flexing the hip to a right angle and at the same time extending the knee. *Brudzinski's Sign* is a tonic neck reflex. When the head is passively flexed on the chest the lower limbs become flexed at the hips and knees. In hemiplegia, rotation or lateral flexion of the head towards the paralysed side may cause extension of the paralysed limbs. Movement of the head to the normal side has the reverse effect. This is *Magnus and de Kleijn's tonic neck reflex.*

Spine and Cranium.—Examine the patient's spine and cranium for *tenderness* or *deformity.* Neglect of this procedure may lead to your missing the fact that a patient's paraplegia is due to early Pott's disease.

Trophic Changes.—The *skin* should be examined for perforating ulcers and bed-sores, and the *bones and joints* for arthropathies, *e.g.*, Charcot joints; and deformities, *e.g.*, pes cavus.

EXAMINATIONS OF OTHER SYSTEMS.

The Cardiovascular, Respiratory, Alimentary and Genito-Urinary Systems should be examined and the urine tested in all cases.

OTHER SPECIAL EXAMINATIONS.

(1) *Cerebro-spinal Fluid.* It is necessary to examine the spinal fluid in many cases of neurological disease, and you should thoroughly familiarise yourself with the technique of performing *Lumbar puncture* (§ 919). Manometric observations on the spinal fluid-pressure must be made during the puncture, and the effect of compression of both jugular veins duly noted. The naked-eye appearance of the fluid should be observed in all cases. In sending fluids to the laboratory the following tests are made as routine: (1) Cell-count, (2) Total Protein, (3) Globulin content, (4) Wassermann reaction, (5) Lange's Gold Reaction (Gold Curve). Where infection of the nervous system is suspected the spinal fluid must be examined bacteriologically as well as serologically and the chlorides and glucose content estimated. *Cisternal* (or *Ventricular*) *puncture* (§ 919) should not be practised by those unfamiliar with the technique: it is not without serious danger.

(2) *The Wassermann Reaction in the Blood* is often necessary in diagnosis.

(3) *Radiological Examination* of the Skull (stereoscopic) or Vertebral Column may be necessary. The technique of encephalography and ventriculography is outlined in § 829, and that of intracisternal injection of iodised oil in § 757. These examinations should always be made by experts only.

(4) *Electro-encephalography* may, in expert hands, provide useful information in cases of head injury, suspected epilepsy or cerebral tumour (§ 667).

§ 708. Electrical Examination of Muscles. The APPARATUS REQUIRED is a faradic and a galvanic battery. In a normal case the faradic (interrupted) current causes a muscular contraction which persists as long as the current is passing. The galvanic

(constant) current causes a contraction only when the current is made or broken, not when it is passing.

To *test the faradic response*, place the large electrode on the patient's chest, on the back of the neck, or some other indifferent region, and another electrode over the motor point of the nerve or muscle to be tested. If the current is too strong for estimating the finer degrees of difference, the operator should take this second electrode in one or other of his hands, and apply his well-wetted finger to the well-wetted skin of the patient. A knowledge of the motor points of nerve and muscles is helpful, but they can be discovered by applying the electrode at different points, and noting those where contraction is most easily obtained. The motor point of a muscle is near the point of entry of its nerve; that of a nerve is generally near its most superficial part. The electrodes and the skin should be very *thoroughly* wetted with salt and warm water. The patient should be placed in a good light so that both sides of the body can be seen equally. Having thoroughly moistened the skin over the part to be tested, as well as the corresponding region of the other side, ascertain first (by gradually sliding up the secondary coil) the *minimum* current necessary to produce a minimum contraction of the muscle or muscles on the healthy side. Then test the side suspected of disease with the same amount of current, to see if the same degree of contraction is produced; if not, what strength of current is requisite. The faradic contraction of a muscle can only be obtained through its nerve, so that when the nerve is completely degenerated, the muscle (though still contracting to the direct stimulus of galvanism) fails to respond to faradism. Hence when a muscle does not respond to faradism, but does respond to galvanism, we conclude that the nerve is profoundly affected. (See § 709.)

To *test the galvanic reaction* the electrodes are placed in the same position as before. For (*a*) *quantitative* alterations, compare the two sides as before, noting what amount of current (as indicated by the number of cells used, or the number of milliampères registered by the galvanometer) is required to produce a minimal contraction on both sides. (*b*) For *qualitative* alterations begin with the kathode (negative pole) placed on or about the motor point under investigation. To distinguish the poles, place the two wires in a glass of water; a lively production of hydrogen gas appears at the kathode. Or place both wires on a piece of wetted blue litmus paper, which becomes reddened around the positive pole (anode) from the liberation of oxygen. Close the current by means of the interrupting handle; the contraction obtained is known as the Kathodal Closing Contraction (K.C.C.). Next convert the electrode on the patient into the anode or positive pole by means of your reverser, and repeat the process of closing the current. The resulting contraction is called the Anodal Closing Contraction (A.C.C.). Normally, with the same strength of current and the same degree of wetting of the skin and pressure of the electrode on the skin, K.C.C. > A.C.C., or what amounts to the same thing, a greater strength of current is required to produce A.C.C. than K.C.C.

Muscular contraction with a galvanic current is only produced at the closing or opening of the current. The normal order of the contractions is as follows:

K.C.C. > **A.C.C.** > A.O.C. > K.O.C. (O.C. = Opening Contraction).

Abnormally, A.C.C. is equal to or greater than K.C.C., and the character of the contraction is altered.

§ 709. The **reaction of degeneration** (R.D.) differs in different stages. When a nerve is severed or is the seat of acute inflammation, after a preliminary increased response to both currents during the first two days, (*a*) the reaction is gradually lost to both currents during the ensuing ten days, the faradic reaction not being regained unless regeneration takes place.

(*b*) In the second or third week, and for some weeks afterwards, the galvanic reaction is restored, and Erb's REACTION OF DEGENERATION occurs in its complete form. It is characterised by—

(i.) No muscular contraction to faradism, however strong the current.

(ii.) A quantitative increased contraction to the galvanic current.

(iii.) The galvanic contraction, which in health is prompt and sharp, becomes sluggish, and often the response is better when the electrode is placed over the peripheral end of the muscle rather than over the motor point.

(iv.) No contraction is elicited by stimulating the motor nerve by either current.

(v.) Qualitative galvanic changes are usual: A.C.C. is equal to or greater than K.C.C.

(*c*) Two or three months later the galvanic contractility gradually disappears (unless regeneration is established), though it may happen that for one or two years A.C.C. can be obtained with a progressively increasing strength of current.

The electrical reactions of muscle are of use in diagnosis and prognosis.

(*a*) *In Diagnosis:* The presence of the reaction of degeneration indicates that there is a lesion in the lower motor neuron. The reaction is partial or complete, according to the amount of damage to the nerve cells or fibres. When there is a *partial reaction of regeneration*, the test is difficult to carry out, and much skill and experience is required to interpret the findings. In certain diseases characteristic types of reaction occur. In *Tetany* the excitability of the nerve and muscle is increased both to faradism and to galvanism. Diminution of excitability to faradism and galvanism is found in conditions of muscular wasting, *e.g.*, *Myopathy*, *Arthritic Muscular Atrophy*, *Disuse Atrophy*, and is usually proportionate to the wasting present. In *Myasthenia Gravis*, continued faradic stimulation rapidly tires out the affected muscles until, at last, no response can be obtained; after a period of rest recovery ensues. The galvanic reactions are unchanged. The Myasthenic Reaction is not present in all cases of the disease. During the paroxysms of *Family Periodic Paralysis*, the muscles respond neither to faradism nor to galvanism. In *Myotonia*, faradic stimulation results in a cramp-like contraction of the muscles which lasts some 5 to 30 seconds after the cessation of the current; galvanism produces a wave-like contraction which also persists after the current has been switched off.

(*b*) *In Prognosis:* In the case of *Facial Palsy* (Bell's Palsy) the electrical reactions are of prognostic value. If, in the third or fourth week after the onset, some faradic response is present, early recovery of function is indicated (*i.e.*, within three months). Where R.D. is present at the end of the fourth week after the onset, long delayed and only partial recovery is to be looked for, with the possibility of secondary contracture in the paralysed muscles.

In *Poliomyelitis* muscle-testing may be undertaken after the painful stage has subsided (usually three to four weeks after the first symptom). Affected muscles may be grouped into three classes: (*a*) Those which will recover; in these the electrical changes are but little altered. (*b*) Those which may recover; in these the electrical responses are typical, *i.e.*, the galvanic response is sluggish, though obtained with a weaker current than normally, while A.C.C.$>$K.C.C., and the faradic reaction is not quite lost. (*c*) Those which probably will not recover; in these there is no response either to faradism or galvanism.

PART C. DISEASES OF THE NERVOUS SYSTEM: THEIR DIAGNOSIS, PROGNOSIS AND TREATMENT

§ 710. Routine Procedure and Classification.

DIAGNOSIS.—(1) With a knowledge of the Applied Physiological Anatomy of the Nervous System and the Methods of Clinical Examination, it is possible to make an ANATOMICAL DIAGNOSIS of the localisation of the lesion.

(2) In order to make a PATHOLOGICAL DIAGNOSIS it is necessary to understand the natural history of diseases affecting nervous structures. These diseases may be classified according to the presenting clinical symptom.

A clinical classification has been used, as set forth in the following scheme:

If there is *Coma, Stupor or Lethargy*	Group I, § 711
If there are *Transient Losses of Consciousness, Fits or Convulsions*	Group II, § 719
If there is *Pyrexia with signs of Organic Nerve Disease*	Group III, § 724
If there is *Defect of Speech or Articulation* ..	Group IV, § 743
If the symptoms relate to the *Motor System* the presenting picture may be:—	
Spastic Paralysis	Group V, § 751
Parkinsonism	Group VI, § 765
Involuntary Movements	Group VII, § 770
Tonic Spasms or Cramps	Group VIII, § 777
Flaccid Paralysis or Muscular Wasting ..	Group IX, § 786
Ataxia or Inco-ordination	Group X, § 810
If the symptoms are those of a *Sensory or Painful Disorder*	Group XI, § 816
If there is *Progressive Headache and Vomiting* ..	Group XII, § 827
If the *Cranial Nerves* and *Special Senses* are affected	Group XIII, § 831

If the symptoms point to a *Psychoneurosis* or to *Unsoundness of Mind* (*Psychosis*) see Chapter XX.

GROUP I. COMA, STUPOR, OR LETHARGY.

The patient is attacked with gradually deepening and prolonged UNCONSCIOUSNESS *from which he* CANNOT BE ROUSED *by shaking or calling.* The case is one of COMA. STUPOR is a less severe degree of coma; the causes are similar. LETHARGY is a condition simulating ordinary sleep, but it may be prolonged for days or weeks.

§ 711. Coma.—In *Coma* the corneal reflexes are absent, the pupils insensitive to light, and the patient does not swallow fluids placed in his mouth. The diagnosis of such a case may present extreme difficulty, especially when no history of the patient's previous health or habits can be ascer-

tained. *If the coma is of sudden onset*, by far the commonest cause is *Cerebral Hæmorrhage.* The cause of such a hæmorrhage must be investigated.

Remember that ordinary examinations will not commonly reveal the cause of the coma. For this purpose examination of the fundi, measurement of the blood pressure, a catheter specimen of urine and spinal puncture, may all be necessary. Remember, too, that patients frequently injure themselves when they fall with a cerebral apoplexy, and the finding of a local scalp lesion, or even escaping blood or spinal fluid from the nose or ears, do not necessarily indicate a traumatic cause. If the patient's breath smells of alcohol, moreover, this does not of necessity mean that the coma is of alcoholic origin.

The Causes of Coma may be divided clinically into two groups:

A. Unilateral Symptoms *are present.*—You may note (1) Conjugate deviation of the head and eyes. (2) Inequality of the pupils. (3) Asymmetry of the mouth and lower face, which puffs in and out with respiration, or other cranial nerve palsies, and (4) Absolute flaccidity of the limbs on one side when you test all four limbs in turn, lifting them and letting them flop on the bed. The cause is probably some Local Intracranial Lesion—viz.:

I. Cerebral Hæmorrhage, Embolism or Thrombosis.
II. Cerebral Tumour or Abscess.
III. Cerebral Compression from Traumatic or Spontaneous Extra-cerebral Hæmorrhage.

B. *The Symptoms are* Bilateral *and* Symmetrical.—The pupils are equal, the face symmetrical, and all four limbs equally flaccid. The cause is a Traumatic, or Toxic or Inflammatory Condition—viz.:

IV. Cerebral Concussion.
V. Post-Epileptic Coma.
VI. Uræmia.
VII. Diabetes Mellitus.
VIII. Insulin Hypoglycæmia.
IX. Poisoning by Opium, Alcohol, Barbiturates or Carbon Monoxide.
X. Acute Yellow Atrophy of the Liver (Cholæmia).
XI. Meningitis or Subarachnoid Hæmorrhage.
XII. Acute Encephalitis and Encephalitis lethargica.
XIII. Cerebral Malaria.
XIV. Heatstroke.
XV. Coma Carcinomatosum.
XVI. Coma Vigil of Typhus, Cholera, and Typhoid Fevers.
XVII. Anaphylactic Coma.
XVIII. Trance States and Katatonia.
XIX. Hypertensive Attacks.

Clinical Investigation of Coma.—Obtain from a relative or friend any *History* available of preceding ill-health or fits, and if the coma came on suddenly or gradually. A sudden onset, *i.e.*, apoplexy, is indicative of a cerebral vascular lesion. Observe the *Age* of the patient. Coma in childhood is due to Meningitis, Epilepsy, or Intracranial Tumour or Abscess; about middle-age suspect a Cerebral Hæmorrhage.

Besides examining for unilateral signs, you should examine the scalp and skull for local trauma, noting if there is escape of spinal fluid and blood from the ears and nose (Fracture of the base). You smell the breath for acetone (Diabetes) and note the colour of the skin (jaundiced in Cholæmia, pink in Coal-gas poisoning), the presence of scars on the face and limbs (Epilepsy) or marks of hypodermic punctures (Insulin hypoglycæmia, Morphinism). You note the size of the pupils (pinpoint in Opium poisoning and Pontine Hæmorrhage) and the presence of head-retraction (Meningitis or Subarachnoid Hæmorrhage).

Next look at the optic discs (papillœdema in Intracranial Tumour and Abscess, albuminuric retinitis in Uræmia) and examine the ears for suppurative disease (Sinus Thrombosis). Estimate the blood pressure and withdraw a catheter specimen of urine, testing it for albumen and sugar. Finally, perform a spinal puncture.

Blood in the spinal fluid indicates hæmorrhage into the subarachnoid space from meningeal or ventricular hæmorrhage, or hæmorrhage from fractured base. In Meningitis, turbid spinal fluid will be present, or the fluid will show pleocytosis.

A. *There is* COMA, *and* UNILATERAL SYMPTOMS *are present.*

I. **Cerebral Hæmorrhage, Embolism and Thrombosis.**—The term apoplexy, or "stroke," is used to indicate a sudden loss of consciousness due to a vascular lesion—hæmorrhage, embolism, or thrombosis—within the skull. The *extent* and the *suddenness* of the vascular lesion, rather than its nature, determine the occurrence of coma. Cerebral hæmorrhage is most frequent between fifty and seventy, but it may occur even in children.

