# Overview of Fetal Alcohol Spectrum Disorders for Mental Health Professionals

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**Abstract**

Fetal Alcohol Spectrum Disorders (FASD), including Fetal Alcohol Syndrome (FAS) and related disorders such as Alcohol Related Neurodevelopmental Disorder (ARND) are the most common form of developmental disability and birth defects in the western world. Early recognition and accurate diagnosis by mental health professionals remains a key issue. This article reviews history, mechanisms of alcohol exposure, epidemiology, diagnosis and management of FASD.

**Abbreviations**

FAS - Fetal Alcohol Syndrome
FASD - Fetal Alcohol Spectrum Disorder
FAE - Fetal Alcohol Effect
PFAS - Partial Fetal Alcohol Syndrome
ARBD - Alcohol Related Birth Defects
ARND - Alcohol Related Neurodevelopmental Disorder
ADHD - Attention Deficit Hyperactivity Disorder

## Overview of Fetal Alcohol Spectrum Disorders for Mental Health Professionals

In the 30 years since the term fetal alcohol syndrome (FAS) was first used by Jones and Smith to describe a group of children, born to alcoholic mothers, who had growth retardation, characteristic facial features and central nervous system involvement, it is now recognized that prenatal alcohol exposure can cause a broad spectrum of developmental, emotional, behavioural and social deficits extending far beyond the original case definition (Jones et al, 1973).

Fetal alcohol syndrome and related conditions are the most common and completely preventable form of developmental disability and birth defects in the western world. The spectrum of deficits associated with prenatal alcohol exposure is now being referred to as "Fetal Alcohol Spectrum Disorders" or FASD (Streissguth et al, 2000). This new umbrella term encompasses other conditions associated with fetal alcohol exposure such as fetal alcohol effects (FAE), partial FAS (PFAS), alcohol related birth defects (ARBD) and alcohol related neurodevelopmental disorder (ARND). All of these terms refer to situations where the patient has some but not all the findings of FAS and a history of alcohol exposure during gestation. The estimated incidence of FAS is between 0.5 and 3 per 1,000 live births. The estimated incidence of conditions within the FASD category is 10 per 1000 live births (Alberta Clinical Practice Guidelines, Preface, 1999). Despite increased public awareness of FASD, early recognition and accurate diagnosis by mental health professionals continues to be a significant issue (Streissguth et al, 2000; Alberta Clinical Practice Guidelines, Preface 1999). The Alberta Medical Association Clinical Practice Guidelines, reprinted in this journal edition, represent one initiative to provide a synthesis of knowledge and best practices for medical professionals. The purpose of this article is to provide an overview of FASD.

### History of FASD

Recognition that alcohol can have harmful effects on pregnancy and child outcomes is not a new finding. Lemoine and colleagues (Lemoine et al, 1968) in 1968 reported changes in a group of 127 children born to alcoholic mothers who had the same constellation of symptoms reported by Jones and Smith (Jones et al, 1973). Streissguth and colleagues describe case reports in the literature from as early as 1726 (Streissguth et al, 2000). However, the naming of the syndrome in 1973 focused new attention on alcohol use and abuse as a serious public health issue and encouraged research into the basic mechanisms of prenatal alcohol exposure and its long-term consequences.

### Alcohol Consumption and the Risk of FAS

Physicians are frequently asked what is the safe "lower limit" for alcohol consumption during pregnancy. The absolute amount of alcohol that will not cause damage to the developing fetus is not known. What is known is that the risk of FASD increases dramatically once the level of "frequent drinking," 6 to 8 drinks per occasion, is reached. We also know that at even lower levels, 1 to 3 drinks per occasion, long term changes in attention, cognition and learning can occur (May, 1995; Olson et al, 1998). Binge drinking during pregnancy, even when infrequent, can cause damage due to high peak alcohol concentration and is itself a drinking pattern associated with an increased risk of FASD. The prevalence of maternal alcohol consumption during pregnancy has increased from 12.4% to 16.3% between 1991 and 1995. Moreover, frequent drinking during pregnancy, defined as 7 or more drinks per week, has increased during the same time period from 0.8% to 3.5% (Ebrahim et al, 1998) In the face of this increasing pattern of maternal alcohol consumption, it is imperative that all women be screened for alcohol use. Appearance, culture or socioeconomic status cannot identify a pregnant woman who consumes alcohol. Hicks and colleagues review key aspects of screening for alcohol use during pregnancy in this journal edition. Some key points for assessing risk dur-

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ing a prenatal alcohol history include:

- Presence of a heavy drinking partner – most women drink with their partners;
- Past history of sexual or physical abuse – a study of 80 birth mothers of children with FAS revealed that 95% were physically or sexually abused during their lifetime (Astley et al, 2000; Astley et al, 2000);
- Presence of mental health disorders – in the same study of 80 birth mothers 96% had 1 to 10 mental health disorders the most prevalent being post traumatic stress disorder (77%) and phobia (44%),
- Polysubstance abuse profile; and
- Social isolation and lack of social support (Astley et al, 2000; Astley et al, 2000).

Interventions should be tailored to deal not only with the alcohol abuse but also the other risk factors identified. In most provinces there is no waiting period for pregnant women in need of addictions treatment. Studies indicate that a supportive counseling and/or case management approach can result in 60 to 80 percent of pregnant women reducing their alcohol intake before the third trimester and 35 to 50 percent stopping heavy drinking (Alberta Clinical Practice Guidelines, Recommendations, 1999).

Once a diagnosis of FASD is made, intensive case management that addresses the medical and social needs of the biological mother and her family must be offered to prevent the occurrence of future affected children. The risk of recurrence of FASD in families with one affected child is 77% (Alberta Clinical Practice Guidelines, Recommendations, 1999). Use of the lay advocate approach, first developed in Seattle by Streissguth and Grant, that supports the mother and facilitates access to resources has been shown to be effective in maintaining abstinence and promoting contraceptive use in high-risk women (Streissguth, 1997). Similar programs are now running in many major centres in Canada (Roberts et al, 2000).

### Spectrum of Alcohol Damage and Alcohol Related Effects

Alcohol is a known teratogen that can cause a spectrum of damage and disrupt fetal development in all three trimesters. Alcohol and its metabolites interfere with DNA synthesis, cell division as well as cell migration and development. Exposure in the first trimester affects organ development and craniofacial development. Structural brain abnormalities are most common followed by cardiac abnormalities, especially septal defects. The whole range of *alcohol related birth defects* (ARBD) is presented in **Table 1** (Alberta Clinical Practice Guidelines, Preface, 1999; Alberta Clinical Practice Guidelines, Diagnosis, 1999; Stratton et al, 1996). Exposure in the second trimester leads to an increased rate of spontaneous abortions. Exposure to alcohol in the third trimester has a more severe effect on birth weight and length. Prenatal exposure to alcohol disrupts fetal brain development at any point in gestation.

Alcohol related brain deficits can manifest as either structural changes such as microcephaly, agenesis of the corpus callosum or cerebellar hypoplasia; or functional deficits affecting behaviour and cognition. Gibbard and colleagues in this journal edition will fully review the range and consequences of these brain deficits.

The Institute of Medicine criteria summarized in **Table 1** lists the various criteria associated with *alcohol related neurodevelopmental disorder* (ARND).

### FAS Diagnostic Categories:

Since FAS was first defined in 1973 researchers and clinicians have struggled to find consistent terminology to describe the spectrum of effects and the individual criteria that should be included in the diagnosis. In 1996, the US institute of Medicine published new diagnostic categories that are summarized in **Table 2** (Jones et al, 1973; Alberta Clinical Practice Guidelines, Diagnosis, 1999; Stratton et al, 1996).

The term *FAS without confirmed alcohol exposure* is used when children have all the growth, facial and CNS characteristics, but there is no way to accurately verify the mother's use of alcohol.

The term *partial FAS* is applied to the patient with a confirmed history of prenatal alcohol exposure who has some but not all the characteristics of FAS. Partial does not mean that the condition is less severe than FAS. Many patients diagnosed as partial FAS would have been designated as Fetal Alcohol Effect or FAE under previous informal classification systems. The use of the term FAE has been discouraged by its originator Dr. Sterling Clarren, since it is non-specific and encompasses a broad range of conditions that have varying severity and outcomes (Aase et al, 1995). A common misconception is that FAE is a less severe form of FAS. Although the patient designated as FAE may not have all the physical abnormalities of FAS, the cognitive and behavioural impairments and hence life long disabilities are similar in severity.

### Primary Features and Disabilities of FAS - Growth, Face and Brain

The growth pattern characteristic of FAS usually presents in the prenatal period and persists as a consistent impairment over time. In contrast, growth deficiency due to postnatal influences will likely present as periodic fluctuations in the growth curve. The most consistent features of the FAS facial phenotype include small palpebral fissures (reflecting small eyes), a smooth philtrum and a thin upper lip. Criteria and norms have now been established by Astley and Clarren that allow for more accurate case definitions (Astley et al, 2000). A manual and CD-ROM have been developed to provide clinicians with specific information on how to examine and measure the facial features (Astley et al, 2000; Fetal Alcohol Syndrome Tutor, 1999).

Additional practice points include the following:

- The likelihood of an FAS diagnosis is greatest when the three features of thin upper lip, smooth philtrum and small palpebral fissures are present;
- Small eyes persist into adolescence and adulthood and may be a reliable physical marker in older individuals;
- Facial features and growth delay diminish in adolescence and hence it is important to obtain past facial photographs and growth records; and
- If there are no facial features, but there is a definitive

history of prenatal alcohol exposure, other conditions within the alcohol related spectrum need to be explored.

**Figure 1** shows the major and associated features of FAS. Other conditions that have similar facial characteristics include Fragile X Syndrome, Velocardiofacial Syndrome and Fetal Dilantin Syndrome.

**Figure 2** shows a graphic representation of the brain manifestations of prenatal alcohol exposure. This photograph has been used widely in FAS public awareness campaigns and compares the brain of a child who died at birth due to severe FAS to that of a normal newborn. This is an extreme example of the effects of prenatal alcohol exposure and would not be a typical finding.

Severity of brain dysfunction can be placed on a continuum from subtle neuro-behavioral deficits to obvious structural abnormalities. The primary functional brain disabilities of FAS can be organized according to a mnemonic "ALARM" (Loock, 2001).

- Adaptive Functioning
- Language/Learning
- Attention
- Reasoning
- Memory

The level of *adaptive functioning* and ability to live independently in individuals with FASD is often less than half the patient's chronological age. Less than 10% of adults with FAS live independently due to impairments in life and social skills, despite having low average or average intellectual abilities (Olson et al, 1998). *Language deficits* result from a global impairment in executive functioning due to prenatal alcohol exposure. The understanding of complex language and figures of speech is markedly deficient (Coggins et al, 1998).Hence the use of language as a social or behavioural mediator is poor and further compromises the adaptive functioning and learning potential of the affected individual (Streissguth et al, 1991). The majority of children with FASD have *attention difficulties* but differ from other types of ADHD in having an earlier onset, more complex comorbidities and a different response profile to psychostimulants (Streissguth et al, 2000). *Reasoning and memory deficits* are also related to basic impairments in executive functioning of the brain and become more noticeable when the FAS affected person reaches school age. Patients with FAS are often slow to learn new skills and do not learn from past experiences. IQ testing may not reveal the reasoning and memory deficits seen in FASD and more specialized neuropsychological testing is often required (Olson et al, 1998).

Each component of the FAS diagnosis can range in severity according to the patient's age, environmental variables and the quantity and quality of prenatal alcohol exposure. Hence it is easy to see why no two individuals with FAS will present with the same constellation of abnormalities and disabilities. In response to these limitations Astley and Clarren have developed a new diagnostic system where growth, facial phenotype, CNS dysfunction and alcohol exposure all vary along separate continua (Astley et al, 2000).

The "4-Digit Diagnostic Grid" allows the features of growth, face, brain and alcohol use to be ranked independently on a 4 point Likert scale, with 1 reflecting complete absence of FAS features and 4 reflecting strong presence of the FAS features. Many diagnostic teams in Canada and the United States are now using this approach.

### Secondary Disabilities, Co-Existing Conditions and Protective Factors

The secondary disabilities of FAS arise after birth as a result of the neurological deficits and come at a high cost to the individual, their family and society.

Common secondary disabilities include:

- Mental health disorders – depression and suicidal ideation
- Disrupted school and employment experience;
- Trouble with the law;
- Inappropriate sexual behavior and involvement in the sex trade; and
- Addictions (Alberta Clinical Practice Guidelines, Diagnosis, 1999).

FASD usually occurs within a constellation of co-existing prenatal and postnatal social and medical comorbidities. Co-existing conditions that are more commonly associated with FASD are attachment disorders as well as sequelae of abuse and neglect such as shaken baby syndrome.

The impact of these secondary conditions may be reduced significantly by the following protective factors:

- Early diagnosis;
- Appropriate interventions for primary and secondary disabilities;and
- Placement in a stable and nurturing environment that is non-abusive (Alberta Clinical Practice Guidelines, Diagnosis, 1999).

However, the mental health disorders that present in patients with FASD do not appear to be impacted significantly by the above protective factors (Streissguth, 1997).

### Long-Term Impact of FASD

Streissguth and O'Malley have summarized the studies looking at the long-term mental disorders and maladaptive behaviours associated with FASD (Streissguth et al, 2000). Streissguth studied 61 adolescents and adults with a FAS/FAE diagnosis and the follow-up showed a wide range of mental and behavioural symptoms. The most common symptoms included: attention difficulties, social withdrawal, impulsive behaviour, dependent behaviour, teasing and bullying behaviour, anxiety, stubborn or sullen mood, lying, cheating and stealing. The authors also report on studies from Europe including Spohr (1996) and Steinhausen (1996) that reveal a similar range of difficulties in the spheres of attention deficits, impulse control, aggression and other deficits in executive functioning. Steinhausen, a psychiatrist from Berlin, reported that 63% of his patients had psychiatric disorders that were long-term and the majority of these patients required intensive therapeutic management. Finally, Lemoine and colleagues were able to identify 105 of the 127 patients of alcoholic mothers from their original study in 1968 and they concluded that mental problems were the

most serious sequelae of FAS in adulthood (Lemoine et al, 1992).

