Third Edition

CORRELATIVE NEUROSURGERY

Edited by

RICHARD C. SCHNEIDER, EDGAR A. KAHN

ELIZABETH C. CROSBY, JAMES A. TAREN

Volume I

While maintaining its unique approach and distinctive style, the Third Edition of this classic neurosurgical work has been dramatically expanded, revised and updated. The expertise and diligence of the editors and contributors have resulted in a work that is certain to assume a prominent slot on the bookshelves of all students, clinicians and researchers in the neurosciences. Making their first appearance in this edition are authoritative chapters on such timely subjects as computed tomography, radionuclide studies, evoked potentials, chemotherapy, and central nervous system infections. Except for historical presentations, material retained from the Second Edition has been uniformly brought into line with the state of the neurosurgical art.

(continued on front of Volume II)

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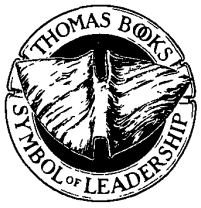

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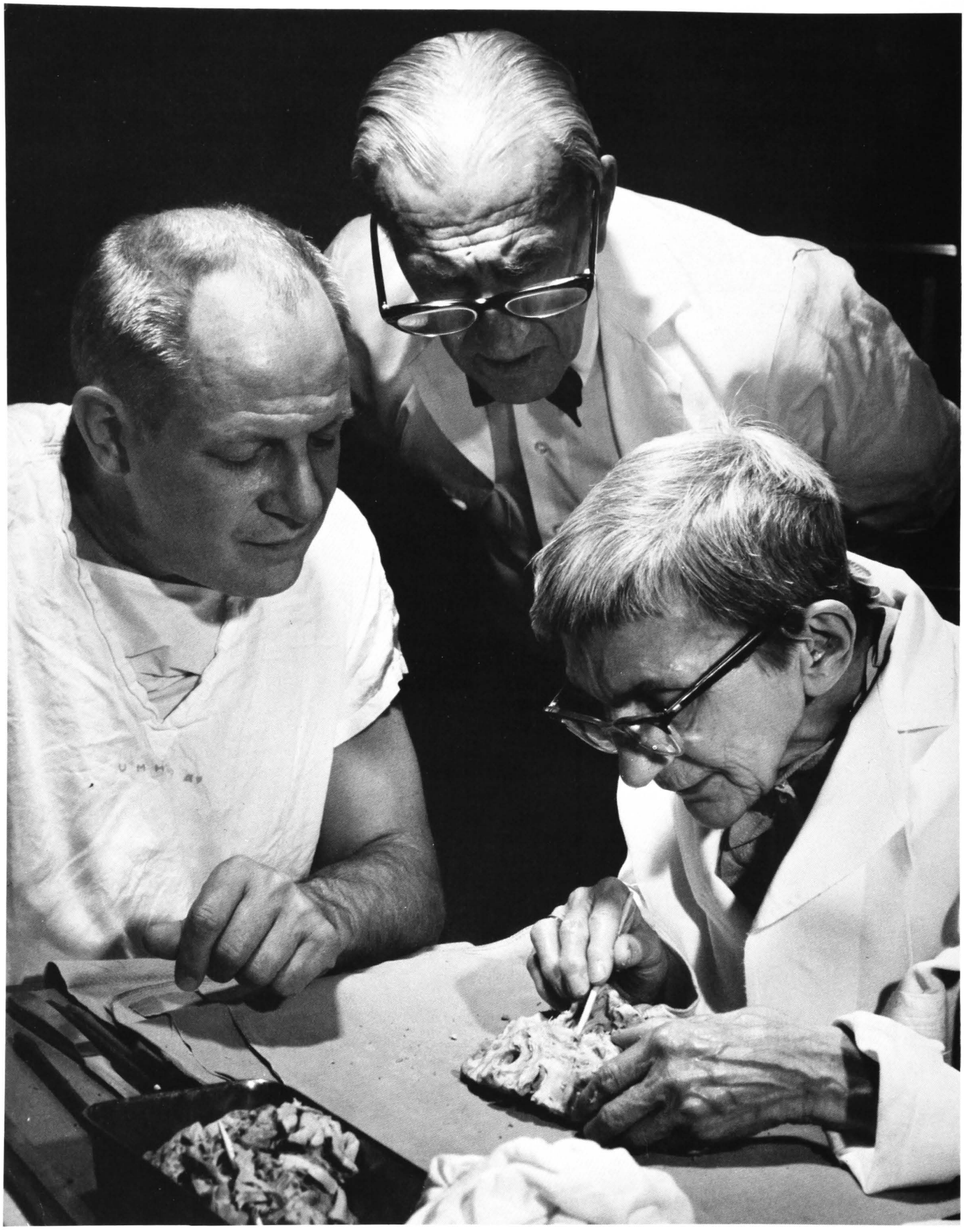
We dedicate this book to Dr. Elizabeth C. Crosby and Dr. Edgar A. Kahn: co-authors, mentors, and colleagues—without their knowledge.

Richard C. Schneider
James A. Taren



· *Richard C. Schneider* *Edgar A. Kahn* *Elizabeth C. Crosby* ·

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This Third Edition of *Correlative Neurosurgery* retains and expands upon the same format effectively devised by Dr. Edgar A. Kahn and Dr. Elizabeth C. Crosby in the previous editions, correlating Neuroanatomy and Neurosurgery. The rapid growth of knowledge in Neuroscience and allied fields has necessitated the increased number of authors who have so kindly and ably contributed to this new edition.

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FOREWORD

This book on Neurological Surgery represents a concerted effort to correlate the various disciplines which are germain to this field. The neurosurgeons who have described the various operative procedures presented here were all trained by Doctor Max M. Peet who died suddenly on March 25, 1949. Doctor Peet was a straightforward, pioneering surgeon possessed of great skill in many technical fields, and distinguished for his directness of approach to all surgical problems. The impact of Doctor Peet's teaching can be seen here in the descriptions of surgical procedures.

While this book will be of interest and value to all who are associated in any way with Neurosurgery, the young neurosurgeon who is now gaining his operative experience will find it to be of particular assistance since many technical procedures are set forth in considerable detail. In addition, there are a number of surgical tips accumulated over the years which may help to keep the beginner out of trouble.

The anatomical descriptions which add so greatly to this book have evolved from the close association of the anatomist, the clinical staff and the patients.

Anatomical diagrams are often extremely complex and difficult to interpret because they are labeled by abbreviations only. In this book, however, the diagrams are presented in extremely large size and the labels are almost invariably written out, a practice which greatly simplifies interpretation. Orientation in gross sections of the brain has been facilitated by presenting many of these in full size. In some cases, these gross sections could well be employed as actual operative guides.

The non-surgical chapters dealing with x-ray, electroencephalography, radioactive isotopes, aphasia and endocrinology have been treated by experts in their respective fields and should be of particular interest to neurosurgeons.

Essentially, this volume deals in an interesting and personal way the manner in which day-by-day neurosurgical problems are looked after in this large teaching center in Ann Arbor.

KENNETH G. MCKENZIE

PREFACE TO THE THIRD EDITION

The Third Edition of *Correlative Neurosurgery* retains the plans so successfully developed by Drs. Edgar A. Kahn and Elizabeth C. Crosby to correlate Neurosurgery with Neuroanatomy. The basic material of the previous edition has been employed and, in most instances, revised. The book has now been expanded into two volumes, to include new chapters on Computerized Axial Tomography, Radionuclide Studies of the Central Nervous System, Electroencephalography, Evoked Potentials, Vascular Disease, Radiation Therapy and Chemotherapy, Infections of the Central Nervous System, Neuroendocrinology, Craniocerebral Trauma, Cervicomedullary and Spinal Cord Trauma, Syringomyelia and other Spinal Disorders, Hydrocephalus, Surgery of the Cerebral Hemispheres and the Cerebellum in the Balancing of Abnormal Tonus and Movement, Acoustic Tumors—Combining Neurosurgery and Neuro-otology Approaches. The chapters are not an all-encompassing review of their topics but reflect the special talents and interests of their authors. Most of the chapters have been written by members of the University of Michigan Medical Center Staff or its alumni. There are others, however, to whom the Editors are also particularly grateful, including Gerard Guiot, M.D., Glenn Sheline, M.D., William Wara, M.D. and the late John M. Converse, M.D., who, although not members of the Michigan Medical family, have made significant contributions to this book. In addition, we are indebted to Professor Stephan Kubik, Anatomisches Institut, Zürich for the photographs of his excellent gross dissections of the brain.

Reviewing past experiences in surgery and evaluating successes and failures are important methods of learning. In several chapters of this book case histories have been freely used to provide a clear, personal insight for the reader interested in treating clinical neurological problems. It is obvious that the rapidly changing field of medical science continues to produce new diagnostic instruments, special equipment and techniques, and drugs that can in a short time revolutionize methods of attacking clinical problems. Some of these case reports of former years, however, illustrate important points and may also be looked upon as being of historical interest. In other instances, such as the surgical treatment of epilepsy, the number of elapsed years for which the patient has remained seizure-free is of primary importance in evaluating the success of the operation.