§ **712.** *Symptoms of Cerebral Hæmorrhage.*—The attack may be ushered in by a stage of headache or giddiness, lasting a few days, connected doubtless with the associated high blood pressure; or it may come on suddenly without warning. Vomiting or a convulsion sometimes occurs. Sometimes the paralysis comes on with faintness and vertigo only; or it may develop more gradually, followed later by unconsciousness (ingravescent apoplexy). Sometimes it comes on during sleep. In severe cases the patient is deeply comatose, cyanosed, and the breathing is stertorous, the skin is cold and covered with sweat. The muscles are completely flaccid, the flaccidity being greater on the paralysed side. The paralysed angle of the mouth drops, and the cheek flaps in and out with respiration. The patient is incontinent, and in hiscoma blisters may develop on the heels, buttocks and sacrum. The pupils and tendon reflexes are commonly absent or a larger pupil may be present on the side of the cerebral lesion. The pulse is slow and the temperature subnormal.

Diagnosis of Cerebral Hæmorrhage.—The *sudden* onset of profound coma in a person of middle age, with the presence of unilateral signs, are points of great diagnostic significance. It should be remembered that cerebral hæmorrhage frequently supervenes in the course of chronic interstitial nephritis, and therefore uræmia and apoplexy may be concurrent. The diagnostic features of the greatest value are the state of the pupils, particularly their inequality, the loss of the conjunctival reflex, and the augmented blood pressure. The diagnosis of the various *forms of vascular lesion* is given in Table XLVI. One is fairly safe in excluding cerebral hæmorrhage if the blood pressure is not high, provided that the patient is not suffering from a hæmorrhage into a vascular and malignant growth, or some severe anæmia, such as acute leukæmia. Though high blood pressure is suggestive of cerebral hæmorrhage, remember that the arterial degeneration which usually accompanies it may give rise to thrombosis as well as hæmorrhage.

As regards the *locality* of the hæmorrhage, the usual position (about

70 per cent.) is the *external* or *internal capsule*. The hæmorrhage comes from the lenticulo-striate artery, especially the left side, giving rise to hemiplegia on the side opposite to the lesion. In most of the cases of hæmorrhage into the *ventricles* there is deep coma and head retraction with paralysis or rigidity of all four limbs, and blood in the spinal fluid: the condition is fatal. Marked contraction of both pupils, convulsions and vomiting, with paralysis of all four limbs, and rapid rise of temperature to the level of hyperpyrexia, suggest hæmorrhage into the *pons*. Hurried or Cheyne-Stokes' respiration often accompanies hæmorrhage in this site, and death ensues. *Conjugate deviation of the head and eyes* towards the paralysed side is frequent when the hæmorrhage involves the motor tract.

Prognosis.—It is held by some authorities that cerebral hæmorrhage almost always goes on to a fatal termination, and that the cases of supposed cerebral hæmorrhage which recover are really cases of thrombosis. About half the cases of supposed hæmorrhage recover from the attack, some with residual paralysis; the other half die within forty-eight hours. The depth and duration of the coma are fair measures of the extent of the mischief, and therefore of the prognosis. As regards *locality*, ventricular hæmorrhage and hæmorrhage into the pons are the most serious.

Etiology.—Cerebral hæmorrhage is more frequent in the male than the female sex, and does not usually occur until after fifty. It is common in those addicted to alcohol. The rarer cases of "apoplectic seizure" in persons under forty are almost invariably due to embolism, thrombosis, or rupture of a "congenital" aneurysm. Heredity plays an important part by reason of the tendency to vascular disease which runs in families. Disease of the vessels is a necessary precursor to their rupture. High blood pressure is a most important factor in the causation of apoplexy; it predisposes to arterial disease, and may also determine the hæmorrhage. The causes of high blood pressure are given in § 87; the commonest cause is chronic interstitial nephritis. Hæmorrhage into the brain may occur in a variety of other conditions. It may occur into a rapidly-growing *glioma* and is rapidly fatal. It may be the terminal incident in *acute leukæmia*, *purpura*, and other blood states. It occurs in *acute infections*, *e.g.*, *diphtheria* and *septicæmia*, *e.g.*, puerperal sepsis, and in these last conditions, embolism (mural endocarditis) and thrombosis also occur.

§ 713. Cerebral Embolism.—Cerebral embolism involving a fairly large artery may give rise to all the symptoms of apoplexy. Embolism is characterised by *instant* loss of consciousness. When a small cortical vessel is blocked, giddiness, or a Jacksonian attack followed by a monoplegia, may replace coma. Sometimes the posterior cerebral artery is involved, with hemianopia and sensory change, rarely the anterior cerebral or the internal carotid (*carotid hemiplegia*—§ 684). The artery usually affected is the middle cerebral. The age of the patient and the presence of cardiac disease, especially auricular fibrillation, mitral disease and ulcerative endocarditis, aid us in diagnosis. Embolism occurs from the pulmonary veins in the puerperium. During induction of artificial

pneumo-thorax a transient hemiplegia may occur from accidental introduction of air into the pleural or pulmonary veins (" Pleural hemiplegia ").

Fat Embolism follows a few hours after fracture of a long bone (Table XLVI). The fat globules lodge first in the lungs, producing cyanosis and pulmonary œdema. Some of the globules may make their way through the pulmonary capillaries to the cerebral vessels, with delirium, coma and localised cerebral palsies.

§ 714. Thrombosis of the Cerebral Arteries.—Thrombosis generally arises from a gradual occlusion of the lumen of a vessel by senile arterial disease, or by syphilitic endarteritis, in younger subjects. The picture is that of a person going about in usual health and awakening early in the morning conscious, but with a hemiplegia. In other cases, a hemiplegia develops over a period of one or more hours with little or no loss of consciousness. There is sometimes a history of previous " faints " or " attacks " indicating thrombotic lesions in tiny cerebral arteries. Thus transient hemianopia, monoplegia, hemiparesis, dysarthria, aphasia or mental confusion may occur, and these symptoms may be repeated many times, either singly or in combination, with partial recovery in the intervals between the attacks. Where the vessel occluded is large and the ensuing necrosis extensive, coma will be profound. Cerebral Thrombosis is much commoner than Cerebral Hæmorrhage or Embolism.

The *Prognosis* of cerebral embolism as regards life is usually good, though the paralysis tends to remain. Paralysis is most likely to clear up in children. If the causal condition remains, a second attack may occur. Cerebral embolism in malignant endocarditis is ultimately fatal. In thrombosis survival is likely, often for many years; but the life of a patient must be regarded as precarious and the field of effort limited. Women seem to survive strokes better than men. Death may occur from coronary thrombosis or from cerebral hæmorrhage into an area previously softened by thrombosis. In syphilitic cases recurrence is unlikely if anti-specific treatment is thoroughly pursued.

The *Treatment of an Apoplectic Seizure.*—Perfect rest and quiet are very important. The patient should, as a rule, be left in the room where the seizure occurred—a mattress being placed on the floor, if necessary—rather than incur the movement necessary to raise him on to a bed. The head and shoulders should be raised, and the patient turned gently over to one side to prevent the tongue falling back into the pharynx. The administration of food is, as a rule, undesirable, at least by the mouth, for fear of its passing into the air passages; alcohol must be absolutely forbidden. The patient will benefit by starvation from food for a day or two; the lips may be moistened. The bladder should be watched, and the catheter carefully passed if necessary. In cases due to *embolism* the condition responsible for the embolism must be treated. Complete rest in bed for several weeks is essential to diminish the risk of further embolism. If embolism occurs in a patient receiving treatment for auricular fibrillation by quinidine, this drug must be discontinued. In *thrombosis* stimulants, *e.g.*, tinct. nucis vom. ℥ 10–15, t.d.s., or niketh-

amide (coramine) hypodermically—are indicated. Prolonged rest in bed is inadvisable. In *hæmorrhage*—indeed whenever the blood pressure is high—a brisk purge is indicated; two drops of croton oil or 4 to 8 grains of calomel on the tongue may be given, followed, if necessary, by a soft soap or turpentine enema. The chief indication is to prevent any extension of the hæmorrhage. If the blood pressure is very high, it is a good practice to bleed to the extent of 10 to 20 ounces. An ice-bag or a cooling lotion to the head may relieve headache.

In all cases, even when coma is present, the paralysed arm and leg should be frequently moved at all joints to prevent contracture. A pillow should be placed in the axilla, or a sling tied to the top of the bed to abduct the arm. The forearm should be frequently supinated and a light cock-up splint applied to the hand and forearm during the morning and afternoon. Passive movements should be carried out at all joints thrice daily with gentle massage, and as soon as voluntary power returns active re-educative exercises can be commenced. If left to himself the patient will walk with his leg extended, circumducting the limb to prevent his toes catching the ground. He should be taught to advance his leg by flexion at hip and knee. In sitting he should avoid the tendency to adduction of the leg and inversion of the foot.

Table XLVI.

Diagnosis of Cerebral Hæmorrhage, Thrombosis, and Embolism.

	Cerebral Hæmorrhage.	*Cerebral Thrombosis.*	*Cerebral Embolism.*
Age.	Middle and Advanced Life.	Middle and Advanced Life or any age.	Any age, but frequent in early life.
Causes.	1. Arterio-sclerosis with high blood pressure. 2. Blood diseases. 3. Acute Infections and Septicæmia.	1. Cerebral atheroma. 2. Syphilitic endarteritis. 3. Acute infections. 4. Exhausting conditions, phthisis, anæmia. 5. Cardiac enfeeblement.	1. Cardiac lesions, especially mitral stenosis, auricular fibrillation and malignant endocarditis. 2. " Fat embolism " in fracture of long bones.
Onset.	Coma usually sudden, with convulsions.	Onset usually gradual, with premonitory vertigo. Sometimes convulsions, rarely coma.	Instantaneous loss of consciousness.
Time of Onset.	During emotional excitement or physical exertion.	Often during sleep.	During exertion.

§ 715. **Thrombosis of the Cerebral Sinuses** may give rise to coma and all the symptoms of apoplexy. It may arise from caries of the skull (syphilitic or tuberculous), extension from an intracranial abscess, *e.g.*, in suppurative ear disease, and occasionally from the pressure of an aneurysm, gumma, or other tumour; or in association with meningitis. Sinus thrombosis is favoured by the feeble cerebral circulation characterising cachectic conditions (chronic diarrhœa, typhoid fever, and marasmus in children). *Septic thrombosis* and the differential diagnosis of thrombosis of the lateral, cavernous and longitudinal sinuses are described in § 738.

II. **Cerebral Tumour or Abscess.**—The symptoms may be unilateral or, less commonly, bilateral. Sudden onset of coma in cerebral abscess indicates that rupture has

occurred into the ventricle, the spinal fluid in such cases being purulent. There will be a preceding history of otitis media, suppurative frontal sinusitis or intrathoracic sepsis, and papillœdema or cranial nerve palsies may be present. *Chronic subdural hæmatoma* following a head injury, or in the aged and arterio-sclerotic, may cause similar symptoms. The onset of coma in these cases generally means a fatal issue (§ 827).

III. **Cerebral Compression from Traumatic Extra-cerebral Hæmorrhage.**—In DELAYED TRAUMATIC APOPLEXY the patient recovers from his initial concussion and collapse. With the rise in blood pressure within a few hours, stupor or coma again intervene, with Jacksonian convulsions and rapidly progressive monoplegia or hemiplegia. The cause is a *rupture of the middle meningeal artery* with extra-dural hæmatoma or a subdural hæmatoma, from rupture of cortical veins. The treatment is exclusively surgical.

B. *There is* COMA, *and the Symptoms are* BILATERAL *and* SYMMETRICAL.

§ 716. IV. **Cerebral Concussion.**—The unconsciousness follows immediately after the head injury without any latent interval. The patient is pale and collapsed, with weak pulse, shallow respiration, pale face, dilated pupils, sweating, flaccidity of the limbs and low blood pressure. In other cases he is merely dazed. In severe concussion the stupor lasts hours or days and is followed by a reactive stage ushered in by vomiting or convulsions. The temperature rises to 100° F. or higher, the pulse becomes full and bounding and the respirations deeper. There is intense headache and photophobia with hypersensitiveness to noise, and the patient lies curled up in bed with his limbs flexed, resentful of interference. This is termed "cerebral irritation," and these symptoms may last for days or weeks, perhaps accompanied by visual hallucinations. Steadily rising temperature to 104° or 105° is a sign of grave omen, indicating extensive contusion. The occurrence of *intracranial hæmorrhage* may be indicated by (1) progressive deepening of unconsciousness, (2) progressive loss of tone or power in the limbs on one side, (3) progressive slowing of the pulse, (4) progressive discrepancy in the size of the pupils—the hæmorrhage being usually on the side of the slowly dilating pupil.

POST-TRAUMATIC AMNESIA is that interval of forgetfulness which may elapse between the moment of impact and the time of subsequent recovery of continuous awareness of surroundings. "Islands" of memory may exist within the span of post-traumatic amnesia. As the patient recovers, the duration of post-traumatic amnesia tends to shrink. During this period rational behaviour may be carried out and subsequently forgotten.

RETROGRADE AMNESIA may also be present for the events immediately preceding the injury. Long retrograde amnesias of months or years, or total amnesia for all events prior to the injury occur usually in hysterical personalities.

Prognosis.—The duration of post-traumatic amnesia is usually a fairly reliable guide to the severity of the brain injury and may be used to estimate prognosis except in cases which are complicated by compensation hysteria. If the post-traumatic amnesia lasts minutes, the brain injury is minor. When it lasts 1 to 3 hours, the brain injury is moderate and complete recovery may be expected in from six to eight weeks. When the post-

traumatic amnesia (P.T.A.) lasts a week or more, the brain injury is severe and intellectual insufficiency or personality change may result as a temporary or permanent finding. Complete recovery in such cases may take three to six months or longer. Other factors to be taken into account in estimating the prognosis are—(1) the pre-traumatic personality of the patient, (2) associated damage to cranial nerves and other structures, and (3) the type and circumstances of the injury.

Treatment.—As soon as possible the patient should be laid flat in bed with his head to one side, and heat applied to counteract shock (§ 239). If there is a bleeding scalp-wound, hæmorrhage should be arrested by a pad and tight bandage, or by deeply suturing the scalp. Should there be a depressed fracture of the skull operation may be necessary (*e.g.*, if there is an overlying scalp wound, which might admit infection to the underlying structures). Search should be made for gross injuries of the limbs and spine, and as soon as the patient has recovered from initial shock, X-ray photographs of the skull should be obtained whenever possible. Noise and light should be excluded from the sick room. Watch the bladder for retention. Fluids should be given by mouth, with a feeding cup if the patient can swallow. The nurse should be instructed to watch for the danger signs of meningeal hæmorrhage enumerated above. Restlessness is treated by rectal paraldehyde, ℳ. 120–240 given in an equal amount of olive oil, or by chloral hydrate gr. 15, potassium bromide gr. 20, three or four-hourly by mouth. Morphine and alcohol should on no account be given. Thirty-six hours after the head injury lumbar puncture should be performed and the fluid pressure estimated by a manometer. If the pressure of the spinal fluid is *above* 150 mm., fluid should be withdrawn until that pressure is reached; this may have to be repeated. Saline purgatives and 6–10 oz. of a saturated solution of magnesium sulphate run into the rectum with a tube and funnel will help to dehydrate the œdematous brain. If the pressure is *below* 150 mm. no spinal fluid should be withdrawn. The foot of the bed should be raised on 6 inch blocks.

