seizures and FSE, with durations of 43, 45, and 60 minutes. CSF findings were normal and bacterial cultures were negative. A child aged 3 months to 5 years who presents with a first or recurrent FS should be considered for LP if one or more of the following indications are present: neurologic signs of meningitis, systemic signs of toxicity, complex seizure with prolonged postictal obtundation of consciousness, or pretreatment with antibiotics. Complex FS alone is not an absolute indication for LP.

In a retrospective study at Children's Hospital Boston to assess the rate of acute bacterial meningitis among 526 children who present with their first complex febrile seizure, 2.7% had CSF pleocytosis and 3 patients (0.9%) had acute bacterial meningitis. One appeared well clinically; of 2 with Streptococcus pneumoniae cultured from CSF, 1 was nonresponsive clinically, and the other had a bulging fontanel and apnea. (Kimia A, et al. **Pediatrics** 2010 Jul;126(1):62-9).

THALAMOCORTICAL STRUCTURAL AND FUNCTIONAL CONNECTIVITY IN JUVENILE MYOCLONIC EPILEPSY

Researchers at King's College, Institute of Psychiatry, London and other centers in the UK, US, and Germany discovered changes in an anterior thalamo-cortical bundle during tests of structural connectivity, as measured by diffusion tensor imaging, in a cohort of 28 subjects with juvenile myoclonic epilepsy. An alteration in task-modulated connectivity was detected in a region of frontal cortex connected to the thalamus via the same anatomical bundle, and overlapping with the supplementary motor area. In patients with active seizures, the degree of abnormal connectivity is related to disease severity in those with active seizures. These results point to abnormalities in a specific thalamo-cortical circuit, with reduced structural and task-induced functional connectivity that underlies this idiopathic epilepsy. (O'Muircheartaigh J, Vollmar C, Barker GJ, et al. Abnormal thalamocortical structural and functional connectivity in juvenile myoclonic epilepsy. **Brain** 2012 Dec;135(Pt 12):3635-44). (Response: Dr Mark P Richardson, E-mail: mark.richardson@kcl.ac.uk).

COMMENT. The characteristic generalized spike and wave discharges in the EEG of juvenile myoclonic epilepsy implicate thalamo-cortical interactions, and the discharges are most prominent in frontal regions. The functional and diffusion MRI and diffusion tensor imaging used above provide anatomic evidence for the role of the thalamus and a specific thalamo-cortical circuit dysfunction in JME. JME is a lifelong disorder and a structural cerebral defect may explain the necessity to continue treatment indefinitely. (Wandschneider B, et al. Frontal lobe function and structure in juvenile myoclonic epilepsy: a comprehensive review of neuropsychological and imaging data. **Epilepsia** 2012 Dec;53(12):2091-8).

MUSCULAR DYSTROPHY-DYSTROGLYCANOPATHY AND EPILEPSY

Investigators from the University of Catania, and other centers in Europe have identified a novel genetic glycosylation disorder, DPM2-CDG (part of the DPM synthase complex) in 3 infants with severe hypotonia, progressive muscle weakness and wasting, elevated CK, absent psychomotor development, intractable epilepsy with onset at 1 week