COMMENT. The definition of hypoglycemia in the newborn is unclear because the lower limit of an accepted ‘safe’ normal has not been determined. Susceptibility to brain injury following hypoglycemia varies with the individual, based on gestational age (GA), maternal glucose metabolism, glucose content of early feeding, and occurrence of hypoxemia and ischemia. Some authorities consider a glucose level of <50 mg/dL should be viewed with suspicion and a threshold level for recommended treatment. (Behrman-Nelson Textbook of Pediatrics, 17th ed. Philadelphia, Saunders, 2004;505-518). Others state that serum glucose of <30 mg/dL (1.65 mmol?l) in the first 24 hours after birth and <45 mg/dL (2.5 mmol/L) thereafter constitute hypoglycemia of the newborn. (Cornblath M et al. Pediatrics 2000;105:141-5). The criteria employed for inclusion in the above Hammersmith Hospital study were a GA of >36 weeks, one or more episodes of hypoglycemia (blood or plasma glucose level of <2.6 mmol/L) associated with acute neurologic symptoms in the first week (eg jitteriness, seizures, hypotonia), and MRI at postnatal age of <6 weeks. Patients with evidence of HIE, congenital infection or brain malformations were excluded. Hypoglycemia of <1.5 mmol/L was considered severe.

The study provides new evidence of variability of MRI-defined brain injury in neonates with symptomatic hypoglycemia, and their neurodevelopmental outcomes at 18-24 months. MRI abnormalities fail to correlate with severity and duration of neonatal hypoglycemia. In contrast, severity of white matter injury, the most common MRI abnormality, is a good predictor of outcome. A review of the literature for case series with longer follow-up found a study in the Netherlands involving 75 term infants with transient hypoglycemia on the first day of life, born to non-diabetic mothers, and examined at 4 years of age by the Denver Developmental Scale and the Child Behavior Check list. No significant differences in psychomotor development are observed between children with normal neonatal glucose levels and hypoglycemic subjects (plasma glucose <2.2 mmol/L/one hr after birth, or <2.5 mmol/L subsequently). (Brand PL et al. Arch Dis Child 2005;90:78-81). In the present series, with shorter follow-up, 85% are walking by 2 years, 14% have moderate cognitive delay, one third have small head size, one third have visual abnormalities, and 27% develop later seizures. Further follow-up is recommended to assess functional impairments at school age.

TRAUMATIC DISORDERS

DIAGNOSIS AND TREATMENT OF TRAUMATIC CAROTID ARTERY DISSECTION

Nineteen reports of pediatric cases of extracranial traumatic carotid artery dissection (CAD), obtained through PubMed search, were reviewed by researchers at Texas Children’s Hospital and Baylor College of Medicine, Houston, TX. Diagnosis made after onset of ischemic symptoms was confirmed by cerebral angiography in 24 of 34 patients, and
reconfirmed by MR angiography in 6 patients; CA Doppler scanning was used alone in 4 patients. Thirty patients were treated with medical therapy or observation. Open surgical or endovascular treatment of CAD was rarely employed in the pediatric population. Of 17 patients treated conservatively with anticoagulation/antiplatelet therapy, 4 recovered completely, and 11 were partially recovered. Of 12 patients treated expectantly without anticoagulation, 2 recovered completely, 8 recovered partially, and 2 died. Of 4 patients treated surgically, 3 showed partial improvement, and 1 died 5 days postoperatively. The authors favor MR angiography as a screening test, anti-platelet therapy to anticoagulation therapy as first-line medical treatment, and endovascular stenting over open surgery in cases of failed medical treatment. (Chamoun RB, Mawad ME, Whitehead WE, Luerssen TG, Jea A. Extracranial traumatic carotid artery dissections in children: a review of current diagnosis and treatment options. J Neurosurg Pediatrics Aug 2008;2:101-108).

COMMENT. An algorithm for the evaluation and treatment of traumatic extracranial carotid artery dissection is proposed, based on a review of the literature. Arterial dissection is identified as the fourth most common cause of arterial ischemic stroke in children, after sickle cell disease, cardiac embolism, and moyamoya disease.

THORACOLUMBAR SPINE SUBDURAL HEMATOMA IN INFANT

A thoracolumbar subdural hematoma (SDH) resulting from nonaccidental trauma in a 4-month-old infant is reported from Women and Children’s Hospital, University of Buffalo, NY. The patient was noted to be apneic and had been shaken by a parent in an attempt to stimulate respiration. In the ED he was unresponsive and was diagnosed with myelopathy. In addition to bilateral clonus and extensor plantar responses, he had a bulging fontanelle, bruising on left shoulder, and retinal hemorrhages. Head CT showed SDH and subarachnoid hemorrhage. MRI of spine showed a SDH dorsal to the spinal cord with mass effect on the conus medullaris and descending nerve roots. Emergency T-12 to L-3 laminectomies were performed, and the hematoma was evacuated. Later, he required a ventriculoperitoneal shunt, and currently, he has severe developmental delay. (Gruber TJ, Rozzelle CJ. Thoracolumbar spine subdural hematoma as a result of nonaccidental trauma in a 4-month-old infant. J Neurosurg Pediatrics Aug 2008;2:139-142).

COMMENT. Spinal cord injury is an uncommon complication of shaken baby syndrome, and may be overlooked when associated with head trauma. The authors explain the injury as a result of hyperflexion and hyperextension of the spine around the rigid rib cage. It is important to perform imaging of the spine, in addition to head and neck in infants with shaken baby syndrome.