Current Surgical Management of Neurologic Disease

Edited by
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and
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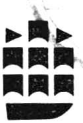

Churchill Livingstone
New York, Edinburgh and London 1980

Distributed in the United Kingdom by Churchill Livingstone, Robert Stevenson House, 1-3 Baxter's Place, Leith Walk, Edinburgh EH1 3AF and by associated companies, branches and representatives throughout the world.

First published 1980
Printed in USA

ISBN 0-443-08042-9

7 6 5 4 3 2 1

Library of Congress Cataloging in Publication Data

Main entry under title:

Current surgical management of neurologic disease.

Bibliography: p.
Includes index.
1. Nervous system—Surgery. 2. Nervous system—Diseases. I. Wilson, Charles B., 1929- II. Hoff, Julian T. [DNLM: 1. Nervous system—Surgery. WL368 C9765]
RD593.C843 617'.48 80-14580
ISBN 0-443-08042-9

Dedication

To R.W. and D.H.,
the non-professional contributors to this book.

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Preface

The publisher has taken a bold and unprecedented step by commissioning a book written for non-neurosurgeons that deals exclusively with the changing pattern of neurosurgery. When we decided to take on the responsibility for this book, we asked ourselves and our colleagues, "Would neurosurgeons be interested in a comparable book dealing with the recent advances in, say, neurology?" The answer was both affirmative and enthusiastic.

Through their reading and attendance at meetings, neurosurgeons stay abreast of contemporary neurosurgical practice—the new procedures, modifications of old procedures, and the timely demise of honored but antiquated operative approaches. Non-neurosurgeons engaged in the diagnosis and treatment of diseases that affect the nervous system may be aware of the changing attitudes of a few colleagues in neurosurgery who cooperate with them in the care of mutual patients, but as a rule the bias is provincial and a broader view is missing. We see this in our own environment.

In selecting subjects for this book, we concentrated on neurological and neurosurgical disorders for which there has developed significant change in either the method of treatment or the neurosurgeon's attitude toward the disorder. To meet our objectives, we decided that each topic should be described authoritatively and succinctly. The reader desiring an update should find sufficient material to become informed. If interest extends beyond the information provided, the reader will find leading references on the subject at the end of each section.

We would like to express our very great thanks to our colleagues who generously contributed their time and their considerable knowledge and experience of neurosurgery in authoring these chapters, and to Susan Eastwood, our editor, who urged us to undertake this book and collaborated with us in its preparation.

We are uncertain—but cautiously optimistic—about this book's reception. We hope that it will find an audience among non-neurosurgical specialists. If this proves to be the case, we will enjoy the task of preparing future editions.

Charles B. Wilson and Julian T. Hoff

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INTRODUCTION
Perioperative Management

PHILIPPA NEWFIELD, M.D.
and
JAMES E. COTTRELL, M.D.

To a greater extent than in virtually any other surgical specialty, perioperative and anesthetic management significantly affect the prognosis of the patient undergoing a neurosurgical procedure. The interaction between pharmacological and mechanical maneuvers and intracranial pathophysiology is critical in determining the results of therapeutic intervention.[14] Heightened appreciation of these relationships accruing over the past decade has fostered sophisticated developments in the practice of neurosurgery. These have been enhanced considerably by advances in four intimately related areas: neuroanesthesia, neuroradiology, monitoring, and the technology of the surgical microscope.

The chapters that follow elucidate the role of such technical and conceptual refinements in enabling neurosurgeons to perform more intricate operations with decreased mortality and improved results. To provide the reader with a framework in which to consider these chapters, we discuss the perioperative care of the neurosurgical patient, and the anesthesia and monitoring techniques that have contributed to the greater safety and success of neurosurgical procedures.

