Anesthetic implications of substance abuse in the parturient

JAY HOROWITZ, RN, BSN
Cleveland, Ohio

Substance abuse has become an increasingly devastating problem in this country and abroad during the last two decades. Huge amounts of time and money have been devoted to the treatment and eradication of this widespread problem. Increasing media attention, brought about by the premature deaths of celebrated athletes, has served to increase the public's awareness of the problem.

Substance abuse crosses all socioeconomic, gender and religious boundaries. In the pregnant patient, substance abuse affects not only the mother, but the unborn fetus as well.

The effects of four commonly abused substances—alcohol, marijuana, cocaine and narcotics—on the mother and fetus will be examined, as well as the implications for anesthetists in administering safe anesthesia for these patients.

Until we can educate the public and eradicate the problem of substance abuse in our society, we will continue to see patients involved with drugs. Awareness of the scope of the problem and the specific effects of the aforementioned drugs should help the anesthetist administer safe and effective anesthesia to substance abusers.

Alcohol abuse

One of every ten people who consumes alcohol is an alcoholic. There are reported to be over nine million alcoholics in the United States, five million of whom are women. At least one million of these women are of childbearing age.

In the past, the negative effects of alcohol consumption on the fetus were attributed to environmental factors, malnutrition and inferior genetic makeup, rather than to the effects of alcohol itself. Now it is believed that malnutrition and vitamin and iron deficiencies are concurrent factors that affect the health of the fetus, but that the ingestion of alcohol itself has devastating consequences. Even moderate amounts of alcohol ingestion have been linked to fetal abnormalities, although one ounce of alcohol per day seems to cause few negative effects on the fetus.

The major outcome of alcohol abuse during pregnancy is Fetal Alcohol Syndrome (FAS), which may be divided into four categories:

1. Central nervous system dysfunction, where mental retardation is the major outcome. Tremulousness, weak grasp, poor hand-eye coordination, irritability, poor sucking ability, head rolling and rocking behavior commonly are seen. Less often seen are microcephaly (dose dependent), anencephaly and meningomyelocele.

2. Growth deficiency, including lower birth weights and decreased head circumference, despite normal levels of growth hormone.

3. Possible renal and hepatic abnormalities.
(4) Well-documented facial characteristics, including abnormal jaw protrusion, flat nasal bridge, turned nasal tip, a thin vermilion and a flat midface. Joint and cardiac abnormalities, including ventricular and atrial septal defects, also have been reported.

The overall perinatal mortality rate in infants born to alcoholic mothers is 17%, compared with 2% in the non-alcoholic population. Both ethanol and its metabolite acetaldehyde have been implicated as teratogens and appear to inhibit neural growth. This may be the result of acidosis, hypoxia, decreased protein synthesis, lessened RNA/DNA content or a combination of these factors.

The metabolism of ethanol increases the $O_2$ consumption of the liver by 100%, leading to collapse of the umbilical vasculature, increased incidence of abruptio placenta, antepartum bleeding and anemia—all factors which decrease the amount of oxygen available to the fetus, resulting in fetal hypoxia and its sequelae.

Alcohol consumption during the first two trimesters greatly increases the incidence of spontaneous abortion. At the time of delivery, a higher incidence of meconium staining, lower Apgar scores and, in some cases, withdrawal symptoms (sweating, shivering, spasticity) have been noted.

Maternal changes associated with alcohol abuse depend upon the amount and duration of alcohol consumption and the recency of alcohol ingestion. Common alterations include peripheral neuropathies, decreased levels of albumin, cardiomyopathy, decreased tolerance to hypoxia, increased gastric acid production, microsomal enzyme induction, coagulation deficiencies (decreased fibrinogen, prothrombin and factors V, VII, IX and X), esophageal varices, ascites and cirrhosis. Vasodilation leading to hypothermia and shivers may be confused with delirium tremens.

There have been two reports from Emory University regarding difficult intubations in newborns and children of alcoholic mothers. These children presented without any of the obvious anatomical abnormalities commonly associated with difficult airways. In one case multiple attempts at intubation were made using various blades, both orally and nasally, without success. A less than satisfactory airway finally was maintained by mask. In both cases, the children presented with ventricular septal defects (VSDs).

Anesthetic considerations for the alcoholic parturient also will depend upon the amount, duration and recency of alcohol consumption. As with any patient, a thorough preoperative evaluation is essential. Serum alcohol levels should be drawn. A result of 100 mg/100 ml is considered legal intoxication, 200 mg/100 ml is severe intoxication, 400 mg/100 ml will leave a patient stuporous and a level of 500 mg/100 ml can be fatal. The use of other drugs also should be explored.

