

NINTH
EDITION

**CORRELATIVE
NEUROANATOMY
and
FUNCTIONAL
NEUROLOGY**

JOSEPH G. CHUSID
JOSEPH J. McDONALD

CORRELATIVE NEUROANATOMY and FUNCTIONAL NEUROLOGY

by

JOSEPH G. CHUSID, M.D.

Attending Neurologist, St. Vincent's Hospital, New York

JOSEPH J. McDONALD, M. S., M. Sc. D., M. D.

Dean of Medical Faculty,
American University of Beirut,
Beirut, Lebanon
Formerly Professor of Surgery,
Columbia University, New York

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Preface

This volume is intended for the beginner in neurology and will serve him best if used as an aid or supplement to standard neurological texts and literature. In recent years it has become popular with practitioners, residents, and graduate physicians preparing for specialty board examinations as a handbook and review of neurology. Our primary objective has been to present simply and clearly some of the structural and functional features of the nervous system related to problems encountered in clinical neurology. Concise format, charts, diagrams, and photographs have been prepared with this purpose in mind.

The authors are particularly indebted to Mr. James Ransom for editorial supervision and other invaluable assistance, and to Dr. Jack D. Lange, publisher and former co-author, for expert and generous guidance in the preparation of this revision. Drs. Paul Sanazaro, Judith Nadell, Otto E. Guttentag, and Professor Harold A. Harper supplied helpful, critical reviews of various portions of the manuscript.

For the Ninth Edition Professor Ralph Sweet has provided more of his highly effective drawings and illustrations. Mr. Rudolph J. Henning assisted greatly with his skill and artistry as a medical photographer.

We are pleased to acknowledge our gratitude and indebtedness to Dr. C. G. de Gutiérrez-Mahoney, Director of the Neurological Division of St. Vincent's Hospital, New York, for the full use of his library and the facilities of the Division, and for his continued kind interest in this book. We extend our sincere thanks to those authors, publishers, and editors who graciously permitted the reproduction of various items.

Joseph G. Chusid
Joseph J. McDonald

New York, April, 1958

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Section I: Central Nervous System

1...

Embryology

Early differentiation. - A thickened plate of ectoderm, the **neural plate**, develops along the middorsal line of the embryo and is transformed by invagination into a neural tube. The **neural tube** detaches from the overlying ectoderm and thickens to develop into the spinal cord and brain. The rostral end of the neural tube, which ultimately forms the brain, differentiates into three primary brain vesicles: (1) the **prosencephalon** or forebrain, which lies closest to the rostrum; (2) the **mesencephalon** or midbrain, which lies behind the prosencephalon; and (3) the **rhombencephalon** or hindbrain, which lies most caudad.

Development of the brain. - From the prosencephalon are formed the telencephalon and diencephalon. The telencephalon forms the cerebral cortex, the striate bodies, the rhinencephalon, the lateral ventricles, and the anterior portion of the third ventricle. The diencephalon gives rise to the epithalamus, thalamus, metathalamus, hypothalamus, optic chiasm, tuber cinereum, posterior lobe of the hypophysis, mammillary bodies, and most of the third ventricle.

From the mesencephalon develop the quadrigeminal plate, the cerebral peduncles, and the aqueduct of Sylvius.

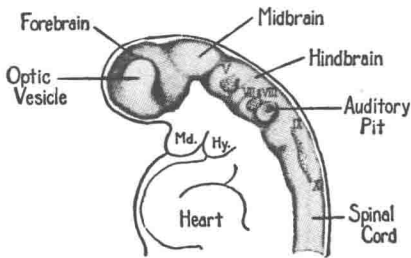
The rhombencephalon gives rise to the metencephalon and the myelencephalon. The metencephalon forms the cerebellum, pons, and part of the fourth ventricle. The myelencephalon forms the medulla oblongata and part of the fourth ventricle.

Development of the spinal cord. - The spinal cord develops from the caudal portion of the neural tube. The earliest tracts of nerve fibers appear in the marginal zone at about the second month. Long association tracts appear about the third month and pyramidal tracts about the fifth month of fetal life. Myelination of nerve fibers of the spinal cord begins about the middle of fetal life and is not completed in some tracts for 20 years. The oldest tracts myelinate first; pyramidal tracts late, largely during the first and second postnatal years.

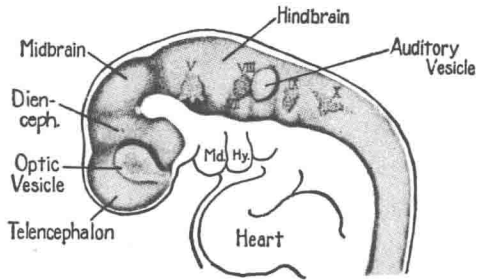
Cellular developmental changes. - Initially, the neural plate consists of a single layer of cells. These divide and proliferate and their cell bodies become indistinct, so that by the time of the formation of the neural tube the wall is formed of several layers of cells with a syncytial appearance. Three layers may be differentiated early: (1) a marginal or nonnuclear outer layer, which in the spinal cord develops into the white substance; (2) a mantle layer with many nuclei, which in the spinal cord differentiates into the gray matter; and (3) an innermost endodermal layer in which may be found large mitotic nuclei of germinal cells. Neuroblasts form, which differentiate into neurons; and spongioblasts, which differentiate into neuroglial and endodermal cells.

