be a fraction of the asymptomatic vigabatrin-induced visual field defects that are unrecognized. At an international meeting in London, sponsored by Hoechst Marion Roussel, it was concluded that routine ophthalmological screening of all patients taking vigabatrin cannot be justified. Confrontational visual field examination is advised at baseline and follow-up of patients on vigabatrin, when practical. The risk:benefit ratio should be calculated for each individual, if visual field defects are uncovered. In infants receiving vigabatrin for the treatment of infantile spasms, the consensus argued that the benefits outweighed the risks. (Harding GFA. Benefit: risk ratio must be calculated for individual patients. BMJ Jan 17, 1998;316:232-233).

NONCONVULSIVE SEIZURES AND BRAIN DAMAGE

Possible brain damage resulting from nonconvulsive seizures is debated "for" by Young GB and Jordan KG (Department of Clinical Neurological Sciences, 375 South St, London, Ontario, Canada N6A 4G5), and "against" by Aminoff MJ (Box 0114, Room M-794, Department of Neurology, School of Medicine, University of California, San Francisco, CA 94143). The brain damage school favors immediate and vigorous tratment of nonconvulsive status epilepticus (NCSE), whereas the non-damage group, while advocating treatment of NCSE, argues against potentially hazardous therapeutic extremes, such as general anesthesia. The section editor (Hachinski V) of these "Controversies in Neurology" concludes that both camps agree that brief absence seizures result in no detectable harm, though subtle cerebral changes may be difficult to detect. In balancing the potential harm of treatment compared with consequencies of nontreatment, antiepileptic side effects are usually transient, whereas sequelae of nontreatment may be cumulative and permanent. (Hachinski V. Nonconvulsive seizures and brain damage. Arch Neurol Jan 1998;55:120). (Respond: Vladimir Hachinski MD, Dept of Clinical Neurological Sciences, London Health Sciences Centre, 339 Windermere Rd, London, Ontario, Canada N6A 5A5).

COMMENT. In pediatric neurology, most practitioners have followed the occasional child with absence epilepsy whose seizures prove refractory to various mono- and polytherapies as well as the ketogenic diet. In some cases it is distressing to observe a gradual though inexorable cognitive impairment, rarely to the level of dementia. The cause of this regression may be unexplained, or linked to the nonconvulsive seizures or to the therapy or both. In some cases the drugs may be more injurious than the seizures, if sedative and cognitive-depressant doses are continued for long periods. In others, patients in unrecognized status absence epilepsy, a dramatic recovery of mental alertness may follow effective vigorous and acute, short-term therapy.

My colleague, Dr Cynthia Stack, Director of Neurophysiology and Electroencephalography at Children's Memorial Hospital, Chicago, was consulted. She is in favor of prompt and vigorous therapy of nonconvulsive status epilepticus in children. Dr Stack supports the theory that nonconvulsive status may signify a non-reactive and more severe state of brain damage than convulsive status epilepsy.

POST-HEAD TRAUMA PROPHYLACTIC ANTICONVULSANTS

The effectiveness and safety of antiepileptic agents in the treatment of acute traumatic head injury were determined at the Institute of Child Health, University College, London, UK, by review of 10 randomized controlled trials involving 2036 patients identified from various databases. The pooled relative risk (RR) for early seizure prevention (within the first week after injury) was 0.34; 10

patients would need to be treated to keep one free from seizures in the acute phase. Seizure control was not accompanied by a reduction in mortality (pooled RR=1.49) or neurological disability. The occurrence of late seizures was not reduced by AEDs; the relative risk of late seizures, based on 4 studies, was 1.28. The risk of skin rashes was increased (RR=1.57). The true net benefit of prophylactic antiepileptic agents was undetermined. (Schierhout G, Roberts I. Prophylactic antiepileptic agents after head injury: a systematic review. J Neurol Neurosurg Psychiatry Jan 1998;64:108-112). (Respond: Dr GH Schierhout, Department of Epidemiology and Public Health, Institute of Child Health, University College, 30 Guilford St, London WC1N 1EH, UK).

COMMENT. Prophylactic antiepileptic drug therapy initiated after acute head injury may reduce the occurrence of seizures in the first week, but has no effect on the development of late seizures, on mortality or neurological disability, and treatment is associated with the risk of skin rash, a potentially serious side effect. For 100 patients treated 10 may be seizure-free in the first week, but 4 will develop skin rash.

See Progress in Pediatric Neurology II, (PNB Publishers, 1994;pp137-138) for reports of prophylactic anticonvulsant drugs after craniotomy. In one study from Walton Hospital, Liverpool, UK, skin rashes occurred in 13% of patients treated with carbamazepine or phenytoin (Foy PM et al, 1992), and the occurrence of seizures within the first post-operative week did not increase the likelihood of late epilepsy. The authors concluded that drug therapy should not be recommended routinely following craniotomy.

Endocrine functions following severe head trauma were assessed in 21 children at Chaim Sheba Medical Center, Tel-Hashomer, Israel (Goldman M et al. Pediatr Neurol Nov 1997;17:339-343). Advanced bone age without other signs of precocious puberty were found in 3 prepubescent children. Biochemical and hormonal determinations were normal, and no endocrine abnormalities were found in children examined 4 months or more following injury. Clinical monitoring is sufficient, and specific hormonal measurements are required only when warranted by abnormal signs.

HEADACHE

FOOD-RELATED HEADACHES

The literature relating to food and headache is reviewed by an authority on migraine, Dr Clifford Rose of the London Neurological Centre, London, UK. Wine was the earliest reference to dietary migraine, a comment attributed to Celsus (25BC-50AD). Fothergill (1712-1780) was the first to incriminate chocolate as a precipitant of migraine. More recent studies have postulated the phenylethylamine content as the active provocative ingredient. Martelletti and colleagues (1994) studied the involvement of the immune system, and found a link between interleukins and other cytokines, immune messengers, with histamine and serotonin, neuromediators of pain in migraine. Hanington (1967) proposed the theory of tyramine contained in cheese as a precipitant of dietary migraine, but subsequent studies provided conflicting results. Caffeine withdrawal is one explanation for weekend migraine sufferers. Octopamine, a biogenic amine, is thought to be the active ingredient in citrus fruit headache. Food additives blamed for exacerbations of migraine in some patients include aspartame (diet-soda headache), nitrites and nitrates (hot dog headache), and glutamate (chinese