It has taken an effective team of many people to bring this book to its completion. Throughout the entire preparation of the revision Mrs. M. Kay Neff has been invaluable in organizing material, typing manuscripts, aiding with indices and many other details, and all of the editors wish to express their heartfelt appreciation to her. Mrs. Siân Coffman, although entering the project near its conclusion, has been most helpful in completing the Author Index and the final phase of manuscript corrections. Mrs. Irina Chernomordik has been thorough and persistent in checking references for the entire book, and without Mr. Allan Dyer's perseverence and experience the Subject Index would never have been so concisely and effectively created. To Mr. Gerald Hodge and his colleagues, Mr. Denis Lee and Mr. Lewis Stadler, of the Medical Illustration Department we are indebted for the advice and fine contributions of art work. The Photography Department under the direction of Mr. Edgar Sherman and his associate, Mr. Robert Bensinger, have been most effective in printing many of our photographs for these

volumes. The most senior editor wishes to express his deep appreciation to his wife, Madeleine T. Schneider, for her major contribution in re-reading a part of this proof and correcting the text and also for permitting him to devote the time to the publication of this work.

Support for the publication has been provided by the Begole-Brownlee Neurosurgical Fund, the E. and S. Power Fund, the Richard C. Schneider Neurosurgical Fund through its various donors, and the Neurosurgery Syringomyelia Research Fund.

The editors also wish to express their special gratitude to Mr. Scott Thomas and Mrs. Susan Meister of the Charles C Thomas Publishing Company without whose expertise and patience these two volumes could never have been printed.

Finally, if this edition of *Correlative Neurosurgery* will have piqued the curiosity of a few students, residents, or staff members in the Neurosurgical field and stimulated an interest in improved patient care, teaching, and pertinent research it will have achieved its goal.

RICHARD C. SCHNEIDER

PREFACE TO THE SECOND EDITION

The Second Edition of this book reiterates the viewpoint that in spite of the advance in mechanical diagnostic methods, neuroanatomy is still an important basis of diagnosis, operative approach and the understanding of neurological signs and symptoms.

Since the First Edition was published, brain scanning has emerged as the most effective single method in the diagnosis of brain tumors. There has been a better understanding of the hypothalamo-hypophyseal target organ system. Human growth hormone has now been used with success clinically. Though the supply of this hormone is scant at present, as was the case with cortisone at an earlier time, an era in medicine comparable to the space age may open up when human growth hormone is synthesized.

Other advances over the past twelve years include an understanding of the role played by extracranial vessels in cerebral thrombosis, though there is still no unanimity as to the surgery of these extracranial vessels. The introduction of percutaneous cordotomy by Sean Mullan is an important advance in the alleviation of pain but should not be undertaken lightly.

Stereotaxic surgery and surgery using the image intensifier can now be carried out with considerable accuracy. We have been impressed with the work of Gerard Guiot in both fields. We are grateful to him for summarizing for the first time his extensive experience with stereotaxic surgery and for detailing his remarkably accurate electrophysiological confirmation of the target area after the latter has already been grossly delineated roentgenographically.

On the dark side of the ledger, the glioblastomas, which comprise one fourth of all brain tumors, remain the *bête noire* of the neurosurgeon. Brain-tumor surgery seems to have been neglected in recent neurosurgical publications. This writer still believes, however, that brain-tumor, intracranial-aneurysm and spinal-cord surgery, where one can see the actual pathology, are still the most interesting and exciting part of neurosurgery.

Unless one has visited underprivileged countries, one has no idea of the enormity of the problems of neurosurgery, not only in terms of numbers, but also in connection with the advanced states which neurosurgical lesions can attain. The chapter on "Exotic Lesions of the Brain," by E. Latunde Odeku, presents an indication of the latter.

We wish to thank Doctors Trygve O. Gabrielsen, Associate Professor of Radiology, John W. Henderson, Chief of the Department of Ophthalmology, Samuel P. Hicks, Professor of Pathology, and Kenneth R. Magee, Professor of Neurology, for their constant help and interest in the Neurosurgical Service.

We are most grateful to Mrs. Evelyn E. Sullivan, medical illustrator, for her intelligent handling of the new figures. Our secretaries, Mrs. Dorothea Goldschmidt, Mrs. Key Neff, Mrs. Doris Reuter and Mrs. Ruth Trimm, have worked hard and understandingly in getting out this manuscript, and we are all deeply appreciative of their efforts.

EDGAR A. KAHN

PREFACE TO THE FIRST EDITION

This book has been written primarily for young neurosurgeons who have finished their formal training and are about to commence practice; consequently, no attempt to teach basic neurosurgical procedures has been made. It is hoped, however, that discussion of certain practical aspects of diagnosis and technique which have been employed in this clinic over a period of years will prove to be helpful. Undergraduate medical students have also been kept in mind. They may find in this book some correlation between what they have learned in the anatomical laboratory and what they see in the hospital.

With the exception of Doctor Converse, the contributors were associated at one time or another with Doctor Max M. Peet, or had their training at the University of Michigan. Doctor Peet was a bold and anatomically-minded surgeon whose ability to go directly to the point stemmed from an uncanny visual memory. He was deeply interested in, and often consulted with, the Department of Neuroanatomy.

About five years ago, our colleague in Neuroanatomy, Doctor Crosby, decided to leave her laboratory one morning a week to make ward rounds on our service. It was evident that clinical problems which had long puzzled us could sometimes be solved by the application of what was, certainly at first, the pure science of anatomy. As a result of our combined ward rounds one could gradually see the clinician becoming more anatomically-minded and the anatomist more clinically-conscious. This has been reflected in the teaching of both.

In Percival Bailey's *Intracranial Tumors,* an attempt was made to correlate Neurosurgery and Neuroanatomy—and this work has become a classic. Since this task was completed by one man, the problem as to what material should be incorporated was simplified. When several people with entirely different backgrounds are involved in putting material together, however, the result is, of necessity, a compromise. The pure anatomist who reads this book may think that certain details of importance have been omitted; the clinician may feel that the anatomy is treated in too much detail. The clinician must remember, however, that some of the finer signs of anatomical dysfunction which the laboratory worker demonstrates in experimental animals are either not looked for in the patient or are seen at a stage when the effects of the lesion have been masked by progression. For example, though glioblastoma multiforme commonly involves the basal ganglia, rigidity and abnormal movements are rarely seen in association with these tumors. One reason for this is that the weakness from involvement of the pyramidal tract in the internal capsule may mask the result of disturbed function of the basal ganglia so completely that the signs of the latter are absent.

It is my personal opinion that the technical side of Neurosurgery has advanced about as far as it can advance on its own. Further progress will probably come only when the knowledge of the neuroanatomist and the neurophysiologist, along with the never biological aspects of physics and chemistry, are utilized by neurosurgeons. This book is intended as a step in that direction.

Few references to the work of others will be found in this book since it has been written from the point of view of personal experience. This does not mean that originality is claimed for many of the opinions set forth. It does mean, however, that we have been influenced, both directly and indirectly, by so many others that it would be impossible to give credit to all of them.

First of all, we wish to thank the late Doctor Kenneth G. McKenzie for his constructive criticism of certain sections of the book which deal with clinical problems. His penetrating views on various phases of neurosurgery have been well known in this country for years.

We are indebted to our colleagues in the Laboratory of Neuropathology in the Neuropsychiatric Institute of the University of Michigan. Doctor Raymond W. Waggoner, the Director, and Doctor Konstantine Scharenberg, Pathologist, placed all the material of that laboratory at our disposal. Most of the photomicrographs were prepared by Doctor Jose Bebin, whose untiring efforts have contributed so much to our work.

Doctor Carl V. Weller, Chairman of the Department of Pathology at Michigan, kindly edited the section of Congenital Dural Sinus and gave us an entirely new concept of pilonidal sinus. Doctor John W. Henderson of the Department of Ophthalmology contributed the discussion on retinal and preretinal hemorrhages in Chapter 1. Doctor William C. Baum of the Urology Service summarized care of the bladder in the paraplegic patient in Chapter 21.

We are deeply grateful to Professor Karl Litzenberg of the English Department of this University who edited, from a stylistic point of view, the chapters written by Doctors Bassett and List and myself.

The photography was completed under the direction of Mr. Roland Burd. We cannot thank him or his staff enough for their cooperation and for their constant striving for perfection.

We wish also to express our very sincere appreciation for the help given in preparing the manuscript and figures for publication by the technical staff in Neuroanatomy; Mrs. Joy M. Mara, artist, Miss Shirley Mesnard, secretary, Miss Bettylou Smith, and Mr. George Smith, technicians. The drawings for the chapter on Trauma were made by Miss Mary L. Cummings, medical illustrator at the University Hospital. We wish to express our appreciation to Miss Patricia Blake, as well, for the excellent illustrations in Chapter 19.

At the University of Michigan Hospital, the Neurosurgical Service is a branch of the Department of Surgery. It is difficult to express in words our appreciation of the support which the Director, Doctor Frederick A. Coller, has continuously given us.

Lastly, we wish to thank Charles C Thomas, Publisher—and I, especially, my friend Charles C Thomas, who suggested that we write this book and who has done his utmost to present it in an attractive format.

