THE HUMAN BRAIN

An introduction to its functional anatomy

JOHN NOLTE, Ph.D.

THE HUMAN BRAIN

An introduction to its functional anatomy

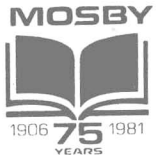
JOHN NOLTE, Ph.D.

Associate Professor of Anatomy, School of Medicine,
University of Colorado Health Sciences Center,
Denver, Colorado

With **379** *illustrations*

The C. V. Mosby Company

ST. LOUIS • TORONTO • LONDON 1981



A TRADITION OF PUBLISHING EXCELLENCE

Editor: John E. Lotz
Manuscript editor: Kendall Wills
Design: Diane Beasley
Production: Linda R. Stalnaker

Cover: Reproduction of a drawing by Rene Descartes in *De Homine*, 1662. Descartes thought that the pineal gland was the seat of the soul, monitoring the movement of "animal spirits" in sensory nerves and controlling the movement of animal spirits through motor nerves.

Printed in the United States of America

The C.V. Mosby Company
11830 Westline Industrial Drive, St. Louis, Missouri 63141

Library of Congress Cataloging in Publication Data

Nolte, John.
The human brain.

Includes bibliographies and index.
1. Neuroanatomy. I. Title. [DNLM: 1. Nervous system—Anatomy and histology. WL 101 N789n]
QM451.N64 611'.8 81-38337
ISBN 0-8016-3702-3 AACR2

GW/VH/VH 9 8 7 6 5 4 3 2 1 03/A/308

PREFACE

The human brain is a marvel in terms of what it allows us to do. Neurologists are fond of saying that the only really important function of the other parts of our bodies is to support the brain. Unfortunately, students often find the brain to be a marvel of complexity as well. I have tried to write a book that will lead beginning students through the basic aspects of the structure and function of their brains. It was designed primarily with students of the health sciences in mind, but I hope others will also find it useful.

In discussing the various parts of the nervous system and its environment, I have tried to restrict myself as much as possible to facts and details that can be correlated with function in some way. To bring home the correlation between structure and function, I have included numerous examples of clinical and experimental findings following damage to or manipulation of the nervous systems of humans and laboratory animals. Because of the difficulty of visualizing the structures and pathways of the brain in three dimensions, a wide variety of diagrams and photographs is also included.

A short and highly selective bibliography accompanies each chapter. The intent in choosing references was not only to document recent findings but also to provide access through those references to the vast neuroscientific literature. For these reasons, the emphasis is on recent reviews and research papers. Many classic and scientifically more important papers have been omitted, although some older or peripherally related articles that make interesting reading are listed.

This book was far from a solitary undertaking and could never have come about without the help and support of many people. I am especially grateful to my friends and colleagues Tom Finger, Claude Selitrennikoff, and Ted Tarby, who read parts (or all) of the manuscript for me and offered many helpful suggestions; to Gary Jenison, Stu Smith, and Jack Willson, who prepared some of the anatomic materials used for illustrations; to Shelley Frisch, Kris McInvaille, and Martha Potter, who watched my gesticulations and somehow came up with drawings; to Betty Aguilar and Sharon Ferdinandsen, who transformed my chicken scratchings into a manuscript; and to all the fellow students of the nervous system, cited in figure captions, who kindly provided illustrations. A very special thanks to Pam Eller, who prepared most of the anatomic materials, did most of the photography, read and commented on the manuscript, and patted me on the head a lot; without her help this would have been a lesser book or no book at all. Most of the photographs in the book depict materials developed for teaching purposes by my just-mentioned colleagues and me; I appreciate the cooperation of the University of Colorado Health Sciences Center in allowing them to be used in this book. Finally, I must acknowledge the students with whom it has been my pleasure to work at the University of Colorado Health Sciences Center; they have made teaching fun, and from the fun arose the notion of writing a book.

John Nolte

PREFACE

The brain, and the brain alone, is the source of our pleasures, joys, laughter, and amusement, as well as our sorrow, pain, grief, and tears. It is especially the organ we use to think and learn, see and hear, to distinguish the ugly from the beautiful, the bad from the good, and the pleasant from the unpleasant. The brain is also the seat of madness and delirium, of the fears and terrors which assail by night or by day, of sleeplessness, awkward mistakes and thoughts that will not come, of pointless anxieties, forgetfulness and eccentricities.

Hippocrates, ca. 400 B.C.

The human mind can be described as a slow-clockrate modified-digital machine with multiple distinguishable parallel processing, all working in salt water.

Philip Morrison: The mind of the machine, Technology Review 75:17, 1973

One of the difficulties in understanding the brain is that it is like nothing so much as a lump of porridge.

From Eye and brain: The psychology of seeing by R.L. Gregory. Copyright © 1966. Used with the permission of McGraw-Hill Book Company.

CONTENTS

CHAPTER 1

INTRODUCTION: THE CENTRAL NERVOUS SYSTEM

The object of this book is to present and explain some basic anatomical facts about how the brain is put together and to discuss a few aspects of how it works. This introductory chapter describes in a very general way the elements that make up the central nervous system (CNS) and some principles according to which these elements are connected to each other. In addition, there is a discussion of how the relatively simple embryonic nervous system is transformed into the much more complex CNS of the adult.

WHAT IS IN IT?

The principal cellular elements of the nervous system are *neurons* and *glial cells*, both present in enormous numbers. There are an estimated 100 billion neurons in the human brain and perhaps 10 times that many glial cells.

Neurons come in a great variety of sizes and shapes (Fig. 1-1); most of them have a long process called an *axon* that conducts information away from the neuronal cell body (or *soma*) and a series of smaller processes called *dendrites* that receive information from other neurons via *synaptic contacts* (or *synapses*). Certain aspects of somatic, dendritic, and axonal morphology give rise to a descriptive terminology for neurons. The vast majority of human neurons are *multipolar*, meaning that there are multiple dendritic projections from the cell body and almost always an axon as well (Fig. 1-1, *C* and *D*). Some are *unipolar* (Fig. 1-1, *A*) or *bipolar* (Fig. 1-1, *B*), having one or two processes, respectively. Neurons are also subdivided into *Golgi type I*, which have a long axon, and *Golgi type II*, which have a short axon or no axon at all.

