THE NEURODEVELOPMENTAL CONSEQUENCES OF PRENATAL ALCOHOL EXPOSURE

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ABSTRACT

During pregnancy, ingestion of alcohol, a known teratogen, can cause harm to the fetus. Prenatal alcohol exposure is one of the leading causes of birth defects, developmental disorders, and mental retardation in children. The fetal central nervous system is particularly vulnerable to alcohol; this vulnerability contributes to many of the long-term disabilities and disorders seen in individuals with prenatal alcohol exposure.

Diagnoses associated with prenatal alcohol exposure include fetal alcohol syndrome (FAS), partial fetal alcohol syndrome, fetal alcohol effects, alcohol-related neurodevelopmental disorder, and alcohol-related birth defects. Once diagnosed, early intervention improves the long-term outcome of affected children. Without documentation of maternal alcohol use, a diagnosis, and consequently treatment, is often difficult to attain. It is imperative that nurses, physicians, and other healthcare providers become comfortable with obtaining a history of, and providing anticipatory guidance and counseling about, alcohol use.

KEY WORDS: prenatal alcohol exposure, fetal alcohol syndrome (FAS), alcohol-related neurodevelopmental disorder, alcohol-related birth defects, early intervention, fetal alcohol effects, family education.

The detrimental effects of maternal ingestion of alcohol during pregnancy have been scientifically documented since the late 1800s.[1] It was not until 1973 that a seminal article published in the *Lancet* by Jones et al[2] labeled the dysmorphology observed in the offspring of alcoholic mothers as fetal alcohol syndrome (FAS). Since that publication, there has been tremendous research on the impact of prenatal alcohol exposure on the developing fetus, infant, and child.

Despite the well-documented teratogenic effects, prenatal alcohol exposure (PAE) continues to be a widespread public health concern. The Centers for Disease Control and Prevention (CDC) report that in 2002 the rate of alcohol use among pregnant women was 10.1%.[3] In pregnant women, binge drinking, defined as 5 or more drinks on 1 occasion, and frequent alcohol use, defined as 7 or more drinks in a week, were both 1.9%.[3] It is estimated that annually 130,000 pregnant women in the United States drink alcohol at levels that may put their fetus at risk of developing alcohol-related disorders.[4] The incidence of FAS in the United States is estimated to be 1 to 2 per 1000 births.[5] The combined estimated numbers of FAS, alcohol-related neurodevelopmental disorder (ARND), and births affected by alcohol-related birth defects (ARBD) is 10 per 1000.[5]

Around the world, PAE is a public health issue as well. The Western Cape province of South Africa has a reported FAS rate of 40 to 46 per 1000 births.[6] A study in Moscow examined 2 groups of children, 1 from a boarding school and the other from an orphanage. These groups were chosen because of their probable exposure to alcohol. The incidence rate of FAS within this small population was estimated to be 14.1%.[7] The incidence rate of FAS in Europe is approximately 0.8 per 1000 births.[8]

The lifetime cost of care for one individual with FAS in the United States is approximately $2 million.[9] In 1998, it was estimated that over $4 billion was spent nationally caring for individuals with FAS.[9] These cost estimates do not include the costs associated with indi-

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Table 1. Definition of Terms at a Glance[1,59,64]

ARBD[59]	**Alcohol-related birth defects** • No facial features of FAS • Known prenatal alcohol exposure • Birth defects associated with alcohol exposure
ARND[59]	**Alcohol-related neurodevelopmental disorder** • No facial features of FAS • Known prenatal alcohol exposure • Must have CNS abnormalities exhibited by 1 of the following: microcephaly, abnormal brain structures, behavioral or cognitive disorders
FAE[1]	**Fetal alcohol effects** • Known prenatal alcohol exposure • Absence of the facial characteristics of FAS
FAS[59]	**Fetal alcohol syndrome** • Presence of facial features (small palpebral fissures, thin upper lip, and a smooth philtrum) • Growth restriction and CNS abnormalities • Confirmed alcohol exposure may or may not be present
FASD[64]	**Fetal alcohol spectrum disorder** • Describes individuals who have a disorder or disability related to prenatal alcohol exposure • Not to be used as a diagnostic criterion
PFAS[59]	**Partial fetal alcohol syndrome** • Some of the facial features of FAS • Known prenatal alcohol exposure • May have some behavioral issues, such as poor impulse control, and some cognitive deficits
PAE	**Prenatal alcohol exposure** • Designates exposure to alcohol in utero • Does not indicate degree of exposure or if individual has an alcohol-related disorder

Abbreviations: CNS, central nervous system; FAS, fetal alcohol syndrome.[1]

viduals diagnosed with ARND, ARBD, or fetal alcohol effects (FAE).

