# PEDIATRIC NEUROLOGY BRIEFS
## A MONTHLY JOURNAL REVIEW

J. GORDON MILLICHAP, M.D., F.R.C.P., EDITOR

Vol. 16, No. 5 May 2002

## TOXIC DISORDERS

### PRENATAL COCAINE EXPOSURE AND INFANTILE PSYCHOMOTOR OUTCOME

The effects of prenatal cocaine exposure on child development outcomes were studied prospectively in an obstetric unit of a large US urban teaching hospital at Case Western Reserve University, Cleveland, OH. Cocaine-exposed (n=218) and unexposed (n=197) infants were identified between 1994 and 1996 in a high-risk, low-socioeconomic, 80% black population screened soon after birth by clinical interview and samplings of urine and meconium for evidence of drug use. Reasons for exclusion included maternal psychiatric history (16), or no meconium (15). Outcome was determined by the Bayley Mental and Motor Scales of Infant Development measured at 6.5, 12, and 24 months of corrected age. The Home Observation for Measurement of the quality of Environment (HOME) test was administered at the 2-year visit by interview of caregivers. Cocaine-using mothers used alcohol, marijuana, and tobacco more often than nonusers, and a mean (SD) of 23.3 (44.0) rocks of cocaine weekly throughout pregnancy. The mean concentrations (ng/g) of cocaine metabolites in meconium were as follows: cocaine 142, cocaethylene 18, benzoylecgonine 552, and metahydroxy-benzoylecgonine 264. Cocaine-using women were older, had more children and less prenatal care, lower IQ scores, and more psychological distress. Cocaine-exposed infants had a lower gestational age, birth weight, head circumference, and length than unexposed infants. HOME scores were not different in exposed and unexposed groups. Eleven infants died during birth to 2 year follow-up, 8 in cocaine-positive and 3 in cocaine-negative children.

When controlled for confounding variables (race, sex, Apgar and HOME scores), cocaine-exposed children performed more poorly on the Bayley Mental Scale than unexposed children, with a 6-point deficit at 2 years, and a 2-fold risk of significant delay (mental development index <80) [p=.006]. The average Mental Development Index score for infants exposed declined by 14 points and for unexposed by 9 points (p=.004); an Index score of <70, in the mental retardation range, was obtained in 13.7% (27) of exposed compared to 7.1% (13) of unexposed children at 2 years of age (p=.04). Tobacco exposure, but not cocaine, predicted

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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