

**Substance Use Guideline 5A
PERINATAL COCAINE USE,
CARE OF THE MOTHER**

INTRODUCTION

In Canada, most female cocaine users are of childbearing age. The exact prevalence of cocaine exposure across Canada is unknown. A Toronto study found the overall incidence of cocaine exposure to be 6.25% as measured by hair and urine tests of 600 neonates born at three hospitals. The incidence was found to be higher among those babies born in the one downtown hospital (12.5%) versus those born in the two suburban hospitals (3.0%). Urine testing done failed to identify 76% of the cases of cocaine exposure in the 3rd trimester. The authors estimate that there are over 5,000 babies born annually in the greater Toronto area being cared for by mothers using cocaine (Forman et al., 1994). One study done in Vancouver showed a prevalence of cocaine and / or opiate exposure close to term at 3.8% as assessed by anonymous meconium screening done in 1995-1996 (Ling, Albersheim & Halftead, In press). Prevalence studies in the U.S. are wide ranging depending on location (0.7%-18%) but data from 1984 to 1992 indicated approximately 1-2% of infants born in the U.S. have been cocaine exposed in utero (Shino, 1996).

PHYSIOLOGY OF COCAINE USE

Cocaine HCl ("up", "blow", "snow") bought on the street is usually only 10-50% pure. Typically, it can be adulterated ("cut") with glucose or lactose to provide weight; amphetamines to increase stimulation; lidocaine to mimic cocaine's anesthetic properties; and heroin to counter cocaine induced agitation and to add to physical dependency thus ensuring a repeat customer. "Crack" is made by precipitating the cocaine HCl with a base like sodium bicarbonate. The impurities remain in the crack, also called "rock". If instead the cocaine HCl is cooked with a volatile substance like ether, it precipitates into pure cocaine called "free base" or "base" (Rottenstein, 1998). Cocaine HCl can be taken nasally, rubbed on the gums, or injected. Free base and crack are usually smoked though sometimes injected and these forms of cocaine are much more lipophilic, thus crossing the blood brain barrier (BBB) more quickly. There is a pyrolysis product of crack called MEG that also crosses the BBB quickly and along with the cocaine, crosses the placenta. There is also another substance called cocethelene which is formed from the coingestion of alcohol with cocaine use which may have fetal toxic effects in addition to those of cocaine and MEG (Plessinger & Woods, 1998).

If cocaine is taken nasally the effects start within minutes and peak by 30 minutes. If taken IV or by inhalation effects can start within a minute and peak within 4-10 minutes. Blood levels then start falling, precipitating more drug seeking. The half life of cocaine in the blood is 40-60 minutes. The urine can show cocaine metabolites for up to 2 or 3 days after a cocaine binge. The quick rise and fall of IV cocaine and smoked crack account for the repeated administration (often

20 times a day or until supplies run out) and its high addiction liability. This is in contrast to heroin, which has a half life of 4-6 hours so is usually administered 2-4 times a day (Gold & Herkov, 1998).

Cocaine HCl powder and its crystalline precipitates (free base and crack) exert powerful effects on the pleasure and "fight or flight" centres of the brain. Cocaine's reinforcing and euphoric effects are mediated through blocking the reuptake of dopamine in the brain's mesolimbic system, especially in the nucleus accumbens (Gold & Herkov, 1998). Thus, there is increased dopamine stimulation of the pathways that normally mediate appetite and sex drive. This is why interest in food is greatly diminished and the pleasure of cocaine is repeatedly sought, even when other risks must be endured. The lack of nutrition during cocaine binges can lead to maternal wasting and contribute to fetal IUGR.

Likewise, the presynaptic reuptake of norepinephrine is blocked in the locus ceruleus in the brain and elsewhere in the body and norepinephrine levels rise causing activation of the sympathetic nervous system. In low doses this activation translates into increased alertness. In higher doses this stimulation of the body's biologic alarm system leads to vasoconstriction, hypertension, tachycardia, dilated pupils, insomnia, agitation, diaphoresis and a host of other signs and symptoms related to norepinephrine release (Gold & Herkov, 1998). It is primarily the vasoconstriction in the placenta that puts the fetus in immediate danger with each cocaine use since this can produce ischemia and trigger abruption. The elevated sympathetic output can also lead to increased uterine tone and premature labour. Chronic use of cocaine can contribute to placental insufficiency and IUGR (Plessinger & Woods, 1998). It is thought that the vasoconstriction effects of cocaine use in early pregnancy is what may contribute to the fetal abnormalities in kidney, GI tract, lungs and brain that have been reported (Hoyme et al, 1990; Jones, 1991; Battin, 1995).

Cocaine also affects the brain's serotonin system. It is the changes in serotonin and dopamine that contribute to the psychosis and paranoia induced by cocaine (Gold & Herkov, 1998).