Prenatal alcohol exposure is the leading preventable cause of birth defects, developmental disorders, and mental retardation in children.[10] Yet evidence of screening for alcohol use in women of child bearing age and documentation in the records of infants with known exposure continue to be inadequate.[11,12] Possible reasons for this are healthcare providers'

- Discomfort discussing the subject with pregnant women;
- Lack of knowledge about the effects of alcohol on the developing fetus, and;
- Lack of awareness of interventions once alcohol use has been discovered.[13]

Since FAS, ARND, and ARBD are completely preventable, screening women of childbearing age for alcohol use is a critical first step. The long-term outcomes of affected children are improved with early diagnosis and early intervention. Caregivers in the newborn and/or intensive care nursery play a pivotal role in identifying, documenting, and providing guidance to those at risk.[14]

Definition of Terms

The term *fetal alcohol syndrome* first appeared in *Lancet* in 1973.[2] It was used to describe a combination of abnormalities that included craniofacial, extremity, and cardiovascular anomalies, as well as growth deficits and developmental delays in 8 unrelated children born to alcoholic mothers.[2] Further research revealed that not all children with PAE develop the physical characteristics of FAS. By 1978, the term *fetal alcohol effects* (FAE) was commonly used to describe children who had some, but not all, of the characteristics of FAS. Table 1 provides an overview of key terms related to FAS.[1]

In 1996, the Institute of Medicine created a 5-category classification system for individuals exposed to alcohol in utero.[15] This classification system added the terms partial FAS (PFAS), ARBD, and ARND, to describe those children who did not precisely fit the FAS category, but had deficits or disabilities that could be linked to PAE.[15]

Recently, the term *fetal alcohol spectrum disorder* (FASD) has been used to include all categories of PAE, including FAS; however, FASD is not intended to serve as a clinical diagnosis.[15,16] A diag-

nosis of FAE, ARND, PFAS, or ARBD does not preclude significant organ damage, often to the brain, equal in severity to the damage seen in the individual with full-blown FAS.[17-20]

Alcohol: A Potent Teratogen

Alcohol is a potent teratogen, capable of causing serious harm to the fetus. Because alcohol causes central nervous system (CNS) damage, it is also classified as a neurobehavioral teratogen.[1,21] Persistent CNS damage is the most debilitating feature of PAE across the lifespan.[18]

The embryo and fetus are dependent on the maternal liver to metabolize alcohol.[22] Alcohol crosses the placenta readily; the embryo and later the fetus are exposed to the same levels of alcohol as in the maternal bloodstream.[22] The timing of exposure determines how and which cells are affected.[23]

Alcohol can cause cell death by both necrosis and apoptosis in the developing embryo and fetus.[23-28] Death of a cell line can affect production, migration, and differentiation of future cell lines.[26] Cells in the CNS have a lower threshold for alcohol and, hence, experience more rapid cell death than other cells in the developing embryo.[26] Consequently some individuals may not have the facial characteristics of FAS, yet have significant CNS damage attributable to alcohol exposure.[24,25]

Another probable contributing factor to FAS is oxidative stress.[29-31] Oxidative stress occurs when the rate of free-radical production exceeds the ability of the cells to detoxify or eliminate them.[32] There are 2 mechanisms by which this stress may occur. First, a byproduct of the metabolism of alcohol is free radicals; in particular, oxygen-containing free radicals referred to as reactive oxygen species.[32] Second, alcohol consumption suppresses antioxidants that are necessary for free-radical elimination.[33] The combination of increased free-radical production and decreased free-radical elimination can cause toxic levels of free-radical exposure, leading to mitochondrial dysfunction, cell damage, and cell death.[34] Some animal and in vivo studies have shown that treatment with antioxidants can reduce the degree of cell damage and death.[33-37]

Alcohol may further interfere with growth factors necessary for normal CNS development. This has been shown in several studies on insulin-like growth factors (IGF) I and II.[38-40] Ordinarily, IGF I and II bind with an IGF receptor on the neuron and a message is sent to stimulate cell division. In the presence of alcohol, the receptor site becomes dysfunctional and the message is never sent.[41] Furthermore, for nondividing nerve cells, IGF I and II receptors are important for maintenance of cell life. Their function is impaired in the presence of alcohol.[42]

Glial cells, important to guide the migration of neurons to their final destination in the brain, are adversely affected by alcohol.[43] In the normal brain, glial cells become astrocytes after all neuronal migration is complete. However, glial cells exposed to alcohol become astrocytes prematurely and remaining neurons are unable to migrate to their proper locations.[44] This explains why some neurons in the alcohol-exposed brain are not in their usual location.[44,45]

The neurotransmitters serotonin and glutamate, both important for fetal brain development, are adversely affected by alcohol exposure. Alcohol appears to delay the development of the serotonin system. Without serotonin, growth-factor–releasing astrocytes are not stimulated and normal brain development does not occur.[46,47] Glutamate is an important excitatory neurotransmitter that interacts with several receptors to control brain function. One of these receptors is N-methyl-D-aspartate (NMDA).[23] Alcohol exposure can reduce the number and functionality of the NMDA receptors, further affecting other neurotransmitter systems.[23] These reductions may cause some of the CNS disorganization commonly seen in FAS.[23]

