and the second to survive. Only one previous fetus has survived a pregnancy complicated by HSE and the mother, treated with idoxuridine, died 5 days postpartum. Acyclovir reduces mortality of neonatal HSE and is well tolerated but morbidity in surviving infants is high especially if treatment is delayed. Early diagnosis is facilitated by MRI. (Scroth G et al. Neurology 1987:37:179). For a review of the natural history of HSV infection of mother and newborn and therapy of neonatal HSV infection, see Whitley RJ et al. and Infectious Disease Collaboration Antiviral Study Group. Pediatrics 1980:66:489-501.

HERPES ZOSTER OPHTHALMICUS

A 17-month-old boy with HZO and delayed contralateral hemiparesis following intrauterine varicella exposure is reported from the Dept of Neurology, Univ Texas Med Sch, Houston, TX. He presented with ataxia and a progressive right-sided weakness. His mother had chicken-pox at 8 months of gestation but he appeared normal at birth. A vesicular rash developed 4 weeks before examination in the distribution of the ophthalmic and mandibular divisions of the left trigeminal nerve. CSF showed mononuclear pleocytosis, CT demonstrated multiple areas of hypodensity in the left basal ganglia, and angiography revealed occlusion of the left lenticulostriate arteries. Treatment with Acyclovir for 10 days was followed by recovery except for minimal right hemiparesis. (Leis AA, Butler IJ. Infantile herpes zoster ophthalmicus and acute hemiparesis following intrauterine chickenpox. Neurology 1987:37:1537-1538). Passive immunization of susceptible women exposed to varicella is recommended to reduce the risks of maternal and fetal varicella. The determination of varicella zoster virus membrane antigen or equivalent anti-varicella antibody status in pregnant women exposed to varicella is a rapid, satisfactory method for determining who should receive varicella immunoglobulin passive immunization (McGregor JA et al. Am J Obstet Gynecol 1987:157:281).

COMMENT: The authors cited only one similar previous case in a child, a 7-year-old boy. Delayed focal cerebral angiitis and infarction may occur after an interval of days to months between HZO and neurologic complications in adults. Passive immunization of exposed susceptible women reduces risks of maternal and fetal varicella. (McGregor JA et al. Am J Obstet Gynec 1987:157:281).

LANGUAGE AND BEHAVIOR

CROSSED APHASIA

A case of crossed aphasia with persistent language disturbances in a right-handed boy aged 5 yr 9 mos is reported from the Centre Hospitalier Universitaire Vaudois, Lausanne, France. An acute left hemiplegia resulted from occlusion of the internal carotid siphon of undermined cause and demonstrated by arteriography. The boy was mute, his auditory comprehension impaired, and tongue and facial movements apraxic. His first intelligible words (maman and non) were pronounced at 2 months after the onset. The language remained agrammatic and the vocabulary and comprehension poor but the tongue apraxia resolved. Twelve years later, language disturbances were still present although his IQ on the WAIS was 100 full scale, 86 verbal (information 6, comprehension 6, digit memory 5,

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 lecticular 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 (ADDH). 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 ADDH seems more conclusive than hypothalmic dysfunction; (5) different sites of dysfunction in the cortical-striatal "circuit" might account for the varying symptoms of the ADDH 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 ADDH 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