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# Manual of Patient Care in Neurosurgery

Second Edition

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# Preface

The **Manual of Patient Care in Neurosurgery** was originally intended to provide the surgical house officer with rapid reference to neurosurgical intensive care techniques. However, comments from readers of the first edition indicate that the work has also been useful for medical students, physician's assistants, and physicians and nurses specializing in critical care.

The purpose of this book continues to be to provide a concise, accessible introduction to the pathophysiology of neurosurgical disease processes and a rational approach to initiating therapy. It is not a comprehensive textbook of neurosurgery, and operative technique is discussed only insofar as it relates to preoperative and postoperative care. The growing emphasis on primary care in medical school curricula make it increasingly likely that little, if any, didactic training in neurosurgical care will have been acquired by the medical student or junior house officer before he or she begins clinical training on a neurosurgical service. This work is not a substitute for that training, but it may ease the trauma of acquiring on-the-job training. It is hoped that improved patient care will be a significant concomitant benefit.

A surprising number of technical innovations have occurred since the first edition was published, and the current edition has been rewritten to incorporate them. Recent work on cerebral metabolism and blood flow has added greatly to our understanding of the neurochemical disruption that is caused by hypoxemia and ischemia, suggesting new forms of treatment now under investigation. Evoked response recording for clinical electrophysiologic diagnosis is rapidly becoming an established diagnostic tool; in the next few years it will likely become a commonplace intraoperative procedure. New concepts of pituitary disease and its biochemical diagnosis have evolved. Computed tomography, digital vascular imaging, intraoperative ultrasonography, and other new imaging techniques have revolutionized neurologic diagnosis. These are just a few of the ex-

citing new developments in clinical neurosurgery that have prompted a near-total revision of this work.

Again, the scientific research is not original. The overall format has not been changed. Bibliographies have been kept small and relevant to the chapter topic by citing review articles only, and references have been updated when applicable.

Special thanks go to my wife, Rosemarie, for her encouragement and guidance, and to Kathleen O'Brien and Chere Bemelmans at Little, Brown for their patience and expert assistance. Thanks also to Jonathan Greene for his fine help with illustrations. Lastly, thanks to the many readers of the first edition who took the time to write to me with their helpful critical comments and suggestions. Many of these suggestions have been incorporated into this revision.

J.R.H.

# Manual of Patient Care in Neurosurgery

# Contents

# I

# Clinical Neurosurgical Physiology

**Notice.** The indications and dosages of all drugs in this book have been recommended in the medical literature and conform to the practices of the general medical community. The medications described do not necessarily have specific approval by the Food and Drug Administration for use in the diseases and dosages for which they are recommended. The package insert for each drug should be consulted for use and dosage as approved by the FDA. Because standards for usage change, it is advisable to keep abreast of revised recommendations, particularly those concerning new drugs.

# 1 Cerebral Metabolism and Blood Flow

Steven L. Giannotta

The brain is the most complex of organs. Although the human brain is certainly not the largest, it is more highly organized than that of other animals; consciousness, memory, and language are all attributable to its function. The basic functional unit of the central nervous system is the neuron, of which there are approximately 10 billion in the mature human being. Each neuron is filled with organelles that are specifically designed to carry out the multitude of biochemical processes required for transmission and storage of information. The neuron has one of the highest metabolic requirements of all mammalian cells; and, in association with perhaps 80 billion other glial and supporting cells, it is the neuron that makes the cerebrum unique in terms of its extraordinary metabolic and circulatory demands.

