and no signs of rejection, pregnancy appears to be well tolerated and can be associated with favorable pregnancy outcome.

Should Hyperbaric Oxygen Be Used to Treat the Pregnant Patient for Acute Carbon Monoxide Poisoning?

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Carbon monoxide (CO) poisoning is the leading cause of fatal poisoning in the U.S., accounting for more than 3,500 deaths/year. CO is a colorless, odorless gas produced by the incomplete combustion of hydrocarbons. The most frequent sources of exposure are automobile exhaust, faulty heating systems and smoke inhalation from fires.

A 17-year old primigravida at 37 weeks' gestation was taken to the E.R. unresponsive following complaints of headache, nausea and vomiting which developed during a 3-hour ride in the backseat of the family car. Similar symptoms, to a lesser degree, were experienced by the two other occupants of the car. She was obtunded but arousable. Physical examination was unremarkable except that the fetal HR was 170 beats/min with decreased beat-to-beat variability and terine contractions occurred every 6 min. While heathing 10 l/min of O₂ by face mask, ABG values **howed a PO₂ of 229, PCO₂ 29, pH 7.34, B.D. 8 mEq/l,** \mathbf{BCO}_3 16 mEq/l, O_2 saturation (Co-oximeter) 52%, and **carboxyhemoglobin** (COHb) 47%. The patient was **miented** to person and time but not to place. Because this neurologic involvement and the apparent fetal Extress, she was treated with $100\% O_2$ at 2.4 atmopheres absolute for 90 min. Ten minutes after the leginning of treatment, FHR decreased to 130-140 heats/min, the uterine contractions and the woman's

complaints of headache and chest pain disappeared. After completion of treatment, COHb was 2.4% which is in the normal range for pregnant women. Five weeks later, the patient delivered a healthy baby with normal neurologic findings. Follow-up examinations at 2 and 6 months of age revealed normal growth and development.

CO is produced endogenously by breakdown of heme pigments. During pregnancy, this is increased due to augmented red cell turnover and production by the fetus. Fetal COHb levels are 10-15% higher at steady state than maternal. In addition, the fetus is more vulnerable than the mother to the hypoxic effects induced by CO. As fetal COHb levels rise, the left-shift of the fetal oxyhemoglobin dissociation curve is increasing thereby exaggerating the effect of hypoxia.

The use of hyperbaric oxygen during pregnancy is controversial because of possible adverse reactions by the fetus induced by high O_2 partial pressures. However, animal studies and human clinical experience, particularly in Russia, have shown that the short duration of hyperoxic exposure attained during therapy for CO poisoning can be tolerated by the fetus in all stages of pregnancy and reduces the risk of death or deformity to mother and fetus.

Thus, hyperbaric oxygen therapy is advocated for the pregnant woman with CO poisoning, if her COHb level is above 20%, if she demonstrates any neurologic signs regardless of the COHb level and if signs of fetal distress are present. If hyperbaric treatment is not available, 100% O₂ by tight-fitting mask should be administered for 5 times as long as needed to reduce the maternal COHb to normal.