brain tumor surgery should be avoided when possible, especially if cranial irradiation is planned.

In a randomized prospective study of carbamazepine or phenytoin in 276 post-craniotomy patients, 37% suffered at least 1 seizure during a 6-24 month trial period, and the incidence of status epilepticus in the first week following surgery was higher in AED-treated patients than in untreated controls (8% cf 2%). The occurrence of seizures in the first post-operative week did not increase the likelihood of late epilepsy. Acute allergic skin rashes occurred in 13% of patients treated with CBZ or PHT. (Foy PM et al. 1992. see Progress in Pediatric Neurology II, 1994;pp137-8; and Vol III, 1997;pp143-4).

VASCULAR DISORDERS

COGNITIVE DEFICITS AND MRI IN SICKLE CELL DISEASE

The risk of subtle brain abnormalities in children with sickle cell disease (SCD) and their relationship to blood hematocrit was determined by prospective comparison of 50 patients and 52 controls studied at St Jude Children's Research Hospital, Memphis, TN. Using quantitative magnetic resonance imaging to measure T1 (spin-lattice relaxation time) in basal ganglia, and the Wechsler test of intelligence, patients by age 4 years showed a significantly lower T1 (evidence of structural changes at the cellular level) in basal ganglia and cortex, but not in white matter, and mild mental deficiency (IQ, 50-70) in 33%, compared to a published prevalence of 1.45% in controls. Routine conventional MRIs were read as normal. Both the subtle T1 abnormalities on MRI and cognitive deficits were associated with a low hematocrit (Hct). Patients with an Hct of less than 27% had significantly lower IQ scores and significantly lower gray matter T1, than those with an Hct >27%. SCD was associated with a 23-fold increase in risk of mild mental deficiency. (Steen RG, Xiong X. Mulhern RK, Langston JW, Wang WC. Subtle brain abnormalities in children with sickle cell disease: relationship to blood hematocrit. Ann Neurol March 1999;45:279-286). (Respond: R Grant Steen PhD, Department of Diagnostic Imaging, St Jude Children's Research Hospital, 332 N Lauderdale, Memphis, TN 38105).

COMMENT. Young children with sickle cell disease and low hematocrits are at risk of subtle brain abnormalities, only detected by quantitative MRI, and complicated by mild mental deficiency. Brain hypoxia is proposed as the mechanism of this subtle brain damage demonstrated in patients with SCD who are spared more obvious brain pathology, including stroke.

Psychometric tests of intelligence can be more sensitive to subtle neurological abnormalities than conventional MRI scanning in SCD. In the absence of quantitative MRI, the Wechsler IQ test should be used routinely to follow children with SCD, not affected by stroke. The authors suggest that aggressive prophylactic therapy should be considered for possible prevention of brain damage and cognitive impairments in young children with SCD.

In an Editorial in the same issue, Dr GJ Dover of Johns Hopkins University School of Medicine discusses the progressive nature of the neuropathology of SCD (Ann Neurol March 1999;45:277-8). Steen and associates, in the present article, demonstrate the earliest detectable evidence of diffuse tissue hypoxia in the gray matter, as measured by quantitative MRI and IQ tests. Heretofore, the progression of brain pathology in SCD was documented in three ways: 1) subclinical large-vessel occlusion shown by Doppler; 2) clinical and subclinical infarcts seen on CT/MRI radiographic imaging; and 3) increased incidence of massive intracranial

hemorrhage in the second and third decades of life.

The inclusion of milder sickle syndromes, and subtle neurologic abnormalities related to a low hematocrit, emphasizes the need for closer patient follow-up and studies of the risk/benefit ratio of chronic prophylactic transfusion therapy or alternative treatments (Adams RJ et al. N Engl J Med 1998;339:5-11).

VERY BRIEF PNB COMMUNICATIONS

"Brief communications" concerning vascular neurologic disorders in the same issue of Ann Neurol include the following:

Stroke-like episodes in autosomal recessive cytochrome oxidase deficiency. (Morin C, Dube J, Robinson BH et al. Ann Neurol March 1999;45:389-392). Three patients with autosomal recessive Saguenay-Lac St-Jean cytochrome oxidase (COX) deficiency from Quebec, Canada, developed acute focal neurologic dysfunction and frontal hypodensities on CT, suggesting cerebral ischemia. Arteriography in 1 patient was normal during the acute episode. Some patients subsequently developed Leigh's disease (subacute necrotizing encephalomyelopathy). COX deficiency with congenital lactic acidosis is characterized by psychomotor retardation, hypotonia, and lactic acidemia.

Radiation-induced cerebral vasculopathy in children with neurofibromatosis and optic pathway glioma. (Grill J, Couanet D, Cappelli C et al. Ann Neurol March 1999;45:393-396). Neurofibromatosis 1 is a specific risk factor for radiation-induced cerebral vasculopathy. In a mean follow-up period of 7 years, 13 (19%) of 69 children with NF-1 and optic pathway glioma (OPG) developed occlusive vasculopathy within 36 months of therapy. Radiation therapy should be avoided as first-line therapy for OPG when possible.

INFECTIOUS DISORDERS

The current issue of Arch Dis Child has three articles concerning neurologic abnormalities associated with infections:

Behavior and cognitive outcomes from middle ear disease. (Bennett KE, Haggard MP. Arch Dis Child 1999;80:28-35). Otitis media with effusion (OME) or "glue ear", the most common cause of hearing loss in children, is associated with an increased incidence of behavioral disorders (mainly neurotic and hyperactive behaviors, and clumsiness) at 5 years of age, and language and speech articulation problems at 10 years. Effects are modest but significant, emphasizing need for increased awareness of parents and preschool teachers, early referral, and perhaps more vigorous physician treatment of recurrent otitis media.

Long term neurological outcome of herpes encephalitis. (Lahat E, Barr J, Barkai G et al. Arch Dis Child 1999;80:69-71). Persistent neurological sequelae occurred in 10 of 28 children with HSE, followed for a mean of 5 years, and 2 died. A low Glasgow coma score is a risk factor for a poor outcome; a score over 10 predicts no neurologic sequelae and a good prognosis. Early diagnosis and treatment are emphasized. PCR is the accepted preferred diagnostic test.

Acute cerebellar ataxia with human parvovirus B19 infection. (Shimizu Y, Ueno T, Komatsu H et al. Arch Dis Child 1999;80:72-73). A first case-report of a 2 year-old boy with ACA and erythema infectiosum caused by PVB19.