EDGAR A. KAHN

CONTENTS

CORRELATIVE NEUROSURGERY

CHAPTER 1

EXPANDING LESIONS OF THE BRAIN AND INCREASED INTRACRANIAL PRESSURE

GLENN W. KINDT AND EDGAR A. KAHN

Expanding lesions of the brain that cause increased intracranial pressure include tumors, intracranial hemorrhages, abscesses, parasitic cysts, and rare granulomas. Cerebral edema and impaired cerebrospinal fluid circulation are often integral parts of the pathological physiology associated with these lesions. Pathologic vasodilatation is also frequently involved with acute brain swelling, particularly in trauma (Chap. 36). Expanding lesions of the brain may manifest themselves by a generalized increase in intracranial pressure, by localizing signs and symptoms, or by both.

The classic manifestations of chronic generalized increased intracranial pressure are *headache, vomiting,* and *papilledema.* Unilateral or bilateral *sixth nerve palsies* can occur and are often associated with obstruction of the pathways of cerebrospinal fluid circulation. Acutely increasing intracranial pressure may initially produce only the headache and vomiting, but invariably, as the intracranial pressure increases in intensity, a *depression of the conscious* level and finally *coma* occur. The common unilateral supratentorial expansion may produce signs of a *tentorial pressure cone,* which include a unilateral dilated pupil, fixed to light, and positive Babinski signs. *Changes in vital signs* become apparent with markedly increased intracranial pressure or brain stem distortion. Loss of pupillary reflexes and respiratory arrest occur ultimately if the intracranial pressure cannot be controlled. An acute increase in intracranial pressure may be superimposed upon a chronic increase when a hemorrhage into a tumor occurs.

Expanding intracranial lesions may also produce localizing signs and symptoms, such as jacksonian seizures, psychomotor and uncinate attacks, focal paralyses or sensory impairments, hemianopsias, and cranial nerve palsies. Other evidence of cerebral dysfunction includes slowing of mental processes, increasing dullness and apathy, carelessness in personal appearance, disturbed vision, hallucinations, and generalized convulsions. *This chapter is mainly concerned with the signs and symptoms of generalized increased intracranial pressure.*

Headache

The headache associated with an expanding intracranial lesion is usually bursting in type. It is often most severe upon awakening in the morning. The location of the headache should be noted but is often not related to the site of the lesion. However, posterior fossa tumors most frequently produce headache in the occipital or suboccipital region. Headache in children is a much more significant symptom than it is in adults. There should be a high suspicion of brain tumor in a child with headaches persisting for three weeks. The headache of a patient with a brain tumor is often aggravated by straining at stool or coughing.

The headaches from an intracranial expansion are frequently not localizing because of the mechanics of pain production. Pain is mainly produced by tension on cerebral veins and dural sinuses, meningeal vessels, and large intracranial arteries. Thus, a lesion in one part of the brain can produce pain referred to a distant part. However, invasion of the dura or large vessels by a neoplastic or inflammatory process can produce pain precisely at the site of the lesion.

Vomiting

Precipitous vomiting occurring with little or no nausea is the most typical kind seen with expanding intracranial lesions. Projectile vomiting is uncommon but is significant when present. Brain stem distortion produces vomiting more quickly than does generalized increased intracranial pressure. Posterior fossa lesions in the region of the area postrema can produce intractable vomiting.

Some patients state that their headaches are temporarily relieved following vomiting. Such relief

may be due to reduction of intracranial pressure by the hyperventilation associated with this action.[46] Patients hospitalized with intracranial lesions are frequently dehydrated from vomiting episodes and poor oral intake. The dehydration may have temporarily controlled their intracranial pressure. Intravenous fluid replacement preoperatively in these patients is hazardous because a rapid increase in intracranial pressure can occur with subsequent decompensation.

Papilledema*

The most significant and reliable sign of chronic increased intracranial pressure is papilledema. Not everyone realizes that the pressure on the central vein of the retina that results in papilledema (if one accepts the mechanical theory of papilledema) occurs within the orbit and not intracranially. The vein actually leaves the optic nerve within 8 to 14 mm of the point where the optic nerve enters the sclera. When the vein leaves the optic nerve it traverses the vaginal sheath of the optic nerve obliquely for several millimeters before perforating it. The vein then runs backwards through the orbit and passes intracranially through the superior orbital fissure to enter the cavernous sinus.

Papilledema results when the increased intracranial pressure is transmitted by the cerebrospinal fluid by way of the vaginal sheath of the optic nerve (a continuation of the intracranial dura and arachnoid) to compress the central vein of the retina. The point is, however, that the compression of the vein occurs roughly only a centimeter from the back of the globe and not intracranially.

Galbraith and Sullivan[21] reported on seven cases in which papilledema was threatening vision and there was no indication for an intracranial procedure. They approached the optic nerve in the orbit and exposed it from the nasal side of the globe. An incision was made in the dural sheath using the operating microscope. The subarachnoid space was entered with ease because the dura and arachnoid were adherent. A 3 by 5 mm window was excised from the sheath and the escape of cerebrospinal fluid was readily visible. The results were striking in five of the seven cases. These cases certainly add to the understanding of the mechanism of papilledema.

Since there is variability in the course of the central retinal vein across the sheath of the optic nerve, the degree of exposure of the vein to increased pressure varies. Where the course is prolonged, papilledema may appear earlier; where it is direct, papilledema may be delayed. This anatomical variation may likewise explain differences in the degree of papilledema between the two optic nerves.

One might wonder why papilledema is not ordinarily associated with cavernous sinus thrombosis where there is certainly obstruction to the venous return from the retina as the vein enters the cavernous sinus. There is, however, an anatomical explanation in that the central vein of the retina, after leaving the nerve and traversing its vaginal sheath, drains not only into the cavernous sinus but by collateral flow also into the pterygoid plexus.

The first funduscopic sign to appear in the development of papilledema is fullness of the retinal veins. A spontaneous retinal venous pulsation (SRVP) is estimated to be present in from 46 to 90 percent of normal individuals. If SRVP disappears, papilledema is further suggested provided the pulsation is not restored by light pressure on the eyeball.

Observation over the past twenty-five years on the phenomenon of SRVP[33] has led us to certain conclusions we believe to be of practical importance:

1. SRVP is almost never seen in papilledema. We have, however, seen two cases in which it was present.
2. We found in a study that if SRVP is present with the patient relaxed and in the horizontal position, the pressure when measured in the ventricle or lumbar sac, in the majority of cases, will ordinarily not be higher than 14 mm of Hg (192 mm of water).[33] In 1969, Walsh et al[80] carried out a similar study. They found that the mean pressure for obliterating the pulsation was about 200 ± 25 mm H_20 cerebrospinal fluid pressure. They concluded that "the presence of a venous pulse indicates no significant elevation of intracranial pressure."
3. SRVP may be present with many benign tumors, chronic subdural hematomas in the older age group, and malignant tumors of the brain, which destroy brain substance rather than increasing intracranial pressure.
4. If papilledema is present in one eye and the fundus is normal in the other eye with SRVP, the intracranial pressure will almost certainly be normal, and the pathological process will

*This material relative to papilledema is in accordance with Dr. J. W. Henderson, Professor and former Chairman, Department of Ophthalmology University of Michigan Medical Center.

be within the orbit on the side of the papilledema.

As a rule, papilledema does not appear for at least forty-eight hours after increased intracranial pressure has developed. Subhyaloid hemorrhages, however, may be seen shortly after rupture of an aneurysm or brain laceration secondary to trauma where there has been a sudden, acute increase in intracranial pressure. We have actually observed these hemorrhages developing while a patient was in the Emergency Room shortly after rupture of an intracranial aneurysm. In this particular case, both retinas with their attached optic nerves, their vaginal sheaths, and the chiasm were removed at autopsy as a single specimen by Dr. J. Reimer Wolter[83] and were studied by him in serial section.

The mechanism by which subhyaloid hemorrhages occur has long been the cause of disagreement. Since such hemorrhages are usually found in the presence of intracranial subarachnoid bleeding, it has been assumed that the hemorrhage is directed forward along the subarachnoid spaces of the optic nerves and is forced further forward to become preretinal. However, it is most unlikely that blood within the vaginal sheaths of the optic nerves can gain access to the interior of the eye. Walsh[79] felt that a sudden rise in intracranial pressure accounts for subhyaloid hemorrhages by obstruction of all the venous channels from the eye and the orbit. A subhyaloid hemorrhage can be recognized by its globular shape and tendency to obscure underlying retinal details. In addition, the blood that lies between the retina and the vitreous humor tends to settle gravitationally, and the lower portion of the hemorrhage will appear more dense. Subhyaloid hemorrhages without papilledema are sometimes seen in chronic subdural hematomas in infants.

In the case previously mentioned, Dr. Wolter concluded that there is no connection between the subhyaloid space of the retina and the vaginal sheaths of the optic nerves. He concluded that the preretinal and the optic nerve sheath hemorrhages are locally independent occurrences and that one can exist without the other. The absence of blood around the intracranial parts of the optic nerves, as well as around the chiasm, was final evidence in Dr. Wolter's opinion that optic nerve sheath hemorrhages are not a direct continuation of subarachnoid intracranial bleeding. Increased venous pressure due to acute backflow difficulties seems to be the best explanation of these independent hemorrhages.