The neural crest, a ridge of ectodermal cells at the junction of the neural groove and the overlying superficial ectoderm, gives rise to the neuroblasts which form the sensory (afferent) fibers and the sensory ganglia. Some ectodermal cells migrate from the neural tube and neural crest along the course of the ventral or dorsal roots. From these is derived the neurilemma or nucleated sheath of the peripheral nerve fiber. Ectodermal cells of similar origin give rise to sympathetic ganglia. The chromaffin tissue (carotid bodies, aortic bodies, adrenal medulla, etc.) and all of the nerve cells outside of the central nervous system, with the exception of those arising from the neural placodes, come from the neural tube and neural crests. The placodes (ectodermal thickenings) give rise to olfactory neuroepithelium, epithelium of the otocyst, and the lens of the eye, and contribute to formation of the trigeminal, facial, glossopharyngeal, and vagus nerves.

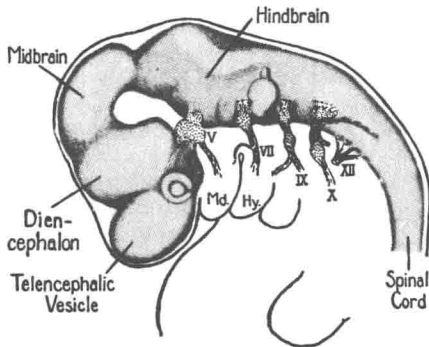
2 Development of the Nervous System



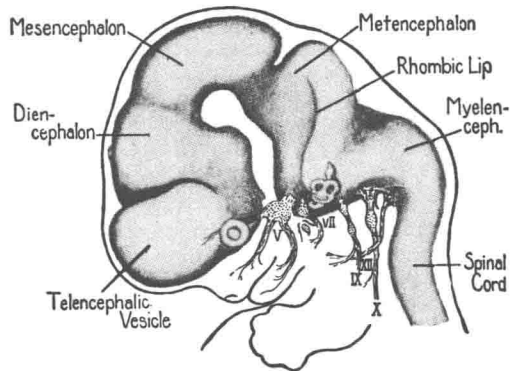
At 20 somites - based on the Davis embryo - probable F.A. of 3 1/2 weeks.



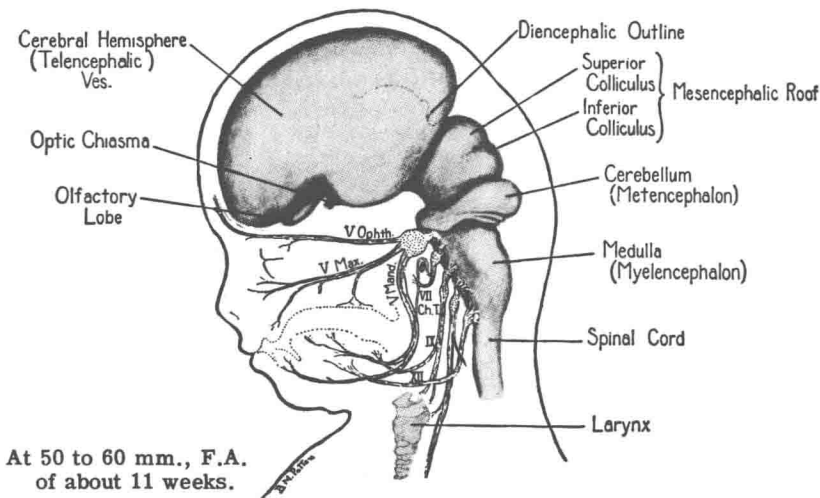
At 4 mm., F.A. of about 4 weeks.



At 8 mm., F.A. of about 5 1/3 weeks.



At 17 mm., F.A. of about 7 weeks.



At 50 to 60 mm., F.A. of about 11 weeks.

Five Stages in Early Development of Brain and Cranial Nerves. (Adapted from various sources, primarily figures by Streeter and reconstructions in the Carnegie Collection.) The cranial nerves shown are indicated by the appropriate roman numerals: V, trigeminal; VII, facial; VIII, acoustic; IX, glossopharyngeal; X, vagus; XI, accessory; XII, hypoglossal. Abbreviations: F.A. = fertilization age; Ch. T. = chorda tympani branch of seventh nerve; Hy. = hyoid arch; Md. = mandibular arch; V Mand. = mandibular branch of trigeminal nerve; V Max. = maxillary branch; V Ophth. = ophthalmic branch. (Reproduced, with permission, from Patten, Human Embryology, 2nd Edition. The Blakiston Co., Inc., New York, 1953.)

2...

Neurochemistry

There is a high percentage of water in neural tissue. The adult brain is about 78% water; the spinal cord, about 75%. Gray matter has a higher water content than white matter.

The solids of neural tissue are made up for the most part of proteins and lipids, with smaller fractions of inorganic salts and organic extractives. **Proteins** constitute up to 40% of the total solids. Most brain protein is linked with lipids in the form of lipoproteins, compounds which resemble living protoplasm much more closely than either free proteins or free lipids. Water-soluble liponucleoproteins (nucleoproteins combined with lipids) are present in brain. The trypsin-resistant and pepsin-resistant protein fraction is known as neurokeratin. Globulin and albumin are present. A large fraction of brain protein is insoluble in water or saline solution but (unlike neurokeratin) is digestible by proteolytic enzyme.