## Cerebral Metabolism

Energy for neural function, as for cells in other organs, comes from splitting energy-rich molecules of adenosine triphosphate (ATP). The majority of ATP is produced in the brain by oxidative metabolism of glucose in the presence of adenosine diphosphate (ADP) and inorganic phosphate ($P_i$). The following equation depicts the production of these energy-rich phosphate-containing compounds:

$$1\ \text{Glucose} + 6\ O_2 + 38\ \text{ADP} + 38\ P_i \rightarrow 6\ CO_2 + 44\ H_2O + 38\ \text{ATP}$$

It can be readily appreciated from this equation that oxygen plays a critical role in the generation of ATP. On a gram-per-gram basis, the brain requires a higher proportion of the body's total oxygen consumption than any other organ, including the heart and kidneys. This is true despite the fact that, by weight, the brain makes up only 2 percent of the body. Fifteen percent of the total cardiac output is required to deliver the appropriate amount of energy-rich material and oxygen to the brain. Unlike heart or skeletal muscle,

the brain does no physical work, despite this incredible appetite for substrate. Energy in the brain is expended simply for the operation of ionic pumps that maintain the proper concentration of ions inside the cells. Energy is also expended for the manufacture of neural transmitter substances, proteins, and the macromolecules that maintain the anatomic integrity of the central nervous system.

*Cerebral Metabolism of Glucose*

Most of the energy necessary for cerebral function comes from the ATP generated in the brain by using glucose and oxygen to combine inorganic phosphate with ADP (oxidative metabolism; aerobic glycolysis). Since the brain can neither store much glucose nor use other substrates (such as fats or proteins) for ATP production, a drop in the serum glucose level below normal may cause confusion or drowsiness [5]. Coma will ensue if the blood glucose falls below 20 mg/100 ml. If glucose is not restored to the brain, cerebral metabolism is severely curtailed and permanent neurologic damage may occur.

One of the ways of expressing cerebral metabolism in quantitative terms is to compute the amount of glucose consumed by the brain. Most of our knowledge of the rate of cerebral metabolism of glucose (CMRglu) comes from data obtained from animal studies. However, a new technique called positron emission tomography (PET) scanning allows the measurement of an analogue of glucose in human patients [10,15]. Investigators have found that the human brain consumes glucose at a rate of 77 mg/min or 5.5 mg/100 gm brain/min, which is 25 percent of all the glucose used by the body.

Although it is necessary to know the total amount of glucose consumed by the cerebrum, it is important to realize two other factors that affect both glucose requirement and rate of oxygen metabolism. First, there is a variation in metabolic rate for different regions of the brain. Motor cortex or thalamic nuclei densely packed with neuron cell bodies may have a resting CMRglu twice that of the average CMRglu of the whole brain. Conversely, white matter uses glucose at a rate that is one half that of the total brain [17].

An equally important concept in understanding cerebral metabolism is the link between energy use and functional activity. PET scanning in human beings, or similarly sophisticated mapping techniques in animals, has demonstrated marked regional elevations of energy use when the organism experiences sensory stimulation, or motor or mental activity. For instance, a complex visual stimulus (such as a geometric pattern) will elicit a 50 percent increase in CMRglu in the visual cortex [15]. Since the organism ex-

periences unpredictable, sudden, and infinitely varied stimuli, it is extremely important that a constant, steady supply of substrate (glucose) be available for instantaneous changes in the metabolic rate. Also, any change in cerebral blood flow (CBF) profoundly affects the brain's metabolism of glucose (see Cerebral Blood Flow).

*Cerebral Consumption of Oxygen*

The brain's energy consumption is produced almost exclusively by aerobic glycolysis, the metabolism of glucose in the presence of oxygen. The cerebrum requires 50 cc of oxygen each minute just to survive ($CMRO_2$). Thus, an organ that is only 2 percent of the body's weight requires 20 percent of the body's total supply of oxygen. Liver and skeletal muscle represent a considerably larger proportion of the body weight but demand only about the same percent of the oxygen supply. Since the demand for oxygen by the brain is continuous and since there is no mechanism for storing oxygen, significant hypoxemia will cause coma within 2 to 3 minutes [17]. If circulation to the brain is interrupted (such as during cardiac arrest), unconsciousness ensues within 10 seconds. Permanent neurologic damage will be incurred if the circulation is not supported properly either by cardiac massage or by some other mechanical means.

