HYDROCEPHALUS

PROGRESSIVE HYDROCEPHALUS: SYMPTOMS AND SIGNS

The etiologies and clinical features of progressive hydrocephalus in 107 children, 56 with and 51 without shunts, were analyzed retrospectively at the Department of Neurology, Royal Hospital for Sick Children, Edinburgh, Scotland. Patients with arrested hydrocephalus, or with ventriculomegaly resulting from atrophic or ischemic brain damage or tumor were excluded. Intracranial pressure was measured percutaneously or through ventriculostomy reservoirs, using a Gaeltec miniature strain gauge transducer. Etiologies included spina bifida (54%), idiopathic (15%), hemorrhage (13%), and meningitis (10%). In those with malfunctioning shunts, symptoms were vomiting, drowsiness, headache, behavioral change, and anorexia; and signs were absent in 25% and included decreased level of consciousness in 18%, acute strabismus (18%), neck retraction (11%), and distended retinal veins (11%). Patients without shunts were asymptomatic in 49%; headache occurred in 33%, and vomiting in 16%. Signs in the nonshunted group included abnormal head growth in 76%, tense fontanelle (65%), scalp vein distention (33%), setting sun sign or absent upward gaze (22%), and neck rigidity (14%). Unusual clinical features included neurogenic pulmonary edema, profuse sweating, macular rash, ptosis, autonomic dysfunction, and neurogenic stridor. Papilledema occurred in only eight cases (8%). The authors emphasize the variability, unreliability, unusual nature, and even absence of clinical symptoms and signs of hydrocephalus with raised intracranial pressure. CT or MRI may not be diagnostic, and direct measurement of intracranial pressure is essential in patients with unexplained clinical features. (Kirkpatrick M, Engleman H, Minns RA. Symptoms and signs of progressive hydrocephalus. Arch Dis Child Jan 1989; 64:124-128).

> COMMENT. The infant referred because of a large head is a fairly common problem in pediatric neurology practice. This instructive article points out that we may be relying too frequently on our colleagues in neuroradiology for diagnostic help and neglecting the much simpler and more economical method of direct measurement of intracranial pressure. The finding that one-half the infantile cases of hydrocephalus were without symptoms is disturbing.

MANAGEMENT OF HYDROCEPHALUS WITH IC PRESSURE MONITOR

Thirteen premature infants with posthemorrhagic hydrocephalus were treated by repeated aspiration of cerebrospinal fluid using a subcutaneous ventricular catheter reservoir at the Departments of Paediatrics and Neurosurgery, University of Heidelberg, Federal Republic of Germany. Criteria for the insertion of the catheter and reservoir were as follows: 1) Increase in head circumference of more than 1 cm/week; 2) Progressive ventricular dilatation on ultrasound scan; 3) Failure of lumbar puncture route of fluid removal; or 4) Bradyardia or apneic complications of lumbar puncture. Hydrocephalus was controlled by aspiration of fluid (median 6ml) one to four times a day for an average of 40 days. Clinical signs (tense fontanelle and increasing head size) and ultrasound were unreliable indicators of the amount and frequency of fluid removal. Direct intracranial pressure measurements made through the reservoir increased the efficacy and

safety of the method. Complications included skin breakdown in one, red blood cells in the cerebrospinal fluid in one, hyponatremia in eight, and hypoproteinemia in two. The authors suggest that shunting should be performed if resolution of the posthemorrhagic hydrocephalus has not occurred in an infant weighing 2000 grams and if spinal fluid protein is low. (Leonhardt A et al. Management of posthaemorrhagic hydrocephalus with a subcutaneous ventricular catheter reservoir in premature infants. Arch Dis Child Jan 1989; 64:24-28).

COMMENT. C. Bannister, Consultant Paediatric Neurosurgeon, Manchester, England, comments that the reservoir allows easy repeated removal of cerebrospinal fluid from the lateral ventricles and is a convenient monitor of intracranial pressure. The disadvantages include risk of infection, skin breakdown, intraventricular bleeding from rapid aspiration and pressure fluctuations, and local cortical damage secondary to catheter insertion.

SEQUELAE OF POSTHEMORRHAGIC HYDROCEPHALUS

The neurologic and developmental outcome of 33 low birth weight neonates with ventriculomegaly and in 39 with no ventriculomegaly after hemorrhage were evaluated prospectively in the Departments of Pediatrics and Neurosurgery, Wayne State University School of Medicine and Children's Hospital of Michigan, Detroit. Ventriculoperitoneal shunts were inserted in 23 of the 33 ventriculomegaly (VM) group infants at a mean age of 26 days. Shunt revisions were performed in 18 of the 23 children for obstruction (71) or infection (11). The total group of 72 children were followed to a mean age of 50 months. More children in the VM group had neurologic sequelae and microcephaly in comparison with the children in the non-VM group. Mild abnormalities included hypotonia and moderate and severe sequelae included spastic quadriplegia in 12 children and right hemiplegia in two children. Visual, language and hearing impairments were significantly increased in the VM group and included strabismus, myopia, nystagmus, and blindness. Developmental delay occurred in 19 patients in the VM group and in only eight in the non-VM group. Among the children with shunts, a higher incidence of sequelae occurred when lack of ventricular decompression was noted immediately after shunt insertion and when shunt infections occurred. The most important predictor of mental and motor outcome in the group with shunts was lack of ventricular decompression immediately after shunt insertion. The authors speculate that in some infants loss of brain tissue, cerebral atrophy or both may occur before insertion of the ventriculoperitoneal shunt even when the shunt is inserted early. (Shankaran S et al. Outcome after posthemorrhagic ventriculomegaly in comparison with mild hemorrhage without ventriculomegaly. J Pediatr Jan 1989; 114:109-114).

COMMENT. There was no delay in the initial ventriculoperitoneal shunt insertion in this group so shunt infection and obstruction appeared to be responsible for the poor neurodevelopmental outcome.