Manual of

Neuroanesthesia

Sperry

Stirt

Stone

Manual of
Neuroanesthesia

Richard J. Sperry, M.D.

Assistant Professor of Anesthesiology
and Surgery (Neurosurgery)
Department of Anesthesiology
University of Utah Medical Center
Salt Lake City, Utah

Joseph A. Stirt, M.D.

Associate Professor of Anesthesiology
Department of Anesthesiology
University of Virginia Health Sciences Center
Charlottesville, Virginia

David J. Stone, M.D.

Assistant Professor of Anesthesiology
and Neurological Surgery
Department of Anesthesiology
University of Virginia Health Sciences Center
Charlottesville, Virginia

B.C. Decker Inc. • Toronto • Philadelphia

Publisher

B.C. Decker Inc
3228 South Service Road
Burlington, Ontario L7N 3H8

B.C. Decker Inc
320 Walnut Street
Suite 400
Philadelphia, Pennsylvania 19106

Sales and Distribution

United States and Puerto Rico
The C.V. Mosby Company
11830 Westline Industrial Drive
Saint Louis, Missouri 63146

Canada
McAinsh & Co. Ltd.
2760 Old Leslie Street
Willowdale, Ontario M2K 2X5

Australia
McGraw-Hill Book Company Australia Pty. Ltd.
4 Barcoo Street
Roseville East 2069
New South Wales, Australia

Brazil
Editora McGraw-Hill do Brasil, Ltda.
rua Tabapua, 1.105, Itaim-Bibi
Sao Paulo, S.P. Brasil

Colombia
Interamericana/McGraw-Hill de Colombia, S.A.
Apartado Aereo 81078
Bogota, D.E. Colombia

Europe
McGraw-Hill Book Company GmbH
Lademannbogen 136
D-2000 Hamburg 63
West Germany

France
MEDSI/McGraw-Hill
6, avenue Daniel Lesueur
75007 Paris, France

Hong Kong and China
McGraw-Hill Book Company
Suite 618, Ocean Centre
5 Canton Road
Tsimshatsui, Kowloon
Hong Kong

India
Tata McGraw-Hill Publishing Company, Ltd.
12/4 Asaf Ali Road, 3rd Floor
New Delhi 110002, India

Indonesia
P.O. Box 122/JAT
Jakarta, 1300 Indonesia

Italy
McGraw-Hill Libri Italia, s.r.l.
Piazza Emilia, 5
I-20129 Milano MI
Italy

Japan
Igaku-Shoin Ltd.
Tokyo International P.O. Box 5063
1-28-36 Hongo, Bunkyo-ku,
Tokyo 113, Japan

Korea
C.P.O. Box 10583
Seoul, Korea

Malaysia
No. 8 Jalan SS 7/6B
Kelana Jaya
47301 Petaling Jaya
Selangor, Malaysia

Mexico
Interamericana/McGraw-Hill de Mexico, S.A. de C.V.
Cedro 512, Colonia Atlampa
(Apartado Postal 26370)
06450 Mexico, D.F., Mexico

New Zealand
McGraw-Hill Book Co. New Zealand Ltd.
5 Joval Place, Wiri
Manukau City, New Zealand

Panama
Editorial McGraw-Hill Latinoamericana, S.A.
Apartado Postal 2036
Zona Libre de Colon
Colon, Republica de Panama

Portugal
Editora McGraw-Hill de Portugal, Ltda.
Rua Rosa Damasceno 11A-B
1900 Lisboa, Portugal

South Africa
Libriger Book Distributors
Warehouse Number 8
"Die Ou Looiery"
Tannery Road
Hamilton, Bloemfontein 9300

Southeast Asia
McGraw-Hill Book Co.
348 Jalan Boon Lay
Jurong, Singapore 2261

Spain
McGraw-Hill/Interamericana de Espana, S.A.
Manuel Ferrero, 13
28020 Madrid, Spain

Taiwan
P.O. Box 87-601
Taipei, Taiwan

Thailand
632/5 Phaholyothin Road
Sapan Kwai
Bangkok 10400
Thailand

United Kingdom, Middle East and Africa
McGraw-Hill Book Company (U.K.) Ltd.
Shoppenhangers Road
Maidenhead, Berkshire
SL6 2QL England