Exposure of fetal cells to alcohol affects glucose uptake. In cell culture studies, the glucose transporter protein GLUT1, found in the brain, was decreased after alcohol exposure.[48] Because glucose metabolism is imperative to brain development and growth, the reduction in glucose uptake may be a major contributor to CNS deficiencies.[48]

The regions of the brain that are affected by alcohol exposure are the basal ganglia, corpus callosum, cerebellum, and, to some degree, the hippocampus.[49-52] These areas of the brain affect motor and cognitive skills, learning, memory, and executive functioning.[53] Deficiencies in the area of executive functioning are often the most devastating during adulthood; the ability to solve problems, plan for the future, and understand abstract concepts such as time and money can all be affected.[53-55] An individual's intelligence quotient is not a direct correlate of executive functioning.[53,54]

The causes of FAS, ARND, and ARBD seem to be multifactorial. Individual genetic makeup plays a part in how alcohol exposure manifests.[1] Dizygotic twins are often affected differently.[56] Furthermore, the frequency, amount, and type of maternal drinking (binge versus frequent low-dose) affect the fetus differently.[57,58] Finally, advanced maternal age, poor maternal health, and comorbid behaviors such as smoking and/or drug use may make the effects of alcohol more potent.[17,57] No studies have determined if there is a safe threshold of alcohol that can be consumed during pregnancy. Current recommendations strongly suggest that pregnant women abstain from alcohol consumption.[10]

Diagnostic Classification

The 5 diagnostic categories of PAE are summarized in Table 2. In all but one, a history of maternal alcohol use needs to be documented. The first category is FAS with confirmed maternal alcohol use. In this category, the individual displays the typical facial characteristics—

Table 2. Institute of Medicine's Diagnostic Criteria for Fetal Alcohol Syndrome/Alcohol-Related Neurodevelopmental Disorder/Alcohol-Related Birth Defects[59]

Diagnosis	Facial Features of Fetal Alcohol Exposure	Maternal Alcohol Use: Regular or Heavy Binge Drinking	Other Information
FAS with known maternal alcohol use	**Yes**	**Yes**	Infants exhibit growth restriction in 1 of 3 areas: small for gestational age, failure to thrive not due to poor nutrition, low weight to height; CNS abnormalities in 1 of 3 areas: small cranial size, brain abnormalities, or neurological abnormalities
FAS with unknown maternal alcohol use	**Yes**	**No**	Same as above
Partial FAS with known maternal alcohol use	**Some**	**Yes**	Same as above, but also includes behavioral or cognitive abnormalities that cannot be explained by environment alone; may include poor impulse control, inability to understand abstracts, poor performance in school
Alcohol-related birth defects (ARBD)	**No**	**Yes**	Anomalies associated with prenatal alcohol exposure include cardiac, skeletal, renal, ocular, and auditory defects
Alcohol-related neurodevelopmental disorder (ARND)	**No**	**Yes**	CNS abnormalities, microcephaly, abnormal brain structure, behavioral or cognitive disorders attributable to prenatal alcohol exposure and not explained by genetics or environment

Abbreviations: CNS, central nervous system; FAS, fetal alcohol syndrome. Modified from Institute of Medicine 1996 diagnostic criteria for FAS.

smooth philtrum, thin vermillion, and small palpebral fissures—along with growth deficiency and CNS abnormalities.[59] The second category is referred to as FAS without confirmed maternal alcohol use.[59] The third category defines PFAS. In this case, the individual has some of the facial features, growth deficiency, and CNS abnormalities, as well as a documented history of maternal alcohol use. The last 2 categories, ARND and ARBD, also require documented maternal alcohol use for diagnosis.[59]

In July 2004, the CDC published *Fetal Alcohol Syndrome: Guidelines for Referral and Diagnosis*, a document that applied to only FAS. Based on these guidelines, 3 criteria must be met for the diagnosis of FAS (Table 3). These include the 3 standard facial abnormalities associated with FAS, documentation of growth deficits, and documentation of CNS abnormalities.[60]

Challenges in and Timing of Diagnosis

Although FAS can be diagnosed at birth, it rarely is for several reasons. First, some of the defining facial features of FAS such as small palpebral fissures, are difficult to detect in the neonate.[20] Second, some pediatricians do not feel competent to make the diagnosis.[12] Third, there is hesitation to assess a newborn for FAS, even when there is known maternal alcohol use, because to do so would label the mother as an alcohol abuser.[12] Finally, there is a lack of documentation in medical records of maternal alcohol use, likely because healthcare providers do not have a full understanding of the spectrum of deficits attributable to PAE.[12] This is extremely problematic because 4 of the 5 diagnostic categories for PAE require documentation of alcohol use.[12,15]

The diagnosis of FAS, PFAS, ARND, or ARBD usually occurs later in infancy or in early childhood.[1,60] It is during this time period that the facial features of FAS are most evident and behaviors typical of PAE begin to manifest.[60] Often the CNS manifestations of PAE lead to an evaluation and ultimately a diagnosis (Fig 1). Infants may be irritable and fail to meet developmental milestones; young children may exhibit hyperactivity, poor fine-motor control, and/or mental retardation; school-age children may have difficulty following directions and poor impulse control, causing