When the amount of oxygen in the blood decreases abruptly (as in cardiac arrest), normal oxidative metabolism ceases. For a brief period of time, anaerobic glycolysis is increased. The metabolism of glucose in the absence of oxygen is described by the following equation:

$$\text{Glucose} + 2\,\text{ADP} + P_i \rightarrow 2\,\text{lactate} + \text{ATP}$$

Unfortunately anaerobic glycolysis produces less than 40 percent of the brain's energy requirement and generates a large amount of lactate, which severely lowers the pH of brain tissue to a toxic level. Prompt reinstitution of oxygen is necessary to revert to aerobic glycolysis before permanent brain damage is sustained.

## Cerebral Blood Flow

*Arterial System*

Approximately 850 ml of oxygenated blood from the heart is delivered to the brain each minute. Oxygenated blood flows through paired carotid and vertebral arteries, which arise as major branches from the aortic arch. The common carotid arteries bifurcate in the neck. The two branches form the external carotid artery, which supplies the face, scalp, and cranial meninges, and the

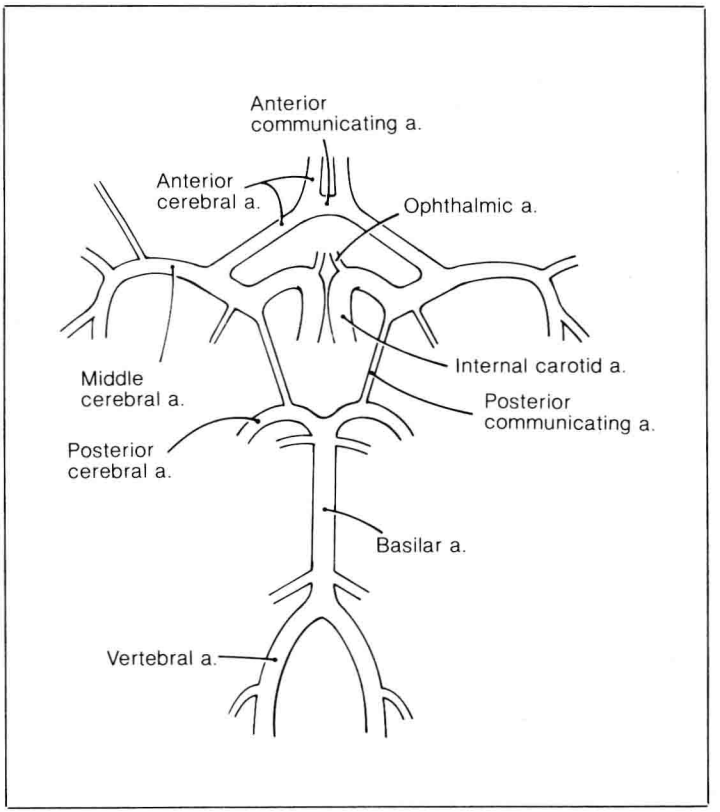


Fig. 1-1. The circle of Willis. The anterior and posterior communicating arteries are important potential channels of collateral circulation.

internal carotid artery, which penetrates the skull base and supplies the brain. Approximately 80 percent of the brain's blood supply travels through the internal carotid artery and is distributed mostly to the frontal, temporal, and parietal lobes, and to the basal ganglia and thalamus through branches of the anterior and middle cerebral arteries.

The vertebral arteries ascend from the aortic arch through the foramina transversaria of the cervical vertebrae and join at the base of the skull to form the basilar artery. Branches of the vertebral and basilar arteries supply the brainstem and cerebellum. The terminal branches of the basilar artery are the posterior cerebral arteries, which irrigate the occipital and inferior portions of the temporal lobe (Fig. 1-1).

*Collateral Circulation*

Idealized drawings of the arterial anatomy of the base of the brain often show well-developed anterior communicating and posterior communicating arteries that link the internal carotid circulation with the vertebral basilar circulation (see Fig. 1-1). Such an anatomical situation allows for collateral blood flow if one or more major arterial conduits are interrupted. The circle of Willis, as it has come to be known, is only a potential mechanism for collateral flow, because one or more of the three major communicating arteries are either rudimentary or missing altogether in approximately 35 percent of patients studied by angiography. Therefore, many clinicians speak of the anterior circulation (internal carotid and major branches) versus the posterior circulation (vertebral basilar system) as though they are physiologically distinct. In patients whose circle of Willis is anatomically incomplete, acute occlusion of either a carotid or vertebral artery (owing to trauma or atherosclerotic thrombosis for instance) may result in an ischemic neurologic deficit. Redistribution of arterial flow may obviate a potential ischemic injury when there is an anatomically complete circle of Willis.

