COCAINE AND HEROIN IN UTERO EFFECTS

A study of 86 infants who were born to women with a history of cocaine and/or heroin use during pregnancy is reported from the Department of Pediatrics, Highland General Hospital, and the Division of Neonatology, Children's Hospital, Oakland, CA. The newborns were observed over a five day hospital period using a standardized abstinence scoring system and urine drug screening of both mother and infant. Urine tests were positive for cocaine only in 35, heroin only in 14, cocaine and heroin in 17, and 20 were negative. In the cocaine group 17% of the newborns had growth retardation and 27% were microcephalic. Microcephaly occurred in 17% of 12 infants in the heroin group, 20% of the 15 infants in the cocaine/ heroin group, and in none of 20 infants in the urine negative cocaine history group. Of 985 newborns in the no drug group, 4% were microcephalic. The incidence of microcephaly was significantly higher in the cocaine group and in the cocaine/heroin group than in the no drug group. Cocaine and heroin were synergistic in causing abnormal behavior of withdrawal as assessed by the Finnegan scoring system which includes tremulousness, tachypnea, decreased sleep and feeding hypertonia. disturbances. (Fulroth R. et al. Perinatal outcome of infants exposed to cocaine and/or heroin in utero. AJDC August 1989; 143:905-910).

<u>COMMENT.</u> The authors conclude that infants exposed to cocaine and/or heroin in utero should be followed up closely after discharge from the nursery since growth retardation, microcephaly, and abnormal behavior may be suggestive of potential long-term neurologic or developmental problems. The cocaine induced microcephaly might be explained by impaired maternal nutrition, vasoconstriction in the placenta, and reduced fetal blood flow and fetal hypoxia. In this study 85% of mothers using cocaine reported the abuse of free base (crack) cocaine which causes significantly more vasoconstriction than that taken intranasally. None of the infants studied had the features of fetal alcohol syndrome.

METABOLIC DISORDERS

NIEMANN-PICK DISEASE TYPE C

The neurologic symptomatology in 22 patients with Niemann-Pick disease type C have been analyzed and reported from the Developmental and Metabolic Neurology Branch, National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, MD. Three phenotypes are described: 1) an early onset, rapidly progressive form associated with severe hepatic dysfunction and psychomotor delay during infancy and later with supranuclear vertical gaze paresis, ataxia, spasticity, and dementia; 2) a delayed onset, slowly progressive form beginning in early childhood with mild intellectual impairment, supranuclear vertical gaze paresis and ataxia, and later associated with dementia, seizures and extrapyramidal deficits; 3) a late onset slowly progressive form beginning in adolescence or adulthood. The classic supranuclear disorder of gaze, initially and predominantly affecting vertical eye movements, is nearly pathognomonic for NPC. The biochemical disorder is a marked deficiency in