Table 3. Centers for Disease Control Criteria for Fetal Alcohol Syndrome Diagnosis With Confirmed or Unknown Maternal Alcohol Use[59]

Facial Dysmorphia +	Growth Problems +	Central Nervous System Abnormalities
Must have all 3 features: • **Smooth philtrum** University of Washington Lip to Philtrum Guide rank 4 or 5 • **Thin vermillion** University of Washington Lip to Philtrum Guide rank 4 or 5 • **Small palpebral fissures** At or below the 10th percentile	Confirmed prenatal height, weight, or both, at or below the 10th percentile, documented at any 1 point in time.	Must have 1 of the following: **1. Structural** Head circumference at or below 10th percentile, adjusted for age and sex. **2. Neurological** Neurological problems not due to postnatal insult or fever, or other soft neurological sign outside normal limits. **3. Functional** Performance substantially below that expected for an individual's age, schooling, or circumstances as evidenced by: Global cognitive or intellectual deficits representing multiple domains of deficit with performance below the 3rd percentile. Functional deficits below the 16th percentile in at least 3 of the following areas: • Cognitive or developmental deficits • Executive functioning deficits • Motor functioning delays • Problems with attention and hyperactivity • Social skills • Sensory problems, pragmatic language problems, memory deficits.

NOTE: Diagnostic criteria recommended by the Centers for Disease Control for fetal alcohol syndrome. There are no discrete categories for alcohol-related neurodevelopmental disorder or alcohol-related birth defects. See Fig 1 for framework for diagnosis and follow-up. Reprinted with permission.

alarm in their parents, healthcare providers, and teachers.[1,60]

The Importance of a Complete History

Thorough prenatal, birth, and postnatal histories are helpful in the diagnosis of FAS, ARND, or ARBD. Foster and adoptive families may not know the child's complete medical history and often need to rely on records from medical professionals, social workers, and adoption agencies. Currently, children who do not have the classic facial features of FAS and do not have documentation of PAE have difficulty receiving an alcohol-related diagnosis. To address this, the University of Washington has developed a more precise 4-digit diagnostic code system. This system, which uses specific criteria to identify individuals with PAE, is used as a screening tool for all children entering the foster care system in Washington state.[62]

Maternal history is an important component in determining if an infant has had PAE. From the history, the examiner can assess maternal risk factors.[28] This assessment is particularly important for the infant who is in the well-baby nursery and shows no signs or symptoms of alcohol exposure. Fetal alcohol syndrome, ARND, and ARBD occur in all racial, cultural, and ethnic groups; however, there are infants who are at higher risk. These risk factors are associated with maternal factors including:

- Age >25 years;
- Documented high blood-alcohol concentration;
- History of alcohol abuse and/or other substance abuse;
- History of living with an alcohol abuser;
- Low socioeconomic class;
- Low self-esteem;
- Loss of children to a foster care system;
- Single status;
- More than 3 children;
- A previous child with FAS;
- Unemployment and social transience[15,17,64]

Anytime a mother has a known history of illegal drug use, alcohol use should be explored, because polydrug use is common.[64]

The infant or child's history is equally important for diagnosis. A history of growth deficits, developmental delays, or CNS abnormalities are suspect for alcohol exposure.[15]