Intravenous dextrose solution (50–100 c.cm. of a 50 per cent. solution) may be administered later to cases with raised intrathecal pressure. Given too early, it may mask symptoms of meningeal hæmorrhage. It causes a sustained fall in intracranial pressure and helps to control acidosis and shock. It should be injected very slowly, at a rate not exceeding 3 c.cm. a minute. Where return to consciousness is delayed for days or weeks (traumatic stupor), the question of decompression may have to be considered. Most cases with cerebral contusion (§ 696), even when aphasic, clear up without operation. Convalescence should be graduated with slow, progressive increase of the field of mental and physical effort. Many cases will be able to leave bed at the end of three weeks and return to work in four to six weeks' time. Others continue to suffer from liability to physical and mental fatigue, difficulty in concentration, headaches, giddiness and sleeplessness and other symptoms of unresolved cerebral

contusion. If possible the patient should be tested out in the performance of his work at home before he returns to business.

V. **Post-Epileptic Coma.**—If there is no history we may have to rely on the finding of old scars for evidence of previous attacks. The coma is usually of short duration, and the patient becomes more and more conscious. After a "congestive attack" in General Paralysis of the Insane the patient may become comatose.

VI. **Uræmia** (see § 372).—There may be albuminuric retinitis or œdema about the face and legs. A catheter specimen will show albumen, tubular or blood casts, and, in acute nephritis, blood. Uræmia produces stupor rather than coma, the deep reflexes can be obtained, there is usually no incontinence, and the plantars are flexor. Uræmic twitchings or convulsions may be observed. Deep coma with unilateral signs may occur in uræmic patients, due to cerebral hæmorrhage, to which they are especially liable.

VII. **Diabetes Mellitus** (see § 416).—The examination of the urine makes the diagnosis simple. It is usually gradual in onset.

VIII. **Insulin Hypoglycæmia** follows an overdose of insulin which leaves the blood-sugar far below its normal value. A small overdose gives rise to prodromal symptoms (§ 418); after a large overdose unconsciousness supervenes in half to one hour, often accompanied by fits. The diagnosis is settled by a blood-sugar examination: insulin coma only occurs if the value is below 0·045 to 0·06 per cent. Consciousness is restored by injecting adrenalin hydrochloride 1 c.c. subcutaneously, or by intravenous dextrose.

IX. **Poisoning by Opium, Alcohol, Barbiturates, Aspirin, or Carbon Monoxide.**—In all forms of narcotic poisoning the deep reflexes are lost and the plantars are extensor. In *Opium poisoning* the pupils are contracted to pin-points. In morphinists, the scars of hypodermic piqures may be seen in the arms and thighs. In *Alcoholism* the smell of the breath is fallacious. If house-surgeons would bear this in mind they would not send dying cases of apoplexy or fractured skull away from hospital as "drunks." If unilateral signs are present the case is not one of alcoholism. The diagnosis of Alcoholism may be sustained by finding alcohol in the urine, or spinal fluid. Mix a specimen with potassium bichromate solution and allow strong sulphuric acid to flow to the bottom of the test-tube. The solution turns a bright emerald green if alcohol be present in quantity. In *Barbiturate* (Medinal, Veronal, etc.) poisoning the picture resembles opium poisoning, except that the respiration rate is often raised: skin rashes are rare. There is great liability to hypostatic pulmonary œdema. The drug may be present in the spinal fluid. *Aspirin* poisoning is characterised by very profuse sweating, hyperpnœa due to acidosis, a reducing substance in the urine and a positive ferric chloride test. It is very liable to be confused with hypoglycæmic coma (§ 418). *Carbon Monoxide* poisoning occurs from coal-gas or petrol fumes, accidentally or otherwise. There is sudden collapse and unconsciousness with stertor and a cherry-red complexion. The blood-spectrum is that of carboxy-hæmoglobin.

X. **Acute Yellow Atrophy** (Cholæmia, § 333).—The associated jaundice makes the diagnosis.

XI. **Meningitis or Subarachnoid Hæmorrhage.**—The presence of head retraction and Kernig's sign point to meningeal irritation. A sudden onset of headache, and deepening coma, indicate a *Subarachnoid Hæmorrhage* from a leaking cerebral aneurysm, usually situated on the Circle of Willis.

§ 717. In **Subarachnoid Hæmorrhage** the presenting symptoms are those of *sudden* intense headache with rigidity of the neck, together with vomiting, head retraction, Kernig's sign, and bilateral extensor plantar responses.

In severe cases there may be focal or general epileptiform convulsions, with rapid coma and death. In some, the spurting arterial jet excavates the inferior surface

of the hemisphere, producing severe hemiplegia or hemianopia. The *diagnosis* closely resembles that of an acute meningitis, but (1) the onset is startlingly sudden, indicating a vascular cause; (2) signs of tuberculosis, suppurative otitis, etc., such as commonly cause meningitis, are absent; (3) retinal hæmorrhage or subhyaloid hæmorrhage and papillœdema may be seen in the fundi; (4) the diagnosis is promptly cleared up by lumbar puncture, which reveals abundant blood intimately mixed with the spinal fluid. When the spinal fluid settles, the corpuscles sink, leaving the supernatant fluid pink with the "laked" blood. During recovery the spinal fluid may be yellowish, and shows a lymphocytosis. The bleeding in many cases comes from a small *non-syphilitic aneurysm* ("congenital" aneurysm) on the Circle of Willis or a cerebral artery near this. Massive albuminuria may be present at the onset for a day or two, suggesting the diagnosis of chronic nephritis. Root-pains in the arms and trunk may be observed as the blood extends downwards in the spinal theca. Before it ruptures, the aneurysm may cause migraine or recurrent unilateral headaches. In others, it may compress and paralyse one of the oculo-motor nerves, commonly the third. Slow leakages of blood from the aneurysm cause recurrent bouts of severe supraorbital pain with gradually increasing ptosis and ophthalmoplegia. Subarachnoid hæmorrhage is a fairly common disease. It occurs at any age and especially in the third and fourth decades.

Subarachnoid hæmorrhage may also occur in (1) Cerebral Arterio-sclerosis, (2) Renal Disease with high blood pressure, (3) Intra-cerebral Hæmorrhage which leaks into the ventricles, (4) Vascular Cerebral Tumour in the neighbourhood of the ventricular system or subarachnoid space, (5) Acute Encephalitis Lethargica, (6) Hæmorrhagic blood diseases, such as Acute Leukæmia, (7) Septic embolic cerebral aneurysm in Infective Endocarditis.

The milder cases recover, but fresh leakages are liable to occur months or years later. More than a third of the cases, however, are immediately fatal. Lumbar puncture should be performed for diagnosis only; as a therapeutic measure it does more harm than good and may precipitate further bleeding. The patient should be nursed in the horizontal position; retention of urine should be looked for. Intramuscular injections of thromboplastin or normal horse serum to increase the coagulability of the blood, are indicated, with ice-bags to the head, carefully suspended, and elimination of light and noise from the sick-room. Pain is relieved by morphine, once the diagnosis is made, or by pyramidon gr. 10 and aspirin gr. 10. The patient should be kept in the horizontal position until all headache and neck stiffness have gone. There should be no return to work for three months. Recurrences are most unlikely if the patient avoids strenuous effort. In severe cases the initial illness may be followed by a transient confusional psychosis. It has been possible to demonstrate the aneurysm by arteriography, and then ligature has been successfully performed.

XII. **Acute Encephalitis** is an occasional cause of Coma (see § 740). The encephalitis may be primary, as in Lethargic Encephalitis or Polioencephalitis, or it may follow specific fevers such as measles, diphtheria, scarlet fever, or vaccination (Vaccinal Encephalitis). It may occur in Cerebral Syphilis.

XIII. **Cerebral Malaria.**—Coma, with hyperpyrexia, occurs from malignant tertian malaria (§ 510) in malarial districts. The plasmodium will be found in the blood.

XIV. **Heat-stroke** (Sunstroke) (§ 508) causes coma, with hyperpyrexia—108° F. —or above this. The spinal fluid is sterile and shows a polymorphonuclear pleocytosis in the acute stages, later lymphocytes appear. Lumbar puncture produces immediate relief of symptoms.

XV. **Coma Carcinomatosum** occurs in patients dying of secondary malignant deposits. The urine shows acetone and diacetic acid. A similar coma occurs as a terminal event in Addison's disease and other exhausting illnesses.

XVI. **Coma Vigil** is a condition occurring in cholera, typhus and severe typhoid fever, where the vital processes are at so low an ebb that the patient may be mistaken for dead.

XVII. **Anaphylactic Coma.**—Following upon the injection of a foreign protein, the patient may become comatose, as the result of severe anaphylactic shock.

XVIII. **Trance States** (§§ 697, 888) should never be mistaken for true coma. The breathing is never stertorous, the pupils react to light and the patient forcibly resists attempts to open the eyes. In **Katatonia** there is mutism, refusal of food and general diminution of activities. Such patients allow their bodies and limbs to be placed in awkward positions which are maintained indefinitely. There is apathy but no unconsciousness. The condition occurs in Schizophrenia as well as in traumatic and other stupors (§ 897).

XIX. The HYPERTENSIVE ATTACK; see § 94.

The *Prognosis* of coma is always grave, the gravity increasing with its depth and duration. The coma after head injury usually comes under the care of the surgeon. The coma of apoplexy and other vascular lesions has been already dealt with. In post-epileptic coma, if the patient does not recover within a few hours then status epilepticus is present and the condition is serious. Coma occurring with tumour of the brain or acute lesions is usually fatal. The prognosis of opium poisoning depends upon the amount of the drug administered and the promptitude and efficiency of the treatment. Uræmic coma is not so unfavourable as might be thought; cases recover with proper treatment, but in granular kidney the condition recurs sooner or later. In diabetic coma, which was formerly fatal, the patient now usually recovers with insulin and glucose.

Treatment.—For *alcoholism* and all *drug poisoning* the stomach and colon should be repeatedly washed out with warm water and warmth and cardiac stimulants applied. In addition, in *barbiturate* poisoning the subarachnoid space should be drained by lumbar puncture, and the patient propped up in bed to prevent pulmonary congestion and œdema. Nikethamide, picrotoxin, or strychnine is given if the heart or respiration be failing. The patient must be kept awake by walking him about, applying electricity to the limbs, ammonia to the nostrils, and artificial respiration. For *uræmia* eliminate the poison in the blood by colon lavage, hot packs, venesection and saline injections. *Diabetic coma* calls for skilled and prompt treatment by insulin. For *insulin coma* give dextrose intravenously and inject adrenalin and pituitrin (§ 418). For *carbon monoxide* poisoning carry out artificial respiration, and give to breathe a mixture of 7 per cent. carbon dioxide and 93 per cent. oxygen. Special inhalation apparatus is provided for this purpose. This may also be used for opium poisoning. In all poisoning cases artificial respiration may be carried out by means of a Drinker or Paul-Bragg apparatus.

§ 718. Coma in Children, apart from injury, may be due, in order of frequency, to tuberculous meningitis, post-basic meningitis, suppurative meningitis, post-epileptic stupor, cerebral tumour, syphilitic pachymeningitis, sinus thrombosis and hæmorrhage; diabetes, abscess and intracranial cysts, are rare causes. The history, mode of onset and associated symptoms, aid the diagnosis. Tuberculous meningitis is by far the most frequent cause (§ 727). Cerebral hæmorrhage or embolism occur chiefly in association with the specific fevers, such as small-pox and whooping-cough, also with rickets and scurvy. In marasmic conditions, thrombosis of the longitudinal sinus (§ 715) may ensue, together with meningeal hæmorrhage, giving

rise to convulsions followed by coma. Thrombosis of the veins of Galen and lateral sinus thrombosis, in association with ear disease, may cause coma (§ 738).

The patient remains in a condition simulating normal sleep for days or weeks—the condition is LETHARGY.

The *causes* of lethargy are similar to the causes of coma. Lethargy is a prominent symptom in *Encephalitis Lethargica* (see § 698) and in *Tumours of the Hypothalamic region.* In both tumours and encephalitis fever and ocular palsies may be present, but gross papillœdema is more common in neoplasm. Mild continued lethargy accompanied by mental confusion is suggestive of *narcotic poisoning.*

GROUP II. TRANSIENT LOSS OF CONSCIOUSNESS. FITS OR CONVULSIONS.

§ **718a.** *The patient has a sudden transient* ATTACK *of* LOSS OF CONSCIOUSNESS. The condition may be a simple "faint," SYNCOPE, or, if it follows a head injury, CONCUSSION. EPILEPSY must always be considered, either *idiopathic*, or *symptomatic* of local or general disease, *e.g.*, cerebral arteriosclerosis, cerebral syphilis, cerebral tumour, uræmia, etc.

(*a*) SYNCOPE. (Fainting.) Apart from those who faint from cardio-vascular instability or insufficiency, due to disease or debility, a small proportion of "nervous" individuals are liable to faint in certain situations. Such faints are conditioned by prolonged standing in crowds and stuffy atmospheres, during medical examinations, listening to medical lectures, on feeling sudden pain, at the sight of blood, or during sudden panic. Others faint after extreme physical over-exertion (see also § 35).

The phenomena observed during some of these syncopal attacks may be indistinguishable from those seen in epilepsy; convulsions and even incontinence may occur. In such rare cases, a constitutional nervous instability is present and epilepsy may subsequently develop. In true "reflex epilepsy" the conditioning stimuli are stereotyped and often bizarre, *e.g.*, certain sounds or sudden noise will precipitate an attack: moreover, in these cases other fits, not so conditioned, will usually occur.

VERTIGO bears a superficial resemblance to fainting. For *Ménière's Disease*, see § 692. In severe aural vertigo consciousness may be lost when the paroxysm is at its height.

(*b*) As the result of HEAD INJURY the patient may be momentarily *dazed* or may *lose consciousness* (see § 716).

(*c*) § **719. Minor Epilepsy** (Synonym: Petit mal).—The attack is preceded—in about half the cases—by an aura or warning, unattended by convulsions, and often without falling, the whole lasting rarely more than half a minute to a minute. In the type of attack now under consideration, it may be that the patient merely pauses in a conversation and does not reply to questions, or there is only a vacant look, a fixity of gaze, dilated pupils or momentary pallor of the face, which none but

a close observer would notice. In more severe cases the patient drops what he is holding, or flings it from him ; his head may sag for a moment and urine may be passed involuntarily, without any convulsion.

(*d*) SENILE SYNCOPE.—In the aged, transient losses of consciousness may occur from minute cerebral thromboses or myocardial failure with arterial degeneration. They indicate a need for increased rest, protection from cold and exhaustion, and small quantities of alcohol with meals and last thing at night.

Convulsions (Fits, Epilepsy) may be due to (A) IDIOPATHIC EPILEPSY, or may be (B) SYMPTOMATIC, or (C) HYSTERICAL.

