propofol, midazolam, or thiopental/pentobarbital); Stage SRSE; after 24 hours in status. Treatments used and recommended for SRSE include the following in this order: anesthetic agents and antiepileptic drugs, identify and treat the cause, magnesium infusion, iv pyridoxine, consider steroids and immunotherapy, consider resective neurosurgery in lesional SE, consider multiple subpial transection, hypothermia, ketogenic diet, transcranial magnetic stimulation, vagal nerve stimulation, consider deep brain stimulation, electroconvulsive therapy, drainage of cerebrospinal fluid, and older drug therapies. Treatment of the underlying cause is stressed. Premature withdrawal of therapy is discouraged, since recovery can occur after even weeks of status epilepticus. A multinational database of outcomes of individual therapies is urgently needed. (Shorvon S, Ferlisi M. The treatment of super-refractory status epilepticus: a critical review of available therapies and a clinical treatment protocol. **Brain** 2011;134:2802-2818). (Respond: Professor Simon Shorvon, Box 5, National Hospital for Neurology and Neurosurgery, Queen Square, London WC1N 3BG, UK. E-mail: s.shorvon@ion.ucl.ac.uk).

COMMENT. The authors offer an interesting review of the mechanism of super-refractory SE. A reduction in GABAergic activity is proposed as a reason for status epilepticus to develop and also for an increasing ineffectiveness of GABAergic drugs (such as benzodiazepines or barbiturates) in controlling status. Other reported proposed causes for SRSE include mitochondrial insufficiency, changes in extracellular ionic environment, inflammatory disease, opening of the blood brain barrier, and possible changes in gene expression. Emergency therapy directed at the cause is crucial in terminating the seizure. (Neligan and Shorvon, 2011).

HYPOTHALAMIC HAMARTOMAS AND GELASTIC EPILEPSY

Researchers at Stanford University and other neurological centers in the United States performed a retrospective review and analysis of the clinical presentation and neuroanatomical features of hypothalamic lesions in 100 cases of gelastic epilepsy. Age of seizure onset was 10.52 +/- 18.12 months. Preoperative brain MRI was delayed a mean of 133.2 +/- 126.7 months after reported onset of seizures. All patients had gelastic seizures; 68 had gelastic epilepsy plus other types of seizures, including partial and generalized. Four had infantile spasms. Cognitive or developmental impairment (IQ <70) occurred in 43% (28% of patients with gelastic seizures only and 50% of those with gelastic plus multiple seizure types; p=0.052). All patients had refractory seizures. Patients with gelastic seizures plus had significantly longer duration of epilepsy ($p<0.001$). Precocious puberty occurred in 23%. Patients with cognitive impairment and those with precocious puberty had significantly larger lesions involving the anterior and posterior hypothalamus, at the level of the mammillary bodies. (Parvizi J, Le S, Foster BL, et al. Gelastic epilepsy and hypothalamic hamartomas: neuroanatomical analysis of brain lesions in 100 patients. **Brain** 2011;134:2960-2968). (Respond: Josef Parvizi MD PhD. E-mail: jparvizi@stanford.edu).

COMMENT. Lesions causing gelastic seizures are all localized to the mammillary level of the posterior hypothalamus. The longer duration of the epilepsy determines the development of other seizure types.