BRAIN MRI ANATOMICAL AND ATTENTION AND BEHAVIOR DISORDERS WITH 22q11.2 DELETION SYNDROME

The brain anatomy of 39 children and adolescents with 22qDS (mean age 11 years; IQ 67) and 26 sibling controls (mean age 11 years; IQ 102) was compared using MRI and automated voxel-based morphometry, and behavioral differences were correlated with affected brain regions in a study at King's College, Institute of Psychiatry, London, UK; Royal College of Surgeons, Dublin, Ireland; and Academic Medical Center, Amsterdam, Holland. Individuals with 22qDS had a significant reduction in cerebellar grey matter, and white matter reductions in the frontal lobe, cerebellum and internal capsule; the volume of the occipital-parietal lobes was significantly reduced bilaterally; the right caudate nucleus and lateral ventricles were enlarged; and the prevalence of midline brain anomalies, such as cavum septum pellucidum, was increased. Significant positive correlations were found in 22qDS subjects between severity of (1) schizotypy symptoms and grey matter volume of temporo-occipital regions and the corpus striatum; and (2) emotional and (3) social behavioral problems and grey matter volume of frontostriatal regions. (Campbell LE, Daly E, Toal F, et al. Brain and behaviour in children with 22q11.2 deletion syndrome: a volumetric and voxel-based morphometry MRI study. **Brain** May 2006;129:1218-1228). (Respond: Linda Campbell, Centre for Mental Health Studies, James Fletcher Hospital, The University of Newcastle, Newcastle, NSW 2300, Australia).

COMMENT. Subjects with 22q deletion syndrome have changes in brain anatomy, especially white matter, basal ganglia and cerebellum, that correlate with behavioral disorders. Children with 22qDS have a higher prevalence of schizotypal traits, emotional symptoms, ADHD and social behavioral disorders. Frontostriatal regions found abnormal in 22qDS are implicated in attention and executive functions.

PERINATAL DISORDERS

ETIOLOGY OF SPASTIC DIPLEGIA

The clinical and etiologic profile of spastic diplegia was studied in a retrospective chart review of 54 patients diagnosed during a 12-year period at Montreal Children's Hospital, Quebec, Canada. Thirty-one (57.4%) were preterm children, and 23 (42.6%) term children. Initial concerns were gait abnormality in 18 (33.3%), global developmental delay in 7 (13%), motor delay in 6 (11.1%), seizure in 3 (5.6%), and hypotonia in 2 (3.7%). ADHD was an associated comorbidity in 7 (13%). The diagnosis was periventricular leukomalacia (PVL) in 24 (44.4%) children; 26.1% were term and 58.1% preterm. The etiology was undetermined in 25 (46.3%) children. Ischemic stroke occurred in 3 (5.6%). PVL correlated with a birth weight of less than 2000 gm, history of neonatal resuscitation, and gestation less than 33 weeks. In term children, PVL was associated with perinatal difficulties, neonatal resuscitation, and a history of neonatal distress. (Tang-Wai R, Webster RI, Shevell MI. A clinical and etiologic profile of spastic diplegia. **Pediatr Neurol** March 2006;34:212-218).

(Respond: Dr Shevell, Room A-514, Montreal Children's Hoispital, 2300 Tupper Street, Montreal, Quebec H3H IP3, Canada).

COMMENT. The authors list further investigations to identify the cause of spastic diplegia in the high proportion (46%) of patients undiagnosed. These investigations include repeat MRI, spinal imaging, voxel-based morphometry, and diffusion-weighted MRI in the acute stage of PVL.

THIRD VENTRICLE ENLARGEMENT IN NEONATES WITH TRISOMY 21

Measurements of routine head sonographic scans of 57 term infants with trisomy 21 born between 2000 and 2005 were performed within 7 days after birth and were compared with scans of 21 randomly selected, healthy, term infants without trisomy 21 at Shaare Zedek Medical Center, Jerusalem; Ben Gurion University of the Negev, Beer Sheva; and Hadassah University Hospital, Jerusalem, Israel. The test and control neonates were the same gestational ages (39+/-1 weeks), but trisomy 21 infants were smaller and had smaller head circumferences than controls (32.9 cm vs 34.9 cm; P=0.001). The width and length of the third ventricle were increased in infants with trisomy 21. Vertical measurements of the lateral ventricle were similar for the 2 groups. (Schimmel MS, Hammerman C, Bromiker R, Berger I. Third ventricle enlargement among newborn infants with trisomy 21. **Pediatrics** May 2006;117:928-931). (Respond: Michael S Schimmel MD, Department of Neonatology, Shaare Zedek Medical Center, PO Box 3235, Jerusalem 91031, Israel).

COMMENT. The authors speculate that the enlargement of the third ventricle demonstrated in neonates with trisomy 21 may reflect hypoplasia of the thalamus, hypothalamus, and deep white matter, which are involved in cognitive processes of attention, verbal and visuospatial memory. Prefrontal, cerebellar, and hippocampal functions are also affected in trisomy 21.

SERUM BILIRUBIN LEVELS AND DEVELOPMENTAL OUTCOME

The neurodevlopmental risks associated with neonatal total serum bilirubin levels of 25 mg/dL or higher in 140 affected infants were compared with 419 randomly selected controls from a cohort of term-infants born 1995-1998 in Kaiser Permanente hospitals in northern California. Peak bilirubin levels were between 25 and 29.9 mg/dL in 130 newborns with hyperbilirubinemia and 30 mg/dL or higher in 10 newborns. Phototherapy was used in 136 cases and exchange transfusion in 5. There were no cases of kernicterus. In subjects followed for at least 2 years, scores on cognition tests were similar in the hyperbilirubinemia and control groups. Questionable or abnormal neurologic findings were present in 14 children (17%) with hyperbilirubinemia vs 48 of controls (29%); P=0.05. Reported behavioral problems were not significantly different in the 2 groups. Those with positive direct antiglobulin tests for immune-mediated hemolytic disease in the hyperbilirubinemia group had lower scores on cognition tests but not more neurologic or behavioral problems. (Newman TB, Liljestrand P, Jeremy RJ, et al. Outcomes among newborns with total serum