When a major extracranial vessel (such as the carotid or vertebral artery) becomes occluded, anastomoses may form between the external carotid circulation and the surface arteries of the brain. Collateral blood flow in atherosclerotic disease often is the result of the anastomoses of the external carotid branches to the ophthalmic artery, which produce retrograde flow to the internal carotid circulation. Intracerebral collateral channels are meager at best. Thrombosis of a parenchymal artery may be followed by very little shunting of flow to the ischemic region. Therefore, some element of infarction usually occurs in such a situation.

*Venous System*

The cerebral venous network is a valveless series of vessels, which can be roughly divided into two divisions. The surface and deep veins drain the parenchyma of the brain, and their anatomy shows a high degree of regional variation. Another division of the venous system is the major venous sinuses, which are large conduits located within the major dural reflections, including the falx and tentorium. The venous sinuses receive blood from the cortical and deep cerebral systems and in turn empty into the internal jugular veins. The sagittal venous sinus is also the main site for reabsorption of spinal fluid back into the bloodstream. Occlusion of a surface vein may be well tolerated owing to the presence of collateral pathways, but occlusion of a deep venous channel or a ma-

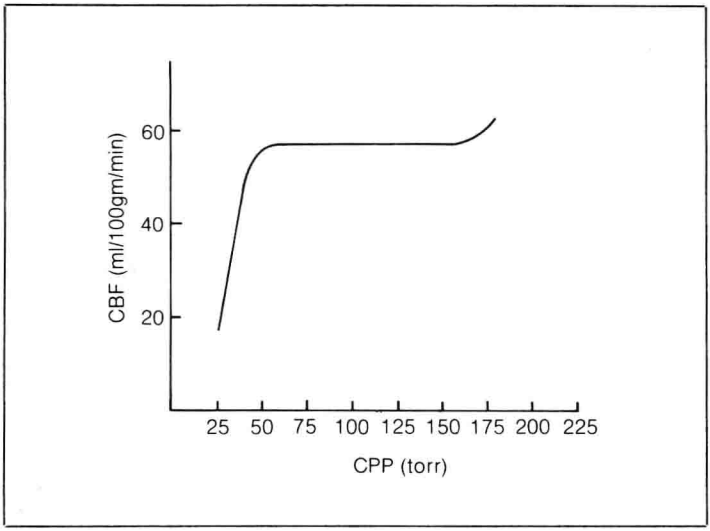


Fig. 1-2. Cerebral autoregulation curve demonstrating constancy of cerebral blood flow (CBF) between cerebral perfusion pressures (CPP) of 50 to 150 torr.

jor venous sinus can result in hydrocephalus in a child or increased intracranial pressure or even venous infarction in an adult.

*Regulation of Cerebral Blood Flow*

Since a constant and ready source of oxygen and glucose is required for cerebral function, total CBF must likewise be continuous throughout all changes in physiologic conditions. In human beings, CBF remains at a fairly constant 55 ml/100 gm tissue/min. A steady CBF is maintained while cerebral structures and regions are perfused roughly in proportion to their metabolic demands which are closely dependent on function. Regional blood flow rates will, therefore, parallel changes in local energy metabolism. The mechanisms responsible for such close coupling of CBF and functional activity are not entirely known.

Changes in physiologic conditions will cause portions of the cerebral vasculature to actively dilate or constrict in order to regulate blood flow. The brain's ability to maintain a stable total flow despite changes in blood pressure is known as cerebral autoregulation. Figure 1-2 is an autoregulation curve demonstrating the constancy of CBF between cerebral perfusion pressures (CPP) of 50 to 150 torr; or CPP = MAP – ICP, where MAP is mean systemic