complicated by purpuric rash. Duration varies from less than 1 week to several months, with occasional episodes of apparent recovery. Severity ranges from self-limited systemic illness (90% patients) to life-threatening illness with jaundice, renal failure, and hemorrhagic pneumonitis (Weil syndrome).

References.
1. AAP. Leptospirosis. In: Pickering LK, ed. Red Book: 2012 Report of the Committee on Infectious Diseases. 29th ed. Elk Grove Village, IL: AAP; 2012:469-71.

HEADACHE DISORDERS

THALAMIC FUNCTION AND VESTIBULAR MIGRAINE

Investigators at Universities of Naples and Salerno, Italy; and Maastricht University, the Netherlands, explored the functional response of vestibular neural pathways using whole-brain blood oxygen level dependent (BOLD) fMRI during caloric vestibular stimulation in 12 patients (mean age 31.2 +/- 5 yrs) with vestibular migraine (VM), in 12 healthy controls, and in a group of age- and sex-matched patients with migraine without aura (MwoA). In all subjects, caloric vestibular stimulation elicited activation in bilateral insular cortex (right > left), right parietal cortex, right thalamus, brainstem, and cerebellum. While all participants demonstrated this general pattern of response, patients with VM showed a significantly increased left medio-dorsal thalamic activation in response to an ipsilateral vestibular stimulation, relative to both healthy controls and patients with MwoA. The magnitude of the thalamic activation was positively correlated with the frequency of migraine attacks in patients with VM. (Russo A, Marcelli V, Esposito F, et al. Abnormal thalamic function in patients with vestibular migraine. **Neurology** 2014 Jun 10;82(23):2120-6).

COMMENTARY. Patients with VM have abnormal thalamic functional response to vestibular stimulation. These findings are consistent with the current view of VM as a migraine subtype clinically characterized by vestibular symptoms and correlated with interictal dysfunctional central vestibulo-thalamocortical processing [1]. Both structural and functional thalamic abnormalities are documented in patients with migraine [2].

References.
1. Russo A, et al. Neurology. 2014 Jun 10;82(23):2120-6.
2. Liu J, et al. PLoS One. 2012;7(12):e51250.

DEMYELINATING DISORDERS

EVALUATION OF REVISED DIAGNOSTIC DEFINITION OF MS

Investigators at Erasmus MC, Rotterdam, The Netherlands, evaluated the 2012 revised IPMSSG consensus definitions in a cohort of children with acquired demyelinating syndromes (ADS) prospectively followed for 2 years from Jan 2007. An MRI within 90 days after first disease onset was a criterion for inclusion. Of 82 children

with ADS, 35 were diagnosed with pediatric MS. The time to MS diagnosis applying the 2007 and revised 2012 definitions was compared. The revised 2012 definitions had sufficient sensitivity (80%) and high specificity (100%). MS diagnosis was made at first MRI and 3.4 months earlier (p=0.004) applying the new definitions. (van Pelt ED, Neuteboom RF, Ketelsiegers IA, et al. Application of the 2012 revised diagnostic definitions for paediatric multiple sclerosis and immune-mediated central nervous system demyelination disorders. **J Neurol Neurosurg Psychiatry** 2014 Jul;85(7):790-4).

COMMENTARY. The new 2012 International Pediatric Multiple Sclerosis Study Group (IPMSSG) consensus definitions of acquired demyelinating syndromes [1] incorporate the 2010 revised McDonald criteria for MS and allow for a reliable and earlier MS diagnosis in all children, including those younger than 12 years.

References.

1. Krupp LB, et al. Mult Scler. 2013 Sep;19(10):1261-7.

SEIZURE DISORDERS

NEUROBEHAVIORAL COMORBIDITIES OF ACTIVE EPILEPSY

Eighty percent of children with active epilepsy in Sussex Schools, UK, who underwent psychological evaluation had a DSM-IV-TR behavioral disorder and/or cognitive impairment (IQ <85). Intellectual disability (ID) (IQ <70) (40%), ADHD (33%), and ASD (21%) were the most common neurobehavioral diagnoses, and only one-third had previously been diagnosed. Seizures in the first 24 months, generalized seizures, status epilepticus, and polytherapy were independently associated with ID, and ID was associated with a diagnosis of ASD. Epilepsy-related factors (e.g. age of onset, seizure frequency) did not independently predict behavioral disorders, suggesting that seizures per se are not the cause of behavioral problems. (Reilly C, Atkinson P, Das KB, et al. Neurobehavioral comorbidities in children with active epilepsy: A population-based study. **Pediatrics** 2014 Jun 1;133(6):e1586-93).

COMMENTARY. The identification of neurobehavioral comorbidities might lead to improved methods of control of both seizures and their associated behavioral disorders. As an example, the use of methylphenidate (MPH) for the management of co-morbid ADHD may be associated with improved control of both seizures and ADHD. The addition of MPH is reported safe in children with ADHD and epilepsy whose seizures are controlled with AEDs [1]. Caution is advisable in the use of stimulants in cognitively impaired hyperactive children whose seizures are AED refractory [2].

References.

1. Millichap JG. Attention Deficit Hyperactivity Disorder Handbook : A Physician's Guide to ADHD. 2nd ed. New York: Springer; 2011:182.
2. Gonzalez-Heydrich J, et al. Epilepsy Behav. 2014 Jun 2;36C:102-107.