COMMENT. The frequency of skin rash with carbamazepine in this study is disturbingly high, particularly in children over 6 years of age. For additional reports, see Ped Neur Briefs Feb 1991, The introduction of dyes in the carbamazepine tablet in recent years may explain an apparent increase in reports of allergic skin reactions.

In a report from The Liverpool Hospital, New South Wales, Australia, the substitution of oxcarbazepine, a keto derivative thought to be less toxic than CBZ, caused a cross-reactive skin eruption in 3 adult patients who had previously developed exfoliative dermatitis with CBZ. (Beran RG. Epilepsia Jan/Feb 1993; 34: 163).

STATUS EPILEPTICUS

SERUM CORTISOL AND PROGNOSIS OF STATUS EPILEPTICUS

The relation of serum cortisol and CSF *B*-endorphin levels to the prognosis of status epilepticus was evaluated in 27 adults treated in the Comprehensive Epilepsy Program of the Medical College of Virginia, Richmond, VA. Poor prognosis was defined as death or severe neurologic or medical complications during or within one week after status epilepticus of more than 30 minutes. Blood cortisol levels were significantly elevated in patients with a poor outcome. CSF *B*-endorphins were significantly increased but did not correlate with prognosis. (Calabrese VP et al. Serum cortisol and cerebrospinal fluid *B*-endorphins in status epilepticus. Their possible relation to prognosis. Arch Neurol July 1993; 50: 689-693). (Reprints: Dr Calabrese, Dept of Neurology, MCV Station-Box 599, Richmond, VA 23298).

COMMENT. Serum cortisol determinations within 12 hours after control of status epilepticus may predict prognosis. The authors also propose that the rise in cortisol levels may contribute to the pathology of status epilepticus.

OCCULT CORTICAL DYSPLASIA AND FOCAL STATUS

Four patients, ages 4, 6, 13, and 21 years, with life-threatening focal motor status epilepticus, normal pre-operative MRI, and focal cortical dysplasia defined only in the surgical specimens obtained by corticectomies, are reported from the Montreal Neurological Institute; Austin Hospital, Heidelberg, Australia; University of California, San Francisco; and Royal Children's Hospital, Melbourne, Australia. Seizures began between 3 months and 18 years of age, and therapy-resistant focal status developed after 18 months to 3 years, resulting in persistent motor deficits. A total of 12 MRIs failed to reveal abnormalities. Ictal SPECT was positive in 2 patients. Seizures were controlled after surgery, and 2 patients were seizure-free. (Desbiens R et al. Life-threatening focal status epilepticus due to occult cortical dysplasia.

Arch Neurol July 1993; 50: 695-700). (Reprints: Dr Frederick Andermann, Montreal Neurological Hosp and Institute, 3801 University St, Montreal, Quebec, Canada H3A 2B4).

COMMENT. Neuronal migration disorder as a cause of focal motor status epilepticus or epilepsia partialis continua in a child may not be excluded by the MRI and may require surgical intervention for diagnosis and treatment.

VASCULAR DISORDERS

MOYAMOYA DISEASE AND HEMIPLEGIC MIGRAINE

A 6-year-old girl presenting with a 6-month history of classical familial migraine, complicated by transitory hemiparesis and moyamoya disease diagnosed by MRI, is reported from the Kaiser Permanente Medical Center, Hayward, CA. Headaches occurred each week and were associated with nausea, vomiting, and photophobia. She awoke on 3 occasions with right-sided weakness, numbness, and garbled speech. All symptoms resolved after two hours of sleep. Neurologic exam was normal between attacks. MRI showed bilateral occlusion of the carotid arteries and increased vascularity of collateral vessels in the basal ganglia, consistent with moyamoya. (Bernstein AL. Hemiplegic migraine and moyamoya disease. AJDC July 1993; 147: 718-719). (Dr Bernstein, Dept of Neurology, Kaiser Permanente Medical Center, 27400 Hesperian Blvd, Hayward, CA 94545).

COMMENT. Children with a diagnosis of hemiplegic migraine may have a vascular malformation or moyamoya disease as the underlying pathology. The clinical course depends on the rapidity and extent of vascular occlusion and the ability to develop a collateral circulation. Early diagnosis and surgical revascularization have been advocated because of the risk of permanent neurologic deficits. (See Ped Neur Briefs July 1987, and Progress in Pediatric Neurology, Chicago, PNB Publ, 1991, pp389-391).

ENDOCRINE DISORDERS

ADRENAL, ALACRIMAL, ACHALASIA SYNDROME

The results of a British and European Paediatric Endocrinology Society, multicenter collaborative, questionnnaire study of the neurological complications of familial glucocorticoid deficiency syndrome are reported from the Hospital for Sick Children, Great Ormond Street, London. Of 20 patients identified, ages 2 to 29 years, all had impaired cortisol secretion, 19 absent tear secretion, 15 achalasia of the cardia, and 17 had neurologic abnormalities including hyperreflexia, hypertonia, Babinski signs, muscle