A. IDIOPATHIC EPILEPSY

I. Major Epilepsy.
II. Minor Epilepsy (Petit Mal).
III. Special Varieties of Epilepsy.

B. SYMPTOMATIC EPILEPSY

IV. Cerebral Tumour.
V. Cerebral Syphilis.
VI. Cerebral Arterio-sclerosis.
VII. Hypertensive Attack.
VIII. Traumatic Epilepsy.
IX. Congenital Nervous Disease.
X. Stokes-Adams' Syndrome.
XI. Cerebral Hæmorrhage or Embolism.
XII. Asphyxia.
XIII. Forced Deep Breathing.
XIV. Toxic Convulsions.
XV. Acute Encephalitis.
XVI. Malaria.
XVII. Intracranial Cysticercus.
XVIII. Presenile Dementia.

FALLACIES.—The convulsions of STRYCHNINE POISONING or TETANUS should never be mistaken for epilepsy. In these conditions *consciousness is never lost.*

§ 720. Investigation of Cases of "Fits."—Confronted with a case giving a history of a "fit" you must decide if the condition is *organic* or *psychogenic.* Psychogenic attacks are of two main varieties : (1) Hysterical Attacks, (2) Vaso-Vagal Attacks (Gowers).

The latter type of attack occurs in BORDERLAND EPILEPTIC patients. There is, however, never true loss of consciousness, as in organic epilepsy, and the onset of the attack is slow. The patient experiences a sense of unreality, of being unable to move or to utter a single sound, with an impending feeling of imminent catastrophe or death. Associated with this are palpitation, pain under the left breast, abdominal discomfort or other visceral sensation, and rapid lowering of blood pressure, nausea or flatulence. The attacks last many minutes or hours. There is often a family history of epilepsy or migraine. The *prognosis* is good. Any psychological factors should be sought and corrected, and luminal prescribed, as for epilepsy.

The table on page 923 gives useful aid in distinguishing between hysterical attacks and organic epilepsy.

Hysterical attacks may follow true Epileptic attacks—*Hystero-epilepsy.* Epilepsy may be feigned in *Malingering.* Such attacks are carefully planned, the attack lasts indefinitely, and resembles true epilepsy only in the imitation of the convulsive movements.

When in doubt, it is best to regard anomalous attacks as epileptic until the subsequent history makes the diagnosis definite.

To obtain the necessary diagnostic information, if you have not observed the attack yourself, *it is essential to interrogate an eyé-witness of the attack in addition to the patient.* In talking to the patient you should be careful not to speak of "fits" but of "attacks" or "seizures."

1. *Interrogation of the Eyewitness.*—The eyewitness should be asked to describe the attack in his own words. Questions are then put—Is there any sound before

TABLE XLVII.

	Organic Epilepsy.	*Hysteria.*
1. Periodicity and onset.	Attacks occur mostly at definite hours of the day or night.	Attacks follow upon an emotional crisis. Never during sleep.
2. Incontinence.	Incontinence of urine during attack, albuminuria found later.	Never incontinence.
3. Self-injury in attack.	Tongue, cheek, or lip-biting, with blood in the mouth.	The patient is not hurt in the seizure, although onlookers may be.
4. Movements.	Involuntary movements are clonic, if present, or have a definite march and are accompanied by conjugate deviation of the head and eyes.	The movements are purposive, and spectacular, the eyes screwed up and the hands clenched. Never conjugate deviation of the head or eyes; often convergent spasm of eyes.
5. Breathing.	Breathing stertorous, with cyanosis	Breathing never stertorous.
6. Duration.	Attack is of short duration usually.	Attack often prolonged, especially if before an interested audience.
7. Plantar reflexes.	Extensor during and immediately after the fit.	Usually absent at the toes.

he falls? What is his breathing like in the attacks, is it snoring? What is the colour of his face in the attacks? Does the patient reply to questions in his attacks? How long do the attacks last? These questions can be answered only by an eye-witness.

2. *Interrogation of the Patient.*—This should include direct questions as—Do you fall in the attacks? Do you get any warning? Has there ever been any blood in your mouth after the attack? Have you ever wet yourself in an attack by passing water? Have you ever hurt yourself in an attack? (An examination of the face, scalp, tongue or limbs may reveal cicatrices, evidences of injury in the fits.) At what time of day or night do your attacks mostly occur?

When observing a fit, push the edge of a handkerchief into the patient's mouth stand back and watch the distribution and spread of the convulsion. If the patient is in bed, throw back the bedclothes. After the convulsion is over, examine the limbs for flaccidity, examine the pupils and test the tendon reflexes and plantar responses.

§ 721. **Epilepsy** may be: A, IDIOPATHIC or B, SYMPTOMATIC.

A. IDIOPATHIC EPILEPSY is a chronic malady characterised by partial or complete loss of consciousness, which is usually the essential feature of the condition. In most cases convulsive movements occur, varying in distribution and degree. Minor Epilepsy (*petit mal*) consists mainly of transitory disturbance of consciousness, Major Epilepsy (*grand mal*) is loss of consciousness with generalised convulsions.

I. **Major Epilepsy.** *Symptoms.*—A complete epileptic fit has the following characters, though they are rarely all present in their entirety: (1) In some cases, during the previous twelve to twenty-four hours, there may be *prodromata*, consisting of headache, giddiness, malaise, or alteration of character or mood. In more than half the cases this stage is absent. (2) The fit, in many cases, is immediately preceded by

an *aura* or warning—*i.e.*, a sensation or movement lasting, at most, only a few seconds, valuable as indicating the point of the cortex whence the cortical nerve-storm starts. The auræ differ infinitely in detail. There are four main groups. *Sensory auræ* are most common—*e.g.*, "a wave passing over the body," numbness, a sensation of movement, flashes of light or of colour, or singing in the ears; *motor auræ*—*e.g.*, twitching of a muscle or limb, occasionally of the trunk, and in rare cases there is a "procursive aura," in which the patient runs forward or turns round and round; *psychical auræ*—*e.g.*, various strange thoughts or hallucinations; and *somatic auræ*—*e.g.*, gastric discomfort, nausea, or fluttering in the stomach. (3) *Loss of consciousness* is the pathognomonic feature of idiopathic epilepsy. It succeeds the aura so quickly that the patient may not have time to place himself out of danger before loss of consciousness is complete. (4) *Convulsions* supervene almost at the same time as the unconsciousness. They are often ushered in with a scream. The true epileptic cry is a "weird, unearthly, hollow sound, produced by inspiratory spasm drawing in air over the nearly closed vocal cords." It comes with the onset of loss of consciousness and the onset of tonic rigidity. In the classical case there is a short stage of tonic convulsions lasting about forty seconds, followed by a stage of clonic convulsions lasting one to three minutes. In the tonic stage the breath is held, the hands are clenched, the back is rigid, and the legs are extended, the pulse is quick and may be imperceptible. The clonic movements soon involve the whole body, and are sometimes of great violence, consisting of rapid extension and flexion of the limbs, opening and shutting of eyes and jaws. The interference with respiration during both the tonic and clonic stages causes stertor and an ever-increasing blueness of the face. The tongue is often bitten. During both these stages the pupils are dilated and inactive to light, and the conjunctival reflex is absent. The light reflex may return during the clonic stage. As the convulsions pass off the respiration becomes stertorous or snoring. Urine, fæces, and semen may be voided. The saliva issues from the mouth as a frothy foam, sometimes blood-stained from injury of the tongue. (5) A stage of *stupor* or *drowsiness* succeeds the convulsions, gradually passing into a deep sleep. The temperature directly after the convulsions is raised, sometimes as much as 4° or 5° F. (6) *Periodicity of Attacks.* It is a marked characteristic of idiopathic epilepsy that the fits occur between definite hours of the day or night. They may occur solely on rising or on retiring or may be strictly nocturnal. This is of importance in treatment. Epileptic fits tend to occur just before or just after menstrual periods. They tend to occur in groups at regular intervals, and may occur several times in one day and then disappear for weeks or months.

Special Symptoms.—Status Epilepticus is a rare condition in which the patient has a series of fits occurring in very rapid succession for several hours or even days, consciousness not being regained in the intervals; the temperature may rise to 107° F., and the issue may be

fatal. Status epilepticus may occur with local cerebral disease, *e.g.*, in G.P.I. and Encephalitis. *Post-Epileptic Automatism* ("Masked Epilepsy").—After an attack, often a minor attack, the patient may perform automatic, irresponsible acts, dressing or undressing himself, or putting the property of others into his own pockets. These automatic phenomena of the post-epileptic state seem to be generally in inverse proportion to the severity of the seizure, which may be so slight as to escape notice. He may pass into a state of dual personality, lasting for days altogether, or sometimes longer, and during this state may perform criminal, or even homicidal acts. No recollection of these automatic actions is afterwards retained. *Epileptic Equivalents.*—Mental disturbances, usually taking the form of attacks of bad temper, sometimes appear to take the place of a fit and are spoken of as "psychic equivalents." These may, however, be part of the mental changes seen in such patients, or may be transitory post-epileptic phenomena. *Mental Changes.*—Many of the patients, but by no means all, show signs of progressive mental deterioration, which may culminate in *epileptic insanity* (§ 903). This is seen chiefly in epilepsy commencing in childhood. The patient becomes egotistical and boastful, irritable, untruthful and spiteful. Transient grandiose delusions may occur, and violent, sometimes homicidal attacks. Acute mania may occur as a transient phenomenon after an attack. These chronic changes are probably due to progressive cortical degeneration. Other cases become dull, lethargic; these symptoms disappearing after a bout of attacks. *Simple jactitation* ("the jerks"), without loss of consciousness, occurs in many chronic epileptics.

II. **Minor Epilepsy** (Petit Mal), see § 719.

§ 722. III. **Special Varieties of Epilepsy.**

(1) **Jacksonian Epilepsy (Focal Epilepsy).**—This variety is common in focal *cortical* lesions, but may occur in idiopathic epilepsy. (*a*) Motor, (*b*) Sensory, (*c*) Visual, (*d*) Auditory, and (*e*) Uncinate attacks are described.

(*a*) *Motor Jacksonian Fits* commence unilaterally in the thumb or index finger, the corner of the mouth or the hallux. The attack spreads in an orderly march, according to the arrangement of movements in the motor cortical area (see Fig. 157). The point of starting indicates the position of the lesion in the cortex. The onset is with tonic spasm; later, broken or clonic twitchings occur, the whole attack occupying, perhaps, twenty minutes. The movements are confined, for a long period, to one limb or one side of the body, and consciousness is retained. Sometimes Jacksonian attacks terminate in a generalised convulsion, with loss of consciousness. The Jacksonian fit may be followed by a transient local paralysis (usually a monoplegia) or, if the convulsion is right-sided, by temporary aphasia. This paralysis is known as *Todd's Paralysis*, and may last hours or even days after the fit. (*b*) *Sensory Jacksonian fits* occur in parietal cortical lesions with numbness and tingling, starting locally, and spreading in an orderly march. These may be followed by transient astereognosis. (*c*) *Visual attacks*, blinding flashes of light of hemianopic distribution, followed by hemianopia, occur in occipital cortical lesions, and (*d*) *Auditory attacks*, sudden hallucinations of sound, followed by deafness, in temporal lobe lesions. (*e*) *Uncinate fits* occur in lesions of the uncinate gyrus or lesions in its neighbourhood (*e.g.*, in pituitary neoplasms) and consist of (i.) an intensely unpleasant flavour or odour, (ii.) champing or spitting movements, (iii.) a characteristic "dreamy" state.

Causes of Jacksonian Attacks.—(1) Any irritative cortical lesion, *e.g.*, depressed fracture of skull, meningeal scarring, with local vascular changes, extra-dural and subdural hæmatoma or abscess, cerebral tumour, granuloma or angioma, cortical softening in cerebral arterio-sclerosis, cerebral syphilis, etc. (2) General Paralysis of the Insane. (3) Uræmia. (4) Infantile Hemiplegia. (5) Idiopathic Epilepsy.

(2) **Flaccid Epilepsy.** The patient may or may not lose consciousness for a moment, toppling suddenly to the ground in a limp condition, often falling so heavily that injury is caused ("drop seizures"). No convulsion occurs and no tonic or clonic contraction of muscles. The attack is usually finished in a second or two, but may last longer.

(3) **Tonic Fits.**—These occur in mid-brain lesions and in lesions of the vermis of the cerebellum, or even in the absence of discoverable local lesion. The patient loses consciousness and the attitude is one of retraction of the head, flexion of the upper and extension of the lower limbs (§ 764). There is no clonic component.

(4) **Psychomotor seizures** are periods of amnesia or automatism which alternate with or may replace grand mal attacks.

(5) **Pyknolepsy.**—In children, between the ages of 4 and 12 years, attacks of minor epilepsy occur up to the number of fifty daily. No mental deterioration occurs, and the attacks cease at puberty. The condition cannot be diagnosed with accuracy until spontaneous cessation of all fits has occurred.

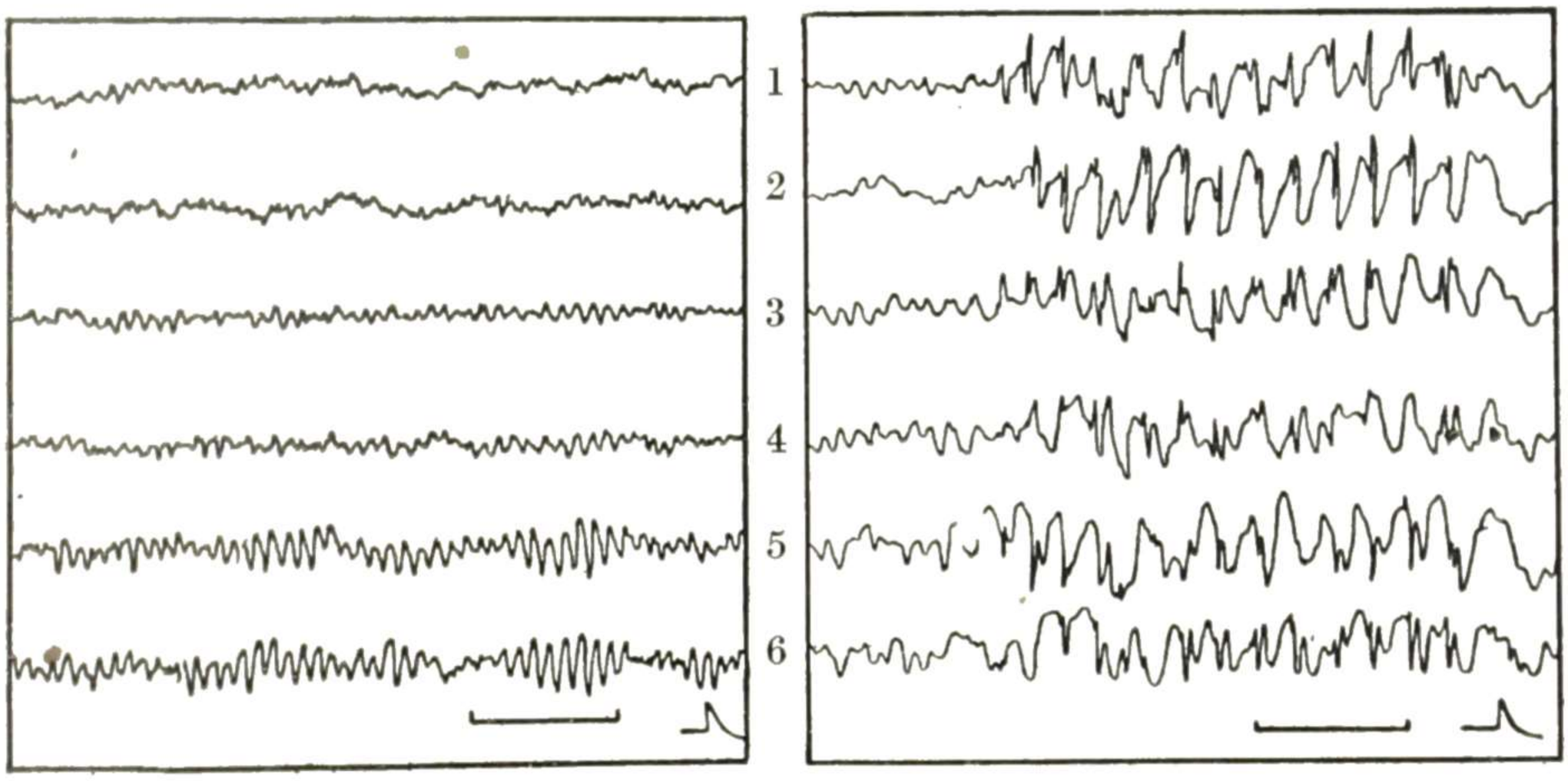
(6) **Reflex Epilepsy.**—Local or general convulsions, with loss of consciousness, start as the result of stimulation of a cutaneous epileptogenic zone. Pinching or pricking the sole of the foot may start a Jacksonian attack, and the attack may be prevented by tying a tight band round the limb above the area stimulated. This phenomenon occurs also with focal cortical lesions. Stranger still are the cases in which a sudden loud noise or bright flash of light will cause the patient to fall to the ground in a fit.