Neurons may also be classified according to their con-

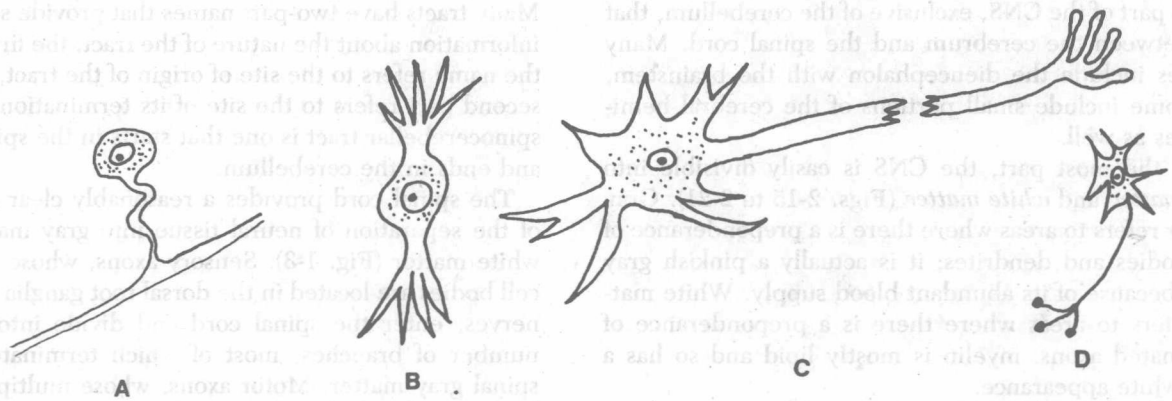


Fig. 1-1. Schematic illustrations of representative neurons. **A,** Unipolar neuron, like those found in the sensory ganglia of spinal and cranial nerves. **B,** Bipolar neuron, like those found in the retina. **C,** Multipolar cell with a long axon (Golgi type I), like the motor neurons found in the spinal gray matter. **D,** Multipolar cell with a short axon (Golgi type II), like the granule cells of the cerebral cortex.

nections. *Sensory neurons* are either directly sensitive to various stimuli (such as touch or temperature changes) or else receive direct connections from nonneuronal *receptor cells*. *Motor neurons* end directly on muscles or glands. *Interneurons* interconnect other neurons. In a strict sense, the CNS is composed almost entirely of interneurons: there are only about 5 million sensory fibers in all the spinal and cranial nerves combined, and only several hundred thousand motor neurons. Thus more than 99.99% of the neurons are interneurons. However, the words "sensory" and "motor" are often used in a much broader sense to refer to cells and axons that carry information related to sensory stimuli and to the generation of movements, respectively.

There are a variety of types of glial cells in the nervous system. Some of them (*oligodendroglia* in the CNS and *Schwann cells* in peripheral nerves) form *myelin sheaths*, which are spiral wrappings around axons that allow the axons to conduct information more rapidly. Other types of glia are thought to perform a variety of functions, such as regulating the composition of extracellular space, complementing neurons in certain metabolic activities, and devouring foreign materials that have gained access to the nervous system.

HOW IS IT ORGANIZED?

The CNS is composed of the *brain* and the *spinal cord* (Fig. 1-2). The brain, in turn, is composed of the *cerebrum*, the *cerebellum*, and the *brainstem*. There is general agreement on what the cerebellum is, but not all authors distinguish the cerebrum from the brainstem in the same way. In this book the cerebrum is treated as composed of the two massive *cerebral hemispheres* (separated by the *longitudinal fissure*) and the *diencephalon;* in an intact brain most of the diencephalon is hidden from view by the cerebral hemispheres. The brainstem is that part of the CNS, exclusive of the cerebellum, that lies between the cerebrum and the spinal cord. Many sources include the diencephalon with the brainstem, and some include small portions of the cerebral hemispheres as well.

For the most part, the CNS is easily divisible into *gray matter* and *white matter* (Figs. 2-15 to 2-21). Gray matter refers to areas where there is a preponderance of cell bodies and dendrites; it is actually a pinkish gray color because of its abundant blood supply. White matter refers to areas where there is a preponderance of myelinated axons; myelin is mostly lipid and so has a fatty white appearance.

Specific areas of gray matter are often called *nuclei*, particularly if the contained cell bodies are functionally related to one another. An area where gray matter forms a surface covering on some part of the CNS (and meets certain other technical criteria) is referred to as a

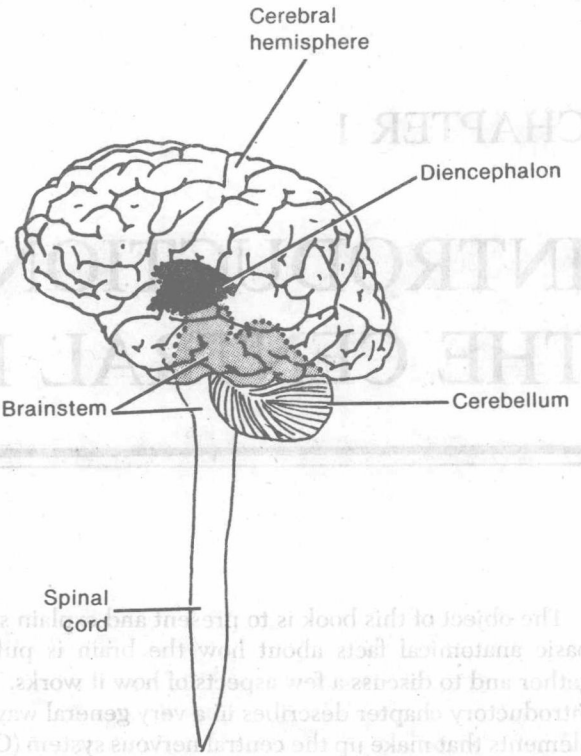


Fig. 1-2. Major divisions of the central nervous system. Stippled areas outlined by dotted lines are normally hidden from view by the massive cerebral hemispheres; they can be seen in a hemisected brain (Fig. 2-3).

cortex. The cerebral and cerebellar cortices are two prominent examples. Occasionally other names such as *body* or *center* are used for areas of gray matter, but these are relatively infrequent.

