PEDIATRIC NEUROANESTHESIA

Hydrocephalus

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Hydrocephalus is one of the most common neurosurgical problems in the adult and pediatric populations, and accounts for approximately 70,000 hospital admissions per year in the United States.[2] The derangement of hydrocephalus is an abnormal accumulation of cerebrospinal fluid in the ventricular system caused by various pathologic processes.

CAUSES

The following are congenital causes of hydrocephalus:

Stenosis of the aqueduct of Sylvius
Myelomeningocele
Dandy-Walker syndrome
Mucopolysaccharidoses[59]
X-linked hydrocephalus
In utero intraventricular hemorrhage[25, 30, 33, 37]
Maroteaux-Lamy syndrome[55]

The following are acquired causes of hydrocephalus:

Intraventricular hemorrhage of prematurity[41, 64]
Space-occupying intracerebral cysts and tumors
Infections (e.g., meningitis)

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Hydrocephalus is present almost universally in patients with myelomeningocele, the most common congenital anomaly of the CNS and a major cause of serious developmental disability. Myelomeningocele is characterized by protrusion of the meninges through a midline bony defect of the spine, forming a sac containing cerebrospinal fluid and dysplastic neural tissue. The Chiari II malformation, including herniation of the cerebellum and hind brain through the foramen magnum into the cervical spinal canal, occurs with this anomaly, and is usually associated with hydrocephalus. The severity of hydrocephalus usually worsens after the neurosurgical repair of the defect in the newborn, requiring placement of a ventricular shunting system.

In the newborn, the most common cause of hydrocephalus is obstruction of the aqueduct of Sylvius, a long, narrow structure that is the most frequent site of obstruction in the ventricular system. Obstruction at this point prevents the free passage of cerebrospinal fluid from the lateral and third ventricles to the fourth ventricle, and from there on to the subarachnoid space.

The newborn's hydrocephalus may require the initial placement of a ventricular reservoir to control the hydrocephalus by daily drainage of cerebrospinal fluid. Conversion of the reservoir to a ventriculoperitoneal shunt with shunt revisions may be necessary to improve survival.[41] The incidence of intraventricular hemorrhage is closely related to the degree of prematurity. In low birth-weight infants, the incidence of intracranial hemorrhage is reported to range from 30% to 50%. Thirty-five percent to 60% of these patients develop an increase in ventricular size, and some require eventual cerebrospinal fluid diversion for progressive hydrocephalus.[41, 44] The operative procedures for shunts in this subgroup have a mortality rate of up to 31%.[34, 44] The outcome, however, is determined by the extent of the intraparenchymal portion of the hemorrhage.

Long-term intellectual and motor development are determined mainly by factors that occur before cerebrospinal fluid removal and diversion for control of hydrocephalus. Motor outcome is significantly related to the extent of hemorrhage, the presence of seizure activity, and the gestational weight. The extent of hemorrhagic infarction at birth is the most significant determinant of survival and cognitive development. The number of shunt revisions in these children also is related significantly to survival; those infants who died early had fewer shunt revisions,[41] suggesting the importance of early and aggressive management of hydrocephalus in the preterm infants.

Hydrocephalus can be congenital or acquired, and the lesion may be of the communicating or noncommunicating type. In the noncommunicating type, there is obstruction to the flow of cerebrospinal fluid. Although there is a free flow of cerebrospinal fluid in the communicating type, there is overproduction or decreased absorption of cerebrospinal fluid.

A genetic form of hydrocephalus also exists, in which the gene is located on the X chromosome. The hydrocephalus is characterized by

stenosis of the aqueduct of Sylvius, and has a frequency of approximately 1 in 30,000 male births.[28]

The neural-cell adhesion molecule L1 (L1CAM) plays a key role during embryonic development of the nervous system, and is involved in memory and learning. Mutations in the *L1* gene are responsible for four X-linked neurologic conditions: X-linked hydrocephalus, MASA (mental retardation, aphasia, shuffling gait, adducted thumbs) syndrome, complicated spastic paraplegia type I, and X-linked agenesis of the corpus callosum. The clinical pictures of these four *L1*-associated diseases show considerable overlap, and are characterized by corpus callosum hypoplasia, mental retardation, adducted thumbs, spastic paraplegia, and hydrocephalus (i.e., the *CRASH* syndrome).[24]

Tumors in and around the third ventricle can obstruct the cerebrospinal fluid before it enters the third ventricle from the lateral ventricles, or can prevent its egress from the posterior portion of the third ventricle into the aqueduct of Sylvius. A small mass can obstruct the free flow of cerebrospinal fluid at the foramen of Monro or at the aqueduct of Sylvius.