The presence of peripheral neuropathies will rule out the use of regional techniques in most institutions, and such neuropathies should be documented. The literature recommends limited use of premedication in alcoholic parturients. Because of the patient's decreased levels of albumin and possible cardiomyopathy, a careful, controlled induction is preferred. Since thiobarbiturates are potentiated by ethanol, large doses of pentothal could be lethal, and potent inhalation agents may depress an already compromised myocardium.

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If the parturient is intoxicated at the time of the delivery, she may require decreased amounts of thiobarbiturates to produce unconsciousness, even though she appears alert and cooperative. Increased doses of non-depolarizing neuromuscular blockers may be required to produce relaxation, yet they will have a longer duration because of the patient's low albumin and heightened gamma globulin levels. Prolonged responses to succinylcholine have been reported as a result of decreased cholinesterase at the motor end plate.

Fluid and electrolyte maintenance may be especially difficult in alcoholic parturients, since they are relatively hypovolemic because of the inhibition of ADH during acute ethanol intoxication. The presence of hypovolemia, along with the anticipated blood loss associated with delivery, may require very large volumes for replacement.

Albumin solutions, which must be carefully administered to avoid congestive heart failure, may provide needed proteins and be beneficial to volume replacement. Solutions containing sodium should be avoided if ascites is present.

The characteristics of alcohol withdrawal can range from mild to severe. Mild symptoms include tremors, anorexia, vomiting, weakness, sweating, nightmares and autonomic imbalances. These symptoms last from six to 50 hours, whereas severe withdrawal or delirium tremens may take three to four days to appear and last seven to 10 days. Symptoms include disorientation, hallucinations, increased catecholamine release, hyperpyrexia, hyperreflexia and seizures. For patients with these symptoms, a C-section with general anesthesia and rapid-sequence induction may be the safest method of anesthesia.

**Marijuana usage**

Marijuana, hashish and hash oil are the three major derivatives of the leaves, stems, fruiting tops.
and resin of the female hemp plant Cannabis Sativa. The active ingredients are Cannabinol (Δ 9-THC) and Cannabidiol (Δ 8-THC). The most potent derivative is hash oil, which contains 60-90% THC (tetrahydrocannabinol). The average marijuana cigarette (joint) contains about 3% THC. THC is a highly lipid soluble substance which accumulates in fatty tissues (i.e., brain, gonads) and has a half-life of 72 hours. The half-life of the metabolites can be seen in the urine for about one week. However, the concurrent use of tricyclic antidepressants can inhibit the transformation of THC in the liver by 20-30%.

The occurrence of marijuana use in the pregnant population ranges from 10 to 37%. Unfortunately, studies done on this population and the general population as well have several innate problems. There often is unreported concurrent usage of other drugs—including nicotine and caffeine—so the reported drug usage may not reflect actual usage. In the pregnant population, the outcome of the pregnancy also may reflect poor prenatal care.

There are several documented effects of chronic marijuana usage in both the mother and the fetus/newborn. There is a higher incidence of marijuana usage during the first trimester of pregnancy, because of the antiemetic effects of the drug. Unfortunately, this also is the most critical period for fetal development.

Cannabinoids do cross the placenta, and their use may lead to fetal alcohol syndrome-like features in the newborn, including tremulousness, a high-pitched cry and an altered response to visual stimuli as a result of an inhibition of the light-sensitive areas of the lateral geniculate nucleus of the thalamus.

THC is known to cross the placenta, and its concentration in breast milk is higher than in maternal plasma, which indicates that the newborn is absorbing THC from breast milk.

The effects of marijuana on the mother reveal themselves both during pregnancy and the delivery itself. During pregnancy, incidence of anemia and poor weight gain has been reported, and a lower birth weight (decreased 105 gm) because of intrauterine growth retardation has been seen. The effects upon delivery seem to be related to the recency of marijuana use. Commonly reported effects upon the labor process include protracted and arrested labor; increased incidence of meconium and fetal resuscitation; abnormal fetal tests, and need for manual removal of the placenta. One study hypothesized that marijuana may increase prostaglandin synthesis, thereby initiating the labor process.

There are many physiologic effects of marijuana which may influence the anesthetist's choice of technique. Marijuana is a cardiac depressant; PVCs, T-wave inversion, decreased P-wave voltage, tachycardias, (the result of increased circulating epinephrine levels) and bradycardias all have been seen. Therefore, marijuana may augment cardiac depression of potent inhalational agents.