Once papilledema has occurred in full form, a few weeks are required after intracranial pressure has returned to normal for it to resolve. The classic appearance of papilledema includes dilated retinal veins; absence of SRVP; elevation of the disc with blurred disc margins; hemorrhages on the surface of the disc, which may extend to the surface of the retina; and disappearance of the lamina cribrosa. Persistent papilledema can lead to a secondary optic atrophy and visual loss. The optic disc then appears gray with proliferation of glial elements over the disc.

It is not always easy to distinguish papilledema from other conditions that give an appearance that is in some ways similar. These conditions include hyperopia, high central retinal artery branching, drusen, myelinated nerve fibers, and optic neuritis. With hyperopia the disc is elevated and the disc margins are blurred. However, other signs of papilledema are absent, and spontaneous venous pulsations may be present. The condition known as high central branching of the entering retinal artery can be associated with elevated glial supporting tissue to produce both nasal elevation and blurring. Embryologically, the retinal artery is a branch of the hyaloid artery, and its origin from this artery is variable. Drusen are hyaline bodies in the optic nerve and cause the disc to appear irregular on its surface and along its margins. Myelinated nerve fibers cause a white, distorted, and feathery disc margin, which may extend beyond the disc on the retina. With optic neuritis the patients complain of rapid visual loss or visual field loss, which may be profound. Hemorrhages on the disc are common, and the appearance on funduscopy may be difficult to distinguish from papilledema.

Paralysis of the Sixth Cranial Nerve

Another common sign of a general increase in intracranial pressure is a unilateral or bilateral paralysis of the sixth cranial nerve. Cushing[12] demonstrated in 1910 that an isolated paralysis of the sixth cranial nerve, in the presence of increased intracranial pressure, is of no localizing significance. He also described how this nerve, in its long course after leaving the pons, is often notched by a branch of the basilar artery as the latter becomes stretched beneath the sixth nerve because of increasing brain volume. Sixth nerve palsies are especially common with increased intracranial pressure due to posterior fossa tumors. If bilateral sixth nerve palsies develop in the absence of increased in-

tracranial pressure, it can be assumed that there is a neoplasm arising from the clivus or an aneurysm of the basilar artery.

Depression of Conscious Level

Patients with expanding lesions of the brain will eventually demonstrate a depressed conscious level and coma. This depression can be due to localized pressure on the reticular activating system of the brain stem, generalized increased intracranial pressure, or a combination of these. Other conditions such as cerebral hypoxia or ischemia, hypoglycemia, toxic states, and convulsive disorders often cloud the picture and make the determination of the causes of coma one of the greatest challenges for a physician.

A reticular formation that is located in the central brain stem might be considered as the center for consciousness. It extends from the lower medulla to the thalamus with indistinct boundaries and incorporates both small and large neurons. Relatively small intrinsic or extrinsic lesions involving the brain stem can produce coma with little or no elevation of intracranial pressure and without damage to the cerebral hemispheres. This central brain stem region is particularly sensitive to petechial hemorrhage or edema from blunt head trauma, perhaps because of the shear forces present in the region of the incisura or the foramen magnum. Compression of the brain stem from the lateral aspect due to uncal herniation resulting in coma is the classic mode of decompensation from any intra- or extracerebral mass involving one cerebral hemisphere.

Generalized increased intracranial pressure can also produce coma without significant shift of the brain structures. Examples of this include communicating hydrocephalus, subarachnoid hemorrhage, meningitis, and perhaps diffuse brain edema. It is oversimplification, but one might consider this type of increasing intracranial pressure as being similar to progressive brain ischemia or hypoxia from any cause. As intracranial pressure rises, the cerebral perfusion pressure (arterial blood pressure minus intracranial pressure) falls and, at the extreme, intracranial pressure and blood pressure are equal, and cerebral blood flow ceases. Examples of this condition in deeply comatose patients are verified angiographically by the nonfill phenomenon.[52] A similar progression of intracranial events occurs with gradual lowering of blood pressure until cardiac arrest ceases all cerebral blood flow. In the case of systemic hypotension, the cerebral perfusion pressure falls with a relatively constant and low intracranial pressure. Another similar sequence of events can occur independent of both blood pressure and intracranial pressure with progressive cerebral hypoxia as might occur when breathing 100 percent nitrogen. All of these are examples of cerebral decompensation due ultimately to lack of oxygen at the cellular level.

The critical level of increased intracranial pressure at which coma or decompensation occurs is difficult to quantify. Patients with pseudotumor or hydrocephalus may be awake at pressures exceeding 50 mm Hg. Other patients with intracranial masses and cerebral edema can become unconscious at pressures of less than 20 mm Hg. If the blood pressure or arterial pO_2 is low, unconsciousness occurs at an even lower intracranial pressure.

Tentorial Pressure Cone

The tentorial pressure cone is the common sequel to many expanding intracranial lesions because most of these lesions compress one or the other cerebral hemisphere. Figure 1-1 demonstrates the result of such a mass at autopsy. As the lesion increases in volume, there is a shift of cerebral tissue to accomodate the mass. The midline structures, such as the upper brain stem, are shifted across the midline away from the lesion. Ipsilateral to the lesion, the brain is also pushed toward the posterior fossa, causing the uncus and hippocampal gyrus of the temporal lobe to herniate between the free edge of the tentorium and the midbrain. The uncal herniation causes pressure on the third nerve and frequently on the posterior cerebral artery against the tentorium. The upper brain stem is shifted away from the side of the lesion compressing the contralateral cerebral peduncle against the somewhat sharp opposite tentorial notch.

The anatomic relationships and the symptom complex of the tentorial pressure cone were summarized from the literature and well described by Sir Geoffrey Jefferson[30] in 1938. The first sign of an impending development of a tentorial pressure cone is often a change in alertness or behavior. The general condition is one of drowsiness, but agitation may also be present intermittently. As the reticular activating system is compressed further, stupor and coma occur. The upper brain stem compression may produce an increase in tone with bilateral positive Babinski signs.

The most consistent and important sign of uncal herniation is ipsilateral third nerve dysfunction.

