

lower motor scores on the Psychomotor Development Index. High levels of cocaine metabolites in meconium were correlated with the lower Mental Development Index scores at 6.5 months, 1 and 2 years, and to a lower Psychomotor Development Index score at 2 years. (Singer LT, Arendt R, Minnes S, et al. Cognitive and motor outcomes of cocaine-exposed infants. JAMA April 17, 2002;287:1952-1960). (Reprints: Lynn T Singer PhD, Case Western Reserve University, Triangle Bldg, 11400 Euclid Ave, Ste 250-A, Cleveland, OH 44106).

COMMENT. Cocaine-exposed children are at risk of significant cognitive deficits and a two-fold increase in the rate of developmental delay during the first two years of life. The impaired cognitive outcome at 2 years is predictive of an increased risk of learning disabilities at school age (Molfese VJ, Acheson S. Int J Behav Dev 1997;20:595-607). In this prospective, longitudinal study, cognitive delays were related to elevated levels of cocaine metabolites in infant meconium and to maternal reports of cocaine use during pregnancy. Laboratory studies of the developing nervous system have shown that cocaine has adverse effects on monoaminergic neurotransmitter systems and cortical circuits important in learning and memory (Volpe J. N Engl J Med 1992;327:399-407). Neonatal abnormalities including premature birth, low birth weight, microcephaly, and behavioral disorders have also been linked to prenatal cocaine exposure (Singer LT, et al. Neurotoxicol Teratol 2000;22:1-14). Dose-related effects of cocaine on neurobehavior may be demonstrated in exposed infants examined at 3 weeks of age; heavily exposed infants show impaired arousal and greater excitability than lightly or unexposed infants (Tronick EZ et al. Pediatrics 1996;98:76-83). Effects of prenatal cocaine on behavior noted in infancy are likely precursors of ADHD in later childhood (Millichap JG. Attention Deficit Hyperactivity and Learning Disabilities. Chicago, PNB Publishers, 2001).

In an editorial, Zuckerman H and colleagues (JAMA 2002;287:1990-1991) question the possible variability in potency and degree of cocaine contamination in different reports of prenatal cocaine exposure. Also, the inclusion of premature infants may present an added risk of impaired development at 2 years that may be difficult to control. Tobacco use during pregnancy, an additional toxic factor and variable found to correlate with delayed motor development in this study, also correlates with a two-fold increased risk of ADHD in childhood (Mick E, et al. J Am Acad Child Adolesc Psychiatry 2002;41:378-385). The editorial cautions regarding the tendency to public bias against drug-exposed infants, especially cocaine and so-called "crack kids" and their mothers. The findings in this and similar studies should not be used to promote stigmatization of cocaine-exposed children that might hinder appropriate interventional services.

PSEUDOTUMOR CEREBRI

STIFF NECK, TORTICOLLIS, AND PSEUDOTUMOR CEREBRI

Three prepubertal children diagnosed with pseudotumor cerebri and presenting with stiff neck and torticollis are reported from Schneider Children's Medical Center, Sackler School of Medicine, Tel Aviv, Israel. Patient 1, a 7-year-old male admitted with stiff neck, had been evaluated at 2 years of age for short stature and treated first with thyroxine and later, with growth hormone injections, starting 3 weeks before complaining of headache, neck pain and head tilt to the left. Funduscopic examination revealed papilledema and hemorrhages. CT and MRI showed no mass effect. CSF opening pressure was 340 mm water, with

normal glucose and protein. Following withdrawal of 6 ml CSF, the rigidity and neck pains resolved and neck movements were normal. Papilledema was reduced after 3 weeks treatment with acetazolamide and dexamethasone. Patient 2, a 9-year-old previously healthy female had a 10 day history of neck pain followed by headache, neck stiffness, and papilledema. CT was normal and CSF pressure 280 mm. Within one half hour of removal of 7 ml CSF, symptoms were completely relieved. Symptoms did not recur during subsequent treatment with acetazolamide and dexamethasone. Patient 3, an 8-year-old male was admitted with a 6 week history of headaches, torticollis for 4 weeks, right sided neck pain and papilledema. Known causes of pseudotumor, including trauma, infection, vitamins, and endocrine factors, were absent. CT showed slit-like ventricles. CSF pressure was 480 mm. Torticollis and neck pain resolved within 1 hour after lumbar puncture. He was asymptomatic and had normal fundi at 3 week follow-up, following therapy with acetazolamide and prednisone. (Straussberg R, Harel L, Amir J. Pseudotumor cerebri manifesting as stiff neck and torticollis. Pediatr Neurol March 2002;26:225-227). (Respond: Dr Straussberg, Department of Pediatrics C, Schneider Children's Medical Center of Israel, Petah Tikva, Israel 49202).

COMMENT. Pseudotumor cerebri should be considered in the differential diagnosis of acute onset of stiff neck or torticollis. A recent review of 10 children with pseudotumor cerebri, cited by the authors, found 4 patients presenting with stiff neck (Cinciripini GS et al. Am J Ophthalmol 1999;127:178-182). The mechanism of the stiff neck and torticollis and the association with prepubertal cases of pseudotumor are undetermined. The more classical presenting manifestations are headache, vomiting, and papilledema. Other more common neurologic disorders that underly neck rigidity and pain and/or torticollis are cervical trauma or inflammation, meningitis, subarachnoid hemorrhage, posterior fossa tumor, spinal cord syrinx, and cervical radiculitis. Funduscopic examination for papilledema is recommended in children presenting with unexplained neck rigidity or torticollis, with or without headache. The rapid relief of symptoms following lumbar puncture and the resolution of papilledema following a short course of acetazolamide and steroids are noteworthy.

MILLER FISHER SYNDROME PRESENTING AS PSEUDOTUMOR

Two female children, ages 9 and 2 years, who presented with pseudotumor cerebri and within 2 to 3 days, were diagnosed with Miller Fisher syndrome are reported from the Hôpital Universitaire des Enfants Reine Fabiola, Brussels, Belgium. *The 9-year old* had sudden onset of frontal headaches associated with nausea, diplopia, and convergent strabismus. Two weeks previously she had developed an acute febrile illness with diarrhea. Neurologic examination on admission was normal except for a left VIth nerve palsy. CT was normal, but opening pressure on lumbar puncture was 300 mm water. Headache and nausea improved initially after removal of 15 ml CSF, but then recurred, with vomiting. Following treatment with acetazolamide, symptoms again improved, but 3 days later, she developed Miller Fisher syndrome, presenting with bilateral III and VI nerve palsies, ataxia, and areflexia. EMG and NCS were normal, and H reflexes were absent, indicating impaired nerve conduction in proximal fibers and acute demyelinating polyneuropathy. Plasma serology was positive for *Campylobacter jejuni* and anti-GQ1b antiganglioside antibodies. Treatment with a 2-day course of iv immunoglobulin resulted in lessening of ataxia in 1 week, and recovery of reflexes and eye movements in one month and at 3 month follow-up. *The 2-year old patient* had a similar history and diagnoses. Plasma serology was negative for *C jejuni*, but positive for antimyelin antibodies. EMG and NCS were abnormal, with