There also is a profound response to agents which sensitize the myocardium to catecholamines. Increased sympathetic nervous system activity and inhibition of the parasympathetic nervous system also may account for tachyarrhythmias and orthostatic hypotension.

Dilation of the respiratory tree has been reported, and in some cases marijuana has been advocated for use in the asthmatic patient. However, toxic doses have been shown to initiate pulmonary depression and pulmonary edema. Respiratory depression associated with narcotics may be potentiated by marijuana.

Two other noteworthy effects of marijuana usage are depression of the temperature-regulating mechanism and a noticeable anticholinergic effect that can lead to potentiation of non-depolarizing neuromuscular blocking agents. Marijuana also has been shown to increase the sleep time with ketamine and thiopental and has been associated with an increased incidence of hallucinations in the presence of intermediate-acting barbiturates. Recent use of marijuana also is associated with a reduction in the MAC (minimum alveolar concentration) of halothane.

The choice of anesthetic technique must be made (as always) after assessing the patient. While regional techniques may offer optimal anesthesia in chronic abusers, administering a regional to an agitated, paranoid, acutely intoxicated patient may not be possible.

**Cocaine use**

The recent cocaine-related deaths of several prominent athletes have drawn public awareness to the increasing prevalence of cocaine abuse in this country. Its greater availability as a result of lower cost and the development of more potent varieties of the drug, such as crack, have led to cocaine abuse among a larger cross-section of our population. Cocaine is no longer just for the rich.

Cocaine is an ester local anesthetic derived from the coca plant. It is a potent central nervous system stimulant that is abused primarily because of the feelings of euphoria and increased energy it produces. Usually, cocaine is inhaled, which leads to destruction of the nasal septum, but its intravenous use—a lone or in combination with narcotics—is relatively common.
Cocaine acts primarily by inhibiting nerve conduction and preventing the reuptake of norepinephrine at the nerve terminal. Increased circulating levels of norepinephrine cause vasoconstriction, tachyarrhythmias and hypertension. Acetylcholine release also is decreased by 33% to 50%. Similar effects are reported with amphetamine abuse.

In the prenatal period, maternal cocaine abuse, with the ensuing peripheral and central vasoconstriction, creates vasoconstriction in the placenta, leading to decreased blood flow to the fetus. If the vasoconstriction is severe enough, it can cause periods of anoxia for the fetus, but there appears to be no overall effect on intrauterine growth of the fetus. However, it has been reported that the newborn may be unable to maintain adequate homeostatic control during the neonatal period.

Cocaine abuse early in pregnancy has been associated with an increased incidence of cryptorchism and hydroureteral in the neonate. There also is a report of cerebral infarction in a term infant in a case where the mother had inhaled five grams of cocaine prior to presentation for delivery. The neonate tested positive for cocaine in the urine, indicating the transfer of cocaine across the placenta.

Maternal cocaine abuse has been associated with an increased incidence of spontaneous abortion. Abuse during the third trimester can lead to contractions, probably induced by the increased levels of norepinephrine. Increases in fetal activity have been noted within minutes of intravenous cocaine use.

The most commonly reported effect of maternal cocaine abuse (inhaled and injected) is abruptio placentae. This effect is thought to be related to the hypertension and vasoconstriction associated with cocaine. Maternal amphetamine abuse also has been linked to serious intraanesthetic complications; Samuels reported one incidence of cardiac arrest during a cesarean section, and Smith reported a case of pulmonary edema under general anesthesia during delivery.

Signs and symptoms of cocaine intoxication include emotional lability, paranoia, loss of appetite, mental impairment, dilated pupils, hypertension, tremors, convulsions and marked hyperpyrexia. This hyperpyrexia is the result of vasoconstriction, increased muscle activity and an altered response of the CNS temperature regulatory center. Succinylcholine should be avoided in patients with such symptoms.

Anesthetic implications related to cocaine abuse must focus on the duration (acute or chronic) of use, the recency of use and the quantity involved. Chronic abusers will develop a depletion of catecholamines, which may decrease their anesthetic needs. In the acutely intoxicated patient, the opposite is true; because of the increased levels of circulating catecholamines, the need for greater concentrations of halothane has been reported. Epinephrine-induced arrhythmias occur at much lower doses in the intoxicated patient.

Traditionally, propranolol has been the treatment of choice for cocaine-associated arrhythmias. Recently, studies in rats have suggested other possibilities. In one study, amitriptyline (10 mg/kg) prevented sudden cardiac arrest associated with cocaine. In another study, calcium channel blockers significantly increased survival time after toxic cocaine injections.