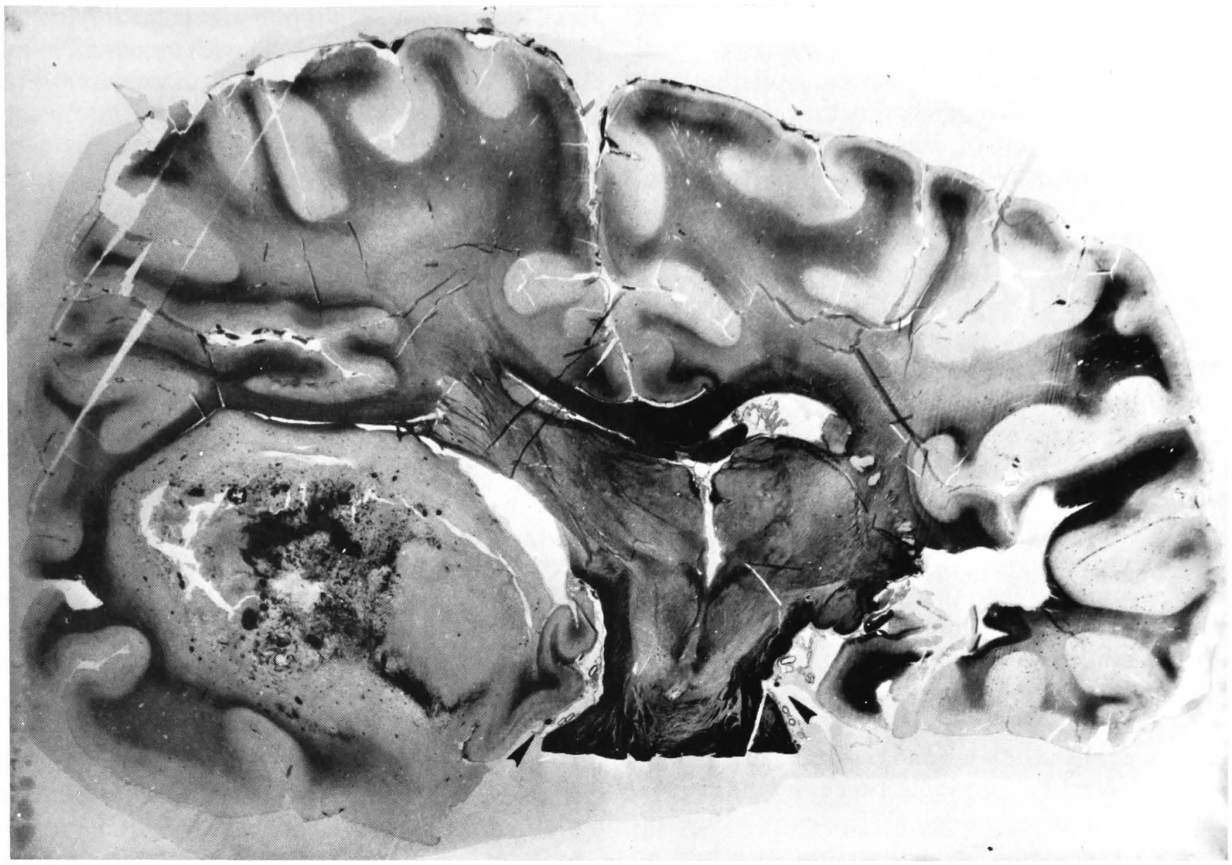

Figure 1-1. Weigert section demonstrating the mechanism of the temporal lobe pressure cone. The patient was a twenty-year-old man with papilledema who was believed to have a posterior fossa tumor. A suboccipital craniectomy was performed, but the failure to find a dilated ventricle by cannulization led to closure of the wound without opening the dura. The following day at 4:00 AM the temperature was 101° rectally, pulse 70 and respiration 22. (The slow pulse in a patient this ill suggests an acute increase in intracranial pressure at this time.) At 8:00 AM the temperature had risen to 104.4°, pulse 130 and respiration 30. The patient died suddenly, this incomplete record shows, and the intern aptly stated, "Died, probably a respiratory death." It can well be seen that this tumor, which was a glioblastoma, had infiltrated the temporal lobe and pushed the hippocampal gyrus (and the uncus) against the cerebral peduncle. The trochlear nerve (arrow) on the same side had been forced downward compared to the position of that of the opposite side (arrow). Though the free edge of the tentorium is not seen, the hippocampal gyrus must have been forced between it and the midbrain, to depress the fourth nerve so markedly. The depression of the trochlear nerve in itself is not of significance, since this nerve has a long and free intracranial course and thus can tolerate considerable displacement without being blocked physiologically. The oculomotor nerve, however, which cannot be seen in this section, since it emerges from the anterior surface of the midbrain, has different anatomical relations. Sunderland and Bradley[74] have shown in their excellent dissections that in the tentorial pressure cone, the herniating uncus forces the posterior cerebral artery down upon the oculomotor nerve, angulating it as it crosses the "tentorial gap" to enter the cavernous sinus. It is the superior portion of this nerve that is first pressed upon, and since Sunderland and Hughes[73] have shown that the pupilloconstrictor fibers are concentrated here, the dilatation of the pupil in the absence of other signs of paralysis of the oculomotor nerve can be explained. The temporal lobe pressure cone, with the resulting decerebrate rigidity and dilated pupil, is frequently seen following trauma, and its delayed development is a cause for immediate action (*see* Chapter 36).

The pupils are initially small or normal in size and constrict briskly, although this constriction may not be apparent with small pupils. The pupil ipsilateral to the lesion then becomes larger, and the light response becomes sluggish. Further progression leads to dilatation of this pupil and no light response. With slow progression there are sometimes signs of complete third nerve palsy with lid lag and the eye turned outward. More often, when progressive deterioration occurs, the contralateral pupil also dilates and the eyes remain in a midposition. Unless therapy is instituted quickly at this point, irreversible brain damage occurs (Chap. 36).

Other eye signs also occur during the developing tentorial pressure cone. The oculocephalic (doll's eyes) and oculovestibular (caloric response) reflexes are affected early by the partial third nerve palsy on the side ipsilateral to the lesion. The oculocephalic reflex is positive in the normal eye until decompensation occurs, and then there is no response to head motion. The oculovestibular reflex becomes sluggish as symptoms progress and disappears with further brain stem compression.

The tentorial pressure cone will sometimes produce hemiplegia ipsilateral to the lesion as the contralateral cerebral peduncle becomes compressed against the tentorial edge (Kernohan's notch).[34] Contralateral hemiplegia due to an ipsilateral hemispheric mass is, of course, most common with the tentorial pressure cone. Compression of the posterior cerebral artery against the tentorium can cause thrombosis of this vessel. Infarction in the distribution of this artery adds to the contralateral hemiparesis and hemianopia, which may already be present from the primary lesion. Late in the development of the tentorial pressure cone, respirations become irregular, the pulse slows, and the blood pressure rises. Ultimately, respiratory arrest occurs (Chap. 36).

Vital Sign Changes

Respirations

The respiratory patterns often seen with expanding lesions in various parts of the brain are well described by Plum and Posner.[57] Cheyne-Stokes respirations are periods of apnea followed by hyperpnea in a quite smooth crescendo-decrescendo pattern. This pattern suggests bilateral involvement deep in the cerebral hemispheres or upper brain stem. Cheyne-Stokes respirations are seen as decompensation occurs from masses within the cerebral fossae such as those which produce the tentorial pressure cone. Central neurogenic hyperventilation is a rapid, deep, and regular respiratory pattern. It occurs occasionally with lesions affecting the midbrain and pons and is most common after acute head injury. A most alarming respiratory pattern is ataxic breathing. This consists of a completely irregular pattern of occasional deep but mostly shallow breaths with a generalized slow rate. This type of breathing precedes apnea and permanent respiratory arrest. Ataxic breathing is due to pressure on the lower brain stem in the posterior fossa. It is also seen just prior to final decompensation due to increased intracranial pressure from any cause. In some instances the patient may be still quite alert and have ataxic breathing if the lesion is quite localized so as to compress the respiratory center in the dorsomedial part of the medulla. A common situation producing ataxic breathing and respiratory arrest is the foramen magnum pressure cone. In this condition, pressure from above causes the cerebellar tonsils to herniate through the foramen magnum and compress the medula.

Blood Pressure and Pulse

A rise in systemic blood pressure with an increase in intracranial pressure was demonstrated in dogs by Naunyn and Schreiber[54] in 1881. Cushing[10] refined these observations and noted that when intracranial pressure was raised artificially to the blood pressure level, the blood pressure increased to a new level. With further increases in intracranial pressure the blood pressure continued to increase in a stepwise manner until decompensation. This cerebral hypertensive response has become known as the Cushing response or Cushing reflex.

An elevated blood pressure and a slow pulse are signs that should be carefully watched for in a patient suspected of having an expanding intracranial lesion. The occurrence of these signs could have a significance equal to development of a dilated pupil. There could be imminent cerebral decompensation calling for immediate intervention. However, the relationship between blood pressure and pulse changes and the level of intracranial pressure is not direct, and other factors must be considered. First, intracranial pressure can sometimes be elevated to very high levels without blood pressure and pulse changes.[5] Also, elevations of blood pressure can occur independent of intracranial pressure.

Many conditions can be responsible for an acute arterial pressure elevation in a critically ill patient.

A hypertensive response can be elicited by (a) increased intracranial pressure;[10] (b) increased cerebral venous pressure;[50] (c) cerebral ischemia;[49] (d) cerebral hypoxia;[49] (e) hypercarbia;[65] (f) localized pressure at several points on the dorsal aspect of the pons and medulla;[27] (g) distortion of the brain stem;[76] (h) spinal cord compression.[1] The carotid and aortic pressure receptors and chemoreceptors influence this hypertensive response but are not essential for its function. The hypertensive response to increased intracranial pressure is mediated via the sympathetic nervous system. Therefore, it is affected by other stimuli that influence this system, such as pain or generalized hypothermia. Figure 1-2 demonstrates the anatomical paths involved in the cerebral hypertensive response.

The significance of an acute hypertensive episode in an individual patient may thus be difficult to evaluate (Fig. 1-2). If increased intracranial pressure is already known to be present, the hypertensive response is likely due to a combination of the above-mentioned factors. Intubation and hyperventilation should be considered early to correct any blood gas abnormalities. Proper diagnosis and treatment of the intracranial pressure and its causes can then be instituted.

THERAPY

The proper therapeutic regimen for a patient with an expanding intracranial lesion and/or increased intracranial pressure depends on the etiology of the problem. Besides location and excision of a mass lesion, if present, the physician must be concerned with related problems such as cerebral edema. Therapy has changed with time as more has been learned about the pathophysiology of increased intracranial pressure and cerebral edema. The following are some general and specific items, not necessarily in order of priority, which today seem appropriate to consider for the patient with increased intracranial pressure:

1. Remove mass
2. Monitor intracranial pressure
3. Drain cerebrospinal fluid
4. Arterial pO_2 control
5. Arterial pCO_2 control
6. Fluid management and use of dehydrating agents
7. Steroids
8. Temperature control
9. Arterial pressure control
10. Venous pressure control
11. Anesthetics and barbiturates
12. Surgical decompression

Remove Mass

In certain patients with a rapidly expanding intracranial mass, such as a postoperative hemorrhage or an epidural hematoma, the first priority must be given to diagnosing and removing the mass as quickly as possible. CT scanning is of the greatest importance here. While intracranial bleeding exists, little benefit is derived from anything other than a direct attack on the source of the bleeding. However, most intracranial bleeding stops soon after it begins as intracranial pressure approaches arterial pressure and clotting occurs.[44] The outlook for the patient then depends, as with other intracranial masses, on the ability of the brain to make adjustments to the intrusion of the mass. Cerebral edema, pathologic cerebral vasodilatation, and obstruction of cerebrospinal fluid circulation then begin to set the pace for further intracranial pressure increases. Operative excision of the mass is still the definitive therapy, but other methods of controlling intracranial pressure can be considered in the interim prior to the operation, as well as postoperatively.

