increased insomnia and decreased appetite. (Stein MA et al. Pediatrics Dec 2003;112:1173-4). Biederman J reports that 54 mg of OROS-MPH (Concerta) is equivalent to 20 mg Adderall XR (Today's Therapeutic Trends 2002;20:311-328).

# NEONATAL DISORDERS

## CHORIOAMNIONITIS: A RISK FACTOR FOR CEREBRAL PALSY

The association between clinical chorioamnionitis and increased risk of cerebral palsy (CP) in term and near-term infants was determined in 109 children with CP not due to postnatal brain injury or developmental abnormalities compared to 218 controls, in a study at University of California, San Francisco, and Kaiser Permanente Division of Research, Oakland, CA. CP was a hemiparesis in 40% and quadriparesis in 38%. Neuroimaging had been obtained in 83%; focal infarct, white matter abnormalities, hypoxic-ischemic injury, and atrophy were the most common findings. Chorioamnionitis or endometritis had been diagnosed clinically in 14% of cases compared to 4% of controls (OR 3.8, CI 1.5-10.1; p=0.001). Independent risk factors for CP in addition to chorioamnionitis included maternal fever, prolonged rupture of membranes, intrauterine growth restriction, maternal black ethnicity, maternal age older than 25 years, and nulliparity. Other factors strongly associated with CP were a 5-minute Apgar score <7, birth asphyxia, and neonatal seizures. The population-attributable fraction of chorioamnionitis for CP is 11%, and for spastic quadriplegic CP, 27%. (Wu YW, Escobar GJ, Grether JK, et al. Chorioamnionitis and cerebral palsy in term and near-term infants. JAMA November 26, 2003;290:2677-2684). (Reprints: Yvonne W Wu MD, MPH, Division of Child Neurology, Box 0136, University of California, San Francisco, 500 Parnassus Ave, MUE #411, San Francisco, CA 94143).

COMMENT. Clinical chorioamnionitis is independently associated with a 4-fold increased risk of CP in term infants. Chorioamnionitis may initiate or exacerbate brain injury from hypoxia-ischemia by leading to an elevation of inflammatory cytokines in the fetus. Prevention of perinatal inflammatory disorders may lower the incidence of CP in term infants.

## VEIN OF GALEN MALFORMATION: OUTCOME AFTER EMBOLIZATION

The neurodevelopmental outcome after endovascular treatment of vein of Galen malformation (VOGM) in 27 patients seen between 1983 and 2002 was assessed by chart review and parental questionnaires at the University of California, San Francisco. The presentation with congestive heart failure (CHF; 16/27) or hydrocephalus (8/27) was prenatal (diagnosed by ultrasound) in 5, neonatal in 16, and post-neonatal in 6. Patients with CHF presented either prenatally or neonatally, 4 died acutely, 6 were significantly delayed, and 6 had no or minor developmental delay. Of 5 presenting perinatally without CHF, all survived, 2 were significantly delayed, and 3 had no delay. Of 6 presenting after the neonatal period, all survived and only 1 was delayed. Those with choroidal VOGM by

angiography had a worse prognosis (3/13 died; 5/13 delayed) than those with mural VOGM (2/10 had significant delay; none died). Of all cases in the series, 52% (61% of survivors) had no or minor delay. The features associated with the worst outcome were perinatal presentation, congestive heart failure, and choroidal angioarchitecture. (Fullerton HJ, Aminoff AR, Ferriero DM, Gupta N, Dowd CF. Neurodevelopmental outcome after endovascular treatment of vein of Galen malformations. Neurology November (2 of 2) 2003;61:1386-1390). (Reprints: Dr HJ Fullerton, Department of Neurology, University of California, San Francisco, Box 0114, San Francisco, CA 94143).

COMMENT. The majority of infants with vein of Galen malformation who are selected for endovascular treatment have a favorable neurodevelopmental outcome. Neonatal presentation, congestive heart failure, and choroidal as opposed to mural angioarchitecture are features carrying the worst prognosis. The overall mortality is 15%. The majority of survivors have a good outcome with 52% having no delay and an additional 9% having only mild delay in neurodevelopment.

## INFANTILE NEUROPATHY WITH DIAPHRAGMATIC PALSY

`A group of 13 patients with early onset diaphragmatic palsy in association with a progressive neuropathy is presented from Great Ormond Street Hospital for Children, London, UK. Weakness and wasting developed over a period of weeks and showed a distal to proximal progression. The patients shared similar characteristics and diagnostic criteria that included early onset respiratory distress, low birth weight, slow motor nerve conduction velocities, and decrease in size of myelinated fibers on sural nerve biopsy. Mutations in 8 cases tested affected the same gene encoding immunoglobulin mu-binding protein 2 in patients with spinal muscular atrophy with respiratory distress type 1 (SMARD1). Histological examination of the spinal cord in one patient showed no evidence of SMA. Genetic and clinical heterogeneity is suggested. (Pitt M, Houlden H, Jacobs J, et al. Severe infantile neuropathy with diaphragmatic weakness and its relationship to SMARD1. Brain December 2003;126:2682-2692). (Respond: Dr Matthew Pitt, Department of Clinical Neurophysiology, Great Ormond Street Hospital for Children NHS Trust, Great Ormond Street, London WC1N 3JH, UK).

COMMENT. Infants and neonates presenting with severe respiratory distress and diaphragmatic palsy may have diaphragmatic SMA (SMARD1) or severe infantile axonal neuropathy with respiratory failure (SIANRF). The syndrome is clinically and genetically heterogeneous, some cases having mutations in the IGHMBP2 gene.

**Clinical features of 29 infants with SMARD1 and 26 novel IGHMBP2 mutations** are reported from Charite, Humboldt University, Berlin (Grohmann K et al. Ann Neurol 2003;54:719-724). Itrauterine growth retardation, weak cry, and foot deformities were the earliest manifestations. Patients presented at 1 to 6 months with respiratory distress due to diaphragmatic paralysis and progressive muscle weakness, predominantly distal lower limbs. Sensory and autonomic nerves are also affected. Diagnosis of SMARD1 is considered in infants with non-5q SMA, neuropathy, and muscle weakness and/or