In contrast, subdivisions of white matter go by a bewildering variety of names, such as *fasciculus*, *funiculus*, *lemniscus*, *peduncle*, and, used most commonly, *tract*. Many tracts have two-part names that provide some free information about the nature of the tract: the first part of the name refers to the site of origin of the tract, and the second part refers to the site of its termination. Thus a spinocerebellar tract is one that starts in the spinal cord and ends in the cerebellum.

The spinal cord provides a reasonably clear example of the separation of neural tissue into gray matter and white matter (Fig. 1-3). Sensory axons, whose unipolar cell bodies are located in the dorsal root ganglia of spinal nerves, enter the spinal cord and divide into a large number of branches, most of which terminate in the spinal gray matter. Motor axons, whose multipolar cell bodies are located in the spinal gray matter, leave the spinal cord and enter spinal nerves. The white matter contains *long descending tracts* (from the brainstem and cerebrum), *long ascending tracts* (to the brainstem, cerebellum, and cerebrum), and local axons intercon-

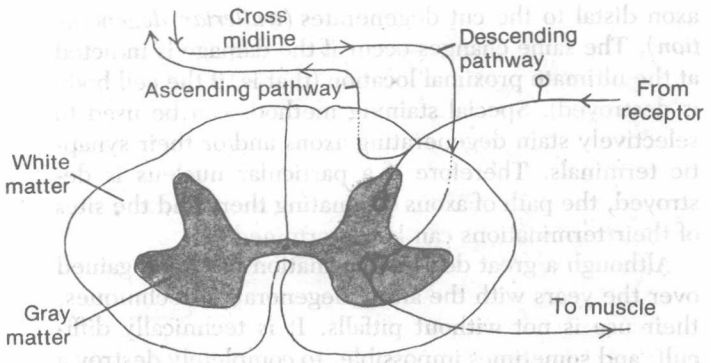


Fig. 1-3. Division of CNS into gray matter (stippled) and white matter, as illustrated by the spinal cord in cross section. Gray matter contains interneurons, motor neurons, and endings of sensory fibers. White matter contains ascending and descending pathways. Ascending pathways usually include interneurons, and descending pathways usually end on interneurons, but these links are omitted in this diagram. One axon in each ascending and descending pathway usually crosses the midline at some point.

necting different spinal levels. The gray matter, on the other hand, contains motor neurons, the endings of incoming sensory axons and long descending tracts, local interneurons, and tract cells whose axons enter long ascending tracts. This division into white and gray matter is not absolute anywhere in the CNS; for example, axons in long descending tracts obviously must pass through some gray matter before reaching their targets.

Ascending (sensory) pathways usually cross to the opposite side of the CNS at some point before reaching the cerebrum (Fig. 1-3). That is, they become *contralateral* to the side on which they originate, so information from the right hand eventually reaches the left cerebral hemisphere.* This crossing of sensory pathways is a curious and unexplained fact of vertebrate evolution. It applies not only to those pathways representing spinal nerves but to those representing cranial nerves as well (except for olfaction; see Chapter 16). A number of hypotheses have been advanced to explain this phenomenon, including the notion that it is an early evolutionary mistake that is still awaiting correction.† Whatever its explanation, it would be even more peculiar if information from the *right* hand reached the left cerebral hemisphere, which in turn controlled the *left* hand. This is not the case, since decending pathways also cross the midline at some point between their origins and their terminations (Fig. 1-3).

*This does not mean that most ascending *axons* cross the midline. The pathway from a peripheral receptor to the cerebral cortex, as will be detailed in subsequent chapters, includes at least three serially arranged neurons; the axon of only one of these crosses the midline.

†Zill, Sasha N.: Personal communication, 1977.

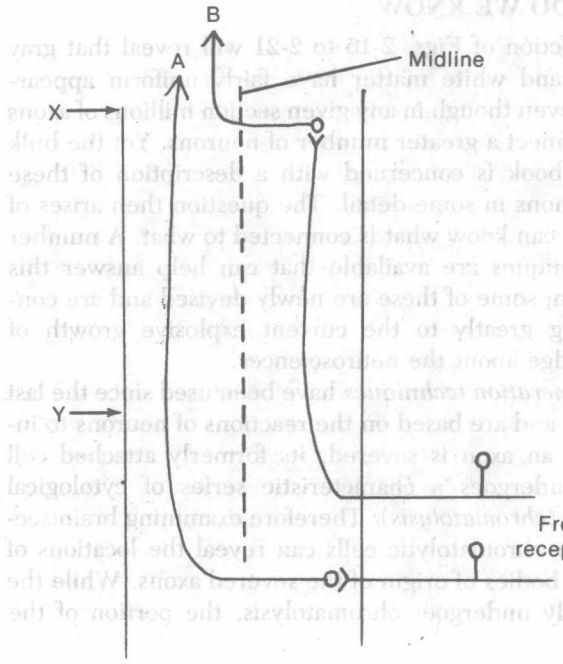


Fig. 1-4. Different pathways may cross the midline at different points in the CNS. Damage to one side of the CNS at point *X* would cause both A- and B-type deficits on the side of the body contralateral to the lesion. Damage to one side of the CNS at point *Y* would cause an A-type deficit on the side of the body contralateral to the lesion and a B-type deficit on the side ipsilateral to the lesion. (The CNS is generally bilaterally symmetrical, but for simplicity pathways *A* and *B* are shown arising on one side only; damage at point *Y* should damage pathway B on the side of the lesion before it crossed.)

It follows from this crossing pattern that damage to the appropriate regions of one cerebral hemisphere will cause sensory and/or motor deficits on the contralateral side. This is also frequently true of damage at lower levels such as the brainstem. However, different pathways cross at different levels within the CNS. Therefore it is possible for a single lesion to affect one pathway before it crosses and another pathway after it crosses (Fig. 1-4). The result would be the curious finding of one type of deficit on one side of the head or body and a second type of deficit on the other side.

As is generally the case, the generalization about crossing pathways has exceptions. Some sensory pathways (for example, auditory) ascend bilaterally, and some motor neurons (for example, trigeminal) are innervated by both cerebral hemispheres. The most outstanding exception is the cerebellum. Damage to one side of the cerebellum produces deficits on the same side of the body (that is, the side *ipsilateral* to the lesion). The anatomical bases of this ipsilaterality are discussed in Chapter 14.

