42. Neurologic Surgery

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General Considerations

Neurologic surgery includes the surgical treatment of diseases of the nervous system, correction of malformations of the nervous system, the care and repair of traumatic lesions of the nervous system, and the surgical palliation of pain and abnormal motor movements. Anatomy and physiology of the nervous system, the pathophysiology of neurologic disease, and surgery in the broadest sense constitute the foundations of neurologic surgery. The goal is to preserve or restore the maximal degree of neurologic function possible within the disease state. This section will discuss a few salient concepts that provide insight into the surgical therapy of nervous system disease.

Consciousness is a prime sign of the working brain, and loss of consciousness can result from many conditions, for the function of the brain is dependent not only upon controlled excitability in many interrelated and interconnected neurons and the integrity of their supporting glia but also upon adequate function in the rest of the organism. Normal central nervous system function presuposes adequate cardiac output of oxygenated blood with normal ionic and molecular constituents and intact vascular channels for perfusion. Loss of consciousness as a presenting symptom requires not only consideration of central nervous system pathology but evaluations of cardiac output competence of blood vessels, causes of aberrant serum chemistries, possible circulating toxic substances, alterations in body temperature, and the myriad causes of each.

One of the important insights into the physiology of the brain in recent years has been the definition of the relationship of the reticular formation of the brainstem to overall function of the nervous system. Small lesions placed in the mesencephalic reticular formation of experimental animals produce a state of coma. Sudden isolation of the spinal cord from descending influences of the reticular core of the brainstem by transsection at the cervical medullary junction or more caudad in the spinal cord produces areflexic paralysis or spinal shock distal to the section, followed by mass withdrawal reflexes when spinal shock ceases. Isolation of the brainstem reticular formation from descending hemispheric impulses by a section at the upper midbrain produces facilitation of antigravity musculature, or the state known as *decerebrate rigidity*. Removal of the cerebellum in the midbrain-sectioned animal decreases the extensor tone, while sensory stimuli of any sort enhance it.

Electrophysiologic studies have shown that the reticular core is activated by all sensory stimuli and, conversely, that with total sensory deprivation, that is, section of all sensory nerves, a sleep record is obtained from the electroencephalogram that is altered or "activated" by stimulation within the reticular core. For this reason the ascending cortical influences of the reticular core of the brainstem have been called the *reticular activating system.* The reticular core is more than this. Its reticulum, or network of neurons, influences cortical and subcortical function in the broadest sense in that consciousness and gnosis (recognition of sensation), and motor function are all dependent upon its integrity. For these reasons small lesions in the reticular core of the brainstem can cause loss of consciousness, as seen with

contusions of the brainstem or infarcts following thrombosis of perforating branches of the basilar artery. These lesions also can alter motor function and motor tone and muscle tone from areflexic paralysis to decerebrate rigidity.

Surgical diseases of the nervous system are manifestations of space-occupying lesions, be they tumor, hemorrhage, abscess, edema, spinal fluid, or foreign body. The technique of their removal or the control of their effects, as well as an understanding of the mechanisms involved in their producing malfunction in the nervous system, makes neurologic surgery a specialty. The fact that the intracranialspace and the spinal canal are nearly closed boxes determines a set of mechanical conditions basic to many considerations of the effect of mass lesions on the nervous system. The intracranial space, compartmentalized by the falx and tentorium cerebri, has communication to the spinal space through the incisura of the tentorium and the foramen magnum of the skull. This represents its only major extracranial communication. The brainstem passes through these areas of communication, ie, from the incisura of the tentorium through the posterior fossa to the foramen magnum. Rostrally it is continuous with the diencephalon and the internal capsule of each hemisphere, where the medial portion of the temporal lobes have immediate lateralrelation to the upper brainstem, with a portion of the circle of Willis, the subarachnoid space, and the oculomotor, or third cranial, nerve between them. Caudad it is continuous with the cervical spinal cord, where at the foramen magnum it lies immediately anterior to the inferior portion of the ansiform lobes of the cerebellum, more commonly known as the *cerebellar tonsils.*

Central nervous system tissue has many of the properties of a fluid; that is, it is deformable but not compressible, and when deformed it flows in the course of least resistance. Deformation over months or years causes "internal" flows, or loss of substance, mainly of myelin, but rapid deformation varying with the imposed pressure causes flow toward sites of decompression, the incisura of the tentorium and the foramen magnum. While the tissue is not compressible, the lumen of its blood supply is, and obstruction of veins, capillaries, and arteries occurs with deformation. The lethal factors of the flow toward decompression areas are in part the limitation of the accommodation allowed by the semirigid infolded meningeal structures of the falx and the tentorium and the sheer stress at their edges, and the unyielding character of the bony rim of the foramen magnum. In addition, the axial as well as lateral shift of the brainstem is functionally limited by the mobility of the circle of Willis, fixed by the carotid arteries where they pierce the dura. The circle of Willis and the basilar arteries supply blood to the brainstem by vessels that enter perpendicular to its surface. With progressive axial shift the normally lax vessels become straightened and eventually tether the brain. With continuing shift there is collapse of the vessels with infarction and rupture of the smaller vessels with hemorrhage. At the foramen magnum compression of the medulla by the decompressing (herniating) cerebellar tonsils causes the same phenomena, but failure of perfusion of this area more commonly causes death before gross visible changes occur.

The situation of a noncompresible tissue nurtured by vessels whose lumens are compressible allows the compressing force to set in motion a cycle that includes production of further mass by edema with consequent increase of the size and compressing force of the original mass. The incisural syndrome of altered consciousness followed by loss of function of the oculomotor nerve, decerebrate rigidity, and death reflects the cycle in supratentorial mass lesions. At the foramen magnum, altered consciousness, followed by depression of

respiration requiring the stimulus of increased blood CO_2 or, Cheyne-Stokes respiration, depression of reflexes, cessation of respiration, and death represent progression of the cycle. The early recognition of any portion of these patterns is necessary if the lethal effects of intracranial masses are to be reversed. Their recognition and treatment is a sine qua non of neurologic surgery.

It is beyond the scope of this section to review all the neurodiagnostic techniques (and their indications) that can or should be used to investigate neurologic diseases requiring neurosurgical intervention. With the recognition that the central nervous system requires a degree of homeostasis inthe patient in order to function normally, it becomes obvious that a complete history, physical examination, neurological examination, and basic laboratory studies are requisites for any evaluation of neurological disease. The history should be carefully done, since there is a high probability of its indicating the cause of the condition being studied. In contrast, neurologic examination indicates the area or areas of the central nervous system involved and when combined with the history will define questions that can be answered by special diagnostic studies.

Diagnostic Studies. These studies may be benign, that is, of no significant risk to the patient, as with skull x-rays or electroencephalogram, or they can be dangerous enough to require the immediate availability of an operating room, as with myelography or pneumoencephalography. The accuracy of the special studies depends not only on the technical skill with which they are performed but also on knowing what question is being asked of the test and what answers can be gained. This latter aspect, as well as knowing what the risks of the test are, is why as much knowledge of the patient as a whole should be gained before using the tests and why their use in neurological evaluation is restricted to physicians knowledgeable in nervous system disease.

X-rays of the skull can be helpful in the diagnosis of neurologic disorders, for increased intracranial pressure may be reflected in springing of the sutures in the infant or decalcification of the sella turcica in the adult. A calcified pineal gland that defines the posterior position of the third ventricle may be shifted from its usual midline position by a supratentorial mass. Many tumors contain radiopaque calcifications which may be visible in skull x-rays. Meningiomas may contain calcification but usually are recognized because they evoke hyperostosis in the adjacent bones of the skull. Other stigmata of brain tumor in skull x-rays are erosions of the internal auditory meatus with an eighth nerve tumor or erosion of the optic foramen or anterior clinoid with an optic nerve tumor.

Special diagnostic studies can indicate the position, the size, and the blood supply of the tumor, and also may give information as to the amount of brain shift. The studies that involve no risk to the patient are computerized axial tomography (CT scan), electroencephalography, echoencephalography, and brain scan.

Computerized Axial Tomography. Computerized axial tomography is a major advance in radiological diagnosis whose unique attributes are particularly advantageous for diagnosis of intracranial disease. Conventional x-rays use photographic film to measure the relative amount of x-ray absorbed by the structures through which the beam has passed. Limitations of conventional x-ray include the fact that with photographic film, quantitative definition of the amount of x-ray absorbed is poor and slanted toward high-absorption matter

such as bone, and the resulting image is only two-dimensional. In the early 1970s, Geoffrey Houndsfield, a British engineer, developed a technique of a carefully controlled small beam of x-ray, a focused crystal scintillation counter, to measure accurately the amount of x-ray remaining after the beam passed through an object, and computer technology along with matrix mathematics and pattern storage techniques to produce a three-dimensional array in a computer of the object being studied through multiple directions, and the matrix is formed in the computer so that by simultaneously solving a number of equations, the amount of x-ray absorbed in each portion of the matrix is determined. The size of the area in each point is determined by the size of the matrix; with recent scanners, the image resolution is approximately 1 mm in diameter. Since brain, cerebrospinal fluid, and bone have different x-ray absorption coefficients, with the technique of computerized axial tomography, the amount and position of each of these structures can be determined, and with the use of radiopaque compounds, areas of abnormal vascularity or breakdown in the blood-brain barrier can be accurately determined. The ability to show enlarged ventricles, altered cortical surfaces, tumors, and abscesses has allowed accurate diagnosis of many neurologic conditions without any significant risk to the patient.

With the addition of a water-soluble contrast agent such as metrizamide (Amipaque, Winthrop Labs), which carries with it the slight risk of irritation of the nervous system, CT scanning can define the surfaces of the central nervous system including the spinal cord, the brainstem, and intracranial cerebral spinal fluid cisterns. Alterations in position or shape of these structures can be used in diagnosis of CNS problems. Since 1974, when Ambrose at the Atkinson Morley Hospital in England demonstrated the value of CT scanning in neurologic diagnosis, continued improvement in instrumentation and computer technology has made CT scanning the principal radiologic diagnostic tool in neurologic disease. It has almost completely displaced pneumoencephalography and is displacing myelography in the diagnosis of intervertebral disc disease. It also adds considerable information in cases of cranial and intervertebral bony abnormalities.

Electroencephalography. The electroencephalogram, or EEG, is the recording of the electrical activity of the brain by amplification of potentials obtained from electrodes places on the scalp. Its interpretation is based on knowledge of the "normal" cerebral electrical activity and the significance of alterations in symmetry, form, frequency, and amplitude of the potentials in nervous system disorders. Its most frequent use is in the diagnosis of epilepsy, with changes in the form of the potentials and the presence of focal or diffuse paroxysmal activity as important aspects of the interpretation. Focal showing is seen in the area of tumors of the cerebral hemisphere, and formerly EEG was a frequently used diagnostic test in evaluating patients suspected of having a brain tumor. Because of the accuracy of the information and the increased amount of information obtained with CT scanning, CT scanning has, to a great degree, replaced EEG in such evaluation, just as it has in other conditions such as intracranial hemorrhage and stroke.

Echoencephalography. The echoencephalogram uses a reflection of ultrasonic waves to localize structures and abnormalities within the cranium. The presence of the intact skull limits the procedure in children and adults to the determination of the position of large or high echo characteristic structures such as ventricles or the pineal gland. In infants with an open fontanelle or a patient with a skull defect, echoencephalography can give considerable information concerning not only ventricular size and position, but also presence of

hemorrhage, tumor, or foreign bodies. Its advantages, particularly for the infant, are that such information can be obtained without exposing the patient to x-ray and that the equipment is portable, allowing studies to be done without moving critically ill patients.

Radioactive Brain Scan. The brain scan uses radioactive materials such as mercury 203 or technetium 99 that will not cross the intact blood-brain barrier. Since most brain tumors are not included within the barrier, an abnormal amount of radioactive material will be present in the area of the tumor and can be localized by counting this radioactive material with a scintillation counter. This counter localizes the abnormal activity through the intact skull, producing a graphic display. Since there are many causes for breakdown of the blood-brain barrier, positive brain scans can be found in conditions which are not caused by tumors, such as in vascular occlusion, infection, or trauma. The problem of false-negative studies of lesions contained within the blood-brain barrier further limits the value of the study.

More invasive neuroradiologic diagnostic studies that have some risk to the patient include pneumoencephalography, angiography, and myelography. Their use is determined by the need for information balanced against the risk involved, and therefore, they are rarely used unless a neurologist or neurosurgeon has determined that they are required.

Pneumoencephalography. Pneumoencephalography, or the x-ray study of the brain with air, utilizes the difference in the absorption of x-rays between air, brain, and fluid. Air studies are done in one of two ways and in general outline the ventricles, the basal cisterns, and a portion of the subarachnoid spaces around the cerebral hemispheres. A patient who is suspected of a major shift of the cerebral hemispheres or who has significant symptoms from intracranial pressure is not a candidate for a lumbar puncture, and therefore the air study is usually done by a technique known as ventriculography. This consists of making openings in the skull, usually in the posterior parietal bone, tapping the lateral ventricles directly, and replacing fluid inthe ventricle with air. A series of x-ray films is then taken with the head manipulated to move the air around to show the different portions of the ventricle and to fill the basal cisterns and subarachnoid spaces.

Pneumoencephalography also may be performed with the patient in the upright position. In this position a lumbar puncture is performed, and small increments of air are placed in the lumbar subarachnoid space. The head is positioned so that the ascending air will fill the different portions of the subarachnoid space. Since both studies alter the cerebrospinal fluid circulation and may precipitate sudden shifting of the intracranial contents with possible sudden loss of brainstem function, it is always best to have an operating room and neurosurgeon available at the start of such studies; rapid decompression of the intracranial pressure and/or removal of a tumor or mass can be accomplished if the patient's condition starts to deteriorate. CT scanning has almost completely replaced both types of x-ray air studies since it is as accurate and does not have the risk of the invasive procedure or herniation of the brain. In the few cases where there is need for better definition of the subarachnoid space, a water-soluble contrast agent or a small amount of air with CT scanning can be used, attendant with some risk; however, even then the risk is smaller than that with the more classic air studies.

Cerebral Angiography. Cerebral angiography, or what is more commonly called carotid or vertebral arteriography, is the x-ray study of the intracranial circulation as

demonstrated by the injection of contrast material. These studies can define the position of the arteries and the presence or absence of abnormal vasculature, and thereby often define not only the presence of a brain tumor but also its blood supply and how it has affected the adjacent structures. Angiography is the main method of demonstrating vascular lesions such as arteriovenous malformations, aneurysms, and vascular occlusive disease. The most common method for performing angiography is by a transfemoral artery puncture, fluoroscopic positioning of a catheter into the aortic arch, carotid arteries, or vertebral arteries, and serial x-rays of the injection of contrast material. The x-rays are taken at intervals to demonstrate arterial, capillary, and venous filling. With injection of a single vessels, such as the internal carotid or vertebral, and with subtraction techniques and various timings of the serial x-rays, information necessary for the surgical approach to tumors, for repair of vascular malformations, or for reestablishing blood supply to the nervous system is obtained. The major danger is a cerebrovascular accident secondary to manipulation of the vessels or in response to injection of the contrast material. Recent computer techniques combined with fluoroscopy, called *digital enhanced angiography*, have decreased the risk by decreasing the amount of contrast material injected. Digital enhanced angiography has decreased the risk in studies done following venous injection of the contrast material for patients with cervical arterial vessel disease, the patients at most risk from arterial studies.

Myelography. Myelography is the radiologic study of the spinal canal, whereby a radiopaque contrast material is placed in the spinal subarachnoid space by a lumbar puncture or a later C_1-C_2 cervical puncture. The contrast agent is then positioned by fluoroscopy, obstructions to flow delineated, and the spinal cord, spinal roots, and soft tissue of the spinal canal outlined. The study is used to diagnose ruptured intervertebral discs, spinal cord tumors, and arterial venous malformations of the spinal cord, and, in conjunction with CT scanning, can define the central cord defects in syringomyelia. Within the past few years, water-soluble x-ray contrast material has been developed and has decreased the complication of arachnoiditis that was seen with nonsoluble agents such as pantopaque. The water-soluble contrast materials can irritate the cerebral cortex, causing seizures. Another possible complication of this study is an increasing neurologic deficit in tumors of the spinal cord or other lesions thatblock the flow of spinal fluid. The cause is alteration in relative pressures on each side of the blocking lesion after a subarachnoid puncture. For these reasons, the study is done only when the neurologic problem requires the information that can be obtained, and it is always done with supervision of persons trained in nervous system diagnosis and treatment. In patients suspected of having a spinal fluid block immediate neurosurgical care to treat precipitate deterioration in function should be available following such an exam.

Head Injury

Included within the term "head injury" are injuries of the scalp, skull, and brain. The immediate care of patients with head injury is no different from that of any injured patient. Primary consideration must be given to the establishment of adequate respiratory exchange, control of hemorrhage, and maintenance of peripheral vascular circulation. Physicians caring for the patient with traumatic injury to the nervous system too often forget these cardinal necessities or blame apparent respiratory difficulty or peripheral vascular collapse on central nervous system factors that they feel unable to alter. During the initial period of treatment of a patient with head injury, as measures are instituted for the control of the vital functions, a preliminary evaluation of the nervous system function is essential and should be followed as

soon as possible by careful repetitive neurologic examinations. The reason for repetitive neurologic examinations will become clear as complications of head-injured patients are discussed, but it suffices to state that the care of patients with traumatic lesions of the nerous system is mainly based on the detection of deterioration or improvement in neurologic function.

Scalp

Although the scalp consists of five layers, from a surgical point of view its two important layers are the dermis and the galea. The principles in the care of scalp lacerations are the same as in the care of any laceration, that is, to change a contaminated open wound into a closed clean wound, but some unique problems arise because of the structure of the scalp and its anatomic relationships. The unyielding character of the scalp with its major blood supply lying between the galea and the dermis has a tendency to hold blood vessels open following laceration with the possibility of considerable loss of blood. Hemostasis of the scalp is easily obtained, if the skull is intact, by compression of the scalp against the skull either in a circumferential fashion at the base of the scalp or by direct pressure on the wound edges. When the skull is not intact, traction on the galea by a clamp which grasps the galea, pulling it back over the dermis, will usually control the hemorrhage. The minor hemorrhage of contusions lying between the galea and the skin rarely causes any significant difficulty, but hematomas of the scalp which lie beneath the galea can attain considerable size, causing elevation of a major portion of the scalp from the skull. Early control of such hemorrhage sometimes can be accomplished by pressure and a firm dressing. Such subgaleal hematomas may take weeks to absorb, but with intact skin over them they are best treated by allowing natural absorption to occur. However, if the hematoma is of significant size to embarrass circulation of the scalp or if there is a question of infection as manifested by heat, swelling, and fever, evacuation of the hematoma is indicated. This should be done under sterile conditions, the hematoma totally evacuated, and any dead space obliterated by a compression dressing.

The approximation of the scalp to the skull not only allows lacerations to occur with blunt injury but also causes increased morbidity with infections of the scalp. Infections of the scalp may spread into the intracranial epidural space through the connecting emissary veins in the diploic spaces of the skull and into the veins on the surface of the brain. With these channels for spread of infection, epidural abscess, subdural abscess, cerebral thrombophlebitis, cerebritis, or brain abscess can occur.

Finally the galea not only is a strong fascial layer that is firmly attached to the skin by numerous fascial bands but is also the attachment of the occipitalis and frontalis muscles. These muscles tend by their contraction to separate any weak area of the galea. The problem of such contraction is that any laceration of more than a few centimeters in size will have a tendency to pull open unless the galea is closed at the time that the dermis is closed; any large laceration of the scalp which is allowed to remain open for more than 5 to 6 days may show a significant amount of contraction with fibrosis, so that it cannot be closed without special plastic techniques.

Thus, the toilet of a scalp wound must be thorough and include shaving of hair in the immediate vicinity, debridement of all the devitalized tissue, and removal of any foreign body.

The wound closure must include closure of the galea, either as a separate layer or as a layer included within the dermis. If the laceration is of a large size with separation of a major portion of the galea from the subgaleal tissue, a compression dressing will make the patient more comfortable and decrease the chances of a subgaleal hematoma and infection.

Skull

Anatomy. The skull is divided into the cranium, which contains and protects the brain, and the facial bones. The cranium consists of eight bones: two parietal and temporal bones and one frontal, occipital, ethmoid, and sphenoid bone. The superior, or rounded, portion of the skull is called the *vault,* and its bones are formed as membranous bone. In the adult the bone consists of firm inner and outer tables with cancellous bone, or diploë, lying between them. The skull contains within the frontal, ethmoid, and sphenoid bones mucous membrane-lined sinuses which connect to the nasal cavities. The basilar, or petrous, portion of the temporal bones contains the middle ear and mastoid air cells, which also connect to the nasopharynx via the eustachian tube.

Classification of Skull Fracture

The clinical term "skull fracture" is used for fractures of the cranium. Such fractures are described by using a term describing the pattern of the fracture (linear, stellate, comminuted), by using the term "depressed" to denote an inward displacement of a portion of the vault, by naming the bone involved, by including the term "basilar" for fractures traversing the base of the skull, and by describing a brak in the scalp or mucous membrane over such a fracture with the term "compound fracture." Thus, a compound comminuted depressed fracture of the left parietal bone states that there is a scalp laceration over a fracture of the parietal bone on the left that consists of multiple fragments of bone driven beneath the surface of the cranial vault.

Fractures of the skull may occur in any area of the skull and have a tendency to radiate from the point of contact into the weaker areas of the skull, eg, basilar or temporal areas. Most skull fractures require no treatment in themselves, but compound and depressed fractures must be treated. A compound fracture of the cranial vault should be cleansed and debrided, and the wound closed. Replacement of large skull fragments in the area of a compound fracture requires surgical judgment and depends upon the degree of contamination, intactness of the dura, the area of the skull involved, and the ability to watch the patient closely after injury. If the surgeon is in doubt in any of these respects, it is always wisest to remove all fragments of a compound fracture.

With depressed fractures the rule is to elevate all of them, but again this should be based on surgical judgment of the size of the depressed segment, the depth to which it is depressed, and the area of the skull depressed, and the ability to be certain that the dura has not been torn. Any fragment which has been depressed more than a centimeter, a fragment which is over the motor strip, and most small fragments that appear sharp on x-ray usually should be elevated, since they may tear the dura and cause damage to the brain. The combination of a depressed and compound fracture will always require elevation to be certain that contamination has not been driven through the dura into the subarachnoid space or the brain.

Another type of compound skull injury, one that is not as obvious as a penetrating injury, is the injury in which a fracture of the skull traverses one or more of the paranasal sinuses, the mastoid air cells, or the middle ear. The indication that this type of open head injury has occurred is the presence of cerebrospinal fluid drainage from the nose or ear. It signifies a rupture of the protective meningeal coverings of the brain and requires observation and prophylactic antibiotics to be certain that infection of the subarachnoid space or brain does not occur. The posttraumatic cerebrospinal fluid fistula of the ear almost always heals within a few days, but healing of rhinorrhea may be more difficult. It is important in both injuries that the patient remain under medical supervision until such drainage ceases, and usually if either otorrhea or rhinorrhea continues for more than 10 to 14 days, surgical repair of the dural tear is necessary.

Brain

Mechanisms of Injury

The most critical aspect of head trauma is what happens to the brain. The immediate brain damage that results from head trauma is dependent upon the force applied to the head, the area of its application, and whether the head is fixed or freely movable. The damage can be caused by the injuring object or a portion of the skull lacerating the brain, the effects of force transmitted to the brain, and the effects of acceleration and deceleration of the head on the brain confined by the semirigid dura and rigid skull. Delayed brain damage is mainly caused by the reactions of the tissues within the skull. When viable tissue receives an application of force strong enough to be injurious, it responds by alteration in intracellular and extracellular fluid content, by extravasation of blood, by increasing blood supply to the local area, and by mobilization of cells capable of removing cellular debris and repairing any disruption. The same mechanisms occur in injuries of the brain, and these mechanisms, as well as alteration of vasomotor control or autoregulation of cerebral circulation, may have secondary deleterious effects on the brain because of the unique anatomic position of the brain within the skull and the brain's unique dependency on a constant supply of oxygen and glucose.

It is simplest and easiest to consider the initial brain injury of head trauma as being caused in one of two ways: by the application of a focal, or local, force tothe skull or by the application of a generalized, or diffuse, force to the skull. There is considerable overlap between the two, since a patient may have both types of injuries at the same time, but the mechanisms and results of the two types of injury are different. In focal injury, a sharp object of low velocity strikes the head and may cause disruption of the scalp, fracture of the skull, and laceration of the brain, either directly or by driving fragments of the skull through the dura mater. The resulting neurologic dysfunction relates to the area of the brain involved, since no significant force is transmitted to the skull or brain generally. There is usually brief loss or no loss of consciousness, and the main problem is the care of the local wound.

Following the application of a more generalized force to the skull, there are three other mechanisms that may cause brain injury and complicate or amplify any focal injury that has occurred. Such an application of force may occur following high-velocity missile injuries, such as a gunshot wound, but is most commonly seen in blunt head injuries or injuries in which the freely movable head is struck by a heavy object or when the head is propelled

against a solid structure as seen in a high-speed vehicle accident. These mechanisms of brain injury in the more generalized application of force to the skull are injury caused by the direct transmission of energy to the brain, injury caused by the inertia and momentum of the brain when the head is accelerated and decelerated causing the brain to strike against the skull and edges of the dura, injury caused by the torque or compressive force applied to the relatively fixed upper brainstem with the rotation of the cerebral hemispheres during such acceleration and deceleration, and the stretching effect of this movement on the various tissues of the intracranial content.

The effects of transmitted energy to the brain relate to the amount of force applied minus the amount of energy dissipated by disruption of the scalp or skull. The force dissipated in fracturing the skull can be considerable and in part accounrs for the paradoxic situation in which brain injury appears inversely related to the skull injury. The remaining energy after disruption of scalp and skull can either be transmitted through the skull or be directly applied to the brain, if the injuring object penetrates the skull. The energy spreads concentrically from the point of application with maximal transmission in the primary direction in which the force was applied. As this energy passes through the tissues of the brain, and particularly through the interphases of the vascular channels and cerebral spinal fluid spaces, a change in direction of the force occurs and may cause disruption or damage to the tissue. This type of damage will usually be most marked at cortical and ventricular surfaces and adjacent to blood vessels within the hemispheres, since these are areas of major changes in tissue density and tensile strength.

