antiepileptic medications were ineffective; the ketogenic diet was unavailable in this center and had not been tried.

Of 314 children enrolled in the Far-East Asia Catastrophic Epilepsy (FACE) study group, age of onset of epilepsy was <12 months in 239 cases (80%), epileptic spasms were the most frequent seizure type (in 42%), followed by generalized tonic seizures (in 20%) [1]. Epileptic syndromes included West syndrome (in 37%), unclassified (21%), Lennox-Gastaut (12%), Dravet (4%), and Rasmussen (2%). Cortical dysplasia and chromosomal anomalies were the two most frequent causes of epilepsy, in 16% and 6%, respectively; in almost one half of patients, the cause was unknown. Psychomotor development was retarded in 62% cases.

References.

**PSYCHOGENIC NON-EPILEPTIC SEIZURES**

Investigators at the National Institute of Mental Health and Neurosciences, Bangalore, India, conducted a retrospective analysis of semiologic patterns of psychogenic non-epileptic seizures (PNES) diagnosed by video EEG in 56 children aged < 18 years (mean age 12.3 yrs; range 2-17 yrs). Age at onset of PNES was 8.9 yrs (range 0.4-15.8 yrs); age at diagnosis 11.9 yrs (range 2-17 yrs); delay in diagnosis 3.2 yrs (range 0-15 yrs). Associated diagnoses included anxiety in 16%, stress in 10%, and depression (10%). Coexistent epilepsy in 16% patients was complex partial in 8.9%, generalized tonic-clonic in 5.4%, and simple partial in 1.8%. Prior to VEEG, 33 (59%) patients were initially misdiagnosed as epilepsy and were treated with AEDs; in 14 patients (25%) the initial diagnosis of PNES was unchanged after VEEG. EEG during a PNES showed various artifacts, depending on the type of movement or coma-like state. MRI performed in 14 patients with PNES alone was normal in 12 (86%) and showed non-specific white matter signal changes or UBOs in 2. Characteristic signs of PNES were flexion/extension movements, moaning and gasping, tremors, flaccidity, vocalization, hyperventilation, and pelvic thrusting. Eyes were closed in 25 (45%) and remained open during the PNES in 55%. The EEG technician’s simple motor commands were followed by 55% during the event. PNES was classified in 5 categories: I. Abnormal motor (hypermotor (23%) and partial (14%)); II. Affective/emotional behavior 3.6% (moaning, grunting); III. Dialeptic 14% (coma-like state, flaccidity); IV. Aura 5.4% (subjective feeling, dizziness); V. Mixed (39%). (Dhiman V, Sinha S, Rawat VS, et al. Children with psychogenic non-epileptic seizures (PNES): a detailed semiologic analysis and modified new classification. Brain Dev 2014 Apr;36(4):287-93).

COMMENTARY. Video-EEG is important in the diagnosis and differentiation of epileptic seizures from PNES. Epilepsy and PNES are coexistent in 16% of cases. In a previous semiologic analysis of 27 childhood PNES cases based on video-EEG monitoring, mean duration of PNES was longer compared to epileptic seizures, eyewitnesses were almost always present, eyes were closed at the onset in only 15% of events, tremor was the most frequent motor sign, and dialeptic PNES was most frequent among younger children [1].
HUMAN METAPNEUMOVIRUS AND STATUS EPILEPTICUS

Investigators at Cincinnati Children’s Hospital, OH, report 2 toddlers, ages 15 and 18 months, with human metapneumovirus (hMPV) infection who presented in status epilepticus and later developed respiratory failure. Both patients recovered over 2 weeks with no sequelae. Infection with hMPV should be considered as a cause of seizures or encephalitis with respiratory symptoms in infants and children. (Webster DL, Gardner AH, Dye TJ, Chima RS. Status epilepticus: a possible association with human metapneumovirus infection. Pediatrics 2014 Mar;133(3):e747-50).

COMMENTARY. Neurological complications of human metapneumovirus infection are not mentioned in the 2012 edition of the AAP Redbook [1], and a review of seizures and hMPV in PubMed uncovers few reports. One earlier study reports an incidence of 6.3% of hMPV cases associated with seizures compared to 0.7% of patients infected with RSV (p=0.031). hMPV may be associated with a spectrum of CNS disease ranging from febrile seizure to status epilepticus and severe, fatal encephalitis [2].

REFERENCES

ENCEPHALITIDES

HERPES SIMPLEX AND NMDA ENCEPHALITIDES

Investigators at University of Texas Southwestern Medical Center, Dallas, TX, report 2 male patients, an infant and adult, with confirmed herpes simplex encephalitis (HSE) and anti-NMDA receptor antibody encephalitis. Testing for anti-NMDA receptor antibodies and autoimmune disorder is recommended in patients with persistent encephalopathy, regression after initial improvement, or persistent movement disorders. Neuronal infections such as HSV may trigger subsequent anti-NMDA receptor antibody formation. Concomitant treatment or testing for immune-mediated encephalitis is indicated when treating viral encephalitis. (DeSena A, Graves D, Warnack W, Greenberg BM. Herpes simplex encephalitis as a potential cause of anti-N-methyl-D-aspartate receptor antibody encephalitis report of 2 cases. JAMA Neurology 2014;71(3):344-6).

COMMENTARY. The association of herpes simplex and anti-NMDA receptor antibody encephalitides is reported in 5 prospectively diagnosed patients (2 female) with relapsing post-herpes simplex encephalitis [1]. In 3 further retrospectively studied patients with HSE and NMDAR antibodies the frequency of autoantibodies increased over time, suggesting that HSE triggers NMDAR antibodies and brain autoimmunity [1].