COMMENT. Children with CVST and severe neurological deterioration despite anticoagulation may have a favorable response to endovascular treatment. Clinical deterioration after medical treatment is an indication for endovascular therapy. Larger scale studies are required to establish the role of endovascular treatment of deteriorating cases of pediatric CVST. Indicators of a poor prognosis are coma, involvement of the deep venous system, and parenchymal hemorrhage. Anticoagulation may be associated with an increase in size of a hematoma or de novo hemorrhage (Moharir MD, Shroff M, Stephens D, et al. Anticoagulants in pediatric cerebral sinovenous thrombosis: a safety and outcome study. **Ann Neurol** 2010 May;67(5):590-9).

Stroke therapy for children is discussed in an editorial (Roach ES. **Pediatr Neurol** 2013 Nov;49(5):301-2). Until age-specific studies are available, pediatric neurologists must accept methods and results of trials in adults. Thrombolysis with tPA in adults must be administered within 4.5 hours of symptom onset for a favorable risk-benefit ratio to be maintained. Administration of tPA after this time increases the risk of hemorrhage. Use of tPA in children is not approved by the FDA, but a trial is now underway (Amlie-Lefond C, et al. Thrombolysis in acute childhood stroke: design and challenges of the thrombolysis in pediatric stroke clinical trial. **Neuroepidemiology** 2009;32(4):279-86).

ENCEPHALITIS AND ENCEPHALOPATHIES

DIAGNOSTIC ALGORITHM FOR ENCEPHALITIS

Diagnostic algorithm for initial evaluation of encephalitis in children is proposed with a consensus statement from the International Encephalitis Consortium, a committee begun in 2010 to serve as a practical aid to clinicians evaluating patients with suspected encephalitis. *Encephalitis is defined as inflammation of the brain parenchyma associated with neurologic dysfunction*. Major diagnostic criterion is an altered mental status lasting >24 hours. Minor criteria include fever, seizures, focal neurologic findings, CSF WBC count >5/cubic mm, MRI parenchymal lesion, or EEG abnormality indicative of encephalitis (>3 required for probable or confirmed encephalitis).

Routine studies proposed include CSF, serum, imaging (MRI preferred), EEG, throat sample for *Mycoplasma pneumoniae* PCR, and throat and stool specimens for enterovirus PCR. Specific signs and symptoms of encephalitis include abnormal behavior, psychotic features, seizures or movement disorder indicating need for NMDAR antibody test in serum and CSF; vesicular rash indicating VZV PCR from CSF; respiratory symptoms indicating *Mycoplasma pneumoniae* PCR in CSF; and limbic symptoms indicating autoimmune limbic encephalitis testing HHV6/7 PCR (CSF). (Venkatesan A, Tunkel AR, Bloch KC, et al. Case definitions, diagnostic algorithms, and priorities in encephalitis: Consensus statement of the International Encephalitis Consortium. **Clin Infect Dis** 2013 Oct;57(8):1114-28). (Response: Ann Venkatesan MD, PhD, Johns Hopkins University School of Medicine, 600 N Wolfe St, Baltimore, MD 21287. E-mail: avenkat2@jhmi.edu).

COMMENT. The definition proposed is chosen to capture both encephalitis and encephalopathy. Encephalopathy is a clinical state of altered mental status, manifesting as

confusion, disorientation, behavioral changes, or other cognitive impairments, with or without inflammation of brain tissue. Encephalitis is characterized by brain inflammation resulting from direct infection of the brain parenchyma (e.g. Bartonella or influenza), a post-infectious process as in ADEM or a noninfectious condition such as NMDAR encephalitis. The definition covers infectious and noninfectious encephalitis and encephalopathy of presumed infectious etiology. Specific etiologies are identified in <50% of cases.

Proteomes in plasma and CSF of children with cerebral malaria were found to differ from those with acute bacterial meningitis and nonspecific encephalopathies. Pathogenic states in children with impaired consciousness in malaria endemic areas could be reflected by changes in protein biomarkers in both plasma and CSF. (Gitau EN et al. **J Infect Dis** 2013 Nov 1;208(9):1494-503).

POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME

Investigators at Children's Hospital of Montefiore, Albert Einstein College of Medicine, NY, determined the incidence of posterior reversible encephalopathy syndrome (PRES) in a pediatric critical care unit. Ten patients <21 years of age with PRES (incidence of 1 in 259 admissions, 0.4%) were studied. Nine patients presented with generalized tonic and/or clonic seizures. Continuous EEG showed generalized slowing but no epileptiform activity. Risk factors included hypertension, cytotoxin medication use, and anemia. Comorbidities included systemic lupus erythematosus, inflammatory bowel disease, ulcerative colitis, liver cirrhosis, pulmonary embolism, renal insufficiency, septic shock, and acute chest syndrome. One-year follow-up showed no residual neurological deficits and resolution of white matter signal abnormalities on neuroimaging. (Raj S, Overby P, Erdfarb A, Ushay HM. Posterior reversible encephalopathy syndrome; incidence and associated factors in a pediatric critical care population. **Pediatr Neurol** 2013 Nov;49(5):335-9). (Response: Dr Raj, The Children's Hospital at Montefiore, Bronx, NY 10467. E-mail: drshashiraj@gmail.com).

COMMENT. PRES, also referred to as hypertensive encephalopathy or reversible posterior leukoencephalopathy syndrome, is a clinical syndrome that results from disruption of the blood-brain barrier and vasogenic edema, demonstrated on MRI with hyperintense signals in the posterior cerebral white matter.

PRES in an infant 35 days old is reported from the Mayo Clinic. The syndrome is rare in children less than 1 year. The infant had a history of obstructive sleep apnea, laryngomalacia, and choanal atresia. While undergoing bronchoscopy, she developed apneic episodes with stiffening of extremities. EEG revealed occipital lobe onset seizures, and MRI showed hyperintense T2 signal in both posterior temporal and parieto-occipital lobes. A labile blood pressure was normalized and seizures abated with fosphenytoin and levetiracetam. At 3 month of age, resolution of MRI abnormality confirmed the diagnosis of PRES. (Mrelashvili A, Watson RE, Wong-Kisiel LC. **Pediatr Neurol** 2013 Nov;49(5):387-8).