W, Smith EC, Mikati MA. Electroencephalographic and seizure manifestations in two patients with folate receptor autoimmune-mediated primary cerebral folate deficiency. **Epilepsy Behav** 2012 Aug;24(4):507-12) (Response: MA Mikati MD. E-mail: mohamad.mikati@duke.edu).

COMMENT. Patients with developmental regression, refractory seizures or spasms, and EEG showing hypsarrhythmia or electrical status epilepticus during sleep should be tested for cerebral folate deficiency and considered for treatment with folinic acid. The authors list seizure onset during the first 2 years, tonic, myoclonic-astatic, absence, or generalized tonic-clonic seizures, and an EEG showing generalized spike-slow waves and multifocal spikes as important in the index of suspicion of this disorder.

VASCULAR DISORDERS

STROKE RECURRENCE IN CONGENITAL HEART DISEASE

Researchers at the Hospital for Sick Children, Toronto, Canada identified 135 patients with congenital heart disease diagnosed with arterial ischemic stroke during 1992-2008 and registered in the Canadian Pediatric Stroke Registry-Toronto site. Of the total cohort with sentinel stroke, 19 (14%) had a recurrence. Of 78 (58%) with neonatal sentinel stroke, 7 (9%) had a stroke recurrence. Ten years following a sentinel stroke, 27% had suffered a stroke recurrence, 26% had died, and 47% were alive without recurrence. Age at sentinel stroke was 0.5 yr (range 0.1-17.0). Stroke recurrence risk was highest immediately following the sentinel stroke and decreased over time. At time of recurrence, 50% were receiving anticoagulation. Recurrence risk factors included a mechanical valve, prothrombotic condition, and an acute infection at time of sentinel stroke. Hazard of mortality after recurrence was similar to mortality after sentinel stroke. More aggressive secondary prophylaxis in the early poststroke period may be indicated in patients at increased risk. (Rodan L, McCrindle BW, Manlhiot C, et al. Stroke recurrence in children with congenital heart disease. **Ann Neurol** 2012 Jul;72(1):103-11). (Respond: Gabrielle deVeber MD, Hospital for Sick Children, 555 University Ave, Toronto, Canada M5G 1XB. E-mail: gabrielle.deveber@sickkids.ca).

COMMENT The Toronto team has demonstrated the relative safety of anticoagulant therapy in 123 children with arterial ischemic stroke, with a 4% risk of intracranial hemorrhage. (Schechter T et al. **Blood** 2012 Jan 26;119(4):949-56).

NEONATAL DISORDERS

SURFACTANT AND NEONATAL ELECTROENCEPHALOGRAM

Researchers at the University of California San Diego evaluated the effects of endotracheal intubation and surfactant on the neonatal brain using a 3-channel neonatal EEG. Surfactant administration was associated with brain wave suppression on EEG in 18 (62%) of 29 infants treated. ($p<0.008$). Nine infants exhibited EEG suppression during

endotracheal intubation, all having received premedication; 5 infants had EEG suppression during endotracheal suctioning. Brain wave suppression was not correlated with SpO2, BP, heart rate, or TcCO2. (Shangle CE, Haas RH, Vaida F, Rich WD, Finer NN. Effects of endotracheal intubation and surfactant on a 3-channel neonatal electroencephalogram. **J Pediatr** 2012 Aug;161(2):252-7). (Reprints: Neil N Finer MD, UCSD, Department of Pediatrics, 200 West Arbor Dr, MPF 1-140, San Diego, CA 92103. E-mail: nfiner@ucsd.edu).

COMMENT. Endotracheal surfactant administration has reduced neonatal mortality and improved lung function in infants with respiratory distress syndrome. The adverse effects on neonatal brain electrical activity require further study of the EEG, using full neonatal electrode placement and including long-term outcome.

MRI IN HYPOXIC-ISCHEMIC ENCEPHALOPATHY

Researchers at University of Melbourne, Australia, and St Louis Children's Hospital, US investigated the effects of hypothermia treatment on MRI patterns of brain injury in newborns with hypoxic-ischemic encephalopathy (HIE) and the prognostic utility of MRI for death or disability at age 2 years. Fewer newborns had white matter and cortical gray matter abnormalities on T1- and T2-weighted images in the 66 treated with hypothermia group compared with 61 normothermic newborns. All T1- and T2-weighted and diffusion MRI abnormalities were predictive of death or major sensorineural disability. The prognostic utility of MRI variables was not altered by hypothermia treatment. MRI abnormalities in the basal ganglia and thalami had the highest sensitivity and specificity for adverse outcome at 2 years. (Cheong JLY, Coleman L, Hunt RW, et al, for the Infant Cooling Evaluation Collaboration. Prognostic utility of magnetic resonance imaging in neonatal hypoxic-ischemic encephalopathy. Substudy of a randomized trial. **Arch Pediatr Adolesc Med** 2012 Jul 1;166(7):634-40). (Respond: Jeanie LY Cheong MD, Neonatal Service, Royal Women's Hospital, 20 Flemington Rd, Parkville 3052, Victoria, Australia (E-mail: Jeanie.cheong@thewomens.org.au).

COMMENT. Treatment with hypothermia is associated with reduction in white matter and cortical gray matter abnormalities on MRI of newborns with HIE. Abnormal MRI findings are prognostic of outcome in moderate to severe HIE in both hypothermia treated and normothermic newborns. MRI is an important biomarker of long-term outcome of newborns with HIE, irrespective of hypothermia.

The importance of timing of imaging and the greater sensitivity of some newer techniques are discussed in an editorial (Chau V et al. Magnetic resonance imaging in hypoxic-ischemic encephalopathy. **Arch Pediatr Adolesc Med** 2012 Jul 1;166(7):669-71). In a previous study (Rutherford MA et al. **Pediatrics** 1998 Aug;102(2):323-8), MRI scans were done at a mean postnatal age of 8 days as opposed to 6 days in the present study, and the positive predictive value was somewhat lower (76%). MR spectroscopy and diffusion tensor imaging will detect brain injury in the first days following an injury and may have better prognostic value than conventional MRI. (Porter EJ, et al. Tract-based spatial statistics of magnetic resonance images to assess disease and treatment effects in perinatal asphyxia encephalopathy. **Pediatr Res** 2010 Sep;68(3):205-9).