HOW DO WE KNOW?

Inspection of Figs. 2-15 to 2-21 will reveal that gray matter and white matter have fairly uniform appearances, even though in any given section millions of axons interconnect a greater number of neurons. Yet the bulk of this book is concerned with a description of these connections in some detail. The question then arises of how we can know what is connected to what. A number of techniques are available that can help answer this question; some of these are newly devised and are contributing greatly to the current explosive growth of knowledge about the neurosciences.

Degeneration techniques have been used since the last century and are based on the reactions of neurons to injury. If an axon is severed, its formerly attached cell body undergoes a characteristic series of cytological changes *(chromatolysis)*. Therefore examining brain sections for chromatolytic cells can reveal the locations of the cell bodies of origin of the severed axons. While the cell body undergoes chromatolysis, the portion of the axon distal to the cut degenerates *(wallerian degeneration)*. The same changes occur if the damage is inflicted at the ultimate proximal location (that is, if the cell body is destroyed). Special staining methods can be used to selectively stain degenerating axons and/or their synaptic terminals. Therefore if a particular nucleus is destroyed, the path of axons originating there and the sites of their terminations can be determined.

Although a great deal of information has been gained over the years with the aid of degeneration techniques, their use is not without pitfalls. It is technically difficult, and sometimes impossible, to completely destroy a particular structure without damaging nearby structures as well. In addition, since the segregation of gray and white matter is not absolute, axons passing through a given nucleus can be destroyed along with the cell bodies forming the nucleus. For these and other reasons, various tracer techniques developed in recent years have been greeted with much enthusiasm. Most of these methods take advantage of the fact that under

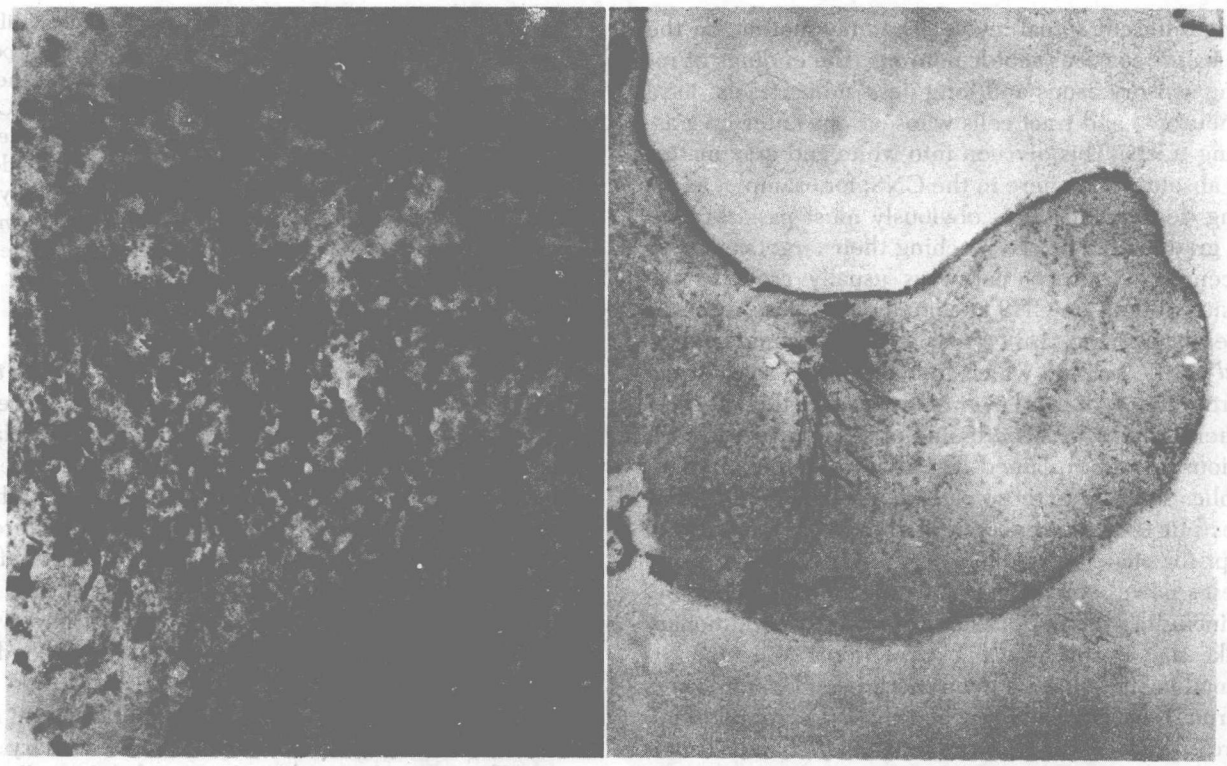


Fig. 1-5. Examples of the use of horseradish peroxidase (HRP) to label neuronal pathways. **A**, HRP applied to the proximal end of the lateral line nerve of a fish after cutting the nerve near its entrance into the brainstem; reaction product fills the entering fibers (thin arrows) and their synaptic endings (thick arrows) within the CNS. (Courtesy of Dr. Curtis Bell, Good Samaritan Hospital.) **B**, HRP applied to the proximal end of the hypoglossal nerve of a frog after cutting the nerve near its entrance into the brainstem; in this case the HRP was transported in a retrograde direction, and reaction product fills the hypoglossal motor neurons (thick arrow) and their axons (thin arrow). (Courtesy of Dr. Thomas Finger, Dr. Stephen Roper, and Barbara Taylor, University of Colorado Medical Center.)

normal circumstances substances are transported from a neuronal cell body down its axon toward its synapses *(anterograde transport)* and in the reverse direction as well *(retrograde transport)*. Appropriate radioactive substances (usually tritiated amino acids) introduced into a nucleus are taken up by the resident neurons, incorporated into macromolecules, and transported down the axons of these neurons. Eventually the synaptic terminals of these axons become radioactive.

In another method, a protein is introduced into areas of synaptic terminals. The terminals take up the protein, which is then transported back to the parent neurons. The protein of choice for such experiments is an enzyme called *horseradish peroxidase*, which can be detected with great sensitivity and resolution by appropriate histochemical procedures (Fig. 1-5).

