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SEIZURE DISORDERS

ANATOMY OF *DEJA VU* IN TEMPORAL LOBE EPILEPSY

Sixteen patients, ages 16 to 32 years, implanted with depth electrodes at Hopital Saint-Anne, Paris, France, had experienced a dreamy state (*deja-vu - deja vecu*, memories of complete scenes, or vague reminiscence) during sterotactic EEG (SEEG). Seizures had been present from 2 to 25 years, they began before the age of 10 years in 50% of patients, and occurred at least once a week. Etiological factors included neonatal injury (6), febrile convulsion (1), tumors (3). Most patients had dreamy states in their spontaneous seizures; the amygdala, anterior hippocampus, and temporal neocortex were all involved in recordings. They were also evoked by stimulation of the temporal neocortex (88%), anterior hippocampus (83%), or amygdala (73%). The superior temporal gyrus was more responsive than the middle temporal gyrus. (Bancaud J, Halgren E et al. Anatomical origin of *deja vu* and vivid 'memories' in human temporal lobe epilepsy. Brain Feb 1994;117:71-90). (Respond: Dr E Halgren, Clinique Neurologique, CHRU Pontchaillou, 35033 Rennes Cedex, France).

> COMMENT. *Deja vu* and other dreamy states in patients with temporal lobe epilepsy may originate in and involve both medial and lateral aspects of the temporal lobe and especially the anterior hippocampus, amygdala and superior temporal gyrus.

RISK FACTORS FOR FEBRILE SEIZURE RECURRENCE

The relation between postulated risk factors and seizure recurrence after a first febrile seizure (FS) was assessed by reanalysis of pooled data from five centers and follow-up studies and reported from the Sophia Children's Hospital, Rotterdam, The Netherlands. Of 2496 children with 1410 episodes of recurrent seizures, 32% had one, 15% had two, and 7% had three or more recurrent seizures after a first FS; 7% had a complex FS. The risk of FS recurrence was increased at ages 12 to 24 months, after a first and second recurrence, with a family history of seizures, and following FSs with a relatively low temperature (<40°C). The risk of complex FS was increased if

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onset of FS was <12 months, if family history was positive for unprovoked seizures, and if the initial FS was focal or partial. (Offringa M et al. Risk factors for seizure recurrence in children with febrile seizures: A pooled analysis of individual patient data from five studies. J Pediatr April 1994;124:574-84). (Reprints: Martin Offringa MD, Room EE 2091, Erasmus University, PO Box 1738, 3000 DR, Rotterdam, The Netherlands).

COMMENT. In a previous report of a follow-up study of 155 Dutch children the principal author had concluded that the predictive value of combined risk factors (age at onset, family history, height of fever) was superior to that of single variables (see Ped Neur Briefs March 1992;6:17). Similar risk factors have been identified previously by a metaanalysis study (Berg AT et al. J Pediatr 1990;116:329-37) and a prospective study (Berg AT et al. N Engl J Med 1992;327:1122-7). A threshold to febrile seizures based on the height of body temperature was first established in animals with seizures induced by microwave diathermy (Millichap JG. Pediatrics Jan 1959;23:76-85), and has been confirmed clinically (Febrile Convulsions, New York, Macmillan, 1968).

None of the patients in the pooled analysis study had received monitored prophylactic treatment, continuous or intermittent. Having established that 54% of children had one or more recurrences of febrile seizures, the authors may be encouraged to conduct trials of intermittent oral diazepam in their patient population at increased risk, especially in those between the ages of 12 and 24 months, with a positive family history, and whose first FS occurred with a temperature <40°C.

BENIGN FAMILIAL CONVULSIONS

Results of linkage analysis between benign infantile familial convulsions (BIFC) and two linked DNA markers, D20S19 and D20S20, in 52 members from eight BIFC pedigrees are reported from centers in Montpellier and Paris, France, and Rome and Treviso, Italy. The gene responsible for benign familial neonatal convulsions (BFNC) has been mapped to chromosome 20q in the close vicinity of these two DNA markers. Several recombinants were observed betweeen the BIFC locus and D20S19-D20S20 markers, whereas none appeared between the BFNC locus and the markers in 11 BFNC families. The gene responsible for BFNC is not implicated in BIFC. (Malafosse A et al. Benign infantile familial convulsions are not an allelic form of the benign familial neonatal convulsions gene. Ann Neurol April 1994;35:479-482). (Respond: Dr Malafosse, Laboratoire de Medicine Experimentale, CNRS UPR 9008-INSERM U249, Institut de Biologie, Bvd Henri IV, 34060 Montpellier, France).

COMMENT. The authors distinguish BFNC and BIFC by clinical and genetic markers, as follows: 1) Onset of BFNC is before 3 months and BIFC, after 3 months of age; 2) Seizures, generalized in BFNC and partial in BIFC; and 3) genetic heterogeneity.

Seizure patterns cannot be used to differentiate these benign familial convulsions without documentation by ictal EEG recordings. Ictal EEGs demonstrated a seizure of right frontal onset with secondary generalization and one of right frontal onset which remained focal in a neonate with BFNC presenting with seizures at 50 hours of age, and reported from the Prince of Wales Children's Hospital, Randwick,