Monitor Intracranial Pressure

Intracranial pressure must be continuously monitored if it is to be properly controlled. Some patients could be expected to benefit more from monitoring than others. Of course, time should not be taken to insert a pressure monitor if there is a requirement for more immediate therapy. Those patients who may benefit from intracranial pressure monitoring include those with closed head injuries, subarachnoid hemorrhage, coma of unknown etiology, hydrocephalus, meningitis, brain swelling from Reye's syndrome and other encephalopathies, as well as certain postcraniotomy patients.

Many methods are available for monitoring intracranial pressure. Ventricular puncture with ventriculostomy pressure recordings is the method of choice if obstruction of ventricular fluid circulation is thought to be part of the problem. Ventricular recordings were used by Lundberg[47] in trauma patients, and important observations of intracranial pressure waves were made. He noted that temporary rises of intracranial pressure to high levels of several minutes duration (plateau waves) occurred

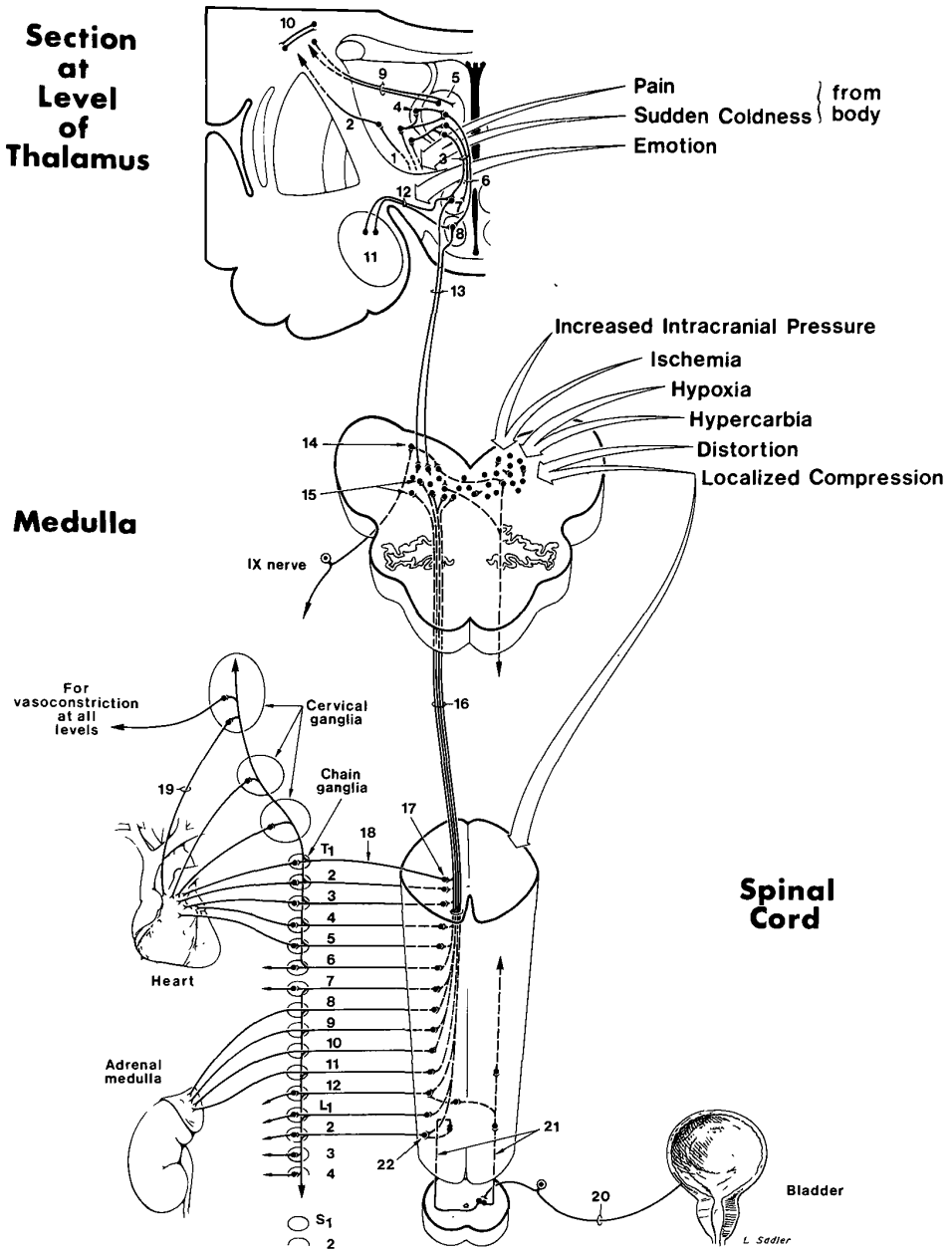

Figure 1-2. Schematic drawing of some of the anatomy involved in an acute hypertensive response from the central nervous system. The hypertensive response is mediated via the sympathetic nervous system but can be initiated from several sources and stimuli. Some of the stimuli include: emotion from cerebrum; pain and sudden coldness from the body; increased intracranial pressure; ischemia, hypoxia, and hypercarbia of the brain stem; distortion and localized compression of both brain stem and spinal cord; carotid sinus reflex via the ninth nerve (also aortic reflex); stimuli from the bladder may progress upward and cause acute pain but also can initiate an acute sympathetic response directly, independent of the brain stem, as is occasionally observed in quadriplegic patients.

The related anatomic structures noted numerically in the diagrams are: 1, lateral spinolthalamic tract; 2, sensory radiations to somesthetic cortex; 3, thalamopreoptic fibers; 4, intralaminar nucleus with relay to dorsomedial nucleus; 5, dorsomedial thalamic nucleus; 6, periventricular thalamohypothalamic fibers; 7, medial preoptic area; 8, anterior hypothalamic area; 9, anterior thalamic radiations to and from cerebral cortex; 10, association fibers between somesthetic and frontal cortex. Frontal cortex projections activate the amygdala through a 2 neuron arc; 11, amygdala; 12, amygdalopreoptic and amygdalohypothalamic tracts; 13, hypothalamotegmental tract; 14, nucleus of fascilicus solitarius; 15, medial reticular gray; 16, tegmental spinal tract; 17, intermediolateral column; 18, preganglionic fibers; 19, postganglionic fibers; 20, sensory neuron for visceral pain; 21, secondary ascending visceral tracts; 22, relay via intercalated neurons to preganglionic neurons for sympathetic discharge at all levels. Courtesy of E. C. Crosby.

spontaneously. A patient exhibiting these waves should be treated promptly to prevent fatal cerebral decompensation. A big advantage of ventricular pressure monitoring is that ventricular fluid can be aspirated as one method of controlling the intracranial pressure. Disadvantages are the difficulty of performing a ventricular puncture, as well as the potential hazards of the procedure in the presence of brain swelling with small and possibly shifted ventricles.

Cerebrospinal fluid pressure can be satisfactorily recorded via a subarachnoid catheter inserted through a lumbar puncture needle.[63] Fluid can then also be aspirated when desired. This method of monitoring is appropriate in some cases of subarachnoid hemorrhage, meningitis, and communicating hydrocephalus. Lumbar drainage is hazardous if an intracranial mass and/or brain swelling is the origin of the increased intracranial pressure.

Perhaps the simplest method of monitoring intracranial pressure is subdural monitoring through a twist drill hole in the skull. We have used this method since 1970 with no apparent complications.[24] The method is demonstrated in Figure 1-3. A 1 cm scalp incision is made under local anesthesia at the hairline. A hand twist drill hole 4.22 mm in diameter is made through the skull, and the dura is perforated beneath this hole. A sterile three-way stopcock, or the standard plastic tip of an intravenous tubing that normally fits into an intravenous needle hub, fits firmly into the cranial opening. A standard fluid manometer is placed in the line to check the subdural pressure directly and to calibrate the strain gauge and recorder. If the signal becomes damped, a fraction of a milliliter of saline can be injected intracranially to clear the line. Usually several hours or even days pass without the necessity of irrigation. If it is thought necessary, a ventricular puncture for drainage can be made through this twist drill hole or one on the contralateral side (Fig. 1-3).

Other methods of monitoring intracranial pressure have been used, such as placing a strain gauge intracranially and recording through wires or remote sensors. A tiny, totally implantable system that could monitor pressure accurately for years would be ideal. Presently some disadvantages with these systems have included a more extensive operation to implant the device, the expense involved, a drift of the baseline with time, and difficulty verifying the zero baseline. Also, ventricular fluid cannot be aspirated.