Other anatomical methods utilize neural tissue that has not been experimentally manipulated prior to processing. The *Golgi technique* is a staining method that completely stains the cell bodies and processes of a small, random sample of neurons in the tissue (Fig. 11-2, *A*). This is useful for determining the sizes and shapes of the cell types in a given area of the CNS. In addition, different classes of neurons have chemically different interiors, some of which can be distinguished by histochemical techniques. For example, neurons that use norepinephrine as the chemical transmitter at their synapses contain this substance throughout their axons and cell bodies. Appropriate fixation and processing causes these neurons to be fluorescent.

Finally, electrophysiological techniques have added a great deal of information about pathways and connections, much of which could not have been obtained by strictly anatomical studies. Such experiments range from those in which the electrical activity of various cortical areas is measured after stimulation of peripheral nerves and receptors to those in which the activity and connections of single neurons is measured.

HOW DID IT GET THAT WAY?

As complex as the human nervous system is, it has its embryonic origin as a simple, tubular, ectodermal structure. Its development provides some useful insights into its adult configuration and organization.

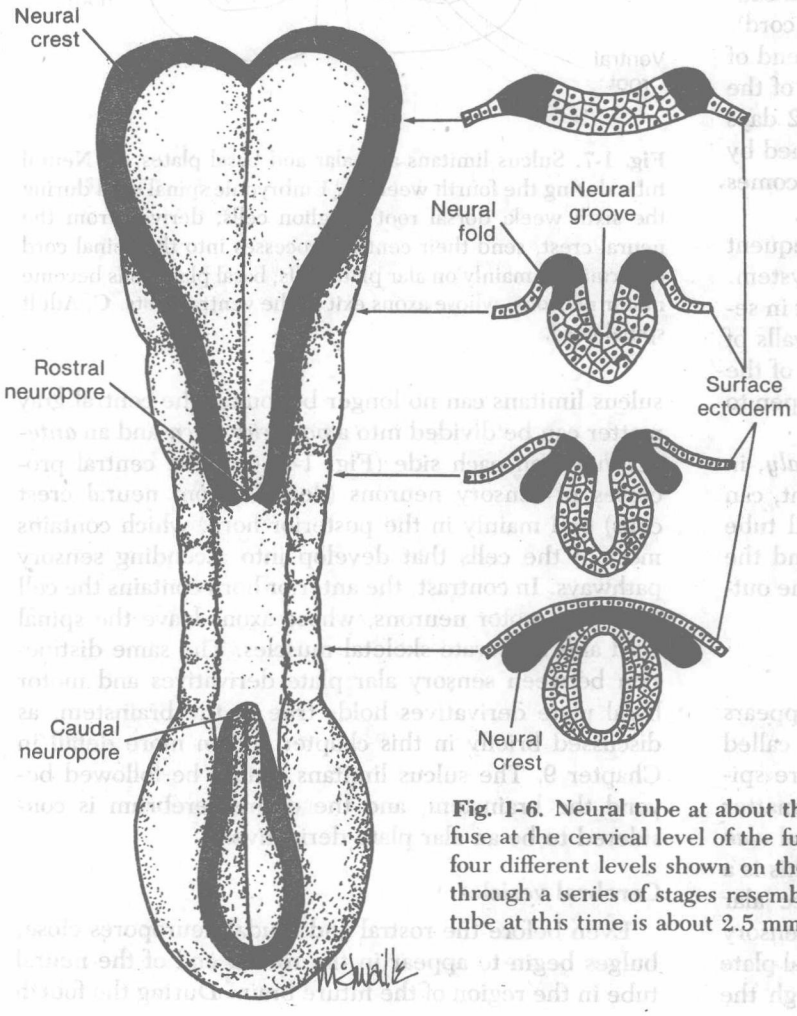


Fig. 1-6. Neural tube at about the end of the third week. Neural folds have begun to fuse at the cervical level of the future spinal cord. Cross sections of the neural tube at four different levels shown on the right; at any given level, the embryonic CNS goes through a series of stages resembling these four cross sections. Total length of neural tube at this time is about 2.5 mm.

Neural tube

During the third week of embryonic development a longitudinal band of ectoderm thickens to form the *neural plate*. Shortly thereafter the neural plate begins to fold inward, forming a longitudinal *neural groove* in the midline flanked by a parallel *neural fold* on each side (Fig. 1-6). The neural groove deepens, and the neural folds approach each other in the midline. At the end of the third week the two folds begin to fuse midway along the neural groove, forming the *neural tube*. As this fusion occurs, groups of cells from the crest of each neural fold are pinched off from the neural tube. These are appropriately called *neural crest cells*, which develop into a variety of cell types, including the sensory neurons of the ganglia of spinal and cranial nerves, the postganglionic neurons of the autonomic nervous system, and the Schwann cells and satellite cells of the peripheral nervous system. The neural tube, on the other hand, develops into the entire CNS; its cavity becomes the *ventricular system* of the brain.

The area of fusion of the two neural folds, which begins in the cervical region of the future spinal cord, rapidly expands rostrally (toward the future brain) and caudally (toward the sacral end of the future spinal cord). The *rostral neuropore*, the opening at the rostral end of the neural tube, closes completely in the middle of the fourth week; the *caudal neuropore* closes about 2 days later. As each successive bit of neural tube is formed by the progressive fusion of the neural folds, it becomes separated from the overlying ectoderm (Fig. 1-6).

Defective closure of the neural tube is a frequent cause of congenital malformations of the nervous system. Failure of the caudal neuropore to close can result in severe forms of *spina bifida*, in which the caudal walls of the neural tube are still continuous with the skin of the back, and the central cavity of the neural tube is open to the outside.

If the rostral neuropore fails to close, *anencephaly*, in which much of each cerebral hemisphere is absent, can result. As in spina bifida, the walls of the neural tube may be continuous with the skin of the head, and the central cavity of the neural tube may be open to the outside.

