infants without hypertonia. (Chiriboga CA et al. Neurological correlates of fetal cocaine exposure: Transient hypertonia of infancy and early childhood. Pediatrics December 1995;96:1070-1077). (Reprints: Dr CC Chiriboga, Division of Pediatric Neurology, College of Physicians and Surgeons, Columbia University, 710 West 168th St, New York, NY 10032).

COMMENT. In our clinic for children with Attention Deficit Disorders at Children's Memorial Hospital, Chicago, I have observed an unusual incidence of a history of fetal cocaine exposure in those placed in foster homes soon after birth. Other complications of cocaine exposure in utero are small head circumference, cerebral infarction or hemorrhage, seizures, and SIDS. Disturbances in corticogenesis have been demonstrated in experiments on laboratory animals (see Progress in Pediatric Neurology I and II, PNB Publishers, 1991, pp452-3, and 1994, pp439-41).

GLUTEN SENSITIVITY AND NEUROLOGICAL ILLNESS

The frequency of IgG and IgA antigliadin antibodies, a measure of cryptic gluten sensitivity, and celiac disease was studied using ELISA in 147 adult patients admitted to the Royal Hallamshire Hospital, Sheffield, UK, for neurologic investigation. Of 53 patients with neurological dysfunction of unknown cause, including 25 with ataxia and 20 with peripheral neuropathy, 30 (57%) had positive antigliadin antibody titers, compared to only 5% of 94 patients with specific diagnoses, such as stroke, MS, and Parkinsonism, and 12% of 50 healthy blood donors. In antigliadin-positive patients with ataxia or neuropathy of unknown cause, duodenal biopsies revealed histological evidence of celiac disease in 35% and non-specific duodenitis in 38%. Only one had low vitamin B12 levels and the biopsy was normal. Gluten sensitivity was a common finding in this group of adult patients with ataxia and peripheral neuropathy of unknown cause. (Hadjivassiliou M et al. Does cryptic gluten sensitivity play a part in neurological illness? Lancet February 10, 1996;347:369-71). (Respond: Dr M Hadjivassiliou, Department of Clinical Neurology, Royal Hallamshire Hospital, Sheffield S10 2JF, UK).

COMMENT. This investigation underscores the importance of nutrition and diet in some neurological disorders of undetermined etiology. Antigliadin antibody estimation should be considered in the investigation of patients with neurological dysfunction of unknown cause, including those with refractory seizures, and especially if associated with occipital calcifications. Patients with histological evidence of celiac disease are treated with a gluten-free diet. However, those celiac patients with seizure complications and occipital calcifications are not always benefited by diet, and surgical resection of the involved occipital cortex may be required. (see Progress in Pediatric Neurology II, PNB Publishers, 1994, pp71-73).

CRANIAL NERVE DISORDERS

CONGENITAL FACIAL PALSY

The association between permanent congenital facial palsy in 61 children and recognized risk factors for traumatic birth was investigated in a retrospective case control study at the Departments of Plastic Surgery, Mount Vernon Hospital, Northwood, Middlesex, and the Hospital for Sick Children, Great Ormond Street, London, UK. The incidence of forceps assisted delivery

used in 13.2% of palsied patients was not significantly different from the 10.2% in the normal population. The prevalence of maternal primiparity (39.6%) among mothers of affected babies was no greater than that expected from national data. Big babies weighing >3500 g (18.9%) were fewer in number in the study group than in the general population (38.6%). This study shows that the risk of birth trauma is no higher in children with permanent 'congenital' facial palsy than in the general population. An intrauterine rather than a traumatic birth etiology is suggested. (Laing JHE et al. Is permanent congenital facial palsy caused by birth trauma? Arch Dis Child January 1996;74:56-58). (Respond: Mr Hamish Laing, 13 Princess Road, Primrose Hill, London NW1 8JR. Reprints: The Secretary, RAFT Institute of Plastic Surgery, Mount Vernon Hospital, Northwood, Middlesex HA6 2RN, UK).

COMMENT. In this large retrospective study, infants born with facial palsy were no more likely to have required forceps to assist delivery, and other risk factors for traumatic birth were not more prevalent than expected from national data. A commonly held assumption that birth trauma is frequently responsible for permanent congenital facial palsy appears to be false.

The authors of a study of 5 cases of unilateral congenital facial palsy identified in a retrospective review at the Massachusetts Eye and Ear Infirmary, Boston, MA, point out that spontaneous recovery is expected within 4 weeks in 90% of traumatic cases, and the prevalence of atraumatic developmental etiologies has been underemphasized in persistent congenital unilateral facial palsies. (Shapiro NL et al. Congenital unilateral facial paralysis. Pediatrics February 1996;97:261-265). The differential diagnosis of developmental cases includes: Mobius syndrome, cardiofacial syndrome, Goldenhar's syndrome (hemifacial microsomia), Poland's syndrome, DiGeorge syndrome, Albers-Schonberg disease, sclerostosis, trisomy 13, trisomy 18, and thalidomide embryopathy. In addition to these complex cases, isolated facial palsies may be complete or affect only the upper or lower lip. Electrophysiological and CT studies and especially electroneuronography (ENOG) are essential in the early differentiation of traumatic facial paralyses and those of developmental origin.

ETIOLOGY OF SENSORINEURAL DEAFNESS

Children with a bilateral sensorineural hearing impairment averaging 30 dBHL or or more were investigated for possible congenital or intrauterine infection as causes at the Centre for Audiology, Manchester University, UK. Of a total of 339 cases studied, 23% had a positive family history of deafness in parents or siblings (genetic group); 13% had suffered from birth asphyxia, respiratory distress syndrome, intraventricular hemorrhage, infections, or hyperbilirubinemia (perinatal group); 8% had congenital rubella or cytomegalovirus infection; 7% had a history of bacterial meningitis; 5% chromosomal anomalies; 5% were diagnosed with Waardenburg, Hurler, Hunter, Klippel Feil, or other syndromes; and 34% were of unknown cause. Autosomal recessive inheritance was probably responsible for most cases of unknown etiology. (Das VK. Aetiology of bilateral sensorineural hearing impairment in children: a 10 year study. Arch Dis Child January 1996;74:8-12). (Respond: Dr VK Das, Centre for Audiology, Education of the Deaf and Speech Pathology, The University, Oxford Road, Manchester M13 9PL, UK).

COMMENT. The authors are critical of the present screening