INTRACRANIAL DYNAMICS

The cranium is a rigid, bony structure containing brain and water (80 percent), blood (12 percent), and cerebrospinal fluid (CSF, 8 percent). The total cerebral blood flow (CBF), supplied by the internal carotid and basilar arterial systems, is 45 to 50 ml/100 g of brain/min, while blood flow to individual areas of brain—the regional cerebral blood flow (rCBF)—varies between 20 and 80 ml/100 g/min. Intracranial blood volume at any given time ranges from 100 to 150 ml.

Regulation of CBF is determined by metabolic, neurogenic, and myogenic (or autoregulatory) factors.[14] Cerebral arteriolar tone reflects the metabolic activity of the brain. The normal cerebral metabolic rate of oxygen consumption is 3 to 3.5 ml/100 g/min, but as hydrogen ion concentration increases with the accumulation of lactate and pyruvate from increased metabolism or decreased oxygen supply, vessels dilate and CBF increases. The elevation of arterial carbon dioxide tension ($PaCO_2$) causes similar hydrogen ion accumulation, vasodilation, and increased CBF. In addition, both sympathetic and

parasympathetic innervation of cerebral vessels contribute to the coarse and fine adjustments of CBF.

Cerebral vascular resistance varies with the muscle tone of the vessel wall in response to changes in cerebral perfusion pressure—the difference between mean arterial pressure (MAP) and intracranial pressure (ICP), calculated as MAP minus ICP. Autoregulation is the process whereby CBF remains constant, despite variations in cerebral perfusion pressure from 60 to 150 torr owing to vascular dilation or constriction. Beyond these lower and upper limits, CBF is affected by the blood pressure such that it decreases as MAP falls below 60 torr, and increases as MAP rises above 150 torr (Fig. I-1).

Anesthetic drugs affect CBF by changing cerebral vascular resistance. Indeed, CBF is the primary focus of neuroanesthetic management. Since the volume of brain, lesion, and CSF cannot be readily altered, cerebral blood volume is the only intracranial component that the anesthesiologist can control, through choice of anesthetic and manipulation of systemic pressure and $PaCO_2$.

PATHOPHYSIOLOGY

The neurological deficits associated with intracranial space-occupying lesions or vascular lesions result from invasion of, or direct pressure on, the brain parenchyma, or from hydrocephalus secondary to obstruction of CSF flow. The degree of intracranial hypertension engendered by hydrocephalus or a mass lesion determines the patient's response to further increase in intracranial volume, whether from pathological or iatrogenic causes.

Normal individuals respond to small, acute increases in intracranial volume with a rapid readjustment to normal levels of ICP through extracranial translocation of blood and CSF. With progressive expansion of intracranial volume, however, compensatory mechanisms are exhausted and the ability to restore the normal ICP is progressively impaired. As volume expands, each new increment in volume causes a more marked elevation in ICP (Fig. I-2). This hyperbolic pressure response is clinically significant because the patient's position on the volume-pressure curve is usually unknown, and the response to any increase in intracranial volume is therefore unpredictable.

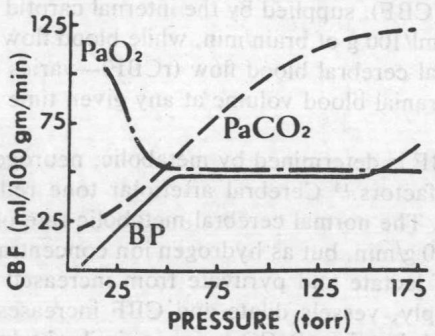

Fig I-1. Cerebral blood flow varies with changes in blood pressure, in arterial oxygen tension (PaO_2), and in arterial carbon dioxide tension ($PaCO_2$).

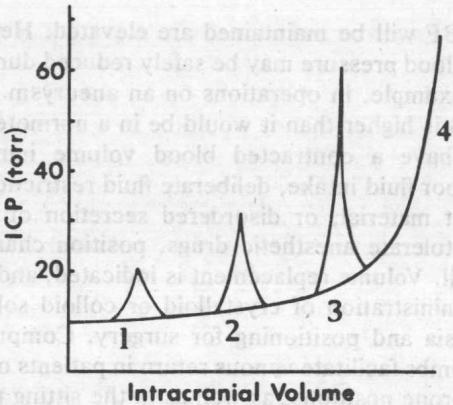

Fig 1-2. Intracranial volume and pressure within the closed cranium are related. As volume expands progressively, compliance is reduced so that each additional increment in volume causes a more marked rise in intracranial pressure (ICP). When ICP is already elevated (point 3), even a small increase in intracranial volume (as from an increase in CBF) will induce a tremendous surge in ICP.

