family members were treated with acetazolamide. The episodic vertigo was either abolished or markedly decreased in frequency and severity. The age of onset of the vertigo varied from age two to 32. The episodes lasted from several hours to several days and were complicated by nausea and vomiting and later, a mildly progressive ataxia. Associated symptoms included horizontal or vertical diplopia, slurred speech, and positional downbeat and rebound nystagmus between attacks. The findings suggested a lesion of the vestibulocerebellum. In seven patients examined the MRI was normal. (Baloh RW, Winder A. Acetazolamide-responsive vestibulocerebellar syndrome: Clinical and oculographic features. Neurology March 1991; 41:429-433).

COMMENT. The familial periodic ataxia syndromes have been divided into two types, 1) a vestibulocerebellar syndrome and 2) diffuse cerebellar ataxia, as described by Parker, in which vertigo is infrequent and ataxia is more pronounced. Both syndromes appear to respond to acetazolamide.

SEIZURE DISORDERS

THE ELECTROENCEPHALOGRAM IN FEBRILE SEIZURES

The clinical value of EEG investigations in children with febrile seizures is reviewed from the Section of Child and Adolescent Psychiatry, Parke Hospital for Children, Old Road, Hedington, Oxford, England. Reports of EEG abnormalities in children with a history of febrile seizures include 1) ictal, generalized spiking, or lateralized spike wave discharge; 2) postictal, slow activity, spike wave or spikes; and 3) serial EEG's showing bisynchronous theta activity, bisynchronous spike wave at rest and during over breathing, bisynchronous spike wave on photic stimulation, focal spikes or sharp waves, and hypnagogic paroxysmal spike wave. The author concludes that an early postictal standard EEG will not be helpful in: a) the distinction between clinically simple and atypical seizures, b) the identification of a cerebral infective etiology, and c) the prediction of later occurrence of either further febrile or later afebrile seizures. A limited place for EEG studies is proposed in connection with febrile seizures associated with suspect cerebral pathology. If the child showed developmental delay, if the first seizure occurred below the age of 12 months, or if the seizures are partial or focal in pattern, a possible structural brain pathology is more likely. In addition, a prolonged febrile seizure, especially if followed by residual neurologic signs or developmental regression, may be evidence of structural cerebral damage and may require further investigation. EEG can be a useful ancillary investigation suggesting a persistent brain pathology, even in the presence of a normal CT scan, if it demonstrates a slow wave abnormality with or without spike or sharp waves that is persistent. This implies the use of serial EEG recordings over a period of weeks rather than a single recording. The EEG may also be helpful in research investigations concerning genetic factors and the possible connection between febrile seizures and benign rolandic epilepsy of childhood.

Also, the possibility of defining clinically important subgroups of febrile seizures might be facilitated by the use of the EEG. It is emphasized that the EEG recordings should include natural sleep and wakefulness, drowsiness, at least stages I and II of nonrapid eye movement sleep, and arousal. Failure to standardize the procedure makes comparisons between different studies very difficult. (Stores G. When does an EEG contribute to the management of febrile seizures? Arch Dis Child April 1991; 66:554-557).

COMMENT. The author correctly emphasizes the need for standardized EEG procedures, including sleep recordings, in assessing the value of the EEG in the management of febrile seizures. The conclusion that EEG findings may lack predictive value for the later occurrence of epilepsy in children with febrile seizures has been based on longitudinal EEG investigations that were sometimes lacking in this necessary standardization and activation procedures, including sleep. Some earlier reports, omitted from the list of references cited in the present review, have demonstrated a predictive value for the EEG and later occurrence of afebrile seizures (Millichap JG. Febrile convulsions. Macmillan New York 1967). Further research would be justified.

PARENTAL REACTION TO FEBRILE CONVULSIONS

The parental reaction to a child's first febrile convulsion was investigated by telephone interview from the Department of Paediatrics, Randers Central Hospital, Denmark. Interviews were conducted in 52 cases from 3-20 months after the convulsion. Fear that the child would die during the fit was volunteered by 44% of parents and another 33% admitted the same reaction when specifically asked about it. Appropriate treatment, i.e. cooling the child and/or placing him in a side position was used in 63% of cases. The child was vigorously shaken during the seizure in 15%. Changes in the behavior of the parents following the child's first seizure included restless sleep in 60%, and dyspeptic symptoms in 29%. The frequency of parental behavioral symptoms rose dramatically if the child had more than a single fit. Many parents wished they had known more about fever and febrile convulsions in children. The general level of knowledge of febrile convulsions among parents of young children is low and the reaction of the parents to the first fit is often severe and persistent. Parents should be given written general information about fever and febrile convulsions, and information to parents who have witnessed a convulsion must be both verbal and written. It is concluded that parents of young children should be better informed about febrile convulsions before they occur. Well informed parents managed febrile convulsions better than those uninformed. (Balslev T. Parental reactions to a child's first febrile convulsion. A follow-up investigation. Acta Paediatr Scand April 1991; 80:466-469).

COMMENT. The controversy concerning the long-term prophylactic use of phenobarbital in the management of febrile seizures