Quantitative MRI in patients with idiopathic generalized epilepsy demonstrated subtle, but widespread, cerebral structural changes (focal cerebral dysgenesis) not identified on routine MRI in a study at the Institute of Neurology, London, UK (Woermann FG, Sisodiya SM, Free SL, Duncan JS. Brain Sept 1998;121:1661-7).

FOCAL CORTICAL OPIOIDS AND READING EPILEPSY

The release of endogenous opioids in 5 patients with reading-induced seizures was investigated using C-diprenorphine PET scans at the MRC Cyclotron Unit Hammersmith Hospital, and the Institute of Neurology, London, UK. During reading a scientific paper, opioid-receptor binding in the left parieto-temporo-occipital cortex (Brodmann area 37) was increased in control patients and decreased in those with reading epilepsy. Opioid-like substances may be involved in the termination of reading-induced seizures. (Koepp MJ, Richardson MP, Brooks DJ, Duncan JS. Focal cortical release of endogenous opioids during reading-induced seizures. Lancet Sept 19;352:952-55). (Respond: Prof JS Duncan, National Society for Epilepsy and Institute of Neurology, 33 Queen Square, London WC1N 3BG, UK).

COMMENT. This novel PET approach to the measurement of neurotransmitter changes associated with focal seizure activity during reading-induced seizures provides further information regarding the anatomical localization of a specific learning disability.

SPET SCAN ABNORMALITIES IN EPILEPTIC APHASIA

SPET scans, using Tc-exametezime, EEG and MRI were evaluated in 25 children with language deficits associated with epilepsy treated at the Royal Hospital for Sick Children, Edinburgh, UK. Seizures, with onset between 0.3 and 12 years (mean, 4 yrs), included atypical absence in 15 and tonic-clonic in 10. All had epileptiform EEGs, with enhanced abnormalities in sleep in 16. MRI was abnormal in 6, including tuberous sclerosis cortical lesions in 1, stroke in 1, cortical dysplasia (1), temporal sclerosis (3). SPET scans were abnormal and hypometabolic in 22, bilateral in 7, and anterior, mainly frontal and temporal, but variable in localization in 15. Aphasia was receptive in 24, expressive in 20, and nominal in 8. The acquired communication disorder, with onset between 1.5 and 12 years (mean, 6 yrs), did not meet strict criteria for Landau-Kleffner syndrome. Clinical and/or EEG seizure activity were responsive to clobazam or nitrazepam in 11 patients, and ACTH, alone or with benzodiazepine, was effective in 19. Benzodiazepine sensitivity testing employed in 21 under EEG control was positive in 18 and negative in 3. An encephalopathy secondary to a persistent epileptic discharge and characterized by regional hypometabolism on SPET scan was thought to underly the onset of acquired aphasia. (O'Regan ME, Brown JK, Goodwin GM, Clarke M. Epileptic aphasia: a consequence of regional hypometabolic encephalopathy? Dev Med Child Neurol 1998;40:508-516). (Respond: Dr ME O'Regan, Department of Paediatric Neurology, Royal Hospital for Sick Children, 9 Sciennes Rd, Edinburgh, EH9 1LF, UK).

COMMENT. Acquired epileptic aphasia in young children may be induced by the epileptic focus, as suggested by Deonna (1991). The Edinburgh SPET- and EEG-monitored study supports this hypothesis, finding evidence for a regional hypometabolic encephalopathy secondary to a persistent epileptic discharge, and advocating treatment and suppression of the EEG epileptiform activity, with or without concomitant clinical seizures. In epileptic aphasia we attempt to treat the