very low birth weight and normal birth weight children. The AP-BP ratio was unrelated to IQ (Kitchen WH et al. Very low birth weight and growth to age 8 years II: head dimensions and intelligence. AJDC Jan 1992; 146:46-50.) (Reprints not available.)

COMMENT. The authors concluded that the National Center for Health Statistics data for occipitofrontal circumference measurements were more appropriate than the Nellhaus data in this study. Head growth achieved by age 8 years was progressively reduced in lower birth weight categories in all measurements except the anteroposterior diameter. Occipitofrontal circumference was closely related to IQ and other head measurements are not recommended in routine clinical practice. In a further report the authors found that maternal height and the birth weight ratio were more important than health after birth in predicting a height or weight below the 10th percentile at age 8 years in children with low birth weights. Occipitofrontal head circumference is related to infant development and later intelligence in childhood and is a useful indication of brain size.

CEREBRAL PALSY

ANTEPARTUM CAUSES OF CEREBRAL PALSY

Six neonates with destructive brain lesions of fetal onset, diagnosed by radiological and neurophysiological studies, are reported from the Departments of Pediatrics, Neurology, Radiology, and Obstetrics and Gynecology, Magee-Womens Hospital, Pittsburgh, PA and Children's Hospital of Pittsburgh. Initial diagnosis of cerebral lesions was made by fetal sonography in 2 patients and CT scan in 4 during the first 30 hours of life. No intrapartum difficulties were noted and 2 patients had definitive evidence of maternal and placental disease that preceded the onset of active labor. The neurological examinations were normal at birth, but 4 presented with isolated seizures at 8-30 hours of life and initial neonatal EEGs showed abnormalities. The authors recommend that cranial imaging and neurophysiological studies should be used during the first days of life for neonates believed to have cerebral lesions based on maternal sonography or isolated seizures. (Scher MS et al. Destructive brain lesions of presumed fetal onset: antepartum causes of cerebral palsy. Pediatrics Nov 1991; 88:898-906.) (Reprints: Dr. Scher, Develop Neurophysiol, Magee-Womens Hospital, Forbes St., Pittsburgh, PA 15213.)

COMMENT. Cranial ultrasonography and CT studies during the first few days of life may document lesions occurring prenatally. Children with antepartal brain injury may be asymptomatic or exhibit few clinical signs during the neonatal period and may later develop cerebral palsy.

MRI IN ATHETOTIC CEREBRAL PALSY

The MRI in 22 children with athetotic cerebral palsy was studied in the Department of Pediatric Neurology, Seirei-Mikatabara General Hospital,

Hamamatsu, the Department of Pediatrics, National Rehabilitation Center for Disabled Children, Tokyo, and the Department of Pediatrics, Nagoya City University Medical School, Japan. Perinatal asphyxia had been diagnosed in 16, neonatal jaundice in 2, and no predisposing condition in 4. MRI lesions possibly caused by asphyxia were found in the basal ganglia, thalamus, and/or cerebral white matter in 14 of 16 children. No abnormalities were seen in the cerebral cortex of the subjects. Six children with lesions in both the thalamus and putamen had moderate or severe motor abnormality. Children with lesions in the cerebral white matter only had mild motor abnormality. (Yokochi K et al. Magnetic resonance imaging in athetotic cerebral palsied children. Acta Paediatr Scand Sept 1991; 80:818-823.) (Corresp: Dr. Yokochi, Seirei-Mikatabara Gen. Hosp., Mikatabara 3453, Hamamatsu, Shizuoka 433, Japan.)

COMMENT. Brain lesions causing athetosis in CP children may not be revealed by CT scan but high intensity areas in the thalamus, putamen and/or cerebral white matter may be demonstrated in T2-weighted images of the MRI. The evidence of perinatal asphyxia in 16 of the 22 children reported was judged by Apgar scores of 3 and below, generalized hypotonia, and convulsions in the neonatal period. The MR imaging was performed at 2-12 years of age and studies obtained soon after birth might have documented a prenatal timing for the lesions.

NEONATAL CSF CREATINE KINASE AND CP RISK

Creatine kinase brain isoenzyme (CK-BB) was examined in the cerebrospinal fluid of 150 neonates at the Departments of Pediatrics and Neonatal Medicine and Clinical Chemistry, University Hospital, Ghent, Belgium. Indications for lumbar puncture were intraventricular hemorrhage, subarachnoid hemorrhage, postasphyxial encephalopathy, convulsions, birth trauma, periventricular echodensities, or question of sepsis. Newborns with a neurologic disorder showed significantly higher concentrations of immunoreactive CK-BB than did normal newborns or those with subarachnoid hemorrhage. (The median CK-BB for 5 neonates with intraventricular hemorrhage and echodensities was 106 mcg/l and the normal control was 2.14.) An independent effect of seizures on the CK-BB concentration was not documented. High concentrations of CSF CK-BB in the neonatal period were correlated with an adverse outcome - that is death or abnormal neurologic exam at discharge. (De Praeter C et al. Creatine kinase isoenzyme BB concentrations in the cerebrospinal fluid of newborns: relationship to short-term outcome. Pediatrics Dec 1991; 88:1204-1210.) (Reprints: Dr. De Praeter, Dept. of Pediatrics, University Hospital, Ghent, De Pintelaan, 185, B-9000, Ghent, Belgium.)

COMMENT. Determination of the CK-BB concentration in CSF appears to be a clinically useful test for the prediction of neurologic disorders in high risk neonates. Infants with high CSF CK-BB concentrations should receive careful follow-up. In a previous study (Fernandez F et al. Acta Paediatr Scand 1987; 76:914) an elevated serum CPK measured within 4 hours after birth was a sensitive indicator of brain damage in asphyxiated term infants but was of limited