Sulcus limitans

During the fourth week a longitudinal groove appears in the lateral wall of the neural tube. This groove, called the *sulcus limitans*, extends throughout the future spinal cord and brainstem and subdivides the gray matter in the walls of the neural tube into a more dorsal *alar plate* and a more ventral *basal plate* (Fig. 1-7). This is a distinction of some functional importance, since alar plate derivatives are primarily concerned with sensory processes, while motor neurons are located in basal plate derivatives. In the adult spinal cord, even though the sulcus limitans can no longer be found, the central gray matter can be divided into a *posterior horn* and an *anterior horn* on each side (Fig. 1-7, *C*). The central processes of sensory neurons (derived from neural crest cells) end mainly in the posterior horn, which contains most of the cells that develop into ascending sensory pathways. In contrast, the anterior horn contains the cell bodies of motor neurons, whose axons leave the spinal cord and innervate skeletal muscles. The same distinction between sensory alar plate derivatives and motor basal plate derivatives holds true in the brainstem, as discussed briefly in this chapter and in more detail in Chapter 9. The sulcus limitans cannot be followed beyond the brainstem, and the entire cerebrum is considered to be an alar plate derivative.

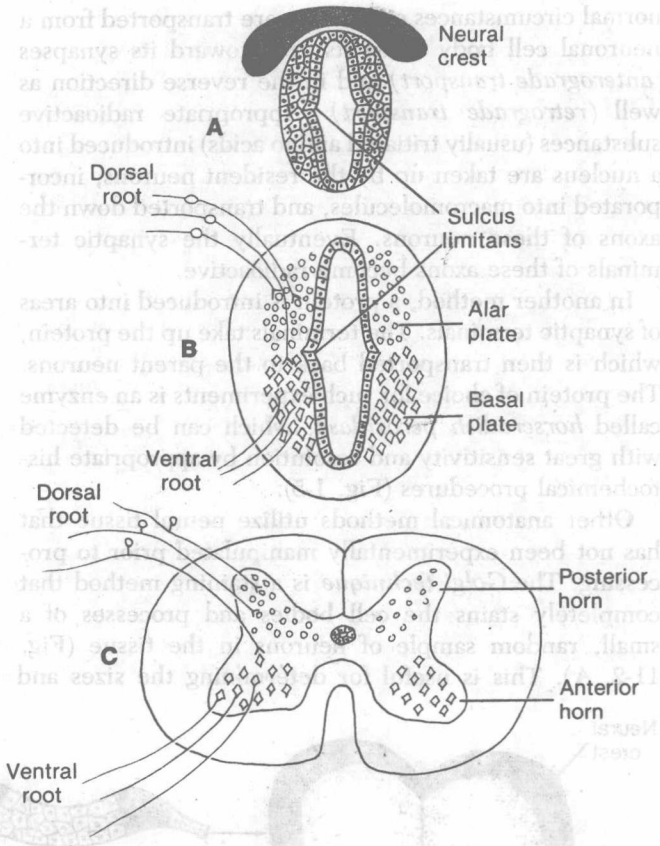


Fig. 1-7. Sulcus limitans and alar and basal plates. **A**, Neural tube during the fourth week. **B**, Embryonic spinal cord during the sixth week; dorsal root ganglion cells, derived from the neural crest, send their central processes into the spinal cord to terminate mainly on alar plate cells; basal plate cells become motor neurons, whose axons exit in the ventral roots. **C**, Adult spinal cord.

Cerebral vesicles

Even before the rostral and caudal neuropores close, bulges begin to appear in the rostral end of the neural tube in the region of the future brain. During the fourth

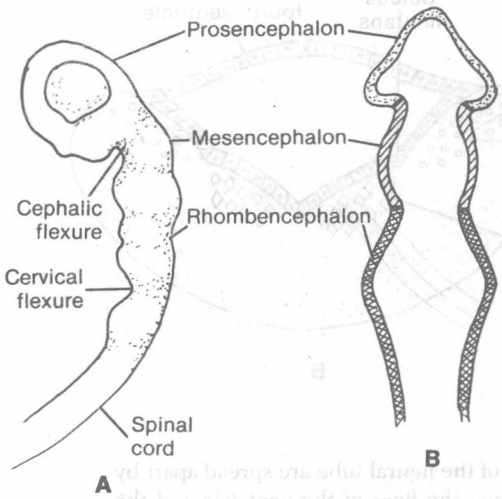


Fig. 1-8. Primary vesicles at the end of the fourth week.

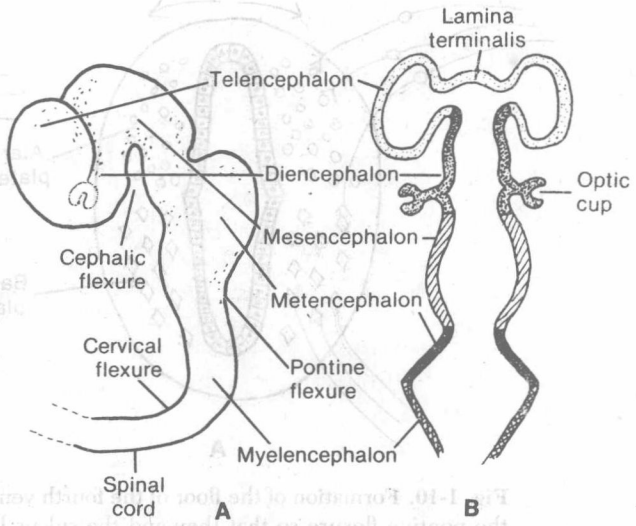


Fig. 1-9. Secondary vesicles during the sixth week.

TABLE 1
Derivatives of vesicles of the neural tube

Primary vesicle	Secondary vesicle	Neural derivatives	Cavity
Prosencephalon	Telencephalon	Cerebral hemispheres	Lateral ventricles
	Diencephalon	Thalamus, hypothalamus, etc.	Third ventricle
Mesencephalon	Mesencephalon	Midbrain	Cerebral aqueduct
Rhombencephalon	Metencephalon	Pons and cerebellum	Part of fourth ventricle
	Myelencephalon	Medulla	Part of fourth ventricle
			Part of central canal

week three bulges, or vesicles, are apparent and are referred to as the *primary vesicles* (Fig. 1-8, *B*). From rostral to caudal, these are the *prosencephalon* (forebrain), the *mesencephalon* (midbrain), and the *rhombencephalon* (hindbrain), which merges smoothly with the spinal portion of the neural tube. The prosencephalon develops into the cerebrum. The mesencephalon becomes the midbrain of the adult brainstem, and the rhombencephalon becomes the rest of the brainstem and the cerebellum (Table 1).

