CONGENITAL CNS MALFORMATIONS

FETAL MRI IN PRENATAL DIAGNOSIS OF CNS ABNORMALITIES

The value of fetal MRI (fMRI) compared to ultrasound in the prenatal detection of CNS abnormalities and impact on counseling were determined in 25 pregnant women examined at University of Dusseldorf, Germany. Examination time was 27 to 51 minutes (41.8+/-6.1 min). Results were correlated with postnatal MRI, ultrasound and clinical follow-up. fMRI was performed 3-10 days after ultrasound between gestational week 22 and 34 (GW 26.1+/-3.6). Abnormalities suspected on fetal ultrasound were confirmed by fMRI in 8 cases. These included 2 cases of aqueductal stenosis hydrocephalus, and 1 each of hemimegalencephaly, microlissencephaly, ventriculomegaly, schizencephaly, brain tumor, and corpus callosum agenesis. Additional diagnoses or exclusions of suspected findings were established in 13 cases. The exclusions were corpus callosum agenesis in 4 cases and myelomeningocele, vermial aplasia, aqueductal stenosis, and Dandy-Walker malformation in 1 case each. Diagnoses were completely revised by fMRI in 4 cases. Postnatal MRI confirmed the fMRI findings in 11 patients. The quality of fMRI is technically comparable to postnatal MRI, and surgical treatment options are better defined than with ultrasound alone. (Messing-Junger AM, Rohrig A, Stressig R, Schaper J, Turowski B, Blondin D. Fetal MRI of the central nervous system: clinical relevance. **Childs Nerv Syst** February 2009;25:165-171). (Respond: AM Messing-Junger. E-mail: m.messing@Asklepios.com).

COMMENT. fMRI is superior to fetal ultrasound in detection of congenital CNS abnormalities. In institutions with trained professionals, fMRI is recommended in addition to ultrasound in patients with suspected pathologies that may require surgical interventions and parent counseling.

FUNCTIONAL MRI OF SENSORIMOTOR CORTEX IN PRETERM INFANTS

Functional MRI (fMRI) findings in a group of 5 pre-term infants were correlated with a unilateral passive forearm extension/flexion to relate the functional data to structural and behavioral data, in studies at the University of Bonn, Germany; and University Medical Center, Groningen, Netherlands. Measurement of blood oxygen level-dependent (BOLD) responses in the sensorimotor cortex showed bilateral activation during unilateral passive sensorimotor stimulation. The prevailing hemodynamic response was a negative blood oxygenation level-dependent signal. Positive blood oxygenation level-dependent response or failure to activate the sensorimotor cortex was found in patients with abnormal brain structural and behavioral problems. (Heep A, Scheef L, Jankowski J, et al. Functional magnetic resonance imaging of the sensorimotor system in preterm infants. **Pediatrics** January 2009;123:294-300). (Respond: Axel Heep MD. E-mail: axel.heep@ukb.uni-bonn.de).

COMMENT. The authors propose that their fMRI findings are compatible with a bilaterally distributed sensorimotor system in the preterm infant. The reductions of oxy/deoxy-Hb ratio in activated brain tissue may reflect ineffective neural processing during this maturational stage of rapid synapse formation. Positive blood oxygenation level-dependent responses or failure to activate the sensorimotor cortex in a preterm infant may predict abnormal cerebral development and need for careful follow-up. fMRI should provide a more effective measure of long-term developmental problems than the neonatal neurological exam.

CONGENITAL HYDROCEPHALUS RISK FACTORS

Risk factors associated with the pathogenesis of congenital hydrocephalus were evaluated in a 10 year retrospective study of 596 cases identified at the University of Mississippi Medical Center between 1998 and 2007. Significant risk factors included lack of prenatal care, multiparous gestation, maternal diabetes, maternal chronic hypertension, pregnancy-induced hypertension, and alcohol use during pregnancy. Hydrocephalus was familial in 12% cases. Except for an increased incidence of multiparous pregnancies and prenatal care in the first trimester in familial cases, no differences in risk factors were identified between sporadic and familial congenital hydrocephalus. The prevalence of familial cases within this cohort is much higher than that reported in X linked congenital hydrocephalus (2-7%), and suggests that the strong genetic factor in etiology is attributed to non-X linked patterns of inheritance. (Landingham MV, Nguyen TV, Roberts A, Parent AD, Zhang J. Risk factors of congenital hydrocephalus: a 10 year retrospective study. **J Neurol Neurosurg Psychiatry** February 2009;80:213-217). (Respond: Dr J Zhang, Department of Neurosurgery, University of Mississippi Medical Center, 2500 N State St, Jackson, MS 39216. E-mail: jhzhang@neurosurgery.umsmed.edu).

COMMENT. Both genetic and environmental factors are involved in the pathogenesis of congenital hydrocephalus. Some risk factors identified in this study should be susceptible to preventive measures, including improved prenatal care and nutrition, avoidance of alcohol, and prompt treatment of hypertension.

MODERATE PREMATURITY AND RISKS FOR CEREBRAL PALSY

The association between moderate prematurity and the incidence of adverse neurodevelopmental outcomes was assessed in a cohort of infants born in the Kaiser Permanente Medical Care Program of Northern California. Data covered 141,321 children born at >30 weeks gestation between Jan 1, 2000 and June 30, 2004, followed through Jan 30, 2005. Decreasing gestational age was associated with increased incidence of cerebral palsy (CP) and developmental delay (DD), even in those born at 34 to 36 weeks gestation. Late preterm infants were >3 times as likely to have CP as term infants. Children born at 34 to 36 weeks were marginally at higher risk of DD and mental retardation but not seizures. (Petrini JR, Dias T, McCormick MC, Massolo ML, Green NS, Escobar GJ. Increased risk of adverse neurological development for late preterm infants. **J Pediatr** February 2009;154:169-176). (Reprints: Joann R Petrini PhD MPH, March of Dimes National Office, 1275 Mamaroneck Ave, White Plains, NY 10605. E-mail: JPetrini@marchofdimes.com).