Diagnosis.—The diagnosis from *Symptomatic Epilepsy* is made by the finding of associated signs or symptoms of the causal disease (*vide infra*). In fits commencing after the age of 25 years, evidence of *G.P.I.*, *intracranial vascular disease*, *cardiac* and *renal* disease, should be sought for. It should never be forgotten that generalised fits may be the first and, for many months, the only sign of an *intracranial tumour*.

Electro-encephalography. A normal electro-encephalogram does not exclude epilepsy. Less than a third of epileptics examined in the interparoxysmal period show characteristic changes. A few show larval attacks: a gradually appearing outburst of fast waves, 30 per second. Cases of petit mal may show characteristic large voltage flat-topped waves formed by one or several rapid deflections at a rate of 3 per second. In those of unstable nervous constitution the electro-encephalogram may be abnormal although not significant of epilepsy. The records require expert interpretation.

Prognosis.—The probability of cure is greater when the patient is willing to co-operate for years in carrying out effective treatment. The outlook is better in the hereditary cases. Spontaneous cessation of epilepsy seems to occur fairly often. Attacks in infancy often disappear, to recur at puberty. The prognosis is bad when there is definite mental impairment or when the attacks start at puberty. Pregnancy affects the disease adversely. Death rarely occurs during the fit, but severe burns, often of the face, fractures and dislocations, occur comparatively frequently in chronic epileptics. Marriage cannot be advised, as the likelihood of occurrence of epilepsy or nervous instability in the family or succeeding generations, is very great. If marriage is undertaken

the condition should be explained to both partners and "protected" marriage advised.



(*a*) (*b*)

1. Right Frontal; 2. Left Fronta ; 3. Right Parietal;
4. Left Parietal; 5. Right Occipital; 6. Left Occipital.

FIG. 174.—(*a*) Normal E.E.G. record. The horizontal line represents 1 second and the vertical signal 50 microvolts. Alpha rhythm of 10 cycles per second and of relatively high voltage is seen arising in the occipital region.

(*b*) Part of a record from a patient with idiopathic epilepsy. An outburst of high-voltage waves of 3 per second, alternating with spikes, occurs simultaneously in all leads and lasts for 4 seconds.

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Etiology.—Both sexes are equally affected. The disease is only rarely heredo-familial, but a history of nervous instability in the family —migraine, eccentricity, alcoholism, insanity, is common.

Treatment.—Three factors will precipitate attacks: (1) Constipation, (2) Overeating, (3) Emotional excitement or worry. These should be avoided. Often, simple restriction of diet, and of eating between meals (many chronic epileptics are gluttonous) and correction of constipation, will diminish the frequency and severity of the attacks. If the occupation can be chosen, an open-air one should be advised. Epileptics should be excluded from all work involving danger. Possible safe occupations are farming and gardening, or indoor work at home. Most epileptics, however, lose their employment. Riding, swimming, and driving a car or riding a bicycle, should be forbidden. Alcohol is contra-indicated. *Where there is an epileptic in the family all fires should be protected with metal screens.*

Diet.—In severe cases a white diet is advisable, red meat is allowed occasionally. In children, a ketogenic diet alone may reduce or entirely stop the attacks. This treatment is not effective in adults. Fat is increased, while carbohydrate and protein are cut down so as to produce ketosis. Fats are given as bacon, suet-pudding, extra rations of butter or margarine, toast dipped in bacon-fat, etc. Not every child can be made to take such a diet. *Drugs.*—The treatment of epilepsy is by medicines. This should be impressed on the patient, who will often

foolishly break the continuity of his treatment after a bout of attacks, attributing his seizures, not to his malady, but to the "drugs" he is taking. In other cases, depression or some other emotional condition is attributed to the medicine, which is stopped by the patient or the parents. If these things are explained at the outset, the likelihood of this happening is less. *In prescribing for epilepsy remember to time the dose of the medicine to precede the occurrence of the fits.* If the fits are all nocturnal, give the medicine last thing at night. If the fits all occur just after the patient gets up in the morning, give the medicine an hour before rising. In female patients, whose attacks occur round about the menstrual periods, the dose of the medicine should be increased, perhaps doubled, just before those times, and a brisk saline purgative, *e.g.*, Seidlitz powder, given in the morning. *Bromide* is probably the most useful drug. It has the disadvantage of causing unsightly, pustular skin eruptions—"bromide acne." This is obviated, to some extent, by giving arsenic in the medicine and restricting salt in the diet. The following mixture may be prescribed once to three times a day: potassium bromide gr. 10–15, liq. arsenicalis ℥ 1–2, tinct. belladonnæ ℥ 5, aquam ad ½ fl. oz. Insoluble *phenobarbitone B.P.* (*luminal*) in tablet form or the soluble sodium phenobarbitone may be given in ½ gr. to 1 gr. doses, once to three times a day. This drug may cause erythematous rashes with dizziness and drowsiness, if employed in larger doses. Patients acquire tolerance to phenobarbitone. It is a useful adjunct to bromide; it is effective in very small doses and can be given in tablet form, which is convenient. In most cases it should be prescribed with a daily dose of bromide, not alone. For cases who do not tolerate bromide, *borax* or potassium borotartrate may be given in 10–15 gr. doses. *In Epilepsy, if the administration of drugs is suddenly stopped, Status Epilepticus may ensue.* The medicines must be continued with religious regularity until the patient has been free from attacks for five or six years. *Sodium-diphenyl-hydantoinate* (*Sodium phenytoin, Epanutin*) is used, particularly for cases of grand mal, but only after all other remedies have been tried first. It is of value in the treatment of psychomotor attacks. Capsules contain 1½ gr. (for infants ¾ gr.). In giving epanutin one dose of phenobarbitone is replaced by a capsule of epanutin for the first week, and the phenobarbitone gradually replaced by epanutin up to 3–5 capsules daily. Spongy gums, tremors, ataxia, diplopia, and scarlatinal or morbiliform rashes are some of the toxic symptoms observed after its use. Should albumen or urobilin appear in the urine during its administration, the dose should be diminished or the drug stopped. *Tridione* (0·3 G.) in some cases will control petit mal attacks (not grand mal), but this drug is toxic and should not be given unless the patient is under continuous expert supervision. Frequent blood counts are necessary as it may cause agranulocytosis.

Treatment in Attacks.—Put the knotted edge of a handkerchief or a wooden pencil between the teeth. Loosen the clothing round the neck,

remove spectacles and false teeth and prevent the patient damaging himself in the clonic stage. *Treatment of Status Epilepticus.*—(1) Give immediately morphine gr. $\frac{1}{4}$–$\frac{1}{2}$, alone or with hyoscine hydrobromide gr. 1/100. (2) Wash out the colon by means of a soap and water enema, followed later by lavage with water or saline. (3) If the fits have not ceased, give 4 drachms of paraldehyde per rectum with an equal quantity of olive oil and repeat in six hours if necessary. Temperature is lowered by tepid sponging. (4) The patient should be fed (nasally, if necessary) with glucose and milk feeds to combat the acidosis. Inhalations of chloroform may be necessary to control the attacks.

§ **723.** B. SYMPTOMATIC EPILEPSY.—Epilepsy may be the presenting symptom, particularly in patients where the first fit occurs after the age of 25 years, of an intracranial tumour, cerebral syphilis, cerebral arterio-sclerosis, or even of cardiac or renal disease. Intracranial cysticercus cellulosæ may cause epileptiform attacks. In all these conditions the attacks may be focal Jacksonian attacks (motor, sensory, visual, auditory, gustatory) or generalised convulsions. The Jacksonian attacks of focal organic cortical lesions are sometimes distinguishable from those of idiopathic epilepsy by the fact that the paralysis following the fit (Todd's paralysis) becomes progressively greater and greater after each seizure, until a permanent residual monoplegia develops.

IV. CEREBRAL TUMOUR (§ 828).—For weeks or months generalised epileptic convulsions may be the only sign of cerebral tumour. With cortical neoplasms, focal Jacksonian attacks occur as the irritative sign, the type of attack varying with the site of the lesion.

V. **Cerebral Syphilis.**—The "congestive attacks" of G.P.I. may be *focal* (followed by transient paresis or aphasia) or *general.* In any form of cerebral syphilis epilepsy may occur. The finding of Argyll-Robertson pupils, cranial nerve palsies, or a history of syphilitic headaches or history of infection, indicate the diagnosis, which is made by examining the spinal fluid. The spinal fluid Wassermann reaction will be positive in nearly all the cases. When the syphilitic disease is mainly vascular (*e.g.*, syphilitic hemiplegia from endarteritis), not meningeal, the Wassermann reaction may be positive in the blood and negative in the spinal fluid.

VI. **Cerebral Arterio-sclerosis** (Synonym : Arterio-sclerotic Epilepsy).—Epilepsy, due to anoxæmia or the formation of mural thrombi in the diseased vessels, occurs in patients with high blood pressure, retinal arterio-sclerosis and hæmorrhages, and a history of "strokes."

VII. The HYPERTENSIVE ATTACK is described in § 94. I. *c.*

VIII. **Traumatic Epilepsy.**—Following a severe head injury (often with sepsis), generalised epileptiform convulsions may occur. In cerebral contusions and depressed fracture, with meningeal and cortical injury, vascular brain scars form and exert traction on the cortical areas, with resulting Jacksonian convulsions. In this latter group surgical removal of the scar may alleviate the condition, otherwise the treatment is as in idiopathic epilepsy.

IX. In CONGENITAL DISEASE of the central nervous system, *e.g.*, syringomyelia, cerebral diplegia and hydrocephalus, fits may occur.

X. STOKES-ADAMS' SYNDROME, in incomplete heart-block, is described in § 69.

XI. CEREBRAL HÆMORRHAGE or EMBOLISM may be ushered in by fits, followed by a flaccid hemiplegia.

XII. Asphyxia, from a foreign body in the larynx, may give rise to unconsciousness, with generalised convulsions, especially in children.

XIII. Forced Deep Breathing will induce epileptic fits in certain individuals.

XIV. Toxic Convulsions occur as the result of (*a*) metabolic poisoning, *e.g.*, *Uræmia* (test the urine, estimate the blood pressure and look for albuminuric retinitis). *Eclampsia* (when the fits occur before, during, or after labour and are associated with albuminuria and signs of toxæmia), *Acute Yellow Atrophy of the Liver* (with jaundice), *Insulin hypoglycæmia* (look for prick-marks on the limbs) or (*b*) *Drug Poisoning, e.g.*, Alcohol, Absinthe, Neoarsphenamine, Lead Poisoning, Belladonna, Hydrocyanic Acid.

XV. Acute Encephalitis, and

XVI. Malaria, especially Cerebral Malaria, may be attended by epileptiform fits.

XVII. **Intracranial Cysticercus Cellulosæ** (*T. Solium*) is a rare cause of focal or generalised convulsions, especially in those who have lived abroad (§ 316). Cysticerci may be present in the subcutaneous tissues or muscles as hard, oval, pea-like bodies. If calcified, the nodules show up in radiograms of the skull or skeletal muscles.

XVIII. Presenile Dementia may first be manifested by recurrent fits in a person past middle age (§ 903).

§ 724. Infantile Convulsions.—In circumstances causing rigors in an adult, convulsions occur in infants. Convulsions in infants may also be due to local disease of the brain or its membranes, or to toxic causes. Recognition of the convulsion is easy. In determining the cause a consideration of the child's age is important.

A. Convulsions in the First Three Months of Life.

1. Birth Injuries, *e.g.*, Tentorial laceration.
2. Cerebral Diplegia and Cerebro-Macular Degeneration.
3. Congenital Hydrocephalus.
4. Congenital Syphilis.

B. Convulsions from Six to Eighteen Months of Life.

5. Rickets with Tetany.
6. Gastro-intestinal Causes—Over-feeding, Unsuitable Food, Constipation.
7. Acute Infective Disorders—Broncho-pneumonia and Measles, Tonsillitis, Bacilluria.
8. Cerebral and Meningeal Causes—Meningitis, Polioencephalitis and other forms of Encephalitis.
9. Reflex Causes—Teething, Round-worms, Rectal irritation from Thread-worms.
10. During Cerebral Venous Congestion—in Whooping-Cough.

C. Convulsions after the age of Two Years.

11. Epilepsy is the commonest cause where the child is otherwise apparently healthy.

The commonest causes are rickets, gastro-intestinal disorders and acute infective diseases. Epilepsy may start at an early age and may be the cause of unexplained convulsions in infancy.

Treatment.—The rectal temperature should be taken and an attempt made to

(1) *Control the Convulsion.*—Immerse the infant in a warm mustard bath for ten to fifteen minutes, then place in a warm bed and give an enema, or insert a rectal glycerine suppository. The administration of chloroform, by inhalation, will immediately control the fits. Chloral

hydrate gr. 4, and sodium bromide gr. 5, may be injected through a catheter into the bowel and repeated in an hour. Next

(2) *Investigate the Cause.*—(*a*) Question the mother or nurse about the *diet*, with special attention to the question of over-feeding, unsuitable food or constipation. (*b*) Search for a history or signs of rickets and tetany. (*c*) Make a general physical examination of the child for rash, any source of sepsis (throat and ears), examine the chest, look for head retraction squint or Kernig's Sign, and lastly (*d*) Examine the stools and urine.

GROUP III. PYREXIA, WITH SIGNS OF ORGANIC NERVE DISEASE

Pyrexial disorders of the nervous system are not numerous, and most of them are due to acute inflammation of the meninges. In this group are considered certain conditions, all of which present *neurological symptoms* with PYREXIA and its attendant symptoms.