**Management**

FASD is a clinical diagnosis that can have varying physical and developmental manifestations across the lifespan. A multidisciplinary team approach is recognized as a best practice standard for making the diagnosis and planning interventions. Psychiatric input into this multidisciplinary team is critical to making an accurate diagnosis of FASD and associated psychiatric comorbidities, and also to the effective management of secondary mental health disabilities

Management in the primary care setting begins with screening for the key manifestations of FASD and facilitating an early and accurate diagnosis by a multidisciplinary team that can address primary and secondary disabilities. It is important to recall that FASD is a diagnosis for two. Biological parents should be identified and supported so that future pregnancies are not alcohol affected.

Additional management strategies include:

- Support to caregivers and educators through the provision of resources and information (see Internet resources; Graefe, 1998; FAS/FAE Technical Working Group, 2001);
- Routine screening of patients with FASD for the primary brain disabilities – ALARM and advocacy for early and intensive intervention;
- Identify advocates for the affected individual who can act as an "external brain" or protector / custodian;
- If medications are necessary provide close follow-up, monitor for side effects and begin at a lower starting dose;
- Regular surveillance for onset of secondary disabilities and aggressive intervention when they occur; and
- Long-term, structured supervised residential, educational and vocational supports (Alberta Clinical Practice Guidelines, Diagnosis, 1999).

In summary, psychiatrists and other mental health professionals can play a key role in the effective prevention, diagnosis and management of Fetal Alcohol Spectrum Disorders. The articles in the remainder of this journal edition are devoted to the topic of Fetal Alcohol Spectrum Disorders and hopefully will provide further insight into current practices, knowledge, screening and the mental health disabilities associated with this condition.

Suggested additional reading is noted in the References with an a.sterix (*).

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**Internet Resources**

Alberta Medical Association- Clinical Practice Guidelines
http//www.albertadoctors.org

Alcohol Related Birth Injury Site (ARBI)
http://www.arbi.org/index.html

BC FAS Resource Society – Community Action Guide for the Prevention of FAS and A Layman's Guide to FAS
http://www.mcf.gov.bc.ca/child protection/fasfae index.htm

BC Ministry of Education – Teaching Children with FAS
http://www.bced.gov.bc.ca/specialed/fas/contents.htm

FAS/FAE Information Service – Canadian Centre on Substance Abuse
http://www.ccsa.ca/fasgen.htm

Health Canada
http://www.healthcanada.ca/fas

Seattle University of Washington FAS Diagnostic and Prevention Website
http://depts.wash.edu/fasdpn

**Table 1**

## Diagnostic Criteria for Alcohol-Related Effects

**1. Alcohol-related birth defects**

**Cardiac:** Atrial septal defects, ventricular septal defects, aberrant great vessels, Tetralogy of Fallot.

**Skeletal:** Hypoplastic nails, shortened fifth digit, radioulnar synostosis, joint contractures, camptodactyly, clinodactyly, pectus excavatum and carinatum, Klippel-Feil syndrome, hemivertebrae, scoliosis.

**Renal:** Aplastic, dysplastic, hypoplastic kidneys, horseshoe kidneys, ureteral duplications, hydronephrosis.

**Ocular:** Strabismus, refractive problems secondary to small globes, retinal vascular anomalies.

**Auditory:** Conductive hearing loss, neurosensory hearing loss.

**Other:** Virtually every malformation has been described in some patient with FAS. The etiologic specificity of most of these anomalies to alcohol teratogenesis remains uncertain.

**2. Alcohol-related neurodevelopmental disorder**

Presence of A and/or B.

A. Evidence of CNS abnormalities in at least one of the following:
- Decreased cranial size at birth
- Structural brain abnormalities (e.g., microcephaly, cerebellar hypoplasia)
- Neurological hard or soft signs (as age appropriate), such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, poor eye-hand coordination.

B. Evidence of a complex pattern of behavior or cognitive abnormalities that are inconsistent with developmental level and cannot be explained by familial background or environment alone, such as learning difficulties, deficits in school performance, poor impulse control, problems in social perception, deficits in higher level receptive and expressive language, poor capacity for abstraction or metacognition, specific deficits in mathematical skills; or problems in memory, attention, or judgment.

Adapted from Stratton K, How C, Battaglia: FAS Diagnosis, Epidemiology, Prevention and Treatment, Washington, D.C. Naional Academy Press, 1996

**Table 2**

## Diagnostic Criteria for FAS

**1. FAS with confirmed maternal alcohol exposure**

A. Confirmed maternal alcohol exposure
B. Evidence of characteristic pattern of facial anomalies including short palpebral fissures and abnormalities in the premaxillary zone (e.g. flattened upper lip, flattened philtrum, and flat midface).
C. Evidence of growth retardation in at least one of the following:
- Low birth weight for gestational age
- Decelerating weight over time not due to other identified cases
- Disproportional low weight to height

D. Evidence of CNS abnormalities in at least one of the following:
- Decreased cranial size at birth
- Structural brain abnormalities (e.g., microcephaly, cerebellar hypoplasia)
- Neurological hard or soft signs (as age appropriate), such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, poor hand-eye coordination.

**2. FAS without confirmed maternal alcohol exposure**
B, C and D above

**3. Partial FAS with confirmed maternal alcohol exposure**

A. Confirmed maternal alcohol exposure.
B. Evidence of some components of the pattern of characteristics facial anomalies and either
C, D or E below.
C. Evidence of growth retardation, in at least one of the following:
- Low birth weight for gestational age
- Decelerating weight over time not due to nutrition
- Disproportional low weight to height

D. Evidence of CNS abnormalities in at least one of the following:
- Decreased cranial size at birth
- Structural brain abnormalities (e.g. microcephaly, cerebellar hypoplasia)
- Neurological hard or soft signs (as age appropriate), such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, poor hand-eye coordination.

E. Evidence of a complex pattern of behavior or cognitive abnormalities that are inconsistent with developmental level and cannot be explained by familial background or environment alone, such as learning difficulties, deficits in school performance, poor impulse control, problems in social perception, deficits in higher level receptive and expressive language, poor capacity for abstraction or metacognition, specific deficits in mathematical skills or problems in memory, attention or judgment.

1. A pattern of excessive intake characterized by substantial, regular intake or heavy episodic drinking. Evidence of this pattern may include frequent episodes of intoxication, development of tolerance or withdrawal, social problems related to drinking, legal problems related to drinking, engaging in physically hazardous behavior while drinking, or alcohol related medical problems, such as hepatic disease.

Adapted from Stratton K, How C, Battaglia: FAS Diagnosis, Epidemiology, Prevention and Treatment, Washington, D.C., National Academy Press, 1996 and Jones KL, Smith DW: Recognition of the fetal alcohol syndrome in early infancy. Lancet 1973; 2(7836): 999-1001.

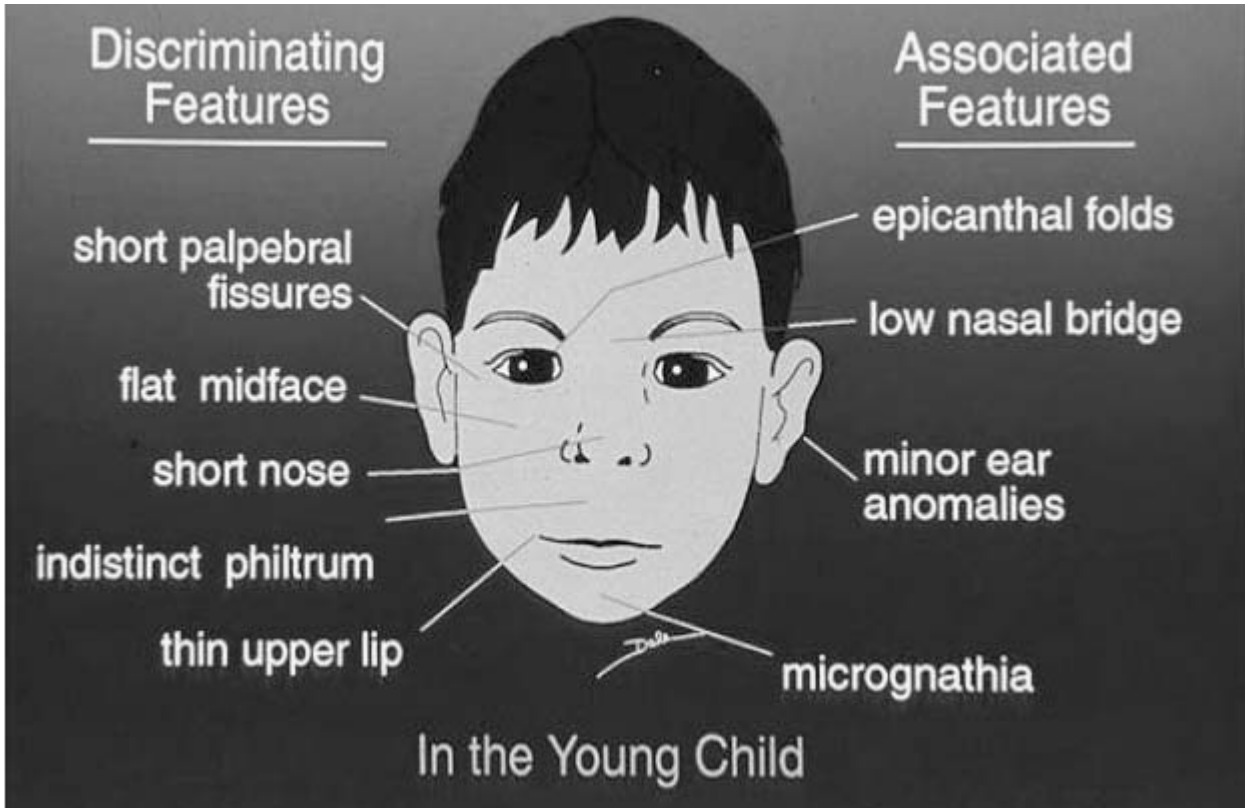


Figure 1: Facial Features of Fetal Alcohol Syndrome

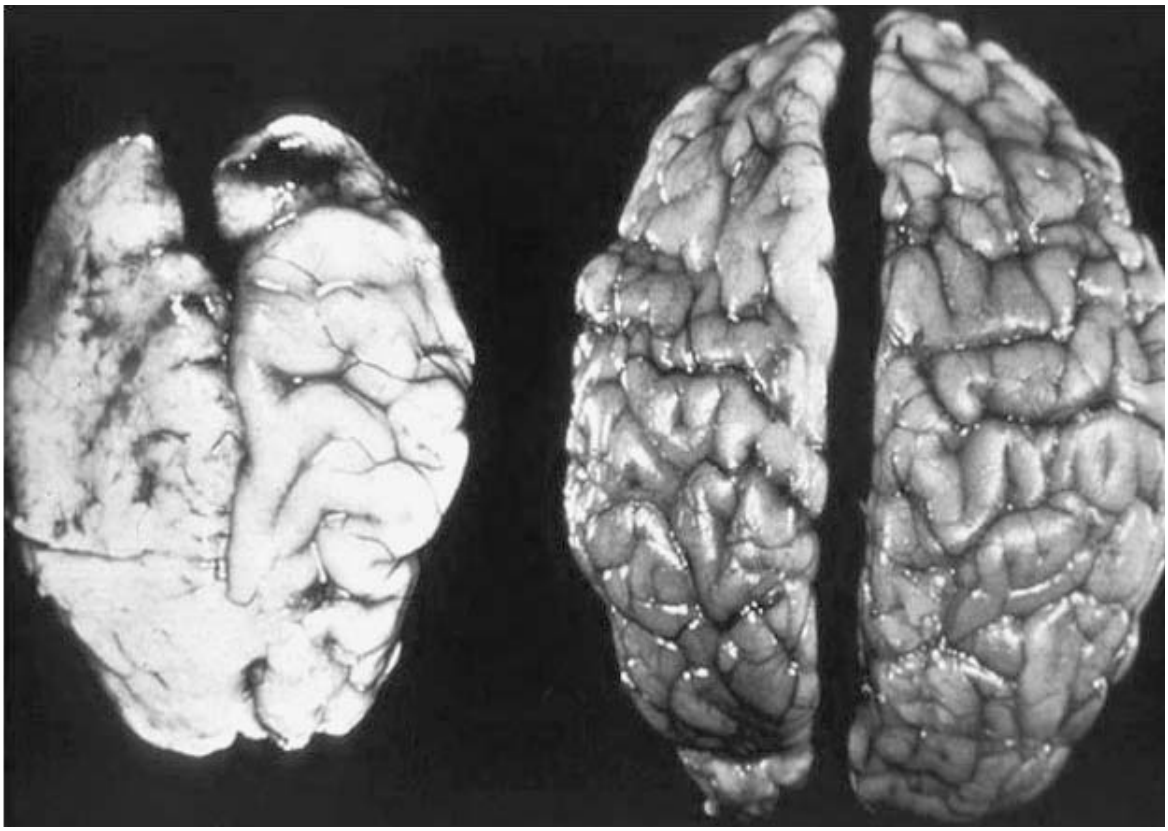

Figure 2: Brain Manifestations of Prenatal Alcohol Exposure

# Knowledge and Attitudes of Canadian Psychiatrists Regarding Fetal Alcohol Spectrum Disorders

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### Background

Current reports regarding increased rates of alcohol use in women of child bearing age and the anticipated impact on the occurrence of Fetal Alcohol Spectrum Disorders (FASD) have highlighted the need to better understand the attitudes and knowledge of health care providers towards alcohol consumption and prevention of FASD. Studies have shown that physicians' knowledge, attitudes and beliefs about a health problem such as alcohol abuse can either predispose or deter them from screening, identifying and managing the problem (Diekman et al, 2000).