The other mechanisms of brain injury following head trauma are more commonly the cause of severe brain damage and neurologic dysfunction in the head-injury patient and therefore are more frequently the cause for major difficulty in the care of these patients. The mechanisms relate to induced acceleration and deceleration of the head, the inertia of the brain at the moment of acceleration, and the momentum of the brain at the time of deceleration. These forces cause motion of the head and its contents, forcing the brain against the inner surface of the skull where the force has been applied, as well as against the opposite inner surface of the skull (countrecoup injury). Such contact may cause damage to any surface of the brain but is usually most severe when it involves a motion which throws the brain against the sphenoid ridges and/or the free edges of the tentorium cerebelli. This latter action is most likely to occur when the head is struck in such a way as to produce rotation of the skull. The areas of the brain most likely to receive severe damage are the anterior portions of the frontal and temporal lobes, and the posterior portions of the occipital lobes, and the upper portions of the midbrain. The intrinsic movement of the brain tissue with the varying degrees of resistance to such movement of blood vessel, fiber tracts, glia, and neurons results in stretching and rupture of the tissue, with maximum effect on the perivascular tissue and the white matter of the hemispheres and brainstem. The torque effect on the upper brainstem is also most easily obtained in the freely movable head that is struck in a manner to cause marked rotation. The effects of such torque applied to the upper brainstem cause almost instant alteration in neuronal function with resulting change in consciousness. This loss of function is probably the cause of the immediate loss of consciousness seen with "concussion." The leverage of a blow applied to the point of the jaw easily gives this rotational effect to the skull and may explain the "knockout" effect of the uppercut in boxing.

In summary, a force applied to the skull may have a local effect of laceration of the scalp, fracture of the skull, and laceration and contusion of the brain, or it may have a more generalized effect caused by the effect of energy transmitted through a semisolid substance, a contusing and lacerating effect when the brain is driven against the inner portion of the skull and edges of the dura, the stretching and tearing effect of the resulting internal movement of tissue, and the loss of function by the compressive effect of the torque or stress applied to the upper portion of the midbrain with rotational movement of the brain. These mechanisms result in the clinical conditions of cerebral concussion, cerebral contusion, and cerebral laceration. They, plus the effect of alteration in total function of the nervous system, the alterations in cerebral circulation, the changes in respiration and vasomotor control, and the reaction of the nervous system tissue to injury, are the main reasons for the clinical syndromes seen in the head-injured patient.

Care of the Head-Injured Patient

The care of the head-injured patient can be divided into temporally sequential aspects. These are first aid; evaluation; protection from the loss of nervous system function; protection from the response of injured nervous system tissue; prevention, recognition, and treatment of complications; and rehabilitation. The eventual morbidity and mortality of the head-injured patient can be markedly improved by knowledgeable and effective care throughout the course of the illness. In general, hospitals with specialized centers for neurosurgical patients and for rehabilitation for neurologically disabled patients should be utilized as soon as it is feasible to move the patient. As a rule the patient is best transferred to such an area for definitive care as early as possible, since with few exceptions the patient is most capable of transport immediately after first aid and evaluation have been completed.

First Aid. The basic principles for first aid of the head-injured patient have already been discussed in the opening paragraphs of this section on head trauma. They consist of maintenance of respiration, control of hemorrhage, and maintenance of peripheral vascular circulation. The importance of such measures cannot be stressed too much, since any failure of these vital functions not only is a significant factor in the mortality of head injury but can be significant in increasing eventual morbidity. The addition of anoxia or loss of circulation, from either respiratory obstruction or peripheral vascular collapse, can cause a marked increase in neuronal damage and may contribute to the degree of reaction the brain has to the original injury.

Another aspect of first aid is the transport of the patient, either from the scene of the accident or to a hospital for definitive therapy. The unconscious head-injured patient should always be accompanied by an attendant who can clear the nasopharynx and assist respiration. The patient is most safely transported in a two-thirds prone position with his head slightly elevated by a firm roll or his arm above a firm surface. This allows secretions from the nasopharynx and vomitus to drain onto the stretcher rather than be aspirated into the trachea.

Evaluation. Evaluation of a head-injured patient includes careful examination for other injuries. Visible lacerations and obvious deformities of the extremities signify lesions which must be treated, but internal injury to the thorax, abdomen, or pelvis may be difficult to diagnose in the unconsciou patient. Continued hypotension and tachycardia are the most consistent signs of such injuries and should always indicate further investigation, since

peripheral vascular collapse from central nervous system damage is seen only as a terminal event. Injury to the cervical spine may occur in head injury, and x-rays of the cervical spine may be necessary to rule out such injuries.

A complete neurologic examination should be recorded as soon as possible, and this must include an accurate evaluation of the state of consciousness. The most common neurologic deficit that occurs with head injury is altered consciousness. Accurate evaluation of the state of consciousness and comparison of the initial examination with subsequent examinations are the most reliable means for evaluating the progress of the head-injured patient. Terms such as "comatose", "semicomatose", and "stuporous" that are commonly used to describe states of consciousness have little meaning and are best used, if used at all, as a means of introducing descriptions that have objective findings as their method of evaluation. The evaluation of the state of consciousness is done by utilizing the response of the patient to various stimuli. If the stimulus and the response are described, the evaluation can be repeated and compared later b the same examiner or others. Table 42-1 offers a general description of the levels of consciousness to be determined by this method. Levels between those described can be easily devised when required.

Table 42-1. Levels of Consciousness

Responds to spoken word, is alert, cooperative, and oriented.
Responds to spoken word, is confused, and obeys simple commands.
Responds to spoken word only after receiving painful stimuli of supraorbital pressure; obeys commands only while receiving painful stimuli.
Does not respond to auditory stimuli before or while receiving painful stimuli but has purposeful, effective motor response to painful supraorbital and sternal pressure.
Responds to painful supraorbital and sternal pressure with purposeful but noneffective motor movements.
Responds to painful supraorbital and sternal pressure with nonpurposeful movement.
Responds to painful stimuli only with alteration in pulse and respiratory rate or with decerebrate posturing.
No response to painful stimuli, but cough and gag reflexes are present.
No response to stimuli.
Lowest level is unconsciousness; the only step below this is death.

By utilizing objective responses to stimuli it is not difficult to recognize progressive neurologic deficit. The difference in neurologic function between the confused patient who follows commands and the patient who responds to painful stimuli with nonpurposeful movements is obvious, and the time it takes for such neurologic deficit to develop indicates how rapidly definitive treatment must be started. Decerebrate posturing as a description of a level of consciousness is the posture assumed by a patient with physiologic or anatomic section of the upper brainstem. It is basically the posture assumed when there is facilitation of all antigravity musculature. The patient therefore extends and internally rotates his extremities, extends his neck, and arches his back. Fragments of decerebrated posturing usually appear first with thrusting of the lower extremities. Since it signifies failure of function of the upper brainstem, it is an ominous sign that requires immediate definitive therapy. It is seen with shift of the brain by a supratentorial mass and therefore is often the preterminal event in the patient with untreated posttraumatic intracranial hemorrhage.

Jennett and coworkers over the past decade have utilized a simple scale to assess the level of severe head injuries in order to study and compare various forms of therapy. Though the outcome of the study comparing various forms of therapy is not complete, the reliability of the "Glasgow Coma Scale" in predicting the outcome of severely traumatized patients who have received good neurosurgical treatment in various clinics and countries has been demonstrated. The scale (Table 42-2) is applied to a patient who has been unconscious for more than 6 hours, thus eliminating relatively minor head trauma. The score is the sum of the highest numbers obtained in each section of the scale, ie, motor response, verbal response, and eye opening. Jennett has demonstrated that patients with less than 5 on the scale have an overall mortality rate of over 50 percent. The scale basically shows that the levels of consciousness and brainstem function closely parallel the severity of the injury and thus the mortality rate. It further points out how evaluation of general function of the brain, ie, consciousness and a portion of the brain that responds to shift and flow of intracranial contents, the midbrain, can give a fairly accurate picture of the amount or progression in amount of injury to the central nervous system.

Table 42-2. Glasgow Coma Scale

Best motor response	Obeys	M6
	Localizes	5
	Withdraws	4
	Abnormal flexion	3
	Extensor response	2
	Nil	1
Verbal response	Oriented	V5
	Confused conversation	4
	Inappropriate words	3
	Incomprehensible sounds	2
	Nil	1
Eye opening	Spontaneous	E4
	To speech	3
	To pain	2
	Nil	1

Note: The coma scale score is the sum of the sectional scores.

Observation and Diagnostic Studies. Therapy is dependent upon the evaluation of the initial and repeated neurologic examination. Most patients can be observed over a short period of time, 30 minutes to a few hours, while first aid is given and other areas of the body evaluated; following observation a more accurate decision can be made as to what is necessary for treatment. Studies other than observation and neurologic examination are helpful but are never indicated if they remove a seriously ill patient from medical and nursing observation.

Radiographs of the skull can be helpful especially if the pineal gland is calcified, since shift of its midline position in the anteroposterior view will signify a mass lesion. The skull radiographs may show depressed fractures or fractures that traverse arterial vessel grooves in the skull (middle meningeal artery), indicating a possible source for bleeding (epidural

hematoma). They may demonstrate air within the skull, indicating a tear in the coverings of the brain, or they may show a foreign body. Computerized axial tomography has altered the evaluation of acute head problems since it can demonstrate recent intracranial hemorrhage quite accurately and indicate both its size and location. The scanner can also define shift of intracranial contents, as well as areas of localized edema. No diagnostic procedure should displace clinical observation, but inthe critically ill head-injured patient, the accurate knowledge that intracranial hemorrhage or mass exists or does not exist allows more accurate control of intracranial pressure and cerebrovascular perfusion pressure without invasive x-ray procedures such as angiography and ventriculography, and quickly defines the need or lack of need for surgical excision of intracranial masses from the injury. If a scanner is not available, further studies such as carotid arteriography or exploratory perforator openings of the skull are indicated only when the patient's condition precludes serial evaluations or a course of progressive neurologic deficit has been established.

During and following the period of initial evaluation frequent recording of the blood pressure, pulse rate, and respiratory rate is indicated, since elevation of blood pressure and decrease in pulse and respiratory rate are seen with increasing intracranial pressure and may indicate impending medullary failure. As these changes in vital signs are less sensitive and usually occur later than alteration in levels of consciousness, they are helpful but not as accurate in defining the need for active intervention.

Lumbar puncture has little place in the early evaluation of head trauma. The risk involved in altering spinal fluid pressure when the possibility exists of mass lesion in the calvarium far outweighs any information gained. The presence or absence of blood in the spinal fluid or the presence or absence of increased spinal fluid pressure does not signify the need for, or the contraindication for, any form of therapy. Lumbar puncture can at times be helpful in deciding that an unconscious patient without history has had a subarachnoid hemorrhage or meningeal infection before, or instead of, head trauma.

Supportive Therapy. During the entire period of treatment of the head-injured patient measures must be instituted to provide substitutes for the protective nervous system reflexes that may have been lost. Any obstruction to the airway or any difficulty with keeping clear the pharynx, larynx, or trachea should be handled by endotracheal intubation. At times with facial or cervical spine injuries an emergency tracheostomy may have to be done. Gastric suction through a nasogastric tube is helpful not only in preventing gastric dilatation and the resulting respiratory difficulty but also in preventing the regurgitation of gastric contents into the nasopharynx and trachea. A Foley catheter for bladder drainage allows accurate measuring of urinary output and prevents overdistension of the urinary bladder.

Control of water balance may be altered so that diabetes insipidus or lack of antidiuretic hormone release may occur. Accurate recording of urinary output, the replacement of fluid, and the use of exogenous antidiuretic hormone may be necessary to control urinary loss. The opposite effect or the inappropriate secretion of antidiuretic hormone is more common. Inappropriate secretion of antidiuretic hormone should be suspected when serum electrolyte concentration decreases, eg, serum Na to 120 meq/L, and confirmed by demonstrating high urinary osmolarity at the same time serum osmolarity is low. While the low serum sodium suggests treatment with hypertonic salt solution, the problem is retained water with increased blood volume; the sodium chloride in such a solution will be excreted

because of the altered aldosterone mechanism, but a considerable portion of the water will be retained. Restriction of water intake below insensible water loss orthe use of solute diuretics that can override the antidiuretic hormone effect is necessary to control this complication.

Temperature regulation may be deficient with head trauma; most commonly the deficiency is in the heat-loss mechanism, that is loss of superficial vasodilation and sweating. The resulting hyperthermia can contribute to the central nervous system damage and to central nervous system response to damage. Frequent rectal or esophageal temperature should be taken. If fever ensues, exposure of major portions of the body, tepid-water sponging, a hypothermic mattress, and/or decreasing the body metabolism by drugs may be necessary to control the fever.

Prevention and Treatment of Cerebral Edema. The nervous system tissue, like other tissue, responds to injury by formation of edema. Since thebrain is encased within the skull and dura, the formation of edema causes increased intracranial pressure which may become of such magnitude that it equals intracranial arterial pressure. At that point circulation through the brain ceases, and death of tissue ensues. When this happens, the situation is usually irreversible, because relief of pressure must be accomplished within 4 to 5 minutes in order to avert neuronal death. Since the formation of edema is gradual, starting a few hours after injury and reaching its maximum in 36 to 48 hours, protective measures instituted promptly can have considerable therapeutic value.

Intracranial pressure in the head-injured patient is dependent upon a number of factors. The main one is the volume of fluid in each of the intracranial compartments, ie, cerebrospinal fluid and fluid in vascular, intracellular, and extracellular spaces. The extracellular space is small, and its contribution tobrain swelling is open to argument; the increase in intracranial pressure appears to relate to alteration in volume of the vascular bed, intracellular space, and the cerebrospinal fluid space. Modification in the reaction of the brain to injury is dependent upon modifying the volume responses of these spaces.

The vascular bed volume is dependent upon venous pressure, CO_2 content of the circulating blood, and arterial pressure. It is generally unwise to alter arterial pressure except to raise it toward normal. The blood CO_2 level and venous pressure can be reduced by making certain there is no obstruction to the airway, by assisting respiration with a respirator, and by elevating the head of the bed 15 to 20°. The intracellular response depends on breakdown of the blood-brain barrier, the metabolism of the cells, the omsolarity of the blood, and the availability of free water. Keeping the head-injured patient at normothermic or slightly hypothermic temperature (34 to 37°C) decreases the chance of hypermetabolism from hyperthermia, but significant decrease in the metabolism of the brain usually requires the use of drugs. Barbiturates (barbiturate coma) combined with a muscle-paralyzing agent are commonly used since they decrease cell metabolism and muscle activity and allow control of respiration without excessive intrathoracic pressure. With this combination of drugs, the ability to monitor neurologic function is lost. A CT scan is required to rule out any significant intracranial hemorrhage or mass before starting the therapy, and monitoring of intracranial pressure as described below is needed to be certain that cerebral perfusion pressure is not impaired.

Removal of fluid in the hydrated or overhydrated patient with solute diuretics such as mannitol or urea, plus limitation of fluid intake to insensible water loss and urinary output, is helpful for the control of intracellular edema. The fluid intake to accomplish this usually is 1.800 to 2.000 mL/24 hours and should include at least 150 meq of sodium chloride. One-half normal saline solution and 2.5% glucose approximates these requirements for intravenous fluids. The inflammatory response may be decreased and the blood-brain barrier protected by the use of glucocorticosteroid compounds. Dexamethasone is commonly used in intravenous doses of 10 mg shortly after injury and 4 mg every 6 hours for the first 36 to 72 hours. The dose is later tapered at a rate dependent upon the course ofthe patient. Fluid restriction also helps control the volume of the cerebrospinal fluid compartment.

Lumbar puncture would appear to be of help, but since the possibility of a mass lesion (hemorrhage) always exists and since asymmetric contusion and swelling frequently exist in the head-injured patient, drainage of spinal fluid via the lumbar route is too dangerous. Such drainage may cause herniation of the temporal lobe into the incisura of the tentorium and compression of the upper brainstem or herniation of the cerebellum through the foramen magnum and compression of the lower brainstem.

Recent studies have shown that posttraumatic edema of the brain can cause such raised intracranial pressure that cerebral perfusion ceases, and that most cases of "malignant" posttraumatic edema after head trauma are caused by repetitive episodes of inadequate cerebral perfusion that relate to transient waves of intracranial pressure. These "plateau waves", first described by Lundberg and coworkers, have been shown to relate to dilatation and later constriction of the cerebrovascular resistant vessels. In the injured brain, such changes in cerebrovascular resistance cause changes in blood flow and blood volume, and since the brain has lost its compliance or ability to tolerate small changes in volume without major changes in pressure, these waves of pressure are seen if intraventricular recording of pressure is done. Since cerebral blood flow relates directly to systemic arterial pressure minus intracranial pressure and inversely to the cerebrovascular resistance

CBF = ((K(SAP-ICP):VR),

during episodes of plateau waves, effective cerebral perfusion is impaired and repetitive inadequate perfusion of the brain occurs. With each occurrence, further damage to the blood-brain barrier, neurons, and glia occurs. When these episodes have caused enough damage or when there is vasomotor paralysis from injury, the increased intracranial pressure causes bradycardia and hypertension systemically or the Cushing effect, which then causes further increase in intracranial pressure from the effect of increased blood volume and pressure intracranially. As mentioned above, this is often a late sign and in the head-injured patient may be the initial sign of impending respiratory arrest and total loss of effective intracranial vascular perfusion.

Since it has been demonstrated that effective cerebral perfusion pressure requires that systemic arterial pressure be at least 50 mmHg greater than intracranial pressure, in the severely injured patient, direct measure of intracranial pressure by placing a catheter in the frontal horn of the lateral ventricle makes it possible, when systemic arterial pressure is also monitored, not only to recognize the presence of plateau waves and inadequate cerebral perfusion pressure but also to undertake treatment to control the pressure and thus avert these

transient episodes. The measures described above to control respirations and thus levels of CO_2, the use of solute diuretics such as mannitol or urea, and since there is a catheter in the ventricle, drainage of spinal fluid can markedly protect the brain against this secondary injury. Many neurosurgical services routinely monitor intracranial pressure in all patients in whom there is major impairment of levels of consciousness for more than 6 to 10 hours and in all patients whose function onthe Glasgow Coma Scale is 6 or less. With these techniques, malignant cerebral edema following trauma is rare and protection of the brain against secondary injury from inadequate cerebral perfusion allows a better functional result, although the mortality rate of severely head-injured patients has not been significantly altered.

Intracranial Hemorrhage

Progressive neurologic deficit, the most common being progressive loss of consciousness, following head trauma should always be assumed to be due to intracranial hemorrhage until proved otherwise. The patterns of neurologic change vary with the type of hemorrhage, but all have as a major component the neurologic deficit of progressive loss of consciousness.

The most common intracranial hemorrhage following head trauma is subarachnoid hemorrhage. It has little surgical significance and usually causes signs of meningismus or stiff neck and headache. It may, in the young male, produce maniacal behavior. It has little significance surgically, because the blood is rapidly diluted by the cerebrospinal fluid and flows throughout the subarachnoid space, so that no significant localized mass effect occurs. It may have significance as a late complication of head injury, causing progressive communicating hydrocephalus. This complication is rare, occurring weeks or months after injury, and presents as a progressive dementia with motor apraxia, usually most marked as a gait disturbance. The mechanism is though to be obstruction of the arachnoid villi and/or basal cisterns so that the absorption of cerebrospinal fluid is altered. The treatment is the same as for infantile hydrocephalus with the construction of an artificial path for the absorption of cerebrospinal fluid. This is done with a shunt from the lateral ventricle to the superior vena cava or peritoneum using a valve to prevent blood reflux and to stabilize the pressure inthe ventricle at 50 to 70 mm of water.

Surgically significant intracranial hemorrhages are best classified by their anatomic positions, ie, subdural hematoma, epidural hematoma, and intracerebral hematoma. While they are relatively rare, unrecognized and untreated posttraumatic intracranial hematomas have almost a 100 percent mortality rate. Since most of this mortality can be averted by surgical treatment, early and accurate diagnosis is important.

Subdural Hematoma

One of the most common types of posttraumatic intracranial hemorrhage is subdural hematoma. It is caused either by rupture of the veins traversing the subdural space from the brain to the dural sinuses or by a laceration of the brain that has torn the overlying piaarachnoid. The symptoms may appear as early as within the first few minutes or as late as 6 to 8 weeks after injury. The pattern of symptoms, the findings, the treatment, and the prognosis vary with the rapidity of formation of the hematoma, so that it is logical to consider the syndrome of subdural hematoma as three different types: acute subdural, subacute

subdural, and chronic subdural. Subdural hematomas and epidural and intracerebral hematomas are the common surgically important intracranial hemorrhagic complications of trauma. Although each has a different symptom pattern, all have somewhere in their history the finding of decreased level of consciousness out of proportion to focal neurologic deficit. Portions of the basic patterns are usually present in all patients with subdural hematoma, but at times discovering them can tax the most astute clinician. The following descriptions are to be taken as outlines of patterns commonly presented by the patient with the different types of intracranial hematoma, but the reader should always keep in mind the protean signs and symptoms possible in a patient with intracranial hematoma.

Acute Subdural Hematoma

Acute subdural hematomas are defined as hematomas that cause significant progressive neurologic deficit within 48 hours of injury. They almost always occur following severe head trauma, and since they may have borth arterial and venous sources for bleeding, the progression of the neurologic deficit can be rapid, often in terms of minutes or hours. The source of the arterial bleeding is frequently a laceration of the brain, and therefore focal neurologic deficit such as a hemiparesis is common.

Clinical Manifestations. When first seen, the patient is usually unresponsive with a focal neurologic deficit. Progressive decrease in sensorium with or without progressive focal neurologic deficit is the warning of increasing intracranial mass. Without treatment the brainstem will be compressed by hemorrhage, edema, and herniation until death results. Death may occur within hours, with cessation of respiration, maintenance of pulse and blood pressure for a short time, and then peripheral vascular collapse as the medulla fails. Diagnosis is made by always considering the possibility of acute subdural hematoma in the severely head-injured patient who shows any deterioration in neurologic status. Aid in the diagnosis and in determining the site of major hemorrhage can be gained by the localization of any focal neurologic deficit, the presence of a lateral shift of a calcified pineal gland, and shift of the midline in the echoencephalogram. As mentioned above, computerized axial tomography is accurate in defining the position and size of recent intracranial hemorrhage and therefore, is the best method, if there is time, of diagnosing acute subdural hematomas. It also can define adjacent intracerebral hematomas, a common concomitant of acute subdural hematomas, making surgical approach to the hemorrhage more accurate and effective. More than half of acute subdural hematomas are bilateral, so that evaluation of both subdural spaces is indicated.

Treatment. Treatment consists of removal of the hematoma through a large craniotomy with control of bleeding areas, decompression by excision of large areas of the skull and relaxation of the compressing dura, and, when necessary, internal decompression by excision of portions of the frontal or temporal lobes. The methods of controlling edema and a secondary injury from loss of effective cerebral perfusion pressure, as outlined in a preceding section, have decreased, but not eliminated, the need for such decompressive procedures and decreased both the problem of shift of the brain and the damage that can occur as an area of brain protrudes through a decompression site, as well as the later need for repair of surgical defect in the skull. Drainage of the hematoma through perforator openings is never satisfactory, since the major portion of the hematoma is solid clot and the problem is a combination of mass from clot and reaction of the brain to severe trauma. Without

prompt surgical care mortality is 100 percent, and evenwith the best care it can be as high as 80 to 90 percent.

Subacute Subdural Hematoma

Subacute subdural hematomas are complications of head trauma that cause significant neurologic deficit more than 48 hours but less than 2 weeks after injury. They are usually caused by venous bleeding into the subdural space.

Clinical Manifestations. The basic pattern of symptoms and signs is a history of head trauma with unconsciousness, gradual improvement in the first few days followed by lack of improvement, fluctuation in levels of consciousness, and then decompensation with progressive loss of consciousness and often partial loss of hemispheric function. The patients are usually not as severely injured as are the patients with acute subdural hematomas. The phase of fluctuation in level of consciousness often heralds that significant shift of the intracranial contents has occurred, and the patient may change from relatively alert to difficult to arouse even with painful stimuli and back to his alert status within a few hours. Herniation of the medial temporal lobe through the incisura of the tentorium and compensation of the midbrain to the pressure of such herniation is a possible mechanism of such fluctuation in levels of consciousness. The presence of a third nerve paresis with dilatation of the pupil is often a warning that midbrain decompensation is imminent. The computerized scanner may not identify subacute subdural hematomas, since they can become isodense within the first 10 to 12 days after injury (ie, the x-ray absorbed is approximately the same as brain). If the hematoma is unilateral or asymmetrical, the shift of the ventricular system as seen on computerized scanning will suggest the location of the hematoma. If the clinical history and signs suggest sudural hematoma, even without confirmation by CT scanning, the diagnosis should be confirmed or excluded by multiple perforator openings or by angiography.

Treatment. Treatment is dependent upon how critical the patient's condition is, how much liquid clot there is, and whether significant temporal lobe herniation has occurred. The patient with solid clot, unresponsive from uncal herniation, requires craniotomy, removal of the clot, and elevation of the temporal lobe herniation with or without incision of the tentorial edge. In the less critically ill patient and in the patient with considerable liquefaction of the hematoma the removal of a major portion of the clot through a small craniectomy and multiple perforator openings followed by external drainage of the subdural space may be all that is required. As with acute subdural hematomas, these hematomas are commonly bilateral, and exploration of both subdural spaces is indicated unless the CT scan or angiograms have ruled out the presence of bilateral hematomas.

Chronic Subdural Hematoma

Although the initial cause of the chronic subdural hematoma is usually rupture by head trauma of one of the veins traversing the subdural space, the symptoms are caused by the increasing mass effect of the hematoma surrounded by a semipermeable membrane. Within 7 to 10 days after bleeding has occurred inthe subdural space the blood is surrounded by a fibrous membrane. As the blood cells within the membrane break down, fluid is osmotically pulled into the hematoma, causing an increase in its volume. This may cause further bleeding

from tears in the membrane or by rupturing other traversing veins with the reestablishment of the same process and an increasing size of the semiliquid-filled membrane.