Preoperative testing of patients with intraventricular cannulae will identify the level of compliance—the degree to which the patient is capable of compensating for increases in the intracranial contents. Compliance is decreased when intraventricular injection of 1 ml of saline causes a 2 mm Hg or greater rise in ICP; this provides evidence of the patient's position on the ascending portion of the volume-pressure curve, where small increases in volume will elicit a marked surge in pressure. When compliance testing cannot be performed, all patients are presumed to have either increased ICP or the potential for dangerous pressure elevation in response to any expansion of intracranial volume.

In addition to a sudden expansion of intracranial volume, abrupt and large increases in blood pressure will cause a breakthrough in the upper limit of autoregulation. The resultant increases of CBF in focal areas of the brain are accompanied by plasma and protein transudation from overdistended capillaries, with disruption of the blood-brain barrier and formation of cerebral edema.

Patients whose condition has developed over a long period of time and who have a large space-occupying lesion have poor cerebral compliance and little compensatory ability, as do those who have sustained a very rapid expansion of intracranial contents. These people respond poorly to any increase in CBF caused by hypertension, hypercarbia, or anesthetic drugs. Even a slight incremental increase in intracranial volume will induce an enormous rise in ICP, perhaps causing compression of vital structures or herniation.[10] Patients with smaller mass lesions will be less adversely affected by such changes.

PREOPERATIVE EVALUATION

A preoperative history of high blood pressure is of significance when evaluating the neurosurgical patient. In the hypertensive patient, autoregulation is altered so that both the upper and lower limits of cerebral perfusion

pressure at which CBF will be maintained are elevated. Hence, the level to which mean arterial blood pressure may be safely reduced during the induction of hypotension (for example, in operations on an aneurysm or arteriovenous malformation [AVM]) is higher than it would be in a normotensive patient.

Patients usually have a contracted blood volume immediately before surgery because of poor fluid intake, deliberate fluid restriction, intravenously administered contrast material, or disordered secretion of antidiuretic hormone. They do not tolerate anesthetic drugs, position changes, or induced hypotension very well. Volume replacement is indicated, and is accomplished with intravenous administration of crystalloid or colloid solution before the induction of anesthesia and positioning for surgery. Compression stockings applied to the lower limbs facilitate venous return in patients operated on in the supine, lateral, and prone positions, as well as in the sitting position.

Because of the significant incidence of postoperative nausea and vomiting, all patients receive a clear liquid diet the night before the operation. Minimal preoperative medication with oral diazepam eliminates the problems of narcotic-induced obtundation, hypoventilation, and resultant carbon dioxide retention. Patients with altered levels of consciousness are not premedicated.

MONITORING

Because of the possibility of extensive blood loss, venous air embolism, cardiac arrhythmias, blood pressure fluctuations, and large urine output during the operation, thorough monitoring is necessary to provide crucial information about the patient's progress. Monitoring includes continuous measurement and display of intraarterial blood pressure, central venous pressure, cardiac rate and rhythm by electrocardiogram (ECG), and temperature by esophageal thermistor; intermittent measurement of arterial blood gases, hematocrit, serum electrolytes and osmolarity, and urinary output; and detection of venous air embolism with the precordial ultrasonic Doppler device. These measures should be instituted before induction of anesthesia and continued into the postoperative period.

ECG monitoring is particularly important during neurosurgical procedures because alterations in heart rate and rhythm result from brain stem and cranial nerve manipulation. Direct arterial pressure measurement provides continuous information about the hemodynamic effects of the surgical manipulation of intracranial contents. Induced hypotension also is accomplished safely and efficiently with beat-to-beat monitoring of blood pressure.