CLINICAL INVESTIGATION.—(1) Obtain a complete *Personal and Family history*, especially with regard to tuberculosis, previous suppurative ear disease, bronchiectasis, pneumonia, empyema, etc. In tuberculous meningitis there is often a history of preceding weeks of malaise. History of a rigor or fit may indicate the formation of a cerebral abscess. (2) The *age* of the patient is of some importance. Post-basic meningitis is almost confined to infants under one year. (3) Look for *signs of meningeal irritation*, *e.g.*, stiffness of the posterior cervical muscles, positive Kernig's Sign, etc. (4) Make as detailed a *neurological examination* as possible with special reference to the cranial nerves, tendon reflexes and plantar responses, signs of paresis in the limbs. Examine the fundi for optic neuritis (intracranial abscess), hæmorrhages (subarachnoid hæmorrhage) or tuberculous deposits (tuberculous meningitis). (5) Examine the *scalp, cranial bones, mastoid and other air sinuses, the ears, throat and lungs*, for evidence of infection. Œdema of the face, scalp or neck should be noted. Suppurative sinus disease is accompanied by facial œdema, extradural abscess by Pott's puffy tumour of the scalp, and sinus thrombosis by orbital œdema or œdema of the neck. (6) In nearly all cases it is necessary to examine the *spinal fluid* for Pressure, Cells, Organisms, Total Chlorides, Glucose, Total Protein, etc. (7) The *temperature* should be recorded on a four-hourly chart.

The following Clinical pictures may be encountered:

A. A patient with a *Chronic Nervous Disease* or an *Acute Non-Infective Nervous Disease* shows *Transient Pyrexia* .. § 725

B. A patient shows *Pyrexia* with signs of *Meningeal Irritation* § 726

C. A patient shows *Pyrexia* with subsequent *Flaccid Paralysis* .. § 732

D. A patient shows *Pyrexia* accompanied by *Rapidly Ascending* or *Descending Paralysis* .. .. § 734

E. A patient with a *Suppurative Focus* in the *Middle Ear, Mastoid, Accessory Air Sinuses, Thorax or elsewhere*, develops *Pyrexia* with *Localising Nervous Symptoms* .. § 737

F. Following upon *Vaccination* or an *Acute Specific Fever*, a patient develops *Pyrexia* and *Nervous Symptoms* .. § 740

G. A patient develops mild *Pyrexia*, with *double vision, ptosis, ocular palsies and lethargy* .. .. § 698

A. *A patient with a Chronic Nervous Disease or an Acute Non-Infective Nervous Disease, shows* TRANSIENT PYREXIA.

§ 725. In **chronic diseases of the nervous system** transient fever may occur from INFECTION OF THE BLADDER OR INFECTED BED-SORES. Patients with the remitting type of DISSEMINATED SCLEROSIS will often give a history of "influenzal attacks" which are doubtless pyrexial attacks associated with the laying down of fresh plaques. Fever of 102°–104° F. may occur in the "congestive attacks" of GENERAL PARALYSIS OF THE INSANE, and it should not be forgotten that there are many such cases with RESIDUAL MALARIAL INFECTION following malarial treatment. In acute cerebral lesions of any kind, notably INTRA- or EXTRA-CEREBRAL HÆMORRHAGE or CEREBRAL CONTUSION, the temperature is subnormal during the initial stages of collapse, but subsequently rises above normal. In hæmorrhage into the *pons* hyperpyrexia is frequent, and may be as much as 108° F. Convulsions occur, affecting chiefly the legs, with coma, bilateral extensor responses and strongly contracted pupils. Death occurs rapidly. The occurrence of "nervous fever" has, in fact, no foundation. Lesions of the *hypothalamic region* and the *tuber cinereum* may cause hyperpyrexia.

We are here, however, chiefly concerned with infective conditions of the nervous system, mostly of acute onset, where there is a combination of nervous symptoms and pyrexia..

B. *A patient shows* PYREXIA *with signs of* MENINGEAL IRRITATION. The condition to be suspected is MENINGITIS.

§ 726. *The Clinical Picture of* **Meningeal Irritation.**—There is intense occipito-cervical headache with photophobia and great sensitiveness to noise, pain in the back, rigidity of the posterior cervical muscles and spine, and retraction of the head. The patient is irritable, later drowsy, and finally comatose. He lies on his side curled up and may be resistive if disturbed. In the early stages retention of urine is a common feature and should always be looked for. There are bouts of restlessness, with a monotonous, rather high-pitched wailing cry.

The only signs may be the characteristic headache and stiffness of the neck (§ 707). *Kernig's Sign* is inability to extend the knee with the thigh flexed at a right angle on the abdomen; it depends on spasm of the hamstrings. *Brudzinski's Sign* is the appearance of similar flexion of the lower limbs and sometimes also the upper limbs, when the head is acutely flexed on the chest.

In meningococcal and tuberculous meningitis other features complicating this clear-cut clinical picture may appear, depending on the spread of the inflammatory process. The knee- and ankle-jerks may disappear, signs of hemiparesis, pupillary abnormalities, squints and other cranial nerve palsies are often found.

Types of Meningitis (Lepto-meningitis).

MENINGOCOCCAL MENINGITIS (Syn. Cerebrospinal fever). See § 503.

§ 727. **Tuberculous Lepto-meningitis** is usually part of a general miliary tuberculosis, the meningeal over-shadowing the other signs. Metastatic infection can also occur by erosion into a vein of caseating material from a mediastinal or mesenteric gland, or from a focus in bone or lung. It is commonest in children under the age of five years, running an acute and

TABLE XLVIII.—CAUSES OF MENINGEAL SYMPTOMS.

	Mode of Onset.	*Clinical Picture.*	*Signs of Disease Elsewhere.*	*Spinal Fluid.*
Subarachnoid Hæmorrhage. (§ 717).	Sudden, with intense headache and deepening coma. History of migraine.	Meningeal irritation, with ocular palsies, hemiparesis, root-pains, massive albuminuria.	Usually none. Sometimes hyperpiesis, renal or blood diseases.	Pink or yellow from laked blood. Sterile.
Tuberculous Meningitis (§ 727).	History of preceding weeks of malaise. Family or personal history of Tuberculosis.	Meningeal irritation with pupillary abnormalities, squint, absence of deep reflexes and abdominal retraction.	Signs of Miliary Tuberculosis.	Clear or yellow, with "web-clot" containing Tubercle Bacilli. Diminished glucose and chlorides.
Benign Lymphocytic Meningitis (§ 728).	Subacute in young adults.	Meningeal irritation but patient otherwise in good health.	None.	Always clear and free from clot. Glucose and chlorides not reduced.
Meningococcal Meningitis (§ 503).	Gradual onset. Perhaps rash.	Intense head-retraction in post-basic variety.	None, or signs of Cerebro-spinal fever.	Turbid fluid with flocculi. Meningococci.
Acute Poliomyelitis (§ 732. I).	Acute Onset, during epidemics.	Meningeal irritation. Later, flaccid paralysis.	None.	Clear, sterile, with lymphocytosis and normal chlorides.
Pneumococcal and Pyogenic Meningitis (§ 729), or *Leaking* Cerebral Abscess (§ 737).	Acute Onset..	Meningeal irritation with ocular palsies and disappearance of deep reflexes. Localising signs in abscess.	Suppurative focus in mastoid, lungs, or elsewhere.	Purulent fluid, with causal organism. Fluid may be sterile in leaking abscess.
Meningism (§ 731).	Acute Onset.	Meningeal irritation only.	Bronchopneumonia or Apical Pneumonia, acute infectious diseases, Bacilluria, Hysteria.	Normal.

fatal course, but it also occurs in adults when it tends, in some cases, to assume a more chronic form.

Symptoms.—In children the early symptoms are most insidious. The child loses appetite, becomes thinner, peevish and apathetic. A slight irregular fever and constipation are common and occasional vomiting occurs. Complaints of pain or discomfort in the neck should never be ignored. Such symptoms *persist* over a period of several weeks and then become rapidly worse. It is in this early stage that treatment may help the patient. In the *irritative stage* the meningeal signs are well-marked with headache, neck rigidity, vomiting and photophobia. Persistent severe vomiting suggests hydrocephalus. Later, signs of *compression* appear—drowsiness, abdominal retraction, flaccidity and loss of tendon reflexes, followed by *coma*. In adults the disease may run a course of weeks or months. Cases of *benign lymphocytic meningitis* closely simulate the tuberculous variety in the cerebro-spinal fluid findings, but in these recovery is usually complete and the chlorides never fall as they do in tuberculous meningitis.

Diagnosis.—Examination of the cerebro-spinal fluid should not be delayed when the disease is suspected. The encouraging results from streptomycin treatment are in cases diagnosed and treated early. At least two adequate samples of fluid should be collected by spinal puncture and the fluid should be examined for cells, total protein, globulin, chlorides, and glucose. Centrifuged deposits should be searched for tubercle bacilli, cultures should be made and a guinea pig injected. In the early stages the fluid is clear and perhaps under pressure. On standing and cooling a "spider web" clot may form, in the meshes of which tubercle bacilli may be entangled. If the cells are more than 300 per cu. mm. the fluid may be opalescent. Up to 30 per cent. of the cells may be polymorphs, but lymphocytes predominate. The chloride level frequently falls to 650 or 600 mgm. per cent. and may be as low as 500 mgm. per cent. These low chloride readings are highly characteristic of the disease (normal is 720–750 mgm. per cent.). The glucose is diminished. Other tests are less helpful but may have to be carried out, viz., (1) Mantoux test (see § 521), (2) radiograms of chest, (3) choroidal tubercles are found in the ocular fundi early in the disease in as many as half the cases, when the spread is a miliary one.

Course and Prognosis.—The discovery of streptomycin by Waksman (1944) has modified the course and perhaps the prognosis of a disease which was almost invariably fatal. In children untreated cases are invariably fatal within three weeks of the appearance of meningeal symptoms. In cases treated with streptomycin the mortality under the age of three years is still very high, but in adults 30 to 40 per cent. are "in good clinical condition" six months after the onset. A few of the remitted cases are left with permanent optic atrophy, squint, dysphasia or mental disorder, but these complications are exceptional. Relapses occur and it is still too early to assess the final results.

Treatment.—Symptomatic treatment consists of repeated spinal puncture to relieve headache, with ice-bags to the head, chloral hydrate, codein and other analgesic drugs by mouth or by nasal tube to control pain and restlessness; with avoidance of noise and bright light. In stuporose patients a nasal tube for feeding is essential.

Streptomycin is available in England at special hospital centres. Combined intramuscular and intrathecal injections are used. *Intramuscularly* the daily dose advised is 0·02 G. per lb. of body weight, given twelve hourly in divided doses, for three to six months. *Intrathecally* the daily dose advised is 0·05 to 0·1 G. in one dose, given six hours after the last intramuscular injection. If spinal block develops, the streptomycin must be given into the ventricles through burr holes made in the skull.

§ 728. Benign Lymphocytic Meningitis. The *Signs* are those of meningitis running a sub-acute course in young adults. Constitutional disturbance is usually slight. At first the illness may be mistaken for tuberculous meningitis or acute poliomyelitis, owing to the large number of lymphocytes found in the sterile spinal fluid. The chloride content of the

fluid, however, does not fall as in tuberculous meningitis and signs of muscular paresis are absent. The disease may be due to a virus similar to that producing chorio-lymphocytic meningitis in mice. In some cases the symptoms come on after acute catarrhal infections of the upper respiratory tract. The illness runs a benign course but an excess of lymphocytes may persist in the spinal fluid for weeks after all clinical signs have disappeared. *Treatment* is symptomatic.

§ 729. Pyogenic and other forms of Acute Lepto-meningitis. Most of these cases are secondary to a focus of infection outside the nervous system. The organisms concerned are the *Pneumococcus*, *Streptococcus*, *Staphylococcus*, *Gonococcus* and *H. influenzæ*.

Pneumococcal Meningitis may be secondary to otitis media, empyema, pneumonia, pneumococcal peritonitis or arthritis. The spinal fluid is sometimes too thick to flow through a lumbar puncture needle: it is greenish-yellow and contains abundance of polymorphonuclear leucocytes amongst which the pneumococcus is found. The condition develops rapidly, coma supervenes early and before the introduction of sulphonamides the disease was nearly always fatal. In many cases there is an associated blood infection.

Streptococcal Meningitis may be secondary to middle-ear or mastoid disease, erysipelas, or other infections of the scalp, face or cranial air sinuses. *Staphylococcal* cases are secondary to skin or bone infections. *Gonococcal* cases occur. *B. Pyocyaneus Meningitis* may be caused through accidental infection during intrathecal administration of penicillin. *Influenzal Meningitis* is a rare cause of purulent meningitis in children.

The C.S.F. findings in such cases are shown in Table LXI.

Treatment of Acute Lepto-meningitis. Before giving either sulphonamides or penicillin, endeavour to identify the causal organism in the C.S.F. This usually means withdrawing at least two adequate samples of spinal fluid and its laboratory examination as soon as possible afterwards. If this is made a rule, much subsequent confusion will be avoided. Oral *Sulphadiazine* is the sulphonamide most commonly used in treatment. It is active against the meningococcus, pneumococcus, streptococcus hæmolyticus and staphylococcus aureus. Give an initial dose of 4 G. by mouth (by nasal tube, or intramuscularly, if the patient is unconscious) followed by 2 G. doses by mouth four-hourly, for four or five days; then half-doses for three days. The patient must be wakened up for his four-hourly doses through the night (see Tables XXVIII and XXIX). Systemic *penicillin* does not pass the blood-brain barrier and is therefore only an adjuvant to treatment with sulphadiazine. Give 120,000 units intramuscularly daily for four or five days. It is safe to do this in an unidentified meningitis if sulphadiazine is given as well. Penicillin should never be the only form of chemotherapy in acute meningitis. Very rarely it may be necessary to give intrathecal penicillin when the causal organism is penicillin-sensitive and is not reacting to other treatment. Penicillin intrathecally is to some extent an irritant and may cause headache, stupor

and fits. Pure crystalline penicillin should be used and the greatest care taken to avoid bacterial contamination, for B. pyocyaneus meningitis is very difficult to cure. Give 10,000 units in 10 c.c. warm sterile saline intrathecally, and repeat in twelve hours. Often these two doses are sufficient: if not, the dose may be repeated once daily for three days. If there is a spinal block, the penicillin may have to be given by ventricular or cisternal puncture, burr holes being drilled in the skull for the ventricular administration. In all cases when the patient is unconscious, a nasal tube should be passed into the stomach so that adequate fluids and feeds can be given.

The primary focus of the meningitis may have to be dealt with surgically.

Anthrax Meningitis is a rapidly fatal form. The spinal fluid is hæmorrhagic. **Blastomycotic meningitis** is a very rare form secondary to infection of the skin or lungs with organisms of the yeast group.

§ **730. Diagnostic Spinal Puncture in Meningitis.**—In all cases of the meningeal syndrome, a diagnostic spinal puncture will be necessary to determine whether there is meningeal infection or whether the syndrome is due to SUBARACHNOID HÆMORRHAGE or MENINGISM. In meningeal infection the fluid analysis will determine the *type of meningitis* and its *bacterial cause.* This laboratory information is essential before treatment can be started. It is reasonable to give oral sulphonamides and systemic penicillin until the bacteriology can be determined as accurately as possible (see Tables XLVIII, LXI).

