brainstem and lack of visualization of the proximal part of the facial nerves. 3D-constructive interference in steady state (3D-CISS) MRI sequences, with reconstructions perpendicular to the bilateral internal auditory channel, were required to demonstrate facial nerve anomalies. (Sasaki M, Imamura Y, Sato N. Magnetic resonance imaging in congenital facial palsy. Brain Dev Feb 2008;30:206-210).(Respond: Dr M Sasaki, E-mail: massaki@nc np.go.jp).

COMMENT. Three-dimensional constructive interference in steady state MRI sequence is useful in the differential diagnosis of congenital facial palsy. 3D-CISS MRI provides T2-weighted images with high spatial resolution.

MOVEMENT DISORDERS

GENETICS OF EARLY ONSET RESTLESS LEGS SYNDROME

Linkage analysis was performed in a four-generational German family with restless legs syndrome (RLS) affecting 15 of 37 family members, in a study at the University of Lubeck, Germany. Age at onset was in early childhood or adolescence in 9 (60%) cases. Clinical findings included a desire to move the legs, paresthesias, motor restlessness at night resulting in sleep disturbance and daytime fatigue. Several family members had severe psychiatric problems, including depression and personality disorder. The inheritance pattern was autosomal dominant. A new RLS gene locus (RLS3) was identified on chromosome 9 in all of 12 patients tested, and 11 of these carried an additional closely linked RLS locus. (Lohmann-Hedrich K, Neumann A, Kleensang A, et al. Evidence for linkage of restless legs syndrome to chromosome 9p. Are there two distinct loci? Neurology February 2008;70:686-694). (Reprints: Dr Christine Klein, Department of Neurology, University of Lubeck, 23538 Lubeck, Germany, E-mail: christine.klein@neuro.uni-luebeck.de).

COMMENT. A linkage to a new locus (RLS3) on chromosome 9p has been identified in a family with RLS of early onset. Five gene loci have previously been mapped in cases of primary RLS to chromosomes 12q, 14q, 9p, 2q, and 20p. To date, no gene mutation has been found. RLS is primary or secondary. The primary form is highly familial; secondary RLS is often associated with iron deficiency, renal disease, or pregnancy. The pathophysiology may be related to dopamine insufficiency and low iron storage in substantia nigra.

NEUROCUTANEOUS SYNDROMES

LINEAR NEVUS SEBACEUM SYNDROME AND INFANTILE SPASMS

Two infants with linear nevus sebaceum syndrome and infantile spasms are reported from Safra Childrens Hospital, Sheba Medical Center, Tel Hashomer, Israel; and Hospital for Sick Children, Toronto, Canada. Case 1 presented at age 4 months with focal motor and generalized convulsive seizures with low-grade fever. Family history was positive for febrile seizures in the mother. A 3-cm gray-yellow scaly patch was noted on the frontal-central scalp area that enlarged and turned red and thickened after discharge. Brain MRI showed bilateral

polymicrogyria and subependymal heterotopia. At age 8 months, she developed infantile spasms and modified hypsarrhythmia on EEG. Seizures were controlled with vigabatrin. At age 2.5 years, she is seizure-free on topiramate and clobazam. Case 2 presented at age 3 months with generalized seizures and two 2.5 cm hyperpigmented nevi in the right parietal-temporal area. Eye exam revealed a right esotropia and coloboma. MRI showed right hemimegalencephaly. EEG recorded right hemisphere slowing and interictal spikes and slow waves. Seizures were controlled with phenobarbital. He was readmitted at age 11 months with infantile spasms and hypsarrhythmia, resistant to vigabatrin and controlled by ACTH. At age 3 years he presented with frequent generalized tonic-clonic and myoclonic seizures and developmental delay. The pigmented nevi had each enlarged to 5 cm in diameter. EEG showed a generalized slow-spike and wave pattern, consistent with Lennox-Gastaut syndrome. The literature on linear nevus sebaceous syndrome is reviewed. (Menascu S, Donner EJ. Linear nevus sebaceous syndrome: case reports and review of the literature. Pediatr Neurol March 2008;38:207-210). (Respond: Dr Menascu, Atad St, PO Box 69, Omer 84965, Israel).

COMMENT. The neurocutaneous linear nevus sebaceum syndrome is characterized by a triad of epidermal nevi, seizures, and mental retardation. A review of 60 cases by Solomon et al, in 1975, described the dermatologic lesions as epidermal nevi associated with neurologic, ophthalmic, skeletal, cardiovascular, and urological abnormalities. Children with a suspected linear nevus sebaceum syndrome should have EEG, MRI, and ophthalmology exams. Seizures occur in up to 75% cases, frequently infantile spasms, West syndrome and evolving into Lennox-Gastaut syndrome. The term linear nevus sebaceum syndrome is usually reserved for cases with midline nevi, while "epidermal nevus syndrome" is more inclusive for all varieties of epidermal nevi.

SEIZURE DISORDERS

HERBAL MEDICINE AND EPILEPSY

The potentially harmful effects of herbal remedies and herb-antiepileptic drug interactions in patients with epilepsy are reviewed by researchers at the Center for Integrative Complementary Medicine, and Division of Neurology and Toxicology, Shaare Zedek Medical Center, Jerusalem, Israel. In the US, the Dietary Supplement Health and Education Act of 1994 removed herbal preparations from the jurisdiction of the FDA. Physicians are unaware of the degree of usage of complementary and alternative medicine (CAM) by their patients. Less than 40% of patients using CAM share this information with their conventional physician. Case reports of herb-induced seizures published between 1993 and 2004 include 14 infants treated with gingko biloba, pennyroyal (in mint tea), or star anise (used for colic). Japanese star anise contains the neurotoxin anisatin, a potent GABA antagonist. Chinese star anise is a spice and tea, used as a sedative for infants with colic. It contains veanisatins that are epileptogenic. Seven infants with star anise-induced seizures were seen at the Miami Children's Hospital ED over a 2-year period (Ize-Ludlow D, et al. Pediatrics 2004;114:653-656). Pennyroyal contains seizure-inducing monoterpine R-(plus)pulegone. Gingkotoxin, 4-O-methoxypyridoxine (MPN) is a vitamin B6 derivative that inhibits GABA synthesis from glutamate. The toxin is contained in either gingko seeds or