Narcotic use

Narcotic abuse is a continuing problem in our society. As with all abused substances, narcotics cut across all demographic boundaries.

Narcotic abuse presents a myriad of problems in the parturient and her offspring. The most common maternal complication is pregnancy induced hypertension (PIH). The most common neonatal complications are decreased birth weight and the neonatal withdrawal syndrome.

Poor prenatal care may account for some postpartum problems. If a narcotic-addicted parturient is identified early in her pregnancy, it is recommended that she undergo psychological counseling, be placed in a methadone program and be educated regarding nutrition, maternal changes, the process of labor and delivery and anesthetic care. No matter what the prenatal treatment, low birth weight still remains a problem; however, the incidence of PIH may be reduced with proper prenatal care and the use of methadone.

Heroin addiction. During fetal life, heroin has been associated with chromosomal breakage and intrauterine growth deficiency. Respiratory depression, hypoxic episodes, growth deficiency, decreased mental and neurological ability and a higher incidence of mortality and morbidity all are present during the neonatal period and may be carried into childhood.

Methadone can be found in both amniotic fluid and breast milk and has been associated with chromosomal damage during the prenatal period. There also is a higher incidence of breech presentations associated with methadone therapy. Once delivered, these neonates have smaller head circumferences, are more depressed, have problems with interactive behavior and homeostatic controls, may exhibit the narcotic abstinence syndrome and have a higher mortality rate overall.
Signs and symptoms of neonatal narcotic withdrawal may appear within 12 to 14 hours when associated with maternal heroin abuse and may appear within one to seven days, if the mother has been on methadone therapy. The symptoms may persist for up to four months. The predominant manifestation in such neonates is autonomic hyperirritability. In acute cases, tremors, restlessness, hyperactive reflexes, increased muscle tone, tachypnea, gastrointestinal irritability and a high-pitched cry commonly are noted. These signs may be misdiagnosed as meningitis, gastroenteritis, hypocalcemia, hypoglycemia or intracranial hemorrhage. Treatment for these infants includes administration of paregoric and phenobarbital. Chlorpromazine, methadone and diazepam also have been used effectively.20

Maternal manifestations of the withdrawal syndrome start approximately four to six hours after the last dose, reach their greatest intensity at about 72 hours and are gone within 10 days. The signs and symptoms of heroin withdrawal include anxiety, craving, yawning, lacrimation, rhinorrhea, piloerection, tremors, hot and cold flashes, muscle and bone aches, anorexia, insomnia and nausea. In addition the heart rate, blood pressure and respiratory rate all are increased. At the peak of the syndrome, the patient presents in the fetal position; vomiting, diarrhea, cramps and backaches are prominent.21

Concomitant problems associated with narcotic abuse include AIDS, endocarditis, tamponade, dysrhythmias, emboli, pulmonary edema, pneumonias, restrictive lung disease, renal disease, adrenal hypertrophy and decreased hemoglobin levels.

In some instances, it may be difficult to differentiate between narcotic abuse and mental problems. If the patient's pupils are not dilated and her skin is warm and dry, it is unlikely that the withdrawal syndrome is present.21

The recommended anesthetic technique in cases of heroin withdrawal is regional anesthesia given as early as possible. These patients should not be given narcotics if it can be avoided, although methadone may be used. The use of mixed narcotic agonist/antagonist drugs also must be avoided, lest the anesthetist find himself with a totally unmanageable patient.

Several reports have been written dealing with episodes of severe hypotension during general anesthesia in narcotic addicts. These patients all had their last dose of their preferred drug two to six hours before the onset of hypotension. The intraoperative injection of 8 mg morphine sulfate, or a dose of the patient's preferred drug (if the substance and dose are known), will abate the symptoms.22

It has been well documented that gastric emptying time is delayed during pregnancy, so it is common practice to perform a rapid-sequence induction with Selleck's maneuver when general anesthesia is to be used for delivery. The delay in gastric emptying in the substance abuser is an indication of the need for rapid-sequence induction, although regional techniques are preferred for such patients when possible.

REFERENCES


AUTHOR

Jay Horowitz RN. is a senior student at the School of Nurse Anesthesia, Mt. Sinai Medical Center, Cleveland, Mr. Horowitz holds a BSN from Kent State University. Prior to enrollment in anesthesia school, he held several positions in critical care areas, with emphasis on cardiac nursing.

ACKNOWLEDGMENTS

The author wishes to thank Jack Kless, CRNA, MA; Douglas Mayers MD, PhD; Robert Kiwi, MD, and Joanne Robin for their assistance in the preparation of this article.