Recurrent meningeal infection may take place from a focus of infection in the extra-dural or sub-dural space or from a leaking intra-cerebral abscess.

§ **731. Meningism** is a condition of meningeal irritability characterised clinically by the picture of meningeal irritation but with normal spinal fluid. The condition closely simulates meningitis and occurs especially in children, in *Broncho-pneumonia* and *Apical Lobar Pneumonia*, at the onset of *Acute Specific Fevers*, *Otitis media* and during severe *Pyelitis.* In adults meningism occurs in *Apical Pneumonia*, *Typhoid*, *Acute Tuberculosis* and without fever in *Hysteria* (hysterical pseudo-meningism).

C. *A patient shows* PYREXIA *with subsequent* FLACCID PARALYSIS. *The condition is either* I. ACUTE POLIOMYELITIS; II. ACUTE INFECTIVE RADICULITIS; *or* III. HERPES ZOSTER.

§ **732.** I. **Acute Poliomyelitis** (Synonyms: Infantile Paralysis, Acute Anterior Poliomyelitis and Polioencephalitis) is an acute specific fever of sudden onset, resulting in loss of power and rapid wasting of one or more groups of muscles. No age is exempt, but the disease usually affects children between the ages of two and five years. During the first year of life there appears to be relative immunity from infection and the disease is uncommon after middle life. The *incubation period* is probably about twelve days. Two stages are recognised: (i.) the pre-

paralytic stage and (ii.) the paralytic stage. The virus seems to invade the nervous system exclusively, and may reach the motor nerve cells before any symptoms appear.

Symptoms.—The symptoms are those of a mild febrile attack and last a few days, rarely more than seven. Paralysis may or may not follow. (1) The *Pre-Paralytic Stage* passes unnoticed in the majority of cases. Pyrexia, general malaise and fretfulness occur, with pain in the limbs. The muscles are tender to pressure and the joints painful when the limbs are moved. Local symptoms: (i.) redness of the tonsils and fauces, (ii.) coryza, with streaming of the eyes or (iii.) gastro-intestinal upset, with anorexia, vomiting and diarrhœa. In more severe cases there is delirium and stupor. Rigidity of the posterior cervical muscles and spine appears, with severe headache and irritability, pains in the limbs, muscular twitchings, positive Kernig's sign, and in severe cases, incontinence. In the early stages the *blood* shows a polymorphonuclear leucocytosis, which may be marked (30,000 per cu. mm.).

The *spinal fluid* is now found to be under increased pressure, clear or opalescent, and it shows a lymphocytosis (occasionally, polymorphs are found), in numbers varying with the severity of the meningeal reaction. The albumen content is increased. The glucose and chloride content is normal; by the end of a fortnight the cell-count is usually normal. The combination of lymphocytosis in the spinal fluid with polymorphonuclear leucocytosis in the blood is quite characteristic.

In some cases (50 per cent. of cases in epidemics) the patient recovers without paralytic symptoms (*Abortive* or *Meningeal* types of the disease). Convalescent serum, even if administered early and intrathecally, does not avert paralysis.

(2) *The Paralytic Stage.*—(*a*) *Spinal Type.*—In infants the paralysis appears two or three days after the onset of the symptoms; in adults the paralysis appears earlier. The paralysis varies in degree: it is (i.) flaccid; (ii.) the corresponding tendon reflexes are lost; (iii.) the affected muscles are very tender, but there is no sensory impairment. (iv.) The paralysis most often affects the legs, and at first appears widespread, but residual paralysis is usually much less. (v.) There may be retention of urine, which soon passes off. Ascending and descending paralysis may occur, especially in adults, and may be rapidly fatal from respiratory paralysis. (*b*) In the *Brain-stem type* the motor nuclei of the brain-stem are affected. This causes facial paralysis, ocular palsies with nystagmus, motor trigeminal or bulbar palsies. The facial paralysis in these cases is usually of the peripheral type and tends to recovery. (*c*) *Cerebral, Cerebellar and Neuritic* forms are sometimes described. The *Cerebellar type* is said to produce cerebellar ataxy without nystagmus; rapid and complete recovery ensues as in other comparable acute cerebellar lesions. Poliomyelitis affecting the cerebrum is called *Polioencephalitis.* In this, mental confusion and restless delirium persist for some days after the pyrexia has subsided. Polioencephalitis is believed

to be a cause of infantile hemiplegia and perhaps some cases of epilepsy.

Diagnosis.—Careful clinical examination usually discloses swelling and redness of joint or joints in *acute rheumatism* and enlargement of the epiphysis in *syphilitic epiphysitis.* In both these diseases the tendon reflexes are brisk, not lost as in poliomyelitis. Examination of the spinal fluid settles the question. In *infantile scurvy* the affected limbs are tender and swollen (§ 545). The ascending and descending types of poliomyelitis closely resemble *acute febrile polyneuritis* and *Landry's paralysis*, but are distinguished by the spinal fluid findings. In *encephalitis lethargica* (§ 698) clot and polymorphonuclear cells are not found in the fluid. In *acute myelitis* there are sensory loss, bed-sores and incontinence (§ 736). In the convalescent stage of flaccid paralysis there is rarely any difficulty in diagnosis. The diagnosis is made certain by spinal puncture. The differential diagnosis from *tuberculous meningitis* is made by the finding of normal amounts of chloride and glucose in the spinal fluid, the absence of tubercle bacilli, and the polymorphonuclear leucocytosis in the blood in Poliomyelitis. Clear fluid and clot are common to the two conditions.

Etiology.—Noguchi and Flexner in 1913 isolated a neurotropic virus believed to be the cause of the disease. It is found in the brain and spinal cord of cases dead of the disease, and also in fæces, the intestinal canal and naso-pharynx. Unfortunately the only method of demonstrating the virus is by injection of emulsions of suspected material into certain monkeys, thus producing disease in the inoculated animals. The disease is believed to spread from healthy carriers and also from one infected individual to another, but whether by infected fæces or by "droplet" infection is unknown. The roles of water, milk and fomites in the spread of the disease is not understood, but patients' fæces should be treated as infective. The virus reaches the nervous system from the naso-pharynx or intestinal canal and the main incidence of the disease is on the motor cells of the spinal cord and brain stem.

Prognosis.—(1) The slight unrecognised forms of Poliomyelitis cause many orthopædic deformities. (2) The mortality in epidemics ranges from 11 to 20 per cent. and is greatest in the spreading form of the disease. (3) The severity of the general or the meningeal symptoms bears no relationship to the amount of residual paralysis, which may be severe when the general symptoms are slight, and *vice versa.* (4) The amount of paralysis present at the beginning of the paralytic stage is commonly much greater than the ultimate residual paralysis, except in the ascending or descending form, where further extension may occur for a few days. (5) Muscles incompletely paralysed will certainly recover. (6) The final prognosis as to ability to use the affected limbs or limb depends on whether the muscles affected perform essential functions in walking or prehension, and on certain orthopædic considerations. No case should be regarded as hopeless until months or years have elapsed, as recovery is often long

delayed. One attack confers immunity. The infectivity varies in degree in different epidemics. For Electro-prognosis, see § 709.

Treatment.—Hexamine gr. 10–30, sodium bicarbonate gr. 20, potassium citrate gr. 20, may be given in a mixture, well diluted, four-hourly. For the pains give aspirin. If the respiratory muscles are involved, and in the spreading types of the disease, the patient should be well propped up and atropine gr. 1/100 given four-hourly, to check the accumulation of bronchial secretion. The nose and nasopharynx should be sprayed with hydrogen peroxide (10 volumes) and an equal part of normal saline. *Isolation.*—This should be as strict as for other specific fevers. The infectivity probably continues for two or three weeks from the onset.

Rest and Posture.—The patient should be kept in bed, even in the mildest cases, for at least three weeks. The patient should be nursed on a water-bed or air-bed during the painful stages of the malady, which may last some weeks. When the erector spinæ muscles are involved, the patient should usually be nursed in the recumbent position, but in all other cases should be propped up. The affected muscles should not be allowed to over-stretch, otherwise appalling deformities from *contracture* will ensue. The relaxation of the affected muscles may be secured by sand-bags, bed boots, celluloid or poroplastic splints and pillows. *All apparatus should be of the lightest possible kind.* Plaster splints, which should be removable, are sometimes used. In a very few cases where the respiratory muscles are severely involved, life may be preserved by Drinker's or the Paul-Bragg Artificial Respiration Apparatus for weeks or months until recovery starts. Many cases will show no recovery, and the horrible situation may arise where life is possible only in the apparatus.

Re-education is of paramount importance in the treatment of the paralytic stage. Any voluntary movement performed by the patient is of much greater value in recovery than the same movement performed passively by a masseuse. Massage must never be regarded as an adequate substitute for active exercises. Faradism certainly cannot hasten recovery; it should never be used in the painful stage, nor can it replace re-educative exercises. Re-educative exercises may be assisted by immersing the limbs in a brine bath or may be undertaken in a salt-water swimming-bath, the water affording additional support to the affected limbs. A walking-machine on wheels is often necessary in re-education of the lower limbs. Contracture and deformity should never occur. When they have appeared they should be dealt with by passive manipulation and stretching, and by tenotomy or other orthopædic measures. In old paralysed limbs, cyanosis, œdema and chilblains may be relieved by lumbar sympathectomy.

Prophylaxis.—In the absence of precise knowledge regarding infection it is difficult to lay down dogmatic rules. When more than one case occurs in a boarding school, the prevailing custom at present is to quarantine the school for three weeks. Parents are given the option of leaving their child at school, or removal home: but as healthy children have to be isolated

for three weeks from other children and adolescents, parents often prefer to leave the child in the quarantined school. Cases which develop the disease should of course be rigidly isolated, treated as contagious and nursed with precautions as for typhoid fever. Nurses should wear gauze masks. The patient's stools and handkerchiefs are infectious. Contacts should not be allowed to handle food for four weeks. Human convalescent serum is not now believed to be of any value in treatment and it is of very doubtful value given prophylactically to contacts.

§ 733. II. **Acute Infective Radiculitis** ("Neuralgic amyotrophy").—After an initial febrile phase usually with pain, there appears flaccid paralysis and sensory loss of root distribution. The disease affects chiefly the roots of the cervical and upper dorsal segments, but sometimes individual muscles may be picked out and it is difficult to explain the findings in terms of affected roots. Paralysis of serratus anterior, rhomboids or trapezius is observed analogous to that seen in Bell's palsy. The disease may attack the lumbar or sacral roots. It is distinguished from poliomyelitis by the associated sensory findings and by the absence of spinal fluid changes.

III. In HERPES ZOSTER (§§ 635, 826) the muscles near the affected area may be paralysed; ocular palsies, facial palsies and upper and lower brachial palsies may occur in herpes of the face and neck. Recovery is usually very slow; paralysis may be permanent. Treatment is by splinting and re-education.

D. *A patient shows* PYREXIA *accompanied by rapidly* ASCENDING *or* DESCENDING PARALYSIS. *The disease may be:* I. ASCENDING *or* DESCENDING POLIOMYELITIS; II. ACUTE FEBRILE POLYNEURITIS; III. LANDRY'S PARALYSIS; IV. ACUTE MYELITIS.

I. The Ascending and Descending Types of POLIOMYELITIS are described above.

§ 734. II. **Acute Febrile Polyneuritis** is a rare condition. Following a short febrile attack, spreading flaccid paralysis of muscles appears, with tenderness of muscles to deep pressure, pains, paræsthesiæ, numbness, tingling, and pins and needles in the chest and limbs, or diminution of deep reflexes. Occasionally fever is absent. Remissions and exacerbations occur. The paralysis differs from other forms of polyneuritis in that (1) it affects chiefly the proximal limb muscles, and (2) cranial nerve palsies, especially peripheral paralysis of the facial, are frequent. Sensory disturbances are of the "glove and stocking" type, or are absent, the plantar responses are flexor, there is no sphincter disturbance, no incontinence and no bed-sores. The affected muscles waste only slightly and retain some excitability to faradism throughout. The *cerebro-spinal fluid* contains excess of albumen and may be yellowish or brown, and may clot spontaneously, but is otherwise normal. (1) Toxic myocarditis may be present. (2) Rapid extension of the paralysis may occur as late as the sixth week and may prove fatal. (3) Some cases recover, but death may occur from respiratory failure.

The *diagnosis* from *Poliomyelitis* is made by (1) the presence of paræsthesiæ, (2) the slower progress of the paralysis, and (3) the spinal fluid findings. Other forms of polyneuritis affect the distal segments, but *diphtheria* and *diabetes* should be excluded.

§ 735. III. **Landry's Paralysis.**—This is probably the same disease as Acute Febrile Polyneuritis. Sensory disturbances are completely absent. The spinal fluid is hyperalbuminous but shows no other changes. In these two conditions there is no consistent pathological lesion present in the spinal cord or peripheral nerves.

§ 736. IV. **Acute Myelitis.**—In this disease a paraplegia develops acutely with a

febrile reaction, due to an unknown infective inflammatory process in the spinal cord. The patient is usually a young adult.

Symptoms.—(1) Marked paraplegia develops in association with fever, (2) pains in the back, and (3) numbness, tingling or a sense of *girdle constriction* round the body at the level of the lesion. (4) At the onset there is flaccid paraplegia with absence of tendon reflexes and retention of urine. (5) Sensory loss is present up to the level of the lesion. (6) Later, the paralysis becomes spastic, with reflex incontinence, the tendon reflexes are exaggerated, with clonus, and the plantars are extensor. Bed-sores develop. The paralysis and anæsthesia may ascend.

The *spinal fluid* may show the syndrome of Froin (*i.e.*, yellow coloration with hyperalbuminosis, formation of clot and relatively little pleocytosis), due to the formation of a zone of arachnoidal thickening and adhesion round the inflamed portion of the cord. If the spinal block persists, the syndrome of Froin may continue indefinitely in the spinal fluid.

Diagnosis.—The sensory loss and definite sensory level distinguishes this disease from other forms of spreading paralysis with fever, described above. This malady must be differentiated from other causes of acute paraplegia. (1) In *disseminated sclerosis*: evidence of disseminated lesions may be present clinically or ascertained by a careful history of previous illness. For other points, see § 755. (2) In *acute syphilitic meningomyelitis*: the Wassermann reaction is usually positive in blood and spinal fluid. (3) In the aged, sudden thrombosis of diseased arteries and veins, *myelo-malacia*, occurs. The presence of arterial disease, the age of the patient and the normal spinal fluid findings serve to differentiate these cases. (4) In *spinal cord compression*, particularly that associated with extension of tuberculous or pyogenic osteomyelitis of the vertebræ, the diagnosis may be very difficult, but is usually made by the slower onset of the symptoms.

Prognosis.—In the spreading and high cervical types of the lesion, death occurs in a few days from respiratory paralysis. There is a tendency in other cases for the disease to become arrested, usually with great residual disability.

Treatment.—See § 761.

E. *A patient with a* SUPPURATIVE FOCUS *in the Middle Ear, Mastoid, Accessory Air-sinuses, Thorax or elsewhere, develops* PYREXIA *with* LOCALIZING NERVOUS SYMPTOMS. The condition suspected is INTRACRANIAL ABSCESS.