Surveys in Canada and the United States have found inconsistencies in diagnosis, treatment, and management of children with FASD due to gaps in physician knowledge and attitudes regarding FASD. A survey by the Michigan Medical Association found that health care professionals felt that alcohol's effect on the fetus was clear, fetal alcohol syndrome (FAS) is not being over-diagnosed, and that making a diagnosis of FAS can improve treatment plans for affected individuals (Abel and Kruger, 1998). A majority of those surveyed said that they felt it necessary to emphasize the dangers of prenatal alcohol use to their patients and that such discussion would not frighten or anger their patients. Physicians also identified that they were not sufficiently familiar with FAS or fetal alcohol effects (FAE). Surveys in Alberta in 1998 and in Saskatchewan in 1991 also provided information on physician understanding and awareness of alcohol use during pregnancy and FASD (Nanson and Bolaria et al, 1995; Alberta Medical Association, 1998; Teskey and Clarke, 1998). The two studies highlight the variation in knowledge and practice in Canada.

Therefore, more information regarding regional and provider variation is required to assist in the development of national guidelines. Psychiatrists have never been included in past FAS surveys, although the recent Canadian Psychiatric Association Practice profile survey showed that they are an important provider group since almost 60% of patients seen are female (Woodside and Lin, 2003).

### Objectives

The objective of the present study was to describe characteristics of psychiatrists with regard to knowledge and attitudes of FASD according to the proportion of their practice allocated to treatment of those <16 years old and to draw some comparisons between psychiatrists and other health care providers.

### Methods

A questionnaire was modified from a prior survey based upon extensive consultation and pilot testing with the National Advisory Committee on FAS and professional groups. The questionnaire consisted of 4 parts including General Knowledge, Prevention and Diagnostic Issues, and Background Information. All but two questions were a forced-choice format with response options varying depending on question content (e.g., yes/no, Likert scales). The survey package was translated into French, formatted in the Teleform data management program, and programmed in HTML for a web-based version.

A mailed survey was sent to a national random sample of 60% of all eligible Canadian psychiatrists selected from the mailing list of the professional organization between October 2001 and January 2002 (N=851/1439). The package contained a cover letter, instructions for completing paper or web-based versions, the survey, and notice of a draw for a Palm Pilot. Mailed questionnaires were followed up with 2 reminder postcards, repeat mailings, phone follow up, and an attempt to reach participants whose original contact information was incorrect. Return of the completed questionnaire was taken to signify consent to participate.

### Statistical Analysis

All data were transferred into SPSS/PC version 10.0 for analysis. Descriptive analysis and bivariate comparisons (2 and 2 trend) were completed to understand psychiatrists' attitudes, knowledge and practices towards FAS by proportion of practice allocated to those aged 16 years or less. 'P' values refer to comparisons made within psychiatrists according to proportion of practice with children <16. As the questionnaire was also completed by midwives, obstetricians, family physicians and paediatricians, the overall results of this cross professional analysis are presented for comparison.

### Results

Of 851 surveys distributed, 391 were returned for a response rate of 45.9%. Respondents in the Prairies may be over-represented while those in Quebec under-represented, as compared to national distributions of psychiatrists, however, the distribution of responses for the West, Ontario and the East reflect the national distribution of psychiatrists (data not shown). Of the 384 psychiatrists who completed the demographic information, only 316 indicated what proportion of their practice was allocated to children <16 (82.3%). Of these, 71.2%

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(n=225) reported that <10% of their practice was comprised of children <16 (group 1), 9.8% reported 10-49% (group 2), 19.0% reported 50-100% (group 3).

### Demographics

There were no significant demographic differences between groups according to the proportion of practice with children <16 years old, years in practice, university appointment, and location of practice. However, among those in group 3, 50% of providers were female, compared to groups 1 and 2 where a higher proportion of providers were male (data not shown).

### Attitudes Towards Alcohol Consumption

Over 65% of psychiatrists agreed with the practice of advising moderate alcohol consumption among non-pregnant women (Table 1). The majority of psychiatrists defined moderate as <=2 drinks per occasion, <=3 times per week (Table 1). There was no difference between psychiatrists' responses and those of respondent from other disciplines (Table 1).

### Awareness and Attitudes

Psychiatrists were asked about awareness, attitudes and prevention of FAS with no significant differences noted between groups. Greater than 75% agreed that the effects of alcohol on the fetus were clear, that prenatal alcohol exposure is a risk factor for permanent brain damage, FAS was identifiable, and it occurs across social strata (Table 2). Over 90% of respondents believed that discussing alcohol use would not deter clients from treatment.

### Level of Preparedness to Deal With Alcohol Related Problems in Practice

Psychiatrists (74%) believed it to be the physician's role to manage problems associated with alcohol use. Within psychiatrist groups, those in groups 2 and 3 were significantly more prepared to care for foster parents and affected individuals, and to access resources for pregnant women, birth mothers and foster parents (all $p<0.05$, Table 2). Psychiatrists with a higher paediatric caseload were significantly more likely to have cared for FAS-affected individuals, have suspected FAS in their caseload, and have referred patients to confirm a diagnosis (see Table 3). Only 14.6% of providers reported using a diagnostic schema for FAS in their practices such as Institute of Medicine criteria. Among psychiatrists, 20% reported using a schema and those with more paediatric clients were significantly more likely to use a schema ($p<0.05$). The most commonly used schema was the DSM-IV.

Over 90% of those in groups 2 and 3 compared to 58% in group 1 agreed that making a diagnosis of FAS was within their scope of practice (Table 4). Within psychiatrist groups significant differences between groups revealed that those who had the greatest contact with children <16 were most likely to indicate that lack of training was a diagnostic barrier, and to identify that diagnosis is unlikely to make a difference to the individual (Table 4). The majority of psychiatrists (15%) did not believe there should be mandatory reporting of FAS or FAE (data not shown).

As noted in Table 5, those in groups 2 and 3 were significantly more likely to recognize the facial features associated with FAS. Psychiatrists in groups 2 and 3 were less likely than those in group 1 to indicated that FAS is associated with addictions and legal problems (secondary disabilities). Of note, psychiatrists in groups 2 and 3 were more likely than those in group 1 to recognize that inappropriate sexual behaviour is associated with FAS (40-55% versus 30%) (Table 5). Diagnostic knowledge about FAE was, in general, significantly higher among those in groups 2 and 3 compared to those in group 1. As well, those in groups 2 and 3 were more likely than those in group 1 to recognize the features of FAE (Table 6).

Over 80% of psychiatrists were able to identify delayed development, birth defects, mental disorders, learning disabilities, lowered IQ, craniofacial deformities, behaviour problems, low birth weight and growth retardation as being correctly associated with prenatal alcohol exposure (Table 7).

### Knowledge

Psychiatrists were asked to identify sources of information about FAS and FAE (Table 8). In general, groups 2 and 3 were significantly more likely than group 1 to obtain information about FAS or FAE from colleagues, medical journals and books, seminars, parents and patients (data for FAE not shown).

### Resources

Resources indicated to be helpful in addressing FAS and related issues by more than half of the psychiatrists included: registry of specialists, referral resources and clinical practice guidelines. Within groups, those psychiatrists in groups 2 and 3 were significantly more likely to indicate that pregnancy history checklists, materials or training on FAS/FAE, access to a psychologist, registry of specialists, clinical practice guidelines and access to telehealth would be advantageous (Table 9). Over 75% of respondents identified that access to educational material, multidisciplinary teams, outreach clinics, and CME training would be of benefit.

### Discussion

This study represents the first survey of psychiatrists in the area of knowledge, attitudes and practices related to FASD. The response rate of 45.6% is adequate for a physician survey and comparable to the Canadian Psychiatric Association Psychiatric Profile (Lin et al, 2003). The distribution of responses suggests that the results are likely generalizable to most regions of Canada, with the potential exception of Quebec, which was under-represented. The majority of psychiatrists had less than 10% of their practice allocated to care of those <16 years old; only 19% of psychiatrists had 50% or more of their practice allocated to care of those <16 years old.

Psychiatrists were aware of the detrimental effects of alcohol on fetal development and agreed that it was the physician's role to manage client issues associated with alcohol misuse. Only half of providers felt prepared to do so, although a greater proportion were willing to access resources. Psychiatrists who have more paediatric clients were more likely to be prepared to access resources and care for clients in areas associated with alcohol misuse.

Psychiatrists who have more paediatric clients were also more likely to indicate that making a diagnosis of FAS was within their scope of practice and that time and training were

barriers to diagnosis. Psychiatrists with larger caseloads of paediatric clients agreed that making a diagnosis changed things for the child. They also indicated that many doctors do not make a diagnosis due to a belief that it will not make a difference for the child. The data suggests psychiatrists who have a greater proportion of paediatric patients and hence a greater likelihood of treating FASD clients have a more realistic understanding of the complexities of care and management of this population. Most psychiatrists did not employ a diagnostic schema specific to FASD, highlighting the need for the Diagnostic and Statistical Manual of Psychiatric Disorders to include specific neuropsychiatric manifestations of this diagnosis

Psychiatrists with higher paediatric caseloads were also more adept at identification of FAS and FAE, including physical, emotional and social/learning issues, however, some still lack understanding of the associated secondary disabilities including inappropriate sexual behaviour and addictions. Educational efforts in the future should concentrate on methods for early identification and management of secondary disabilities.

Psychiatrists who have more paediatric clients were more likely to have obtained information about FAS/FAE from a variety of sources, including parents, patients and traditional medical venues. Furthermore, psychiatrists, particularly those with larger paediatric practices identified the need for Clinical Practice Guidelines, access to psychologists and educational information. Over 75% of all psychiatrists indicated that, in addition to the above, access to a multidisciplinary team, outreach clinics and CME training would be of value. Less than 30% of all providers wanted training on addiction counselling, still suggesting there is a gap between the scope of practice of psychiatry and the field of addictions medicine.

This study represents the first survey of psychiatric practice in the area of FASD in Canada. There is a great need to help psychiatrists recognize the primary and secondary disabilities, especially in affected individuals who do not have mental retardation or dysmorphic features as part of their diagnosis. There will be a great need in the future for psychiatrists to work closely with colleagues in the fields of addictions, pediatrics and neuropsychology to develop a comprehensive multidisciplinary approach to this significant and ultimately preventable condition.

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# Tables

**Table 1. Alcohol use, moderate consumption and preparedness to care for clients with alcohol related issues.**

| | Overall N (%) | Psychiatrist N (%) | Group 1 0-9% | Group 2 10-49% | Group 3 50-100% | p |
|---|---|---|---|---|---|---|
| Agree with telling patients to drink in moderation | 1302(60.7) | 255(67.8) | 153(70.5) | 16(53.3) | 38(67.9) | .432 |
| Moderate number of drinks per occasion among non-pregnant women <=2 | 1825(91.0) | 324(93.4) | 191(94.6) | 23(92.0) | 48(87.2) | .319 |
| Moderate number of occasions per week among non pregnant women is <=3 | 1472(74.3) | 256(74.8) | 148(75.5) | 19(76.0) | 46(82.2) | .152 |
| Agree physicians role to manage problems in area of alcohol use | 1670(76.1) | 288 (73.8) | 164(72.6) | 28(90.3) | 42(70.0) | .641 |
| **Prepared to care in area of alcohol abuse or dependency for :** | | | | | | |
| Pregnant women** | 831(54.2) | 154(58.1) | 83(52.2) | 17(77.3) | 19(57.6) | .876 |
| Birth mothers ** | 879(55.6) | 158(58.3) | 84(52.2) | 20(87.0) | 23(62.2) | .068 |
| Foster parents | 789(36.2) | 171(44.6) | 89(54.3) | 20(83.3) | 32(72.7) | .007 |
| Affected individuals | 987(45.4) | 210(54.6) | 107(60.8) | 26(92.9) | 39(73.6) | .020 |
| **Prepared to access resources in area of alcohol abuse or dependency for:** | | | | | | |
| Pregnant women** | 1167(70.9) | 246(80.9) | 137(75.7) | 24(100.0) | 37(88.1) | .017 |
| Birth mothers** | 1195(70.9) | 247(81.8) | 139(78.1) | 25(100.0) | 38(88.4) | .034 |
| Foster parents | 1063(49.1) | 239(62.7) | 129(72.1) | 26(92.9) | 40(83.3) | .037 |
| Affected individuals | 1273(58.7) | 275(72.2) | 152(78.4) | 28(93.3) | 45(83.3) | .229 |

Agreement defined as agree and strongly agree
*56.3% of all health care professionals felt that 1-2 occasions per week was moderate
**Among these who care for these patient populations

**Table 2. FAS Awareness and Attitudes about Prevention (Agreement)**

| | Overall N (%) | Psychiatrist N (%) | Group 1 0-9% | Group 2 10-49% | Group 3 50-100% | p |
|---|---|---|---|---|---|---|
| Alcohol's effects on fetus are clear | 1651(74.9) | 296(75.9) | 172(77.8) | 24(77.4) | 46(78.0) | .992 |
| Prenatal alcohol exposure is a risk for permanent brain damage | 2059(93.3) | 360(92.1) | 214(94.3) | 28(90.3) | 58(96.7) | .698 |
| FAS is an identifiable syndrome | 2066 (94.0) | 362 (92.6) | 211(95.5) | 29(96.7) | 54(93.1) | .522 |
| FAS occurs in all strata of society | 2107(95.4) | 368(94.1) | 212(94.6) | 29(93.5) | 59(98.3) | .289 |
| **Discussing alcohol use will not:** | | | | | | |
| *Deter from treatment* | 1895 (86.1) | 353 (90.5) | 211(93.0) | 25(83.3) | 54(90.0) | .123 |
| *Frighten/anger patients* | 2065 (93.6) | 374 (95.7) | 218(96.0) | 31(100.0) | 59(98.3) | .265 |