Clinical Manifestations. Chronic subdural hematomas may occur at any age but are most frequent in the infant or the elderly. The causative trauma, particularly in the elderly, may be so slight that the patient does not remember it and gives no history of trauma. The symptoms and signs are best classified as a progressive alteration in mentation and level of consciousness of 4 to 6 weeks' duration out of proportion to the focal neurologic deficit. Therefore, any patient presenting with a progressive change in mental faculties and fluctuation or decreasing level of consciousness should be considered a subdural hematoma suspect.

Treatment. Since the hematoma is liquid, drainage through perforator openings is usually all that is required. In the past many neurosurgeons have felt that removal of the membranes is required, since they may be quite thick. However, experience has shown that there is increased mortality and morbidity from craniotomy removal of membranes and no improvement in the long-term results over those achieved by simple drainage of chronic subdural hematomas.

Subdural Hygroma

Although subdural hygromas are not a part of intracranial hemorrhage, they can produce similar symptoms. Subdural hygroma is usually caused by a tear in the piarachnoid that acts as a one-way valve with leakage of cerebrospinal fluid into the subdural space. The hygroma can increase in size quite rapidly and therefore can mimic an acute or subacute subdural hematoma. Perforator openings of the skull with external drainage is the treatment of choice, but at times formation of an artificial fistula between the subarachnoid space and the subdural space must be made in order to control continued formation of the hygroma.

Epidural Hematoma

Epidural hematomas may be caused by either venous or arterial bleeding. They lie between the skull and the dura. The most common cause is venous bleeding, but the surgically important epidural hematomas most frequently are formed by arterial bleeding. Occasionally the patient with a skull fracture may have a significant venous epidural, the removal of which allows decrease in intracranial pressure or release of focal compression of the brain, but generally venous epidural hematomas are limited by the firm adherence of the dura to the inner surface of the skull. The central point of the venous epidural is almost always at the site of a fracture.

The epidural hematoma from arterial bleeding commonly occurs from rupture of the middle meningeal artery. This may be caused by a fracture of the temporal bone where the artery is in close proximity to the bone or by a tear of the middle meningeal artery when a blow causes angular acceleration of the head or sudden decrease or increase in any diameter of the skull. The tear may occur at the foramen spinosum, where the artery enters the skull, or anywhere along its branches.

Clinical Manifestations. The basic historical pattern of the epidural hematoma is a young adult who has received a relatively minor blow causing momentary alteration in

consciousness, followed by a lucid interval extending from a few minutes to a few hours. This interval terminates by rapid progressive loss of consciousness, dilatation of the pupil on the side of the epidural hematoma, evidence of compression of the upper midbrain, by either the production of a hemiparesis or decerebrate rigidity, and then evidence of compromise of the entire brainstem and death. The frequency of the hematoma being over the temporal area, the direct pressure onto the temporal lobe and therefore the maximal chance of herniation of the medial temporal lobe through the incisura, and the arterial pressure as a source of bleeding are the main reasons for the rapidity with which the entire syndrome can be completed.

Treatment. Treatment consists of early recognition, a temporal craniectomy, evacuation of the hemorrhage, and control of the bleeding artery either at the foramen spinosum or at the point of tear in the dura. The mortality from epidural hematoma remains approximately 50 percent, partly because of the difficulty in recognizing the syndrome and partly because even upon recognition rapid control of the hemorrhage is necessary to avert a fatal outcome.

Intracerebral Hematoma

Posttraumatic intracerebral hematomas are most often seen in patients with severe head trauma, often mimic the symptom patterns of an acute or subacute subdural hematoma, and frequently are found in conjunction with them. In some series of posttraumatic intracranial hemorrhage intracerebral hematomas are as frequent as subdural hematomas, especially if the series includes many patients injured in high-speed vehicle accidents. The hematoma usually presents either beneath a cortical laceration or as a confluence of small hemorrhages in a contused area of the brain. Intracerebral hematomas are most commonly found in the anterior third of the temporal lobe where the temporal lobe may strike the sphenoid bone, and less frequently in the tips of the frontal or occipital lobes. They can be seen in any area of the cerebrum. The symptoms of clinically significant posttraumatic intracerebral hematomas are similar to the symptoms of an acute or subacute subdural hematoma. There are many intracerebral hematomas that do not alter the clinical course of a patient, are not diagnosed, and resolve by liquefaction, phagocytosis, and gliosis. The surgically significant intracerebral hematomas present with progressive decrease in consciousness and/or progressive focal neurologic deficit. Most frequently, focal deficits are third nerve palsy and hemiparesis. These symptoms may develop within a few hours or a few days of the injury. An important concept is the fact that these hematomas can cause significant neurologic deficits in the head-injured patient in whom exploratory trephines have failed to reveal subdural hematoma. As with acute subdural hematomas, computerized axial tomography accurately diagnoses, localizes, and determines the extent of intracerebral hematomas. If a scanner is not available, arteriography is usually the best method to localize the hematoma, and ventriculography the next best. Occasionally an intracerebral hematoma is an incidental finding in surgery for acute subdural hematoma or is diagnosed by exploring the areas where occurrence is anticipated.

Treatment. The only effective treatment of surgically significant posttraumatic intracerebral hematomas is craniotomy or craniectomy with incision into the hemisphere at the most superficial area of the hematoma and evacuation of the clot. Simple aspiration of the lesion is not adequate since the majority of the hemorrhage is usually solid clot. The evacuation often must include excision of part of the lobe involved in order to remove devitalized brain and to obtain effective decompression of the injured cerebrum.

Rehabilitation. Rehabilitation of the head-injured patient starts with his initial care and finishes when his function is stabilized at its highest possible level. The rehabilitation of the brain-injured patient is a specialty in itself, but fortunately few patients require retraining or reconstructive surgical procedures to overcome their neurologic deficits. A majority of head-injured patients require only the support and care of a knowledgeable physician. Special problems of the head-injured patient during convalescence fall within three categories: psychologic problems, convulsions, and problems with cerebrospinal fluid circulation.

Psychologic Problems. Changes in personality and mood during the period of convalescence are one of the most common and often one of the most difficult symptoms for both patient and physician. The symptoms usually fall into the broad psychiatric category of a reactive depression with an overlay of anxiety. Complaints of headache, fatigue, loss of memory, excessive difficulty with performing tasks, transient despondency, and overreaction to emotional stimuli demonstrate a mixture of possible organic and psychologic bases for the symptoms. On the organic side is the suggestion of temporal and frontal lobe malfunction, while on the psychologic side is the reaction to physical and mental fatigue with a superimposed anxiety from the fear that the change may be permanent.

Gradually increasing physical activity in a planned fashion with limited goals that can be reached in reasonable periods of time and a planned return to increasingly difficult mental tasks is usually the best method for overcoming the problem. The settlement of any pending litigation is important when psychologic problems become paramount, for unnecessary delay can make a psychologic cripple of the patient. The physician should not abandon these patients or allow them to become dependent but rather insist that they get psychiatric help when necessary and that they work toward any physical and mental goals that seem possible. The best method of giving such support is for the physician to see the patient at planned intervals and during the visits to evaluate the neurologic progress of the patient to be certain that a portion of the problem is not coming from seizures or altered cerebrospinal fluid circulation.

Convulsions. Approximately 30 to 45 percent of patients with a compound injury that involves a laceration of the brain will have seizures if they are not on anticonvulsive medication. In contrast, the incidence of convulsions in the closed-head-injury patient is much lower. It is difficult to obtain any significant figures on the incidence of convulsion in the closed-head-injury patient, for in large series it varies from 0.5 percent to as high as 10 percent. In general it is safe to assume that a patient who has had less than 4 hours of either markedly altered consciousness or focal neurologic deficit will have no significant chance of convulsions developing late in the convalescence that relate to the head injury. In the group with more serious head injuries there remains a small but persistent percentage in whom convulsions will develop that appear to relate to the head injury. The period in which convulsions may develop extends over many years. It is often difficult to recognize that focal or partial seizures are occurring, but they always should be considered when any paroxysmal symptoms occur, that is, symptoms of relatively brief duration that end with excessive fatigue. Electroencephalograms may be of help in diagnosing the cause of such symptoms as seizures, but at times trials of anticonvulsants are necessary to rule them out. Control of posttraumatic seizures with Dilantin and phenobarbital is usually sufficient, but occasionally resection of the damaged area is necessary for control with anticonvulsant drugs.

Problems of Cerebrospinal Fluid. Alteration in cerebrospinal fluid circulation is probably common in the early convalescence after head injury and may accout for some of the complaints of headache, postural vertigo, and feelings of lightheadedness. These symptoms usually require no therapy other than the passage of time. Occasionally mild elevation of cerebrospinal fluid pressure continues and requires investigation by CT scanning or arteriography. If no mass lesion is found to explain the increased pressure, intermittent lumbar spinal drainage over a few days will often control this. Prolonged elevation of cerebrospinal fluid pressure with enlargement of ventricles is a rare but serious complication. Its cause is probably obstruction of the arachnoid villi or basal cisterns by blood. It usually presents weeks or months after the injury with a gradual dementia and motor apraxia or loss of complex coordinated motor movements. The treatment is to shunt the cerebrospinal fluid from the lateral ventricles to the superior vena cava or peritoneum using a pressure-regulating valve to maintain the cerebrospinal pressure at 50 to 70 mm of water.

In general, with patience, support, and guidance the head-injured patient will recover and return to useful existence. This may take many months and requires that both the physician and the patient not become discouraged. The degree of recovery of even the very seriously injured patients will justify for the physician the amount of effort and time that he must give to this group of patients.

Spinal Cord Injuries

In the treatment of spinal cord injuries the prime objectives are the preservation of neural function and, when possible, restoration of neural function, followed by the reestablishment of the integrity of the vertebral column and the rehabilitation of the patient.

Mechanisms of Injury. In contrast to skull injuries, in which fracture and brain damage may have an inverse relationship, bony vertebral damage and spinal cord injury have a high correlation, for the bony vertebrae and their attached soft tissue are usually the agents directly causing the spinal cord injury. The construction of the vertebral column with its circumferential bony ring provides ideal protection for low-velocity penetrating injuries or concussive blows, but the intervertebral articulations are weak points for flexion, extension, or rotary stresses. Dislocation and fractures that do not break the vertebral ring allow the vertebrae above and below the area of injury to act as a fulcrum for another vertebra and its attached soft tissue to concuss, stretch, contuse, or disrupt the spinal cord. The rarer fracture-dislocation that separates the posterior arch from the vertebral body demonstrates how much dislocation of the body can occur without permanent spinal cord damage and suggests methods of protecting the damaged cord, namely, laminectomy or removal of the posterior segments of the vertebrae. The stress of flexion, extension, and rotation and the relative weakness of the articulations of the vertebrae operate to make fractures and dislocations most common at points of junction between mobile and relatively fixed segments of the spinal column. These are the lower cervical region and the upper lumbar region, related tot he thoracic spine, that is relatively fixed by the ribs.

Most of the spinal cord damage occurs at the time of injury. However, recent studies of experimental spinal cord trauma in animals suggest a progressive hemorrhagic destruction of the central gray matter followed by edema in the white matter. Progressive damage to the white matter may occur during the first 4 to 6 hours after injury and plays a role in eventual

spinal cord dysfunction. The mechanism of the pathophysiology is not clear, but in the experimental animal cooling of the injured segment, steroids, and myelotomy have protective value and alter this progressive posttraumatic process. This suggests possible therapeutic approaches to the problem of spinal cord injury, and clinical studies are in progress. Secondary spinal cord damage can occur from further movement of the unstable vertebral column, the movement of the spinal cord against sharp fragments of bone in the canal, and continued compression of the spinal cord. This may be missed on routine spine films and detected by CT scan. Therapy to prevent any secondary damage to the spinal cord requires stabilization of the vertebral column, removal of any foreign body or fragments of bone from the spinal canal, and decompression of the spinal cord when necessary.

Reduction and Stabilization of Vertebral Column. In the cervical region stabilization of the vertebral column is most effectively obtained by skeletal traction, using skull tongs (Barton, Vinke, or Crutchfield tongs). Stabilization is achieved by anatomic reduction and by tension of the spinal ligaments and soft tissue of the cervical region. Slight extension of the neck will give tension to the anterior spinal ligament, the strongest of the spinal ligaments, and therefore is preferred unless there is evidence of rupture of the ligament. This is usually apparent from an x-ray that shows avulsion of the anterior lip of a vertebral body, the site of insertion of the ligament. Stabilization is promoted by placing the patient on a firm surface. A Stryker or Foster frame bed makes an excellent support, since the patient can be turned from the prone to supine position without changing the relative position of the spinal column.

Reduction of fracture-dislocations of the thoracic or lumbar spine should not be done unless the spinal cord or cauda equina is exposed by laminectomy before the reduction, since the force necessary for such closed reduction carries too high a risk of further damage to the spinal cord or cauda equina. Simple compression fractures of the body of the vertebra, with flexion angulation of the spine but without spinal cord deficit, may be gently extended by positioning in a Foster frame, utilizing extension to stretch the anterior spinal ligament and expand the vertebral body. A foreign body of bone fragment in the canal is a contraindication for any manipulative maneuver, since it may act as a disrupting agent during such maneuvers. The presence of foreign bodies adjacent to or in the spinal cord also allows possible continuing damage from any body movement or from the natural movements of the spinal cord with respirations and pulse. If any continuity of spinal cord function remains across such an area, laminectomy and removal of the fragments is indicated.

Decompression of Spinal Cord. The most difficult judgment in the early care of spinal cord injury is the decision to decompress the spnal cord. Some surgeons have solved this problem by always doing a laminectomy in any severe spinal cord injury, while others are firmly convinced that it is of value in only the rare case with later progression of neurologic deficit. The first group base their judgment on the concept that there is always edema and swelling of the spinal cord and surrounding tissue and therefore laminectomy with decompression is always of some potential value. Complications of such therapy have to be considered, and in unskilled hands they may be considerable. An argument used by the latter group is that severe injury of the spinal cord rarely can be reversed, since neuronal loss is complete within a short time of the injury and the life of the patient is placed in jeopardy by surgical therapy with little chance of helping spinal cord function.

Experimental evidence in animals demonstrates that the tracts of the spinal cord can tolerate many hours of anoxic compression and recover, even though the neurons within the anoxic area cannot. Since the total functional result is mainly dependent upon the tract traversing the segment, preservation of viability in these tracts is worthwhile. Evidence that there is compression consists of any progression in neurologic deficit, or a block of cerebrospinal fluid circulation. The Queckenstedt test consists of a measurement of lumbar cerebrospinal fluid pressure before and after compression of the jugular veins of the neck by a blood pressure cuff. If rapid rise and fall in the lumbar pressure does not occur with the compression and relase of the neck veins, the test shows evidence of a block in the cerebrospinal fluid circulation between the foramen magnum and the lumbar puncture needle. The Queckenstedt test is both inaccurate and dangerous, since an opening at the site of compression just larger than a spinal needle may give a negative test (no evidence of block) despite considerable compression, and increasing cerebrospinal fluid pressure above a block of the spinal canal can cause further spinal cord damage. For these reasons, when a compressive block is suspected, contrast myelography with air or metrizamide, usually placed in the spinal canal through a lateral C_1-C_2 spinal puncture, is indicated. With such evidence of block in cerebrospinal fluid circulation, laminectomy with opening of the dura is indicated unless rapid improvement in neurologic function is occurring. With trained neurosurgeons the morbidity and mortality of the procedure are far below the risk of allowing progressive spinal cord damage from further compression of the cord.

Compound Injuries. Trauma to the spinal cord may also result in open or compound injuries. The principles for the care of both closed and open injuries are not substantially different except that compound injuries require debridement and closure of the wound and they rarely have vertebral instability. Compound wounds of the spinal cord may occur from sharp penetrating objects but are most commonly from gunshot wounds. The concussive force of the bullet causes widespread damage and therefore more widespread reactive edema. If there is any possibility of cord continuity, early exploration, wide decompression, and removal of devitalized tissues both within and around the spinal cord are indicated in order to give the patient any significant chance of recovery. Without such surgical measures secondary compressive anoxia of the tracts is certain to occur.

Bony Union. Reestablishment of vertebral integrity requires time for bony union. If instability is marked, fusion is usually indicated. Marked instability occurs when the articulating facets and ligaments are disrupted or when there is some combination of injury to the vertebral body, pedicles, and facets. If open fusion is indicated, it is best to allow 2 to 3 weeks for reaction of injury within the cord to subside before performing a spinal fusion, since further reaction within the surrounding tissue occurs with such a procedure. Total immobilization of at least 6 weeks is indicated in most cervical injuries, with 6 to 8 weeks of support following this to ensure firm union. In other areas of the spinal column where weight-bearing stresses are greater, the time for sufficient union varies with the area and the type of bony injury but in general is longer than in the cervical area.

Rehabilitation. Rehabilitation of the patient with a spinal cord injury is a specialty by itself. The early care of the skin and bladder, however, can be as important for the time required for rehabilitation as any factor other than neurologic deficit. Following cord injury, spinal shock of varying degree occurs, and protective skin and bladder reflexes are lost. Support of the body is necessary so that the pressure points, such as the sacrum and the heels,

take as little weight as possible. A dry smooth covering against any weight-bearing skin, good nutrition, cleanliness, and, most important, turning the patient at least every 2 hours are the necessary nursing measures to protect the skin of the cord-injured patient. Initial constant catheter drainage of the bladder should be followed by intermittent catheter drainage plus high intake to protect the urinary tract from distension and infection.

Illustrative Case

Perhaps the easiest way to summarize the acute neurosurgical care of the cord-injured patient is to review the care of a patient who, immediately following an automobile accident, entered the hospital with weakness and sensory loss from the arms down. Initial examination revealed normal movement of the deltoid and biceps muscles, weakness of both triceps muscles, and only slight movement of the fingers, legs, and toes. Sensory examination showed preservation of deep pain and some touch sensation in both lower extremities but marked hypesthesia and hypalgesia from the ulnar side of the hands and arms caudad bilaterally. There were absent deep tendon and superficial reflexes except for the biceps reflexes. X-rays of the cervical spine were reported as negative.

The history and examination indicated a C_6-C_7 or C_7-T_1 vertebral column injury, and with this knowledge the x-rays were found to be not negative but inadequate, since they showed only the upper portion of C_6 and more cephalad vertebrae. Further x-rays showed a fracture dislocation with the C_6 vertebra anteriorly displaced on the C_7 vertebra. The patient was placed on a Stryker frame; the bladder was catheterized and connected to constant drainage. Barton tongs were applied to the skull, slightly anterior to the coronal plane traversing the mastoid tips, and 15 lb of weight was applied. Neurologic examination showed no change in function. X-rays showed no evidence of reduction. Five more pounds of weight was applied. Neurologic examination remained unchanged, and x-rays showed slight reduction of the dislocation. Five more pounds, or a total of twenty-five pounds, was applied, and neurologic examination remained unchanged, but x-rays showed reduction of the dislocation. Because of the lack of improvement, a myelogram was done and showed compression of the spinal cord at approximately the C_6 level. The patient was taken to the operating room for laminectomy, and at operation the laminae and facets of C_6 and C_7 were found to be fractured. The laminae and spinous processes of C_5, C_6, and C_7 were removed, and the dura was opened. The spinal cord appeared posteriorly displaced and swollen. Because closure of the dura appeared to cause constriction of the spinal cord, a fascial graft was placed for dural relaxation, and the dura, muscles, fascia, and skin were sutured with interrupted silk sutures. The patient made a gradual recovery over the next 6 months.

The points illustrated are the same as those discussed in the initial portion of this section. It is important that the area of damage be identified from the neurologic examination, so that the examiner is certain that x-rays of the spinal column include the area. When any manipulation is done, both the x-rays and the neurologic function should be checked. If despite reduction there remains evidence of cerebrospinal fluid circulation obstruction and there is no sign of neurologic improvement, decompressive laminectomy is indicated at that time, and the decompression should be complete, so that not even the dura binds the spinal cord.

Peripheral Nerve Injuries

The common types of peripheral nerve injuries are laceration, contusion, and stretch or compression, with or without disruption. The agents causing such injuries are numerous but from a therapeutic view can be divided into those causing a focal injury, such as a knife or a piece of glass; those causing relatively localized injury but imparting force to adjacent areas, such as a gunshot wound; and those causing damage to long segments of the nerve, as with stretch following a dislocation of extremity or compression from swelling under a rigid cast or dressing. Evaluation of nerve injuries includes accurate knowledge of the anatomy and function of the nerve suspected of injury, identification of the agent causing the injury, and recognition of the type of injury resulting. Treatment is based on the evaluation as well as knowledge of the factors influencing the comparative prognosis of the different forms of therapy available and the skill of the surgeon, not only to repair the nerve involved and the adjacent tissues, but also to utilize all reconstructive measures available.

Evaluation. The evaluation of peripheral nerve injuries is in part similar to the evaluation of central nervous system injuries, for comparison of serial observations and recognition of progressive neurologic deficit or improvement often determine the type of therapy required. The treatment differs from that for central nervous system injuries in that regeneration with reinnervation can occur and therefore the treatment of peripheral nerve injury includes neurorrhaphy, or the surgical repair of the nerve. Since comparison of later examinations with the initial one is important in therapy, it is essential to have an accurate examination that includes the sensory and motor function of all nerves thought to be injured. Peripheral nerve injuries often occur as part of more extensive injuries, and since an accurate examination can be done only on a cooperative patient, all patients suspected of nerve injury should be reexamined when well enough to cooperate.

Accurate knowledge of the anatomy and function of the nerve suspected of injury is a minimal requirement for a competent examination and evaluation. Nerve injuries are a relatively rare experience for the average physician; since the accuracy of even an experienced neurosurgeon can be enhanced by reviewing the anatomy and functions of nerves suspected of injury, the physician making the initial examination or treating the patient at any time should not hesitate to consult an anatomy book and review the anatomic relations and the motor and sensory function of the nerves involved. With this help accurate localization and accurate definition of the neurologic deficit can usually be made.

Treatment. The problem after diagnosis is what to do and when to do it. There are only a few alternatives for treatment of peripheral nerve injury: suture of the nerve, resection of damaged areas and resuture, or no treatment of the nerve but reestablishment of function of the part by utiziling other muscles and internal or external support.

A few axioms should be kept in mind. Complete functional loss in a nerve can occur without anatomic disruption, and functional return can occur within 60 to 90 days. This segmental loss of conduction in the nerve following trauma is caused by alteration in the ability of the axons to reestablish their membrane potential and is called *neuronapraxis.* The mechanism of recovery is not known, but the nerve recovers its function in all segments distal to the injury at approximately the same time, and therefore the recovery cannot be secondary to regrowth of the axons.

Although immediate repair gives the best functional results, little is lost by a delay of 3 to 5 weeks. After disruption and reapproximation of a nerve the axons require about 3 weeks to grow across the suture line and then grow at about 3 cm/month. In general, counting the delay across the suture line and the time for reinnervation of the muscle end plates or peripheral end organs, an estimated functional growth of 1 in/month is usually accurate. Following denervation, muscle end plates atrophy and muscle fibers progressively degenerate, so that within 20 to 24 months no significant muscle contraction can be expected from reinnervation. While muscle tone reappears and some movement will occur up to that time, significant strength in a muscle rarely can be regained past 15 months. Since functional nerve regeneration proceeds at approximately 1 in/month, a nerve suture that is more than 15 in from the denervated muscle cannot be expected to give significant motor return, and tendon transplants, joint fixation, or bracing should be considered as the treatment for the motor loss. Tone in facial muscles will return up to 24 months past denervation, but as the end of the 24-month period approaches, no movement of expression can be expected.

All nerve injuries are painful, and the pain will continue with varying intensity for many months. Narcotics have no place in the treatment of chronic pain and therefore should not be used after the immediate wound pain has ceased. Functional use of the extremity is the best therapy for the pain and is an important reason for using passive and active motion and functional bracing as soon as possible. It is also a major reason for not waiting for an improbable result, such as reinnervation of a muscle too distant from the point of suture, before using other reconstructive methods.

In contrast to the time limits on return of motor function, sensory function of a significant degree can return whenever reinnervation of the part occurs and often is of help in relieving the discomfort as well as in providing protection of an extremity. This is especially true of the protection offered by sensory innervation of the foot.

In almost all nerves, gaps of 8 to 10 cm can be bridged by proximal and distal dissection to release the nerve from surrounding tissue and by positioning of the nerve or the extremity to minimize the distance across joints. This may require wide dissection and positioning of more than one joint of the extremity. When neurorrhaphy is planned, skin preparation and draping should allow for such maneuvers. The problems with these maneuvers are that they may not allow closure of the ends of the nerve without tension, or may, after initial healing of the neurorrhaphy, allow tension and interference with blood supply of the regenerating nerve as the joints are extended. A recent microsurgical technique of perineural suture, ie, approximation of the perifascicular tissue of the nerve rather than closure of the epineural tissue, suggests improved distal growth both in number and rate of regenerating axons. The technique allows no tension at the site of closure, since very fine suture and low-tensile-strength tissue are used in the approximation. Interfascicular grafts, composed of homologous nerve, usually the sural nerve of the patient, interposed in a defect and sutured by these techniques, are reported to give better results than end-to-end epineural neurorrhaphy under tension. These grafts, by averting the mixture of interfascicular fibers and altered blood supply of a nerve under tension, appear to be an advance in nerve repair and have improved the expected results from some neurorrhaphies that had been done under tension and from some nerve grafts that had been required because of a long area of deficit. Interfascicular (perineural) neurorrhaphy allows better approximation and has reactivated interest in homologous nerve grafts.

Evaluation of Functional Recovery. In the evaluation of the injured nerve, evidence for functional recovery of the nerve is important, for it determines the success or failure of the therapy. Evidence that the nerve functions is not the same as functional recovery, for nerve function means only that some portion of the nerve has anatomic continuity. Definite evidence of functional recovery consists of:

1. Voluntary motor function of a muscle innervated by the nerve distal to the injury. This means that a major number of the motor fibers have reinnervated the muscle and that with time increase in strength will occur, either through increased strength of the individual reinnervated muscle fibers or by further innervation.

