confirmed that the malformation was causing an intermittent obstruction and increased intracranial pressure. In one attack in which the patient experienced several typical episodes of right hand and right chest paresthesias, headache, and blurred vision the simultaneous intracranial pressure readings were elevated to a maximum of 580 mm H_2O. The pressure was sustained for approximately 2 minutes; as it decreased the patient's symptoms abated. Following posterior fossa decompression and a C1 to C3 laminectomy the pressure was relieved and the patient was asymptomatic. (Vrabec TR et al. Intermittent obstructive hydrocephalus in the Arnold-Chiari malformation. (Ann Neurol September 1989; 26:401-404).

COMMENT. Although this patient is outside the pediatric age group, Chiari I malformation becomes symptomatic in children and the transient nature of the signs and symptoms may prove misleading in diagnosis. Conventional CT and MRI may fail to reveal the malformation and posterior fossa directed MRI using multiple thin sagittal sections including the midline view may be necessary for diagnosis.

ORIGINS OF CEREBRAL PALSY

The causes of cerebral palsy were determined in a prospective study of 43,437 full term children from data collected in the Collaborative Perinatal Study of the National Institute of Neurological and Communicative Disorders and Stroke, Bethesda, MD, and published from the Department of Pathology and Center for Biostatistics and Epidemiology, M.S. Hershey Medical Center, Pennsylvania State University College of Medicine, Hershey, PA. The diagnosis of cerebral palsy had been made by neurologic examination at seven years of age in 127 and at one year of age in 23 who then died before the seven year follow-up. The incidence of cerebral palsy in the group studied was 1 in 290 (0.34%). Based on attributable risk estimates, congenital malformations explained over half (53%) of the cases of quadriplegic cerebral palsy. Only 14% of the cases of quadriplegic CP was attributable to birth asphyxia and 8% to CNS infections. Of 116 nonquadriplegic patients with cerebral palsy congenital disorders explained about one-third and CNS infections about 1 in 20; no cause could be identified for nearly 60% and birth asphyxia was not a significant antecedent. Characteristic consequences of birth asphyxia were more often the result of nonasphyxial disorders. These included meconium in the amniotic fluid, low 10 minute Apgar scores, neonatal apnea spells, seizures, persisting neurologic abnormalities, and slow head growth after birth. Congenital disorders explained nearly four times as many cases of quadriplegic CP then did birth asphyxia (attributable risks, 53% vs 14%, respectively). Congenital disorders were the only factor that explained a large number of the nonquadriplegic CP cases. Oxytocin, whose use was followed by an increased frequency of neonatal seizures, was not followed by a corresponding increase in the frequency of CP. (Naeye, RL et al. Origins of cerebral palsy. AJDC October 1989; 143:1154-1161).

COMMENT. Many previous studies have invoked asphyxia as the cause of CP on the presence of fetal distress and low Apgar scores.

In the present study, low ten minute Apgar scores were more often associated with congenital disorders than with birth asphyxia in CP cases. Fetal distress and low Apgar scores cannot be used to distinguish CP of asphyxial origin from CP due to congenital malformation. Birth asphyxia or hypoxia that is severe enough to damage the fetal brain usually causes death before or soon after birth. The present authors underscore the importance of making accurate measurements and observations on neonates to avoid misattributing nonasphyxial CP to birth asphyxia. Carefully recorded observations of kidney, heart, and lung function can help to determine cause, because birth asphyxia that is severe enough to damage the brain usually damages the kidneys, lungs, and often the heart.

The purpose of the Collaborative Perinatal Study of the NINCDS was to determine the causes and methods of prevention of cerebral palsy. This present analysis of the data collected from the study shows that no cause could be identified for the majority of cases of cerebral palsy in term infants. Has the Collaborative Study failed in its designed purpose? In an editorial comment, Bedrick AD of the University of Arizona, Tucson, states that "prematurity and low birth weight are strong risk factors for CP. Prevention of preterm delivery would be a tremendous stride in preventing CP." This same comment was voiced by some of the committee members, myself included, 34 years ago in the early planning stages of the Collaborative NINCDS Study, Bethesda, 1955. The proposal of a prospective study restricted to the causes and prevention of prematurity in relation to CP which might have provided early answers was overruled in favor of the more general and involved study of all pregnancies. Much time and energy have been expended in the analysis of the data collected by this extensive study only to conclude that we probably do not know what causes most cases of cerebral palsy .

INTRACRANIAL TUMORS

WEST SYNDROME AND CEREBRAL TUMORS

Two infants, six and seven months of age, with West syndrome associated with cerebral tumors are reported from the Department of Neurology, Pediatric Hospital, Buenos Aires, Argentina. Initial neurologic examinations were normal and the diagnosis of the tumors was by ultrasound and CT. One infant had a Grade III glioma in the right thalamus and the other had an anaplastic ependymoma and cyst in the right hemisphere. EEGs revealed generalized hypsarrhythmia in both cases. Infantile spasms responded to ACTH 5 IU/kg/day. One patient died at 18 months of age and the other was seizure free after complete surgical resection of the ependymoma. (Ruggieri V et al. Intracranial tumors and West syndrome. Pediatr Neurol Sept/Oct 1989; 5:327-9).