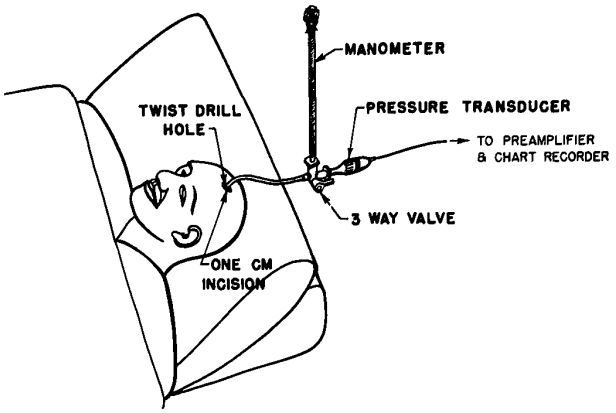

Figure 1-3. Schematic drawing of simplified intracranial pressure monitoring system. Under local anesthesia, a 4.22 mm twist drill hole (No. 9 drill) is made through the skull 3 cm from the midline at the hairline after making a 1 cm scalp incision. The dura is perforated and a 3-way stopcock or plastic tip of intravenous tubing is plugged firmly into the cranial vault. A standard manometer can be placed in the line to measure the intracranial pressure directly and calibrate the strain gauge and recorder. The stopcock at the base of the manometer is then turned to obtain direct intracranial pressure recordings.

The main advantage of continuous intracranial pressure monitoring is the early warning effect. If the intracranial pressure is rising gradually or pressure waves are occurring, therapy can be instituted promptly to reverse the process. Massive pressure increases and structural shifts such as tentorial pressure cone development, which can be damaging in themselves, are avoided. When monitoring patients in which cerebral edema is thought to be present, our goal has been to maintain intracranial pressure below 20 mm Hg.

Drain Cerebrospinal Fluid

The drainage of cerebrospinal fluid can at times be lifesaving and may be helpful in many other instances with increased intracranial pressure. A patient with hydrocephalus, who is already apparently decompensated with dilated pupils and respiratory arrest, can sometimes be returned to normal function in a few minutes after ventricular drainage. A shunting procedure or direct treatment of the cause of hydrocephalus can be done later.

Many other patients can be helped by drainage but in a less dramatic manner. The neurologic deficit of some patients who have had a subarachnoid hemorrhage is improved by the drainage of cerebrospinal fluid.[72] Obstruction of the circulation or poor absorption of cerebrospinal fluid probably contributes to deterioration from many conditions.

Patients having their intracranial pressures monitored, particularly trauma patients, will have at least a temporary reduction in pressure with ventricular fluid aspiration. The least help from ventricular drainage can be expected from those patients with generalized brain swelling and small ventricles.

Arterial pO_2 Control

In the presence of an expanding intracranial mass, hypoxia probably plays a predominant role in the production of both temporary and permanent brain damage. Brain tissue at the margin of the mass may be hypoxic from ischemia due to localized tissue pressure, as well as generalized increased intracranial pressure. This hypoxic brain is likely to become edematous,[56] causing further increase in tissue pressure unless some type of therapy is instituted. Any lowering of systemic arterial pO_2 could cause necrosis and further edema of the brain tissue at the margin of the lesion. As the process enlarges, a generalized increase in intracranial pressure occurs.

Besides the local effect, hypoxia has a tendency to increase intracranial pressure by its effect on normal brain. When the pO_2 falls below a critical level, even without the usual associated rise in pCO_2, cerebral vasodilatation occurs and cerebral blood flow increases.[35] This vasodilatation increases intracranial blood volume and could increase intracranial pressure markedly if there is already diminished compliance because of the presence of a mass.

It is not easy to state what the ideal level of pO_2 should be for a patient with increased intracranial pressure. Hyperbaric oxygen would seem to be a beneficial adjunct with brain swelling and, indeed, experimental studies have shown this.[71] However, with or without a hyperbaric chamber, one must be concerned when raising the pO_2 with the ever-present danger of pulmonary oxygen toxicity. Although 100 percent oxygen can be tolerated for short periods, for prolonged ventilation the inhaled oxygen concentration should be kept below 40 percent. The main emphasis should be placed on the prevention of any periods of hypoxia rather than elevating the pO_2 above normal.

Arterial pCO_2 Control

Changes in arterial pCO_2 have a profound effect on cerebral hemodynamics. Cerebral blood flow will more than double in normal brain when arterial pCO_2 changes from 25 mm Hg to 60 mm Hg.[61] This increase in blood flow is produced through cerebral vasodilatation, which also increases cerebral blood volume. If the intracranial compliance is already reduced because of an intracranial mass or other lesion, this elevation of pCO_2 could result in a disastrous increase in intracranial pressure and cerebral decompensation.[36] Figure 1-4 demonstrates the effect of changes in arterial pCO_2 on intracranial pressure. If the baseline intracranial pressure is low, elevating the pCO_2 has little effect on intracranial pressure. However, if the baseline intracranial pressure is higher, i.e. 30 mm Hg, a dramatic increase in intracranial pressure occurs with an elevated pCO_2 (Fig. 1-4).

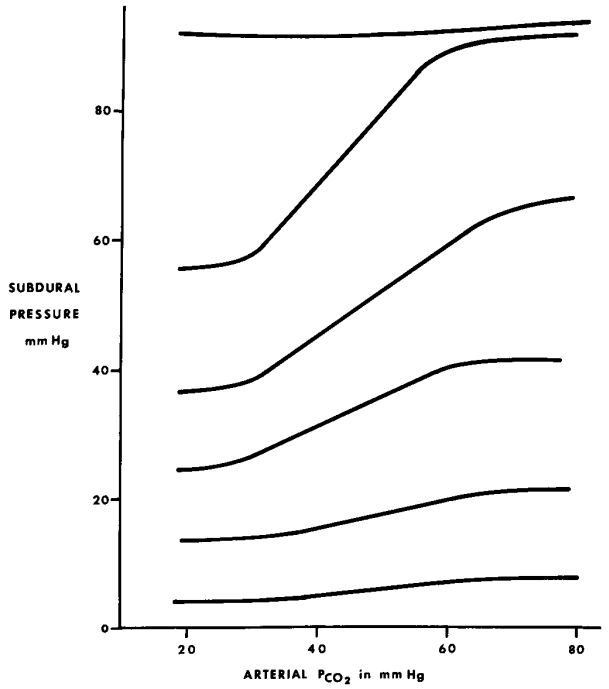

Figure 1-4. The effect of variations in arterial pCO_2 on intracranial pressure at different baselines of acutely increased intracranial pressure in the monkey. The higher the baseline intracranial pressure, the greater the effect of variations in pCO_2 until decompensation when there is no longer a response to pCO_2 as shown by the top line. From G. Kindt and H. Gosch, Arterial pCO_2 effect at various levels of intracranial pressure. In M. Brock, H. Dietz (eds), *Intracranial Pressure*, 1972. Courtesy of Springer-Verlag, Berlin.

There is another deleterious effect of an elevated pCO_2 besides the effect on intracranial pressure. Raising the pCO_2 can produce little or no change in blood flow in ischemic or damaged brain because there may already be maximal vasodilatation in these areas due to hypoxia. In fact, an elevated pCO_2 will dilate the vessels of the normal brain and

shunt blood away from the hypoxic brain where it is needed (steal phenomenon).[4] A reduction in arterial pCO_2 would thus seem advisable.

During therapy of patients with increased intracranial pressure, we have maintained the arterial pCO_2 between 20-25 mm Hg. It is generally necessary to have the patient intubated, on a respirator, and paralyzed to maintain these blood gas levels. Further acute hyperventilation by hand is used when sudden pressure spikes appear. Disturbances in acid-base balance from prolonged reduction in pCO_2 have not been a noticeable problem despite weeks of hyperventilation in some instances. The beneficial effect on intracranial pressure from a lowered pCO_2 does not disappear with time.

Fluid Management and Use of Dehydrating Agents

The goal of the administration of dehydrating agents is to substitute one disease state, dehydration, for another more lethal state, increased intracranial pressure. This therapy is expected to be temporary until the cause of the pressure is corrected surgically or the inflammatory process subsides. Dehydrating or hypertonic agents can be given as a lifesaving measure for rapid relief of intracranial pressure, or a more chronic state of dehydration can be maintained for several days, if necessary. For the chronic state, it is important that electrolyte concentrations not be greatly altered from normal and toxic levels of dehydrating agents not be instituted for prolonged periods. Hypotension, with associated renal failure, is to be avoided by monitoring central venous pressure and maintaining intravascular volume with colloid.

Fluid Management

One must consider the general problem of fluid replacement prior to discussing dehydrating agents. Considerable disagreement has occurred in the past about whether glucose solutions or saline solutions should be administered as fluid therapy for patients with increased intracranial pressure. The problem diminishes in relevance if the approach is taken that the goal is fluid replacement rather than fluid therapy. Only the minimal amount of intravenous fluids is given to maintain an adequate urine output. A lowered central venous pressure or hypotension is corrected with colloid such as albumin, plasma, or whole blood. Rapid administration of an isotonic glucose solution has been shown to increase intracranial pressure more than an equivalent amount of a saline solution.[20] However, it is seldom necessary to give either solution rapidly except in cases of multiple injuries. It should be remembered that both salt and water are normally conserved by the kidneys after a traumatic event.

In our early experience treating patients with persistent brain swelling such as Reye's syndrome, a rapidly increasing serum sodium was frequently encountered. A falling serum sodium was never a problem. Measured urine sodium was near zero. For these reasons, we have avoided the administration of parenteral sodium to patients with cerebral edema for several days unless the serum sodium begins to decrease. A typical intravenous fluid regimen for an adult with brain swelling is 1000 to 1500 ml of 5% g/w or 5% g in .2 NaCl per 24 hours.