Lipids make up a large part of the solid content of neural tissue (variously estimated at 40 to 75%). Very little simple lipid is found. Compound lipids are abundant and include phospholipids (lecithins, cephalins, and sphingomyelin), cholesterol, cerebrosides or galactolipids (glycolipids), sulfur-containing lipids, and amino lipids. Lipids present in brain are synthesized there rather than transported to the brain from other sources. Neural tissue lipids may be unique in that certain of their important component fatty acids (e.g., 24-carbon fatty acids) have not been demonstrated elsewhere in the body. Lipids are metabolized faster during early development of brain than later; in adult brain there is a slow turnover of fatty acids which penetrate into the brain very slowly if at all. The rate of exchange of brain lipids is slow compared with that of liver lipids.

Inorganic salts are found in the combustion products (1% ash) of neural tissue. The principal inorganic salts found are potassium phosphate and chloride. Sodium and other alkaline elements are found in lesser amounts. There are high potassium and magnesium concentrations intracellularly but little or no sodium or chloride, which are found extracellularly. The Nissl bodies of cytoplasm are considered to be centers of protein production. Portions of the endoplasmic reticulum supply various enzymes and substrates, and the fine granular component of the cytoplasm supplies some of the requirements for protein synthesis. Oxidative and synthetic activities of the mitochondria and glycolytic activities of the fluid matrix may be coordinated by structural alterations within the cytoplasm (mitochondrial movements, cytoplasmic streaming, and sol-gel changes in the matrix). Microsomes (particulates obtained from nerve cytoplasm by differential centrifugation) are rich in phospholipids and contain most of the ribonucleic acid of the cytoplasm.

The nuclei of nerve cells are rich in **nucleic acids**. Two general types of nucleotides are found in nucleic acids: ribonucleic acid (RNA) and desoxyribonucleic acid (DNA). The tissue of the central nervous system contains about twice as much RNA as DNA. The DNA is confined to the nuclei of nerve and glial cells, with a considerable part of the nuclear DNA in the chromosomes. RNA is found in both the nucleus (mainly in the nucleolus) and in the cytoplasm. RNA may be identified histologically by the orcinol green reactions for pentoses; DNA by the color reaction of Feulgen. Quantitative spectrophotometry can be performed on various cellular constituents with the use of the quartz microscope, which is capable of transmitting ultraviolet light. Nucleic acids absorb ultraviolet light strongly at a wavelength of 2600 Å.

Histologic studies of neural tissue permit the identification of various pigments and substances. **Melanin** is the deep black pigment found in the nerve cells of the substantia nigra, of the locus caeruleus, in some of the cerebrospinal and sympathetic ganglion cells, and in the chromatophore cells of the leptomeninges. Melanin is usually not present in the newborn but appears toward the end of the first year, increases in amount until puberty, and remains more or less constant thereafter. Depigmentation of the substantia nigra is a frequent finding in postencephalic parkinsonism. **Lipochrome** or **lipofuscin**, a yellow pigment, appears in spinal ganglia neurons about the sixth year; a few years later it appears in the spinal cord, and after the twentieth year is found in

4 Brain Metabolism

cerebral cortical neurons. It increases with advancing age and is quite marked in old age. It appears as droplets around the nerve cell nucleus, stains deeply with osmic acid and Sudan III, and is insoluble in the usual fat solvents.

Hemoglobin derivatives are sometimes found in the central nervous system. Yellow-brown granules of **hemosiderin**, an iron-containing pigment, appear following extravasations of blood and in hemochromatosis. **Hemofuscin**, a light yellow granular substance containing no iron, is found in excessive quantities in hemochromatosis. **Hematoidin**, a decomposition product of heme, forms biliverdin, a green pigment which imparts a light green color to white matter surrounding a hemorrhagic site.

Calcification occurs normally within the pineal body during adult life. Small granules or large masses of calcium phosphate and carbonate occur pathologically in the central nervous system. Calcification of the cerebral cortex occurs in the Sturge-Weber syndrome; within the vascular tree, meninges, and the choroid plexuses as a degenerative process; and in some brain tumors such as meningiomas, oligodendrogliomas, and craniopharyngiomas. **Iron compounds** are normally present in the globus pallidus and substantia nigra. Some of the brain iron has been identified as ferritin, a crystallizable protein containing 23% iron. The tissue iron of brain probably is a product of iron metabolism, although it can also arise from extravasated red blood cells.

BRAIN METABOLISM

The metabolism of the embryonic brain is characterized by a great capacity to synthesize the proteins and lipids needed for growth. Oxidative mechanisms are deficient, but the brain is highly capable of utilizing carbohydrates by glycolysis. During fetal life glucose oxidation systems become more active, extending progressively from the lower to the higher centers and continuing after birth. As development proceeds successive changes in the activity of individual enzymes take place, with new enzymes appearing, increasing in activity, and then declining.

In the adult brain the ability to synthesize certain proteins and lipids is greatly reduced, but the dependence upon carbohydrate as its main fuel persists. The brain is characterized by a high over-all oxygen consumption, with metabolic activity generally highest in the cortex and cerebellum. The high energy requirement of most portions of the brain is related to the transport of ions, the synthesis of acetylcholine, and the metabolism of glutamic acid. Concomitant changes affecting phospholipids and nucleoproteins occur, but the metabolic processes associated with functional activity of the brain are poorly understood.