Venezuela
McGraw-Hill/Interamericana, C.A.
2da. calle Bello Monte
(entre avenida Casanova y Sabana Grande)
Apartado Aereo 50785
Caracas 1050, Venezuela

Manual of Neuroanesthesia

ISBN 1-55664-105-2

Library of Congress catalog card number: 89-50931

10 9 8 7 6 5 4 3 2 1

Contributors

David L. Bogdonoff, M.D.
Assistant Professor of Anesthesiology
University of Virginia Health Sciences Center
Charlottesville, Virginia

Donald L. Boos, M.D.
Research Fellow in Neuroanesthesia
University of Virginia Health Sciences Center
Charlottesville, Virginia

William C. Broaddus, M.D., Ph.D.
Research Fellow in Neurological Surgery
University of Virginia Health Sciences Center
Charlottesville, Virginia

C. Morgan Cooper, M.D.
Research Fellow in Neuroanesthesia
University of Virginia Health Sciences Center
Charlottesville, Virginia

Johnny B. Delashaw, M.D.
Research Fellow in Neurological Surgery
University of Virginia Health Sciences Center
Charlottesville, Virginia

Adrian J. Hobbs, M.B.
Assistant Professor of Anesthesiology
University of Virginia Health Sciences Center
Charlottesville, Virginia

Bruce A. Mannes, M.D.
Chief Resident in Anesthesiology
University of Utah Medical Center
Salt Lake City, Utah

Scott L. Mears, M.D.
Fellow in Neuroanesthesia
University of Utah Medical Center
Salt Lake City, Utah

Hilary A. Noble, B.M., F.F.A.R.C.S.
Assistant Professor of Anesthesiology
University of Virginia Health Sciences Center
Charlottesville, Virginia

T. S. Park, M.D.
Professor of Neurological Surgery and Pediatrics
University of Virginia Health Sciences Center
Charlottesville, Virginia

Richard J. Sperry, M.D.
Assistant Professor of Anesthesiology and Surgery (Neurosurgery)
University of Utah Medical Center
Salt Lake City, Utah

Cary S. Sternick, M.D.
Chairman, Department of Neurology
Tomball Regional Hospital
Houston, Texas

Joseph A. Stirt, M.D.
Associate Professor of Anesthesiology
University of Virginia Health Sciences Center
Charlottesville, Virginia

David J. Stone, M.D.
Assistant Professor of Anesthesiology and Neurological Surgery
University of Virginia Health Sciences Center
Charlottesville, Virginia

Foreword

In the last three decades there have been enormous strides in neurosurgery. Many lesions which heretofore were beyond the realm of technical feasibility can now be boldly approached, and other challenging but somewhat more routine problems can be treated with considerably greater safety. A large component of the progress in technical neurosurgery is a direct result of the advances in neuroanesthesia.

This new *Manual of Neuroanesthesia* presents succinctly the state-of-the-art in practical terms that can be readily applied in the operating room. Written by three leaders in the field, this work promises to become an essential text for practitioners and teachers of neuroanesthesia, and for their students.

The emphasis of this text is clearly upon the basis of the clinical practice of neuroanesthesia. The authors are superb clinicians who have written a beautifully organized and interesting book. With the tremendous growth in neurosciences, it is refreshing to have a volume that relates the progress at the basic level to the actual giving of anesthetics to neurosurgical patients.

Neal F. Kassell, M.D.
John A. Jane, M.D., Ph.D.

Preface

Why another neuroanesthesia book? While attempting to design and implement teaching programs in neuroanesthesia at our institutions, we quickly realized that none of the available texts met our needs. Although there are excellent books available in this field, we felt they did not emphasize the practical aspects of current neuroanesthesia practice in the United States. Too many times, a chapter could burst with facts yet leave the student with the uncomfortable question of "How do I actually do the anesthetic for a given neurosurgical procedure?"