Absorption of cerebrospinal fluid depends on bulk flow, passive or facilitated diffusion, and active transport of specific solutes. With the rare exception of cerebrospinal fluid overproduction by a choroid plexus papilloma, hydrocephalus results from impaired cerebrospinal fluid absorption. Production of cerebrospinal fluid in hydrocephalus is normal or nearly normal.[43]

PRESENTATION

Presenting signs and symptoms of hydrocephalus are related to the increase in intracranial pressure, and depend on whether the hydrocephalus is acute or chronic. When acute, hydrocephalus may be rapidly fatal.[58]

Acute hydrocephalus develops in the setting of sudden obstruction of the ventricular system, with lack of compensation for the increase in intracranial volume which may be caused by intraventricular hemorrhage in the premature infant, hemorrhage into a tumor, or expansion of a colloid cyst of the third ventricle. The resultant sudden severe headache may go undiagnosed in the nonverbal and developmentally delayed population. Vomiting, dehydration, lethargy, neurogenic pulmonary edema, and coma are signs of impending catastrophy. If appropriate therapy, such as decompression, is not instituted in time, the increase in intracranial pressure may lead to herniation of the brainstem, respiratory and cardiac arrest, and possibly death.[58]

The more chronic the hydrocephalus is, the slower the development of signs and symptoms. Chronic hydrocephalus can occur because of congenital aqueductal stenosis, meningitis, and spinal cord tumors. Slowly progressive signs range from irritable behavior, poor school performance, intermittent headaches, rambling speech, bizarre behavior,

and confusion to lethargy, weakness, unsteady gait, seizures, and incontinence. If intracranial pressure is increased grossly, papilledema also may be present.

In the neonatal period, the cranial sutures are not fused, causing widening of the sutures and enlargement of the head circumference. In extreme cases, the resultant large size of the head may cause airway problems in the neonate. The symptoms are often nonspecific and minor, and include irritability, poor feeding, and vomiting. In infants and small children, vomiting and decreased oral intake usually are misdiagnosed as a viral or flulike illness or as gastroenteritis.[38]

In pediatric patients, acute obstructive hydrocephalus caused by expansion of a previously undiagnosed intracranial neoplasm may be the cause of sudden and unexpected death.[58] The mechanism of this sudden death is an acute increase in intracranial pressure with resulting cerebral herniation. Pathophysiologically, the increase in intracranial pressure may occur because of small intracranial tumors located at critical positions obstruct drainage of cerebrospinal fluid and cause acute obstructive hydrocephalus[11, 13, 40, 58] or because of hemorrhage into and acute enlargement of a previously unsuspected intracranial tumor with or without cerebrospinal fluid blockade.[10, 48, 58] Particular attention is owed to those children with persistent lethargy, which should be considered a neurologic rather than a nonspecific clinical sign. Heightened awareness of these rare presentations of childhood intracranial neoplasms (e.g., vomiting, headache, lethargy) may lead to swift diagnosis and potentially life-saving intervention.[58]

DIAGNOSIS

Fundoscopic Examination

Fundoscopic evaluation may reveal bilateral papilledema when intracranial pressure is high.[38] The examination may be normal, however, with acute hydrocephalus, and may provide false reassurance.[58]

Lumbar Puncture

Lumbar puncture can be hazardous if the relief of pressure causes herniation of the brainstem, and should not be performed when an increase in intracranial pressure is suspected.[38]

Computed Tomography

Although it is not always easy to detect the cause with this modality, the size of the ventricles is determined easily. Computed tomography may reveal hydrocephalus, cerebral edema, or mass lesions, such as a

colloid cyst of the third ventricle or thalamic or pontine tumor.[58] A CT scan is mandatory when any suspicion of an acute neurologic process exists.[58] The finding of ventriculomegaly warrants immediate neurosurgical consultation.

Magnetic Resonance Imaging

Scans may reveal dilated ventricles or the presence of a mass lesion.

Ophthalmodynamometry

Venous ophthalmodynamometry, although not suitable for continuous monitoring, is a simple and noninvasive method of assessment of intracranial pressure. This technique can be repeated easily, and can be used whenever increased intracranial pressure is suspected in a patient suffering from hydrocephalus, brain tumor, or head injury. It may be used in the differential diagnosis of malfunction of ventricular shunts, gastrointestinal disorders, hypertensive hydrocephalus, and brain atrophy.[23]

Transcranial Doppler

Transcranial Doppler is a noninvasive method of evaluating hydrocephalus. By producing ventriculomegaly and an increase in intracranial pressure, hydrocephalus causes changes in cerebral vasculature and cerebral blood flow velocity. Diastolic velocity decreases and pulsatility index (systolic velocity-diastolic velocity/mean velocity) increases.[31] Transcranial Doppler does not give direct information about changes in cerebral blood flow, but a compromised flow velocity pattern as evidenced by an increase in the pulsatility index can be a sensitive index of impending ischemic injury.[27] The pulsatility index has been used in the diagnosis of blocked shunts in children. Postoperative measurement of the pulsatility index demonstrated a return to values comparable with the control group after shunt revision.[45, 50]

