

shunted for post-haemorrhagic ventricular dilation. Dev Med Child Neurol August 1988;30:451-456).

COMMENT. Assessment and therapeutic management of neonatal posthemorrhagic hydrocephalus is also reported from the Universitäts-Kinderklinik Mannheim (Arnold D et al. Klin Padiatr July-August 1988;200:299-306). In this series, 40 of 135 neonates with intraventricular hemorrhage developed hydrocephalus. Treatment was by serial lumbar puncture in 70% and only 40% required a shunt. Acetazolamide and furosemide were used in 10%. At follow up in 25 children, 40% were normal or had mild developmental delay, and 60% were seriously handicapped. As in the study from Hammersmith, poor outcome was related to severe hemorrhage and pre-operative brain damage. Cerebral damage may occur without an increase in intracranial pressure, and normal fontanelle and head circumference do not rule out the development of hydrocephalus.

PERIVENTRICULAR LEUKOMALACIA AND BILIRUBIN TOXICITY

The role of bilirubin toxicity and other factors in the etiology of extensive periventricular leukomalacia of 5 preterm infants was investigated in the Department of Paediatrics, Tampere University Central Hospital, Tampere, Finland. Diagnosis was made by routine ultrasound screening, and the perinatal courses and later development of affected infants was compared with 12 normal controls. Infants with leukomalacia were delivered more often by the vaginal route, their mean highest serum total bilirubin and blood pH were significantly higher than the controls. The authors speculate that bilirubin toxicity may play an important role in addition to ischemia in the severe cases of periventricular leukomalacia. (Ikonen RS et al. Possible etiological factors in extensive periventricular leukomalacia of preterm infants. Acta Paediatr Scand July 1988;77:489-495).

COMMENT. The periventricular white matter in preterm infants is susceptible to ischemia and factors leading to hypotension can cause leukomalacia. Vaginal delivery and compression of the head of the preterm infant caused by uterine contractions during labor may lead to cerebral ischemia, but other causes include birth asphyxia, hypoxia, and hypocarbia. Although the basic cause of periventricular leukomalacia is ischemia, bilirubin toxicity may occur when the blood brain barrier is damaged by anoxia, permitting bilirubin to enter the brain even when bound to albumin.

NEONATAL STROKE

The prevalence of left middle cerebral artery involvement in 15 patients with neonatal stroke has been investigated at the Department of Neurology and Pediatrics, Strich School of Medicine, Loyola University of Chicago, Maywood, Illinois. Of 15 patients reviewed retrospectively, 12 had left middle cerebral artery infarction, and this preponderance was the same as that observed in 36 previously

reported cases. Transcranial Doppler measurement of mean blood flow velocity in the right and left middle cerebral arteries of 20 normal newborns demonstrated no significant difference between the 2 sides. The authors postulate that the turbulent flow of blood from the ductus arteriosus in the perinatal period favors the passage of an embolus travelling up the arch of the aorta into the left common carotid artery. (Coker SB et al. Neonatal stroke: Description of patients and investigation into pathogenesis. Pediatr Neurol July-August 1988;4:219-223).

COMMENT. Neonatal seizures were reported in 12 of the 15 infants with stroke, and the birth was complicated in 13. Cesarean section was performed in 8 (53%). Subsequent examination at 1-4 years of age showed hemiparesis in 10, near normal function of the involved extremity in 8, and persistent epilepsy requiring anticonvulsant treatment in 4 patients. Neonatal stroke is not usually correlated with birth asphyxia, birth trauma, and type of delivery, and an embolic etiology is more likely.

CEREBRAL VASCULAR MALFORMATIONS

ARTERIOVENOUS MALFORMATION (AVM)

The clinical characteristics and microsurgical approach to treatment of 39 children with AVMs are reviewed from the Neurosurgical Unit, Queen Elizabeth Hospital, Hong Kong. The pediatric cases represented 23% of 175 AVMs in all age groups. Age at diagnosis ranged from 1 month to 16 years, the majority between 11-16 years. Male to female ratio was 1.8:1. Hemorrhage in 87.5% was the most common presenting symptom. Total surgical excision was the treatment of choice, more than 80% leading a fully functional life. (Fong D, Chan S. Arteriovenous malformation in children. Child's Nerv Syst August 1988;4:199-203).

COMMENT. The reported mortality from the first hemorrhage in AVM is 10% and 12% from the second. AVMs in children are more apt to bleed than those in adults. Surgery is usually advised on operable lesions.

Cerebral cavernous malformations are discussed in a recent article (Rigamonti D et al. N Engl J Med August 11, 1988;319:343). In a study of 24 patients with histologically verified CCMs at the Barrow Neurological Institute, Phoenix, Arizona, 13 were members of 6 unrelated Mexican-American families, and 11 had no evidence of a heritable trait. In the familial cases, 11% of relatives had seizures and 83% were asymptomatic. MRI revealed cavernous malformations in 14 of 16 studied, and 11 had multiple lesions. MRI was superior to CT and angiography in diagnosis of CCM. The authors conclude that CCMs are not rare, particularly in Mexican-Americans. There are 2 forms - sporadic and familial. The more prevalent familial form is transmitted as an autosomal dominant, and multiple lesions are common.