infants but was of limited prognostic value in assessment of neurological outcome. These results contrast with those reported by Walsh P (J Pediat 1982;101:988), who found that serum CPK-BB activity measured in cord blood and at 6-12 hrs of life correlated with neurological outcome after severe asphyxia and compared favorably with CT scanning as a prognostic indicator. Normal CPK-BB activity was a predictor of good neurologic outcome in both studies (see Ped Neur Briefs 1987;1(3):17).

BRAINSTEM INJURY FROM PERINATAL ASPHYXIA

The clinical, radiological and neuropathological features of selective hypoxic-ischemic injury of the brainstem with relative sparing of cortex and subcortical white matter in an asphyxiated term infant are described in a case reported from the Division of Neurology and Depts of Pediatrics, Pathology and Radiology, British Columbia's Children's Hospital, Vancouver, Canada. The infant was pale, flaccid and without respiratory effort at birth and seizures occurred during the first hour. The Apgar score was one at 1, 5 and 10 min. The signs of brainstem dysfunction included abnormal horizontal eye movements, facial diplegia and ptosis, tongue fasciculations, and abnormal auditory evoked potentials. CT showed increased attenuation in the basal ganglia at 2 wks, and dilation of the third ventricle at 1 mo. Lateral ventricles and cortical sulci were normal, showing no atrophy.

The infant died of pneumonia at 4 mo of age. Neuropathological examination revealed scarring and pallor of the thalamus, basal ganglia and brainstem with neuronal loss and gliosis. (Roland EH et al. Selective brainstem injury in an asphyxiated newborn. Ann Neurol Jan 1988;23:89-92).

COMMENT. In animal studies, selective brainstem damage occurs after acute total asphyxia whereas the cerebral cortex and subcortical white matter are predominantly affected by prolonged partial asphyxia. In the human infant, the localization of hypoxic-ischemic encephalopathy is generally more diffuse (Volpe JJ Neurology of the Newborn 2nd ed, Philadelphia, Saunders, 1987) and selective brainstem injury is rare and frequently fatal.

CEREBRAL PALSY

A professor of obstetrics at the Univ of California at Davis School of Med, Sacramento, reviewing the relationship of obstetric care and management of asphyxia to the subsequent development of cerbral palsy (CP), refers to his own previously published study at Oxford University (Lancet 1984;2:827) and a similar study in progress at the Univ of Newcastle, England. Babies who were at risk for development of CP were compared with matched normal controls. The frequencies of substandard obstetric care were determined in the controls and in all cases of fetal death from asphyxia or trauma, those with severe asphyxia, convulsions in the first 48 hrs of life, and in children recognized to have CP at 18 mo of age.

Quality of care during labor proved to be less important than prenatal care. Substandard care during labor was not related to severe asphyxia, neonatal convulsions, or CP. A delay in the initiation of treatment for diagnosed asphyxia was not observed in CP cases, was uncommon in the control group (1.4%), but was frequent in cases of fetal death (20%), convulsions (7.9%) and severe birth asphyxia (5.4%). Substandard intrapartum care and especially the lack or failure to react appropriately to electronic fetal monitoring was causally related to neonatal seizures but not to CP.