The practice of neuroanesthesia depends heavily on the basic sciences of anatomy, physiology, and pharmacology, and we have not intended to downgrade the importance of these fields which lend a rationale to what we do and make it more enjoyable. However, we did not wish to shortchange clinical practice, leaving a brief piece on "how to do it" buried at the end of a long chapter. This book should be a useful guide for a resident undertaking a one-month rotation in neuroanesthesia, as well as a resource for the practicing CRNA or anesthesiologist who does not anesthetize neurosurgical patients with great frequency. The recovery phase and intensive care period have been covered in detail as well. Although not originally intended as such, the book should form a solid basis for review for both written and oral board exams in anesthesiology. Once the basic text has been absorbed, use of the references should allow those with a greater academic inclination to dig deeper into some of the current research in the field.

We believe that anesthesia for neurosurgery is especially gratifying, as our function interacts with that of the surgeon in a way not present in many other fields of anesthesia. Neuroanesthesia provides the practitioner with a real clinical test with challenging patients who require the physician's skill in *all* phases of anesthesia. We hope this book makes the practice of neuroanesthesia more rational, more accessible, and more fun.

Richard J. Sperry
Joseph A. Stirt
David J. Stone

Contents

William C. Broaddus, M.D., Ph.D., Johnny B. Delashaw, M.D., and T. S. Park, M.D.

Anatomic, Physiologic, and Neurosurgical Considerations in Neuroanesthesia 1

The central nervous system (CNS) is unique in its vulnerability to both direct and indirect consequences of trauma, hypoxia, ischemia, and other pathophysiologic phenomena. Furthermore, it is unlike body systems in its relatively poor ability to recover from injury. For these reasons, several aspects of normal CNS anatomy and physiology play critical roles in the choices and effects of anesthesia. The wide range of pathologic conditions requiring neurosurgical treatment also frequently have mechanical or physiologic consequences that affect the choice of or response to anesthetic techniques.

Clearly, each patient represents a complex interplay of physiology, pathology, and the consequences of various interventions. The task of the surgical team is to develop a set of priorities by which to make decisions regarding the management of the individual patient. The purpose of this chapter is thus fourfold. First, selected aspects of CNS anatomy and physiology are briefly reviewed for their significance with respect to neuroanesthetic techniques. Second, a brief overview of mechanisms of CNS injury due to neurosurgical pathology is provided as a framework for discussion of surgical and medical treatment strategies undertaken by the neurosurgeon and neuroanesthesiologist. Third, a neurosurgical perspective of the significant issues in anesthetic management of patients is provided. Finally, we emphasize the importance of communication between neuroanesthesiologists and neurosurgeons prior to and during a procedure requiring anesthesia.

Anatomic and Physiologic Considerations

Central Nervous System Compartments

The craniospinal compartment is effectively a single contiguous space bounded by the calvaria and spinal canal. Although it is well suited for protecting the soft vulnerable neural tissue of the brain and spinal cord, its lack of distensibility is a major reason for the low compliance of the CNS. Brain parenchyma, cerebrospinal fluid, and intravascular fluid are the primary components contained within the intradural compartment. A pathologic increase in one of these compartments (e.g., tumor growth, hydrocephalus,

or intraparenchymal bleeding) can result in compression and shift of surrounding structures. In addition, the increase in volume produces a rapid rise in intracranial pressure (ICP) and may result in deleterious consequences. The craniospinal space is anatomically separated into three intradural compartments: the supratentorial compartment, the posterior fossa, and the spinal intradural compartment (Fig. 1–1).

The Supratentorial Compartment

The supratentorial compartment comprises the largest component of the craniospinal space and is demarcated by the calvaria and the tentorium cerebelli. It is partially divided in the midline by the falx cerebri. The latter structure also divides the major contents of this compartment, the cerebral hemispheres. The importance of these structures in subserving higher aspects of function of the various sensory modalities, organized complex motor behaviors, and personality and intelligence is well known. Paradoxically, however, only relatively large lesions in this compartment are usually life-