The metabolic activity of the brain varies somewhat with the state of functional activity. When there is a general increase in neuronal activity, as in convulsive states and in states of diffuse neuromuscular activity, metabolic activity is increased. However, there is no significant change in over-all metabolic activity in highly localized types of cerebral function. The decrease in oxygen consumption of brain and of activity of enzymes associated with glucose utilization in the older age groups may be due to progressive decrease in the ratio of neurons to glial cells.

Serotonin (5-hydroxytryptamine) may be one of the important regulatory amines of the body, similar in this respect to histamine, epinephrine, and norepinephrine. It is present in high concentration in the hypothalamus, midbrain, and caudate nucleus. It is probably synthesized from the amino acid, tryptophan, although by a different metabolic pathway from that which leads to nicotinic acid, and is disposed of through deamination to 5-hydroxyindoleacetic acid. It has vasoconstrictor and pressor effects and may also be found in the mammalian gastrointestinal tract and blood platelets. The action of some tranquilizing drugs, such as reserpine, may be by the release of bound serotonin in the brain. A structural analog of serotonin is *d*-lysergic acid diethylamide (LSD), which in small doses is capable of evoking mental symptoms similar to those of schizophrenia. The vasoconstrictive action of LSD is inhibited by serotonin.

Among the studies made in an attempt to correlate functional brain status and enzymatic findings are those of Flexner on the guinea pig. In early fetal life, the cerebral cortex of the guinea pig has been found to contain low, constant concentrations of respiratory enzymes, cytochrome C, succinic dehydrogenase, and adenylypyrophosphatase (apyrase). The concentrations of these enzymes increase sharply at the time of morphologic differentiation and the onset of

electrical activity in nerve cells. The adult level is reached or approximated at birth. A similar close relationship in other vertebrate species has been noted between functional development and brain enzyme concentrations of cholinesterase and carbonic anhydrase.

Carbohydrate metabolism of nerve tissue is similar to that of muscle. Lactic acid and pyruvic acid appear under anaerobic conditions; they disappear very slowly, and oxygen does not accelerate this process. Very little storage of glycogen occurs in neural tissue, and brain extracts react more readily with glucose than with glycogen. The respiratory quotient of neural tissue is 1.0 which suggests that ordinarily the tissues of the central nervous system utilize carbohydrate almost exclusively, burning sugar with oxygen and introducing energy into cells via high-energy phosphate esters. In some circumstances, however, the brain can apparently remain active without the use of extrinsic or intrinsic carbohydrates.

Acetylcholine and cholinesterase activity have been demonstrated in every cortical layer of the normal brain and are roughly proportional to neuron density and size. Epileptic cortical foci are reported to have elevated cholinesterase activity, and changes in the bound acetylcholine of the brain may be demonstrated before and during experimentally induced seizures in animals. These findings have been interpreted to indicate that alteration in acetylcholine metabolism occurs in conjunction with epileptic brain abnormalities. However, other neurochemical mechanisms unrelated to acetylcholine metabolism may also be important in the production of convulsive seizures. Increased production of ammonia may immediately precede the onset of experimental seizures, and abnormalities of potassium distribution have been found in convulsed brain segments. Toxic epileptogenic agents (e.g., fluoroacetate) block the citric acid (Krebs) cycle while producing convulsions. Inhibition of glutamine synthesis in the brain occurs after treatment with methionine sulfoxime, the toxic convulsant agent of nitrogen trichloride (agene). A deficiency of pyridoxine (vitamin B₆) causes seizures in infants and animals, and certain convulsant drugs such as the carbazide series act by inducing pyridoxine deficiency. Glutamine and asparagine can reverse the defective glutamic acid metabolism of certain types of epileptogenic cortex, thus inhibiting seizures. Chronic experimental epilepsy is readily induced in monkeys by treatment of cerebral cortex with aluminum hydroxide; other metals may also be effective.

Increased knowledge of the metabolic characteristics of mammalian embryonal tissues has resulted from the studies of Hicks and others on experimental induction of brain malformations of small laboratory animals. Developing cells change their response to metabolic injury as they grow, and the organism changes metabolically as it develops. Although developmental patterns are primarily genetically determined and latent genetic abnormalities may be precipitated by injurious agents, different agents may produce different types of malformation at the same stage of development. In general the rat embryo is resistant to anoxia and hypoglycemia, but interference with nucleic acid metabolism of primitive differentiating cells causes their destruction. In late fetal and neonatal life resistance to anoxia persists, but the interruption of some phases of glucose metabolism has serious consequences.

The **blood-brain barrier** may influence brain function by determining the level of metabolism and the ionic composition of tissue fluids. Certain types of abnormal brain function could conceivably result from an abnormal blood-brain barrier. The function of the blood-brain barrier may be influenced by the metabolism of brain cells as well as by the composition of the circulating blood, and it may hinder the free passage of many metabolites into the brain, thus protecting the brain from variations of blood composition and from the entry of toxic compounds. As the brain matures, changes occur in the relative ease with which substances can enter it. Thus the brain of the premature human infant is quite permeable to bilirubin; kernicterus develops readily in these infants, but not at all in adults with greatly increased blood bilirubin levels. Trypan blue, an azo dye, and ferricyanide both penetrate freely into the brain of very young but not of mature laboratory animals following intravenous injection. Radioactive phosphorus (P³²) enters more readily and in greater amounts into the brains of newborn and very young animals.