2. Muscle contraction on electrical stimulation of the nerve distal to the point of injury when performed more than 7 days after injury. Since the distal segment of a divided nerve will conduct impulses for only 5 to 6 days, evidence of ability to conduct later than 7 days after injury indicates functional continuity or regeneration. Muscle contraction following the stimulation means a major portion of the motor fibers are conducting. Direct electrical stimulation of the muscle does not give evidence of functional recovery, since contraction with this type of stimulation will occur until atrophy of the muscle is complete.

3. Recovery of sensation in an autogenous area of innervation. There are few such areas for individual nerves. The distal phalanx of the index finger for the median nerve and the distal phalanx of the little finger for the ulnar nerve are the only two in the upper extremity. The distal phalanges of the middle three toes of the foot are autogenous for the sciatic nerve but only if both peroneal and tibial divisions are involved.

Evidence of anatomic continuity but not of prognostic significance for functional recovery consists of:

1. Tinel's sign distal to the site of injury. Tinel's sign is elicited by percussing the nerve and obtaining paresthesias referred to the superficial area of skin innervation. It requires activation of only a small percentage of the fibers, far less than is required for functional recovery.

2. Alteration is electromyographic activity with attempted voluntary motion or nerve stimulation. As with Tinel's sign, the activation of far fewer fibers than is necessary for functional return of a muscle is required to elicit alteration in the recorded electromyographic activity.

Evidence that indicates neither anatomic nor functional continuity of an injured nerve consists of:

1. Shrinkage of the area of sensory loss. The marked overlap of sensory innervation in all areas of the body has been alluded to with the description of the very few autogenous zones of peripheral nerve innervation. Decrease in an area of sensory loss after nerve injury can occur from recovery of the nerve function, but even without reinnervation adjacent nerves reestablish sensation in the area. The mechanism appears to be both utilization of fibers which function with the damaged nerve but which without the spatial summation of the damaged

nerve activity do not reach consciousness, and branching of the superficial nerve fibers of the remaining nerves with growth into the denervated area.

2. Improved use of the extremity. The adaptability of the child to use any remaining function to accomplish coordinated tasks is a major example, but use of gravity, momentum, and muscles with remaining function to simulate the lost function must not be confused with reinnervation.

Illustrative Cases

The application of these concepts is most easily understood in relation to examples of the three major groups of peripheral nerve injury, laceration, or focal sharp injury, focal contusion, and compression or stretch injury.

Focal Laceration. An example of focal sharp injury is a knife wound of the upper extremity at approximately the midhumeral level, resulting in loss of median nerve function. Motor loss would consist of paralysis of all flexors of the wrist and fingers except the flexor carpi ulnaris and the ulnar portion of the flexor digitorum profundus. These latter two muscles would give wrist flexion with ulnar deviation and flexion of the ring and little fingers. Sensory loss would consist of loss of superficial sensation of the palmar surface of the hand extending to the thumb and first two fingers and the radial half of the ring finger. Deep as well as superficial sensation would be lost in the distal phalanx of the index finger.

Immediate repair is indicated *except* when any of the following apply: (1) The nerve appears contused for more than a few millimeters on either side of the injury. (2) There is blood loss from arterial injury that would jeopardize the life of the patient in undergoing a long (2- to 3-hour) operation for repair of the artery and nerve. (3) The wound is older than 5 to 6 hours, and dissection might spread contamination. (4) The surgeon is not certain of his ability to perform adequate neurorrhaphy.

If inadequate repair is done so that regeneration does not occur (no matter what the reason), it will take approximately 6 months, or the time it takes the nerve to grow 6 in to the flexor muscles of the forearm, before the inadequacy of the repair is recognized. Since the opponents of the thumb is beyond the distance where significant strength can be expected to be regained, as soon as the flexor muscles of the forearm are reinnervated, tendon transplant of one of the flexors to strengthen the apposition of the thumb is indicated.

Focal Contusion. The most common focal contusion of nerve is that secondary to a gunshot wound, and the treatment of a gunshot wound of the midthigh resulting in loss of sciatic nerve function is an example of such an injury. Immediate repair of the nerve is not indicated, since the damage extending up and down the nerve cannot be determined by inspection and after a high-velocity-missile injury it is always present in varying degrees.

The wound is treated to prevent infection, and the patient is fitted with a short leg brace for stabilization of the ankle. Stabilization is necessary, since motor function in all flexors, extensors, evertors, and invertors of the foot, as well as in the intrinsic muscles of the foot will be lost. The patient is ambulated with a brace as soon as possible and taught to inspect the skin of the foot three or four times a day in order to protect it from unrecognized

injury in the anesthetic area. Four to five weeks later, if no function has returned or if only minimal function in one portion of the sciatic nerve has returned, exploration is indicated. By this time fibrosis or neuroma formation will define the area of injury. Resection and resuture may be indicated. Three weeks after repair, when the anastomosis is secure, active use of the leg with the same precautions that were observed preoperatively should be started. Fifteen months later the stability of the ankle should be evaluated, and internal stabilization or arthrodesis may be indicated because of failure of adequate reinnervation.

Stretch or Compression Injury. The problem of the nerve with stretch or compression injury is more difficult. The segment of injury is often longer than can be bridged by any technique, and the loss is usually incomplete, so that even if the area might be bridged by surgical technique, comparison of the deficit from the injury with what might be obtained by resection and suture is essential in the decision for treatment. A common area of stretch injury is the brachial plexus, as might be seen following a shoulder injury. Careful recording of all sensory and motor loss is necessary, and if the pattern of loss suggests a radicular pattern, that is, loss of root function such as at C_5 and C_6, myelography or the fluoroscopic study of the spinal arachnoid space with radiopaque substance is indicated. Rupture of the arachnoid root sleeve with formation of a traumatic meningocele or pouching of the arachnoid through the intervertebral foramen may be demonstrated, indicating avulsion of the roots from the cord. Root avulsions are not repairable, and reconstructive measures such as fusion of the shoulder and triceps transplantation for elbow fixation are indicated rather than surgical exploration of the area of injury.

The possibility of surgical repair of compression or stretch injury is slight, since the area of injury is so long, and 2 to 3 months of evaluation are indicated to determine that the loss is not due to neuronapraxis, with spontaneous recovery occurring. In the brachial plexus injury loss of motion in the forearm would not be significantly improved by resection and suture, and loss of intrinsic muscles in the hands is not an indication for surgical intervention, since return of function cannot be expected. The usual early treatment indicated is protection of the denervated muscles from overstretching by a supporting brace, while later functional bracing, fusion of joints, and transplantation of remaining functioning muscles are the appropriate means of gaining maximum possible function.

Brain Tumors

Although uncommon in the experience of the average physician, brain tumors are not rare. As a clinical entity they are best brought into perspective by realizing that brain tumors constitute almost 10 percent of all benign and malignant tumors requiring hospitalization for surgical removal and that just under 1 percent of all deaths are caused by primary intracranial neoplasm. One reason that brain tumors seem to be uncommon in the experience of physicians is that they are not suspected as the cause of the patient's complaints, for the presenting symptoms are diverse and confusing, and their recognition can be difficult.

Classification and Characteristics. The classifications of brain tumors are at best confusing, partly because the naming of tumors has usually been on the basis of cellular characteristics. The difficulties of such classifications arise not only from the many different names that can be applied to the same tumor in different situations but also from the difficulty of describing the different characteristics of even the same cell type within a tumor. Clinically

a useful classification of brain tumors is the one proposed by Kernohan and Sayre based on naming the tumors for the cells present in the adult nervous system, vascular tissue, and developmental defects, combined with a grading of the malignancy of the tumor from grade I to grade IV, with IV the most malignant (Table 42-3).

Table 42-3. Brain Tumors

Gliomas	40-50
Astrocytoma, grade I	5-10
Astrocytoma, grade II	2-5
Astrocytoma, grades III and IV (glioblastoma multiforme)	20-30
Medulloblastoma	3-5
Oligodendroglioma	1-4
Ependymoma, grades I-IV	1-3
Meningioma	12-20
Pituitary tumors	5-15
Neurolemmomas (mainly eighth nerve)	3-10
Metastatic tumors	5-10
Blood vessels tumors	
Arteriovenous malformations	
Hemangioblastomas	
Endotheliomas	0.5-1
Tumors of developmental defects	2-3
Dermoids, epidermoids, teratomas	
Chordomas, paraphyseal cysts	
Craniopharyngiomas	3-8
Pinealomas	0.5-0.8
Miscellaneous	
Sarcomas, papillomas of the choroid plexus, lipomas, unclassified, etc	1-3

Certain types of tumors are more frequent at certain ages and occur with greater frequency in those ages in different areas of the brain. For example, 70 percent of adult tumors are supratentorial, that is, in the middle or anterior fossa, while 75 percent of childhood tumors occur in the posterior fossa. The most common primary tumor of the brain in the middle-aged and elderly is a grade III or grade IV astrocytoma (glioblastoma multiforme), while the most common tumor of childhood is the grade I and II astrocytoma of the cerebellum. Malignant astrocytomas are by far the most common primary brain tumor in adults, followed by meningiomas, pituitary tumors, and neurilemmomas, while in childhood the most common tumors are the relatively benign astrocytomas of the posterior fossa, followed very closely by the highly malignant medulloblastomas, and then by ependymomas and craniopharyngiomas. Tumors of childhood occur characteristically near the midline of the brain, as with the tumors of developmental defects, the medulloblastomas, and the astrocytomas of the brainstem and cerebellum. Gliomas in adults occur at a frequency that is related to the volume of the brain itself and therefore are more common in the cerebral hemispheres. Meningiomas have a predilection for areas of arachnoid villi and arachnoid invaginations that contain cells similar to those seen in the meningioma. These areas of

predilection are along the sagittal sinus, the sella, the olfactory groove, the tentorium, and the petrous ridges.

Pathophysiology. The basic pattern is one of progressive neurologic deficit. This deficit can be a progressive focal deficit or a progression in the neurologic dysfunction that occurs secondary to increased intracranial pressure. Since these clinical patterns are a continuum, the historical examination of the patient must include the factor of time and its relationship to the development of the symptoms and signs. Their development may vary within the basic patterns because of the different mechanisms by which tumors disturb normal function, the different positions the tumor may occupy within the skull, and the diverse growth potentials of the different tumor types. These variables, when combined withthe concepts discussed in the opening portion of this chapter, namely, the anatomic confinement within compartments of the skull of a deformable but relatively noncompressible brain and the changes in neural function that occur from compression of the brainstem passing through the incisura of the tentorium and the foramen magnum, explain in part the diversity of the presenting symptoms produced by brain tumors. With so many combinations of variables possible, learning the symptoms for the different types of tumors intheir different clinical stages is almost an endless task unless it is related to functional anatomy of the brain, the mechanisms used by tumors in producing signs and symptoms, and a classification of the tumors by frequency of occurrence and growth potential.

Tumors of the central nervous system manifest themselves most frequently by effects caused by one or more of the following mechanisms:

1. Compression of neural tissue, either direct or secondary to displacement of the brain.

2. Infiltration or direct invasion with destruction of neural tissue.

3. Alteration in the blood supply to neurons.

4. Alteration in neuronal excitability.

5. Increase in mass within the skull.

6. Alteration in cerebrospinal fluid circulation.

Clinical manifestations. Direct compression of neural tissue produces a progressive focal neurologic deficit which is the most easily recognized sign of brain tumor. The deficit will vary depending upon the area of the brain involved and is usually partial rather than complete. Thus, hemiparesis rather than hemiplegia is more frequently seen, and sensory loss is more commonly a partial rather than a total deficit.

Progressive focal neurologic deficit can be produced by other mechanisms, such as direct destruction of neural tissue by malignant cells and loss of neural function by alteration in local blood supply. Direct destruction of neural tissue by involvement of malignant cells is uncommon, but alteration in local blood supply does occur and particularly may be seen in the older patient with a highly malignant glioma. Interference with arterial blood supply

by tumor usually manifests itself as an acute loss of function, suggesting primary vascular disease, and only the later focal progression from increasing mass effects, or further involvement of local blood supply, competes the pattern of progressive focal neurologic deficit, indicating the diagnosis of brain tumor.

Compression, invasion, and altered blood supply may also cause altered neuronal excitability which is manifested by convulsions. Convulsions are paroxysmal episodes of uncontrolled neural activity, and since the neurons of the cerebral cortex have the lowest threshold for such paroxysmal activity, seizures caused by tumors are most commonly seen in lesions adjacent to or within the cerebral cortex. A focal or a Jacksonian seizure and especially a postictal neurologic loss (Todd's paralysis) indicate the localization of the lesion, and progression in either frequency or severity of the seizures or in the postictal neurologic deficit is strongly suggestive of a brain tumor.

The signs and symptoms of generalized increase in pressure are cloudy mentation and consciousness, headache, papilledema, vomiting, bradycardia, and systolic hypertension. It is rare to have all these findings except as terminal events. Increased pressure can also produce abducens, or sixth cranial nerve palsy, which may have no localizing value, for the abducens nerve is particularly prone to loss of function secondary to increased intracranial pressure. Increased intracranial pressure from brain tumors can result from mass increase, impaired cerebrospinal fluid circulation, or a combination of these.

The increase in mass may result from a number of factors. There is the neoplastic growth itself and edema formation. Malignant tumors especially produce edema in the adjacent brain. The cause of this edema is not understood, but it can result in massive swelling at considerable distances from the tumor. Some tumors form cysts that have osmotic gradients that cause an enlargement of the cyst by absorption of fluid. Infrequently rapid enlargement may result from hemorrhage within a tumor. Changes may occur in the surrounding nervous tissue, such as edema from venous obstruction or edema from breakdown of the integrity of the blood-brain barrier, secondary to either arterial or venous insufficiency.

The increased intracranial pressure caused by alteration in cerebrospinal fluid circulation usually occurs from obstruction of the passage of cerebrospinal fluid from the lateral ventricles to the subarachnoid space. This can occur when tumors arise within the ventricular system. Obstruction in the flow of cerebrospinal fluid may also occur when there is a shift of the brainstem so that the aqueduct of Sylvius is compressed or the outlets of the fourth ventricle are blocked. Tumors in the basal cisterns may also obstruct cerebrospinal fluid flow.

There are compensatory intracranial mechanisms for altering the effects of increased mass and pressure. Most of them require days or months to be effective and therefore are best utilized in controlling pressure from a slowly growing, benign tumor. These mechanisms include decreases in intracranial blood volume, cerebrospinal fluid volume, intracellular fluid contents, and parenchymal cell numbers.

Evaluation. The primary necessity in the diagnosis of brain tumor is the suspicion which should be initiated by the patient's history of a progressive neurologic deficit. The more knowledgeable the physician is of the functional anatomy of the brain, the more accurately

he will recognize progressive neurologic deficit. While hemiparesis and hemisensory loss or lack of motor function in a cranial nerve are easily recognizable, slow progressive dementia, decreasse in pituitary function, or progression in the severity of seizures are more difficult neurologic deficits to recognize as indicating brain tumor.

It is beyond the scope of this section to review the diagnostic investigation that should be completed in evaluating a patient suspected of having a brain tumor, but complete physical and neurologic examinations are the minimal requisites to the use of special diagnosti tests that have been described in the opening section of this chapter. CT scanning has become the major method of diagnosing brain tumors, replacing air studies and other scanning techniuqes. Arteriography is used as an aid in making surgical decisions and, in combination with CT scanning, aids considerably in the final judgment of what should be done with a brain tumor.

Treatment. The primary goal of tumor therapy is cure, and surgical excision is the method most likely to accomplish a cure. Unfortunately, in the case of brain tumor surgical removal is not always possible or even desirable, since the resulting neurologic deficit may leave the patient so impaired that saving his life is of no significance. The treatment of brain tumors therefore is not just their surgical removal, and it requires experience and knowledge combined with surgical skill in order to obtain the maximal functional result. At times the repetitive use of these special diagnostic tests and evaluation over a period of time are necessary to be certain of what would be the optimal therapy. As often as possible total removal is attempted, but when this is deemed impossible, palliation by partial remova of the tumor for internal decompression, relief of intracranial pressure by shunting of blocked spinal fluid, or treatment of the tumor by x-ray therapy and/or chemotherapy may control the tumor or give significant relief and allow the patient extended periods of normal existence. Combinations of these palliative and curative techniques allow a large percentage of patients to obtain significant relief of symptoms or cure of the brain tumor.

Pituitary Tumors

Pathology and Pathophysiology. Pituitary tumors are often classified as brain tumors since the pituitary is an intracranial structure, and their mass effects cause neurologic loss. The tumors are rarely of neurotissue origin since only the posterior lobe is composed of neural tissue, and the vast majority of pituitary tumors are adenomas inthe anterior lobe. Pituitary tumors are almost always benign and cause symptoms by overproduction of hormones, mass effects, loss of anterior pituitary gland function, or a combination of these mechanisms. Formerly, pituitary adenomas were classified by the histologic affinity to anyline dyes, with chromophobe adenomas having little or no affinity, eosinophilic adenomas having an affinity for acid dyes, and basophilic adenomas having an affinity for basic dyes. They are better classified as endocrine-active or -inactive, with identification of the hormone or hormones produces, and described by their size or growth characteristics since both methods correlate with the symptoms they produce and relate to the therapy effective in their control.

Endocrine-active tumors may produce prolactin (PR), growth hormone (GH), adrenocorticotrophic hormone (ACTH), thyroid stimulating hormone (TSH), or luteinizing and follicular stimulating hormone (LSH-FSH). Prolactin-secreting adenomas are the most common endocrine-active tumors, followed by growth hormone and the much less frequent ACTH-producing tumors. TSH- and LSH-TSH-producing tumors are extremely rare.

Endocrine-inactive tumors are the second most common pituitary adenoma, occurring with frequency between prolactin- and growth hormone-producing tumors. The other description of pituitary adenomas is on the basis of size and/or evidence of invasive growth. Tumors less than 1 cm in diameter are called microadenomas, while those greater than 1 cm are called macroadenomas. The latter may have suprasellar or parasellar extension, and when they invade the skull base and paranasal sinuses or the cavernous sinus, they are termed invasive adenomas. Thus, a patient may have a prolactin-secreting invasive macroadenoma with suprasellar extension, signifying a large endocrine-active tumor that produces prolactin that has destroyed part of the base of the skull or invaded the cavernous sinus with extension into the sella.

Diagnosis. The diagnosis of pituitary tumor should be considered when endocrine symptoms and/or parasellar neurologic deficits are found by history or examination. The endocrine symptoms depend on the hormone produced and/or destruction of the anterior pituitary. Rarely do pituitary tumors cause the symptom of posterior pituitary lobe dysfunction of diabetes insipidus except when the suprasellar extension damages the hypothalamus. The commonest parasellar neurologic deficit is caused by compression of the crossing fibers of the optic chiasm that arise from the nasal portion of the retina causing a bitemporal visual field loss (bitemporal hemianopsia). Depending on the size and rapidity of growth of the tumor, optic nerve dysfunction, or frontal or medial temporal lobe signs and symptoms may occur. As with central nervous system tumors, recognition of a progressive endocrinopathy that includes more than one system, such as hypothyroidism followed by hypogonadism and adrenal insufficiency, should suggest a pituitary tumor.

The most common endocrine symptoms result from an excess production of prolactin. In the female the excess production causes menstrual irregularities or amenorrhea, infertility, and galactorrhea. The same hyperproduction in the male causes decrease in potency and fertility, and since these symptoms are often not reported or recognized, the prolactin tumors of male patients are often larger, causing hypopituitarism and neurologic deficit before being diagnosed.

The next most common endocrine symptom is hypopituitarism, which can be caused by endocrine-inactive tumors and large endocrine-active tumors. The second most common endocrine-active tumor produces growth hormone. Excess growth hormone produces gigantism in the young and acromegaly in the patient who has finished growing. Acromegaly literally means enlargement of the distal parts, eg, hands, feet, jaw, nose, etc. Excess growth hormone also alters glucose and fat metabolism, and the patients have a high incidence of diabetes mellitus and atherosclerosis along with changes in facial features and enlargement of the hands and feet. This alteration in glucose and fat metabolism shortens their life span because of cardiovascular and cerebrovascular disease. Patients with pituitary tumors that produce growth hormone have a high incidence of elevated prolactin, and females with acromegaly may have galactorrhea and menstrual dysfunction. Both prolactin-secreting tumors and growth hormone-secreting tumors can be diagnosed by direct radioimmunoassay of the circulating levels of the respective hormone, and the size of the tumors can be determined by x-ray studies of the sella including polytomography and CT scanning.

An infrequent tumor of the anterior pituitary lobe is an ACTH-producing tumor that produces Cushing's disease, that portion of Cushing's syndrome of hypercortisolism caused

by hypothalamic pituitary dysfunction. Recent series of Cushing's disease indicate that very small, 1 to 2 mm in diameter, or larger adenomas of the pituitary are the etiological factor of the disease in 85 to 90 percent of patients. The smaller tumors may be difficult to demonstrate by x-ray or other studies, but with the low morbidity and mortality of a transsphenoidal approach to the sella, many surgeons believe exploration of the sella, with a search for such tumors, is indicated when Cushing's disease is diagnosed despite lack of x-ray evidence of the presence of pituitary tumor. Hypercortisolism causes hypertension, obesity, hirsutism, and abdominal stria as well as interferes with calcium metabolism and resistance to infection. The average length of life of a patient with severe Cushing's syndrome is approximately 5 years, and thus radical treatment such as exploration of the sella or adrenalectomy is indicated.

TSH-producing and LSH-FSH-producing pituitary adenomas usually are seen in patients with thyroid gland or gonadal insufficiency and often will respond with decrease in size when the hormone deficiency is replaced with exogenous hormone. Prolactin is the only anterior pituitary hormone that is mainly under a suppressive effect by the hypothalamic neurons. The inhibitory factor is either dopamine or a dopamine-like substance, and dopamine agonists such as bromocryptine decrease the production of prolactin and often decrease the size of prolactin-secreting tumors of the pituitary. Bromocryptine also can suppress reduction of growth hormone in growth hormone-producing tumors but has been less successful in decreasing the size of growth hormone-producing tumors. The success of bromocryptine in treating pituitary tumors, both the endocrinopathy and mass effect, has led to the search for other drugs that may control pituitary tumors, but the major effective therapies remain surgery and x-ray therapy.

Treatment. In the past few decades, the reestablishment of the transnasal and transsphenoidal operation for tumors of the sella turcica combined with microsurgical techniques and the operating microscope has markedly decreased the mortality and morbidity of pituitary surgery and allowed the removal of microadenomas without damage to the remaining pituitary gland. In larger tumors, it has allowed the removal of the major mass without the risk of craniotomy, and when this surgery if followed by x-ray therapy, it has allowed control of the pituitary adenoma in 80 to 90 percent of patients for as long as 10 years.

In many autopsy series, 20 percent or more of pituitary glands studied contained adenoma, and as far as can be ascertained from the hospital charts, without premortem endocrine symptoms. This may indicate that many pituitary adenomas have little or no growth potential and result from the activity of the hypothalamic pituitary releasing substances, aging, and/or the propensity for all glandular tissue to form adenoma. No technique has been devised that can differentiate between pituitary adenomas that have growth potential and those that do not. The high incidence of adenoma formation in the pituitary, along with clinical experience, indicates that the presence of pituitary adenoma which is not causing endocrinopathy is probably not an indication for surgical removal, and since a clinically significant adenoma may require 5 to 10 years to develop symptoms, there is need for long-term follow-up of any patient suspected of or diagnosed as having a pituitary adenoma.

Summation. In summary, it is the evidence of a progressive neurologic deficit that should raise the suspicion of diagnosis of a brain tumor, for progressive neurologic deficit is

the most common presenting symptom of brain tumor. This deficit may be of a focal nature which varies according to the area of the brain involved or may be a more generalized type secondary to increased intracranial pressure. The focal neurologic deficit may be a loss of function or can be altered excitability. Loss of neural function secondary to brain tumor is most frequently incomplete, and altered excitability usually presents as a convulsion. The effects of generalized increased intracranial pressure can be secondary to increased mass, alteration in cerebrospinal fluid circulation, or a combination of these. The deficits seen in brain tumor have a common denominator, that is, evidence of progression in symptomatology and signs over a period of time. A knowledge of the functional anatomy of the brain, classification of brain tumors, their frequency of occurrence and growth potential, and the mechanisms by which tumors manifest themselves will facilitate a high degree of accuracy in suspecting brain tumors, thereby allowing the physician to have the aid of the specialist in diagnosing and treating the tumor.

Spinal Cord Tumors

The term *spinal tumors* includes all tumors encroaching on the spinal cord. Primary tumors of the spine are approximately one-sixth as common as intracranial tumors but have a much better prognosis, since almost 60 percent are benign and a high percentage of the remainder respond to therapy, so that prolonged functional palliation can be expected. Metastatic spinal tumors are reported to present as often as primary spinal tumors. Any malignant tumor may metastasize to the bones of the spinal column or to the spinal epidural space, but the most common are from lung, breast, lymphoid tissue, prostate, kidney, and thyroid. Metastatic spinal tumors usually cause pain, followed by neurologic deficit. Since these tumors rarely cause death, palliation by surgical and radiation therapy is indicated in order to preserve neurologic function and decrease suffering during a terminal illness.

Classification and Characteristics. The symptoms, the type of tumor, and the prognosis correlate with where the tumor is in relation to the dura and spinal cord. Therefore, the classification of tumors into extradural and intradural groups and the subdivision of the intradural group into extramedullary and intramedullary has significance from both a clinical and pathologic point of view. Ninety percent of extradural tumors are malignant, while sixty percent of intradural tumors are benign. Seventy-five to eighty percent of extradural tumors are metastatic, while ninety-eight percent of intradural tumors are primary tumors. The common clinical course of extradural tumors is one of rapid compression of the spinal cord, either by the tumor itself or from collapse of involved vertebrae. The patient may present with rapidly progressing flaccid paraparesis and sensory loss that requires rapid recognition and surgical decompression in order to have significant palliative effect. The common clinical course of the intradural tumor is usually much slower and may extend over months or years with the patient presenting with spastic paraparesis and partial sensory loss.