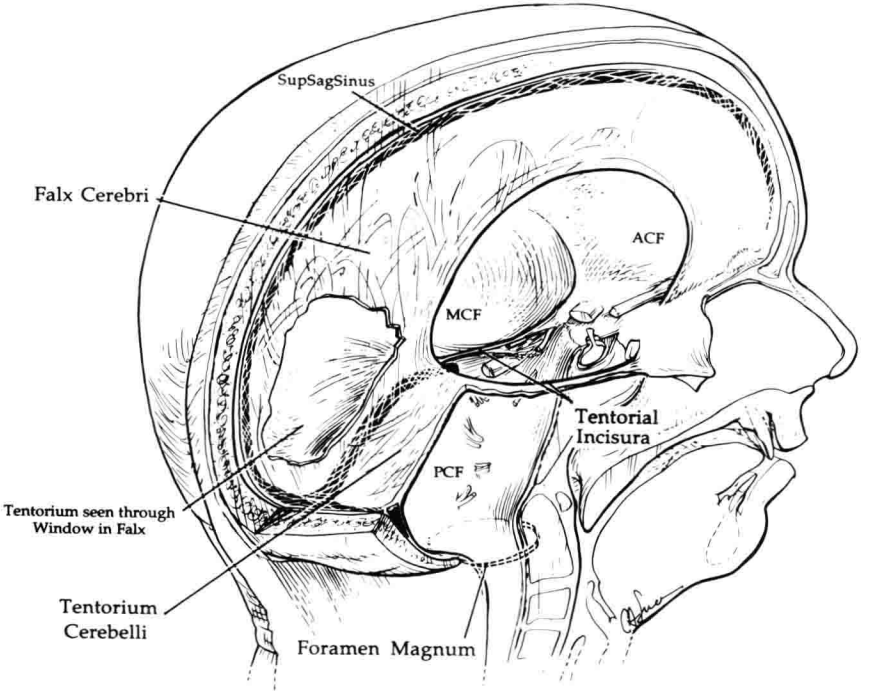


Figure 1–1 Illustration of the compartments of the craniospinal space in a drawing of the hemisected head. The dural structures, including falx cerebri and tentorium cerebelli, are left partially intact to illustrate how the cranial compartment is subdivided. Note that the supratentorial compartment is divided by the falx cerebri and is contributed to by the anterior cranial fossa (ACF) and middle cranial fossa (MCF). The supratentorial compartment communicates with the infratentorial compartment or posterior cranial fossa (PCF) via the opening of the tentorial incisura. The posterior cranial fossa in turn communicates with the intraspinal compartment by way of the foramen magnum.

threatening. Smaller lesions, which in the posterior fossa might be devastating to vital functions, may have only subtle consequences in the supratentorial compartment.

Both the inferior edge of the falx cerebri and the incisura of the tentorium are important sites for secondary injury to the CNS. When differential pressure develops as a result of a frontal lobe mass, subfalcial herniation can develop. This can lead to direct injury to the region of the cingulate gyrus, as well as cerebral ischemia by impingement of the anterior cerebral artery (Fig. 1-1 and Fig. 1-2)

Herniation through the tentorial incisura is of even greater clinical importance because of the other structures that are at risk of injury. In particular, these include brain stem structures which are important for maintenance of consciousness, the motor and sensory pathways within the cerebral peduncles, the oculomotor nerves (CN III), as well as the proximal portions of the posterior cerebral arteries (see Figs. 1-2 and 1-3).

Lateral masses cause downward and medial pressure on the temporal lobe resulting in herniation of the uncus (on the medial surface of the temporal lobe) over the edge of the tentorium. This results in ischemic injury to the cortex of the uncus and may result in pressure on the oculomotor nerve, posterior cerebral arteries, and cerebral peduncle (Fig. 1-3). The ominous consequences of these phenomena are well known:

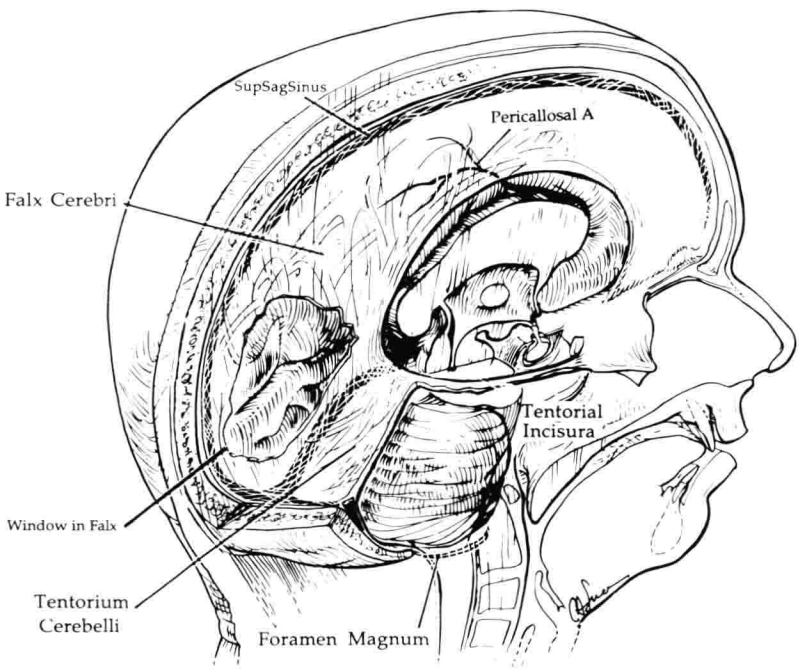
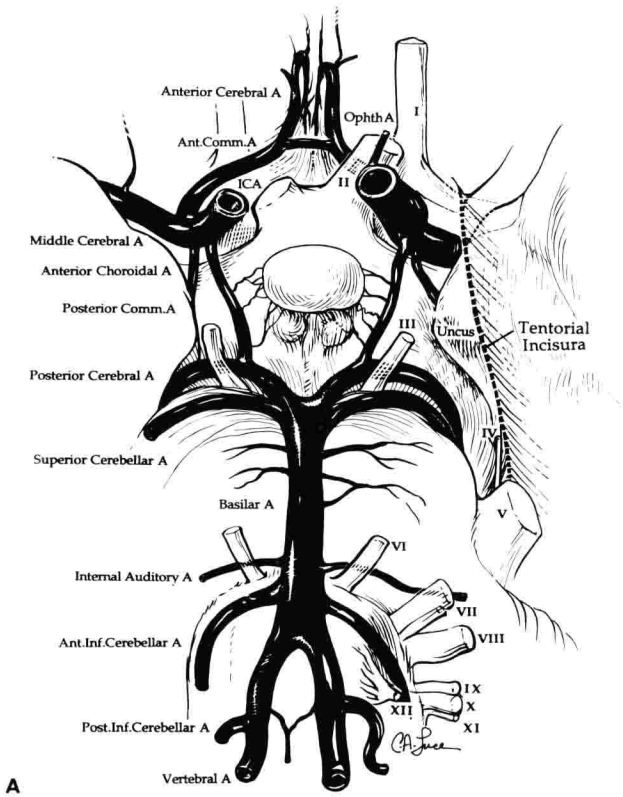
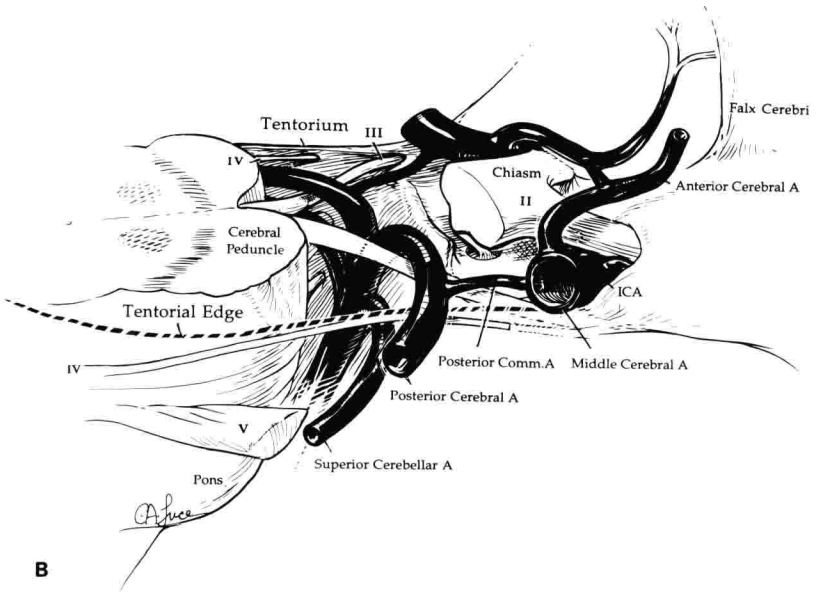