Hypertonic Glucose

The administration of a hypertonic glucose solution has long been used effectively for temporary reduction of intracranial pressure.[81] The hypertonic glucose produces a rapid shift of intracranial water into the bloodstream. The adult dose of 100 ml of 50% glucose can be injected in one bolus without significant side effects. The solution is almost universally available and no time is required for mixing. The reduction in intracranial pressure that occurs with hypertonic glucose is not as great as with other agents,[70] or as great as might be expected based on its clinical effect. Improvement in the patient's condition sometimes occurs quickly, within three to five minutes. Perhaps it is the excess glucose available for cerebral metabolism that has a beneficial effect independent of intracranial pressure.

It is essential that when hypertonic glucose is used for increased intracranial pressure, a definitive surgical procedure is performed soon, or a longer-acting dehydrating agent such as urea is also administered. A rapid rebound in intracranial pressure can be expected in about one hour as the serum glucose is cleared and fluid reenters the brain. It is also not practical to use hypertonic glucose for chronic control of intracranial pressure because of the potential hazards of prolonged high level of serum glucose.[55]

Urea

Urea can be used both acutely and chronically to control intracranial pressure. It was demonstrated in 1950 that urea produces a more profound, as well as prolonged, reduction in intracranial pressure

than does 50% glucose solution.[70] Javid[29] popularized the use of urea clinically and recommended the combination presently used of 30% urea in 10% invert sugar. The therapeutic dose of urea is 1 gm/kg body weight. Our clinical experience indicates that urea produces a greater reduction in brain bulk, as well as in measured intracranial pressure, than any of the other hypertonic or dehydrating agents administered in the recommended doses. Prolonged observations on patients with severe brain swelling, again with Reye's syndrome, have demonstrated situations in which 50% glucose, furosamide, mannitol, and glycerol were ineffective in returning intracranial pressure to normal or desired levels. In all these situations, a therapeutic dose of urea returned the intracranial pressure to normal, at least for a few hours. Also, the chronic hyperosmolar state produced by repeat doses of urea did not appear to be as toxic as with some other agents such as mannitol.

Urea is stored in the dehydrated state and requires five to ten minutes to dissolve in a warm solution. This delay is a disadvantage that could be crucial. There is also a rebound of intracranial pressure a few hours after urea administration when the brain urea concentration is higher than that in the blood. However, this rebound effect, as with other hypertonic agents, depends on the rate and quantity of fluid given after the dose of urea. Urea causes a profound diuresis, and if the patient is kept in the dehydrated state the rebound effect may not occur for several hours. Urea solutions can cause venous irritation and even skin slough with extravasation. The dose of urea should be administered over a period of 10 minutes or more. Cardiac abnormalities or even arrest can occur if a bolus is injected via syringe.

Mannitol

Mannitol is probably the most popular cerebral dehydrating agent used by neurosurgeons. It is a 6-carbon alcohol of the sugar mannose and has long been used as a diuretic by internists. Mannitol is said to be confined to the extracellular space, only slightly metabolized, and rapidly excreted by the kidneys. Its use in neurosurgery became popular after 1961 with the publication by Wise and Chater.[82] The recommended dose for reduction of intracranial pressure is 1–2 gm/kg administered over a 30-minute period.

There are advantages, as well as disadvantages, to the use of manitol as a dehydrating agent in neurosurgery. One advantage is that it is supplied in a solution that can be promptly administered without the necessity of mixing. There is less of a rebound in intracranial pressure when fluids are given following a dose of mannitol. This lack of rebound is because mannitol does not easily cross the blood-brain barrier so it exerts its effect and is excreted. There is not a period of time when mannitol is within the brain in a higher concentration than in the plasma, causing overhydration of the brain, such as occurs with urea.

However, mannitol can be dangerous if a prolonged high blood level is maintained or if the patient has renal insufficiency. Death apparently due to central nervous system toxicity from mannitol was reported in both man and experimental animals by Silber and Thompson.[68] Becker and Vries[2] noted a high mortality of patients whose osmolality was maintained above 310 mOsmoles/l with mannitol. Dodge et al[15] found that hypersomolarity could be tolerated to a level 100 mOsmoles higher with urea than with sucrose or sodium chloride. For these reasons, and because of our own experience, we have not recommended the use of mannitol for chronic control of intracranial pressure in patients with cerebral swelling. Instead, we recommend the use of urea in bursts of full or half doses as the intracranial pressure spikes occur and only then as a last resort in conjunction with other methods of reducing the pressure.

Glycerol

Increasing interest has developed in the use of glycerol to control increased intracranial pressure. Virno et al[77] described the action of oral and intravenous glycerol in reducing brain swelling in rabbits in 1961. Its use in man was begun soon afterward, and the advantage of oral administration over long periods was stressed.[7] Oral administration of glycerol can produce gastric instability predisposing to nausea and vomiting. Otherwise, it would appear to be very safe, since doses up to 25 gm/kg have been tolerated. The normal oral dose is 1 gm/kg every 6 hours.

The use of parenteral glycerol in experimental animals has resulted in reports of hemolysis[6] and renal damage.[19] However, when glycerol is administered intravenously in the proper concentration and vehicle, it is probably no more toxic than urea or other dehydrating agents.[78] Data are not available at present comparing the toxicity of hyperosmolarity produced by glycerol with that of

urea or mannitol. It is possible that intravenous glycerol could compare well with the other drugs and could be used in place of or simultaneously with these drugs.

Furosemide

Furosemide is a potent nonosmotic diuretic that was described by Kleinfelder[40] in 1963. It is rapid acting and can be used during neurosurgical procedures in a manner similar to mannitol or urea.[75] It has also been used for brain swelling independent of operative procedures.[62] Furosemide has another potential advantage in that it reduces cerebrospinal fluid production.[48] Both salt and water are excreted, and a hyperosmolar state is not as readily produced as with mannitol. The dose of furosemide for cerebral dehydration is 1-2 mgm/kg.

Serum electrolytes and glucose should be checked during furosemide therapy. The central venous pressure should also be monitored to avoid sudden circulatory collapse. Furosemide is particularly helpful when fluid overload is part of the problem in a patient with increased intracranial pressure. We have not found the drug to be very useful during the chronic monitoring and controlling of intracranial pressure in patients with severe cerebral edema. It has not appeared to be very effective in controlling intracranial pressure after the first dose.

DMSO

Dimethyl sulfoxide (DMSO) is an industrial solvent that has a multitude of pharmacologic effects. It easily crosses the dermal barrier[41] as well as the blood-brain barrier.[14] It acts as an anti-inflammatory as well as a diuretic agent, resulting in the reduction of edema. The effect of DMSO on cerebral edema in man has not been determined, but some encouraging results have been reported using an acute brain injury model in the primate.[14] Further studies are needed to determine the value of this drug.

Steroids

Most neurosurgeons have seen patients with brain tumors who have shown dramatic improvement and reversal of neurologic deficit with steroid therapy. Prevention of brain edema by using adrenal cortical extract was demonstrated as early as 1947.[25] Kofman et al[42] described an improvement in status of patients with metastatic tumors using prednisolone in 1957. The widespread use of steroids in neurosurgery began after a report by Galicich et al[22] in 1961 on the treatment with dexamethasone of cerebral edema associated with brain tumors. Using cold lesions in experimental animals, a 50 percent reduction in edema has been demonstrated at twenty-four and forty-eight hours with dexamethasone.[45] The adult dose of dexamethasone is usually 10-20 mgm initially followed by 4-6 mgm q.6h. A lag period of twelve hours after administration is necessary before a significant effect can be expected.

It has not been definitively proven how steroids work to maintain the blood-brain barrier and prevent or reverse brain edema with brain injury. Part of the problem is that the factors involved in the production of brain edema have not been clarified. Klatzo et al[38] demonstrated that a basic mechanism in brain edema development is an increase in cerebrovascular permeability to ions and proteins. Steroids function to restore this permeability toward normal.

Steroids, particularly dexamethasone, have been used freely by many neurosurgeons for all brain operations, as well as for closed head injuries and spinal cord injuries. This practice is probably reasonable, based on the potent anti-inflammatory effects demonstrated in experimental animals. However, a clear benefit of steroids in trauma patients has not been established, probably because of the wide variability of injuries in patients. Gobiet et al[23] reported that high doses of dexamethasone (100 mgm/day) significantly benefited patients with head injuries, but the usual dose (16 mgm/day) did not. The main complication of steroid administration for brain edema has been gastrointestinal hemorrhage. We have seen this complication only rarely in recent years, but perhaps this is related to the routine administration of antacids with steroid therapy.

Temperature Control

Hypothermia produces a reduction in intracranial pressure by affecting the cerebral metabolic rate. Figure 1-5 demonstrates the effect of temperature on cerebral oxygen uptake. A reduction in temperature from normal to 30°C reduces cerebral metabolism by approximately 30 percent.[60] An associated reduction in cerebral blood flow occurs through vasoconstriction resulting in reduced cerebral blood volume. This reduced blood volume provides the desired reduction in intracranial pressure or regional tissue pressure (Fig. 1-5).