arachnoid cyst in the right anterior temporal lobe. (Kaga M. Language disorders in Landau-Kleffner syndrome. J Child Neurol Feb 1999;14:118-122). (Respond: Dr Makiko Kaga, Department of Developmental Disorders, National Institute of Mental Health, National Center of Neurology and Psychiatry, 1-7-3 Kohnodai, Ichikawa, Chiba 272, Japan).

COMMENT. Long-term follow-up of children with Landau-Kleffner syndrome into adult life shows a sequence of language disorders beginning at 4 to 6 years of age with sensory aphasia, and auditory verbal agnosia (word deafness), nonverbal agnosia, with or without anosognosia or denial of disability, and later showing improvement in auditory comprehension and speech but awareness of the impairments. The severity of language disorders, epileptic seizures, and EEG epileptiform discharges are not necessarily directly related, and antiepileptic medications which may control the seizures do not usually benefit the language deficits. Landau WM and Kleffner FR, in their original article (Syndrome of acquired aphsia with convulsive disorder in children. Neurology Aug 1957;7:523-530), state that "EEG improvement tends to parallel improvement in speech reeducation."

Magnetoencephalography in Landau-Kleffner syndrome has been studied prior to subpial transection in 4 children in Helsinki, Finland. The earliest spike activity on the EEG originated in the intrasylvian cortex, and MEG data focussed on small otherwise overlooked EEG potentials, helping to identify the primary epileptogenic source and influencing the planning of surgery. ((Paetau R, Granstrom M-L. Blomstedt G et al. Magnetoencephgalography in presurgical evaluation of children with the Landau-Kleffner syndrome. Epilepsia March 1999;40:326-335).

ANTIEPILEPTIC DRUGS

ENDOCRINE FUNCTION AND ANTIEPILEPTIC DRUGS

The effects of valproate (VPA) in 40, carbamazepine (CBZ) in 19, and oxcarbazepine (OXC) in 18 girls with epilepsy on growth and maturation were compared to 49 healthy untreated controls examined at 8 to 18 years of age at the Departments of Neurology, Pediatrics, and Radiology, University of Oulu, Finland. VPA, CBZ, or OXC did not affect linear growth, followed longitudinally from the age of 1 year, or pubertal development. An increase in weight in VPA-treated girls occurred in those starting treatment either before or during puberty, and was not associated with hyperinsulinemia. High circulating concentrations of plasma insulin-like growth factor-1 in girls taking CBZ or OXC were of uncertain significance, and unassociated with abnormal weight gain. (Rattya J, Vainionpaa L, Knip M, Lanning P, Isojarvi JIT. The effects of valproate, carbamazepine, and oxcarbazepine on growth and sexual maturation in girls with epilepsy. Pediatrics March 1999;103:588-593). (Reprints: Johanna Rattya MD, Department of Neurology, University of Oulu, Kajaanintie 50, 90220 Oulu, Finland).

COMMENT. Neither VPA medication itself nor the associated weight gain affect linear growth or pubertal development of girls with epilepsy. Hyperinsulinemia, reported in obese adult women taking VPA for epilepsy, is not observed in girls treated for an average of 2.8 years. Weight gain in girls treated with VPA is slow and progressive and should be monitored regularly. These authors have reported on valproate-induced obesity and polycystic ovarian syndrome in women with epilepsy (see Progress in Pediatric Neurology III, 1997).