The three primary vesicles are not arranged in a straight line but rather are associated with two bends or flexures in the neural tube (Fig. 1-8, *A*). One of these, the *cervical flexure,* occurs between the rhombencephalon and spinal cord but does not persist in the adult CNS. The second, the *cephalic* (or *mesencephalic*) *flexure,* occurs between the mesencephalon and the prosencephalon; it persists in the adult as the bend between the axes of the brainstem and the forebrain (Fig. 2-1).

As the brain continues to develop, two of the primary vesicles become subdivided. During the fifth week five *secondary vesicles* can be distinguished (Fig. 1-9, *B*). The prosencephalon gives rise to the *telencephalon* and the *diencephalon;* the mesencephalon remains undivided; the rhombencephalon gives rise to the *metencephalon* and the *myelencephalon*. The telencephalon becomes the cerebral hemispheres of the adult brain. The diencephalon gives rise to the *thalamus* (a large mass of gray matter interposed between the cerebral cortex and other structures), the *hypothalamus* (an autonomic control center), the neural part of the eye, and several other structures. The metencephalon becomes the *pons* (part of the brainstem) and the cerebellum. The myelencephalon becomes the *medulla* (the part of the brainstem that merges with the spinal cord).

In addition, a *pontine flexure* appears in the dorsal surface of the brainstem between the metencephalon and the myelencephalon (Fig. 1-8, *A*). This flexure does not persist as a bend in the axis of the brainstem, but it does have important consequenees for the configuration of the caudal brainstem. As the flexure develops, the walls of the neural tube spread apart to form a diamond-shaped cavity (hence the name "rhombencephalon") so that only a thin membranous roof remains over what will become the *fourth ventricle* (Fig. 1-10). Thus the alar and basal plates, still separated by the sulcus limitans,

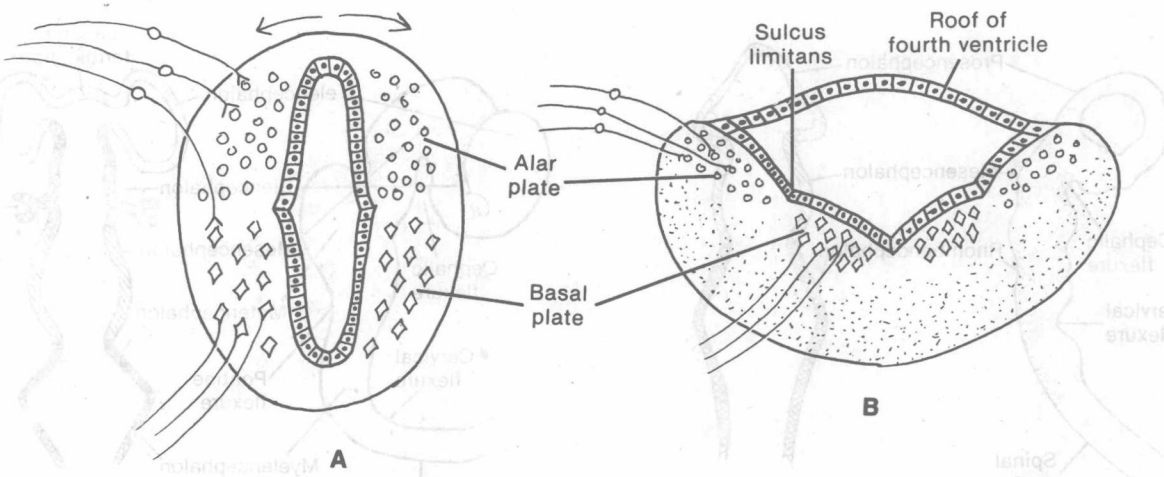


Fig. 1-10. Formation of the floor of the fourth ventricle. Walls of the neural tube are spread apart by the pontine flexure so that they and the sulcus limitans become the floor of the ventricle and the roof becomes a thin membrane.

come to lie in the floor of the fourth ventricle. The result is that in the corresponding part of the adult brainstem (rostral medulla and caudal pons), sensory nuclei are located lateral, rather than posterior, to motor nuclei. As discussed further in Chapter 9, this is of some utility in making sense of the arrangement of cranial nerve nuclei.

Lateral portions of the alar plate in the rostral metencephalon thicken considerably to form the *rhombic lips*. These continue to enlarge, finally fusing in the midline to form a transverse ridge that will eventually become the cerebellum.*

Further development

Subsequent events are dominated by the tremendous growth of the telencephalon. This portion of the neural tube begins as two swellings connected across the midline by a thin membrane, the *lamina terminalis* (Fig. 1-9). The basal part of the wall of the telencephalon, adjacent to the diencephalon, thickens to form the primordia of gray masses called the *basal ganglia* or *basal nuclei*. At the same time, the walls of the diencephalon thicken to form the thalamus and hypothalamus, separated by the *hypothalamic sulcus*. With continued growth, the telencephalon folds down alongside the diencephalon until eventually the two fuse (Fig. 1-11). The telencephalic surface overlying the area of fusion develops into a portion of the cerebral cortex called the *insula*. The remainder of the telencephalon grows rapidly in all available directions (Fig. 1-12), until the insula is completely hidden from view and each cerebral hemisphere has the shape of a great arc encircling the insular cortex (Fig. 1-11; compare Figs. 2-2 and 2-4). As discussed and demonstrated in the next chapter, knowledge of this growth of each cerebral hemisphere in a great C shape is of considerable importance for understanding the anatomical organization of the forebrain.

*Although the cerebellum develops from the alar plate, it is involved in motor functions in the sense that cerebellar lesions cause impairments of posture and movement but not of sensation (see Chapter 14).

Ventricles and choroid plexus

The cavity of the neural tube persists as the ventricular system of the adult brain (Fig. 4-1). Except for the rudimentary central canal of the spinal cord and caudal medulla, the ventricles comprise a continuous, fluid-filled series of spaces extending through all the major divisions of the CNS. The cavity of the pons and rostral medulla is the fourth ventricle; that of the diencephalon is the *third ventricle;* a large C-shaped *lateral ventricle* occupies each cerebral hemisphere. Each lateral ventricle communicates with the third ventricle through an *interventricular foramen* (Fig. 1-11), and the third ventricle communicates with the fourth ventricle through the *cerebral aqueduct* of the midbrain.