Knowledge of the types of tumors, their potential for growth, and their mechanisms of production of symptoms is the means by which the finer aspects of diagnosis of spinal cord tumor are best attained, but the clinician should always consider the diagnosis of spinal cord tumor when any bilateral progressive neurologic loss occurs below any transverse level of the body. The value of such constant suspicion is the high degree of functional return and the high percentage of cure or palliation of symptoms resulting from the early surgical treatment.

Evaluation. A suspected spinal cord tumor is localized by utilizing the findings of the neurologic examination, the bony changes in spinal x-rays, and the results of spinal contrast studies, or myelography.

Clinical Manifestations. The diagnostic neurologic examination findings are of two types, local signs and tract signs. The local signs are segmental changes and localize the lesion along the rostral-caudal axis. The local motor signs are weakness and loss of reflexes with normal above and normal or hyperactive reflexes below the level, fasciculations over a segmental distribution, and atrophy in a myotome pattern. These indicate involvement of the anterior motor horn cells or the anterior spinal roots. Local sensory signs are localized pain and spinal tenderness, radicular or radiating pain, and loss of sensation over a dermatome pattern, indicating involvement of the dorsal root or dorsal root entry zone. Segmental loss of pain and temperature with preservation of touch (sensory dissociation) is also a local sign and indicates involvement of the crossing fibers in the anterior commissure of the spinal cord from a central lesion of the cord.

Tract signs only indicate that the lesion must be cephalad to the highest point of involvement, and the motor changes may vary from increased tone and reflexes to flaccidity with absent reflexes. The difference usually relates to the rapidness of onset, since with rapid loss of spinal cord function spinal shock with flaccid, areflexic paralysis may result whereas spastic paraparesis is more common with the slow, progressive loss of motor function. The other tract signs may relate to involvement of any portion of the spinal cord and thus can consist of loss of pain and temperature (anterolateral fasciculus); loss of position and vibratory sense (dorsal column); weakness, spasticity, hyperreflexia, and Babinski signs (posterolateral fasciculus).

Diagnostic Studies. Radographs are of diagnostic aid if they are taken of the correct area. When doubt exists or when there is a question of multiple levels, the entire spinal column should be visualized. Diagnostic findings in the radiographs relate to the type of tumor present. With malignant tumors areas of invasion and destruction, particularly of the vertebral body and pedicles, may give localization. With benign or slowly growing tumors, widening of the interpedicular and anteroposterior diameter of the neural canal, erosion of a pedicle or of a body, and enlargement of the intervertebral foramina are diagnostic.

Definitive localization, however, is done by meylography. It is usually best to do the lumbar puncture and obtain fluid, at the time that myelography is done. Prompt surgical intervention may be required with lumbar puncture or myelography, since increase in deficit may follow removal of spinal fluid below the level of the lesion. For this reason none of these should be done unless patient is in an area where definitive neurosurgical care is available and the patient is under the observation or care of a neurosurgeon.

Extradural Tumors

The different types of patterns of symptoms and the local and tract signs are best remembered in relation to the different types of spinal cord tumors, namely, extradural, intradural-extramedullary, and intradural-intramedullary.

Aside from a rare extradural meningioma, neurofibroma, and benign osteoma of the vertebra, extradural tumors are malignant with rapid growth and a tendency to destroy the spinal column. The symptom pattern of the extradural tumor is one of pain, both localized to the area of involvement and radiating over the dermatome level of the adjacent spinal roots (local sign). This is folloed by rapidly progressive transverse loss of spinal cord function (tract sign). The local pain is frequently more severe at night and at rest, while the radicular pain relates to movement, coughing, and straining. Both types of pain may be present for weeks or months before spinal cord involvement. The progressive transverse loss of spinal cord function can be as rapid as a few hours or as slow as a few days, and effective palliation is possible only if surgical decompression is done before total loss of spinal cord function has occurred.

Since these tumors frequently involve the adjacent vertebrae, x-rays of the spine with particular attention to the integrity of the vertebral body and pedicles will often suggest the diagnosis before as well as after the onset of progressive neurologic deficit in the spinal cord. Decompression by removing the posterior neural arch or laminectomy followed by x-ray therapy gives the best palliative result in this malignant group of tumors, and laminectomy with excision of the lesion is the treatment of choice for the few benign tumors.

Intradural-Extramedullary Tumors

Approximately 65 percent of all intradural tumors are extramedullary, and 90 percent are either neurofibromas or meningiomas. Neurofibromas are slightly more common and often are multiple. They have no sex predilection and are more common inthe cervical and thoracic areas, while meningiomas are more frequent in females and more common in the thoracic region. It has been stated that the frequency of occurrence of both these tumors in the thoracic areas is in the same ratio as the length of the thoracic spine to the length of the spinal column.

Angiomas are also common in the thoracic area. They are arteriovenous malformations and not true tumors but may at times present like an extramedullary tumor. This is particularly true during the last trimester of pregnancy. Other tumors that occur in the extramedullary-intradural area are epidermoids, dermoids, and lipomas. The basic pattern of symptoms of the extramedullary-intradural tumor is similar to the extradural tumors, namely, local pain and radicular pain (local sign), followed by progressive spinal cord malfunction (tract sign). The course is slower, and the development of the spinal cord deficit is often prolonged so that partial patterns of loss are seen. A common partial pattern is the modified Brown-Séquard pattern in which loss to temperature and pinprick is more marked on one side below the level of the lesion and weakness and spasticity are more marked on the opposite side (tract signs).

Intramedullary Tumors

Over 95 percent of intramedullary tumors are gliomas. In contrast to intracranial gliomas they tend to be more benign histologically and to have a more benign course. This is in part because of the low grade of their malignancy and in part because of their frequently nonlethal position in the spinal cord. Except for the preponderance of ependymomas occurring in the conus medullaris and the filum terminale, they occur equally frequently in all areas of

the spinal cord. Approximately one-half of intramedullary tumors are ependymomas, and 45 percent are astrocytomas, with a few oligodendrogliomas, hemangioblastomas, and ganglioneuromas making up the remainder. Arterial venous malformations and syringomyelia may present as intramedullary spinal cord tumors but are developmental abnormalities and not neoplasms.

The basic pattern of symptoms for the intramedullary tumor is different from the extradural and extramedullary tumor in that radicular or radiating pain is rare and the complaint of local pain is uncommon. These tumors tend to grow into the central part of the spinal cord and to destroy crossing fibers and neurons of the gray matter. The destruction of crossing fibers causes segmental loss of pain and temperature sensation with preservation of touch (sensory dissociation), resulting in damage to peripheral skin areas because of lack of protection of pain and temperature sensation (local sign). Trophic changes with alteration in vasomotor control and sweating may cause symptoms that resemble Raynaud's phenomenon or erythromelalgia. Alteration in the function of descending motor pathways or ascending sensory pathways is usually late in appearance, but alteration in sexual function and bladder function may occur early (tract sign). Segmental lower motor neuron destruction is common, and the signs of such destruction, namely, weakness, atrophy, loss of reflex, and fasciculations, should always be searched for in these patients, since they have localizing value (local sign).

Summation. In summary, spinal cord tumors have a good prognosis if treated early. It is important to keep in mind that a progressive decrease in spinal cord function must always be considered to indicate a spinal cord tumor until proved otherwise. Early diagnosis, accurate localization, and surgical therapy can be expected to give a large percentage of cures with good functional result and in the remainder to give considerable and prolonged palliation of symptoms.

Vascular Diseases of the Nervous System

Etiology. Vascular disease constitutes a set of major causes of neurologic morbidity and mortality spanning several pathologic categories. Congenital lesions are represented by saccular aneurysms which appear and enlarge consequent to defective muscular and elastic layers at vessel bifurcations, and by arteriovenous malformations that result from failure of formation of a capillary bed between segments of the arterial and venous circulations. Infectious and inflammatory conditions of the vessels of the central nervous system occur. These include arteritis and phlebitis accompanying chronic meningitis, thrombophlebitis or dural venous sinus thrombosis accompanying subdural and epidural infections, mycotic aneurysms secondary to septic emboli, and the less specific arterities as exemplified by temporal arteritis. The degenerative or metabolic disease complex subsumed by the term "atherosclerosis" constitutes one of the characteristic afflictions of twentieth-century humans. Brain ischemia or infarction due to thrombosis of involved vessels or their occlusion by emboli from vessels more proximal with diseased endothelium constitutes a frequent form of this disease. A less frequent but even more catastrophic form of the disease is the intracerebral hemorrhage that can occur with the same basic set of conditions upon which has been superimposed an elevation in blood pressure.

Anatomy. The major arterial supply to the brain consists of paired internal carotid and vertebral arteries. The internal carotid arteries begin in the neck at the division of the common carotid artery into the external and internal carotid arteries. The internal carotid arteries can be divided into cervical, petrous, cavernous, and intradural portions. The cervical portion has no branches, but the remaining portions have branches that can be significant in collateral blood supply to the brain following obstruction in the internal or common carotid artery. Branches of the petrous portion that anastomose with the internal maxillary artery are the caroticotympanic and pterygoid canal arteries. These are relatively small branches and rarely have significant anastomotic capacity. In the cavernous portion are branches to the cavernous sinus, the hypophysis, semilunar ganglion, meninges, and orbit. The semilunar and meningeal arteries can have significant anastomosis with the meningeal branches of the internal maxillary artery, and the ophthalmic artery is commonly a significant anastomotic channel with the terminal branches of the external maxillary artery. The cavernous sinus branches may be a source of communication between the carotid artery and the cavernous sinus, a carotid cavernous fistula, when they are ruptured by trauma or disease. The intradural branches of the internal carotid artery are the anterior cerebral, the middle cerebral, the posterior communicating, and the choroid arteries. The posterior communicating artery forms an anastomosis with the posterior cerebral artery, and the anterior cerebral artery with the anterior communicating and the opposite anterior cerebral artery to complete the anterior portion of the circle of Willis. The vertebral arteries in their cervical or extradural portion have anastomotic branches with the thyrocervical trunk of the subclavian artery and with the posterior branches of the external carotid. After traversing the dura, they join to form the basilar artery giving branches to the brainstem. The division of the basilar artery into the posterior cerebral arteries completes the posterior portion of the circle of Willis.

There are, therefore, extensive anastomoses between the vertebral basilar system and the carotid system. The collateral circulation can be divided into the extracranial, extracranial-intracranial, and intracranial. The extracranial anastomoses have been described in the paragraph above but, in general, consist of posterior branches of the external carotid and the vertebral and the thyrocervical trunk of the subclavian, the internal maxillary branch of the carotid through the meningeal branches of the internal carotid, and between terminal branches of both external carotid arteries. Most of these anastomoses are normally small but have the capacity to enlarge following proximal or distal occlusion of a carotid or vertebral artery. For example, after obstruction of the cervical common carotid artery below the bifurcation, anastomosis through the external carotid arteries can result in retrograde flow in the ipsilateral external carotid artery to its origin and consequent forward flow in the external carotid artery to the brain, bypassing the obstruction. Extracranial-intracranial collaterals are principally by anastomosis of the external carotid artery to the intracranial circulation by the ophthalmic artery and by collaterals developed between meningeal vessels and the intracranial arteries of the pia on the convexity of the cerebral hemispheres. The major intracranial anastomosis is the circle of Willis described above. In only 18 percent of brains is the circle of Willis fully developed, there being various degrees of hypoplasia in one or more vessels in the majority of cases. Extensive end-to-end anastomoses of arterial branches of the anterior, middle, and posterior cervical arteries have been demonstrated by arteriography following occlusion of major intracerebral vessels. Although these anastomoses are, in general, not present in the normal state, they develop rapidly following vascular compromise and may be, in part, the reason for neurologic recovery following cerebrovascular accidents secondary to occlusion of a major vessel. All the anastomoses, but particularly the larger extracranial-intracranial and

intracranial anastomoses, play a significant role in the protection of the brain against neurologic dysfunction in vascular disease and major occlusion.

Physiology. Intracranial circulation is dependent upon many factors, but the main determinants are arterial and venous pressure at the level of the brain, intracranial pressure, blood viscosity, and the state of the vascular bed and its resultant resistance. The brain is one of the most metabolically active organs in the body, requiring a constant supply of oxygen and glucose and, therefore, constant blood flow containing these substances. Intracerebral blood flow has been calculated by several techniques with variations between 44 and 80 mL/100 g of brain/minute, but the generally accepted value in the adult normal brain is approximately 50 to 55 mL/100 g of brain/minute, or approximately 750 mL of blood/minute in the adult. Thus, the brain, representing only 2 percent of the body weight, ordinarily receives between 17 to 18 percent of the heart's output and consumes nearly 20 percent of the oxygen supply of the body. Gray matter blood flow has been measured at 80 mL/100 g of brain/minute, while that of the white matter is 20 mL/100 g of brain/minute or roughly one-fourth of the gray matter, indicating further the metabolic activity of the two areas.

There are several intinsic factors regulating the cerebral blood flow which are unique to the cerebral vasculature, and extrinsic factors which relate to blood flow and its constituents, both of which tend to keep cerebral blood flow and availability of oxygen and glucose constant despite wide variation in systemic flow to other areas of the body. The intrinsic factors are referred to by the term *autoregulation.* In experimental animals with normal autoregulation of the cerebral vessels if the parameters of PCO_2, PO_2, and body temperature are kept constant, cerebral blood flow remains constant between systolic pressures of 80 to 180 mm Hg. Not all the factors affecting autoregulation are clear, but two are well substantiated and a third has been suggested by recent experimental work. Evidence of a local myogenic factor is based on the demonstration that the intrinsic smooth muscle cells of intracranial arteries and arterioles respond to change in intraluminal pressure by contracting with increasing pressure and dilating with decreasing pressure. It has also been demonstrated that intracranial arteries constrict in response to increased levels of oxygen and to decreased levels of PCO_2 and expand to the opposite changes in these gases. Alteration in pH causes similar changes to PCO_2 alteration, ie, vasodilation with increase and vasoconstriction with decrease in level of pH. A neurogenic basis for intrinsic control of the intracerebral arteries has been proposed based on physiologic experiments demonstrating areas of the brainstem, whose stimulation or ablation affects the autoregulation of the arteries and larger arterioles of the cerebral circulation.

Loss of autoregulation is seen in toxic states, following injury, and after hypoxia or hypotension; if autoregulation is lost, hypertension or hypotension can in itself be a factor for further brain injury. Methods for determining the intactness of autoregulation are available in the experimental animal and may be estimated in the clinical situation when intracranial pressure (ICP) is being monitored. It has been demonstrated that ICP will passively follow a change in mean arterial blood pressure (MABP) in animals and humans who have lost cerebral autoregulation. Thus, one may observe simultaneously recorded ICP and MABP during physiologic fluctuations, or the physician may artificially lower or raise the MABP, noting the effect on ICP. The importance of these measurements is rapidly becoming more apparent in clinical problems such as head trauma or vascular disease.

Extrinsic factors for control of cerebral circulation are intracranial pressure, and vasomotor and cardiac reflexes from special receptors in the carotid artery, the aortic arch, pulmonary arch, vena cava, and splenic artery, via brainstem nuclei to raise or lower systemic pressure toward adequate cerebral perfusion. Elevated PCO_2 levels or reduction in oxygen tension via these receptors tends to raise systemic blood pressure. Hypoxia or decrease in blood flow in the hindbrain by direct effect on the muscle causes slowing of the heart, peripheral constriction, and, therefore, relative increase in available circulation to the brain.

In the past, knowledge of the action of autoregulation and reflex activity have been used for diagnosis and prognosis of disease states. More accurate methods of determining their intactness and functions and pharmacologic agents now have therapeutic possibilities.

Clinical Manifestations. The history of neurologic dysfunction in the patient with vascular disease is one whose course in time is characterized by an abrupt change in neurologic function. There may be sudden deterioration, followed by relatively immediate or full recovery, called *transient ischemic attacks* (TIA). The term *stroke* denotes sudden loss of perfusion to a portion of the brain, which may be followed by a prolonged return to the premorbid state (reversible ischemic neurologic deficit) or leave the patient with residual deficit (completed stroke). The vascular accident of intracerebral hematoma often results in progressive neurologic dysfunction and a decreasing level of consciousness as the mass expands. This same abrupt onset is seen with the subarachnoid hemorrhage as sudden headache, alteration in consciousness, and changes in neurologic function occurring in a very few moments. The symptomatology resulting from vascular disease infrequently can be quite nonspecific, but more commonly the gradual loss of function in the cerebral hemisphere thought to be caused by "little strokes" is found at postmortem not to be vascular but to be a neuronal degeneration. Changes in function of the spinal cord considered to be vascular are rarely proved to be so unless the vascular compromise is secondary to mechanical factors involving the blood supply passing through the intervertebral foramen or mechanical factors in the canal, such as hypertrophic osteoarthritic spurs at the intervertebral spaces causing both vascular and nervous system tissue compression.

Occasionally the patient with a history typical of cerebrovascular disease, on more complete workup, proves to have a tumor. Conversely, the progressive neurologic deficit which has caused a patient to be suspected of having a tumor turns out to be recurrent transient ischemic attacks, secondary to extracranial vascular disease.

Surgically significant lesions of the cranial vasculature, that is, lesions in which surgical therapy can improve the prognosis for life or can decrease morbidity, are relatively rare compared with the common occurrence of cerebrovascular accidents or strokes. However, since surgery may decrease the morbidity and mortality of cerebrovascular disease and since there is relatively little other than supportive therapy, it is important to keep in mind the possibility of surgical intervention whenever cerebrovascular disease cases are seen. Surgical lesions fall into four groups of conditions with overlapping categories, such as infarction from congenital lesions and hematomas with infarcts. These groups are:

Transient ischemic attacks.
Cerebral infarctions.
Cerebral and cerebellar hematomas.
Congenital lesions.
Aneurysms.
Arteriovenous malformations.

Cerebral Ischemia and Infarction

Cerebral ischemia is a multifaceted problem which may originate with progressive atherosclerosis and which encompasses cerebral thrombosis, embolism, altered metabolic substrates, and hypoxia. Its ultimate pathologic end point, a stroke, is the third leading cause of death and disability in the USA and accounts for the disability of some 2 million persons today. The logic of surgical intervention in this disease is predicated upon the reversibility of the process and demonstration of statistically significant prophylaxis.

The sites of atherosclerosis are generally bifurcations of major vessles. In decreasing order they are the carotid bifurcation in the neck; the vertebral basilar junction; the middle cerebral (MCA), posterior cerebral (PCA), and anterior cerebral (ACA) arteries; and the major ascending vessels from the aorta. Hypertension increases the predilection for atherosclerosis at each of these sites. Embolic phenomena are generally fibrin-plated aggregates, and most of them arise in the heart, but many originate from one of the "local" vessels mentioned above. Embolic episodes usually affect the more distant vessels of the intracranial circulation, such as MCA and PCA, whilte the internal carotid (ICA) and vertebral artery are more subject to thrombosis.

The diagnosis of an occluded artery based on focal neurologic deficit was accepted for a long time, though it rarely was proved to be accurate at postmortem. Since arteriography, the assignment of the site of vascular disease on the basis of neurologic dyfunction has become even more insecure. A wide range of capability for adequate perfusion of the brain is maintained by the anastomoses described above, and these can alter the resulting neurologic dysfunction following occlusion of any of the major vessels.

The syndrome of "middle cerebral artery occlusion", described as a hemiparesis most severe in the arm with cortical sensory dysfunction, a partial visual field defect, and, if the hemisphere involved is the dominant hemisphere, dysphasia, does not necessarily require occlusion of the middle cerebral artery. It may result from narrowing of proximal vessels, the common sites being in the internal carotid artery at its origin in the neck or intracranially in its cavernous portion. In contrast, an individual may have a clinically silent carotid occlusion with maintenance of normal cerebral function distal to it because of adequate perfusion of the vessels by segments of the circle of Willis. Such a patient may eventually present with a "middle cerebral artery" syndrome when his opposite internal carotid has become sufficiently narrowed by atheromas to impair the adequacy of collateral flow. Thus, the effects of ischemia may be first apparent in the most distant reaches of the circulation on the side of the originally silent carotid occlusion. Anomalies and imperfections of the circle of Willis may remove its safety factor for some and make then early victims of arteriosclerosis, for they are less likely to have been protected from the effects of the loss of individual vessels.

The premorbid recognition of cerebrovascular disease often begins with an awareness of marginal cerebral blood flow presenting as transient ischemic attacks (TIAs), defined as focal neurologic signs or symptoms of less than 24 hours in duration which have a tendency to recur. In the region of carotid perfusion, they are associated with local embolic phenomenon and herald a completed stroke in one-third of patients within 5 years. Some patients progress to stroke after only one or two warning TIAs. Symptoms include hemiparesis, hemisensory disturbance, and, commonly, decrease or loss of vision in the eye of the involved side (amaurosis fugax). Vertebrobasilar TIAs less frequently progress to stroke with statistical predictability; symptoms include vertigo, dysarthria, facial paresthesia, diplopisa, sometimes paresis, and drop attacks. Immediate mortality rates from intracerebral hemorrhage, embolism, and thrombosis are approximately 93 per cent, 60 per cent, and 37 per cent, respectively. For those that survive, death rates from 16 to 18 percent per annum should be expected. A portion of this group with TIAs and more major infarctions may have a focal problem amenable to surgical treatment.

Identification of this group which may be helped by surgery is difficult, but a few quidelines are apparent. The workup of patients with TIAs should begin with a physical examination, including auscultation for a focal bruit over the suspected vessels - the carotid in hemispheric lesions, the subclavian or vertebral in brainstem disease. Next, regional cerebral blood flow is ascertained either with conventional nuclear brain scans or with more accurate xenon washout techniques that quantitate cerebral blood flow in milliliters per minute per 100 g cerebral tissue. Numerous Doppler systems now available are also able to pinpoint stenotic extracranial vessel disease and determine flow direction. Arch and cerebral angiography, however, must be performed to delineate accurately all the vascular pathologic change. There is a 5 to 7 percent morbidity from angiography in most series. It is important, therefore, to choose those patients in whom the chance of finding a remedial lesion is high enough to warrant this risk. If a stenotic of ulcerative lesion is localized, surgery may be appropriate. Treatment involves excision of the ulcerative or stenotic plaque, with or without vascular reconstruction or bypass around the obstruction. The most ideal patient for extracranial vascular surgery has TIAs, an isolated lesion, and no neurologic deficit. In Ojemann's endarterectomy series, 104 patients with TIAs had a 1/104 mortality, 2/104 had neurologic morbidity, and 101/104 resumed normal function. Fifty patients with persistent neurologic deficit before surgery showed a 45/50 recovery, 2/45 mortality, and 5/45 neurologic morbidity. A low surgical morbidity and mortality are dependent upon preoperative blood flow studies to identify the patient with borderline cerebral ischemia. At Yale, intraoperative distal internal carotid artery pressures are obtained and correlated with EEG and evoked cortical response recordings. Temporary bypass shunts are placed when the EEG or cortical evoked responses indicate inadequate perfusion.

Extracranial focal vascular lesioins are accessible to direct intervention; however, a joint study of extracranial arterial occlusion concluded that about 33 percent of patients suffered from a surgically inaccessible lesion such as intracranial ICA stenosis, MCA occlusion, or complete ICA occlusion. In 1967, Donaghy and Yasargil successfully anastomosed the superficial temporal artery to a cortical branch of the MCA in two patients with inaccessible vascular disease. Since then, this operation has been performed in hundreds of patients, with low morbidity and mortality rates but without long-enough follow-up for statistical analysis of results. Recently, not only has the STA-MCA bypass been utilized, but also occipital to posterior inferior cerebellar artery anastomoses have appeared in the

treatment of verebral basilar disease. At this time, indications for bypass procedures may include low-perfusion syndromes which are accompanied by ICA occlusion, carotid siphon stenosis, and MCA stenosis or occlusion. It has also been successfully employed as an additional source of flow to cerebral regions perfused by major intracranial vessels which may require occlusion during the treatment of another primary disease such as aneurysm, carotid cavernous fistulas, or neoplasms.

The other indication for surgical therapy for patients with cerebrovascular occlusion is in those patients with completed infarcts in whom symptoms of progressive focal neurologic deficit and brainstem involvement occur. The progression most commonly occurs from 12 to 36 hours after the stroke; it consists of increase in the initial focal deficit, such as hemiparesis to hemiplegia; decreasing levels of consciousness; and signs of third nerve paresis (dilated pupil) and midbrain dysfunction. This progression occurs since occasionally a major infarct constitutes a mass because of the volume of swollen edematous and, at times, hemorrhagic brain. In such circumstances, it behaves like other cerebral tumors in producing clinical signs and symptoms of progressive focal neurologic deficit and progressive brainstem involvement. The condition may be amenable to surgical removal or internal decompression of the swollen brain by craniotomy for relief of the pressure.

Nonsurgical management following cerebral ischemia and infarction should be directed at controlling associated brain edema and altered metabolism. With reference to ischemic brain edema, vigorous efforts should be made to control the mean arterial blood pressure. Loss of autoregulation in the ischemic brain can result in further brain damage with hypotensive or hypertensive episodes. Hyperglycemia often occurs during or following cerebral vascular episodes. The hypoxemia of cerebral ischemia results in more anaerobic glucose metabolism and lactate accumulation, which can cause further brain injury. It has been suggested, therefore, during the peri-ischemic period (1 to 3 days) that only glucose-free intravenous solutions be given to patients. Dexamethasone has been shown by several reports to be efficacious inthe management of cerebral ischemic edema. The mechanisms suggested include reestablishment of cell membrane function and decrease in fatty acid peroxidation. With several recent reports demonstrating release of free fatty acids (which induce edema) into brain tissue during ischemic episodes, the addition of antioxidants to steroids may prove to be beneficial in controlling some progressive neurologic deficits due to ischemic cerebral edema.

