in the above patient's seizure disorder, and a change in life style was important in seizure control. Macdonald Critchley (1942) in his report of cases of musicogenic epilepsy considered emotion an important precipitant. (In Lennox WG. Epilepsy and Related Disorders. Boston, Little, Brown, 1960;p362). In his hagiographic obituary of Macdonald Critchley, who died Oct 15, 1997, aged 97, Robert J Joynt described him as "a reminder of the great heritage of our specialty and a vibrant contributor to it." (Arch Neurol Jan 1998;55:122).

LORAZEPAM-INDUCED NEONATAL SEIZURES

Two cases of lorazepam-induced neonatal seizures are reported from the University of Rochester, NY. Patient 1, a full-term male infant intubated for transient tachypnea was given lorazepam (Ativan), 0.1 mg/kg iv, for sedation. Within minutes, the infant developed clonic jerks of both legs and right arm, occurring in bursts of 1 to 3 min for 1 hour. A 24-hour EEG recorded 2 hours after movements resolved was normal. Patient 2 had necrotizing enterocolitis at 3 days of age and was treated briefly with antibiotics. A single dose of lorazepam, 0.1 mg/kg iv, was given as a sedative for placement of a percutaneous intravenous catheter. Within 1 min, intermittent clonic movements of all extremities were followed by flaccidity and pallor that lasted for 5 min, and clonus recurred every 5 minutes for one half hour. Follow-up examinations at 12 months were normal in both infants. (Chess PR, D'Angio CT. Clonic movements following lorazepam administration in full-term infants. Arch Pediatr Adolesc Med Jan 1998;152:98-99). (Respond: Dr Patricia R Chess, Department of Pediatrics, University of Rochester, Rochester, NY 14642).

COMMENT. Myoclonus associated with lorazepam therapy in premature neonates is a well known side effect. This appears to be the first report of seizure-like activity following lorazepam injection in otherwise neurologically normal, full-term infants. Additional etiologic factors considered included the propylene glycol adjuvant in the injection and the rate of administration.

VIGABATRIN-INDUCED VISUAL FIELD DEFECTS

A drug surveillance database at Hoechst Marion Roussel, manufacturers of vigabatrin, identified 92 cases of symptomatic visual field defects associated with vigabatrin (usually as add-on therapy) between 1990 and 1997. Two further asymptomatic cases have been identified in patients treated at the Prince of Wales Hospital and University of Sydney, Australia. Patient 1, a 21-year-old man with complex partial seizures had received carbamazepine for 12 years and vigabatrin 2 g/day for three years. Clinical visual field testing was normal, but Goldman perimetry showed bilateral nasal field defects and some superior peripheral field constriction. Electroretinography was also abnormal, showing reduced b wave amplitude. Patient 2, a 36-year-old woman with tonic-clonic seizures treated with valproate and carbamazepine for 12 years, followed by carbamazepine and vigabatrin 2 g/day for two years, had questionable defects on clinical visual field testing and definite peripheral binasal field loss on Humphrey perimetry. Electroretinography showed reduction in b wave amplitudes in nasal fields. (Mackenzie R, Klistorner A. Severe persistent visual field constriction associated with vigabatrin. Asymptomatic as well as symptomatic defects occur with vigabatrin. BMJ Jan 17, 1998;316:232-233). (Respond: Dr Rod Mackenzie, Director, Comprehensive Epilepsy Service, Prince of Wales Hospital, Sydney, Australia).

COMMENT. Vigabatrin antiepileptic therapy poses the risk of visual field damage that may be persistent. The number of symptomatic cases identified could

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