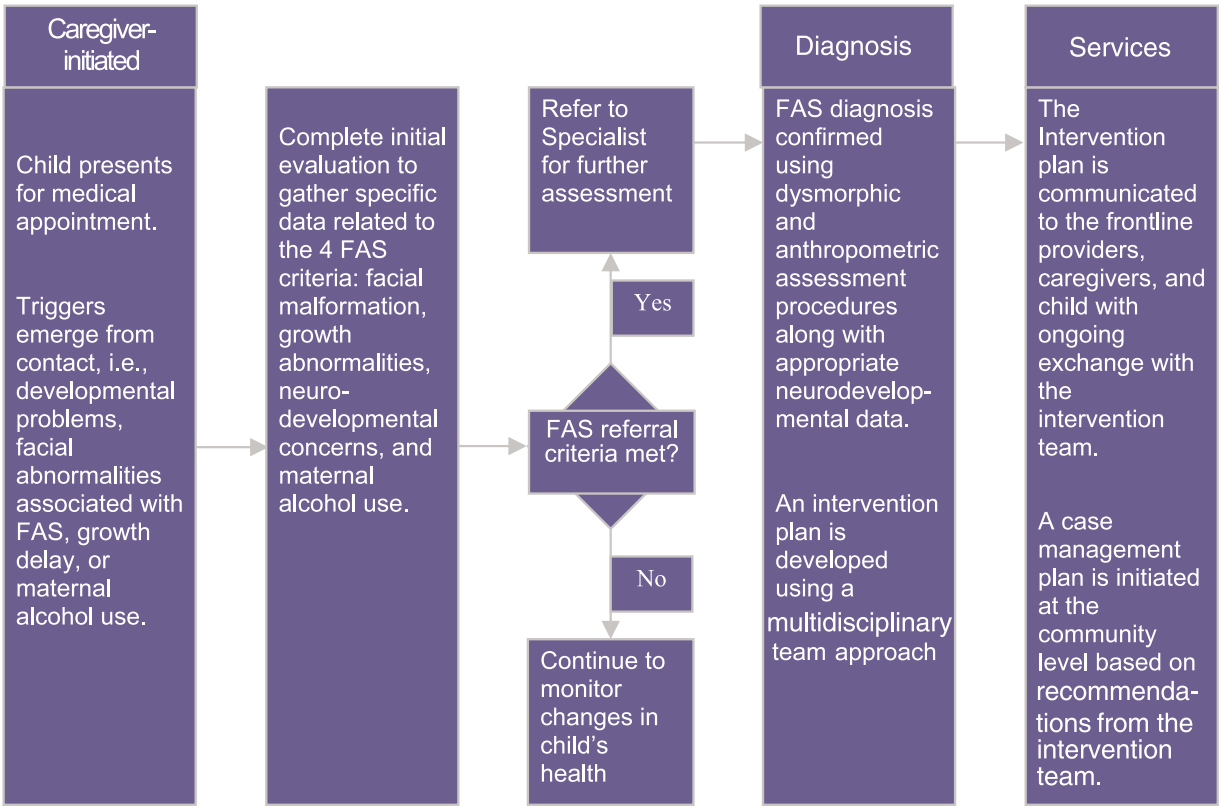

Figure 1. The Centers For Disease Control and Prevention's framework for fetal alcohol syndrome (FAS) diagnosis and follow-up care. Reprinted with permission.[8,9]

The diagnosis of FAS is based on a clinical examination.[1,15] The philtrum and the vermillion are compared to a University of Washington standard key. Palpebral fissures are measured and compared to standards.[60] The classic facial characteristics of FAS are a manifestation of early exposure; a fetus exposed after the first trimester may not exhibit the typical face of FAS, yet may still have significant neurological damage.[15,20] Head circumference at birth and throughout early childhood is documented as this is one of the simplest means of measuring abnormal brain growth (Fig 2).[1]

Other professionals who may be included in the diagnostic process include a geneticist, a medical social worker, a psychologist, a physical therapist, an occupational therapist, and a speech and language therapist. Together these disciplines collect data to determine if the child exhibits the physical, neurodevelopmental, and/or cognitive signs and symptoms associated with FAS or an FASD.[65]

Primary and Secondary Disabilities

For individuals who have PAE, receiving a diagnosis is paramount. A formal diagnosis is the eligibility passcard that unlocks the doors to early intervention, the most effective treatment. Prenatal alcohol exposure can cause significant neurological damage. This damage manifests as primary disabilities and cognitive or behavior deficits that can be directly attributed to maternal alcohol use. Examples of primary disabilities are mental retardation, hyperactivity, memory difficulties, perseveration, attention deficit disorder, poor judgment, impulsiveness, and difficulty with abstract concepts and problem solving.[1]

Secondary disabilities are the long-term behavioral issues often associated with FAS. Six secondary disabilities commonly observed are mental health problems, disrupted school experience, trouble with the law, imprisonment, inappropriate sexual behavior, and substance abuse problems.

Early diagnosis, early intervention, and a stable home decrease the risk of secondary disabilities in individuals with PAE.[14] These factors are particularly important for individuals with normal intelligence, because they generally do not qualify for special education and other traditional services.[14]

Assessing for Signs and Symptoms of Alcohol Exposure

Some infants may be asymptomatic and display no signs of exposure. Others may present with irritability, hypertonia or hypotonia, opisthoto-

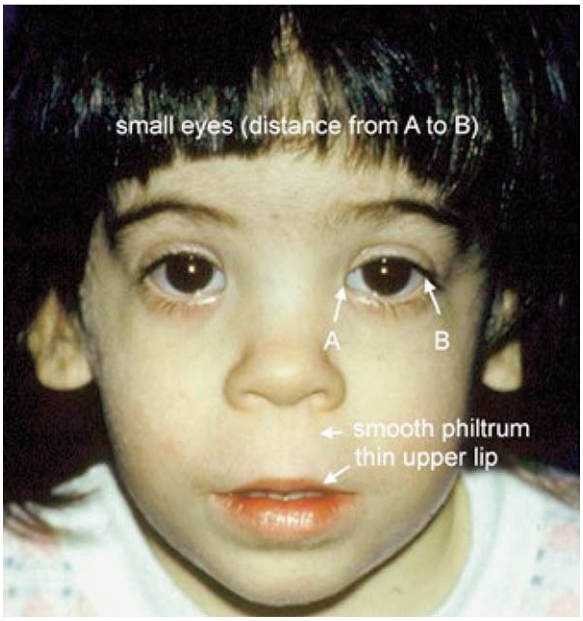

Figure 2. The typical facial features of a child with fetal alcohol syndrome. Courtesy of Susan Astley, PhD. Photo © 2005 by The University of Washington.