The rate of uptake of dyes, anions, and cations from the circulating blood by the intact adult central nervous system is slow compared with the uptake by other organs. This applies for inorganic substances (potassium, sodium, etc.) as well as organic substances (e.g., glutamic acid). There is a relatively rapid gas exchange and uptake of lipid-soluble compounds and of glucose. Glutamic acid and its amide, glutamine, are present in the brain in large amounts, comprising almost half of the nonprotein nitrogen. The blood-brain barrier appears to prevent glutamic acid from penetrating into the intact brain, but glutamine enters readily. The source of the large amounts of glutamic acid and glutamine in the central nervous system is not known.

NERVE FUNCTION

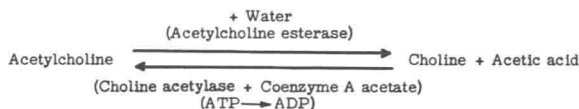
Electrolytes may influence nerve function. High external potassium and low external calcium concentrations decrease the resting potential of peripheral nerve, presumably increasing its excitability. Nerves lose calcium in low-calcium solutions and gain potassium when exposed to high concentrations of external potassium. Excessive magnesium has a general depressant effect on nervous system function.

Neural activity, such as occurs in nerve conduction, may lead to altered inorganic ion concentrations. A loss of potassium due to activity has been demonstrated in ganglia and unmyelinated nerve. According to the membrane theory of nerve action, the surface of a nerve is permeable to potassium but relatively impermeable to sodium; upon excitation there is an alteration in permeability of the membrane, permitting sodium and perhaps other ions to enter. When a nerve conducts an impulse following stimulation, a small amount of heat is produced. The rapidly released initial heat may represent energy associated with transmission of the impulse; the recovery or delayed heat (up to 45 minutes) may be related to the mechanisms of energy restoration. Under anaerobic conditions (nitrogen atmosphere), a nerve may conduct impulses and develop heat; recovery, however, depends upon oxygen utilization.

Chemical mediators may be elaborated at the myoneuronal junction in association with the action of a nerve impulse. **Acetylcholine** is produced in parasympathetic and voluntary nerves to skeletal muscles. **Sympathin** results from sympathetic nerve stimulation, and its effects are opposite to those of acetylcholine. Sympathin may have an excitatory (Sympathin E) or an inhibitory effect (Sympathin I). Sympathin resembles epinephrine in its activity; sympathetic nerve stimulation may cause release of epinephrine from the adrenal medulla.

The adrenal medulla is a derivative of the sympathetic portion of the autonomic nervous system; in general, the hormone of the adrenal medulla (epinephrine, adrenalin) duplicates the effect of sympathetic stimulation of an organ. About 80% of the hormonal activity of the adrenal medulla is due to epinephrine and the remainder to norepinephrine (arterenol), a closely related hormone, which is a precursor of epinephrine. Epinephrine produces vasodilatation of the blood vessels of the skeletal muscle and vasoconstriction of the arterioles of the skin, mucosa, and splanchnic viscera; norepinephrine exerts an over-all vasoconstrictor effect. Both epinephrine and norepinephrine produce elevation of blood pressure, which is more marked in the case of norepinephrine.

Acetylcholine esterase, found within nerve fibers and at nerve endings, readily hydrolyzes acetylcholine to choline and acetic acid. The inactivating hydrolyzing effect of acetylcholine esterase is believed to control the action of acetylcholine in the body. This substance must be distinguished from pseudocholinesterase, found in blood serum, which hydrolyzes other esters. For resynthesis of acetylcholine, energy is required. Active acetate (coenzyme A acetate) serves as acetate donor for acetylation of choline. Choline acetylase, activated by potassium and magnesium ions, catalyzes the transfer of acetyl from coenzyme A acetate to choline. Regeneration of adenosine triphosphate (ATP) from adenosine diphosphate (ADP) is accomplished by phosphocreatine, which is resynthesized from creatine and free phosphate with the aid of energy produced in glycolysis.



Physostigmine (eserine) inhibits acetylcholine esterase, thus prolonging parasympathetic activity. Neostigmine (Prostigmin®), an alkaloid, is believed to act similarly. Diisopropyl-fluorophosphate (DFP) is a synthetic substance which irreversibly inhibits acetylcholine esterase. This compound is believed to be one of the most powerful and specific enzyme inhibitors known. The toxic properties of some "nerve gases" and some insecticides (such as Parathion®) depend upon their action as anticholinesterases. A highly effective antidote for certain nerve gases and insecticides is pyridine-2-aldoxime methiodide (PAM), which is especially effective with atropine.

3...

The Brain

The brain is the greatly modified and enlarged anterior portion of the central nervous system. It is surrounded by three protective membranes (meninges) and enclosed within the cranial cavity of the skull. Division into cerebral cortex, basal ganglia, thalamus and hypothalamus, midbrain, brain stem, and cerebellum provides a useful basis for the study of brain localization.

THE CEREBRAL HEMISPHERES

The two cerebral hemispheres, which make up the largest portion of the brain, are separated by the deep **longitudinal cerebral fissure**. The **falx cerebri**, a crescent-shaped extension of dura mater, projects into the longitudinal cerebral fissure. The **corpus callosum** is the great white central commissure which crosses the longitudinal cerebral fissure. The body of the corpus callosum is arched; its anterior curved portion, the **genu**, continues anteroventrally as the **rostrum**. The thick posterior portion terminates in the curved **splenium**, which overlaps the mid-brain.