**Table 3. FAS Diagnosis**

| | Overall N (%) N=1957 | Psychiatrist N (%) N=384 | Group 1 0-9% N=223 | Group 2 10-49% N=31 | Group 3 50-100% N=60 | P |
|---|---|---|---|---|---|---|
| Proportion who use a diagnostic schema | 282 (14.6) | 75 (20.0) | 29 (13.2) | 12 (38.7) | 15 (26.8) | .003 |
| Diagnosed any patient with FAS | 690 (35.3) | 141 (36.7) | 63 (28.3) | 18 (58.1) | 42 (70.0) | .001 |
| Cared for FAS affected individuals | 991 (50.5) | 176 (45.7) | 84 (37.7) | 20 (66.7) | 48 (80.0) | .001 |
| Suspected (not diagnosed) FAS | 947 (48.6) | 197 (51.4) | 98 (43.8) | 22 (73.3) | 50 (83.3) | .001 |
| Referred patients to confirm a diagnosis | 668 (34.3) | 84 (22.0) | 23 (10.3) | 11 (37.9) | 40 (66.7) | .001 |

**Table 4. Global issues related to Diagnosis, including Barriers.**

| | Overall N (%) T=2209 | Psychiatrist N (%) T=391 | Group 1 0-9% | Group 2 10-49% | Group 3 50-100% | P |
|---|---|---|---|---|---|---|
| Diagnosis changes thing for child (agree) | 2007(90.9) | 346(88.5) | 205(94.9) | 26(86.7) | 53(89.8) | .100 |
| Making a diagnosis of FAS was within scope of practice | 1498(75.0) | 257(65.7) | 131(57.7) | 29(93.5) | 54(90.0) | <.001 |
| **Many doctors do not make a diagnosis of FAS in their practice due to:** | | | | | | |
| *Lack of time (agree)* | 436(20.0) | 50 (13.2) | 20(8.8) | 8(25.8) | 14(23.3) | .001 |
| *Lack of specific training (agree)* | 1234(56.4) | 203 (52.9) | 111(48.9) | 20(64.5) | 46(76.7) | <.001 |
| *A belief that making the diagnosis will not make a difference to the individual (agree)* | 265(12.2) | 70 (18.4) | 32(14.1) | 9(29.0) | 20(33.3) | <.001 |

Some cells may not add to n=391l as some questions were not answered by all participants.

**Table 5. Diagnostic Knowledge about Characteristics of FAS**

| | Overall N (%) T=1901 | Psychiatrist N (%) T=368 | Group 1 0-9% | Group 2 10-49% | Group 3 50-100% | P |
|---|---|---|---|---|---|---|
| Flat Philtrum | 1412(74.3) | 214 (58.2) | 110(51.6) | 23(74.2) | 49(87.5) | <.001 |
| Thin Upper Lip | 1316(69.4) | 177 (48.8) | 84(39.8) | 25(80.6) | 43(76.8) | <.001 |
| Short Palpebral Fissures | 1211(64.1) | 192 (52.5) | 96(45.1) | 25(80.6) | 38(69.1) | <.001 |
| CNS Dysfunction | 1678(87.7) | 306 (82.3) | 173(80.5) | 27(87.1) | 55(96.5) | .003 |
| Prenatal Growth Deficiency | 1536(80.4) | 254 (68.8) | 143(67.1) | 23(74.2) | 41(73.2) | .266 |
| Combination of growth, brain and facial abnormalities provide the most accurate info about diagnosis of FAS (agree) | 1084(59.7) | 215 (61.4) | 122(59.8) | 22(75.9) | 33(57.9) | .782 |
| **Secondary Disabilities** | | | | | | |
| Emotional disorders | 1345 (70.8) | 265 (70.5) | 147(66.5) | 21(75.0) | 42(75.0) | .632 |
| Disrupted school experience | 1303 (67.3) | 263 (69.0) | 144(64.6) | 19(67.9) | 46(78.0) | .278 |
| Addictions | 1190 (61.7) | 235 (62.0) | 139(62.3) | 16(59.3) | 28(49.1) | .050 |
| Legal Problems | 1336 (69.2) | 256 (67.4) | 154(69.1) | 21(75.0) | 38(65.5) | .029 |
| Inappropriate Sexual Behaviour | 671 (34.8) | 140 (36.8) | 74(33.2) | 16(55.2) | 23(40.4) | .001 |

**Table 6. Diagnostic Knowledge of FAE**

| | Overall N (%) T=2151 | Psychiatrist N (%) | Group 1 0-9% | Group 2 10-49% | Group 3 50-100% | P |
|---|---|---|---|---|---|---|
| Recognized FAE is a partial expression of FAS (agree) | 1508(70.1) | 246(65.3) | 131(60.1) | 25(86.2) | 50(83.3) | <.001 |
| **Recognized that FAE is not used when:** | | | | | | |
| *Birth defects diminish over time* | 1269(59.2) | 191(50.9) | 100(45.9) | 20(69.0) | 44(74.6) | .126 |
| *There are no IQ deficits* | 1122(52.3) | 186(49.3) | 92(42.0) | 24(82.8) | 38(63.3) | <.001 |
| *A child has birth defects but maternal alcohol use is unclear* | 1146(53.4) | 193(51.2) | 105(48.2) | 19(65.5) | 40(66.7) | <.001 |
| *A child is too young to make a diagnosis of FAS* | 1035(48.2) | 180(47.6) | 87(39.5) | 22(75.9) | 44(73.3) | .147 |
| *A less severe form of FAS* | 310(14.4) | 45(11.9) | 27(17.1) | 5(19.2) | 8(15.4) | .829 |
| Recognized that using the term FAE **does not** result in better long term outcomes than FAS | 856(39.9) | 151(40.2) | 72(63.7) | 21(84.0) | 34(82.9) | .010 |

**Table 7. Proportion of respondents who correctly identified the following as associated with prenatal alcohol exposure.**

| | Overall N (%) T=1901 | Psychiatrist N (%) T=368 | Group 1 0-9% | Group 2 10-49% | Group 3 50-100% | P |
|---|---|---|---|---|---|---|
| Infantile withdrawal symptoms | 1404(65.4) | 254(67.4) | 150(68.5) | 19(67.9) | 36(61.0) | .523 |
| Delayed development | 2121(96.9) | 366(94.3) | 209(92.5) | 29(100.0) | 56(93.3) | .360 |
| Birth defects | 1929(88.6) | 339(88.1) | 193(86.2) | 25(89.3) | 56(93.3) | .127 |
| Mental disorders | 1939(88.9) | 336(87.0) | 187(83.1) | 26(89.7) | 55(91.7) | .120 |
| Learning disabilities | 2136(97.4) | 370(95.4) | 209(92.5) | 28(96.6) | 60(100.0) | .018 |
| Lowered IQ | 2091(95.5) | 363(93.6) | 210(92.9) | 28(96.6) | 58(96.7) | .167 |
| Craniofacial deformities | 2025(92.5) | 335(86.8) | 193(86.2) | 27(93.1) | 54(90.0) | .255 |
| Behavioural problems | 2119(96.8) | 364(93.8) | 208(92.0) | 28(96.6) | 56(93.3) | .676 |
| Low birth weight | 1968(89.9) | 332(85.8) | 186(82.3) | 28(96.6) | 53(89.8) | .052 |
| Growth retardation | 1946(88.9) | 325(83.8) | 182(80.5) | 28(96.6) | 50(83.3) | .372 |
| Premature birth | 1429(65.6) | 256(66.3) | 148(65.8) | 23(79.3) | 35(59.3) | .891 |
| Seizures | 1250(57.1) | 232(59.9) | 142(62.8) | 18(62.1) | 26(43.3) | .043 |
| Vision problems | 836(38.3) | 123(31.9) | 69(30.7) | 12(41.4) | 19(31.7) | .276 |
| Structural brain damage | 1475(67.5) | 268(69.6) | 156(69.6) | 20(69.0) | 42(70.0) | .995 |
| Spontaneous abortion | 1127(51.6) | 216(55.8) | 127(56.2) | 20(69.0) | 30(50.0) | .459 |

**Table 8. Identification of Information Sources Regarding FAS**

| | Overall N (%) T=2200 | Psychiatrist N (%) T=387 | Group 1 0-9% | Group 2 10-49% | Group 3 50-100% | P |
|---|---|---|---|---|---|---|
| Gained knowledge of FAS from: | | | | | | |
| Colleagues | 1003(45.3) | 176(45.0) | 86(37.9) | 16(51.6) | 38(63.3) | <.001 |
| Medical journals and books | 1694(76.4) | 306(78.3) | 170(74.9) | 27(87.1) | 56(93.3) | .001 |
| Medical school, residency or fellowship | 1410(63.6) | 205(52.4) | 128(56.4) | 11(35.5) | 32(53.3) | .379 |
| CME seminars or rounds | 1128(50.9) | 156(39.9) | 73(32.2) | 14(45.2) | 37(61.7) | <.001 |
| Parents or patients | 535(24.1) | 105(26.9) | 48(21.1) | 9(29.0) | 31(51.7) | <.001 |

**Table 9. Supports Perceived as Helpful for Clinical Practice**

| | Overall N (%) | Psychiatrist N (%) | Group 1 0-9% | Group 2 10-49% | Group 3 50-100% | P |
|---|---|---|---|---|---|---|
| Literature on impact of prenatal alcohol use | 1087(50.0) | 191(49.5) | 111(49.6) | 18(60.0) | 28(47.5) | .872 |
| Pregnancy history checklists including alcohol use | 851(39.4) | 138(35.8) | 68(30.5) | 14(46.7) | 22(37.3) | .048 |
| Materials or training on FAS/FAE | 1119(51.7) | 181(47.3) | 88(39.6) | 18(60.0) | 38(64.4) | .002 |
| Addiction counselling training | 558(25.9) | 99(26.1) | 59(26.9) | 5(16.7) | 11(19.0) | .585 |
| Registry of specialists for FAS/FAE consultation | 1341(61.8) | 227(53.9) | 128(57.4) | 13(44.8) | 43(72.9) | .046 |
| Referral resources for women of childbearing age with alcohol problems | 1357(62.6) | 226(58.7) | 130(58.3) | 19(63.3) | 37(62.7) | .798 |
| FAS Clinical Practice Guidelines | 1323(60.8) | 214(55.7) | 109(49.1) | 17(56.7) | 44(74.6) | <.001 |
| Assistance with diagnosis of FAS/FAE through Telemedicine | 521(24.1) | 81(21.1) | 38(17.2) | 8(26.7) | 20(33.9) | .004 |
| Access to information about FAS/FAE through Telemedicine | 558(25.8) | 86(22.6) | 41(18.6) | 9(30.0) | 20(33.9) | <.001 |
| Internet Resources | 774(35.8) | 127(33.4) | 62(28.2) | 12(40.0) | 28(47.5) | .001 |
| Access to psychologist | 1077 (71.6) | 204 (75.6) | 100(69.4) | 22(81.5) | 49(89.1) | .003 |
| Access to educational information | 1367 (89.7) | 256 (93.4) | 134(90.5) | 26(96.3) | 53(96.4) | .090 |
| Access to a multidisciplinary team | 1311 (85.2) | 242 (88.0) | 126(84.6) | 23(85.2) | 51(92.7) | .138 |
| Outreach clinic with an expert diagnostic team | 1143 (75.6) | 209 (77.7) | 110(76.4) | 21(75.0) | 41(78.8) | .714 |
| CME training | 1358 (90.7) | 252 (93.0) | 131(89.7) | 26(96.3) | 50(94.3) | .077 |
| Use of Telehealth for diagnosis by an expert diagnostic team | 725 (49.1) | 139 (53.9) | 70(50.0) | 15(53.6) | 28(58.3) | .577 |

# The Neuropsychological Implications of Prenatal Alcohol Exposure

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### Introduction

Alcohol is a neurobehavioral teratogen in that it causes brain damage and modifies behavior (Riley and Vorhees, 1986). The teratogenic effects of alcohol on the developing brain can occur at lower doses and frequency of exposure than required for the physical manifestations of FASD such as the characteristic facial features or growth restriction. Because the brain is constantly developing throughout gestation, the effects of prenatal alcohol exposure on the developing brain can occur at any point. Therefore, exposure at different times and with different doses during gestation may explain why FASD presents as a spectrum of central nervous system dysfunction. In addition, individual differences in the mother and child modify the effect of prenatal exposure in the individual, and not every child exposed is affected (Streissguth, 1997). Overall, the behavioral and cognitive difficulties seen in FASD are modulated by other biologic and environmental factors, making a diagnosis of FASD only one of many contributing factors to a particular individual's profile (Stratton et al, 1996). Prenatal alcohol exposure can result in primary deficits in cognition and behavior. In addition, these primary disabilities can cause secondary disabilities and psychiatric co-morbidities.

### Primary Disabilities

The primary disabilities seen in FASD arise from direct brain damage. The most consistent finding from prenatal alcohol exposure is reduction in brain weight and this can result in microcephaly (Jacobson and Jacobson, 2003). Prenatal alcohol exposure is known to disrupt many areas of brain development, including the cerebellum, hippocampus, basal ganglia, and the corpus callosum (Sowell et al, 2001; Mattson, Schoenfeld, and Riley, 1999). Other pathologic changes to the central nervous system include enlarged ventricles, abnormal neural/glial migration, and changes in the microvasculature in regions of the brain such as the cerebellum and hippocampus (Miller, 1992).

