Substance Use Guideline 5B

PERINATAL COCAINE EXPOSURE, CARE OF THE NEWBORN

INTRODUCTION

While the search for a causal link between maternal cocaine and adverse infant outcomes is an ongoing and active area of research, it is important to recognise the role of multiple factors that accompany cocaine use during pregnancy that also adversely influence infants. **Cocaine use crosses all socio-economic boundaries and should not be considered safe in any setting.** Although affluence can protect some women against some of the adverse effects that women in poverty experience, cocaine use is frequently associated with malnutrition, polydrug use, homelessness, violence and infectious/communicable diseases that can also adversely influence child development.

Although there is no consensus among experts on all of the harmful effects of cocaine use in pregnancy on the wellbeing of the fetus, there is a growing number of studies that raise serious concerns about adverse outcomes. The difficulty in determining adverse effects is due in part to the difficulty of studying the effects of cocaine alone, as many cocaine users also use alcohol and other drugs. The issue is complicated further by the fact that there are very differing patterns of cocaine use, from occasional use (usually intranasal), to more "hard core" use that involves smoking "crack" cocaine, or using it IV. There is a dose-response curve, with the degree of harm influenced by:

1) The amount of drug to which the fetus is exposed.
2) The timing of exposure in gestation.
3) The length of time over which the exposure occurs.
4) Potential interaction with other drugs used. Of particular concern is the combination of cocaine and alcohol which produces a toxin called cocaethylene that may be more toxic to the central nervous system than cocaine itself. There is no evidence of a safe lower limit of consumption below which there is assurance of *no* harm to the fetus.

From our current understanding, prenatal cocaine induces vasoconstriction of various vascular beds that predispose the fetus to injury at key sites during embryogenesis (e.g. brain, eye, heart, kidneys, gastrointestinal tract). Because prenatal cocaine exposure lacks a specific outcome(s), it has been suggested that a “cocaine syndrome” does not exist, in contrast to clear, well defined outcomes that follow prenatal alcohol or opioid exposure. However, exposed infants should be considered at risk for effects of cocaine-induced vasoconstriction, possible long-term behavioural effects and secondary social / environmental consequences for the child and family that are associated with continued parental use. Further, it should be recognized that cocaine use is frequently accompanied by polydrug use (alcohol, heroin, etc.) and all the associated sequela and withdrawal symptoms that may follow.

FETAL/NEONATAL RISKS

1) Cocaine use does not lead to any pattern of *predictable* neonatal withdrawal.
2) Irritability and poor feeding in the first days of life are frequently reported (Bell & Lau, 1995; Volpe, 1992).

Many studies report specific outcomes that may be a consequence of the powerful vasoconstrictive effect cocaine has on organ development (Bandstra & Burkett, 1991). These include:

1) 10 fold increase in stillbirths (Bingol, Fuchs, & Diaz, 1987).
2) Prematurity - probably related to increased incidence of placental abruption (Ney, Dooley, & Keigh, 1990).
3) Intra-uterine growth retardation (Zuckerman et al, 1989).
4) Smaller head size - up to 8 fold increase in microcephaly (Nulman & Koren, 1994).
5) Increased incidence of genitourinary malformations (Battin et al, 1995; Chavez et al, 1989).
6) Possible increase in incidence of CNS malformations, including cerebral infarction (Bandstra & Burkett, 1991).
7) Possible increase in bowel malformations, such as gastroschisis (Bandstra & Burkett, 1991).
8) 3.9 increase in incidence of Sudden Infant Death Syndrome (Fares et al. 1997).
9) Increased incidence of neurological abnormalities in the neonate including global hypertonia, coarse tremor, and extensor leg posturing. Cocaine appears to have a dose response effect on these neurological impairments as well as a lowering head circumference (Chiriboga et al. 1999).

NEWBORN ASSESSMENT

1) A careful newborn physical and neurological exam and all appropriate investigations should be carried out.
2) **In addition** , because of the increase in incidence of malformations, consideration should be given to performing:
   - head ultrasound if there is abnormality in the CNS exam, seizures or other abnormal CNS behaviour, macrocephaly or microcephaly
   - renal ultrasound (Battin et al., 1995) (particularly if cocaine use was early or heavy during pregnancy)

NEWBORN MANAGEMENT

Cost for care of cocaine-exposed infants is 10 times the cost of matched urban controls and 3 times as much for those matched for other prenatal risk factors (Behnke et al., 1997; Calhoun & Watson, 1991).