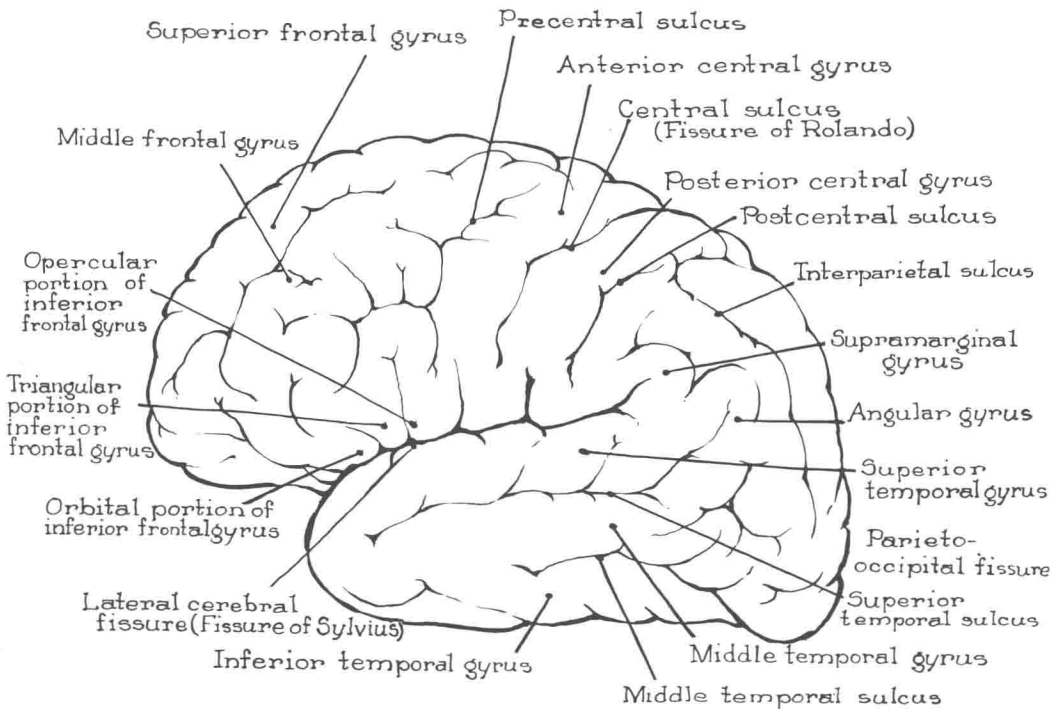
The surfaces of the cerebral hemispheres are dorsolateral, medial, and basal. They contain many grooves or furrows, known as fissures and sulci. The portions of brain lying between these grooves are called convolutions or gyri. Some gyri are relatively constant in location and contour, whereas others show considerable variation. The **lateral cerebral fissure** (fissure of Sylvius) separates the temporal from the frontal lobe. Starting at the base of the brain as a deep cleft lateral to the anterior perforated substance, it divides into three branches: the anterior horizontal ramus, which ascends into the inferior frontal gyrus; the anterior ascending ramus, which also ascends into the inferior frontal gyrus farther posteriorly; and the posterior ramus, which continues backward and upward to terminate in the parietal lobe.

The **central sulcus** (fissure of Rolando) arises about the middle of the hemisphere, beginning near the longitudinal cerebral fissure and extending downward and forward to about an inch above the lateral cerebral fissure. The **parieto-occipital fissure** passes along the medial surface of the posterior portion of the cerebral hemisphere, runs downward and forward as a deep cleft with much buried cortex, and joins the calcarine fissure. The **calcarine fissure** begins on the medial surface, near the occipital pole, and extends forward to an area slightly below the splenium of the corpus callosum. The rostral portion is deeper and more constant in location and structure. The **cingulate sulcus** begins below the anterior end of the corpus callosum on the medial surface of the hemisphere, continues parallel to the corpus callosum, and finally curves up to the superior medial border a short distance behind the upper end of the central sulcus. The **circular sulcus** (circuminsular fissure) surrounds the insula, or island of Reil, and separates it from the adjacent frontal, parietal, and temporal lobes.

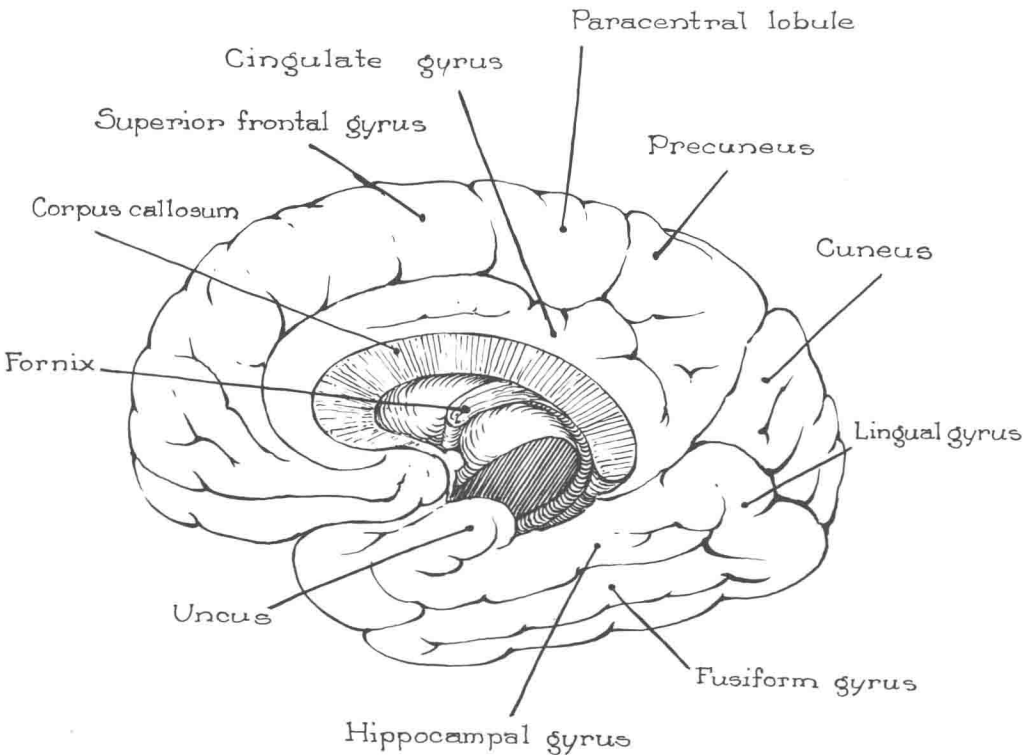
Main Divisions of the Cerebrum.