Transcranial Doppler also may be used as a noninvasive monitor in assessing cerebrospinal fluid shunt function. A fall in the pulsatility index correlates with a change in ventricle size in all age groups.[35] The advantage of using this modality lies in the fact that the lack of specificity of the symptoms of shunt obstruction necessitates reliance on examination and measurement. Aspiration of the reservoir may be helpful, but carries the risk for introducing infection.[50] A brain CT scan may show enlarged ventricles, but a previous scan is necessary for comparison; CT scanning also involves a moderate dose of ionizing radiation and transporting the patient to the CT area. Transcranial Doppler provides an inexpensive, portable modality that can be used repeatedly at the

bedside. The accuracy is limited, as with other measurements, by the expertise of the person performing the test.[35]

TREATMENT MODALITIES

A ventriculostomy[43] or an extracranial ventricular drainage system[43, 58] may be inserted before the direct surgical introduction of a ventricular shunt in the initial management of hydrocephalus associated with tumors in or about the third ventricle when the neurologic condition of the patient is deteriorating rapidly. Ventriculostomy is preferred by some neurosurgeons as a temporizing measure, followed by an attempt to reestablish the cerebrospinal fluid circulation at the time of tumor removal, obviating the need for a shunt, if successful.[43] Insertion in the right frontal region is preferred because it is rarely the dominant hemisphere and the ventriculostomy can be more easily placed when the patient is in the ICU. Ventriculostomy also allows for monitoring of intracranial pressure in the postoperative period.[43]

SHUNTING

The following are types of shunting procedures:

Ventriculostomy (frontal or parietal region)
Ventriculoperitoneal shunt
Ventriculopleural shunt
Ventriculoatrial shunt

The following are complications of shunt placement:

Infection[43]
Obstruction[43]
Hematoma[43]
Valve malfunction[43]
Disconnection[15, 52]
Overdrainage[52]
Outgrown shunt[15]
Shunt fracture[15, 39]
Allergic reaction to material[60]
Seizures[43]

Cerebrospinal fluid shunts are the standard for treatment of hydrocephalus; however, they are prone to complications, with up to 16% of shunts requiring revision within 1 month of insertion.[49] Controversy still surrounds the relationship of the cause of hydrocephalus and the incidence of multiple shunt failures.[14, 17, 20, 47, 56, 63]

Several factors are responsible for cerebrospinal fluid shunt system failures, including a particular subgroup of patients,[12, 56] timing of the procedure,[36] types of shunt equipment,[5, 21] and surgical technique.[17] Identification of these risk factors is difficult; however, a recent study indi-

cates that none of the easily remedial factors, such as shunt-valve design, shunt implantation site, length of surgery, and use of antibiotic agents, seemed to be significant. Instead, immutable patient characteristics, such as age and the cause of hydrocephalus, were found to be significant risk factors. There was a higher rate of failure among patients who had undergone shunt implantations at younger than 6 months of age.[20, 62] After initial shunt insertion, the failure rate by 1 year postimplantation is up to 40%.[22]

There is also an increased risk for failure of shunts that were revised fewer than 6 months after implantation.[47] A general response to shunt revision, rather than an infection, is considered to be the cause. The trauma of shunt revision and perhaps the reaction of the ventricular tissue may incite an inflammatory response that predisposes to subsequent shunt obstruction. Shunt failure as a general response to shunt revision suggests that more inert shunt materials or anti-inflammatory medication might reduce repeated shunt failure.[62]

Concurrent surgical intervention also may be associated with an increased risk for shunt failure.[62] Postoperative complication caused by shunt failure after surgical correction of scoliosis in the myelodysplasia population may be as high as 9.1%.[26] Shunt failure after scoliosis correction is believed to be caused by calcification and tethering of the spinal cord, which causes tubing to fracture when mechanical stresses, such as torso lengthening during deformity correction, are applied.[4, 18] In patients with myelodysplasia in whom neurologic deterioration occurs after deformity correction, examination of the shunt tubing for disconnection or fracture should be conducted because shunt malfunction may lead to acute hydrocephalus.[32]

High cerebrospinal fluid protein concentration also may impair shunt performance. Several mechanisms, including reduced flow because of high cerebrospinal fluid viscosity,[46, 54] sticking of the valve components, peritoneal malabsorption,[1, 46] and protein deposition obstructing the lumen,[46] have been described. Recent studies, however, have invalidated these as possible mechanisms.[7-9]