Figure 1-2 A view of the hemisected head similar to that in Figure 1-1 is shown with the cerebellum, caudal brain stem, and left cerebral hemisphere in place. This shows the important anatomic relationships of brain and vascular structures to the falx cerebri, tentorial incisura, and foramen magnum. Note that the pericallosal artery is an extension of the anterior cerebral artery and passes above the inferior edge of the falx as it courses posteriorly. Also note the potential for impaction of the cervicomedullary junction and cerebellar tonsils at the foramen magnum in the event of a downward shift of the brain.


Anterior Cerebral A
Ant.Comm.A
Ophth A
I
ICA
II
Middle Cerebral A
Anterior Choroidal A
Posterior Comm.A
III
Uncus
Tentorial Incisura
Posterior Cerebral A
IV
Superior Cerebellar A
Basilar A
V
VI
Internal Auditory A
VII
VIII
Ant.Inf.Cerebellar A
IX
X
XII
XI
Post.Inf.Cerebellar A
Vertebral A
A


Tentorium
III
IV
Falx Cerebri
Chiasm
Anterior Cerebral A
II
Cerebral Peduncle
ICA
Tentorial Edge
IV
Posterior Comm.A
Middle Cerebral A
Posterior Cerebral A
V
Superior Cerebellar A
Pons
B

ipsilateral large irregular poorly reactive pupil, contralateral hemiplegia and posturing, and sometimes ipsilateral medial occipital lobe infarction. If the lateral to medial pressure is more diffuse, the contralateral cerebral peduncle may be pressed against the medial edge of the contralateral portion of the tentorial incisura, resulting in Kernohan's notch phenomenon (ipsilateral hemiplegia). In fact, bilateral neurologic signs with predominance of one side over the other are more common than unilateral findings, and suggest a combination of the above described pathophysiologic phenomena.

Paramidline supratentorial masses can also result in herniation through the tentorial hiatus, and they may produce displacement of structures through the foramen magnum.

The Posterior Fossa

The posterior fossa is the portion of the cranial cavity below the tentorium, and it is notable for the vital structures contained within it. The compartment contains the cerebellum and caudal brain stem. The caudal brain stem lies ventrally along the clivus connecting with the rostral brain stem through the tentorial incisura and the cervical spinal cord through the foramen magnum. Relatively small mass lesions of the cerebellum and even smaller mass lesions adjacent to or within the caudal brain stem can have devastating consequences after presenting with only subtle symptoms. Injury to the reticular activating system frequently can result in a decreased level of consciousness, while injury to centers for respiratory and hemodynamic control can cause apnea and marked hemodynamic instability. In addition, lesions along the brain stem can result in impairment of function of the cranial nerves and ascending and descending pathways.

The tendency of large supratentorial masses to cause shifts of the brain through the tentorial incisura have been discussed. Central herniation caused by paramidline supratentorial masses, however, may also cause severe injuries by two other mechanisms. Differential movement of caudal brain stem structures caused by the relative immobility of the basilar artery as compared to the overlying pons may result in secondary pontine hemorrhages due to rupture of small perforating vessels. Second, the "pressure cone" phenomenon that occurs in herniation at the foramen magnum may result in impaction of the cerebellar tonsils on the cervicomedullary junction, frequently with fatal results (see Fig. 1-2 and Fig. 1-3)

The premonitory signs of impending herniation, particularly in the posterior fossa, may be relatively subtle and require a high index of suspicion for diagnosis. Computed tomography (CT) or magnetic resonance imaging (MRI) may be helpful in demonstrating asymmetric effacement of the ambient cisterns due to uncal herniation,