Research to date has not identified a single prototypical cognitive profile, and it is unlikely that one specific behavioral or cognitive phenotype for FASD will emerge. More likely, there will be identifiable patterns related to differential alcohol exposure (timing, amount, and frequency), combined with other genetic and environmental factors. These patterns may be reflective of general patterns of atypical prenatal brain development due to a combination of factors, and may not be specific to alcohol as a teratogen. However, some of the more commonly identified problem areas in FASD include attention, learning and memory, abstract problem solving and strategy generation. While individuals with FASD will often be within normal limits on measures of IQ, they often have other significant neurocognitive deficits. Many areas of cognitive functioning are only peripherally assessed through an IQ measure, such as attention and concentration. In addition, IQ testing does not assess other domains, such as higher order executive functions. These deficits will have a profound effect on the ability of a person with FASD to function, and without appropriate supports and interventions this can lead to secondary impairments.

In regard to general intellectual functioning, early work in this field led to the misperception that individuals with FASD fell within the mentally retarded range (Darby, Streissguth, and Smith, 1981). More comprehensive studies indicate that approximately 25% of persons with FAS and less than 10% of persons with FAE would qualify for funding if a diagnosis of mental retardation was required (Streissguth et al, 1996). This later study, based on a large cohort of individuals with FASD, reported full scale IQ's in the FAS group that ranged from 20 to 120, with a mean IQ score of 79. In the FAE group full scale IQ scores ranged from 49 to 142, with a mean IQ score of 90. It is also not unexpected to see an apparent decrease in IQ performance on sequential assessment in an individual with FASD. However, this is not related to an underlying deteriorative process. Rather, these individuals often fail to make age-appropriate gains in intellectual functioning based on normative performance, even though they continue to show improvements in their raw scores. Part of this failure is related to other deficits such as impaired learning and memory, as well as secondary disabilities that interfere with progress in mainstream academic programming.

Memory impairments are another common deficit in individuals with FASD. More specifically, they are prone to intrusion errors and confabulation, difficulties with strategic manipulation of information to improve recall, and difficulties with initial encoding of information. The nature of memory impairments related to encoding difficulties is due to hippocampus damage, while impaired frontal lobe functioning can cause free recall and intrusion/confabulation difficulties. However, short-term immediate verbal recall is often well developed in individuals with FASD, which can lead to a false impression of "good memory" functioning. Individuals with FASD will also have

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problems with working memory. While many aspects of explicit memory functioning or conscious memory recall have been found to be impaired, implicit memory functioning or procedural or unconscious recall typically functions within normal limits (Mattson & Riley, 1999), which could have significant implications for remediation and vocational training.

Basic language abilities are often found to be a relative strength in FASD. It is often recognized that they are very adept at "parroting back" verbal information that they have heard. However, while they tend to be loquacious, their verbal communication is lacking in complex meaningful content and their actual comprehension of complex material often is significantly compromised. Mixed expressive-receptive language disabilities are often diagnosed in these children. Severe language delays are seen more commonly in children diagnosed with the full manifestations of FAS (Adnams et al, 2001).

Visual-motor integration and visual-perceptual deficits are also commonly reported in FASD. Some potential problems with deficits in this area can include reading disorders, impaired appreciation of spatial orientation, and these can impede the ability to detect and understand nonverbal communication that is critical to successful social interaction.

FASD individuals are often identified with learning disabilities. Specifically, mathematics skills are often impaired, especially relative to their single word reading and spelling abilities. This often manifests as difficulties with money management and telling time. Significant reading comprehension and written expression, and learning impairments are also common, and these typically become more evident as the child progresses through school.

Poor response inhibition, impulsiveness, and perseverative behavior also present a problem for an individual with FASD, often giving them the appearance of having Attention Deficit Hyperactivity Disorder. In a review of the literature on the link between ADHD and FASD, it has been asserted that in children with FASD, ADHD is more likely to be of earlier-onset, inattention subtype, and to have comorbid developmental, psychiatric, and medical conditions (O'Malley and Nanson, 2002). Other researchers have found that FASD children do not show deficits in sustained attention, the ability to remain alert over time, but have deficits in focused attention, the ability to maintain attention in the presence of distraction (Coles et al, 1997; Coles, 2001). Hyperactivity may also be attributable in FASD to social and environmental factors that frequently accompany the diagnosis including attachment disorder, post traumatic stress disorder and anxiety disorders (Jacobson and Jacobson, 2003). ADHD in children with FASD is also more often poorly responsive to standard medical intervention, including psychostimulants (O'Malley et al, 2000).

Executive functioning, including the ability to plan, cognitive flexibility, selective inhibition, and concept formation, has consistently been reported to be impaired in FASD. These difficulties can be apparent in measures designed to be more sensitive to dorsolateral frontal lobe functioning such as the Wisconsin Card Sorting Test, as well as on measures sensitive to orbital medial frontal lobe functioning such as response inhibition testing. These impairments will often lead to marked adaptive deficits. For example, their ability to problem solve and make appropriate choices in emotionally-weighted or social situations is significantly compromised. This is further confounded by their compromised ability to generate effective alternate strategies to solve a problem, even if they recognize that their first attempt was not successful.

Individuals with FASD can also be compromised in their ability to take the perspective of another person. Self-reflection and insight into their actions is typically very limited. Therefore, they will often fail to demonstrate effective reciprocal social behavior, which serves to alienate them from others.

Adaptive skills are also consistently reported to be deficient in individuals with FASD (Thomas et al, 1998). Particular areas of adaptive impairment include social skills, emotional maturity, money and time concepts, and comprehension. On the other hand, physical maturity, basic reading ability, and basic expressive language abilities tend to be normally developed or only mildly impaired. As a result, these individuals may appear physically mature, are able to meet basic literacy demands, and are able to use relatively sophisticated language. This combination of relative strengths will give them the appearance of functioning at a level consistent with their chronological age. However, their other deficits often impede their ability to function independently. This mismatch between the demands and expectations placed upon them as compared to their actual underlying ability likely contributes to the development of secondary disabilities common in FASD.

### Neuropsychological Assessment of Primary Disabilities

A comprehensive neuropsychological assessment is important to assess all areas of neurocognitive and adaptive functioning, and should explore patterns of strengths and weaknesses. This should include an assessment of sensory perceptual functioning, gross and fine motor skills, visual-motor integrative abilities, visual-spatial and visual-perceptual skills, attention and processing speed, expressive and receptive language, auditory and visual learning and memory, executive functioning, and both IQ and academic testing. This information should be combined with adaptive and behavioural data, using tools such as the Behaviour Assessment System for Children, Conner's, and the Adaptive Behaviour Assessment System. Patterns of weaknesses can be used to help determine the presence of chronic organic brain dysfunction, which is a critical component of arriving at a FASD diagnosis. Identified strengths can provide valuable information to help the treatment team develop individualised treatment programs.

Comprehensive neuropsychological assessment may not be available in all cases due to limited resources or a lack of available professionals. Another means of undertaking a comprehensive neuropsychological assessment is through the use of a team of interdisciplinary health care providers, including a speech-language pathologist, occupational therapist, and psychologist. A full psychological assessment will be able to provide useful information for treatment planning and helping other professionals better understand the psychological functioning of the person identified with FASD. Unfortunately, the diagnostic risk

of false negatives when relying solely on a general psychological assessment or basic cognitive screening tools is high due to the probability of their IQ and basic rote verbal skills being within normal limits, while many other critical areas of cognitive functioning are significantly impaired. Therefore, when the clinical picture of disability is not fully captured by a basic psychology assessment, consideration should be made to undertake a comprehensive neuropsychological assessment. It is important to note that the cognitive and behavioural profile of children with FASD can change over time. Therefore, repeated neuropsychological assessment may be needed at different times during the life of an individual with FASD to refine their FASD brain-related diagnosis, and to accurately capture their evolving strengths and weaknesses, and to plan appropriate interventions.

### Diagnostic Models for Brain Dysfunction

In the past, diagnostic approaches and guidelines were based on a gestalt approach (Sokol and Clarren, 1989; Rosett, 1980; Clarren and Smith, 1978). At present there are two different models for diagnosing fetal alcohol spectrum disorders: the Institute of Medicine (IOM) model (Stratton et al, 1996), and the FAS Diagnostic and Prevention Network (DPN) model (Astley and Clarren, 1999). Both of these diagnostic models have specific approaches to understanding brain dysfunction in individuals with FASD, and were developed to make FASD diagnoses more objective, explicit, reliable and valid.

### The Institutes of Medicine Model

The IOM model was the first attempt to define a consensus approach to the diagnosis of FASD (Stratton et al, 1996). The IOM committee delineated five diagnostic categories for FASD. These include FAS with a confirmed maternal alcohol exposure, FAS without confirmed maternal alcohol exposure, partial FAS with confirmed maternal alcohol exposure, alcohol–related neurodevelopmental disorder (ARND), and alcohol-related birth defects (ARND) (Stratton et al, 1996). One of the key questions raised by the IOM committee was "can behavioral or cognitive features be used to define the disorder?" The IOM diagnostic criteria for FASD does include evidence for central nervous system or neurodevelopmental abnormalities as one component of the diagnostic framework. They state: "No single expression of structural or functional brain damage is universal or pathognomonic when patients with the FAS face and a clear history for substantial alcohol exposure are reviewed. Evidence of abnormality in this field may be structural, neurologic, or functional (Stratton et al, 1996)." For FAS, this is stipulated as at least one of the following: decreased cranial size at birth, structural brain abnormalities (microcephaly, partial or complete agenesis of the corpus callosum, cerebellar hypoplasia), or neurological hard or soft signs such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, or poor eye-hand coordination (Stratton et al, 1996). The diagnosis of partial FAS or ARND requires evidence of neurodevelopmental abnormalities as described above, but these diagnoses can also be made based on the finding of evidence for a complex pattern of behavioral or cognitive abnormalities not consistent with developmental stage, and that cannot be explained by familial background or environment. This "complex pattern of behavioral or cognitive abnormalities" is further specified to include learning difficulties, deficits in school performance, poor impulse control, problems in social perception, deficits in higher level receptive and expressive language, poor capacity for abstraction or metacognition, specific deficits in mathematical skills, or problems in memory, attention or judgment (Stratton et al, 1996).

Clearly, the IOM recognized that their work in this area was incomplete as a key recommendation for further research was the "investigation of the differences in expression and specificity of behavioral and cognitive deficits in FAS and ARND (Stratton et al, 1996). They state that "a variety of behavioral and cognitive features have been proposed as indicators of brain dysfunction in FAS . . . at present, however, no consensus has been achieved as to which features are most appropriate for the diagnosis of FAS, or indeed whether any are appropriate (Stratton et al, 1996)." The IOM also recognized that the behavioral and cognitive effects of prenatal alcohol exposure fall on a continuum that ranges from normality to impaired, that they change with time, that severity of presentation is not necessarily reflective of type of FASD diagnosis, and that FASD can occur in association with other developmental and psychiatric conditions (Stratton et al, 1996). They also acknowledged that the behavioral and cognitive deficits seen in individuals with FASD are also influenced by other factors such as genetic contributions, educational experience, impoverished postnatal environment, and other social and cultural influences (Stratton et al, 1996).

### The FAS Diagnostic and Prevention Network Model

The DPN model was developed to address previous limitations in FASD diagnosis via a quantitative approach. Aims of this model are to provide a specific, accurate and precise diagnosis, to provide objective quantitative scales to measure the magnitude of key diagnostic features, and to separate outcome and exposure (Astley and Clarren, 1999; Astley and Clarren, 2000).

The DPN model provides case-definitions for brain damage or dysfunction along a continuum of probability using a Likert scale from 1 (unlikely) to 4 (definite). The 4-point brain dysfunction scale allows patients with clear evidence of brain damage to be differentiated from patients without evidence of brain damage. The higher the number the more certain the patient's cognitive and behavioral problems stem from brain damage. But a higher score does not necessarily mean a more severe expression of functional disability. A brain ranking of 4 (definite brain damage) and 3 (probable brain damage) are defined as "static encephalopathy." Static encephalopathy means non-progressive brain dysfunction, but the term does not define or suggest any specific pattern of structural abnormality of cognitive/behavioral dysfunction. (Astley and Clarren, 1999). A Likert rank of 4, or definite organic brain damage, is based on evidence for structural or neurological brain damage, including microcephaly, structural abnormalities on brain imaging likely of prenatal origin, evidence of persistent neurologic findings likely to be of prenatal origin (such as seizure disorder, or cerebral palsy), or a full-scale intelligence quotient (FSIQ) less than or equal to 60 (which was felt to fall clearly below the normal dis-

tribution). A Likert rank of 3, or probable organic brain damage, is supported by psychometric test outcomes that document abnormal brain function in three or more areas with test scores that are less than 2 standard deviations below the mean. Although this criterion is not based on scientific data, the authors state that using the cut off of three or more deficits is more reflective of brain damage than one or two areas of deficit, and that this assertion is supported by face validity in their study population. A brain ranking of 2 (possible brain damage) is defined as "neurodevelopmental disorder." The term neurobehavioral disorder is used when the patient presents with cognitive/behavioral dysfunction, but structural, neurologic and psychometric measures do not unequivocally support the presence of structural brain abnormalities (Astley and Clarren, 1999). A Likert score of 2 is given either to those who are too young to receive psychometric testing, or to those individuals for which there is a strong possibility for brain damage, based on psychometric testing deficits. A brain ranking of 1 (unlikely brain damage) does not receive an FASD brain-related diagnostic label, and is given when there are no identified structural, neurological or cognitive/behavioral problems.