The principal complication of ventriculostomy is infection, with an incidence of 2% to 3% when the duration of monitoring averages 7 days.[43] The infection rate depends on factors that lower the patient's resistance to infection. Another major complication is epidural, subdural, or intraparenchymal hematoma at the site of the ventriculostomy; the hematoma is most often associated with a known or unsuspected coagulation disorder.[43] Seizures originating at the site of insertion are another risk.[16, 19]

Ventriculoperitoneal shunt infections remain a major source of morbidity for patients with hydrocephalus. Most shunt infections occur within a few months of operation. Approximately 10% of shunt infections occur more than 1 year postoperatively.[3]

Certain complications, such as disconnection, obstruction, and infection, are common to all types of shunts; obstruction is the most common problem.[43] Overdrainage of cerebrospinal fluid can occur if the distal slit valve becomes disconnected from the shunt system. The proximally

placed low-pressure valve allows overdrainage of the ventricles by way of a patent subcutaneous fibrous tract from the end of the disconnected shunt into the peritoneal cavity. The syndrome of shunt overdrainage may be recognized by a history of headaches, nausea, and vomiting that improves on lying recumbent, in association with slit-like ventricles on CT scan.[52]

Allergic reaction to foreign material may be responsible for causing cerebrospinal fluid eosinophilic granulocytosis without accompanying inflammation or pyrexia. Prompt steroid treatment with systemic prednisolone can produce dramatic and spontaneous regression of symptoms.[60]

PERIOPERATIVE ANESTHESIA MANAGEMENT

Perioperative anesthesia management depends on the underlying cause of hydrocephalus, associated congenital anomalies, and their effect on the neurophysiology of the child, and whether the signs and symptoms of raised intracranial pressure are present. One must know if these are acute or chronic in nature. A history of meningitis, seizures, altered level of consciousness, posturing, intracranial hypertension, headache, nausea, vomiting, or any signs of dehydration, nystagmus, diplopia, abnormal respiratory pattern, arterial hypertension, or bradycardia also must be elicited. Sudden neurologic deterioration in the pediatric patient is always a concern, and must be investigated and managed promptly.

Premedication

Sedation usually is not required because of patients' altered level of consciousness that is being monitored. Any resultant respiratory depression may cause hypercapnia with further increases in end-tidal CO_2 and grave consequences. As soon as possible, intravenous access should be established in preparation for any possible emergency and also can serve for induction of anesthesia.

Anesthesia

Latex precautions are recommended for patients with myelomeningocele undergoing shunt placement.[57, 61] Inhalation induction of anesthesia is avoided because all inhalation agents dilate cerebral vessels in a dose-dependent manner and may cause increased intracranial pressure. The patient is preoxygenated, and a modified rapid-sequence induction is preferred to minimize the risk for aspiration caused by recent consumption of food or decreased gastric emptying from increased intracranial pressure. Induction of anesthesia usually is done with intravenous thiopental, 3 to 4 mg/kg, followed by a nondepolarizing

neuromuscular blocking agent, such as rocuronium, 0.5 to 0.8 mg/kg,[42] that has a rapid onset and intermediate duration after intravenous administration. This drug has been given in the deltoid intramuscularly (1 mg/kg in infants and 1.8 mg/kg in children) to intubate the tracheas of pediatric patients, and provided satisfactory conditions.[51] The effect on intracranial pressure, however, was not studied.

An inhalational agent is introduced in low concentrations once adequate hyperventilation is established. Muscle relaxation is maintained throughout the procedure, and an intravenous antibiotic, such as vancomycin or ceftriaxone, is given slowly over 60 minutes after checking sensitivity to the drug. If required, a short-acting analgesic is used in small doses. Surgeons usually infiltrate the surgical site to reduce the requirement for intravenous analgesics so that a neurologic assessment may be carried out in the immediate postoperative period. At the end of the procedure, the stomach is well suctioned, and the trachea is extubated when the patient is fully awake.

POSTOPERATIVE CARE

The patient is transported to the recovery room with an oxygen mask, and the vital signs are monitored for 1 hour. Neurologic stability is confirmed before transfer to the floor for continued care.

SUMMARY

Hydrocephalus, one of the most common adult and pediatric neurosurgical disorders, is an abnormal accumulation of cerebrospinal fluid in the ventricular system as a result of obstruction to the flow of cerebrospinal fluid. Causes of hydrocephalus include congenital obstruction, hemorrhage, infection, cysts and tumors, and associated neural tube deformities (i.e., myelomeningocele, Arnold-Chiari malformation). Treatment of hydrocephalus involves surgical implantation of shunt systems to drain the cerebrospinal fluid. Anesthetic considerations involve attention to the possibility of increased intracranial pressure and prevention of aspiration through rapid-sequence intravenous induction and modest hyperventilation until the ventricles have been decompressed.

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