The cerebral hemisphere may be divided into the frontal, parietal, occipital, and temporal lobes, the insula, and the rhinencephalon.

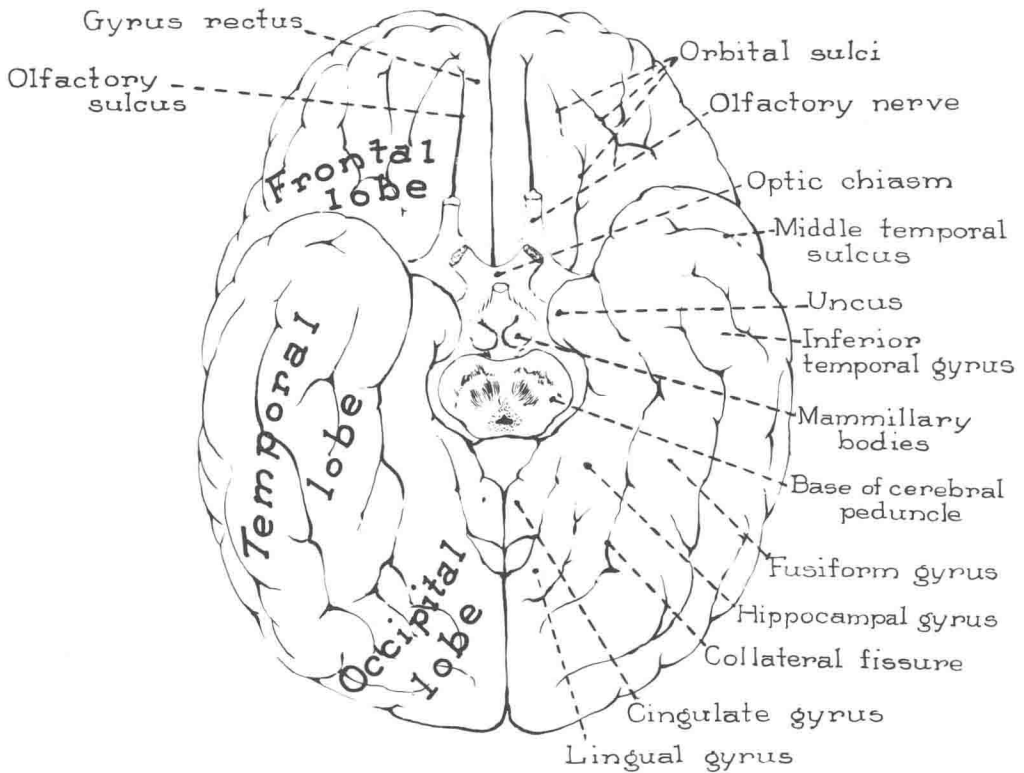
Frontal lobe. - The frontal lobe extends from the frontal pole to the central sulcus behind and the lateral fissure at the side. The **precentral sulcus** passes anterior and parallel to the central



Lateral View of Left Cerebral Hemisphere



Medial View of Right Cerebral Hemisphere



Basal View of Cerebrum

sulcus. It is subdivided into the superior and inferior precentral sulci. The **superior and inferior frontal sulci** extend forward and downward from the precentral sulcus, dividing the lateral surface of the frontal lobe into three parallel gyri: the **superior, middle, and inferior frontal gyri**. The inferior frontal gyrus is divided into three parts by the anterior horizontal and ascending rami of the lateral cerebral fissure: The orbital part lies rostral to the anterior horizontal ramus; the triangular part is the wedge-shaped portion between the anterior horizontal and anterior ascending rami; the opercular part is between the ascending ramus and the precentral sulcus.

The **orbital sulci and gyri** are irregular in contour and location. The **olfactory sulcus** lies beneath the olfactory tract on the orbital surface; lying medial to it is the gyrus rectus or **straight gyrus**. The **cingulate gyrus** is the crescentic or arched convolution on the medial surface between the cingulate sulcus and the corpus callosum. The **paracentral lobule** is the quadrilateral gyrus around the end of the central sulcus on the medial surface of the hemisphere.

