FETAL ALCOHOL SYNDROME AND OTHER ALCOHOL-RELATED BIRTH DEFECTS

THIS MAY BE USED AS SUPPLEMENTARY INFORMATION FOR THE TRANSPARENCIES AND POWER POINT PRESENTATION

AN INTRODUCTION TO THE PROBLEM OF FETAL ALCOHOL SYNDROME (FAS) AND OTHER ALCOHOL-RELATED BIRTH DEFECTS (ARBDs)

When a mother drinks, her unborn child is exposed to alcohol. As opposed to a common misconception, the baby is not protected in the uterus from alcohol exposure. Excessive drinking by the mother at any time after fertilization of the egg may result in damage to the developing child.

The problem of Fetal Alcohol Syndrome (FAS) and other alcohol-related birth defects (ARBDs) is very large. In fact, **maternal alcohol consumption is the leading known cause of mental retardation in the Western world (transparency #1)**. Although the range of intellectual deficits is wide, the average IQ of individuals with FAS is approximately 70.

The prevalence of FAS is typically quoted as 1 in 750 live births in the general population. However, the reported incidence varies, depending on the study population and design. The incidence of FAS currently exceeds that of Down Syndrome, spina bifida, as well as cerebral palsy. The incidence of all ARBDs, including FAS, is estimated to be 1 in 100 live births in the general population (www.nofas.org, 2004).

In spite of the fact that FAS and other ARBDs can be prevented by women simply avoiding alcohol consumption throughout their pregnancies, the problem remains.

FETAL ALCOHOL SYNDROME (FAS)

FAS is one of several consequences of maternal alcohol abuse. A diagnosis of FAS is based on the combination of 1) prenatal or postnatal growth deficiency or both (weight or length or both below the 10th percentile when corrected for gestational age), 2) central nervous system disorders including neurological abnormality, developmental delay, intellectual impairment, and structural abnormalities, and 3**) a distinctive pattern of facial anomalies, including short palpebral fissures, a thin upper lip, an elongated, flattened midface, and an indistinct philtrum (transparencies #2 &3).**

Recently, **magnetic resonance imaging (MRI) has made it possible to examine the brains of living individuals with FAS. Scientists have discovered that the brains of individuals with FAS illustrate specific structural abnormalities (transparency #4).** In addition to deficiency in the corpus collosum, other areas of the brain including the basal ganglia and the rostral portion of the cerebellar vermis have been shown to be structurally deficient. These deficiencies are not reparable. The effects of maternal alcohol abuse last a lifetime!

ALCOHOL-RELATED BIRTH DEFECTS (ARBDS)

FAS represents only a fraction of the consequences of maternal alcohol abuse. Prenatal alcohol exposure leads to a continuum of reproductive health effects ranging from infertility, miscarriage and stillbirth to low birth weight, prematurity, and a variety of neurobehavioral deficits in addition to physical malformations. Individuals with ARBDs may not have all of the features noted in those with full blown FAS (i.e. they have fetal alcohol effects, FAE). They frequently have attention deficits, language difficulties, learning disabilities, behave impulsively and have poor judgment. Attention deficits are

the most consistent neurobehavioral effect of prenatal alcohol exposure observed in older children. Cognitive deficiencies and other alcohol-related neurobehavioral deficits are the most common, but least diagnosed sequellae associated with prenatal alcohol exposure. Behavioral and mental problems of children with FAE can be just as severe as those with FAS. Many children with FAS/FAE are not able to understand cause and effect relationships and long-term consequences. These characteristics predispose the affected individuals to delinquency.