Cerebral and Cerebellar Hemorrhage

Although intracranial hemorrhage may result from any structural cause of an abnormality in the blood vessels or circulating blood, the most common cause of intracranial hematoma is hypertensive arteriosclerotic vascular disease. In patients succumbing to hypertensive intracranial hemorrhage, the larger arteries show hypertrophic and degenerative changes in their media and intima; the arterioles demonstrate demonstrate thickness of the vessel walls and reduction in lumen caliber secondary to hypertrophy of the initimal lining; and only minor structural changes are present in the capillaries. The pathogenesis of hypertensive hemorrhage is unclear and thought to involve many factors. The blood pressure fluctuates in hypertension and, during periods of decreased systolic pressure, ischemia of tissue supplied by these diseased vessels and/or the arteries themselves may occur. The lack of arterial support from surrounding tissue (evidenced by "état lacunaire") and weakness in

the vessel wall result in hemorrhage when the increased intraluminal pressure returns to the damaged segment. Arterial spasm with distal ischemia and decreased tonus may be significant mechanisms with hemorrhage resulting at the devitalized or necrotic channel after relaxation of the spasm.

The commonest sites of hypertensive hemorrhages are in the regions of the lentiform nucleus, deep white matter or cerebellar hemispheres, pons, and midbrain. Hemorrhage into the basal ganglion can be divided into lateral and medial involvement. The latter arises medial to the putamen and destroys the hypothalamus, usually resulting in death; however, the lateral group are in the putamen, external capsule, and claustrum and often are compatible with survival. In contrast to hemorrhagic infarction in which all tissue in the area is destroyed, hemorrhage has a tendency to track along tissue planes, separating rather than destroying the nervous tissue. This phenomenon is especially true when the hematoma has its origin in white matter and may result in relatively minor permanent neurologic deficit.

Clinical Manifestations. Initial symptoms of intracranial hemorrhage may be nonspecific and include headache, dizziness, paresthesias, or mild speech disturbances. These symptoms are followed by a sudden "strokelike" syndrome of paresis, severe headache, severe speech disturbances, vomiting, and/or loss of consciousness. The space-occupying hemorrhage may continue to enlarge and result in herniation of brain, secondary brainstem hemorrhage, and death. In instances where hemorrhage is situated in the medial basal ganglia, hypothalamus, brainstem, and dominant hemisphere temporal parietal speech areas in patients over fifty-five years of age, surgical intervention is probably not indicated because of the severe mortality and morbidity. However, intracranial hemorrhage at other sites may be surgically evacuated, frequently with little or no neurologic residual.

Approximate localization of the surgically accessible hematomas in the cerebral hemispheres is not difficult, since the patients present with neurologic dysfunction of the area involved. Usually carotid angiography is necessary to accurately localize the lesion in order to minimize surgical trauma. Hematomas of the cerebellar hemisphere are more difficult to recognize. A history suggestive of acute cerebellar dysfunction is most helpful but frequently not obtained, since initial loss of consciousness in patients with cerebellar hemorrhage is not uncommon. Cerebellar dysfunction with altered consciousness followed by progressive lower brainstem dysfunction, but without the pupillary signs and deep unconsciousness seenwith pontine hemorrhages, should raise the suspicion of cerebellar hematoma. The brainstem signs most frequently seen in these conditions are slow respiratory rate, often with Cheyne-Stokes characteristics, slow pulse, hiccups, and difficulty with swallowing secretions. Early recognition of a cerebellar hematoma before major compromise of the lower brainstem occurs is necessary if a favorable outcome is to be achieved with surgical intervention.

Congenital Lesions

The vascular diseases most amenable to surgical intervention are those due to congenital lesions, namely, the saccular, or berry, aneurysm and the arteriovenous malformation.

Aneurysms

Most cerebral aneurysms are silent lesions that do not cause clinical disease and are often therefore incidental findings at autopsy. However, rupture of an aneurysm initiates a distinctly morbid train of events. The natural history of patients with ruptured aneurysms is still the subject of brisk controversy, since the reported mortality during the initial hemorrhage and during subsequent therapy varies from 20 to 75 percent. It is fair to state that there is a high initial mortality and that some of the lower figures do not take into account patients who never arrived at a surgical service. Although the prognosis is directly related to the degree of neurologic deficit following subarachnoid hemorrhage, approximately 27 percent of patients will die in the first week subsequent to ruptured aneurysm, the percentage declining thereafter. A second hemorrhage has a mortality of 42 percent, the incidence having its peak between 7 and 12 days following the initial hemorrhage. The likelihood of rebleeding then decreases through the ensuing 4 weeks to drop off sharply after 6 weeks; however, 10 percent of patients will rehemorrhage after 1 year. From the onset of the rupture to the second or third month after the rupture there appears to be a mortality between 30 and 40 percent.

The majority of intracranial aneurysms arise from the larger intracranial arteries of the circle of Willis and at the origin of large vessels from the vertebrobasilar system. Intracranial arteries have a thin, poorly developed, adventitial layer, a media which is partially or completely devoid of elastic fibers, and an intima consisting of an elastic lamella and thin collagenous tissue covered by an endothelial lining. Aneurysms develop at the arterial bifurcation and are probably caused by the effect of hemodynamic factors on defects in the medial musculature and elastic layers of the artery.

The most common sites of intracranial aneurysms are the internal carotid artery (42 percent), anterior cerebral artery (43 percent), and middle cerebral arteries (20 percent), with 6 percent of aneurysms arising from the vertebrobasilar system in the posterior fossae. The majority of aneurysms are single; however, there is a 20 percent incidence of multiple aneurysms. When two aneurysms are present, they have a proclivity either for being symmetric in location or for arising from the same parent vessel.

The immediate cause of rupture of aneurysms usually cannot be determined, but the coincidence of hypertension and a congenital aneurysm appears to increase the possibility of rupture no matter what the cause of hypertension. Thus, in young patients with coarctation of the aorta with hypertension in the cerebral circulation ruptured aneurysms are common. There also seems to be a correlation between systolic hypertension and rupture of the aneurysm. Silen aneurysms are seen with approximately the same incidence in patients with or without hypertension, while rupture is more frequent in patients with hypertension.

Clinical Manifestations. The syndrome of nontraumatic subarachnoid hemorrhage is one of sudden onset of headache followed by altered consciousness. Focal neurologic deficit may occur, but, in contrast to the occlusion of a major intracranial vessel, headache, meningeal signs, and altered consciousness are usually much more prominent and the focal neurologic deficit less prominent. The most common cause of this syndrome is a ruptured berry aneurysm. Other possible causes are hypertensive hemorrhage and a bleeding arteriovenous malformation.

The most benign syndrome of subarachnoid hemorrhage occurring from aneurysm is a sudden headache; maintenance of, or perhaps only transient loss of, consciousness; and the absence of any objective neurologic signs. The presence of the stiff neck and perhaps photophobia, reflecting the presence of blood in the cerebrospinal fluid, are the only signs that a major pathologic vascular syndrome has occurred. A patient with this syndrome is considered to be grade I in each of several schemas for evaluating patients with subarachnoid hemorrhage and presents an excellent opportunity for surgical intervention to alter the natural course of the disease. On the other end of the spectrum of subarachnoid hemorrhage, when a large amount of bleeding occurs in a short period of time, the basal cisterns may be filled with clotted blood, or dissection of the hematoma into the hemispheres may occur. At this time the patient is unconscious, and therapeutic manipulation or angiography has a high probability of increasing morbidity or mortality.

Treatment. A major factoor in the morality and morbidity of subarachnoid hemorrhage is vasospasm of the branches of the circle of Willis due to an irritating effect of the blood on the vessels. This may produce neurologic deficit without the presence of mass and has been considered by most surgeons to be a contraindication to early surgical treatment, since it is associated with a high operative mortality. The goal of the treatment is to remove the aneurysm from the force of systolic blood pressure; since recent evidence suggests that the probability of a second hemorrhage increases in a linear manner with time, the earliest possible accomplishment of this is indicated. The most favorable lesion is one in which the aneurysm has a narrow neck which can be occluded without interference of the parent vessel circulation. Not all aneurysms can be so treated; some may be coated with synthetic plastics to reinforce their walls, and others may be treated by occlusion of parental vessels, such as the common or internal carotid artery in the neck, to decrease the pulse pressure applied to an aneurysm of the internal carotid artery intracranially.

Arteriovenous Malformations

A second major type of congenital vascular disease is arteriovenous malformation, which results from replacement of the capillary bed by clusters of abnormal vessels interposed between arterial and venous systems in a segment of the circulation. Such malformations tend to enlarge with time and may present in a variety of ways. Because the contiguous cerebral tissue may be chronically ischemic, neurologic symptoms and signs will include focal loss of neurologic function and altered excitability. For this reason focal neurologic deficits of a minor or major nature and seizures are frequently seen in these patients. Arteriovenous malformations also are causes of subarachnoid hemorrhage and intracerebral hematoma.

Subarachnoid hemorrhage due to arteriovenous malformation occurs generally at a younger age than that due to aneurysm. The majority of cases occur between twenty and forty-nine years of age without a peak incidence, whereas with aneurysm the majority occur between forty and sixty-four years of age with a peak incidence between fifty and fifty-four years.

Intracerebral hematoma resulting from "cryptic" vascular malformations is probably more common than generally appreciated and is frequently mistaken for hypertensive hemorrhage. These small arteriovenous malformations, measuring less than 3 cm maximum size, are usually clinically silent and may be more common than the classic arteriovenous

anomaly. The cryptic anomaly may be destroyed by the hematoma and its pathogenesis impossible to determine. In contrast to hypertensive hemorrhage, hematoma secondary to arteriovenous anomalies tend to occur outside the basal ganglion in the cerebral hemisphere white matter and may be present in the cerebellum, spinal cord, and brainstem.

Treatment. Because of the possibility of increase in size of the malformation with increased shunting of blood to impair further the oxygenation of surrounding tissue, the continued problem of seizures, and the constant possibility of hemorrhage, surgical excision of the malformation is indicated whenever it is feasible. Feasibility of resection relates to size, position, and source of arterial supply. Arteriovenous malformations that are deep in the hemisphere are in general nonresectable, since according to their natural history they would have a lower morbidity than would be expected from surgical treatment. Excision of arteriovenous malformations in the tip of the occipital, frontal, or temporal lobe not only may be lifesaving inthe face of hemorrhage but may control seizures and improve circulation in the involved hemisphere.

The Surgery of Epilepsy

Epilepsy is perhaps the oldest recorded neurologic disorder. Its history traces a fascinating path from mysticism to accurate descriptions of cortical physiology and pathology. Although its study has provided insight into cerebral pathways and cortical functional localization, the cure or control of this disorder continues to frustrate the physician devoted to caring for seizure victims. Epilepsy affects at least 1 in 200 people. Therefore, the population of epileptics in the USA today totals more than 1 million. Of these, more than 600.000 have partial seizures, eg, having focal origin. Two-thirds of patients with seizures have their initial seizure before the age of twenty. The older the patient, however, at the onset of epilepsy, the more likely a focal etiologic cause will be found. In patients presenting with their first seizure after age twenty, between 10 and 25 percent will have a focal lesion (such as a tumor or arteriovenous malformation) demonstrated on a complete neurologic workup.

The cornerstone of seizure control is medical therapy for those patients without obvious cerebral pathologic change. Anticonvulsant drugs will control seizures in more than half of the patients with epilepsy and achieve partial to almost complete control in another 25 percent. It is estimated that there are approximately 200.000 patients with uncontrolled focal epilepsy in the USA. Perhaps 45.000 to 100.000 of these patients are potential surgical candidates. Surgical therapy is reserved for patients whose seizures are not well controlled with medication and whose history, description of the seizures, neurologic examination, or electroencephalogram suggests a focal cerebral origin. Success of surgical treatment correlates best with accurate identification of these patients with focal seizures, precise localization of the point of origin, and careful surgical excision of the area. Accurate localization of the area and identification of any motor, sensory, or intellectual deficit that may be caused by excision of the focus require a team approach, utilizing the neurosurgeon, neurologist, electroencephalographer, neuroradiologist, and neuropsychologist, as well as the proper electrophysiological equipment and techniques.

The evaluation of a patient being considered for surgical treatment consists of four phases. In phase 1, the patient has a careful physical and neurologic examination with evaluation of past medication and additional medical trials if medication has not been used

adequately. When the decision is made to continue the surgical workup, the aims are to establish scalp localization if possible and to exclude those with EEG and clinical evidence of multifocal disease. This requires continuous electroencephalographic monitoring in order to record spontaneously the electrical seizures. Audiovisual monitoring is essential to provide clinical correlation with electrical evidence of seizure events. To enhance the opportunity of recording spontaneous seizures, the patient may undergo sleep deprivation, and anticonvulsant medication is often stopped or markedly decreased. This methodology, of course, requires constant observation by specially trained nursing and medical personnel. Nasopharyngeal or sphenoidal leads are often used in conjunction with scalp electrodes during this phase. In phase 2, the patient undergoes investigation to document other localizing findings. Structural central nervous system abnormalities are searched for, which might lead directly to surgery, circumventing additional workup. The patient undergoes plain skull x-rays, computerized axial tomography with double-dose contrast, cerebral angiography, examination of the cerebral spinal fluid, neuropsychologic evaluation, and formal visual fields. At the time of arteriography, a short-acting barbiturate, such as Amytal, is injected selectively into each internal carotid artery, and behavioral testing is carried out by the neuropsychologist and neurologist to determine speech dominance and the memory capacity of each temporal lobe (modified WADA test). If a space-occupying mass lesion is not identified during this phase but there is continued evidence of focal epilepsy, the patient enters phase 3, which aims to establish definitive localization or to exclude those patients with multifocal disease or surgically inaccessible disease by the use of depth electrodes. In this phase, electrodes are implanted stereotactically and the patient again undergoes prolonged, continuous audiovisual electroencephalographic recording. In a special nursing unit, medication is again withdrawn if necessary to precipitate ictal clinical and subclinical seizures, and at least three such events are recorded before a decision is reached regarding the patient's appropriateness for surgical treatment. There is still a great deal of controversy regarding the use of scalp versus depth electrodes to localize focal cerebral epilepsy. We have recently reviewed the accumulated literature on depth and scalp localization. In this review, depth EEG identified some candidates who had localized depth EEG recordings but unlocalized scalp EEGs. This increased the number of operative candidates by 36 percent of the patients reported. Conversely, there were patients who had scalp localization but in whom the depth EEG was unlocalized, and these patients were excluded from surgical consideration. Thus, the depth EEG seemed to alter surgical plans in more than 50 percent of patients where scalp and depth patterns could be compared.

At Yale, 60 patients were evaluated with depth EEG, and 32 underwent surgery. Of the 32, 24 have been followed for more than 5 years. The overall surgical success rate was 57 percent, success meaning either cure of seizures or a reduction in seizure frequency greater than 75 per cent. The depth electrodes provided the major prognostic information, indicating that when 100 percent of clinical seizures originated electrically at one site, surgical success was 95 percent or greater. However, when less than 80 percent of seizures arose from one focus, even when this focus was still a predominant one and was excised, success dropped markedly to less than 20 percent. Most patients falling in the surgical success category had temporal lobe foci. Those with less good control were determined to have predominantly frontal lobe or multifocal onset of their seizure disorder. A frontal lobe focus usually indicates more diffuse pathology and has been one of the most frustrating groups with which to deal surgically. Of patients without radiographic lesions, gliosis was the most common pathologic finding at surgical excision, being seen in all cases. Of the 24 patients undergoing surgical

excision, two hamartomas and one vascular malformation were unexpected findings at the time of surgical excision. All patients are maintained on their anticonvulsants for at least 5 years postoperatively, and if they remain seizure-free, the drugs may then be tapered. Thus, patients with well-localized complex seizures, especially in the temporal lobe, may do very well following cortical excision. However, this leaves a number of patients who are localized to either the frontal, parietal, or occipital lobe who do less well, perhaps because their disease process is more diffuse and certainly because complete resection is impossible without unacceptable neurologic deficit.

Those patients who present with radiographic mass lesions, such as tumors or calcifications, are an exception to this rule. Of 171 patients followed at Yale for medically intractable partial epilepsy, 15 percent were ultimately found to have an intracranial mass. These masses included 16 neoplasms and 9 nonneoplastic structural lesions (2 calcified arteriovenous malformations, 3 arachnoid cysts, 1 granuloma, and 3 calcifications of unknown etiology). Although any treatment directed at the mass lesions, such as biopsy and radiation in unresectable neoplasms, gave good results in terms of reduction of uncontrolled seizures, patients undergoing lobectomy consistently showed more than 95 percent reduction in seizures, regardless of the cerebral lobe involved. This leaves a group of patients with multifocal epilepsy who are much more difficult to treat. Their seizure disorder may emanate from a diffuse process in one hemisphere or may have bilateral cortical onset. It is in this group of patients that more experimental forms of treatment are being attempted, such as stereotactic subcortical radiofrequency ablations, cerebellar stimulation, and, perhaps most promising, limited or complete section of the corpus callosum. This latter surgical procedure has been found most efficacious in those patients presenting with congenital infantile hemiplegia whose seizures originate diffusely from the affected hemisphere. Division of the corpus callosum confines the abnormal electrical activity to the bad hemisphere and stops secondary generalization and, therfore, loss of consciousness in the majority of patients. Although all the indications for this operation are not clearly defined, the prospect for this being a standard and recommended operative procedure in some patients is clearly evident.

Intracranial Infections

Infections of the central nervous system and its coverings have surgical significance if they produce a mass, hydrocephalus, or osteomyelitis, or are the result of a break in, or absence of, continuity of the nervous system coverings.

A mass can be caused by pus, as in abscess or empyema, by the edema of the reaction of the nervous system to infection, by the edema of venous occlusion, or by an effusion in the subdural space. Hydrocephalus may be caused by the interruption of cerebrospinal fluid flow by blockage at the villi or basal cisterns, or within the ventricular system. Osetomyelitis of the skull and spine has surgical significance because of the propensity for the infected bone to become necrotic and sequester, forming the nidus for chronic inflammation, abscess, and progressive infection of adjacent bone and intracranial contents.

Surgical therapy of infections of the nervous system falls into one or more of the following categories: excision of infected bone, drainage or excision of abscess or empyema, decompression to relieve the effects of edema, shunting to relieve obstruction of cerebrospinal fluid circulation, and the repair or establishment of anatomic barriers to infection. This last

category may involve surgical procedures as diverse as repair of a congenital heart lesion, eradication of a chronic middle ear infection, or removal of a pituitary tumor that has eroded into the sphenoid sinus.

Osteomyelitis of the Skull

Infections of the skull are relatively rare. The pathogenesis of osteomyelitis of the skull includes three major avenues of origin: (1) direct extension of preexisting infection in a paranasal sinus, middle ear, or mastoid air cells; (2) infection from a wound of the scalp that extends into or below the subgaleal space; and (3) hematogenous spread from elsewhere in the body. Any organism can be the causative agent, but by far the most common are *S. aureus* and the gram-negative contaminants of ear infection, such as *Bacillus proteus* and *B. pyocyaneus.* Once the infection is established, it usually spreads through the diploic spaces and the epidural space. Although spread under the outer periosteal layer or pericranium does occur, the pericranium is so firmly attached to the skull that it limits this as an important avenue of spread. This same attachment of the pericranium sometimes gives a false impression of the extent of the skull infection. Part of the mechanism of spread of the infection includes damage to the blood supply of the skull, and therefore necrosis and sequestration of the bone is common.

Clinical Manifestations. The signs and symptoms of pyogenic infection of the skull are those of generalized infection, that is, fever, malaise, and leukocytosis, with focal tenderness and swelling. At times the infection can be indolent and manifest itself first by drainage through an area of the scalp, but more often it is highly virulent with spread into the intracranial structures, resulting in irritation and inflammation of the brain.

Treatment. The only effective treatment is excision of the involved bone and drainage of the area. Care must be taken to be certain that no infection remains in the epidural space to strip away the dura or in the diploic spaces to further necrose and infect adjacent bone. Appropriate antibiotics are helpful in protecting the bone edges and epidural space from further spread of the infection but are of little help without surgical excision of infected necrotic bone.

Epidural Abscess

Intracranial epidural abscesses are most commonly seen adjacent to infections of the skull and therefore are common near the middle ear, mastoid air cells, and nasal sinuses. Although the dura is a good protective barrier against inward spread of infection and although many epidural abscesses are discovered as incidental findings of surgical treatment for mastooid and sinus infection, they should not be considered benign processes. The infection can and often does break through the dural barrier, meningitis being the most common result and brain abscess the next most common. As already mentioned, the epidural space is an avenue for progression of osteomyelitis of the skull.

Clinical Manifestations. The symptom of epidural abscess are quite similar to skull infections, except that seizures and focal neurologic deficits are more common. These are commonly secondary to thrombophlebitis of superficial cortical veins.

Treatment. Treatment of epidural abscess is trephination, drainage, excision of infected bone, and the use of appropriate antibiotics. The excision of any infected bone and the drainage of any infected sinus or mastoid air cells are mandatory if the infection is not to recur.

Subdural Empyema

Although intracranial spread of paranasal sinus, ear, and mastoid infections to the epidural and subarachnoid spaces is more common, the high rate of morbidity and mortality seen with delayed or inadequate treatment with spread to the subdural space requires that this possibility always be kept in mind. Infections of the paranasal sinuses are the most common source of subdural empyema, and the spread of the infection to the subdural space is felt to include direct extension along emissary veins and via dural sinuses.

Clinical Manifestations. The symptom complex of subdural empyema is related to five factors: First is the pathogenicity of the organism. Although staphylococci, *B. proteus,* and *B. pyocyaneus* are common chronic infecting agents of the sinus, ear, and mastoid areas and may cause subdural empyema, the infection is more often caused by staphylococci or streptococci that are acutely superimposed on a chronic infection. With these highly pathogenic organisms the course of subdural empyema may proceed to serious neurologic deficit in hours and to death within days. Second, there is no natural barrier to spread of the infection within the subdural space. Because of this the surface of an entire hemisphere may be involved within a very short period of time, and progression beneath the falx to the opposite hemisphere occur as soon as significant pus has accumulated. Third, the veins traversing the subdural space from cortex to the dural sinuses are particularly vulnerable to inflammatory response from the infection, with progressive thrombosis of the cortical veins and dural sinuses resulting. Fourth, the reaction in the subarachnoid space and surface of the hemisphere through the thin piarachnoid causes a sterile meningitis and an acute encephalitis. Finally, there is the mass effect, mainly secondary to the inflammatory reaction and the occlusion in venous drainage but also in part secondary to the volume of pus.

The first factors relate to rapidity of onset and the involvement of the entire hemisphere within a short period of time, while the last three explain in part the high incidence of seizures, signs of meningitis, neurologic deficit, and massive edema. Thus the symptom pattern of a patient with subdural empyema may vary but is best characterized as follows: A patient with a history of chronic sinusitis or chronic ear infection has an acute upper respiratory tract infection. Two or three days later when he should be making an uneventful recovery, he has a sudden secondary rise in temperature, shaking chills, and a convulsion. On regaining consciousness he complains of severe lateralized headache and of mild weakness or numbness on the opposite side of the body. Within a short period of time, a few moments to a few hours, he has another convuslion and then progressive decrease in his sensorium. At this point he is frequently either brought to a hospital or seen by a physician. Initial examination reveals an acutely ill patient with signs of meningitis. Lumbar puncture shows increased cellular response, but the spinal fluid sugar is within normal limits, and the microscopic smears fail to reveal any evidence of bacteria.

Within a few hours the patient can be in severe difficulty, often with total loss of function in the involved hemisphere, evidence of herniation of the temporal lobe with a dilated pupil on the side involved, and the beginning of decerebration.

Treatment. Treatment varies with the severity of the symptoms at the time that the patient is seen and the rapidity with which they have progressed. However, early trephination and drainage alone would be a satisfactory treatment only if the patient shows no sign of increased intracranial pressure, major alteration of conscious level, or shift of the midline structures. If the patient already shows any of these signs, drainage of the subdural space, external decompression by removal of a major portion of the skull on the side involved, and opening of the dura to allow expansion of the hemisphere are mandatory if the patient is to survive with good neurologic function. Although this appears to be a rather heroic treatment, the fact that the major deficit is secondary to inflammation and venous occlusion allows a degree of recovery to occur that is most gratifying, provided the treatment is started before the upper brainstem becomes irreversibly damaged.

Brain Abscess

Intracerebral pyogenic infection can be categorized as acute, subacute, or chronic. The symptoms and signs of suppuration within the brain encompass a range that extends from rapidly progressive focal neurologic deficit with a systemic response indicative of severe infection to evidence of an expanding intracranial mass with no suggestion of infection. In the former, the symptoms usually relate to a focal pyogenic encephalitis with little or no frank pus that will either resolve into an abscess or cause death if it continues to spread unchecked, while in the latter the abscess has a well-formed capsule of fibrous tissue and glial reaction.

The brain is resistant to infection by bacteria. Experimentally it is difficult to produce an abscess or focal encephalitis within the brain unless the area in which bacteria are placed is damaged beforeheand, as by trauma, hemorrhage, or anoxia. Although the brain initially responds to infection in the same fashion as any tissue, its ability to wall off the infection by granulation and glial tissue is so effective that it often isolates the infection not only from the brain but even from significant contact with the systemic blood supply. For this reason the contents of a localized brain abscess usually cannot be sterilized by systemic antibiotics.

The pathogenesis of brain abscess includes, in the order of frequency of occurrence, three main routes of entry of the infecting organism: by direct extension from middle ear, mastoid, and paranasal sinus infections; through the blood supply to the central nervous system; and by direct traumatic penetration. Posttraumatic brain abscess is now rare, since adequate surgical care of the wound and antibiotic protection are excellent preventives. The ear is three or four times more common as a source of the infection for brain abscess than are the sinuses, and the temporal lobe and cerebellar hemispheres are the more common sites for abscesses secondary to these infections. Hematogenous abscesses can occur following any bacteremia but are more commonly seen with lung abscess, infected bronchiectasia, and cyanotic heart disease. In the patient with cyanotic heart disease the presence of a right-to-left heart shunt with the loss of the lung as a filter to remove blood-borne bacteria is felt to be the mechanism responsible for the high frequency of occurrence of brain abscess. Since in cyanotic heart disease one of the commonest causes of cerebral dysfunction is abscess, a patient with symptoms of cerebral disease, either seizures or neurologic loss, should have a

contrast CT scan to rule out abscess. The organisms commonly seen in brain abscess are streptococci, pneumococci, and staphylococci. The most common streptococci are the anaerobic strains.