---

Figure 1-3 Vascular and neural anatomic relationships of the base of the brain are depicted from (*A*) a basal view and (*B*) a posterolateral view. Components of the anterior and posterior arterial circulations of the brain are shown. The anastomosis of these two circulations by way of the posterior communicating arteries bilaterally comprises the circle of Willis. Cranial nerves are labeled by Roman numerals, and the important relationship of the tentorial incisura and uncus of the temporal lobe are demonstrated. Abbreviations used are as follows: ICA = internal carotid artery; A = artery; Ophth A = ophthalmic artery; Ant. Comm. A = anterior communicating artery; Posterior Comm. A = posterior communicating artery; Ant. Inf. Cerebellar A = anterior inferior cerebellar artery; Post. Inf. Cerebellar A = posterior inferior cerebellar artery.

effacement of the fourth ventricle due to a cerebellar mass, or presence of cerebellar tonsils adjacent to the spinal cord in the foramen magnum. In the context of neuroanesthesiology, these signs should be used as indications for interventions that lower ICP, and protection against large increases in ICP during manipulations such as intubation or endotracheal suctioning.

The Spinal Intradural Compartment

The spinal intradural compartment represents a significant portion of the total volume of the craniospinal compartment. The spinal cord extends only down to the first lumbar segment, the lumbar and sacral subarachnoid space (L2 to S2) represents a cerebrospinal fluid (CSF) reservoir through which the lumbar and sacral nerve roots pass. Surrounding the dura within the spinal canal is the epidural venous plexus, which represents a displaceable extradural vascular volume. A slight distensibility of the spinal dura and the compressibility of the epidural venous plexus thus appear to underlie the ability of the spinal subarachnoid space to accommodate CSF displaced by the rapid expansion of an intracranial mass. This greater compliance of the spinal intradural compartment would also appear to be a significant factor in the pathogenesis of transforaminal herniation. With this in mind, it is obvious how further decompression of the spinal intradural compartment by lumbar puncture could precipitate a transforaminal herniation, with disastrous consequences. Conversely, it should be noted that a head CT scan frequently allows the clinician to proceed safely with lumbar puncture, even in the presence of a known intracranial mass, by demonstrating the lack of significant mass effect.

Structures of the Central Nervous System Parenchyma

The Cerebral Hemispheres

The cerebral hemispheres are responsible for cognitive function, personality, and meaningful sensory and motor function. Discrete neurologic deficits can result from direct destructive lesions. Although patients with lesions of the frontal lobes may present with subtle neurologic deficits, patients with lesions of the cerebral cortex adjacent to the Rolandic fissure, internal capsule, or basal ganglia may exhibit profound deficits in motor or sensory function. Furthermore, lesions that involve the speech areas in the dominant hemisphere, while not life-threatening, have the potential to render a patient unable to communicate with the outside world. Large lesions of the cerebral hemispheres have the potential for causing transtentorial or central herniation syndromes. Such patients are thus extremely vulnerable to otherwise insignificant changes in intracranial dynamics.

The Diencephalon

The diencephalon is the rostral portion of the brain stem that lies centrally within the supratentorial compartment. The diencephalon includes the thalamus, hypothalamus, and pineal gland, and encloses the third ventricle. These structures play an important role in relaying and integrating CNS function. The diencephalon is vulnerable to neoplastic involvement, ischemic injury due to impairment of blood flow in perforating arteries, and direct compression due to hemispheric mass effect.

The Cerebellum

The cerebellum is the largest structure occupying the posterior fossa and is involved in coordination of motor function. Although it is not vital for survival, midline or bilateral lesions may cause permanent disabling motor impairment. Unilateral lesions of the cerebellar hemispheres usually cause ipsilateral limb ataxia, which will partially resolve over time. Cerebellar lesions and associated edema can interfere with the vital functions of the brain stem by direct compression.