THE BIOLOGICAL BASIS FOR FAS AND OTHER ARBDS

The presentation of alcohol-induced prenatal damage is dependent on the timing of the insult and the amount and pattern of maternal drinking (peak blood alcohol concentration). Although excessive alcohol exposure can cause damage at all stages of in utero development, the embryonic period (weeks 3-8 after fertilization) is the time when the conceptus is most susceptible to the development of major abnormalities. This is not surprising considering that during this short period of time, the embryo changes remarkably in form. It transforms from a “worm-like” shape the size of Roosevelt’s ear on a dime at four weeks of development to the distinctly human form that is the size of a quarter by the eighth week after fertilization. At early stages, animals such as rodents that researchers use in order to study the effects of alcohol, are very similar in their development to humans. This is one of the reasons that the results of experiments on these laboratory animals can be extrapolated to humans. In fact, **exposure of mouse embryos to a high dose of alcohol at the time of development that corresponds to the third week of human development (when the human embryo is about the size of the inside of the 9 in the year “1994” on the dime) can cause all of the facial features in mice that are typical of children with FAS (transparency #5).**

Research has shown that **alcohol can kill cells of the developing embryo. Different cells of the embryo are sensitive at different stages of development. Some of the cells in the developing brain and face are particularly sensitive (transparency #6).** Finding the pattern of cell death caused by ethanol at various stages of development provides important clues for understanding the long-term consequences of maternal alcohol abuse in affected humans.

Research in animals has made it clear that alcohol can be very damaging even at very early stages of development. In fact, at some of the early, vulnerable stages, most women are unaware that they are pregnant.

DOSAGE AND WARNINGS

Everyone wants to know “how much is too much”. Although alcohol-related birth defects are believed to be induced in a dose-response manner, low dose effects are very difficult to scientifically assess in human populations. Whether there is a threshold below which alcohol can be consumed without harming the conceptus is not known. Also, due in part to individual variability (susceptibility), research will not be able to provide an accurate answer for everyone. To date, studies indicate that most neurobehavioral effects can be caused by a pregnant woman drinking from 0.5 to 2 ounces of absolute alcohol per day (7 – 28 drinks with each containing 0.5 ounces of absolute alcohol per week; an 8 oz. can of beer contains the same amount of alcohol as a glass of table wine or a serving of fortified wine or a 1 oz. shot of liquor). Although this would indicate that even one drink per day can cause measurable consequences to the offspring, the drinking patterns of many of the women studied were such that the majority of the drinks consumed in one week were on only one or two occasions, rather

that one drink each day. It is expected that self-reported data showing a relationship between moderate use and alcohol-related birth defects may often underestimate the true level of drinking. High peak blood levels of alcohol are important predictors of adverse outcome. Binge exposures (at least 5 standard drinks on any occasion) result in a greater frequency of neurological sequellae than the same amount of alcohol distributed across a greater time course.

Certainly, the best advice is to totally abstain from alcohol use during pregnancy, even at stages prior to the time that pregnancy is recognized. Although some clinicians believe that recommending total abstention for pregnant women may subject them to unwarranted guilt about drinking small amounts of alcohol, most accept the need for clinical caution. **Because it is not known at what dosage alcohol damage begins, it is prudent to recommend that pregnant women abstain from alcohol use (transparency #7).** In spite of public warnings the Centers for Disease Control reports a fourfold increase in frequent drinking from 1991-1995 among pregnant women

In addition to considering consequences of alcohol exposure prior to birth, it is also important to note that alcohol that a lactating mother consumes is present in her milk and may affect the brain of her nursing infant.

ARBDs are expensive. For one person with FAS, the institutional and medical costs over a lifetime have been estimated to be between $800,000 to $2 million (www.nofas.org, 2004; and Addiction Biology, Vol 9., No. 2, 2004). Recent estimates indicate that total annual costs of care to US society are as high as $7.8 billion for individuals with FAS (Addiction Biology, Vol 9., No. 2, 2004).

PREVENTION

The traditional ARBD prevention approach of focusing intervention efforts during pregnancy is after much damage has already occurred. Physicians and other health care providers should encourage sexually-active childbearing age alcohol consumers to prevent pregnancy or to avoid any alcohol use during preconceptional and prenatal periods.

Ideally, appropriate screening of all childbearing age patients for alcohol use combined with preconception health promotion, contraceptive counseling, and referral to substance abuse programs for high risk consumers should become a routine standard of care in primary care settings. Serving this role, physicians and other health care providers will play a critical role in the primary prevention of FAS and other ARBDs.

Education of physicians, other health care providers, parents, and prospective parents is essential!!