§ 737. **Intracranial Abscess.**—The majority of cases arise from a chronic infection in the middle ear or mastoid antrum usually with cholesteatoma. Infection spreads directly through the carious tegmen tympani to the under surface of the temporal lobe, or posteriorly into the lateral lobe of the cerebellum. Frontal lobe abscess arises in association with pansinusitis or chronic suppuration in the frontal or ethmoidal air-cells especially in children. Metastatic abscesses arise from chronic suppurative infections in the pleura, lungs or long bones.

All degrees of infectivity exist. The most virulent infections cause *acute suppurative encephalitis* with extensive diffluent cerebral softening and rapid death. Milder and more chronic infections from contiguous foci in the air-sinuses cause *pachymeningitis* with formation of sub-dural or extra-dural abscess. Metastatic intracerebral abscesses are often multiple. Otitic abscesses tend to be loculated and if chronic, they become enclosed in a fibrous capsule, in which state they are more amenable to surgical drainage or excision.

Symptoms.—Very rarely the formation of a pyæmic brain abscess

causes a rigor with meningeal or focal neurological signs. In almost all cases of otitic and pyæmic abscess the signs are latent for several weeks. The earliest changes are an alteration in the patient's mental and general condition. Apathy, defective attention, difficulty in expressing ideas and defect of memory are found. The patient begins to look ill, to lose flesh and the tongue becomes coated. There may or may not be elevation of temperature. In intracerebral abscess the pulse is slow; in extra-dural abscess the pulse rate is raised. In all cases the blood usually shows a polymorph leucocytosis. Papillitis is a late sign and headache is rarely marked. *Local Symptoms* are never marked in *Temporal Lobe Abscess.* Slight paresis of the lower face, diminished or absent abdominal reflexes and extensor plantar responses on the contra-lateral side may be found. Abscesses extending backwards in the temporal lobe involve the optic radiations with production of upper quadrantic hemianopic field defects. If the abscess is in the left temporal lobe and the patient is right-handed, there will be dysphasia, sometimes "jargon aphasia." Jacksonian fits may occur from cortical irritation. Occasionally signs of mid-brain compression are present, squint with diplopia or paralytic dilation of the ipsilateral pupil. In *Cerebellar Abscess* coarse horizontal nystagmus may be present on looking to the side of the lesion. Flaccidity of the ipsilateral upper limb with undue extensibility of the muscles may be present. There is no alteration in the tendon reflexes and the upper and lower abdominal reflexes remain brisk; a point which may be valuable in distinction from temporal abscess. The progressive apathy and drowsiness of the patient soon render clinical examination almost impossible. *Spinal fluid.*—Cells are perhaps 5–30 per cu. mm. and are mostly lymphocytes with a few polymorphs. This is the characteristic finding, but if the abscess is thick-walled, the cytology of the fluid may be normal. Protein may be increased. Glucose and chlorides are normal unless the abscess leaks, when signs of recurrent meningitis become apparent clinically, with fall in the cerebro-spinal fluid chlorides and rise in cell count.

Diagnosis is often extremely difficult. In selected cases air ventriculogram may be of great assistance in diagnosis, revealing abnormal dilation, displacement, or filling defects of the ventricles.

Treatment.—The treatment of intracranial abscess is surgical and the results have been very materially improved (1) by the use of penicillin and sulphonamides before, during and after operation, and (2) by the technique of aspiration and excision of the abscess with its capsule intact. Medical treatment pending surgical removal or drainage is as for purulent meningitis.

§ 738. **Pyogenic Sinus Thrombosis** causes (1) severe headache, vomiting and high fever of a pyogenic type, accompanied by rigors and sweats (see chart in § 515), (2) optic neuritis supervening in a day or two, and often photophobia, (3) drowsiness, deepening into coma and, if operative measures are not prompt, ending in death.

In *lateral sinus thrombosis* there are pain and tenderness in the mastoid region, together with other signs of suppurative otitis media; the inflammation spreads down the jugular vein on the same side, and backwards behind the mastoid; con-

sequently there is generally some hard brawny swelling in these positions. If there has previously been a discharge from the ear it usually ceases. When the *longitudinal sinus* is thrombosed the localising signs consist of œdema of the scalp, distension of the veins over the forehead and sometimes strabismus, associated with convulsions at the onset. There may be a spastic paraplegia or bilateral hemiplegia. With pyogenic thrombosis, the cause is usually some septic lesion of the face or scalp. When the *cavernous sinus* is affected the localising signs are œdema of the eyelids and root of the nose, sometimes also of the pharynx, exophthalmos and paralysis of the third, fourth, ophthalmic division of the fifth, and sixth nerves. There may be blindness from infarction of the retina, with retinal hæmorrhages and thromboses. Pyogenic thrombosis of this sinus may arise from some septic lesion of the orbit, nose, pharynx or face.

The *Diagnosis* of pyogenic sinus thrombosis is difficult unless the local signs are pronounced. In uncomplicated lateral sinus thrombosis there may be papillœdema, and this is not necessarily a sign of co-existing cerebral abscess. It may clear up when the sinus has been opened and drained and the jugular vein ligatured. The *Treatment* of lateral sinus thrombosis is a matter for an expert aural surgeon. In cavernous sinus thrombosis mortality is lowest when conservative treatment is employed. In septic cases, penicillin and sulphonamides are of great value.

§ **739. Cortical Thrombo-phlebitis.** Although pathological confirmation is scanty, a spreading non-suppurative thrombo-phlebitis of the cortical veins is believed to exist. It is usually met in association with suppurative otitis, when it may cause foci of superficial encephalitis. The physical signs and spinal fluid findings are indistinguishable from those of abscess formation. In most cases, however, the condition resolves and the patient recovers.

Some cases of hemiplegia and other cerebral symptoms in women during the puerperium or after abortion are attributed to venous occlusions in the superior longitudinal sinus and cortical veins. Pelvic and femoral thromboses are often present. The hemiplegia comes on with convulsions or coma. In the sinus and cortical venous thromboses of puerperal women, the C.S.F. is under increased pressure but is normal in composition.

Otitic Hydrocephalus is met with in children and gives rise to papilloedema with signs of increasing intracranial pressure and a pleocytosis in the C.S.F. There are no signs of any focal brain lesion. It occurs in association with chronic otitis and is believed to be due to partial thrombosis of the superior longitudinal sinus with defective re-absorption of cerebro-spinal fluid.

F. *Following upon* Vaccination *or an* acute specific fever *a patient develops* Pyrexia and Nervous Symptoms. The disease is Acute Encephalitis (Synonym: Acute Encephalomyelitis).

§ **740. Post-Vaccinal Encephalomyelitis.**—Six to fourteen days after vaccination with glycerinated calf-lymph the patient becomes acutely ill with fever, coma and delirium (§ 480). Meningeal signs may be present, with convulsions, trismus, papillœdema and various squints and pupillary abnormalities. The disease may end fatally, or the patient recovers with or without mental or physical sequelæ. A *spinal type* with sudden paraplegia and retention of urine also occurs but more rarely. Post-vaccinal encephalomyelitis has to be distinguished from severe constitutional reactions to vaccination seen in normal individuals and in the debilitated and mentally unstable. Pathologically, the brain and cord show in the white matter *perivascular zones of*

demyelination, with relatively little cellular infiltration. The grey matter suffers even more, and shows numerous punctate hæmorrhages. The disease has a definite incubation period after vaccination against small-pox, and has been produced experimentally in rabbits. It is probably due to the virus of vaccinia. Two members of a family, vaccinated on the same day, may develop the disease simultaneously.

§ 741. **Encephalomyelitis following Acute Specific Fevers.**—During the course of various acute specific fevers there may be a recrudescence of the fever with development of clinical signs, similar to those of Post-Vaccinial Encephalomyelitis. Cases are described in association with Measles, Chicken-pox, Small-pox, Mumps and Whooping-Cough.

Symptoms.—The onset is sudden during the first or second weeks of the illness. There is an exacerbation of fever and headache; delirium, diplopia and head retraction occur. Cranial nerve palsies, hemiplegia, paraplegia, with incontinence, or involuntary movements, may be observed. Papillœdema is common. After some days, during which the symptoms advance, recovery sets in and may be almost complete. In the *spinal form*, paraplegia, with retention of urine and incontinence of fæces develops. Sensory symptoms, ascending paralysis or root palsy may occur. If death does not take place within the first week, recovery ensues. The *spinal fluid* shows a lymphocytic reaction, with normal chloride and glucose content. The residual symptoms of the encephalomyelitis may be hemiplegia, spastic weakness of the legs, with or without sensory loss, involuntary movements or ataxia, fits, deafness or optic atrophy, and various cranial nerve palsies and pupillary abnormalities.

Treatment.—The appropriate convalescent serum may be given intrathecally, after withdrawing an equal amount of cerebro-spinal fluid.

§ 742. **Acute Disseminated Encephalomyelitis** is described (Brain and Hunter) as a clinical entity, probably distinct from disseminated sclerosis. These cases may be due to polioencephalitis. In all cases the spinal fluid shows excess of lymphocytes, but the characteristic clinical changes of acute meningitis are not found.

G. *A patient develops mild* PYREXIA *with* DOUBLE VISION, OCULAR PALSIES *and lethargy.* The disease is ENCEPHALITIS LETHARGICA.

ENCEPHALITIS LETHARGICA is fully described in § 698. The acute phases may be characterised by a febrile illness of some days' duration, with headache, pains in the limbs, malaise and constipation. Later supervene the striking drowsiness by day, with muttering occupational delirium by night and diplopia, ptosis, squint, pupillary abnormalities and nystagmus.

Any of the foregoing illnesses may be attended by **Febrile Delirium** (see § 469.)

GROUP IV. DEFECTS OF SPEECH AND ARTICULATION

I. *The patient has difficulty in exteriorising his ideas in speech or writing, or you are unable to communicate with the patient because he cannot recognise words spoken to or written for him.* The condition is APHASIA.

§ 743. **Aphasia** is the term applied to severe speech defects of cortical origin. *Dysphasia* is a milder degree of aphasia. Aphasia is of two kinds —motor and sensory. In *Motor Aphasia* there is loss of power to exteriorise thought in spoken or written words—the patient knows what he wishes to say but cannot say it, although the peripheral neuromuscular mechanism of articulation is intact. In *Sensory Aphasia* there is defect of comprehension of spoken speech (word-deafness) or written speech (word-blindness) in the absence of deafness or blindness; the words are heard or

seen by the patient but convey imperfect ideas or nothing at all, to him.

The symbols of speech are vocal words or written words. The movements which govern the production of vocal or written speech are under the controlling influence of sensory impressions. When we speak we use our auditory memories of words to control the correctness of our speech movements, and when we write we use our visual memories to control the correctness of our writing movements. Hence, it is that aphasias are rarely purely motor or purely sensory, but more often a combined defect.

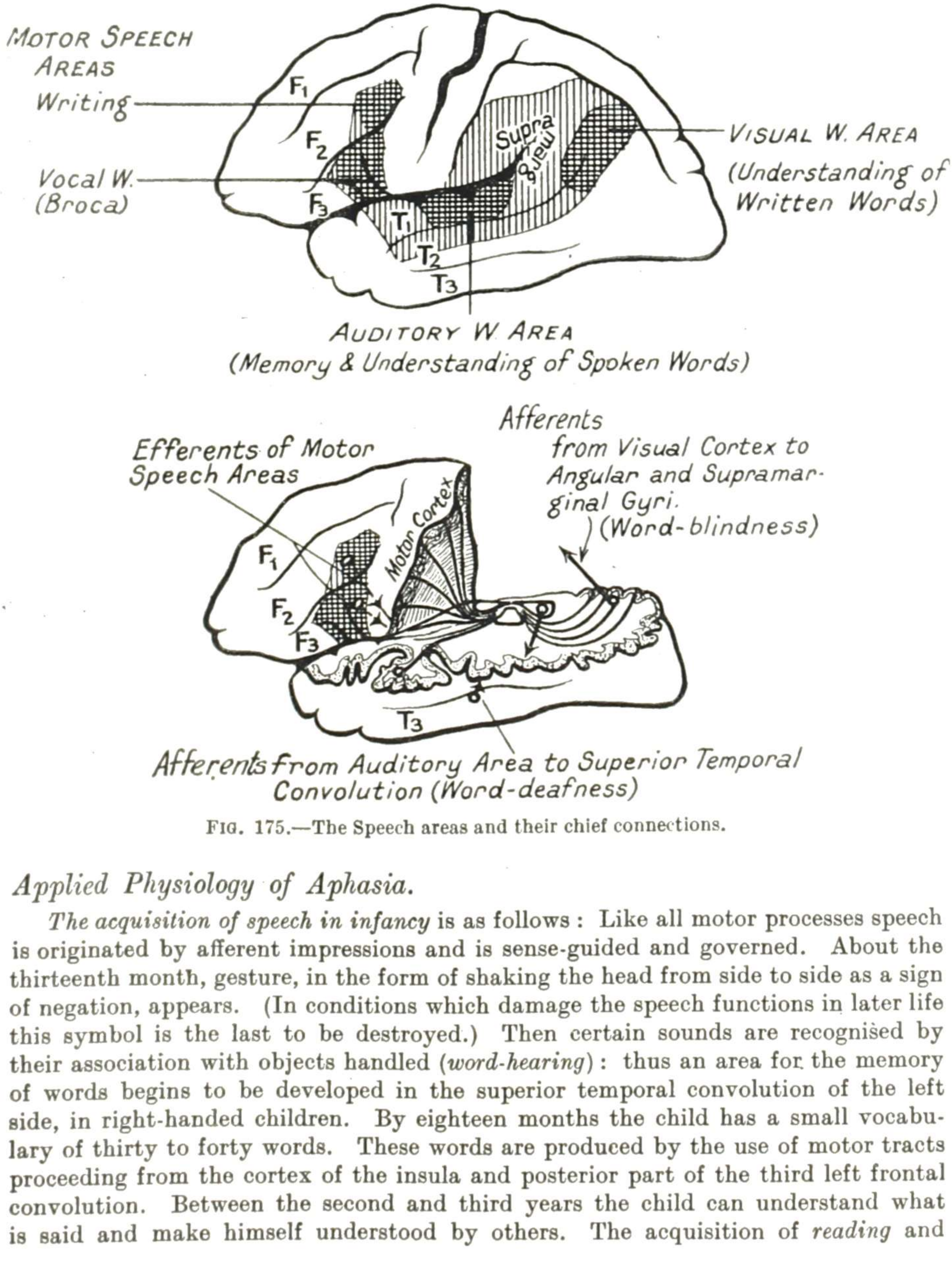

FIG. 175.—The Speech areas and their chief connections.

Applied Physiology of Aphasia.

The acquisition of speech in infancy is as follows: Like all motor processes speech is originated by afferent impressions and is sense-guided and governed. About the thirteenth month, gesture, in the form of shaking the head from side to side as a sign of negation, appears. (In conditions which damage the speech functions in later life this symbol is the last to be destroyed.) Then certain sounds are recognised by their association with objects handled (*word-hearing*): thus an area for the memory of words begins to be developed in the superior temporal convolution of the left side, in right-handed children. By eighteen months the child has a small vocabulary of thirty to forty words. These words are produced by the use of motor tracts proceeding from the cortex of the insula and posterior part of the third left frontal convolution. Between the second and third years the child can understand what is said and make himself understood by others. The acquisition of *reading* and