nus, tremors, poor feeding, poor state regulation, poor habituation, and electroencephalographic (EEG) changes.[1] There may be differences in the threshold, latency, and pitch in the cry of infants with PAE.[66,67] Infants withdrawing from alcohol may have seizures.[1]

A number of laboratory tests currently under investigation may prove to be helpful in identifying exposed infants. Fatty-acid ethyl esters, a byproduct of ethanol metabolism, can be found in the meconium of ethanol-exposed infants during the second trimester. This test is not helpful in cases in which exposure occurred only during the first trimester of pregnancy.[68] Maternal blood can also be tested for 4 markers that have a high correlation with maternal alcohol abuse. These markers include whole-blood–associated acetaldehyde, carbohydrate-deficient transferrin, gamma-glutamyl transpeptidase, and mean red blood cell volume.[68] Women with 2 or more positive markers for alcohol use had infants with smaller head circumferences, shorter lengths, and lower weights. These blood markers are better predictors of infant outcome than self-reports of alcohol use.[68]

Obtaining a history of alcohol intake is an important, albeit sensitive, task. The accuracy of self-reports are often in question. Table 4 describes 2 screening tools that have been developed for use with pregnant women.[64,69] The T-ACE is a 4-question tool that more readily identifies women with alcohol risk than staff assessment alone; its specificity is 69%.[69,70] The TWEAK is a 5-item tool with a 79% specificity.[70] The key component of both of these tools is the reference to "tolerance." In pregnant women, questions about tolerance seem to elicit the most honest responses; there is less stigma attached to how much alcohol a person can drink before feeling drunk.[70] If maternal alcohol use is revealed, it is important to document the frequency and the amount of maternal alcohol consumption in both the mother's and the infant's charts.[12]

Table 4. Tools to Assess Maternal Alcohol Use During Pregnancy[69,70]

TWEAK[69]

1. How many drinks can you hold (**T**olerate)?
2. Does your spouse (parents) ever **W**orry about your drinking?
3. Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover (**E**ye opener)?
4. Have you ever awakened the morning after some drinking the night before and found that you could not remember a part of the evening before (**A**mnesia)?
5. Have you ever felt you should cut (**K**ut) down on your drinking?

T-ACE[70]

1. **T**olerance - How many drinks can you hold?
2. Have people **A**nnoyed you by complaining about your drinking?
3. Have you ever felt that you ought to **C**ut down on your drinking?
4. **E**ye opener - Have you ever had a drink first thing in the morning to steady your nerves or to get rid of a hangover?

NOTE: Question number 1 in both surveys is useful for determining the degree of alcohol use/abuse.

Impact of the Diagnosis

The sooner early intervention services begin the better, particularly for the child who does not exhibit the typical face of FAS.[1] Once diagnosed, families will be better able to care for their child.[1,18] That said, there is little that can prepare a family for how difficult raising a child with PAE can be. See Sidebar 1 for a personal account of parenting a child with FAS.

At the time of the intensive care nursery admission, the long-term implications of PAE may seem remote and irrelevant to many presenting parents. Families will want to believe that once the acute health problem is addressed, their child will be a typical rather than a special-needs child. As difficult as it may be for the healthcare providers to say, and for the families to hear, the impact of PAE needs to be clearly explained and reiterated over time.[1] It is imperative that parents understand that neurological damage attributable to PAE is permanent. Even

infants who appear alert and morphologically normal may develop neurodevelopmental disorders attributable to PAE.

As advocates and positive role models, parents can lessen the degree of secondary disabilities for their child.[1,18] They can protect their child from harm, utilize early intervention services, provide a constructive means for their child to express frustration and anger, remain calm during temper tantrums, and work with the school system to ensure that their child has a positive educational experience.[21,71]

Nursing Implications

An important responsibility of the intensive care nursery nurse is to obtain a thorough prenatal history and document alcohol exposure in the infant's chart.[12] For the infant who will be going home with his or her biological parents, the nurse must assess parental coping skills and identify support services and resources for the family.[63] Table 5 provides a list of excellent resources that are available for professionals and parents who care for infants with FAS or ARND.

Biological parents may need referrals to substance abuse treatment programs.[63] The biological mother may experience guilt for causing harm to her child. Alcohol abuse is often transgenerational, adding further complexity to the home environment and limiting the availability of effective social support. The mother may also have other comorbid psychiatric disorders, such as bipolar disorder, depression, post-traumatic stress disorder, or psychosis.[72] A referral to mental health services for further evaluation, counseling, or a support group may be necessary.