One strength of the DPN model is that it does not establish a causal link between behavioral and cognitive deficits and prenatal alcohol exposure, but rather seeks to establish what an individual's disabilities/abilities are, and then suggests what etiologic factors are at play. The DPN model recognizes the complexity of development and behavior, and that alcohol related brain damage is only one piece of a dynamic process that can contribute to the complex primary and secondary disabilities seen in FASD.

### Secondary Disabilities

Secondary disabilities are the behavioral, cognitive, and psychiatric results of living with brain damage. In individuals with FASD, these include mental health problems, disrupted school experience, trouble with the law, confinement, inappropriate sexual behavior, and problems with dependent living and employment in adults (Streissguth et al, 1996). It is recognized that these problems also include an increased risk of addictive behaviors such as alcohol abuse, thereby potentially continuing the cycle of FASD into the next generation (Baer et al, 2003).

Although mental health problems are the most commonly reported secondary problem across all age groups in FASD, and are seen in up to 90 percent of subjects, studies to date have focused primarily on neurocognitive deficits (Streissguth, 1996; Streissguth and O'Malley, 2000). For many of the problem areas, higher IQ increased the probability of having problems, with the exceptions of dependent living and problems with employment. It was also quite striking that the probability of experiencing secondary disabilities was higher if a FASD diagnosis was made after age six. Overall, the strongest protective factors were a diagnosis before 6-years of age and living in a stable nurturing home (Streissguth, 1997). European long-term follow-up studies have had similar findings, including a high rate of mental health disorders in adulthood and a greater need for social and employment support due to deficits in adaptive functioning (Lemoine and Lemoine, 1992; Spohr, 1996; Steinhausen, 1996). In a population-based study in Seattle, researchers found that prenatal alcohol exposure was the major explanatory variable related to antisocial behavior as well as classroom behavior and learning problems in 14-year-olds. This same study also reported a link between prenatal alcohol exposure and adolescent drug and alcohol use (Olson et al, 1997). Psychiatric illness and DSM-IV diagnoses have also been studied in a sample of children between the ages of 5 and 13 years with heavy prenatal alcohol exposure. Children with IQ's of 70 and above were assessed and approximately 87 % of the sample met criteria for a psychiatric disorder. The majority (61%) were diagnosed with a mood disorder. Criteria for bipolar disorder were met in 35%, and 26% met criteria for a major depressive disorder or an adjustment disorder with depressed mood. This same study also found that children with partial FAS or no physical stigmata of FAS were just as likely to have significant mental health problems as those with obvious physical characteristics of FAS (O'Connor et al, 2002). Studies in adults with FASD have reported similar findings suggesting that these problems persist in adulthood. In a recent study of adults exposed to alcohol prenatally, 44% were diagnosed with a major depressive disorder, 40% had psychotic disorders, and 20% had bipolar disorder (Famy et al, 1998). One possible explanation for the increased risk of mood disorders comes from recent MRI studies of children prenatally exposed to alcohol compared to adults with depressive disorders. In both groups, there is underdevelopment of the structures controlling mood in the fronto-cortical network, the basal ganglia (in particular the caudate nuclei) and the cerebellum (O'Connor et al, 2002).

### Conclusion

Psychiatrists, pediatricians and neuropsychologists must collaborate closely to sort out the complexities of brain and behavior in individuals diagnosed with FASD. Ultimately, it must be recognized that the degree of impairment, including an accurate physical diagnosis, comprehensive testing of the primary disabilities, and close surveillance for secondary disabilities for each person with FASD must be fully explored. A common misperception is that the person with the full manifestations of FAS will be more impaired in all areas as compared to the person diagnosed with another FASD diagnosis. To the contrary, persons with FAS exhibit fewer secondary problems than other FASD, likely due to the fact that they have the physical stigmata of FAS, and hence their impairment is recognized and interventions provided at an earlier age. Without a comprehensive neuropsychological assessment, as this article has outlined, there is a risk of underestimating the degree of impairment in individuals with FASD, and thereby placing inappropriate demands and expectations on them.

It will be necessary in the field of psychiatry to include the FASD diagnosis in the Diagnostic and Statistical Manual of Psychiatric Disorders, so that the unique neurobehavioral and cognitive features of this condition can be accurately identified and diagnosed. Understanding the underlying brain-based aspects of this disorder should assist in improved medical, pharmacological and societal management of individuals affected by this common and ultimately preventable condition.

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# Alcohol Use and Abuse in Pregnancy: An Evaluation of the Merits of Screening

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### Introduction

The current recommendation from Health Canada is that women abstain completely from alcohol if they are pregnant or are attempting to conceive, as a safe level of alcohol consumption during pregnancy has not been determined (Health Canada, 1996). Maternal alcohol use in the prenatal period is of concern as it may result in Fetal Alcohol Spectrum Disorder (FASD), which represents the most common form of mental retardation and birth defect in Canada (Health Canada, 1996 & 2000). If alcohol is avoided during pregnancy then FASD can be prevented (Health Canada, 2000b). Approximately 16.6% of women who were surveyed in the 1996-1997 National Longitudinal Survey of Children and Youth reported some drinking during pregnancy with regional variation of 24.9% in Quebec and 7.7% in the Atlantic provinces (Health Canada, 2000a). These prevalence values are believed to be underestimates given the stigma associated with prenatal alcohol use (Health Canada, 2000a).

Research into the relationships between alcohol type and frequency of use, gestational age, nutritional status, maternal and fetal genetic susceptibility, and the occurrence of FASD is ongoing with the hope that this will help target prevention activities. Meanwhile, screening for the main explanatory variable in FASD, prenatal alcohol consumption, continues to be inadequate. A recent Canadian survey of health care providers showed that fewer than 50% of providers frequently discussed risks of alcohol use during pregnancy among all women of childbearing age or obtained a detailed history of addictions (Health Canada, 2003). Additionally, fewer than 15% of providers frequently obtain a detailed history of sexual abuse among women of childbearing age, a known risk factor in alcohol use in pregnancy (Astley et al, 2000).

There are suggestions for routine screening programs that can assist physicians both to detect and deter alcohol consumption by pregnant women and to intervene with affected children at an early age (Health Canada, 2000b). Alcohol screening program development is particularly important to psychiatrists because improved diagnosis or clinical suspicion of FASD by referring physicians post-screening may dramatically increase those identified with the diagnosis in psychiatry practices. Psychiatric assessment of patients may benefit from records of alcohol exposure in utero and improve clinical diagnosis and management.

At this time there are no universal programs for screening for in utero alcohol exposure in Canada, however there are several screening approaches that can be initiated by healthcare professionals including clinical interview, standardized questionnaires, and biochemical tests. An alcohol-screening program could incorporate screening and treatment elements currently available and be applied universally to mothers and babies. It is important to identify all those with disease, therefore, the program or combination of tests should be highly sensitive (i.e., those with disease screen positive) while specificity (i.e., those without disease screen negative) is less important.

### Evaluation of the criteria for screening

While screening for alcohol use in pregnancy may have some benefits, it should be carefully evaluated for scientific, medical, economic and ethical merit. This review will use the World Health Organization's (WHO) criteria for health screening programs to briefly evaluate if a screening program for alcohol exposure in pregnancy is warranted (Wilson & Jungner, 1968).

**1. Suitable tests should exist.**

***No single appropriate test has been developed, but the combination of several existing tools could be used in a screening program.***

***Clinician interview/Self-report*** - The accuracy of self-reporting of alcohol use during pregnancy in clinician interviews can be highly variable (Chang et al, 1998; Chasnoff, 1989). Self-report depends on both a women responding truthfully and on a clinician asking the question. Women who drink heavily during pregnancy may not admit to alcohol consumption due to guilt, shame, or fear of consequences. Chasnoff (1989) notes that an informal interview of women inquiring about alcohol and drug exposure results in under-reporting, whereas a more formal and organized interview increases reporting five-fold.

***Standardized Questionnaires*** - Standardized questionnaires that are easy to use and quickly administered have been developed including the AUDIT, CAGE, TWEAK, and T-ACE (Bradley et al, 1998; Chang et al, 1998). The TWEAK and T-ACE were found to be sensitive in the prenatal population. However, these tools alone do not accurately identify all mothers and infants at risk. The Alberta Medical Association has published guidelines, 'Recommendations on Prevention', related to FASD, which include the use of standardized questionnaires during pregnancy (see the reprint in this issue).

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***Biological Markers*** —Biological markers may offer an objective assessment of infant exposure to alcohol in utero. There are many biomarkers for alcohol use that have been identified: blood alcohol, alcohol metabolites (e.g., fatty acid ethyl esters), enzyme levels (e.g., gammaglutamyl transferase), and altered proteins (e.g., carbohydrate deficient transferrin) (Bearer, 2001). Recently, the technical potential to assess fatty acid ethyl esters (FAEE) in meconium as a measure of alcohol exposure in utero was described (Bearer et al, 1999). Fatty acid ethyl esters are produced by an enzymatic process involving esterification of alcohol with free fatty acids. Meconium, a neonate's first stool, contains a biological record of the total quantity of prenatal alcohol exposure in the last half of pregnancy. However, it is unclear how timing and type of drinking affects the amount and type of FAEEs in meconium.

In a study of nonalcoholic women who self-reported varying amounts of alcohol use during pregnancy, Bearer et al (1998) analyzed FAEE levels and types in meconium. They found that the sensitivity of FAEE analysis of meconium was 72% and the specificity was 51% in distinguishing the consumption of at least one drink per week in the third trimester. In later studies the author found that levels of FAEE increased in a dose-dependant manner with an increase in maternal self-report of alcohol use (Bearer, 2001). This potential tool for prenatal alcohol exposure is being further evaluated.

**2. The disease or condition that is being screened for should be important medically, socially, or economically.**

The prevalence of Fetal Alcohol Syndrome in Canada is estimated at 1-3 per 1,000 live births while the prevalence of FASD is estimated to be approximately 10 times higher (Health Canada, 1996, 2000b). Secondary disabilities of FASD may include mental health problems, disruptive school experience, alcohol and drug addiction, criminal behavior, and inappropriate sexual behavior (Streissguth et al, 1990 and 1991). The estimated cost for additional education, disability payments, incarceration, and health care per individual with FASD is as high as $3.0 million over their lifetime (Alberta Health, 2000). In Alberta, up to 29% of children in government care and 60% of the prison population suffer from the effects of FASD (Alberta Health, 2000).

**3. The natural history of the disease should be understood and the population at risk should be identifiable.**

***The natural history of FASD is fairly well understood, however more work is required to identify the role of genetics, diet, and type and frequency of alcohol use.***

Alcohol is a teratogen which disrupts normal development in offspring through exposure during pregnancy. The specific body system affected and the long-term outcome seems to depend on when the exposure occurs. No single type of CNS damage has been identified that characterizes FASD and the association between exposure and outcome is dependent on a host of factors including genetic and maternal characteristics, diet, patterns of alcohol use, genetic susceptibility, and maternal lifestyle. Neurobehavioral deficits associated with alcohol exposure may result from drinking at any time during pregnancy.

There are reports that indicate that women who engage in binge drinking during pregnancy are more likely to smoke cigarettes, have a history of sexual abuse and addictions, be in a relationship with a heavy drinker, and be young and single (Gladstone et al, 1997). Other studies found that as maternal age and parity increase, the risk of FASD increases (Jacobson et al, 1996). No clear and consistent risk factors for FASD, with the exception of prenatal alcohol use, have been identified.

**4. The test should be acceptable to the population.**

***There is insufficient evidence on the acceptability of test methods within the perinatal population and with the clinicians who would be administering them.***

The tests described are non-invasive. The factors that may influence acceptability in the population are how a screening program is presented, level of education and knowledge about the program, how test results are used and repercussions for a positive screen. Resources to educate women about testing in the prenatal period and counsel women about a positive test result after birth would have to be in place.

**5. The condition should be recognizable at an early stage.**

***Early identification is possible but challenging. This may be aided by documented alcohol use or exposure.***

Currently, FASDs are believed to be under-diagnosed and many children are not diagnosed until schoolaged; however, FASD can be recognized early, when interventions may be most successful (Sampson et al, 1997; Clarren et al, 2001). Some children may not have the full complement of physical or developmental characteristics of FAS but may have spectrum deficits which are not easily identifiable. Early identification of affected infants is difficult because the characteristic facial dysmorphia may not be evident until school age, if at all (Astley et al, 1995; Clarren et al, 2001).

**6. There must be an accepted and effective treatment for the condition.**

Two interventions must be considered: interventions to reduce alcohol use during pregnancy and interventions to reduce secondary disabilities in children with FASD.

Brief interventions delivered by a care provider that incorporate elements of counseling, feedback, monitoring of drinking habits, self-efficacy and exercises on counting drinks are effective in reducing prenatal alcohol use (Grant et al, 1996). A recent multi-site pilot study showed that four sessions of motivational interviewing and a contraception counseling session decreased the risk of an alcohol exposed pregnancy in 68% of participants (Ingersoll et al, 2003). These interventions are more successful than simple advice. The more elements of intervention used, the more effective the intervention.

Protective factors that can minimize secondary disabilities include early diagnosis, access to resources, involvement in special education, and a stable and nurturing caregiving environment (Streissguth et al, 1996). Typical interventions include provision of funding for classroom assistants, therapy, and respite care for parents. In many cases the guardian is an adoptive or foster parent who may require access to resources and aid in caring for an affected individual. However, there is only moderate evidence supporting better outcomes and less severe and lower

frequency of secondary disabilities with early detection of FASD. One study of a large sample of children and adults with FAS indicated that those diagnosed before age six had lower rates of secondary disabilities than those diagnosed later (Streissguth et al, 1996). There is a consensus in the literature and among FAS experts that early diagnosis is a protective factor that can minimize secondary disabilities. However, further research on the effectiveness of interventions is required.

