are allowed to develop with increasing age, the likelihood of postoperative improvement is only 25-50%. Ultrasound may be used to diagnose tethered cords but magnetic resonance is usually required for better visualization. In addition to the association with cutaneous hemangioma, tethered cord occurs with subcutaneous lipoma, a hairy tuft, a prominent dimple, or a midline sinus tract or skin defect.

TRAPPED VENTRICLE AND SHUNTED HYDROCEPHALUS

Occluded fourth ventricle (trapped ventricle) is reported in eight of 47 children (17%) receiving repeated shunt revisions for hydrocephalus at the Stritch School of Medicine, Loyola University of Chicago, Maywood, IL. The hydrocephalus was caused by intraventricular hemorrhage but the fourth ventricular enlargement developed only after shunting. Massive dilatation of the ventricle occurred in four, three developed a progressive spastic quadriparesis, and two had increased intracranial pressure with lethargy and vomiting. Two children underwent a fourth ventricular shunt; one became more alert and less quadriparetic, and the other showed gradual improvement in motor function. (Coker SB and Anderson, CL. Occluded fourth ventricle after multiple shunt revisions for hydrocephalus. Pediatrics June 1989; 83:981-985).

COMMENT. Trapped ventricle following repeated shunting may be manifested by headache, lethargy, vomiting, ataxia, spastic quadriparesis, cranial nerve palsies and head tilt. This complication appears to be common among children with intraventricular hemorrhage who have received ventricular peritoneal shunting. Progressive fourth ventricular enlargement may be silent and diagnosis requires post shunt neural imaging and brain stem auditory evoked responses. Shunting of the fourth ventricle results in clinical improvement.

TOXIC DISORDERS

HYPERBILIRUBINEMIA AND NEURODEVELOPMENTAL OUTCOME

The results of the Collaborative Project on Preterm and Small for Gestational Age Infants in the Netherlands, 1983, in regard to hyperbilirubinemia and neurodevelopmental outcome at two years of age are reported from the Division of Neonatology, Department of Pediatrics, University Hospital, Leiden, the Netherlands.

Children with minor and major handicaps had significantly greater maximal serum total bilirubin concentrations than children with a normal neurodevelopmental outcome ($P = 0.02$). An increase in prevalence of handicaps was found for each 50 mmol/L (2.9 mg/dL) increase of maximal serum total bilirubin concentration. The neurological abnormalities included cerebral palsy, seizures, hearing defects as well as retinopathy of prematurity. The risk of a handicap increased by 30% for each 2.9 mg/dL increase of maximal serum total bilirubin concentration ($P = 0.02$) suggesting a causal relationship. (van de Bor M et al. Hyperbilirubinemia

in preterm infants and neurodevelopmental outcome at 2 years of age: Results of a national collaborative survey. Pediatrics June 1989; 83:915-920).

COMMENT. In the same issue, Newman TB and Maisels MJ comment that the strength of the association between bilirubin levels and brain damage in the present study was insufficiently precise to provide evidence of a strong causal relationship. The 95% confidence interval was wide, the association has not been consistent in other studies, and panic about bilirubin in the 6-12 mg/dL range is premature. They note that the NICHD collaborative phototherapy trial showed that bilirubin levels were reduced significantly by 4 mg/dL but this did not reduce the incidence of motor deficits or cerebral palsy and there was no effect on IQ scores. They estimate that even if the hyperbilirubinemia was toxic and causally related to neurologic damage, about 33 premature infants would need to be treated to prevent one case of neurologic handicap. To achieve similar benefit in term infants the number to be treated would be 333. Further studies are suggested.

CARBAMAZEPINE INDUCED MALFORMATIONS

A study of the pattern of malformations in children of women treated with carbamazepine during pregnancy is reported from the Division of Dysmorphology, Department of Pediatrics, University of California-San Diego, La Jolla, CA. The authors evaluated eight infants identified retrospectively as having had prenatal exposure to carbamazepine, alone or in combination with other drugs except phenytoin. In addition, in a prospective study they documented the outcome of the pregnancies of 72 women who were concerned early in their pregnancies about the potential teratogenicity of carbamazepine. The pattern of malformation including minor craniofacial defects, fingernail hypoplasia and developmental delay identified in the eight children in the retrospective study was confirmed through the evaluation of 48 children born alive to the women in the prospective study. The incidence of craniofacial defects was 11%, fingernail hypoplasia 26% and developmental delay 20%. The pattern of malformation with carbamazepine was similar to that of fetal hydantoin syndrome, suggesting that the epoxide intermediate metabolite is the teratogenic agent rather than the drug itself. (Jones KL et al. Pattern of malformations in the children of women treated with carbamazepine during pregnancy. N Engl J Med June 22, 1989; 320:1661-6).

COMMENT. We can now add carbamazepine (Tegretol) to the list of anticonvulsants with teratogenic effects.

A new use for carbamazepine is described from the Department of Pediatrics, 1011 Lausanne Switzerland (Roulet E Deona T. Pediatrics June 1989; 83:1077). Hereditary dominant chorea in an 11½ year old girl and in her mother was treated successfully with carbamazepine, confirming the experience of some other authors that this drug may have an effect on various choreas in a lower dose than that required for an antiepileptic effect.