Clinical Manifestations. The symptom complex of brain abscess will vary according to whether the infection is acute, subacute, or chronic. As was noted earlier, in the chronic walled-off brain abscess there may be no signs of systemic reaction to infection, that is, no fever or leukocytosis. Brain abscess should be considered as a possible diagnosis in any patient with progressive focal neurologic deficit and increasing mass with a short, that is a 2- to 3-week, history. Often with questioning a source of possible brain abscess or an episode suggestive of bacteremia can be found in the recent past. Acute pyogenic encephalitis has a course quite similar to a subdural abscess, from which it is often difficult to differentiate.

The subacute abscess varies between these two extremes, so that the combination of signs of inflammation, progressive focal neurologic deficit, and increasing mass effect not only suggests the presence of a brain abscess but also frequently from the neurologic signs localizes it. A CT scan is the most accurate method to identify the presence of a brain abscess, to differentiate cerebritis from abscess, and to demonstrate the presence or absence of a capsule, and it is the mandatory test when brain abscess is suspected.

Treatment. Although in the past it was taught that suppurative intracranial encephalitis could not be treated surgically, decompression, removal of portions of the infected brain, and the use of antibiotics may be lifesaving and function-saving when applied to the critically ill patient in the acute stage. Usually the patient can be carried through the acute stage by the use of antibiotics and antiinflammatory agents, such as steroids, and by careful control of fluids. The progress of the treatment can be monitored by repeated CT scans, and the formation of a capsule demonstrated. This usually takes 10 to 12 days.

Once localization has been accomplished, drainage of the abscess by intermittent tapping through a perforator opening, by constant drainage, or by excision of the abscess will give a high percentage of cures. In hematogenous abscesses more than half will be multiple, and care must be taken to be certain that an abscess is not missed. The major dangers to the patient with abscess of the brain are (1) the early acute edematous response the brain has to pyogenic infection, (2) the continued problem of a mass lesion from both the edema and the suppuration itself, and (3) the continued possibility of rupture of the abscess into the ventricular system. This latter complication can be rapidly fatal. With the advent of antibiotics and anti-inflammatory agents such as steroids, brains abscess should have low mortality and, depending upon its location, low morbidity rates. However, failure to diagnose its presence until late in its course, failure to realize the dangers of mass effects from the abscess, the surrounding reaction of the brain, and the concurrrence of cardiac disease and systemic infection have kept the mortality rate in most reported series between 35 and 45 percent.

Pain

Pain, the sympotom that most commonly brings a patient to a physician, can be simultaneously the protector and the debilitator. As a protector it calls for diagnostic inquiry, but when it no longer has protective value, it can be a disease in itself. The best treatment of pain is the removal of its cause. When this is not possible or must be delayed, drugs, and

particularly opiates and their derivatives, can be used for relief. All potent analgesics, however, have addicting qualities and therefore decreasing effectiveness with continued use, so that if more than a few months of pain relief is required, drugs not only are ineffective but also can contribute to physical deterioration and mental depression. The need for prolonged effective control has been the impetus for surgeons to devise surgical means for relieving pain. Horsley's resection of the gasserian ganglion (1891), section of dorsal roots by Bennett and Abbe (1889), and Martin's incision into the anterolateral portion of the spinal cord (1911) were milestones in this quest, but further success has been limited by the same lack of information that has limited the control of pain by any method. This lack of knowledge exists not only in the spheres of anatomy and physiology but even in defining pain. Is pain a sensory modality with its own anatomic and physiologic correlates, or is it a learned response to environmental alteration which threatens the organism? Is pain an emotional reaction evoked with or without stimuli, or is it the recognition that a central nervous system pathway or area has been activated, indicating that a portion of the organism has reached or exceeded its functional limits? There is justification for the concept that pain contains some or all or these attributes.

A mark of a good clinician is the ability to separate pain caused by stimulation of peripheral nerves from complaints of pain that signify a psychologic or psychiatric disorder. The major cause for failure of any treatment of pain is the failure to differentiate pain from suffering. Even in the face of obvious disease that can cause pain, such as cancer, the complaints of pain may relate more to depression over the disease and its progress than to nerve stimulus. Failure to treat both aspects will give little relief, and denervating the offending part may increase suffering by reinforcing the patient's concept of progressive deterioration. Drugs given for anxiety may increase depression and increase suffering. Careful evaluation of the situation and the response to each therapeutic maneuver is necessary to obtain maximum benefit of any treatment for pain. In general, patients with a life expectancy of 2 months or more are best treated with drugs. The treatment should include drugs of sufficient potency and in sufficient amounts to relieve pain. Such treatment, while a blessing to a terminal patient because of the changes in sensorium, does not allow a patient with longer life expectancy to enjoy the remaining time. Surgical interruption of pain pathways can both relieve the pain and allow a drug-free existence and should be considered in any patient with severe pain that cannot be relieved by treatment.

Surgical Relief of Pain

Despite the paucity of knowledge concerning pain, empiric observations combined with physiologic studies have given neurosurgeons effective methods for surgically relieving pain. They fall into one of five different categories:

1. Section of the peripheral nerve pathway: neurectomy, sphlanchnicectomy, dorsal rhizotomy.
2. Section of central nervous system pathways: anterior lateral chordotomy and trigeminal tractotomy.
3. Procedures to alter affective response to pain: thalamotomy, lobotomy, cingulotomy.
4. Section of the efferent arc of the vasomotor reflex: sympathectomy.
5. Suppression by stimulation.

Despite many variations in technique and many claims for various procedures, clinical experience has demonstrated that all the procedures for relief of pain from lesions of the nervous system fall into one of the first four categories and that each category has its area of application, its inherent rate of success, and its complications. Experience with suppression of pain by stimulation, if related to acupuncture, has accumulated for centuries. However, there is still too little objective evidence to determine the extent of its usefulness and its long-term effectiveness. Application of the correct procedure at the correct time to a suitable patient is the most difficult aspect of the surgery for pain.

Section of the Peripheral Pathway

Section of the peripheral pathway for pain causes loss of all sensation distal to the area of section. Permanent loss occurs only when the pathway is sectioned proximal to the sensory ganglion, that is, section of the dorsal roots, termed *rhizotomy*. Any procedure that damages primary sensory neurons gives subjective sensory loss, which has as a complication painful paresthesias or spontaneous pain in the anesthetic area in 5 to 8 percent of cases. The explanation for this pain may be altered excitability of the dorsal root entry zone neurons. However, since further pain pathway interruption usually intensifies the pain, a more logical explanation appears to be that in this group of patients the subjective sensation of sensory loss signifies something wrong with the area and is painful. Any further alteration in sensation increases the pain. The psychologic basis of this may be that anything painful is abnormal, and therefore anything abnormal is painful. Stressing the lack of significance of such feelings at times helps, but alteration in affective response by either psychotherapeutic drugs or surgical intervention sometimes is the only recourse when the paresthesias become unbearable.

Section of multiple dorsal roots is seldom considered for relief of pain in a functioning limb, since loss of position sense, gamma afferents, and sense of touch leave the limb useless. It is of value in the thoracic acra or where only one root is directly involved. In the former, touch and position senses are not of functional significance, and in the latter, sensory overlap of adjacent roots makes the peripheral sensory loss minimal. This sensory overlap of adjacent roots makes section of at least two and usually three roots necessary for significant deafferentation of any peripheral area.

Rhizotomy of the trigeminal nerve is used to control pain of malignant tumors of the face and trigeminal neuralgia. It can be performed by direct section of the trigeminal roots in the middle or posterior fossa or stereotaxically in the middle fossa with a radiofrequency electrode through the foramen ovale. Trigeminal neuralgia, or tic douloureux, is a painful condition of the trigeminal nerve of unknown cause, characterized by paroxysms of pain over one or two adjacent divisions of the fifth cranial nerve. The paroxysms are often initiated by any stimulus in localized areas of the division involved, known as "trigger zones". Section of the preganglionic fibers of the trigeminal nerve or of the descending trigeminal tract in the brainstem permanently controls the pain, but as with other peripheral nerve section painful paresthesias may arise.

An observation by Jannetta that patients with trigeminal neuralgia frequently have vascular compression of the trigeminal rootlets as they exit from the pons suggests altered conduction and excitability at the root entry zone of the brainstem as thje cause of the syndrome of trigeminal neuralgia. Jannetta also has suggested that other paroxysmal disorders

of cranial nerves, such as glossopharyngeal neuralgia and facial tic, have the same cause. Compression of the trigeminal nerve more distally does not correlate with the syndrome, and decompression of the compressing vessels from the trigeminal rootlets has achieved a high percentage of relief without significant sensory loss.

Peripheral pathway section was the first procedure used for relief of pain, but because of the annoyance of the feeling of numbness, the incidence of paresthesias, nerve regeneration, and the availability of other procedures, it is now rarely used except for denervating trigger areas in the control of trigeminal pain by avulsion of either the supraorbital or infraorbital nerve.

Splanchnicectomy effectively denervates the upper abdominal viscera and is used for relief of intractable pain of the upper abdomen. Although the slpanchnic nerves are part of the sympathetic nervous system, it is the interruption of the visceral afferent fibers coursing within the sympathetic nerves that gives the relief of pain. Their cells of origin are in the dorsal root ganglion, as are the cells of all peripheral sensory nerves, but since they have no surface innervation and paresthesias of denervation are always referred to the surface, painful paresthesias are not seen following their interruption. Since at times with malignant disease of the upper abdomen somatic sensory fibers in the intercostal nerves are involved, dorsal root section or chordotomy may be more effective than splanchnicectomy.

Section of Central Nervous System Pathways

When Martin in 1911 made an incision in the anterolateral quadrant of a patient's spinal cord, the era of surgical control of intractable pain commenced. Anterolateral chordotomy has remained an effective means of controlling intractable pain, and many surgerons have contributed their skill and knowledge to modify Martin's original procedure so that it is a simple and reliable method for production of analgesia. Section of the tract in the spinal cord causes no subjective sensory loss and therefore rarely causes dysesthesias or paresthesias, but it has limitations. Since the fibers projecting to the anterolateral quadrant of the spinal cord ascend three to four segments before crossing in the spinal cord, incision of the quadrant gives loss of pain and temperature sensation on the opposite side of the body three or more segments below the level of the chordotomy. Thus, even a perfectly performed high cervical chordotomy fails to block pain in the upper arm, shoulder, or neck; therefore the procedure is best used for control of pain below the upper thoracic level. The loss of pain, although marked, can be demonstrated to be incomplete, and with time islands of pain perception appear, and with them the original pain usually returns. This may take a year or more to occur and limits in part the usefulness of the procedure for pain from benign conditions.

Lesions further cephalad in the spinothalamic tract, that is, at the midbrain, although causing loss of pain without subjective sensory loss, may also cause dysesthesias or the sensation of pain from nonpainful stimuli. The cause of this dysesthesia is unknown.

Section of the descending, or spinal, tract of the trigeminal nerve in the medulla caused loss of pain and temperature sensation in the ipsilateral face. It is used to control pain in the face, but since the operation has a higher morbidity and mortality than preganglionic section of the trigeminal nerve in the middle fossa, it is usually used only when it is feared

a patient would not tolerate anesthesia of the face or when paralysis of the motor portion of the trigeminal on the opposite side is already present. In this later condition the danger of bilateral paralysis of the muscles of mastication by approaching the nerve distad is higher than the morbidity of medullary tractotomy.

Stereotaxic lesions have been placed in the posterior medial thalamus in the area of the centramedian and nucleus parafascicularis, structures suggested by electrophysiologic and axonal degeneration studies as projections of the medial spinal reticular tracts. These procedures have been reported to relieve pain without any loss of pinprick or temperature sensation in the periphery, but their effectiveness is short-lived. It can be hoped, however, that with more basic information lesions for control will be devised that either interrupt major projections of the impulses defining a stimulus as noxious or increase the blockade of such impulses as they attempt to pass each controlled synaptic junction.

Procedures to Alter Affective Response to Pain

On a basis of the work in primates of Fulton and Jacobson, Egas Moniz in 1935 persuaded his neurosurgical colleague Lima to perform a prefrontal lobotomy on a psychiatric patient with marked anxiety as part of the basis for the psychiatric problem. The success of the procedure prompted many other surgeons to try prefrontal lobotomy for psychiatric illness. Altered affect, particularly in response to peripheral stimuli, and relief of anxiety were seen in the patients just as they had been seen in the animals, and it seemed probable that the reaction of the patients to pain might be similarly altered. Van Wagenen in 1942 performed bilateral prefrontal lobotomies on a mentally ill patient who had phantom limb pain, and his success in altering the patient's complaint prompted other surgeons to use the same procedure in patients with intractable pain. Experience with the procedure of prefrontal lobotomy has shown that the lobotomy patient is unconcerned with the pain but when questioned still reports the pain as present. This lack of concern is no different for pain than it is for other stimuli, and therefore the procedure alters not only the response to pain but the entire affect of the patient. Lesions of the nonspecific nuclei of the thalamus, as in the dorsal median nucleus, lesions of the limbic system, such as cingulomotomy, or excisions of gyri in the prefrontal cortex all produce the same response with varying degrees of affective change.

The major limitation of these procedures is this change in personality. The same effect can be obtained with psychotherapeutic drugs, such as chlorpromazine, but with drugs it can be reversed. For this reason, the procedures for altering affective response to pain are rarely used but still should remain a part of the physician's armamentarium for care of the terminally ill patient whose fears make the situation and the pain unbearable for the patient and the family alike, and for the occasional patient where relief of anxiety will make it possible to resume a more normal life.

Section of the Efferent Arc of the Vasomotor Reflex

Pain that occurs in diseases which cause alteration in blood flow of an extremity, such as Raynaud's syndrome or erythromelalgia, or conditions which cause marked vasospasm, such as causalgia following nerve injury, and the pain of obstruction of the major vessels in an extremity where circulation can be reestablished by dilatation of collateral vessels will respond to sympathectomy. Much has been written about possible sensory function in the

sympathetic nervous system; however, aside from the visceral afferent fibers in the splanchnic nerves, no evidence of sensory function has been demonstrated. The clinical experience that sympathectomy does not relieve pain unless evidence of altered blood flow is present before sympathectomy bears this out. The occasional exception experienced by surgeons is more likely related to the placebo effect of any form of therapy rather than to alteration in peripheral afferent pathways.

Surgical treatment for pain is a useful adjunct to the care of patients and can be effective in prolonging useful life in many situations. An understanding of what it can and cannot do and an awareness of its limitations are an important aspect of its use. The striking relief afforded a patient with intractable pain of malignant pelvic tumor and the return of such a patient to normal interpersonal relationships provide an experience that can demonstrate to any physician the effectiveness of chordotomy. The failure of ablative surgical procedures for pain in the patient using pain as a means of removing himself from an intolerable situation should demonstrate just as strikingly to the physician that no therapy should be applied just because a symptom is present.

Suppression of Pain by Stimulation

Counterstimulation based on theories foreign to most Western neurophysiologic concepts has been used for centuries. Unfortunately, scientific evaluation of the results is not available, and allegorical reports not recognizing placebo effects, emotional factors, and the striking immediate effect of one pain to suppress a less severe pain are the only evidence available for evaluation. More recently, electrical stimulation applied to peripheral nerves, dorsal columns of the spinal cord, and various subcortical cerebral structures have been used for a variety of intractably painful states with varying clinical results.

Although the initial rationale for using these techniques was based on physiologic concepts, the development of the techniques appears to be more empirically than theoretically based. Stimulation of peripheral nerves is done for painful dysesthesias limited to an area innervated by a single nerve. The technique is based on electrophysiologic evidence that the larger nonpainful fibers of peripheral nerves inhibit the subsequent spinal cord activity secondary to smaller fibers, the latter being essential for pain conduction. Stimulation of nonpainful myelinated fibers by implanted cuff electrodes on peripheral nerve has had limited success in alleviation of pain, and the method is limited to painful entities in a single nerve distribution. It has been more effective in the upper extremity than in the lower extremity.

Electrical stimulation of the spinal cord dorsal coumn by implanted electrodes has been used for more diffuse intractable pain, particularly of the lower extremities. A theoretic basis for this type of stimulation includes increasing descending inhibition in the dorsal root entry zone, and experimental evidence that such stimulation inhibits the central conduction of smaller fiber activity has been demonstrated. Using nonpainful paresthesias to determine the threshold, implanted dorsal column stimulators have had effective short-term results for relief of intractable pain in many patients.

Evidence of the production of effective analgesia in rats by stimulation of the raphe's nuclei of the brainstem, the presence of opiate receptors in the paraventricular and brainstem gray matter in vertebrates, and the identification of endogenous polypeptides, called

endorphins, with opiate properties have led to techniques for stimulating the medial thalamic and upper brainstem nuclei to control pain. These techniques consist of stereotaxically placed electrodes and implanted self-contained stimulators that the patient can control with a radiofrequency generator placed over the implanted stimulator antenna. Evidence that a system relating to the action of opiates is being activated by such stimulation includes the blocking of analgesic effects with naloxone, an opiate antagonist. Initial reports of the results of this technique are encouraging, but long-term results are not yet available and evaluation of complications awaits further experience.

Structural Abnormalities of Axial Skeleton

General Considerations

Anatomy. The vertebral column with its ligaments and musculature serves two functions: the support of body weight and protection of the neuraxis. The same dual role is played by the basiocciput, which may be considered from an embryologic and functional standpoint as an extension of the spine. In health, the spine is a sturdy yet flexible weight-bearing structure. Most of its weight-bearing property is provided by the vertebral bodies and intervertebral discs. The neural arch formed by the pedicles and laminae complete a bony ring at each vertebral level defining the vertebral canal, while the articular processes and attached facets bridge the intervertebral spaces posterolaterally, providing further support and protection. The spinous and transverse processes of the neural arch serve as attachments for the spinal musculature. The ligamentum flavum and ther posterior atlantooccipital ligament, both thick elastic structures, complete the bony and ligamentous tube which extends from the foramen magnum to the lowest part of the sacrum. In the normal skeleton there is ample room within the foramen magnum and the vertebral canal for the contained neural and vascular elements, and the intervertebral foramina provide unrestricted passage of the nerve roots and blood vessels.

There is a considerable discrepancy between the cross-sectional area of the vertebral canal and that of the spinal cord at all levels, the additional space being occupied by the cerebrospinal fuid-filled subarachnoid space and the fat-filled epidural space. The fluid can fat suspect and cushion the neuraxis and, because they are displaceable, provide room for lateral and anteroposterior movement of the neuraxis that must take place with normal motion of the axial skeleton. Radiologic and postmortem studies have shown that the spinal cord and nerve roots also undergo grossly perceptible axial movement when the spine is flexed or extended. The spinal cord and roots must, therefore, be free to move up and down the vertebral canal, and the stress applied to the nerve roots through the movement of the peripheral nerves must be relieved by movement within the intervertebral foramina.

Pathophysiology. Structural abnormalities which markedly decrease the dimensions of the foramen magnum, the vertebral canal, or the intervertebral foramina may result in damage to the nervous system. The mechanism of injury is primarily that of direct compression of nervous tissue, although in some instances concomitant interference with blood supply may be a factor. Injury to the nervous system may be caused by acute angulation of the vertebral axis, subluxation of one part of the axial skeleton upon another, or encroachment of a mass upon the vertebral canal or intervertebral foramina. Not only the degree but the rate of compression is important. Angulation of the spin, for example, in

idiopathic scoliosis may be extremely severe but rately causes neurologic deficit because of the ability of the cord and roos to accommodate to their distorted position. In contrast, less pronounced angulation from an acute process such as collapse of vertebra in an osteoporotic spine may result in complete destruction of the spinal cord.

The causes of structural abnormalities of the spine which affect the nervous system are extremely diverse. The bones, joints, and associated muscles of the axial skeleton are subject to the same diseases as they are elsewhere in the body. They may be congenital or acquired, local or diffuse. Symptomatic abnormalities often result from a combination of these factors. As an example, the forament magnum and the vertebral canal in achondroplasia is malformed and disproportionately small as compared with the normal size of the spinal cord. A relatively minor degree of disc protrusion or scoliosis in these patients often causes severe neurologic deficit. It is becoming increasingly apparent as careful measurement of the spinal canal becomes more prevalent that individual differences in response to structual disease in the general population can, at least in part, be accounted for by differences in the size of the vertebral canal. Those persons with congenitally small but undistorted vertebral canals are much more liable to major neurologic deficit in response to degenerative disc disease or relatively minor spine trauma.

It is not the intention of this section to discuss all the etiologic agents that may result in structural abnormalities of the spine. What will be emphasized are the often misunderstood structual lesions occurring at the craniovertebral border and the common lesions resulting from disease of the intervertebral discs.

Abnormalities of the Craniovertebral Border

Structural abnormalities involving the basiocciput and the cephalic portion of the cervical spine are uncommon as compared with those of the remainder of the axial skeleton. Their importance lies in the fact that they may produce profound neurologic dysfunction, which is not infrequently misinterpreted as the result of other causes. Craniovertebral abnormalities are usually congenital but may be acquired. Anatomically these lesions divide into two basic categories, those which affect the basiocciput and those which primarily involve the first and second cervical vertebrae. Both limit the space available for the cervicomedullary junction and can produce similar neurologic dysfunction.

Pathology. Maldevelopment of the occipital bone resulting in an abnormally small and irregularly shaped forament magnum occurs in achondroplasia and occasionally in craniosynostosis. More common deformities of the basiocciput are those termed *platybasia (platys,* flat) and *basilar impression.* To accomodate the cerebellum and the brainstem, which lie in approximately the same axis as the spinal cord, the level of the floor of the posterior fossa is normally well below that of the anterior and middle fossa. In platybasia, the entire floor of the posterior fossa appears elevated. As seen in a lateral x-ray of the skull, the angle formed by the floor of the anterior fossa and the posterior border of the clivus approaches 180°, and the posterior fossa is therefore extremely shallow. In basilar impression the margins of the foramen magnum are indented as if the weight of the head had caused it to sink toward the vertebral column. This may be the case in acquired basilar impression associated with rickets and Paget's disease. In both platybasia and basilar impression the capacity of the posterior fossa is reduced, and the dimensions of the foramen magnum, especially the

anteroposterior diameter, are decreased. In accomodating to the small and abnormally shaped posterior fossa, the hindbrain may be distorted with resulting pressure applied, especially to its ventral surface. Thus the lower cranial nerves and the cervicomedullary junction may be compressed directly or stretched, and hydrocephalus can result from local obstruction to cerebrospinal fluid pathways.

The most significant structural abnormality of the upper cervical spine is atlantoaxial dislocation. This can occur for a variety of reasons. As with dislocations at other levels, it may be the result of trauma, infection, or degenerative bone disease. It most commonly, however, occurs in association with maldevelopment of the atlas. The atlas may be fused to the base of the occiput, and the odontoid, which represents the body of the atlas, may be maldeveloped. Failure of fusion of the odontoid process to the body of the axis may occur without other abnormalities. In occipitalization of the atlas, the head can no longer nod at the atlantoocipital joint, and the added stress ont he ligaments holding the odnotoid process in place may eventually produce a dislocation. The same ventral dislocation of the atlas on the axis may occur if the odontoid process is rudimentary or ununited to the atlas. With atlantoaxial dislocation the upper cervical spinal cord may be compressed between the odontoid process and the posterior arch of the atlas. The lower medulla may become compromised as well, presumably by resulting angulation at its junction with the posteriorly displaced spinal cord.

Clinical Manifestations. Structural abnormalities of the craniovertebral border frequently remain asymptomatic until adult life and then frequently are in response to relatively minor trauma. Cranial nerve signs include disassociated sensory loss over the face (descending trigeminal tract) and palatal and vocal cord weakness. Nystagmus and spastic weakness of the extremities are common, and the patient may have loss of position sense, ataxia, bladder dysfunction, and atrophy of the shoulder musculature and small muscles of the hand. Although examination may reveal a short neck and restriction of head movement, diagnosis is confirmed by x-ray studies. Myelography in questionable cases usually reveals a partial or complete obstruction of passage of the contrast agent.

Treatment. Immobilization of the neck is indicated for patients with minor degrees of impairment. If the dysfunction is progressive or is already severe, decompression of the craniovertebral border is indicated. The mechanisms of continued compression, however, must be demonstrated. Most frequently, the mechanism is occlusion of the outlet of the cerebrospinal fluid from the fourth ventricle, causing hydrocephalus and increased intracranial pressure. A shunt from the ventricle to the venous system, as described later under Hydrocephalus, will often reverse this process and allow a return toward normal neurologic function. If hydrocephalus is not found, air myelography or metrizamide myelography with CT scanning will demonstrate, in most cases, the direction of the compression of the cervical medullary junction and thus indicate either anterior or posterior decompression of the area. In atlantoaxial dislocation, surgical procedures should be done with the patient in skeletal traction and stabilization obtained by bony fusion.

Degenerative Disease of the Intervertebral Disc

Degenerative disease of the intervertebral disc is one of the most common, yet one of the least understood and often one of the most mistreated, disorders of the spine (see Chap

43). Although many patients have benefited from removal of herniated portions of an intervertebral disc compressing neural tissue, too many have been crippled by ill-advised operation for symptoms mistakenly interpreted as being due to this same cause. Just as not all right lower quadrant abdominal pain is caused by appendicitis, not all neck and low back pain is caused by surgical disease of the intervertebral disc.

Anatomy. The intervertebral disc is well suited for its task of supporting considerable weight while still allowing mobility of the spine. It is classically and practically described as consisting of two parts: a tough yet slightly flexible outer ring, the annulus fibrosus, which joins the periphery of adjacent vertebral bodies to one another, and a semisolid plastic center, the nucleus pulposus, interposed between the hyaline cartilage faces of the vertebral bodies. The joint is further reinforced by the anterior and posterior longitudinal ligaments, which extend the length of the spine. Actually, the intervertebral disc is a composite sturcture in which three zones grading into one another may be recognized. The outer zone, which may be regarded as the joint capsule, is composed of lamellae of interlacing bundles of fibrous tissue that blend with the longitudinal ligaments. Beneath this fibrous capsule and intimately adherent to it lies a thick envelope of fibrocartilage that in turn surrounds and attaches to the less dense nucleus pulposus. This latter substance is composed primarily of collagenous fibrils and cartilage cells suspended in a fluid matrix. In younger persons remnants of the notochord may be found here as well. Motion between vertebral bodies is allowed by the flexibility of the outer layers, the more fluid center responding to variations in weight distribution by flowing into areas of least pressure within the confines of the annulus.