Central venous catheterization is valuable for diagnosis and treatment of venous air embolism, measurement of right-sided filling pressures, and administration of fluid and drugs. The catheter position may be verified by chest x-ray, by the transduced pressure waveform, or by the p-wave configuration on the ECG.

Intraoperative determinations of arterial blood gases, hematocrit, and serum chemistries facilitate rapid assessment of the patient's respiratory and

metabolic status, and guide acute adjustments of ventilation and of intravenous infusion rate and content.

AIRWAY MANAGEMENT

Maintaining an adequate airway is a pivotal aspect of the care of neurosurgical patients. Airway obstruction must be alleviated because it increases intrathoracic pressure, which in turn exacerbates intracranial hypertension. Furthermore, in preventing adequate oxygenation, airway obstruction causes secondary injury to the brain, compounding the biomechanical trauma of operation. Accumulation of CO_2 in the presence of obstruction dilates cerebral vessels and increases CBF, cerebral blood volume, and ICP. Both improved oxygenation and adequate ventilation are achieved by airway control.

The decision to intubate a comatose patient depends on the depth of coma. In the event of airway obstruction, loss of reflexes, or inability to cough up secretions, the patient should be intubated to ensure oxygenation and prevent aspiration. If the patient exhibits purposeful movement in response to a painful stimulus, the protective reflexes are probably intact. Close observation, rather than immediate intubation, may be indicated—bearing in mind that the patient's neurological status is dynamic and may deteriorate precipitously at any time.

There are potential difficulties with airway management and intubation in patients with cervical spine fractures, cervical spine fixation, head and neck trauma, and impaired mandibular motion. The necessity for avoiding flexion and extension of the neck may make airway maintenance impossible after an anesthetic-induced loss of muscle tone. For this reason intubation is accomplished with the patient awake, using topical anesthesia of the larynx and trachea.

Patients who cough and strain on the endotracheal tube require sedation to avoid ICP elevation and regional cerebral perfusion deficits. In ventilated patients, poor synchronization with the ventilator and inadequate muscle relaxation may also raise ICP.

An endotracheal tube with a pressure-controlled cuff can remain in place for 2 to 3 weeks without causing tracheal stenosis from pressure necrosis and resultant scarring of tracheal cartilage. Tracheotomy is indicated when intubation is impossible because of extensive airway trauma, when prolonged intubation is necessary, when improved tracheal access is required for pulmonary toilet, and when the patient is transferred to hospital areas where the staff is not prepared to care for patients with endotracheal tubes.

Damage to the ninth, tenth, and twelfth cranial nerves during operations in the area of the posterior fossa interferes with airway integrity, depresses reflexes, and promotes aspiration. The possibility of operative trauma requires cautious assessment of airway and swallowing after extubation and during the first feeding. Complete inability to cough or swallow effectively may necessi-

tate tracheotomy, but patients usually compensate for unilateral paralysis of the vagus nerve over a period ranging from days to months.

POSITIONING

Successful surgical intervention is predicated on obtaining adequate exposure of, while causing minimal trauma to, the central nervous system (CNS). To achieve these objectives during the neurosurgical procedure, patients are placed in one of several operative positions, often for long periods of time. Common to all these positions is elevation of the head to decrease bleeding and ICP.

The sitting position provides excellent access to the brain, improves venous and CSF drainage, and facilitates hemostasis. Among patients operated in this position, however, venous air embolism, depressed cardiac output, hypotension, and cerebral ischemia, caused by a reduction in cerebral perfusion pressure, occur with increased frequency. To avoid hypotension with position change, patients must have an adequate circulating blood volume, and their legs must be wrapped from toes to groin. The sitting position is achieved gradually, with constant monitoring of blood pressure and heart rate.

