electrocorticographic as well as visual identification of intrinsically epileptogenic dysplastic cortical tissue. Completeness of excision of tissue showing I/CEDs was important for seizure control. The authors found that dysplastic cortex was more epileptogenic than other structural lesions, and patients with cortical dysplasia have a greater tendency to intractable seizures and a higher incidence of status epilepticus than those with other lesions. Previous reports from Montreal have found status epilepticus in 30% of patients with cortical dysplasia compared to 3% with epilepsy caused by supratentorial tumors. Life-threatening focal status epilepticus due to occult cortical dysplasia, not revealed by MRI, was successfully treated by surgical excision in 4 patients (Desbiens R, Andermann F et al, 1993; see Progress in Pediatric Neurology II, 1994, p292).

CONGENITAL MALFORMATIONS

PRENATAL CEREBRAL DYSGENESIS AND CEREBRAL PALSY

The MRIs of 70 cerebral palsy patients, aged 2 - 16 years, performed between 1989 and 1993 at Kansai Medical University Otokoyama Hospital, were analysed to evaluate the causative roles of pre-, peri-, and postnatal events. The CP was related to neuronal migration disorders in the embryonal stage in 26 patients. These included pachgyria and polygyria in 8, schizencephaly in 4, heterotopia in 4, agenesis of the corpus callosum in 4, cerebellar hypoplasia 3, and disorders of neuronal proliferation, differentiation and histiogenesis in 3. Vascular disorders were diagnosed in 30, intra-uterine infection in 5, and birth asphyxia in only 9. The authors conclude that CP of term infants is frequently the result of prenatal factors, either migration defects or cerebral infarction, and birth asphyxia is a relatively uncommon cause. (Sugimoto T et al. When do brain abnormalities in cerebral palsy occur? An MRI study. Dev Med Child Neurol April 1995;37:285-292). (Respond: Dr Tateo Sugimoto, Department of Paediatrics, Kansai Medical University Otokoyama Hospital, Izumi 19, Yawatashi, Kyoto 614, Japan).

COMMENT. The MRI may be used to identify causes of brain lesions underlying cerebral palsy, and birth asphyxia resulting from obstetrical factors is frequently excluded. In 31 of the 70 infants in this study the CP-related brain abnormalities were clearly developmental and prenatal in origin. In 10 of 30 with vascular lesions the damage had probably occurred in the prenatal period, and in 13 the time of damage was undetermined. The World Federation of Neurology cautions that the term birth asphyxia should be applied only to cases with definite evidence of an asphyxial origin for the neurological disability. Neonatal seizures are the most reliable evidence of intrapartum asphxia. The Apgar score is not the best indicator and most children with CP do not have low Apgar scores at birth. A possible causal relationship of perinatal asphyxia and CP should require the following: 1) severe newborn acidosis, 2) damage to other organs, and 3) severe neurologic abnormalities in the first 24-72 hours. (see Progress in Pediatric Neurology I, 1991, p333-6).

HIPPOCAMPAL CHANGES IN DOWN'S SYNDROME

Semiquantitative scales and quantitative computerized image analyses

were used to determine the neurofibrillary tangle formation and AB amyloid deposition in the hippocampal formation and inferior temporal gyrus in 36 Down's syndrome cases, aged 4 to 73 years. Neuropathological material was collected from several sources in Great Britain and America, and results are reported from the Massachusetts General Hospital, Boston, and the University of Manchester, England. Neurofibrillary tangles (NFTs) accumulated in patients with Down's syndrome over the age range 35 to 75 years, in the same anatomic locale as individuals with sporadic Alzheimer's disease. The entorhinal cortex, area CA1/subiculum, and other hippocampal subfields were especially vulnerable. Amyloid deposition is more widespread, accumulating over the years 30 to 50, and then reaching a plateau. Inheritance of the apolipoprotein E (Apo E) e4 genotype predisposed to more than double the amount of amyloid burden and was associated with increased numbers of senile plaques in Down's syndrome individuals with Alzheimer's disease. (Hyman BT et al. Neuropathological changes in Down's syndrome hippocampal formation. Effect of age and apolipoprotein E genotype. Arch Neurol April 1995;52:373-378). (Reprints: Dr Bradley T Hyman, Neurology Service, Massachusetts General Hospital, Fruit Street, Boston, MA 02114).

COMMENT. In a study at Mount Sinai School of Medicine, NY, the University of Kentucky, and the University of Geneva, Switzerland, quantitative analyses of neuropathologic changes in cerebral cortex of 16 patients (aged 6 to 74 years) with Down's syndrome and in 10 elderly individuals with Alzheimer's disease showed a similar time course of neurofibrillary tangle formation. Older patients with Down's syndrome had more neurofibrillary tangles and senile plaques than patients with Alzheimer's disease. Amyloid deposition preceeded neurofibrillary tangle formation. (Hof PR et al. Age-related distribution of neuropathologic changes in the cerebral cortex of patients with Down's syndrome. Quantitative regional analysis and comparison with Alzheimer's disease. Arch Neurol April 1995;52:379-391).

MIGRAINE HEADACHES

PERSISTENT VISUAL PHENOMENA IN MIGRAINE

Ten patients with migraine and persistent positive visual phenomena lasting months to years are reported from the University of Pennsylvania, Scheie Eye Institute, the Children's Hospital of Philadelphia, University of Miami Bascom Palmer Eye Institute, and the Cleveland Clinic, OH. Ages ranged from 9 to 67 years but visual phenomena were very similar in their simplicity, quality, and involvement of the entire visual field. They consisted of diffuse small particles, such as TV static, snow, lines of ants, dots, and rain. Treatment with various medications was of no benefit. (Liu GT et al. Persistent positive visual phenomena in migraine. Neurology April 1995;45:664-668). (Reprints: Dr GT Liu, Division of Neuro-Ophthalmology, Department of Neurology, Hospital of the University of Pennsylvania, 3400 Spruce Street, Philadelphia, PA 19104).

COMMENT. Visual processing in 12 migraineurs has been studied at the Mass General Hospital (Wray SH et al. Brain Feb 1995;118:25-35). An inherited abnormal threshold to visual stimuli is suggested. The high speed of the migraineur's brain in discerning a single target in low-level visual tasks is consistent with an oversensitivity to visual stimuli.