to chromosome 15q14, similar to that involved in families with juvenile myoclonic epilepsy. An autosomal recessive mode of inheritance with heterogeneity was suggested. (Neubauer BA, Fiedler B, Himmelhein B, et al. Centrotemporal spikes in families with rolandic epilepsy. Linkage to chromosome 15q14. Neurology Dec 1998,51:1608-1612). (Reprints: Dr Bernd A Neubauer, Dept of Neuropediatrics, University of Kiel, Schwanenweg 20, 24105 Kiel, Germany).

COMMENT. Both benign rolandic epilepsy, a common partial, idiopathic epilepsy syndrome, and juvenile myoclonic epilepsy, a generalized idiopathic syndrome, have been linked genetically to chromosome 15q14.

NEONATAL DISORDERS

OUTCOME OF NEONATAL CEREBRAL INFARCTION

Antenatal and perinatal factors, early clinical signs, electroencephalograms (EEG), and magnetic resonance imaging (MRI) findings were compared with neurodevelopmental outcome in 24 infants with neonatal cerebral infarction followed at the Dept of Paediatrics, Hammersmith Hospital, London, UK. Infarcts defined by MRI involved a major cerebral vessel in 19 and borderzones in 5. Duration of follow-up ranged from 15 months to 5 years. Of 7 (29%) infants with abnormal neuromotor outcome, 5 were hemiplegic and 2 showed asymmetry of tone or function. None developed seizures. Adverse antenatal factors, present in 11 (46%), perinatal continuous decelerations below 90 with slow recovery in 14, meconium staining in 11, cord blood pH<7.1 in 2, and Apgar <5/1min in 5 were not related to outcome. Abnormal signs on neonatal neurologic exam, chiefly hypotonia, were poor prognostic indicators. Both EEG and MRI were predictors of abnormal outcome. Abnormal neonatal EEG background was associated with later hemiplegia whereas epileptic discharges were not predictive. MRI showing involvement of hemispheres, basal ganglia, and internal capsule, but not one or two of these regions, tended to develop hemiplegia or asymmetry of tone. Concomitant thalamic involvement did not increase the risk of poor outcome. (Mercuri E, Rutherford M, Cowan F et al. Early prognostic indicators of outcome in infants with neonatal cerebral infarction: a clinical, electroencephalogram, and magnetic resonance imaging study. Pediatrics Jan 1999;103;39-46). (Reprints: Dr Eugenio Mercuri, Dept of Paediatrics, Hammersmith Hospital, Du Cane Rd, London W12 OHN, UK).

COMMENT. Neonatal EEG and MRI findings may be predictive of later outcome and development of hemiplegia in infants born with cerebral infarcts. Abnormal antenatal and perinatal factors and neonatal hypotonia fail to identify infants with a poor prognosis.

ANTE/INTRAPARTUM RISKS OF NEONATAL ENCEPHALOPATHY

The role of antepartum and intrapartum factors in the etiology of neonatal encephalopathy (NE) in 164 term infants was investigated in a Western Australian case-control study, with 400 randomly selected controls. The prevalence of NE was 3.8/1000 term live births, with a 9.1% case fatality. The features of NE included seizures, abnormal tone, apneas, feeding difficulties, abnormal consciousness, and ventilatory support. Independent risk factors before conception and in the antepartum period included lower socioeconomic status, family history of seizures or other neurologic disease, conception after infertility treatment, maternal thyroid disease, severe pre-eclampsia, bleeding during pregnancy, viral illness,

abnormal placenta, intrauterine growth retardation, and postmaturity.

Intrapartum risk factors included maternal pyrexia, persistent occipito-posterior position, and acute intrapartum events. Operative vaginal delivery and emergency cesarean section were risk factors whereas elective cesarean carried a reduced risk. Intrapartum hypoxia was absent in >70% of cases of NE.

Causes of newborn encephalopathy are heterogeneous and almost 70% have only antepartum risk factors; 24% have both antepartum and intrapartum factors; 5% have only intrapartum factors; and 2% have no recognizable risk factors. (Badawi N, Kurinczuk JJ, Keogh JM et al. Antepartum and intrapartum risk factors for newborn encephalopathy: the Western Australian case-control study. BMJ 5 Dec 1998;317:1549-53 and 1554-8). (Respond: Dr N Badawi, Dept of Neonatology, New Children's Hospital, Royal Alexandra Hospital for Children, PO Box 3515, Parramatta, New South Wales 2124, Australia).

COMMENT. The majority of causes of neonatal encephalopathy occur in the antepartum period and are not primarily birth-related. Elective ceasarean section is associated with a reduced risk of NE, presumably by avoidance of some intrapartum risk factors, including post-maturity, maternal pyrexia, bleeding, and acute labor events.

NEUROLOGIC COMPLICATIONS OF FETAL COCAINE EXPOSURE

The effects of prenatal cocaine exposure on intrauterine growth and neurologic function of 253 infants was evaluated prospectively at 1 to 7 days of age at the Division of Pediatric Neurology, College of Physicians and Surgeons, Columbia University, New York. Mothers with alcoholism, parenteral drug use, and AIDS, and infants with Apgars of <4 at 5 min, malformations, seizures, or stroke were excluded. Cocaine exposure was determined by radioimmunoassay of maternal hair collected in the last trimester. Of 240 woman and infant pairs with hair samples, 104 were cocaine-exposed and 136 unexposed. Cocaine-exposed infants had higher rates of intrauterine growth retardation (24% vs 8%), small head circumference <10th percentile (20% vs 5%), and neurologic abnormalities (hypertonia, tremor, and extensor leg posture). These abnormalities were dose-related, with increased odds of small head and neurologic impairment with increasing levels of cocaine exposure. (Chiriboga CA, Brust JCM, Bateman D, Hauser WA. Dose-response effect of fetal cocaine exposure on newborn neurologic function. Pediatrics Jan 1999;103:79-85). (Reprints: Dr Claudia A Chiriboga, Neurological Institute, 710 West 168th St, New York, NY 10032).

COMMENT. Fetal cocaine exposure has adverse neurologic effects that follow a dose-response relationship. Higher levels of prenatal cocaine exposure are associated with higher rates of reduced head growth, abnormal tone and posture, and tremor in the neonatal period.

Motor development of cocaine-exposed children at age two years was studied in 199 subjects (98 prenatal cocaine-exposed and 101 unexposed) at the Dept of Pediatrics, Case Western Reserve University, Cleveland, OH (Arendt R, Angelopoulos J, Salvator A, Singer L. Pediatrics Jan 1999;103:86-92). Scores on the Peabody Developmental Motor Scales showed that cocaine-exposed children performed significantly less well in gross and fine motor indices, with impaired balance and receipt and propulsion, poorer hand use and eye-hand coordination, and lower developmental motor quotients.