In general, the management of an infant exposed to prenatal cocaine should be directed at managing concerning clinical symptoms, regardless of whether they are considered secondary to prenatal drug exposure. These may include:

- Irritability
- Poor feeding
- Diarrhea
- Temperature instability

Once a differential diagnosis (sepsis, opioid withdrawal, etc.) of other concurrent conditions has been considered, symptomatic management could include:

- Reduced environmental stimuli
- Increased caloric diet
- Pharmacologic measures for cocaine exposure are rarely indicated. Only for extreme irritability or abnormal CNS behavior should an agent such as phenobarb 3-5 mg/kg/day be used. This dose is maintained until signs and symptoms abate, and then the infant is weaned 10% of the original dose every day, depending on symptomatology. Again, consideration should be given to other causes of CNS irritability prior to any pharmacologic intervention.

I PRACTICE POINTS FOR NEWBORN CARE

A. Recognize the risk of adverse effects on organ development and undertake investigations as clinically warranted (e.g. ultrasound of head or kidneys, etc.).

B. Recognize possible withdrawal symptoms, regardless of whether they are associated with cocaine exposure e.g. concurrent opioid exposure, etc., and manage appropriately (See Guideline 4B – Perinatal Opioid Exposure, Care of the Newborn).

C. Recognize the social and environmental risk associated with continued parental cocaine use and its potential adverse effect on the growing infant and child. As with other high risk settings, early identification and communication with appropriate community supports is essential.

LONG TERM OUTCOMES

1) Increased incidence of behaviour problems in early school aged children exposed to cocaine in utero (Delaney-Black et al, 1998).
2) Lower scores in expressive language and verbal comprehension (Nulman & Koren, 1994).
3) Deficiencies in both gross and fine motor development at 2 years in cocaine exposed children (Arendt et al, 1991).
4) There is understandably a high risk of emotional and behavioural problems in children raised in homes where drug use persists, which in turn leads to problems in school performance.

5) Maternal cocaine use should be recognized as a "red flag" for the social and environmental variables associated with cocaine that may adversely influence growth, health and development of the infant, regardless of their prenatal exposure. These frequently include inconsistent care giving, nutritional concerns, violence, homelessness, and secondary cocaine smoke exposure.

LONG-TERM MANAGEMENT

Recognising that cocaine - exposed infants are at increased risk for learning and behavioural disabilities, long term management should include:

1) Infant Development Program Follow-up.
2) Medical evaluations.
3) Social and educational support in the school age period.
4) Other community support services that allow for timely and appropriate evaluation and management of "at risk" children.

REFERENCES

Arendt, R , Angelopoulous, J., Salvator, A., & Singer, L. (1999). Motor Development of Cocaine Exposed Children at Age 2 Years. Pediatrics (103). No 1, January, 86-92.

Bandstra ES, Burkett, G. (1991). Maternal-fetal and Neonatal Effects of In Utero Cocaine Exposure. Seminars in Perinatology (15). No. 4, August, 288-301,

Battin M et al. (1995) Congenital Genitourinary Tract Abnormalities following Cocaine Exposure In Utero. American Journal of Perinatology (12). No 6, 425-428.

Behnke M., et al. (1997). How fetal cocaine exposure increases neonatal hospital costs. Pediatrics (99). 204-208.

Bell GL, Lau K. (1995). Perinatal and Neonatal Issues of Substance Abuse. Pediatric Clinics of North America (42) No.2, April, 261-81.

Bingol N, Fuchs M, Diaz V. (1987). Teratogenicity of Cocaine in Humans. Journal of Pediatrics (110). p. 93-96.

Calhoun, B., & Watson, P. (1991). The cost of maternal cocaine abuse. Perinatal Cost Obstetrics and Gynecology (78). 731-734.

Chavez GF, et al. (1989). Maternal Cocaine Use during Early Pregnancy as a Risk Factor for Congenital Urogenital Anomalies. JAMA (262). No 6, August 11, p. 795-798.

Chiriboga, C., Burst, J., Bateman, D., & Houser, W. (1999). Dose-response effect of fetal cocaine exposure on newborn neurologic function. Pediatrics (103), NO. 1, 79-85.

Delaney-Black,V, et al. (1998). Prenatal Cocaine Exposure and Child Behaviour. Pediatrics (102). No 4, October, p. 945-950.

Fares, I., McCulloch, K., Raju, T. (1997). Intrauterine cocaine exposure and the risk of sudden infant death syndrome: A meta analysis. Journal of Perinatology(17), 179-182.

Ney JA, Dooley SL, Keigh LG. (1990). The Prevalence of Substance Abuse in Patients with Suspected Preterm Labour. American Journal of Obstetrics & Gynaecology (162). P.1562-1567.

Nulman I, Koren G. (1994). Neurodevelopment of Adopted Children Exposed In Utero to Cocaine. Canadian Medical Association Journal (151). No11, 1591-1596.

Volpe, J. (1992). Effect of Cocaine on the Fetus. New England Journal of Medicine Vol 327. No 6, 399-405.

Zuckerman B et al. (1989) Effects of Maternal Marihuana and Cocaine on Fetal Growth. New England Journal of Medicine (320). p. 762-768.