WITHDRAWAL

After days of repetitive use of cocaine the brain stores of dopamine have been depleted and cocaine cannot produce the same high. By this point the user is often exhausted (both physically and financially) and stops using. Withdrawal consists of the "crash" of the above symptoms as blood levels of cocaine fall and dopamine levels in the brain are low. The woman experiences lethargy, hypersomnolence, hyperphagia, depression, anxiety, and more cravings often including drug using nightmares ensuing over 1-5 days. A lower level protracted withdrawal syndrome can occur lasting for weeks consisting of anhedonia (lack of pleasure), depression, sleep disturbance, and drug craving (Gold & Herkov, 1998). Unlike alcohol, heroin or benzodiazepine withdrawal, the acute cocaine withdrawal is not physically dangerous to mom or fetus. However, relapse and mental health issues can pose risks for individual women. After a few days of rest and restoration the craving for cocaine often once again overwhelms the person and the cycle is repeated. It is this cycle we see as women appear for admission only to eat and sleep for a few days then be gone again to the street repeating cycles of behaviors that put her and her fetus at risk. However,

independent of other factors cocaine appears to have a dose-response effect on neurobehavioral outcomes and elevate the risk of anomalies as outline in Substance Use Guideline 5B: Perinatal Cocaine Exposure, Care of the Newborn.

MATERNAL RISKS OF COCAINE USE (Miller, 1998; Plessinger & Woods, 1998)

- 1) Cardiac arrhythmias.
- 2) Sinus tachycardia and bradycardia.
- 3) Myocardial ischemia and infarction.
- 4) Seizures.
- 5) Stroke.
- 6) Lung damage, "crack lung" and asthma.
- 7) Sexually transmitted diseases.
- 8) Rhabdomyolysis (muscle breakdown that can lead to renal failure).
- 9) Psychosis.
- 10) Placental abruption.
- 11) Spontaneous abortion.
- 12) Death.

FETAL / NEONATAL RISKS OF COCAINE EXPOSURE

(See Substance Use Guideline 5B: Perinatal Cocaine Exposure, Care of the Newborn)

Most women who abuse substances in pregnancy use more than one drug. It is often difficult to tease out the effect of cocaine alone from those of say, cigarettes and alcohol. In addition, there tends to be bias in the literature to report positive findings and not publish negative results with regard to perinatal cocaine effects (Koren et al., 1989). The "causative agent" seems to be a mixture of social parameters like nutrition, housing, and lack of prenatal care along with actual drug use behaviors (type, frequency, amount, route).

For an overview of the effects of cocaine use on the neonate and child, see Hans, 1998; Plessinger & Woods, 1998; and Nulman et al., 1994.

CLINICAL MANAGEMENT

For general management, see Substance Use Guideline 3: General Clinical Management of Pregnant Substance Using Women.

There is a paucity of evidence for the safety and efficacy of drug therapies for use with cocaine withdrawal in pregnancy. Below are suggestions based on experience, convention and some literature in non-pregnant individuals (Miller, 1998; Center for Substance Abuse Treatments, 1995).

I ANTENATAL HOSPITAL ADMSSION

Cocaine abuse or dependence constitutes a high risk pregnancy. A woman having a problem with cocaine should be offered shelter in a residential setting (e.g. hospital) for several reasons:

- 1) Cocaine craving and use are very environmentally cued so withdrawal in the home setting may be difficult.
- 2) Often there has been little prenatal care and admission is the most time efficient way to address the variety of health and social concerns.
- 3) Women may need shelter from their partners (if drug using or abusive).
- 4) Time is needed to sort out options. Admission is a vital window of opportunity to assess and assist with needs and make links with community resources. It is also an opportunity to build trust, have conversations about the baby, and allow a momentary haven in the chaos.

A. History, Physical and Investigations

See Substance Use Guideline 3 – General Clinical Management of Pregnant Substance Using Women.

B. Physician Orders and Instructions

- No medication is usually indicated or advised.
- If marked agitation ensues or patient's cravings put her at high risk of leaving hospital and/or relapsing then **one** of the following medications can be used for about 5 days:
 - 1) diazepam 5 mg up to qid prn, **or**
 - 2) lorazepam 0.5-1 mg qid prn, **or**
 - 3) doxepin 25 mg bid, **or**
 - 4) phenobarbital 30-60 mg q4h prn (d1-2) then q6h (d3-4)
- **Note:** bromocriptine is not approved for use in pregnancy.
- Order double food trays each meal, facilitate access to abundant snacks.
- Daily nonstress tests when acute, then 2-3x/week.
- Emotional support and encouragement, low stimuli, baths.
- Acupuncture, Acupressure, Therapeutic Touch as available.

Assisting in connections to community resources including ongoing prenatal care, nutrition, safe housing, parenting classes, addiction rehabilitation programs, and child welfare workers, when applicable, need to be addressed (see Guideline 2: *Discharge Planning Guide for Substance Using Women and their Newborns*).

