COMMENT. The prognostic value of video-EEG monitoring and estimation of risk of SUDEP in patients with refractory generalized seizures is demonstrated in this study. Patients at risk should receive more aggressive medical and possibly surgical therapy as well as close postictal observation and stimulation to minimize the occurrence of central inhibition, apnea and SUDEP. Dr LJ Hirsch, Neurological Institute, New York, NY, in an editorial, recommends the use of multimodal ambulatory monitoring (respiration, oxygen saturation, CO2, EKG, and EEG) in epilepsy patients at risk of SUDEP. He cites a prospective, international study in Europe and Australia, using cardio-respiratory and EEG monitoring in 16 cases of SUDEP, reporting central shutdown and respiratory arrest as the primary mechanism of death. (Findings presented at recent European Congress of Epileptology). The quality of life of patients with epilepsy needs protection, but the increasing interest in SUDEP indicates the need for closer monitoring of patients at risk.

RISK FOR DEVELOPING EPILEPSY

Lifetime risk and cumulative incidence of epilepsy were examined among Rochester, MN, residents between 1960 and 1979, and are reported from Columbia University, New York; and Mayo Clinic. Rochester, MN. Among 412 individuals identified with incident epilepsy, lifetime risk was 1.6% to age 50 and 3.0% to age 80; cumulative incidence 0.9% to age 20, 1.7% to age 50 and 3.4% to age 80. Lifetime risk through 87 years of age increased over time from 3.5% in 1960-69 to 4.2% in 1970-79. One in 26 people will develop epilepsy during their lifetime. (Hesdorffer DC, Logroscino G, Benn EKT, Katri N, Cascino G, Hauser WA. Estimating risk for developing epilepsy. A population-based study in Rochester, Minnesota. **Neurology** January 2011;76:23-27). (Response and reprints: Dr DC Hesdorffer, Columbia University, 630 West 168th St, P&S Unit 16, New York, NY 10032. E-mail: dch5@columbia.edu).

COMMENT. Lifetime risk forecasts the need for epilepsy services in a community. The greatest need for these services is in infants and the elderly. The incidence curve varies with age, with high rates in infants under 1 year of age, low rates from age 20 to 60, and an increase from 60 to age 80, a time period that is rapidly expanding in the general population. The authors estimate that 12 million individuals in the US will develop epilepsy in their lifetime.

WEST SYNDROME REMISSION FOLLOWING ACUTE VIRAL INFECTION

Researchers at Nihon University School of Medicine, Tokyo, Japan, report 11 children with intractable epilepsy (West syndrome in 6 and myoclonic seizures in 5) who showed clinical and electrographic improvement following acute viral infection. Seizure remission occurred following exanthem subitum in 5 patients, rotavirus gastroenteritis in 2, measles in 2, herpetic stomatitis in 1, and common cold in 1. In patients with West syndrome, salaam and/or tonic spasms resolved within 6 days after onset of viral infection, and hypsarrhythmia was modified and evolved to localized spikes. Myoclonic seizures resolved rapidly and completely and the EEG gradually normalized or improved.

Four of the 11 patients became seizure free for 5 to 20 years, 1 child remained seizure free for 12 months after viral infection, and 6 relapsed and had seizure recurrence within 14 days to 1 month after remission. Characteristic findings in the 4 patients with prolonged remission included: 1) normal brain CT/MRI, 2) normal development prior to onset of epilepsy, and 3) a short duration of epilepsy before occurrence of viral infection. One mechanism for seizure remission proposed is a change in immune state following viral infection similar to the effect of immunoglobulin therapy. Alternative hypotheses include: 1) direct suppression of seizures by virus, 2) elevation of serum cortisol level, or 3) anti-inflammatory cytokine response to viral infection. (Fujita Y, Imai Y, Ishii W et al. Improvement of intractable childhood epilepsy following acute viral infection. **Brain Dev** January 2011;33:62-68). (Respond: Dr Yukihiko Fujita, Nihon University School of Medicine, Tokyo, Japan. E-mail: yfujita@med.nihon-u.ac.jp).

COMMENT. Several reports of seizure remission following viral infection have involved patients with West syndrome, and some cases have a spontaneous remission. Among cases of viral-induced remission of epilepsy, exanthem subitum and human herpes virus-6 appear to be involved most commonly, and West syndrome is the most susceptible epilepsy. Previous reports of viral-induced seizure remission have described the clinical findings but have lacked the details of electrographic and hypsarrhythmia modification. The mechanism and therapeutic possibilities of this report deserve further study.

PROLONGED EEG DEPRESSION AS A PREDICTOR OF WEST SYNDROME IN TERM INFANTS WITH HIE

Seventeen term and near-term infants with hypoxic ischemic encephalopathy (HIE) were studied at Okazaki City Hospital and other centers in Japan to clarify the relationship between prolonged depression of the EEG and later development of West syndrome (WS). Group A, 4 patients with prolonged EEG depression over 21 days, and Group B, 13 with disappearance of EEG depression by 21 days of age, are compared. WS developed in all 4 infants in Group A, but only 1 of 13 in Group B. Abnormal irregular faster waves (irregular spiky beta or sharp theta) in 11 infants from both groups between 2 and 28 days of age were related to an adverse developmental outcome but not to West syndrome. Prolonged depression of EEG over 21 days of age in term infants with HIE is a predictor of later development of WS (sensitivity 0.80, specificity 1.0, pos predictive value of 1.0, neg predictive value 0.92). In comparison, MRI lesions in basal ganglia, thalamus, and white matter had a sensitivity of 0.80, specificity 0.92, pos predictive value of 0.80, and negative predictive value of 0.92. (Kato T, Okumura A, Hayakawa F et al. Prolonged EEG depression in term and near-term infants with hypoxic ischemic encephalopathy and later development of West syndrome. **Epilepsia** Dec 2010;51(12):2392-2396). (Respond: Toru Kato MD, Department of Pediatrics, Okazaki City Hospital, Okazaki, Aichi, 444-8553, Japan. E-mail: kato-jes@umin.ac.jp).

COMMENT. Watanabe and colleagues (1987, 2001) previously reported neonatal EEG abnormalities that preceded WS, but the predictive value of EEG depression was not established. The abnormal irregular faster waves, not significantly related to the later