

vocabulary 7, arithmetic 8) and 116 performance scale.

The CT scan showed an atrophic right hemisphere and dilated lateral ventricle with cortical and subcortical low densities involving the base of the 3rd frontal, supramarginal, insular and middle part of 1st temporal convolutions, the lectular and caudate nuclei and the anterior limb of the internal capsule. (Assal G. Aphasie croisee chez un enfant. Rev Neurol (Paris) 1987;143:532-535).

COMMENT: Crossed aphasia is the combination of right hemiparesis with aphasia in a left-handed patient or left hemiparesis and aphasia in a right-handed patient. It is rare in dextrals, only 9 cases cited in a review article by Brown JW and Hecaen H (Neurology 1976;26:183). Diagnosis requires the following: a pathologic lesion limited to the right hemisphere, absence of early childhood brain damage, strong right-handedness, and a negative family history of left-handedness. These criteria were satisfied in the author's case. A state of incomplete left lateralization is suggested to explain crossed aphasia in a right-handed patient. Although recovery of fluency is quicker and more extensive than in adults, later academic problems are common in children with aphasia even with those caused by left hemisphere lesions. (Cranberg LD et al. Neurology 1987;37:1165).

ATTENTION DEFICIT DISORDER

Progress over the past 50 years in our understanding of the neurobiology of attention deficit disorder with hyperactivity is reviewed by child psychiatrists at the National Institute of Mental Health, Bethesda, MD. Since Bradley first described the paradoxical calming effect of the stimulant benzedrine on hyperactive children (Amer J Psychiat 1937;94:577), more than 20 neuropharmacological agents have been used for the study and treatment of children with attention deficit disorder with hyperactivity (ADHD). Biochemical, pharmacological, and anatomical hypotheses are analyzed and may be summarized as follows: (1) stimulants are the treatment of choice and all beneficial drugs have effects on catecholamine metabolism; (2) alteration in noradrenergic function appears necessary for clinical efficacy; (3) a role for norepinephrine but not serotonin metabolism in the pathophysiology is likely; (4) support for a frontal lobe anatomical location of CNS dysfunction for ADHD seems more conclusive than hypothalamic dysfunction; (5) different sites of dysfunction in the cortical-striatal "circuit" might account for the varying symptoms of the ADHD syndrome. (Zametkin AJ, Rapoport JL. Neurobiology of attention deficit disorder with hyperactivity: Where have we come in 50 years? J Amer Acad Child and Adolesc Psychiat 1987;26:676-686).

COMMENT: The possible importance of brain injury or other neuropathological lesions in the pathogenesis of hyperkinetic behavior is often discounted in favor of environmental factors, and the authors emphasis on the neuroanatomical hypothesis and especially frontal lobe dysfunction in ADHD is refreshing. Some of the earlier experimental neuroanatomical studies of hyperkinesia have been concerned with the effects of ablation or destruction of different cortical and subcortical structures on locomotor activity. Bilateral removal of the prefrontal and frontal areas in the monkey and smaller

animals causes the greatest total increase in activity (Kennard MA et al. *J Neurophysiol* 1941:4:512. Millichap JG et al. *Excerpta Medica* 1974:130-139). We found an increase in locomotor activity of mice with prefrontal cortical lesions and those animals with the highest level of post-operative activity responded to methylphenidate with a reduction in locomotor activity. We suggested that animals with prefrontal cortical lesions should make valuable experimental models for testing new drugs. The beneficial effect of methylphenidate on hyperactivity in our patients was related to the level of motor activity before treatment and the degree of neurologic abnormality. ADDH patients with the greatest number of neurologic signs were most active and were most likely to benefit from stimulant therapy. (*N.Y. Acad Sci* 1973:205:321). A neuroanatomical basis for ADDH in some children might be substantiated by the MRI.

METHYLPHENIDATE AND ATTENTION DEFICIT DISORDER

The relative effects of sustained release (Ritalin [SR-207]) and standard methylphenidate (Ritalin 10 mg, BD) on cognitive and social behavior in 22 boys with ADD were investigated at a summer treatment program supervised by the Western Psychiatric Institute, Univ of Pittsburgh School of Medicine, PA. Group analyses of data showed that both drugs were effective but standard methylphenidate was superior to SR-20 on measures of disruptive behavior and SR-20 had a slower onset on a continuous performance task. Analyses of individual responsivity showed that most boys responded more positively to the standard compared to the sustained-release preparation of methylphenidate. The authors note that in contrast to advertising material, the effects of SR-20 and standard methylphenidate are not equivalent. They recommend pemoline or slow-release dextroamphetamine in preference to SR-20 if a single daily dose sustained effect is required. (Pelham WE Jr et al. Sustained release and standard methylphenidate effects on cognitive and social behavior in children with attention deficit disorder. *Pediatrics* 1987;80:491-501).

COMMENT: Stimulant medications used in the treatment of ADDH have different half lives: methylphenidate 2.6 hours on a dose of 0.60 mg/kg; dextroamphetamine 6.8 hours with 0.45 mg/kg; and pemoline 8.36 hours after doses up to 110 mg (see Zametkin AJ, Rapoport JL in *Ped Neur Briefs* 1987:1(5):37). Clinical responses, however, are not always correlated with the half life of the drug or with plasma levels which may vary considerably from day to day in patients with a fixed dose. Previous studies have shown that time release Dexedrine may not act for longer time periods than the standard tablet form. These inconsistencies, together with the findings in the present study, suggest that standard methylphenidate or pemoline remain the treatments of choice in ADDH.