

TREATMENT OF BENIGN ROLANDIC EPILEPSY

The outcome of ten untreated and 20 treated patients with benign childhood epilepsy with Rolandic spikes was studied retrospectively after recovery at the Institute of Neurology, University of Bologna, Italy. The patients had been seizure free for at least five years. The mean period of active epilepsy was 16 months for patients and 28 months for controls. All patients and controls had Rolandic and/or centrottemporal spikes on their awake EEG recording during the active epilepsy period. Seizure frequencies, seizure occurrence and duration of active epilepsy were similar in both the treated and untreated groups. No differences were found in social adjustment of the patients. The authors conclude that antiepileptic treatment may be unnecessary in most cases of benign epilepsy with Rolandic spikes (Ambrosetto, G, Tassinari CA Antiepileptic drug treatment of benign childhood epilepsy with Rolandic spikes: is it necessary? Epilepsia Nov/Dec 1990; 31:802-805).

COMMENT. The authors comment that the results should be interpreted with caution because they are retrospective. If the data are confirmed by prospective studies antiepileptic drugs should be withheld unless seizures are frequently recurrent. Since the seizures are generally nocturnal and usually involve the oropharynx, parental anxiety and dangers of suffocation are factors that may dictate the need for treatment.

MOVEMENT DISORDERS

DYSTONIA DUE TO PERINATAL ASPHYXIA

Delayed onset dystonia associated with perinatal asphyxia in two patients and asphyxia in early childhood in two patients is reported from the Neurological Institute of New York. In the perinatal group the mean age of onset was 12.9 years; dystonia progressed for a mean of 7 years and as long as 28 years. The patients had moderate motor disability and none was wheelchair bound. The prognosis was better than that of childhood onset idiopathic torsion dystonias. In the absence of any identifiable cause of dystonia the authors considered the asphyxia as related either directly or indirectly to the development of dystonia. (Saint Hilaire MH et al. Delayed onset dystonia due to perinatal or early childhood asphyxia. Neurology Feb 1991; 41: 260-222).

COMMENT. Despite the known difficulties in relating adverse perinatal events to static encephalopathies of childhood, the authors claim a relationship between perinatal asphyxia and late onset dystonia based on exclusion of other causes of dystonia and documentation by birth records or a verbal history that asphyxia occurred.