Etiology and Pathology. Although the intervertebral disc may be damaged by infection, collagen disease, and severe trauma, it is, in comparison with other weight-bearing structures, particularly liable to early degenerative changes. Knowledge of the basic cause of this deterioration, which secondarily involves surrounding bone as well, is lacking, but it is at least presumably in part a response to the trauma of daily normal activity. It is more prevalent in men than in women and in laborers than in sedentary workers. Degenerative changes occur most frequently in portions of the spine where there is a transition between a relatively mobile segment and a less mobile one, namely, in the lower cervical and lower lumbar region. This distribution again implicates trauma as a causative factor, since there is relatively more motion and therefore more stress at these levels.

Pathologically there is thinning of the fibrous layers of the annulus, destruction of fibrocartilage, and dehydration of the nucleus pulposus. The interspace becomes narroed, and presumably because of loss of normal joint function, there is frequently new bone formation (osteoarthritis) at the margins of the interspace. These changes are often relatively asymptomatic, producing only transient local pain and the progressive loss of spine mobility usually taken as a matter of course with advancing age. Under certain circumstances, however, essentially the same degenerative processes may have more serious consequences. The nucleus pulposus may herniate through the weakened annulus and/or large bony spurs may develop to project into the vertebral foramina. Of the two processes, herniation of the nucelus pulposus is a much more common cause of neurologic dysfunction and occurs at an earlier age. Approximately three-quarters of symptomatic herniations of the nucleus pulposus occur between the age of thirty and fifty years. Neurologic disability resulting from osteoarthritis occurs predominantly in the individuals fifty years of age or older.

The effects of herniation of the nucleus pulposus depend on its location, both in respect to the transverse plane and the disc involved, its size, rate of development, and individual differences in surrounding structures. The most common type of herniation of the nucleus pulposus is not through the annulus but rather into the cancellous portion of the vertebral bodies, the so-called Schmorl's nodules. Such herniation produces no neurologic deficit but by impairing joint function often results in reactive bone formation at the disc margins. Herniation of the nucleus pulposus through the annulus may likewise be asymptomatic. It most commonly occurs at its weakest portion just lateral to the posterior longitudinal ligament near the intervertebral foramen. Herniation into the center of the vertebral canal is relatively uncommon. When it does occur, however, it may result in compression of the spinal cord or of multiple nerve roots if it is below the conus medullaris. The size of the herniated mass varies considerably; it ranges from a protrusion still covered by thinned-out annulus to complete extrusion of the nucleus pulposus as well as the fibrocartilage layers of the annulus into the vertebra canal. The largest intervertebral discs are those in the lumbar region, and, as might be expected, the largest herniated masses occur there. Herniations of the same size are better tolerated in the lumbar area than in other regions, since the spinal cord ends at the thoracolumbar junction, and the lumbar vertebral canal and the intervertebral foramina are larger.

Occasionally in response to severe trauma, especially flexion injuries, an acute rupture of the annulus occurs with massive herniation of the nucleus pulposus and immediate onset of severe neurologic deficit. In the vast majority of patients, however, signs and symptoms resulting from herniation of the nucleus pulposus are more chronic. Although patients often relate their disability to a recent event, such as bending, turning, or lifting, they almost invariably also give a history of past neck or back pain with or without symptoms of nerve root compression. Herniation of the nucleus pulposus is therefore usually a gradual and intermittent process. Signs and symptoms appear when surrounding pain-sensitive structures, such as the annulus, longitudinal ligaments, and dura, are stimnulated or nervous tissue, usually roots, is compressed. Anatomic and pathologic adjustments then often occur, and the symptoms regress, perhaps to reappear when the mass enlarges or anatomic relationships change.

Clinical Manifestations. The clinical picture produced by the common posterolateral disc herniation is characteristic. Diagnosis often can be made by history alone. Signs and symptoms are primarily those of extradural mass: pain over the distribution of the nerve root involved and evidence of lower motor neuron functional loss. Complaints of sensory disturbance over the peripheral distribution of the nerve root involved is commonly of a "pins and needles" type or just a slightly numb feeling. While weakness may be a symptom, it is much less common. The other root signs are loss or decrease in the deep tendon reflex and weakness and atrophy. Examination of the involved area of the spine reveals muscle spasm that may cause loss of normal lumbar or cervical lordosis. In lumbar lesions scoliosis is frequently present, the convexity usually occurring on the side of the lesion. Motion in the affected portion of the spine is limited and painful, especially on lateral flexion toward the lesion. Palpation over the corresponding spinous process and along the major nerve trunks produces pain. Maneuvers such as straight-leg raising which increase the tension of the nerve roots are painful.

Diagnostic Findings. Plain x-rays may be normal or reveal distortion of alignment of the spine produced by muscle spasm. They may, however, show narrowing of the interspace, lipping of the margins of the vertebral bodies bordering on the disc, and osteoarthritic spurs. X-rays also serve to rule out bone erosion that may be present with spinal neoplasms and congenital bony anomalies such as spondylolisthesis. Computerized axial tomography defines the cross-sectional diameter of the spinal canal including osteoarthritic and facet hypertrophy encroachment on the spinal canal and intervertebral foramen. In cases of myelopathy secondary to stenosis, CT scans both can be diagnostic and can outline, in combination with positive contrast, the extent of spinal cord compression. The later-generation CT scanners can be used to diagnose ruptures of intervertebral discs, but myelography with or without scanning may be necessary for final verification of a herniated disc, especially in the cervical and thoracic region. This procedure is best carried out after making the decision to operate based on the course of the disease and the findings on examination.

Herniated Lumbar Disc

The highest incidence of symptomatic herniation of the intervertebral disc is in the lumbar region. Ninety-five percent of lumbar herniations occur at the last two interspaces. The relative frequencies of herniations at these two interspaces are approximately equal. Most of the remainder of the herniated lumbar intervertebral discs occur at the third lumbar interspace. The vast majority of herniations in the lumbar as well as in other locations are unilateral. The disposition of the nerve roots in the lumbar space is such that posterolateral herniation does not compress the nerve existing at the corresponding intervertebral foramen but rather impinges on the set of anterior and posterior nerve roots, contained in a single dural sleeve, which cross the disc in their course of the foramen immediately caudad.

Clinical Manifestations. Herniation at the interspace between the fourth and fifth lumbar vertebral bodies usually compresses the fifth lumbar roots, and herniation at the disc located at the lumbosacral junction produces signs and symptoms referable to the first sacral roots. The fifth lumbar anterior root serves as a major supply to the anterior crural muscles, which dorsiflex the foot, and the peroneal muscles, which evert and plantarflex the foot. The dermatome served by the fifth lumbar posterior root includes the anterolateral aspect of the leg and crosses anteriorly at the ankle to supply the medial aspect of the foot including its dorsum. Compression of the fifth nerve root by a herniated nucleus pulposus occasionally produces a foot drop. The motor deficit, however, is usually less severe; the most common finding is weakness of dorsiflexion of the great toe. Hypesthesia and hypalgesia are usually most evident on the dorsum of the foot between the great and second toes. The motor root of the first sacral nerve supplies some innervation to the gluteal and hamstring muscles but gives a large supply to the muscles of the calf and the small muscles of the foot. Its sensory distribution covers a narrow strip on the posterior aspect of the leg and the lateral aspect of the foot. The most common motor deficit in patients with herniation at the lumbosacral interspace is weakness of plantal flexion. Minor degrees of weakness may be brought out by having the patient attempt to walk on his toes. In addition, the ankle reflex is diminished or absent, and sensory deficit is found on the lateral aspect of the foot.

Treatment. For the majority of patients with herniated lumbar intervertebral discs the appropriate treatment includes a regimen of strict bed rest on a firm mattress, local heat, and anaglesics. Pain usually subsides in 1 to 2 weeks, and the patient may then be started on a

graded program of exercises designed to strengthen the back musculature. The patient should be instructed to refrain from heavy lifting and from activities that involve sudden bending or twisting of the lumbar spine. Early operation is reserved for those patients who have evidence of major neurologic dysfunction such as a foot drop or bowel and bladder disturbances which indicate a massive disc protrusion. If the patient has not responded to conservative management in a few weeks or is losing considerable time from work each year from repetitive episodes, operation should probably be carried out. Back pain alone is not an indication for operation. The aim of the operation, done through a limited hemilaminectomy, is to relieve nerve root compression by excision of the mass of herniated nucleus pulposus. Relief of signs and symptoms referable to such compression is usual, but many of the patients have residual low back pain. In our experience fusion of the vertebrae does not often solve this latter problem.

Herniated Cervical Disc

The lower cervical region is the next most common site of herniated discs. Approximately 90 percent of the herniations in this region occur at the interspaces between C_5 and C_6 and between C_6 and C_7 with the lower level predominating. In contrast with the lower lumbar region, cervical nerve roots are short and almost horizontal.

Clinical Manifestations. Relatively small herniations are often symptomatic, usually lying just beneath the corresponding roots as they enter the intervertebral foramen. It is fortunate that large or medially placed herniations are rare, for when they do occur, they often result in quadriparesis.

With lateral herniations at the lower cervical levels, patients complain of neck pain radiating into the corresponding shoulder and arm. Referred interscapular pain is also common. Localization of the compressed roots by physical signs is somewhat less precise than in the lumbar area. Compression of the sixth cervical roots by posterolateral herniation at the C_5-C_6 interspace usually results in clinically evident weakness of the biceps muscle, depression or absence of the biceps tendon reflex, and sensory loss on the dorsal and lateral aspect of the thumb and radial aspect of the hand often including the index finger. The syndrome of the seventh cervical roots includes weakness of the triceps muscle, decrease or absence of the triceps reflex, and sensory deficit of the distal portions of the corresponding dermatome, which is medial to that of the sixth cervical dermatome. It usually includes both the dorsal and palmar aspects of the index and middle fingers.

Treatment. The rationale of treatment of cervical disc herniation is similar to that in the lumbar region. Signs and symptoms in the majority of patients will regress with conservative management. Neck traction is especially useful. Operation is indicated only in patients with evidence of spinal cord compression and those not responding to conservative measures. Central herniations are best removed through the interspace from the anterior aspect of the spine. The common lateral herniations can be removed effectively either from the same anterior approach or by hemilaminectomy.

Herniated Thoracic Disc

Disc herniation in the thoracic region is rare. When it does occur, it is usually in the lower thoracic spine and is apt to produce major neurologic deficit, often with little associated pain. This lesion may be suspected on the basis of a past history of trauma to the thoracic spine, but it is usually clinically indistinguishable from the more common neoplasms occurring in this region. Diagnosis is made by x-ray studies. There may be narrowing of an interspace and, in contrast to disc disease in other regions, calcification in the interspace. Myelography is essential for diagnosis. Results of operation by the classic approach, ie, laminectomy, are in general poor with no improvement or with increased deficit in about one-half of the patients reported in larger series. The reasons for these poor results include the fact that most of these lesions are anterior to the cord and are firm and partially calcified. Their removal with a posterior exposure therefore often necessitates major retraction of an already compressed spinal cord. Removal of the herniated disc by approach through the interspace, either by removing the adjacent ribs and transverse processes or by going through the chest cavity, has been more effective in relieving the myelopathy without causing further neurological deficit.

Spondylosis

Hypertrophic bone changes are not infrequently associated with disc herniation even in younger individuals. They are especially prevalent in cervical herniations in which spurs of bone projecting into the intervertebral foramina are often a contributing cause of nerve root compression. In relation to its almost uniform presence in the edlerly, hypertrophic bony change alone as a cause of significant neurologic dysfunction is uncommon. However, in some patients with advanced hypertrophic bone disease of the cervical spine, often termed *spondylosis*, major spinal cord and nerve root dysfunction develop. This entity, which usually affects two or more lower cervical interspaces, is characterized by narrowing of the interspaces and extensive bony proliferation on the lips of the vertebral bodies adjacent to the discs, producing transverse ridges which project into the vertebral canal. The corresponding intervertebral foramina are narrowed, and in advanced disease minor degrees of subluxation of one vertebra on another may exist.

Why many persons tolerate advanced cervical spondylosis without significant disability while in others a severe spastic quadriparesis may develop is largely unknown. It has been demonstrated that a greater number of patients with symptomatic spondylosis have congenitally narrow cervical vertebral canals than those without spinal cord signs. A simple compressive mechanism, however, does not entirely suffice to explain the problem, since return of function often does not follow seemingly adequate decompression which includes extensive laminectomy combined with removal of bony spurs and fusion from the anterior approach. Chronic vascular insufficiency resulting from a combination of such factors as arteriosclerosis, compression of a long segments of spinal cord, and compression of the arterial supply entering through the intervertebral foramina may explain the difference in the results of surgical therapy for cervical spondylosis as compared with more localized mass lesions in the same region.

Clinical Manifestations. The clinical picture of symptomatic cervical spondylosis varies considerably. Neck pain radiating bilaterally into the shoulders is frequently but not

invariably present. Examination reveals evidence of compression of multiple cervical nerve roots and signs of cervical spinal cord compression. The course of the disease is also variable. Occasionally there is rapid progression which may lead to a severe spastic quadriparesis within a year or less. More often the onset of signs is insidious, and progression is slow. Spontaneous regression of neurologic deficit is rare, but on the other hand progression of deficit may halt at any point.

Treatment. The variability of the course makes it difficult to evaluate the effectiveness of operative procedures. In the early stages of the disease immobilization of the cervical spine by a well-fitted collar brace may be of benefit. Traction occasionally may reduce root pain but often is not successful. Operation early in the course of the disease should probably be reserved for those patients with involvement of only one or two interspaces and those with obviously small vertebral canals. With severe and seemingly unrelenting progression of neurologic deficit associated with widespread bony changes, the patient should be informed that a halt in progression may be all that can be hoped for from operation.

Pediatric Neurosurgery

Charles C Duncan, Laura R Ment

Children with neurologic and cranial disorders present in most instances a distinct group of problems. The presentation, examination, and care of the young further require different approaches as one progresses from the fetus to the preterm infant, neonate, toddler, and school-aged children. The patterns of the adolescent, in terms of presentation and range of diseases processes, more closely follow those of the young adult than those of the child.

Fetus

Aggressive high-risk obstetrics and real-time ultrasound have provided a means for the diagnosis and treatment of a wide range of congenital lesions of the nervous system including hydrocephalus, dysraphism, and other brain anomalies. Decisions regarding the fetus with congenital CNS anomalies may now be made before birth, with several options available to the parent. Previous to these capabilities, such problems required that decision in most instances be made in the delivery room or during the immediate newborn period.

With prenatal diagnosis, potential options include termination of the pregnancy, early delivery, fetal shunting, cephalocentesis, or no intervention. Which option is chosen is dependent upon the particular findings of a case and parental decision. As yet, no data exist as to improved outcome with aggressive fetal therapy for hydrocephalus or early delivery and shunting.

Preterm Infant

The preterm (PT) infant weighing less than 1.500 g has a 40 percent risk of intraventricular hemorrhage (IVH), and the PT weighing less than 1.250 g has a 50 percent incidence of IVH. Bleeding occurs in most instances in the germinal matrix region, which by

this point in development represents a watershed area of cerebral blood flow. Such hemorrhage has significant influence on the infant's neurodevelopmental outcome.

Diagnosis was carried out earlier by CT scan and more recently by real-time ultrasound. The incidence of hydrocephalus requiring shunting varies considerably from center to center. The use of serial ultrasounds and lumbar punctures appears to decrease dramatically the incidence of hydrocephalus requring shunt. Some of the PT infants may block their aqueduct with clot and require earlier shunting. More frequently, communicating hydrocephalus develops from the decreased absorption of CSF at the level of the arachnoid granulations. With time, in the majority of these children, the error in CSF absorption diminishes and the hydrocephalus resolves.

Infant

For the PT and full-term infant, neurologic evaluation in a meaningful fashion is quite different from that of the older child and adult. Initially, the only cortical function observed is whether the infant is able to fix and follow a visual stimulus. The remainder of the examination is subcortical. Several pediatric neurology texts detail the examination in this age group. For brief repetitive objective examinations by all levels of personnel, the Neonatal Arousal Scale is useful. The scale is based on the infant's response to sound, light, and motor response and may be used from 26 to 52 weeks postconceptual age (Table 42-4).

Table 42-4. Neonatal Arousal Scale

Best response to bell	
Facial and extremity movements	5
Grimaces/blinks	4
Increase in RR/HR	3
Seizures/extensor posturing	2
No response	1
Best response to light	
Blind and facial/extremity movements	4
Blink	3
Seizures/extensor posturing	2
No response	1
Best motor response	
Spontaneous	
Period of activity alternating with sleep	6
Occasional spontaneous movements	5
Sternal rub	
Extremity movements	4
Grimace/facial movements	3
Seizures/extensor posturing	2
No response	1
Total	3-15

Pediatric neurosurgery in this group is primarily directed toward congenital anomalies, although acquired disorders may rapidly appear which would include posthemorrhagic hydrocephalus, subdural hematoma, and depressed fractures. Additionally, occasional tumors may present very early.

Myelomeningocele. Dysraphisms along the entire neuraxis may occur. Myelomeningocele is the most common of these and provides an approach to these disorders. With myelomeningocele an incomplete closure of the neural tube occurs between 21 and 25 days postconception. Diagnosis in early stages of pregnancy may be suspected by an elevated maternal serum alpha-fetoprotein (AFP) or diagnosed with amniocentesis and AFP measurement or real-time ultrasound. Hydrocephalus occurs in 80 to 90 percent of cases, with the exception of the lipomyelomeningocele where hydrocephalus occurs only rarely. Virtually 100 percent of patients with myelomeningocele have Arnold Chiary Type II malformations as well, which means that the structures of the posterior fossa are caudally displaced, such that the cerebellar tonsils, medulla, and IV ventricle are in the cervical canal.

Treatment of these patients has been the subject of extended controversy in neurosurgical literature for many years. The accepted approach for most pediatric neurosurgeons in the USA is for aggressive treatment if there is any significant chance for survival. Closure of the back does not improve lower extremity function. Rather, it protects the neural placode and the nervous system from mechanical trauma and infection. The neurodevelopmental outcome of children with myelomeningocele is significantly better than that of unselected cases of hydrocephalus. The care of patients with myelomeningocele is a multidisciplinary approach which must continue throughout the patient's life.

Craniosynostosis. Abnormalities of head shape, or craniosynostoses, are also recognized in the early postnatal weeks. The development of a normal head shape requires two primary factors. The first is the growth of normal brain beneath the skull, and the second is functional cranial sutures which permit increase in head size. When craniosynostosis occurs, the brain continues to grow but not in place of the abnormal suture. Total craniosynostosis is quite rare. Other causes of microcephaly are far more common and result from lack of brain growth. Urgent opening of the suture is required in the rare case of total craniosynostosis.

For the more usual cases of metopic, sagittal, coronal, and lambdoidal synostosis, the appearance of the infant's head usually yields the diagnosis. Plane films show the anomalous suture in less than half the cases. Although other investigations, including xeroradiographs and bone scans, may be advocated for diagnosis, the clinical evaluation is the most reliable. With coronal and lambdoidal synostosis, sutures in the skull base are more likely to be involved than with sagittal synostosis.

Patients with sagittal synostosis have very long, narrow heads and usually a prominent ridge along the sagittal suture. Coronal synostosis presents with a narrow anterior portion of the cranial vault and orbits which are angled such that their lateral portion is much more posterior then the medial. Metopic synostosis presents with a similar picture and a prominent midline forehead ridge. Lambdoidal synostosis presents with the ipsilateral occipital being guite flat, the ear on that side protruding and anteriorly placed with respect to the contralateral. As well, the ipsilateral forehead protrudes more than the opposite side. Therapy

is directed toward providing an artificial suture in the form of a linear craniectomy cotaed with silastic film and maximal time for brain growth to reshape the head. Coronal and metopic synostosis, which frequently involves the anterior skull base, requires forward displacement of the lateral superior orbital rims for a good cosmetic result.

Operations such as these for a single fused suture are basically for appearance. In order for the surgery to be as effective as possible, the operation should be carried out quite early in life. As a result, major surgery is carried out in infants on an elective basis. Such operations should be done with maximally experienced anesthesia, pediatric, and pediatric neurosurgical personnel to assure that the safest procedure possible is being done.

Further complex varieties of craniofacial anomalies and incompletely treated patients with simpler synostosis at an older age are beyond the scope of this discussion and frequently require specialized multidisciplinary operative teams.

Young Children

Trauma. Trauma is the most frequently encountered neurosurgical problem in this age group. Preventive measures, such as suitable restraints for automobile, instructing regarding roadways, household and play safety, bicycle helmets, and the identification of children at risk for abuse, are far more effective lifesaving maneuvers than any particular neurosurgical intervention.

Formal neurologic examination of this group theoretically should be like that of older individuals, but it must be couched in play and patience to be effective. With a few notable exceptions, disease processes of a congenital origin will continue to be found in younger adults. Similarly, some symptoms in patients of this age group are quite different from those of older and younger individuals.

Trauma in the young presents three major distinctions from older populatioins. In this group head injury is likely to be a cause of shock. The intracranial compartment with a partially closed head with a fontanelle that may bulge and sutures which will separate is sufficient to contain enough blood to result in hypovolemic shock. Computerized axial tomographys has provided a means for rapid accurate assessment of intracranial pathology. In the instance of trauma even with a widely open fontanelle, real-time ultrasound is not an appropriate diagnostic approach because of the difficulty of visualizing peripheral lesions. A second distinction is the relation of skull fracture to subsequent intracranial pathology. With the malleability of the young skull, dural vessels, for example, may be torn and other significant intracranial injury can occur without fracture. As well, detailed neurologic examination may be very difficult to carry out following head injury. As a consequence, most children sustaining loss or alteration of level of consciousness should be carefully observed in hospital. The third distinction relates to child abuse. Any child who has an injury the cause of which is not clearly understood should be considered as having sustained potential abuse. Care should involve clarifying the mechanism of injury, checking for associated recent or old injury, and giving appropriate follow-up.

Tumors. In this age group headache should be considered the most dramatically different symptom from older populations. While in adolescents and adults this may be one

of the most frequently found complaints, in young children such a complaint requires serious consideration. With the availability of computerized axial tomography, appropriate workup may be carried out with minimal risk and reasonable expense. Tumors in this age group usually present as headache and not as seizure. As the children become older, seizure becomes a more likely presenting symptom. Conversely, seizure was rarely thought to be a heralding event for an intracranial structural lesion in the young. However, as computerized axial tomography has become widely available, far more structural lesions in this age group are found than were previously expected.

Tumors tend to be midline. These include medulloblastoma and ependymoma in the IV ventricle and pinealomas, craniopharyngiomas, and neuroentodermal tumors supratentorially. Other tumors may present laterally. Choroid plexus papilloma, which is the classic example for the overproduction of CSF, usually occurs in the lateral ventricle. A vast variety of other intracranial masses also occur.

Vascular malformations and aneurysms occur in these age groups as well. The general approach is as in older individuals. Aneurysms, while unusual at this age, may have a tendency toward a higher and more rapid rebleed rate which encourages earlier operative care.

Older Children and Adolescents

From the surgical standpoint the care of these patients begins to approach that of the young adult. Dealing with the adolescent and older school-age child may be quite difficult. The problem is not so much a different category of diseases, or a different type of neurologic examination; instead, the problem is how to deal with these patients on an appropriate level such that trust, honesty, and rapport are developed.

Reye's syndrome is the most significant distinctive disease process in this age group. It frequently follows a recent or resolving viral illness, especially chicken pox or influenza B, and is characterized by an altered level of consciousness with elevated values of arterial ammonia. When the altered level of consciousness approaches coma, aggressive control of the intracranial pressure is required.

Hydrocephalus

Much of pediatric neurosurgery deals with the shunt for hydrocephalus. In a number of large hospitals in the USA the shunt is likely to be the second or third most commonly performed operation in the hospital. Similarly, the children requiring this care are likely to be inpatients in essentially all of the age groups of the pediatric service. Hydrocephalus is not a primary problem; rather it is a second-order manifestation of some other process. The primary processes include aqueductal stenosis, dysfunction of the arachnoidal granulation, scarring of the subarachnoid space over the cerebral convexities or base, block of the flow of CSF by tumor, and overproduction of CSF from a choroid plexus papilloma. Situations such as arachnoidal granulation, dysfunction, and scarring of the subarachnoid spaces may result from bleeding, infections, or tumor. Enlargement of the ventricular system to produce hydrocephalus is the result of these processes.

By convention hydrocephalus has been divided into two broad categories: communicating and noncommunicating hydrocephalus. In noncommunicating hydrocephalus there is an obstruction to the flow of CSF from inside the ventricular system. In communicating hydrocephalus the obstruction is outside the ventricular system.

In most instances, therapy of hydrocephalus requires the placement of a pressure-regulated shunt system. This consists of the insertion of a catheter into the ventricular system, a valve, and a distal catheter to an area where CSF is absorbed. A wide range of distal sites have been tried and used. At the present time most shunts are ventricular-peritoneal. The distal catheter lies free in the peritoneal cavity where fluid absorption takes place.

Shunt systems have many disadvantages, but none can compare with the usual devastation of untreated hydrocephalus. The shunt crosses enormous anatomic barriers, the plastic does not grow, and the valve system treats a volume transfer problem by pressure regulation. These situations indicate the need for continued care of these patients as well as the need for advances in treatment modalities.

The outcome of these patients has steadily improved over the past two decades. Follow-up studies carried out in the 1960s indicated that approximately one-third of these children would have a normal intelligence. Follow-up studies in the 1970s indicated that close to two-thirds may have a normal intelligence. In the 1980s follow-up studies indicate that these children have a mean IQ of 90 ± 19. These figures are significantly below the general population. Some of these children do very poorly and may require special education; some are very bright. Overall shunted patients do not perform as well sibling matched control groups. To some extent how well an individual performs depends upon the basic disease process. Performance also depends on several variables such as the prevention of ventriculitis and maintenance of shunt function.