Parietal lobe. - The parietal lobe extends from the central sulcus to the parieto-occipital fissure and laterally to the level of the lateral cerebral fissure. The **postcentral sulcus** extends behind and parallel to the lateral (Rolandic) fissure, and consists of a superior and inferior portion. The **intraparietal sulcus** is a horizontal groove which sometimes unites with the postcentral sulcus. The **superior parietal lobule** lies above the horizontal portion of the intraparietal sulcus, and the **inferior parietal lobule** lies below.

The **supramarginal gyrus** is that portion of the inferior parietal lobule which arches above the ascending end of the posterior ramus of the lateral cerebral fissure. The **angular gyrus** is that part which arches above the end of the superior temporal sulcus and becomes continuous with the middle temporal gyrus. The **posterior central gyrus** lies between the central and postcentral sulci. The **precuneus** is the posterior portion of the medial surface between the parieto-occipital fissure and the ascending end of the cingulate sulcus.

Occipital lobe. - The occipital lobe is the pyramid-shaped posterior lobe situated behind the parieto-occipital fissure. The **lateral occipital sulcus** extends transversely along the lateral surface, dividing the occipital lobe into a **superior** and **inferior gyrus**. The **calcarine fissure** divides the medial surface of the occipital lobe into the **cuneus** and the **lingual gyrus**. The wedge-shaped **cuneus** lies between the calcarine and parieto-occipital fissures. The **lingual gyrus** is between the calcarine fissure and the posterior part of the collateral fissure. The posterior part of the **fusiform gyrus** is on the central or basal surface of the occipital lobe.

Temporal lobe. - The temporal lobe portion of the cerebral hemisphere lies inferior to the lateral cerebral (sylvian) fissure and extends back to the level of the parieto-occipital fissure. The **superior temporal sulcus** extends across the temporal lobe parallel to the sylvian fissure. The **middle temporal sulcus** runs parallel to the superior temporal sulcus at a lower level. The **superior temporal gyrus** is the part of the lateral surface of the temporal lobe between the sylvian fissure and the superior temporal sulcus. The **middle temporal gyrus** lies between the superior and middle temporal sulci. The **inferior temporal gyrus** is below the middle temporal sulcus and extends posteriorly to connect with the inferior occipital gyrus. The **transverse temporal gyrus** (Heschl's gyrus) occupies the posterior part of the superior temporal surface (the inferior border of the lateral cerebral fissure). The **inferior temporal sulcus** extends along the inferior surface of the temporal lobe from the temporal pole in front to the occipital pole behind. The **fusiform gyrus** is medial and the **inferior temporal gyrus** lateral to the inferior temporal sulcus. The **hippocampal fissure** extends along the inferomedian aspect of the temporal lobe from the area of the splenium of the corpus callosum to the uncus. The **hippocampal gyrus** lies between the hippocampal fissure and the anterior part of the collateral fissure. Its anterior part curves in the form of a hook and is known as the uncus.

Insula. - The insula (island of Reil) lies deep within the fissure of Sylvius and can be exposed by separating the upper and lower lips of the fissure. The deep **circular sulcus** bounds the insula. Several **short gyri**, formed by shallow sulci, occupy the anterior portion of the insula; a **long gyrus** occupies the posterior part.

The **opercula** of the insula are portions of the lips of the lateral cerebral fissure. The orbital operculum is anterior and inferior to the anterior horizontal ramus. The frontal operculum lies between the orbital operculum and the anterior ascending ramus. The parietal operculum lies between the frontal operculum and the end of the posterior ramus. The temporal operculum lies below the posterior ramus.

Rhinencephalon. - The rhinencephalon, a phylogenetically old portion of the cerebral hemisphere, includes the portions associated with the perception of olfactory sensation. The **olfactory bulb**, an oval structure, lies on the cribriform plate of the ethmoid bone and receives the olfactory nerves which have passed upward through the cribriform plate from the olfactory zone of the nasal cavity. The **olfactory tract** lies in the olfactory sulcus on the orbital surface of the frontal lobe. As it passes posteriorly, it divides into the **lateral olfactory stria**, which passes laterally, then medially, to enter the uncus; and the **medial olfactory stria**, which passes medially and up toward the subcallosal gyrus near the inferior aspect of the corpus callosum. The **olfactory trigone** is the small triangular attachment between the medial and lateral olfactory striae, just anterior to the anterior perforated substance. The **anterior perforated substance**, a depressed area of gray matter, extends from the olfactory striae to the optic tract. The **pyriform area** includes the anterior portion of the hippocampal gyrus, the uncus, and the lateral olfactory gyrus. The **subcallosal gyrus** is the portion of gray matter which covers the under aspect of the rostrum of the corpus callosum and is continuous about the genu of the corpus callosum with the supracallosal gyrus. The **supracallosal gyrus** (indusium griseum) is the thin layer of gray matter that extends from the subcallosal gyrus and covers the upper surface of the corpus callosum. The **medial and lateral longitudinal striae** are delicate longitudinal strands which extend along the upper surface of the corpus callosum. The **dentate fascia**, a thin crenated strip of cortex, lies on the upper surface of the hippocampal gyrus. The **hippocampus**, composed chiefly of gray substance, extends the length of the floor of the temporal horn of the lateral ventricle and becomes continuous with the supracallosal gyrus at the splenium of the corpus callosum. The **paraterminal body** is a triangular area of cortex lying just anterior to the lamina terminalis.

The **fornix** is an arched white fiber tract extending from the hippocampal formation. The **alveus** is the white layer on the ventricular surface of the hippocampus containing fibers from the