Where the walls of the rhombencephalon spread apart to form the fourth ventricle, the roof of the ventricle becomes extremely thin (Fig. 1-10). An area covering the roof of the third ventricle and extending onto the surface of the telencephalon becomes similarly thin (Fig. 1-13). At each of these locations, tufts of small blood vessels invaginate the ventricular roof to form the *choroid plexus*, which is responsible for the production of most of the *cerebrospinal fluid* that fills the ventricles. As each cerebral hemisphere grows around in a C shape, so too does the choroid plexus, which protrudes into its lateral ventricle.

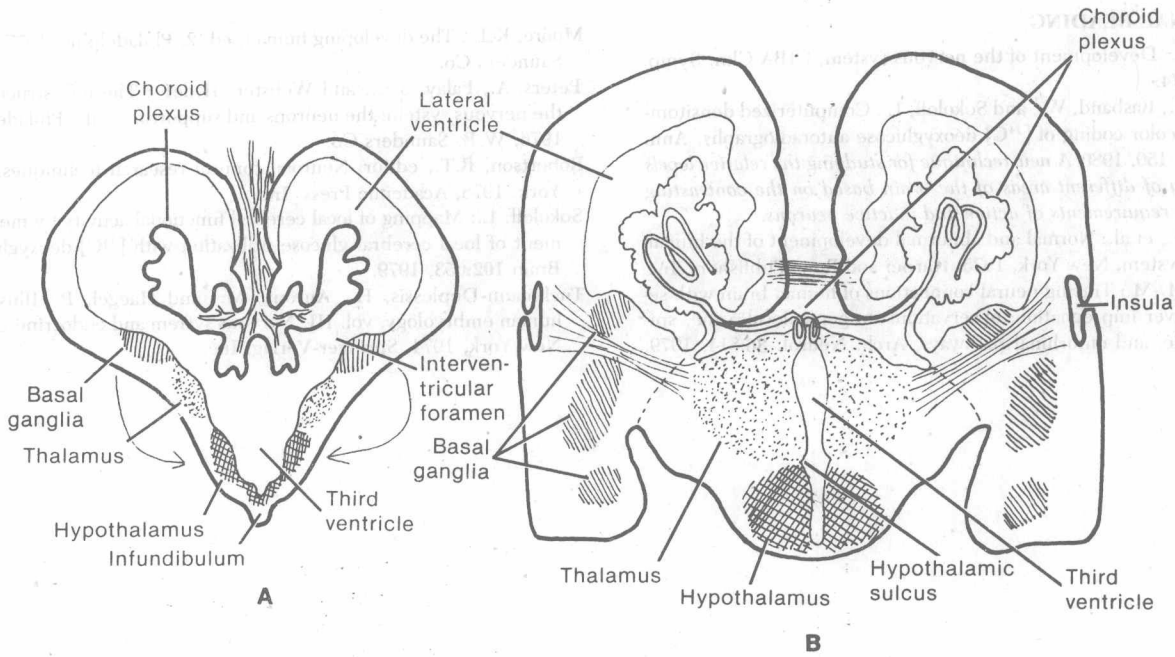


Fig. 1-11. Formation of choroid plexus and of the fusion between diencephalon and telencephalon. **A,** Condition at the end of the second month; vascular connective tissue has invaginated the third and lateral ventricles to form choroid plexus, and rapid growth of telencephalon begins to fold its basal ganglia down toward diencephalon, as indicated by arrows. **B,** Condition at the end of the third month; telencephalon and diencephalon have fused, as indicated by dashed line, and the insula, overlying the point of fusion, begins to be overgrown by other cerebral cortex. (Modified from Carpenter, M.: Human neuroanatomy, ed. 7, Baltimore, 1976, The Williams and Wilkins Co.)

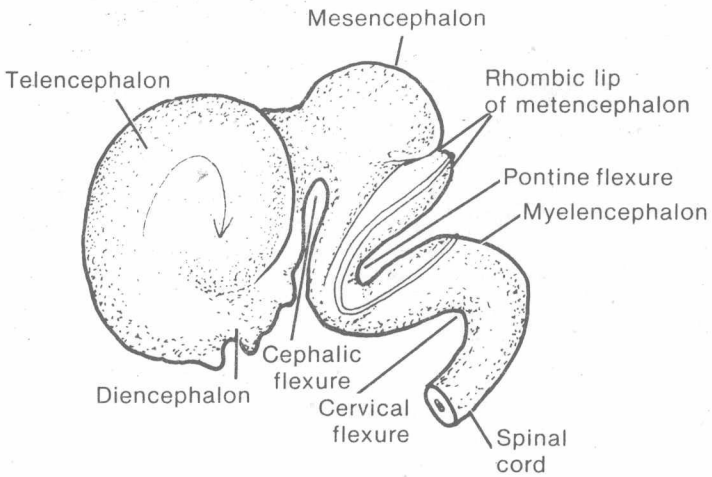


Fig. 1-12. Neural tube at the end of the second month. Development from this point is dominated by rapid growth of telencephalon in a C shape, as indicated by arrow.

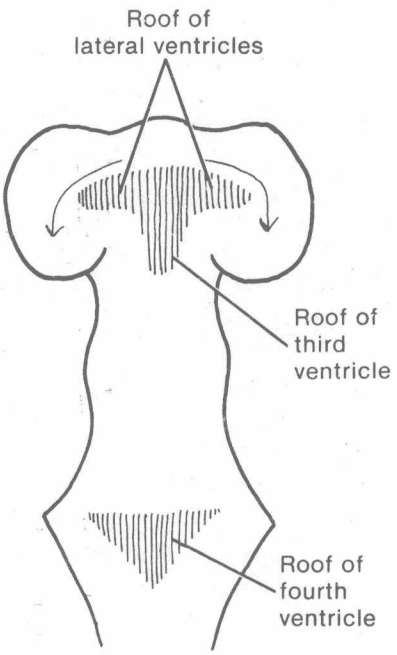


Fig. 1-13. Sites of formation of choroid plexus. As each cerebral hemisphere grows around in a C shape (indicated by arrows), so does its choroid plexus.