Children with PAE frequently become foster children.[60,72] Many placement agencies provide minimal education for foster parents about the implications of PAE. Nurses will need to teach foster parents how to care for these infants.[60,72]

Infants with PAE are often easily overstimulated. Teach the parents the infant's cues that signal overstimulation and help parents to make appropriate environmental modifications.[63] Demonstrate how to assist the infant to self-regulate, i.e., by helping the infant to return to midline by placing their infant's hands near her or his mouth.[63] Prepare parents for the potential episodes of inconsolable crying and provide them with safe coping strategies.[63] Encourage parents to ask for help if they need it. The Family Teaching Toolbox, *A Parent's Guide to Prenatal Alcohol Exposure*, on pages 230-231, may be a useful teaching adjunct.

Children with PAE have an increased risk of being insecure and developing attachment disorders.[73] Insecurity is linked to the degree of exposure to alcohol.[73] They often have difficult temperaments, making positive parental interaction difficult.[1] Provide the parents with practical methods to help the infant feel secure, such as swaddling, periods of quiet time, and opportunities for caregiving.[1,73] With positive support, the infant will be more likely to form secure attachments and coping skills.[73]

Early intervention services should begin as soon as a diagnosis is made.[60] Become familiar with services available in your region. Part C of the Individuals With Disabilities Act is a federally mandated, usually county-administered, program that provides services to children from 0 to 3 years of age. The diagnosis of FAS is a presumptive eligibility diagnosis based on future risk. This means that the infant is eligible for services even if he or she does not meet the usual eligibility criteria at the present time.[60] Physical therapy, occupational therapy, speech and language therapy, parent/infant interaction groups, and respite care may all be available.

Home visits may be indicated for medically challenging infants or when parents need additional support.[63] Home visits provide additional information about the safety and resources of home environment and how both the parents and infant are transitioning and coping with their new situation.[63]

Educate all women of childbearing age about the risks of PAE. Women who are having unprotected sex, are trying to become pregnant, or those who are already pregnant should be encouraged to abstain from alcohol use. Although it has not been determined if an occasional drink during pregnancy will definitely harm a fetus, neither has the safe versus unsafe threshold for any given fetus been determined.

Conclusions

Prenatal alcohol exposure can cause significant harm to the human fetus. It is the leading preventable cause of birth defects, developmental disorders, and mental retardation. It is a public health concern throughout much of the world. The long-term neurodevelopmental manifestations of PAE are often the most devastating for individuals both with and without the typical facial features of FAS.

Prevention of PAE is the goal. The nurse plays a part in prevention through the education of all women of childbearing age about the risks associated with PAE. Early diagnosis of FASD increases the chances of a positive long-term outcome for the child. The intensive care nursery nurse, who often has extended contact with mothers at the bedside, is well positioned to obtain a detailed history, to alert the medical team of PAE, and to ensure that the medical record accurately reflects PAE. Once a diagnosis is established, ongoing assessment of parenting skills and educational needs, anticipatory guidance, and modeling of advocacy skills, as well as

SIDEBAR 1. A PERSONAL STORY: RAISING A CHILD WITH ARND

In 1999 I brought my adoptive, then foster, daughter home from the newborn intensive care unit. Carly was 9 days old. She was placed in our home because her urine tested positive for cocaine. The social worker told us that her biological mother also had a long history of alcohol abuse. She had already begun physical therapy because she was hypertonic on her left side.

I read anything I could find about FAS; Carly did not fit the classic picture. She was alert, engaging, of normal weight, and not microcephalic. I convinced myself that her brain had suffered no injury.

Because her urine was positive for cocaine, Carly was eligible for early intervention services. She received physical therapy until she was 15 months of age for some motor delays and ongoing hypertonicity on her left side. She also attended a school for special needs children through Part C of the Individuals With Disabilities Act. By 3 years of age, her delays were negligible; however, she attended school through Part B, as she was identified as a child "at risk."

Carly, age 5 years, diagnosed with ARND.

It is hard to remember exactly when my husband and I started to notice that her temper tantrums were beyond what we had become accustomed to with our 4 other biological children. Carly would have tantrums that were impossible to deescalate. It did not matter if she was sent to her room for quiet time, the tantrum would just continue. I felt guilty and frustrated because I did not want her to be so miserable; it was easy to take it personally, as if somehow I was failing as a parent.

When Carly was almost 5, my husband and I decided to have her tested for FAS to ensure that if she did have an alcohol-related disorder, she would continue to receive services. Carly underwent a battery of examinations that included a motor-skills assessment, a psychological examination, a language-skills assessment, and a physical examination. A team specializing in alcohol-related disorders reviewed the results.

We were not surprised to find that Carly had normal intelligence. Of great surprise was that she had sentinel features of FAS, meaning she had "the face." It was the first time any medical provider had noted that she had the facial features typical of FAS. Because Carly had normal growth and did not have microcephaly, she did not meet the diagnostic criteria for FAS. Instead she received the diagnosis of ARND. I interpreted this diagnosis as a good thing and had an initial sense of relief. Then the doctor explained to me that, embryologically, the face and the frontal lobe develop at the same time. I do not know how I had missed this in all that I had read, but I had. At that moment, I felt completely overwhelmed.