The patient is actually semireclining, resting back 60° from the horizontal, the knees flexed at heart level, and the head supported in a three-point pin head-holder. Care is taken to avoid excessive neck flexion because it obstructs venous drainage and causes elevation of ICP. The sitting position usually is not used for older patients who have cardiopulmonary disorders because postural hypotension is positively correlated with poor physical condition.

The lateral decubitus position affords wide exposure of the posterior fossa for tumor resection and cranial nerve microvascular decompression, and of the temporal region for operating on aneurysms and tumors. Inserting a soft roll of towelling through the dependent axilla prevents injury to the brachial plexus. Jugular compression, and the attendant increase in venous bleeding and ICP, is avoided by providing adequate distance between the neck and the dependent shoulder.

The prone position offers access to midline and lateral structures in the brain. Despite support of the thorax and abdomen with parallel bolsters from shoulder to thigh, ventilation must be controlled because spontaneous respiration may be inadequate. Although air embolism is more frequently a complication of operations performed with the patient in the upright position, it has occurred with patients in the lateral or prone position.

TEMPERATURE CONTROL

During aneurysm clipping, hypothermia is induced to 31°C because this decreases the cerebral oxygen requirement 7 to 15 percent for every degree to which temperature is lowered below 37°C. Consequently, the brain can safely

tolerate circulatory arrest with no perfusion for 4 minutes at 38°C, for 8 minutes at 30°C, for 16 minutes at 22°C, and for more than 30 minutes at 16°C.

Hypothermia has few deleterious systemic effects if the body temperature remains above 28°C. Progressive impairment of myocardial function and increasing myocardial irritability occur below 28°C, requiring extracorporeal circulatory support. Function of liver, kidneys, and endocrine glands is decreased during hypothermia, but returns to normal within 24 hours after rewarming. Delayed metabolism and excretion of depressant drugs and muscle relaxants also occur secondary to cooling of liver and kidneys.

Shivering, common during cooling and rewarming, causes a 50 to 200 percent increase in oxygen consumption, and must be treated to avoid anaerobic metabolism, progressive metabolic acidosis, and cardiac depression. Significant metabolic alterations of the acid-base balance only occur, however, when shivering is accompanied by inadequate tissue perfusion and prolonged circulatory arrest. Even without shivering, decreasing the temperature causes an increase in the solubility and combining power of CO_2, and a decrease in buffer capacity. Maintenance of levels of ventilation appropriate to the normothermic patient during cooling will result in progressive respiratory alkalosis. The increased pH and lower temperature combine to cause a leftward shift of the oxygen-hemoglobin dissociation curve. Although the greater affinity of oxygen for hemoglobin is counteracted in part by the increased solubility of oxygen, ventilation should be reduced during cooling to maintain a more normal pH.

BRAIN VOLUME

Hyperosmotic agents, including mannitol and glycerol, diminish brain volume by increasing plasma osmolarity, which moves water from brain to intravascular space and facilitates surgical manipulation. Although mannitol is effective and may be used for patients who have renal disease, it does cause transient hypervolemia, with a rise in CBF and ICP. Mannitol (0.50 to 1.0 g/kg) is given 15 minutes before skin incision. Its action begins within 10 to 15 minutes and remains effective for 2 hours.

Furosemide also decreases brain volume because it effects a systemic diuresis, decreases CSF production, reduces cerebral chloride transport, and causes capacitance relaxation. Since furosemide, while providing adequate operating conditions, produces neither the ICP elevation nor the osmolar and electrolyte changes that occur with mannitol, it is used as a primary (1 mg/kg) or adjuvant (0.15 to 0.30 mg/kg) diuretic.[9]

Steroids reduce cerebral edema by affecting CSF dynamics, decreasing membrane permeability, stabilizing the blood-brain barrier, and improving intracranial compliance. Dexamethasone and methylprednisolone are most effective in treating patients who have perifocal edema surrounding a tumor or abscess, and in ameliorating the associated neurological deficit. In conventional doses, steroids do not influence survival after head trauma, but large initial and maintenance doses may facilitate recovery. Clinical improvement and in-