**7. There should be facilities for assessment, diagnosis, and rehabilitation.**

Facilities exist for the assessment, diagnosis, treatment and referral of affected individuals in Centres across Canada. However, these facilities are not able to quickly see all individuals currently identified and care provision is not standardized. Following the implementation of a screening program, the capacity of these facilities would be further strained. Screening at several levels with trained community physicians and paediatricians assessing individuals at risk and referring to clinical FASD specialists would be required. There are Canadian programs to develop capacity and introduce standardization related to assessment, diagnosis, and treatment of FASD.

**8. Interventions should be acceptable to the population.**

Interventions with women are non-invasive and do not usually involve increased financial costs for the individual. However, there is an increased time-cost for the clinician delivering the intervention. While it is compelling that interventions to decrease alcohol use in pregnant women can be effective, women have identified legal and ethical rights to use alcohol throughout pregnancy, which confounds the acceptance of maternal screening strategies and maternal interventions by the general public and healthcare professionals (Flagler et al, 1998).

Interventions with children may be less acceptable to the population. Guardians may have concerns related to identification and treatment of affected individuals as FASD may be an undesirable label, even leading some to prefer an alternative diagnosis of attention deficit or hyperactivity disorder. A diagnosis of FAS identifies a mother as a woman who drank during pregnancy and harmed her child, which has associated social stigma.

**9. The cost of screening should not be disproportionate to cost of caring for affected individuals.**

The cost of implementing a screening program cannot be adequately addressed within the scope of this review. However, some comments on the nature and magnitude of costs are possible. Many of the questions related to alcohol use are included on several provincial Prenatal Records. The opportunity exists to collect this data from all pregnant women. These standardized forms provide opportunities to counsel women regarding alcohol use and to identify infants potentially at risk for FASD. Currently, the standardized forms are not used in a standardized fashion. Therefore, an initial low cost opportunity includes improved usage of existing forms through clinician training relating to assessment and follow-up of women. However, the cost for universal biomarker screening would be substantial, and as per existing screening programs for newborn hearing deficiency there would be additional costs for follow up evaluation of patients that screen positive.

Considering that an estimated 60% of individuals in Alberta penal institutions have FASD the potential cost benefits for screening are easily conceptualized (Alberta, 2000). If secondary disabilities related to FASD, including violent crime, were ameliorated by early detection and intervention then a decrease in the number of individuals in prison or care would be expected. This decrease and the accompanying societal savings may offset the cost of a screening program. However, there would be a substantial time lag between inception of a program and evaluation and realization of benefits.

**10. Screening programs should be a continuing process.**

A screening program for FASD would have to be used universally and become part of ordinary and ongoing care to be effective.

**Conclusion**

This article briefly reviews the evidence for screening for alcohol use and abuse in the perinatal period using WHO criteria. There is some evidence of the benefits of such a program but limited evidence of the effectiveness of screening tools, interventions, and the current capacity of the health care and mental health systems to deal with individuals identified at risk. This review highlights the importance of using standardized screening methods for alcohol use and abuse during pregnancy and with women of childbearing age. The Canadian Psychiatric Association Practice Profile survey showed that women comprise almost 60% of psychiatry practices, hence there may be a public health benefit for psychiatrists to routinely screen for alcohol use as part of the psychiatric intake process. This screening should also encompass other known risk factors associated with alcohol abuse during pregnancy. Psychiatrists have an important role in the management of high-risk pregnant women, including treatment of the addictions and the management of the often multiple and severe psychiatric conditions. Further research is needed to identify screening methods, respectful, acceptable and effective interventions with mothers, and effective intervention and support programs to help children with FASD who are at risk for secondary disabilities.

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The following pages (81-91) are a reprint of the Alberta Clinical Practice Guidelines — a publication of the Alberta Medical Association. Thank you to the AMA for allowing The Canadian Child and Adolescent Psychiatry Review to publish them here.

ALBERTA PARTNERSHIP ON Fetal Alcohol Syndrome — Together Let's Find a Solution

ALBERTA CLINICAL PRACTICE GUIDELINES

Administered by the Alberta Medical Association

# Guideline for the

# DIAGNOSIS OF FETAL ALCOHOL SYNDROME (FAS)

*This guideline was developed by a working group based on best available evidence and from a province-wide survey of physicians. The development of this guideline was funded as part of the Alberta FAS Initiative and in cooperation with the Prairie Province FAS Initiative.*

**For the purpose of this guideline, the term FAS (FAE, ARBD) is used to describe the full continuum of abnormalities attributed to prenatal exposure to alcohol (see preface).**

## GOALS

This guideline is intended to:

- assist health care professionals to recognize the disorders associated with fetal alcohol exposure.
- promote early (infancy and preschool) and accurate diagnosis.
- prevent secondary disabilities[1] through early diagnosis.
- prevent future FAS children in affected families by offering interventions to families which will enable them to abstain from alcohol use when planning or during pregnancy.

*Secondary disabilities:*[1]
- *mental health problems*
- *disruptive school experience*
- *trouble with the law*
- *inappropriate sexual behaviour*
- *drug/alcohol problems*

*Protective factors:*
- *early diagnosis before age 6*
- *stable and understanding caregiver in a non-abusive environment.*
- *access to resources for person's with disabilities.*

## RECOMMENDATIONS

- The standard for diagnosis of FAS includes the following clinical indicators (Tables 1 and 2):
  - a history of maternal alcohol consumption during pregnancy;
  - prenatal and/or postnatal growth retardation
  - neurodevelopmental and behavioral characteristics
  - characteristic facial features (see diagram in background section)
- Primary care providers should refer any child, adolescent or adult suspected to have FAS to an appropriate specialist such as: a pediatrician, psychiatrist, psychologist, for further assessment.
- Once a diagnosis has been made:
  - specific advice and contraceptive counselling can prevent further births of alcohol affected children.
  - aggressive intervention measures with the help of a multidisciplinary team* can improve the outcome for the individual; and
  - provide information and support to family/caregivers.

* *A multi-disciplinary team for care and management could include, at minimum, two or three professionals, depending on need and availability within the area; and could be comprised of the following professionals: physicians, nurses, psychologists, speech pathologists, occupational therapists, educators, and social workers.*[2] *(Refer to Figure 2 on back cover)*

## BACKGROUND ON DIAGNOSIS

### History of Maternal Drinking

Establishing the history of alcohol consumption is one of the most difficult issues in diagnosing FAS. The pregnant woman who consumes alcohol is not always easily identified. Patients usually are not forthright about their drinking habits nor are they necessarily able to recall the precise quantities and timing of their drinks. However, in the absence of a specific biomarker to detect alcohol exposure, the history remains pivotal in the diagnosis.

The challenge for the physician is to identify women who are drinking alcohol during pregnancy. Problem drinkers cannot be identified by appearance or by socioeconomic characteristics. **A systematic drinking history is essential and should be obtained from all patients during the initial history and in subsequent prenatal care.**

Taking a history of maternal drinking can be helped by specific screening tools, included in the Prevention of FAS Recommendations.

### Physical and Neurological Features and Characteristics

In the most severely affected children, FAS can be diagnosed at birth, however, the characteristic physical features are most pronounced between eight months and eight years of age.[3] Facial abnormalities observed in affected children are the key cluster of physical features of FAS.[4] As the child approaches adolescence, the typical facial features become less pronounced. In some adults, facial characteristics have become so normalized that early childhood photographs must be used to confirm diagnosis.[5] Some authors suggest that FAS may not be recognized until postnatal growth retardation and developmental delay become apparent.[6] Abnormalities in neurodevelopment and behavior are usually evident. Alcohol related birth defects (cardiac, skeletal, renal, ocular, auditory) occasionally occur as well (Table 2).

*No single feature alone can be used to diagnose FAS*

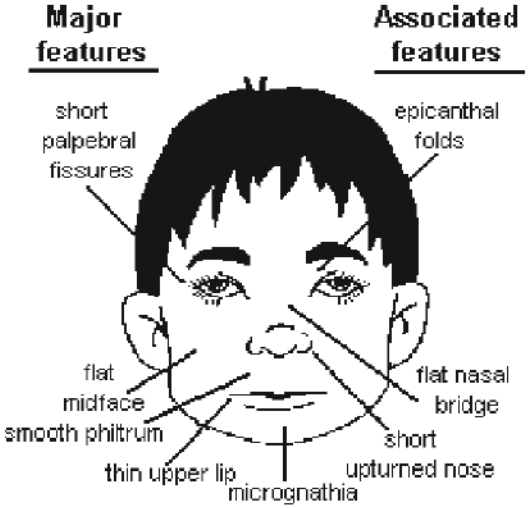
#### *Growth failure*

Alcohol exposure in-utero can cause growth failure either apparent at birth or postnatally.

#### *Facial features*

No single facial feature is diagnostic of FAS, but the constellation of short palpebral fissure, smooth philtrum and thin vermilion upper lip are characteristic features.

**Figure I**




*Redrawn from Streissguth & Little, 1994*

### Neurodevelopmental and behavioral characteristics

FAS results in abnormalities of cognition, language, and behavior. The expression of these abnormalities changes from birth to adulthood.

In infancy and early childhood (0-5 years) they include delayed developmental milestones, poor sleep/wake cycle, attentional deficits, impulsivity, and difficulty adapting to change.

From ages 6 to 11, the following may also appear: significant learning difficulties, cognitive delay, an inability to appreciate cause and effect, and poor understanding of social expectations.

In adolescence and adulthood, these difficulties lead to problems with independent living, competitive employment, social integration, and involvement with the legal system.

Concomitant secondary disabilities, including mental health disorders, problems of substance abuse, and behavior disorder are most obvious during adolescence and adulthood.

**No single diagnostic test is available to confirm FAS. Appropriate investigations need to be undertaken as necessary.**

### Differential Diagnosis

Other medical, psychosocial, and psychiatric conditions/disorders may present similarly to FAS. Usually they can be differentiated by an adequate history and investigation. However, co-morbidities with FAS is a common occurrence.

In an individual presenting with a behaviour disorder or Attention Deficit Disorder, it is important to consider the maternal alcohol use history and a diagnosis.

The diagnostic criteria outlined in Tables 1 and 2 covers the full spectrum of the continuum, recognizing that it is sometimes impossible to confirm maternal drinking. Rather than excluding these cases, it is imperative that the diagnostic criteria enable professionals to make a diagnosis identifying specific areas of difficulty.

### In Conclusion

The diagnosis of FAS relies on a composite of specific physical, psychological and behavioral tests. Specific programs or services for the individual and the caregiver are required for accurate diagnosis and appropriate long-term management.

## REFERRAL SOURCES

Contact your Regional Health Authority, AADAC or the College of Physicians and Surgeons of Alberta for a list of current resources.

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The Working Group encourages your feedback. If you need further information or if you have difficulty applying this guideline, please contact:


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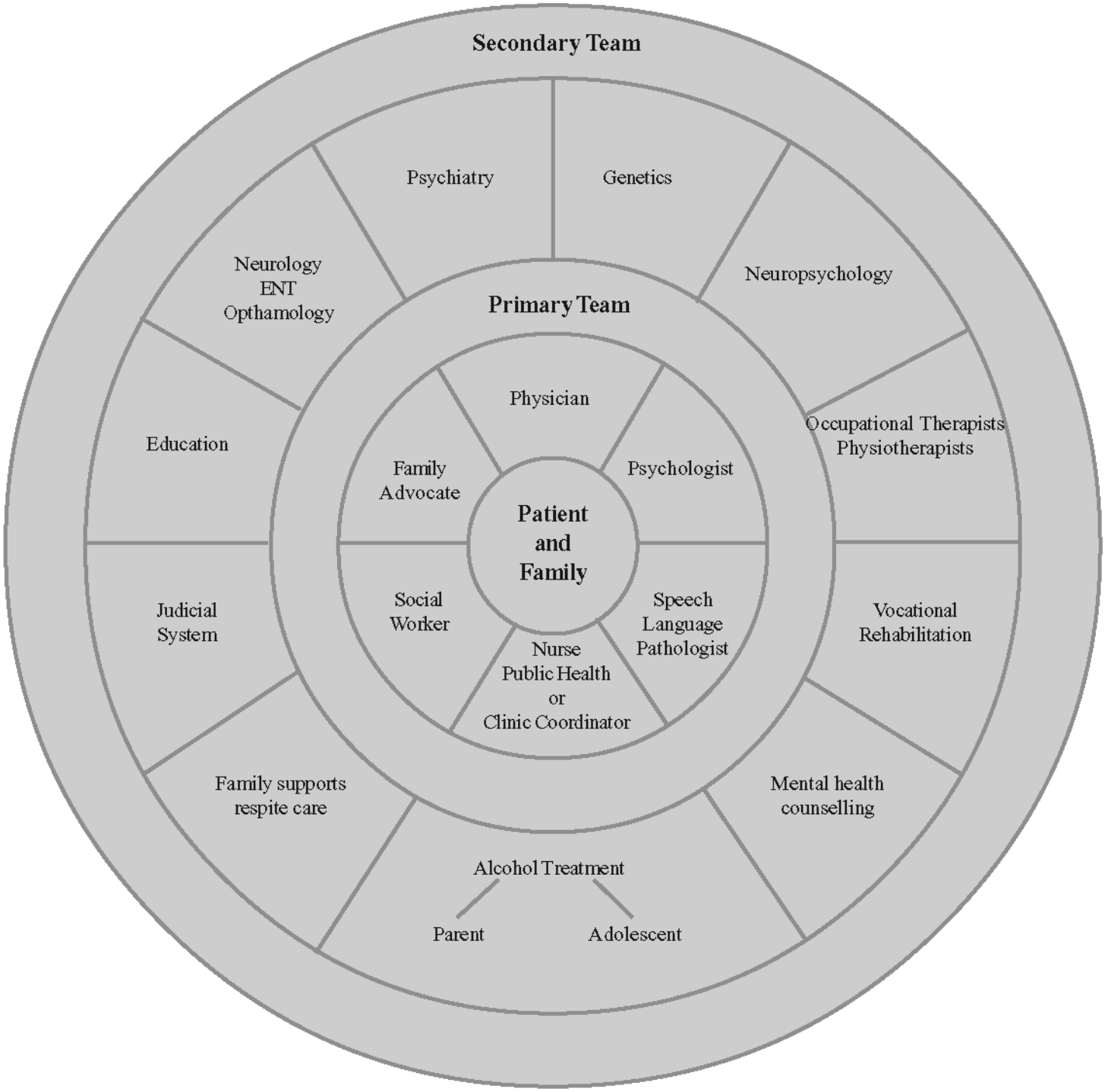
*Diagnosis of FAS - June 1999*

