VERBAL MEMORY AND THE HIPPOCAMPUS

The relationship of memory impairment and hippocampal damage was studied in 35 patients with medically refractory epilepsy localized to the temporal lobe at the Departments of Neurosurgery, Neuropathology, and Neurology and Psychiatry, Yale University School of Medicine, New Haven, CT. The temporal lobe lesion was left-sided in 18 and right-sided in 17. A history of febrile convulsions was obtained in 27. The mean age at which seizures became recurrent was ten years and the mean age at surgery was 29 years. The groups (left foci versus right foci) did not differ significantly with regard to the history of febrile convulsions, age when recurrent seizures developed, and age at surgery. All patients were seizure-free after surgery at a minimum follow-up of six months. Patients completed the Verbal Selective Reminding Test and the WAIS-R before surgery which involved anteromedial temporal lobectomy and radical hippocampectomy. Contrasted with normative standards for the verbal memory test, the means of patients with left temporal seizure foci (78.9) and right temporal seizure foci (101) were less than that achieved by healthy adults (115). Left temporal seizure foci were associated with significantly greater preoperative verbal memory impairment than right temporal seizure foci. Volumetric cell densities of hippocampal subfields (CA3 and the hilar area) were reduced in all patients with temporal lobe epilepsy when contrasted with autopsy controls and measures of long term memory retrieval were correlated significantly with pyramidal cell densities in CA3 and the hilus for patients with left temporal seizure foci only. No significant correlations were found between measures of memory retrieval and the cell densities of CA1, CA2 or the granular layer. Left temporal seizure foci were associated with significantly greater preoperative verbal memory impairment than right temporal seizure foci. (Sass KJ et al. Verbal memory impairment correlates with hippocampal pyramidal cell density. Neurology Nov 1990; 40:1694-1697).

> COMMENT. This study demonstrates impaired verbal memory in patients with confirmed hippocampal damage. The verbal memory impairment was significantly correlated with hippocampal pyramidal cell density in patients with left temporal seizure foci. A history of febrile seizures in 77% of this group of patients with subsequent complex partial seizures is noteworthy. Lennox WG (Pediatrics 1953; 11:341) found a significantly higher incidence of psychomotor seizures among patients with a history of febrile seizures compared to those with generalized tonic-clonic and absence patterns; psychomotor seizures were diagnosed in 17% of the febrile seizure group and in 5.9% of the grand mal and petit mal groups.

CEREBRAL GLUCOSE METABOLISM AND ADHD

Whole brain and regional rates of glucose metabolism were assessed by PET scanning in 25 adults with hyperactivity of childhood onset at the Section on Clinical Brain Imaging and Child Psychiatry Branch, National Institute of Mental Health, Bethesda, MD. Global

cerebral glucose metabolism was 8.1% lower in the adults with hyperactivity than in normal controls. The largest reductions in glucose metabolism were in the premotor cortex and the superior prefrontal cortex, areas of the brain shown to be involved in the control of attention and motor activity. No significant differences were found in global cerebral cortex metabolism between patients with hyperactivity who had current learning deficits and those who did not. (Zametkin AJ et al. Cerebral glucose metabolism in adults with hyperactivity of childhood onset. N Engl J Med Nov 15, 1990; 323:1361-6).

COMMENT. The frontal lobes are important in maintaining attention, and disorders of the prefrontal regions may result in inattentiveness, distractibility, and an inability to inhibit inappropriate responses, such as motor restlessness, calling out in class, verbal interruptions, and acting before thinking.

Experimental neuroanatomical studies of hyperkinesia have been concerned with the effects of destruction of different cortical and subcortical structures on locomotor activity. (Millichap JG. Internal J Neurology 1975; 10:241-251). Bilateral removal of the prefrontal and frontal areas in the monkey causes the greatest total increase in activity. (Kennard MA et al. J Neurophysiol 1941; 4:512). Lesions in Walker's area 13 of the orbital surface produce the most extreme degree of hypermobility. Hyperactivity induced by parietal lobe lesions is not as marked as in frontal lobe lesions. Destruction of subcortical structures including the striatum, interpeduncular nucleus, and parts of the hypothalamus may also induce hyperactivity. Diffuse brain lesions have been thought to be a major cause of a large percentage of clinical cases of hyperactive behavior. The correlation between functional and anatomical development and pathology is still unclear. Monaminergic transmitters such as norepinephrine are in high concentration in the frontal lobes and increase as the child grows older. (Njiokiktjien C. Pediatric Behavioural Neurology Suyi Publications, Amsterdam 1988). The hypometabolism in prefrontal areas noted in the above study might possibly extend to monaminergic metabolism and may explain the beneficial effects of methylphenidate which increases the neurotransmitters and activity of cortical inhibitory systems in hyperactive children. The efficacy of methylphenidate in hyperactive children has been related to the level of motor activity before treatment and the incidence of abnormal neurological signs and evidence of brain dysfunction. (Millichap 1975). It is apparent from these studies that ADHD must be distinguished diagnostically from behavioral disorders that may appear similar but are reactions to environmental crises or inappropriate school placement. (Weiss G. Hyperactivity in childhood. Editorial. N Engl J Med Nov 15, 1990; 323:1413-1415).