brain volume abnormalities and long-term cognitive outcome in preterm infants. JAMA October 18, 2000;284:1939-1947). (Reprints: Bradley S Peterson MD, Yale Child Study Center, 230 S Frontage Rd, New Haven, CT 06520).

COMMENT. Regional cortical volumes measured at 8 years of age in preterm children are significantly smaller than in term controls, and abnormalities, especially in the volumes of sensorimotor and midtemporal cortices, are related to cognitive impairments.

NEUROIMAGING AND NEURAL BASES OF LEARNING AND MEMORY

The use of positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) studies in identifying brain regions involved with learning and memory is reviewed from the University of Alberta, Canada, and the Umea University, Sweden. Prefrontal and parietal regions are involved with working memory; the left prefrontal and temporal regions with semantic memory; the left prefrontal and medial temporal regions with episodic memory encoding; right prefrontal, posterior midline and medial temporal regions with episodic memory retrieval; and the motor, parietal, and cerebellar regions with skill learning. (Cabeza R, Nyberg L. Neural bases of learning and memory: functional neuroimaging evidence. Current Opinion in Neurology August 2000;13:415-421). (Respond: Roberto Cabeza, Department of Psychology, University of Alberta, P220 Biological Sciences Building, Edmonton, T6G 2E9, Canada).

COMMENT. Memory functions are served by various brain regions, as determined by neuroimaging studies, mainly in healthy young adults. *Working memory,* the processing of information in short-term memory, is subserved by prefrontal and parietal regions. *Semantic memory,* referring to general knowledge, and *episodic memory* for personal experiences, are based in prefrontal and temporal regions. The acquisition of *Skill learning* abilities involves motor, parietal and subcortical regions.

SEIZURE DISORDERS

EPILEPSY IN JUVENILE NEURONAL CEROID LIPOFUSCINOSIS

The clinical characteristics of epilepsy and optimal antiepileptic drug therapy were surveyed in 60 patients (mean age 16 years, range 5-33) with juvenile neuronal ceroid lipofuscinosis (JNCL), followed at the University of Helsinki, Finland. Epilepsy, mainly generalized, was diagnosed in 50, and the first seizure occurred at a mean age of 10 years (range 5-16). Median seizure frequency was 4 per year, and seizure control was satisfactory in 72%. Lamotrigine as first choice and valproate were equally effective in seizure control, and carbamazepine was useful as add-on therapy. (Aberg LE, Backman M, Kirveskari E, Santavuori P. Epilepsy and antiepileptic drug therapy in juvenile neuronal ceroid lipofuscinosis. Epilepsia October 2000;41:1296-1302). (Reprints: Dr Laura Aberg, Hospital for Children and Adolescents, Pediatric Neurology, PL 280, 00029 HYKS, Finland).

COMMENT. JNCL is now regarded as a lysosomal disorder, characterized by an intralysomal accumulation of storage material, subunit c of mitochondrial adenosine triphosphate (ATP) synthetase. The disease is recessively inherited, with the gene locus mapping to chromosome 16, and with several different

mutations. Onset is with visual failure due to retinal degeneration, noted between the ages of 5 and 8 years. This is followed by epilepsy, psychomotor deterioration, sleep disturbance, and extrapyramidal symptoms. Affected females have acne, hirsutism, and obesity. Death follows in the early twenties.

EARLY DIAGNOSIS OF EPILEPSY SYNDROMES

The classification of epilepsy syndromes made initially on the basis of information at time of diagnosis was compared to that made 2 years later in a cohort of 613 children, followed by participating physicians in Connecticut, between 1993 and 1997. After 2 years, syndrome classifications were the same in 86% of the cohort. The diagnosis was changed in 10% (mainly incomplete syndromes), and syndrome evolution, mainly West to Lennox-Gastaut, occurred in 4%. Significant changes were rare. (Berg AT, Shinnar S, Levy SR et al. How well can epilepsy syndromes be identified at diagnosis? A reassment 2 years after initial diagnosis. Epilepsia October 2000;41:1269-1275). (Reprints: Dr Anne T Berg, Department of Biological Sciences, Northern Illinois University, DeKalb, IL 60115).

COMMENT. The identification of epileptic syndromes, for the most part, may be made accurately at the time of the initial presentation and diagnosis. Changes in diagnosis at follow-up, necessary in only 14%, are explained by difficulties in classification of incomplete syndromes and the evolution of West to Lennox-Gastaut syndomes with age and maturation.

Epileptic syndromes posing problems in diagnosis. Hirsch E et al (Strasbourg, France) review the heterogeneous nature and clinical management of partial epilepsies and incomplete syndromes. BECTS are the most common idiopathic localization-related epilepsy, and may be triggered by carbamazepine in some cases. Primary reading epilepsy and idiopathic occipital lobe epilepsies with photosensitivity are an overlap of idiopathic localization-related and generalized epilepsies, and respond to sodium valproate. Other variants of idiopathic localization-related epilepsies include autosomal dominant nocturnal frontal lobe epilepsy and benign familial infantile convulsions. AED resistance can be due to errors in diagnostic classification of these epilepsy syndromes. EEG-video evaluation may be necessary in refractory seizures. (Hirsch E et al. New insights into the clinical management of partial epilepsies. Epilepsia Oct 2000;41(suppl 5):S13-S17).

Post-ictal paralysis in BECTS. Dai A et al (State University of New York, Buffalo, NY) found a 9% association of post-ictal paresis among 68 children with benign rolandic epilepsy, and 50% had brief post-ictal aphasia. Todd's paresis and aphasia do not exclude the diagnosis of BREC, and these transient complications are clinically benign. (Abstracts from the Annual Meeting of the AES, Los Angeles, CA, Dec 1-6, 2000. Epilepsia Oct 2000;4, suppl 7:88).

ANOXIC AND VASCULAR DISORDERS

HYPOTHERMIA TREATMENT FOR NEONATAL ENCEPHALOPATHY

Treatment with mild whole body hypothermia after birth asphyxia was evaluated in 10 of 16 newborns with EEG burst suppression evidence of a bad prognosis, followed at the Imperial College School of Medicine, London, UK. All infants selected for treatment had convulsions and a severe encephalopathy. Hypothermia was instituted within 6 hours of birth and continued for 48 hours.