Currently, my husband and I visit with a behavioral therapist once a month to discuss methods to modify our daughter's behavior. She still has temper tantrums, but we have been handling them better after the therapist explained that, in many ways, our daughter's brain is developmentally more equal to that of an 18-month-old than a 5-year-old. We sometimes wonder if she will ever be able to live on her own. Carly is smart, but she is also manipulative, lies frequently, and does not understand consequences very well. The therapist has said that Carly may always need to have some sort of external boundaries set by a mate, her siblings, friends, or us. We remind ourselves that Carly is still only 5 years old. We remain optimistic.

Having a child with an alcohol-related disorder has changed our lives. It impacts everything. We have to consider how many of the decisions we make will affect our daughter. We try to run our household of 7 by routine, because Carly functions better that way. We have learned that we must be extremely consistent with her. Every time we go anywhere, we need to consider if Carly will be able to handle it. We seldom take vacations because the change in routine can lead to her spiraling out of control.

There is a level of hypervigilance in parenting Carly that does not have to occur with our other children. Carly needs to be watched and monitored all the time, in part because she is emotionally disconnected from herself. If something happens to her, for example if someone makes fun of her on the school bus, she is unable to later verbalize any emotional response. My greatest fear as a parent is that someone might do harm to Carly, and she would be unable to communicate this to me and thus, I would never know. Another reason for hypervigilance is that Carly has little impulse control; if she wants to do something, she just does it, no matter what the rules or the possible outcomes might be. We have told everyone in our community that Carly has ARND so that they, the village, can help monitor her with us.

Despite the level of energy it takes to raise Carly, she endears herself to us. She has a wonderful laugh. She loves to take care of her brothers if they are sick. She is extremely affectionate. She can run like the wind and run forever. Carly is really an amazing child. I just wish that she had never been exposed to alcohol; life would have been easier for everyone, but especially for her.

Table 5. Fetal Alcohol Syndrome (FAS) Web Resource List

Resource and Internet Address	Description
Centers for Disease Control Fetal Alcohol Syndrome, NCBDDD, CDC Mail-stop E-86 1600 Clifton Road Atlanta, Ga 30333 Phone: 404-498-3947 http://www.cdc.gov/ncbddd/fas/default.htm	Information in English and Spanish about FAS including *Guidelines for Referral and Diagnosis*. New US Surgeon General's Advisory on alcohol use in pregnancy. New educational curricula.
FAS Community Resource Center http://www.come-over.to/FAS/	Books, videos, posters about FAS. Links to other sites. Personal stories about living with FAS. National Public Radio interview of Claire Coles about fetal alcohol syndrome (http://www.come-over.to/FAS/NPR2003.htm).
The FAS Family Resource Institute http://www.fetalalcoholsyndrome.org/index.html	Nonprofit organization committed to identifying, understanding, and caring for individuals with prenatal alcohol exposure.
Fetal Alcohol Syndrome Diagnostic and Prevention Network University of Washington, Seattle, Wash http://depts.washington.edu/fasdpn	Screening and diagnosis of FAS. Training on how to diagnose FAS will soon be available online.
Medline Plus http://www.nlm.nih.gov/medlineplus/fetalalcoholsyndrome.html	Links to publications about FAS.
National Institutes of Health, National Institute on Alcohol Abuse and Alcoholism 5635 Fishers Lane, MSC 9304 Bethesda, Md 20892-9304 http://www.niaaa.nih.gov/	Information about alcohol abuse and FAS.
National Organization of Fetal Alcohol Syndrome 901 17th St NW, Suite 910 Washington, DC 20006 Phone: 800-66NOFAS. Phone: 202-785-4585	Information for healthcare providers about screening and treatment. Information for families about living with FAS.
Substance Abuse and Mental Health Services Administration, The Fetal Alcohol Spectrum Disorder Center for Excellence Phone: 1-866-STOPFAS http://fascenter.samhsa.gov http://www.findtreatment.samhsa.gov/	Federal initiative dedicated to preventing and treating fetal alcohol spectrum disorders. Information on substance abuse treatment.

referrals for early intervention and community support services, are essential elements of care.[61]

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New Warning About Leaving Infants in Cars Even on Mild Days

Serious injury and death from heat stroke continue to occur each year in the United States despite warnings about the risks of leaving infants and children unattended in vehicles. Parents might be aware that it is unsafe to leave a baby in a car on a very hot day, but mistakenly believe that it would be harmless to do so on a milder day or if the windows are cracked open.

A recent observational study determined that on a clear sunny day, even if the outside temperature is mild, there is rapid and extreme heating of the interior of the vehicle.[1] With an ambient temperature of 72°F, the internal vehicle temperature reached 117°F within 60 minutes; 80% of that temperature rise occurred in the first 30 minutes.[1] Cracking open the windows did not decrease either the rate of heat rise or the maximum temperature attained.

Routine safety and discharge teaching should include the facts about this dangerous practice.

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