The Caudal Brain Stem

The caudal brain stem resides in the posterior fossa and is composed of the midbrain, the pons, and the medulla oblongata. This area of the brain stem serves as a conduit with significant processing functions of afferent and efferent activity between the brain and spinal cord. It serves similar functions for cranial nerves III through XII, and contains structures that are vital to maintenance of consciousness, equilibrium, and respiratory and hemodynamic control. These structures are vulnerable to injury by transtentorial, central, and transforaminal herniation, or by direct compression by posterior fossa lesions.

The Spinal Cord

The spinal cord extends from the cervicomedullary junction at the foramen magnum to the conus medullaris and cauda equina at the T12–L1 level. This structure is vulnerable to trauma, compression by intradural or extradural tumors, and occasionally vascular injury. The danger of such injuries to motor and sensory function below the segmental level of the injury is well known. In addition, certain important functions require intact innervation from specific segmental levels of the spinal cord. For instance, the ability of an individual to breathe independently requires intact phrenic nerve function as a minimum. The phrenic nerves, in turn, are formed by branches of the anterior nerve roots at the third to fifth cervical levels. For full respiratory function, thoracic spinal cord innervation of the muscles of respiration is also required. Thus, respiratory dysfunction is a concomitant of any spinal cord injury in the thoracic or cervical segments, with the degree of impairment related to the level of injury.

Another example of segmental dependence is the outflow of the sympathetic nervous system in the thoracic and lumbar nerve roots. Thus, lesions of the upper thoracic level or above will disrupt sympathetic tone. Practical consequences of such lesions include postural hypotension and bradycardia.

Intracranial Pressure

Determinants of Intracranial Pressure

The ICP can be expressed in terms of the volume of the contents of the craniospinal compartment and its compliance ($\Delta ICP = \Delta volume/compliance$). As previously discussed, the intracranial compartment has little if any compliance, while the spinal canal has a small degree of compliance related to distensibility of the spinal dura mater and surrounding epidural venous plexus. As a result, significant changes in total volume of the craniospinal contents are accompanied by large changes in ICP. In fact, significant

changes in volume of any of the components occupying the craniospinal compartment are usually accompanied by passive changes in volume of the other components so that the change in total volume is minimal. The rise in ICP due to a given pathologic process is determined by the net increase in volume after primary pathologic changes and compensatory changes in volume of the other components are balanced.

There are three components that make up the craniospinal contents (Table 1–1). These are the brain and spinal cord parenchyma, the cerebral and spinal blood volume, and the CSF. Changes in the CNS parenchyma that may result in increased ICP include tumor growth and cerebral edema. The latter may be due to ischemic injury or trauma, or may be surrounding a hematoma, tumor, or inflammatory process such as an abscess.

Changes in the brain vascular compartment can have a major effect on ICP, particularly when involving venous blood volume. This is directly affected by systemic venous pressure as well as cerebral vascular tone related to autoregulatory functions. Both of these parameters are amenable to manipulation for amelioration of intracranial hypertension and, by the same token, are potential mechanisms by which existing intracranial hypertension may be exacerbated.

The CSF volume depends on a balance between production and disposition. The fact that production of CSF is poorly responsive to changes in ICP is responsible for the development of hydrocephalus in patients who develop problems in disposition of CSF. In the normal individual, the total CSF volume is approximately 140 ml, with daily production of about 500 to 600 ml. Since the majority of this production arises from the

Table 1–1 Determinants of ICP and Therapeutic Techniques to Lower Elevated ICP

Determinant	Therapeutic Intervention	Mechanism	Duration of Effectiveness
CSF volume	Ventricular or lumbar puncture	Volume reduction	Hours
	Ventriculostomy or lumbar drain	Volume reduction	Days
	Ventricular or lumbar shunt	Volume reduction	Indefinite
Cerebral blood volume	Hyperventilation	Cerebral vasoconstriction due to decreased P_{CO_2}	Hours
	Barbiturates	Cerebral vasoconstriction	Hours to days
Volume of parenchyma	Mannitol infusion	Osmotic reduction of brain water content	Hours to days
	Corticosteroids	Reduction of peritumoral or peri-inflammatory edema	Days to weeks
	Excision of mass	Volume reduction	Indefinite
	Craniectomy	Increased craniospinal compliance	Indefinite