Harm reduction strategies need to be discussed as well as the other learning areas identified in Guideline 2.

II ANTENATAL HOME DETOX

It is medically safe to withdrawal from cocaine outside of hospital, however this may be a window of opportunity for more intensive care in a hospital/detox setting. At home a benzodiazepine can be used to combat agitation and assist with sleep for the first 5 days or so. Beware of the addicting and disinhibiting effects of benzos.

III LABOUR AND DELIVERY

The woman can be treated the same as above but care needs to be applied with phenobarbital so that the neonate is not too sedated.

IV POSTPARTUM

- 1) Breastfeeding provides optimal infant nutrition however, in the context of substance use many other considerations are involved. There needs to be a discussion of the risks and benefits and the mother needs to make an informed choice. Breastfeeding is contraindicated if HIV +, or if active substance use of certain substances is present (e.g. heroin, cocaine, amphetamines) (Howard & Lawrence, 1998). Cocaine can remain in breast milk up to 60 hours after last use so the woman needs to be stable in recovery to breastfeed (Howard & Lawrence, 1998). There is still debate about breastfeeding if the mother has HCV. The ACOG Committee Opinion titled Breastfeeding and the Risk of Hepatitis C Virus Transmission (1999) states, "Studies to date evaluating the effect of breastfeeding on HCV transmission indicate that the average rate of infection is 4% in both breastfed and bottlefed infants. Therefore, it appears that breastfeeding does not appreciably increase the risk of transmitting HCV to a neonate."
- 2) **If cocaine use has been recent then a postpartum stay of 5-7 days is reasonable** to rest, eat, and make plans. If cocaine use only occurred early in the pregnancy and the social situation is currently stable and good family, medical, addiction, and mental health supports are in place, then an earlier discharge is appropriate.

See Guideline 2: *Discharge Planning Guide for Substance Using Women and their Newborns.* Follow up support is crucial.

REFERENCES

Battin, M. et al. (1995). Congenital genitourinary tract abnormalities following cocaine exposure in utero. American Journal of Perinatology (12). NO.6, 424-428.

Centre for Substance Abuse Treatment. (1995). Guideline 5 – Cocaine withdrawal. In: Marion, J. (ed). *Pregnant, Substance-Using Women. Treatment Improvement Protocol (TIP) Series #2*. U.S. Department of Health and Human Services: Rockwall, IL. 22-23.

Forman, R., Klein, J. et al. (1994). Prevalence of fetal exposure to cocaine in Toronto, 1990-1991. Clinical Investigative Medicine(17), NO.3, 206-211.

Gold, M. & Herkov, M. (1998). The pharmacology of cocaine, crack and other stimulants. In: Graham A., Schultz T. eds. Principles of Addiction Medicine. Second edition. Chevy Chase, MD: American Society of Addiction Medicine. 137.

Hans, S. (1998). Developmental outcomes of prenatal exposure to alcohol and other drugs. In: Graham A., Schultz T. eds. Principles of Addiction Medicine. Second edition. Chevy Chase, MD: American Society of Addiction Medicine. 1223-1237.

Hoyme, H. et al. (1990). Prenatal cocaine exposure and fetal vascular disruption. Pediatrics (85). 743-747.

Howard, C., & Lawrence, R. (1998). Breast feeding and drug exposure. In: Graham A., Schultz T. eds. Principles of Addiction Medicine. Second edition. Chevy Chase, MD: American Society of Addiction Medicine. 195-217.

Jones, K. (1991). Developmental pathogenesis of defects associated with prenatal cocaine exposure: Fetal vasculature disruption. Clinical Perinatology (18). 139-146.

Koren, G. et al. (1989). Bias against the null hypothesis: The reproductive hazards of cocaine. Lancet (2). 1440-1442.

Ling, E.W., Albersheim, S.G., and Halftead, A.C. (In press). Prevalence of In Utero Drug Exposure by Meconium Screening and Infant Outcome.

Miller, L. (1998). Treatment of the addicted woman in pregnancy. In: Graham A., Schultz T. eds. Principles of Addiction Medicine. Second edition. Chevy Chase, MD: American Society of Addiction Medicine. 1199-1209.

Nulman, I., Rovet, J., Altmann, D., et al. (1994). Neurodevelopment of adopted children exposed in utero to cocaine. Canadian Medical Association Journal (151). NO.11, 1591-1596.

Plessinger M., & Woods, J. (1998). Cocaine in pregnancy: Recent data on maternal and fetal risks. Obstetric and Gynecologic Clinics of North America (25). NO.1, 99-118.

Rottenshein, M. (1998). Cocaine and amphetamines. ASAM's Review Course in Addiction Medicine. 22-24. Chicago, IL.

Shino P., (1996). Prevalence of drug exposed infants. The Future of Children (6). NO. 2, 159-163.