# Figure 2: FAS Multidisciplinary Team Approach




Primary Team = for all patients
Secondary Team = on consultation for individual needs

Adapted from the FAS Clinic Model by Dr. Sterling Clarren as presented at 1999 Alberta: Prairie Province Conference on Fetal Alcohol Syndrome

# Table 1: Diagnostic Criteria for FAS

| | |
|---|---|
| **1.** | **FAS with confirmed maternal alcohol exposure** [1] |
| A | ■ Confirmed maternal alcohol exposure |
| B | ■ Evidence of characteristic pattern of facial anomalies including: short palpebral fissures and abnormalities in the premaxillary zone (e.g., flat upper lip, flattened philtrum, and flat midface) |
| C | ■ Evidence of growth retardation, in at least one of the following:<br>• low birth weight for gestational age<br>• decelerating weight over time not due to other identified causes<br>• disproportional low weight to height |
| D | ■ Evidence of CNS abnormalities in at least one of the following:<br>• decreased cranial size at birth<br>• structural brain abnormalities (e.g., microcephaly, cerebellar hypoplasia)<br>• neurological hard or soft signs (as age appropriate), such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, poor hand-eye coordination |
| **2.** | **FAS without confirmed maternal alcohol exposure** [1] |
| | ■ B, C, and D above |
| **3.** | **Partial FAS with confirmed maternal alcohol exposure** [1] |
| A | ■ Confirmed maternal alcohol exposure |
| B | ■ Evidence of some components of the pattern of characteristic facial anomalies AND either C or D or E below |
| C | ■ Evidence of growth retardation, in at least one of the following:<br>• low birth weight for gestational age<br>• decelerating weight over time not due to nutrition<br>• disproportional low weight to height |
| D | ■ Evidence of CNS abnormalities in at least one of the following:<br>• decreased cranial size at birth<br>• structural brain abnormalities (e.g., microcephaly, cerebellar hypoplasia)<br>• neurological hard or soft signs (as age appropriate), such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, poor hand-eye coordination |
| E | ■ Evidence of a complex pattern of behavior or cognitive abnormalities that are inconsistent with developmental level and cannot be explained by familial background or environment alone, such as learning difficulties; deficits in school performance; poor impulse control; problems in social perception; deficits in higher level receptive and expressive language; poor capacity for abstraction or metacognition; specific deficits in mathematical skills; or problems in memory, attention, or judgement. |

1. A pattern of excessive intake characterized by substantial, regular intake or heavy episodic drinking. Evidence of this pattern may include frequent episodes of intoxication, development of tolerance or with drawal, social problems related to drinking, legal problems related to drinking, engaging in physically hazardous behavior while drinking, or alcohol related medical problems such as hepatic disease.

## Table 2: Diagnostic Criteria for Alcohol Related Effects

| | |
|---|---|
| **4.** | **Alcohol-related birth defects** |
| Cardiac | ■ Atrial septal defects, ventricular septal defects, aberrant great vessels, tetralogy of Fallot |
| Skeletal | ■ Hypoplastic nails, shortened fifth digits, radioulnar synostosis, joint contractures, camptodactyly, clinodactyly, pectus excavatum and carinatum, Klippel-Feil syndrome, hemivertebrae, scoliosis |
| Renal | ■ Aplastic, dysplastic, hypoplastic kidneys, horseshoe kidneys, ureteral duplications, hydronephrosis |
| Ocular | ■ Strabismus, refractive problems secondary to small globes, retinal vascular anomalies |
| Auditory | ■ Conductive hearing loss, neurosensory hearing loss |
| Other | ■ Virtually every malformation has been described in some patient with FAS. The etiologic specificity of most of these anomalies to alcohol teratogenesis remains uncertain |

| | |
|---|---|
| **5.** | **Alcohol related neurodevelopmental disorder** |
| | Presence of A **and/or** B |
| A | ■ Evidence of CNS abnormalities in at least one of the following:<br>• decreased cranial size at birth<br>• structural brain abnormalities (e.g., microcephaly, cerebellar hypoplasia)<br>• neurological hard or soft signs (as age appropriate), such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, poor eye-hand coordination |
| B | ■ Evidence of a complex pattern of behavior or cognitive abnormalities that are inconsistent with developmental level and cannot be explained by familial background or environment alone, such as learning difficulties; deficits in school performance; poor impulse control; problems in social perception; deficits in higher level receptive and expressive language; poor capacity for abstraction or metacognition; specific deficits in mathematical skills; or problems in memory, attention, or judgement. |

Tables adapted from Stratton K, How C, Battaglia. FAS Diagnosis, Epidemiology, Prevention and Treatment Washington, D.C. National Academy Press, 1996

ALBERTA PARTNERSHIP ON Fetal Alcohol Syndrome
Together Let's Find a Solution

Recommendations

ALBERTA CLINICAL PRACTICE GUIDELINES
Administered by the Alberta Medical Association

# PREVENTION OF FETAL ALCOHOL SYNDROME (FAS)

*This guideline was developed by a working group based on best available evidence and from a province-wide survey of physicians. The development of this guideline was funded as part of the Alberta FAS Initiative and in cooperation with the Prairie Province FAS Initiative.*

**For the purpose of this guideline, the term FAS (FAE, ARBD) is used to describe the full continuum of abnormalities attributed to prenatal exposure to alcohol. (see preface)**

## GOALS

This guideline is intended for use by physicians, midwives and other health care professionals to reduce the incidence and disability from FAS by increasing:

- knowledge about screening for alcohol consumption in pregnancy;
- awareness about common attitudes regarding alcohol use and FAS; and by
- promoting productive dialogue and action between healthcare professionals and their patients (and when possible, their partners).

## RECOMMENDATIONS

- A consistent message must be given to all women of childbearing years and their partners that:
  - a prudent choice for women who are pregnant or who are considering pregnany is to abstain from alcohol;
  - inform women who consumed small amounts of alcohol occasionally during pregnancy that the risk to the fetus in most situations is minimal; and
  - reducing or stopping alcohol use at any time during pregnancy will reduce the harmful effects of alcohol.
- Physicians/midwives engage with other health care professionals in basic strategies to prevent FAS in their practices.
- Given the risk of recurrence is high for women (couples) who have had an FAS child, that they are counselled in recognizing and changing their alcohol use patterns in order to reduce impairments and disabilities in future children.

## PREVENTION STRATEGIES

### Primary Prevention

- Engage in education regarding FAS and the adverse effects of alcohol on the fetus with all women and their partners
- Ask all female patients, of childbearing age, the basic question about their use of alcohol
- Be aware of and use promotional materials in offices and as handouts for patients
- Be aware of and access community resources
- Discuss and enhance access to contraceptive strategies with all women and their partners

### Secondary Prevention

- Identify women who are using alcohol during pregnancy and assess level of risk (refer to Algorithm and Screening Details)
- Counsel pregnant women who are using alcohol about the effects on the fetus and their own health
- Counsel pregnant women regarding the benefits of stopping or reducing the use of alcohol at any time during pregnancy
- Refer women who are using alcohol for appropriate treatment
- Provide contraceptive counselling

### Tertiary Prevention

- Identify those women at high risk in future pregnancies
- Ask the woman why she drinks?
- Refer women at risk, especially pregnant women, for appropriate treatment
- Counsel women about the benefits of stopping or reducing alcohol consumption at any time during pregnancy
- Provide contraceptive counselling

***Practice Tip:***
*Alcohol use in pregnancy should be discussed as part of risk reduction counselling.*

The above recommendations are systematically developed statements to assist practitioner and patient decisions about appropriate health care for specific clinical circumstances. They should be used as an adjunct to sound clinical decision making.

## BACKGROUND ON PREVENTION

### Prevention Strategies

The goal of a comprehensive prevention program is to provide overlapping levels of reinforcement (education and persuasion), incentives and interventions to prevent FAS.[1]

### Primary Prevention

Primary prevention involves the elimination of the root causes of a problem by broad-based efforts to promote the health and well-being of a community. These strategies can be applied to the entire population.

In the case of FAS, the goal of primary prevention is to have no fetuses exposed to alcohol, thus eliminating the problems of FAS before they ever develop. Because no safe level of drinking has been identified, ideally all females would cease drinking before conception and during pregnancy.[2] This abstinence would guarantee the primary prevention of all FAS.

#### *Knowledge and Understanding*

To carry out effective prevention strategies, health professionals need knowledge and understanding of the probable etiologic mechanisms of FAS and the factors contributing to the use of alcohol.

Evidence[3-5] shows that information provided by physicians and others in office and prenatal clinic settings is seen as credible and effective in relaying the message.

Clarren[6] suggests that physicians and other health care providers should discuss the notion, prior to conception, that alcohol consumption is potentially harmful to a developing fetus.

#### *Education*

One of the most basic techniques used in primary prevention is public education at all levels.

Information should be directed slightly differently for women and men. It is important to emphasize to women abstinence from alcohol and drugs during pregnancy.[7] For partners and families, the emphasis should be placed on the supportive role that they play in a healthy pregnancy.

**FAS is 100% preventable**
***********
**A prudent choice for women who are pregnant or who are considering pregnancy is to abstain from alcohol**

### Secondary Prevention

Secondary prevention seeks to reduce the duration and severity of maternal drinking by identification of the person at risk. Thus, strategies would include screening, early intervention programs, and services for pregnant women and women of childbearing potential who may be at risk for having a child with FAS.

#### *Professional Education*

Many physicians are reluctant to inquire about alcohol use. One explanation is that they wouldn't know what to do if they found problems.[8] This attitude may be overcome when physicians learn that patients often appreciate professional concern and when they know how to refer problem patients to community resources for treatment.

**Studies[9, 10] indicate that a supportive counselling and/or case management program can result in 60 to 80 percent of pregnant women reducing their alcohol intake before the third trimester and 35 to 50 percent stopping "heavy" drinking**

#### *Screening*

The pregnant woman who consumes alcohol cannot be identified by appearance or by socioeconomic characteristics therefore a systematic drinking history is essential and should be obtained from all patients during the initial history and in subsequent prenatal care.

In response to this clinical need several screening tools have been developed such as the T-ACE screening questionnaire.[11] Although it is understood that many physicians currently use the CAGE screening tool, this committee finds the T-ACE to have higher sensitivity and specificity when used to assess periconceptual heavy drinking. (Refer to the Screening Algorithm)

### *Intervention*

Pregnancy is an ideal time for the physician to identify drinking problems and to intervene. It is reported that alcohol consumption often decreases spontaneously during pregnancy.[12]

> *Once alcohol consumption has been identified, physicians should discuss the fetal risks with the woman and her family in the same way that other risks are discussed. Everything possible should be done to discourage drinking during the pregnancy to prevent harm to the fetus*

In several clinical trials, intervention as a part of prenatal care has been successful in reducing alcohol consumption. Approximately 50 percent of heavy drinkers were able to reduce their drinking to the moderate or rare category and these women had infants with higher birth weights and fewer characteristics of FAS.[13]

> ***Practice Tip:***
> - *Disulfiram should not be used during pregnancy.* [14,15]
> - *Special care in detoxification procedures during pregnancy are needed.*

### Tertiary Prevention

Tertiary prevention is aimed at reducing the complications, impairments, and disabilities caused by FAS, and includes activities that prevent recurrence of the condition in subsequent children. Strategies should be designed and reserved specifically for children with FAS and their caregivers.

### *Treatment/Therapy*

It is important to assist the mother (couple) to recognize and change their alcohol consumption pattern. Specific education and advice for voluntary referral to therapy for alcohol and drug misuse should be pursued with the couple who have an FAS child. This advice should be offered empathically but firmly. A more direct approach is needed if change is not evident over a reasonably short period of time.[16]

> **AADAC has available resources in the Alberta communities that are easily accessible.**

### Future Pregnancies

Once a diagnosis of FAS has been made, aggressive measures are needed to reduce the injury to future children. This is best accomplished by helping the mother (and partner) in changing their alcohol abuse pattern.[17] Studies indicate **that women who have had one definite FAS child and continue to drink will have equally or progressively more severely damaged children in subsequent pregnancies**.[18] Both parity and age are contributing factors. In such cases, isolating the experience of pregnancy from alcohol and substance abuse is the logical and safe course of prevention.

Intensive case management of women who have borne one or more FAS children has been shown to help protect against future FAS children, allow for the care of the specific social and medical needs of the woman and her family, and improve the future health of the mother.[16]

## REFERRAL SOURCES

- Contact your Regional Health Authority, AADAC, or the College of Physicians and Surgeons of Alberta for a list of current resources.

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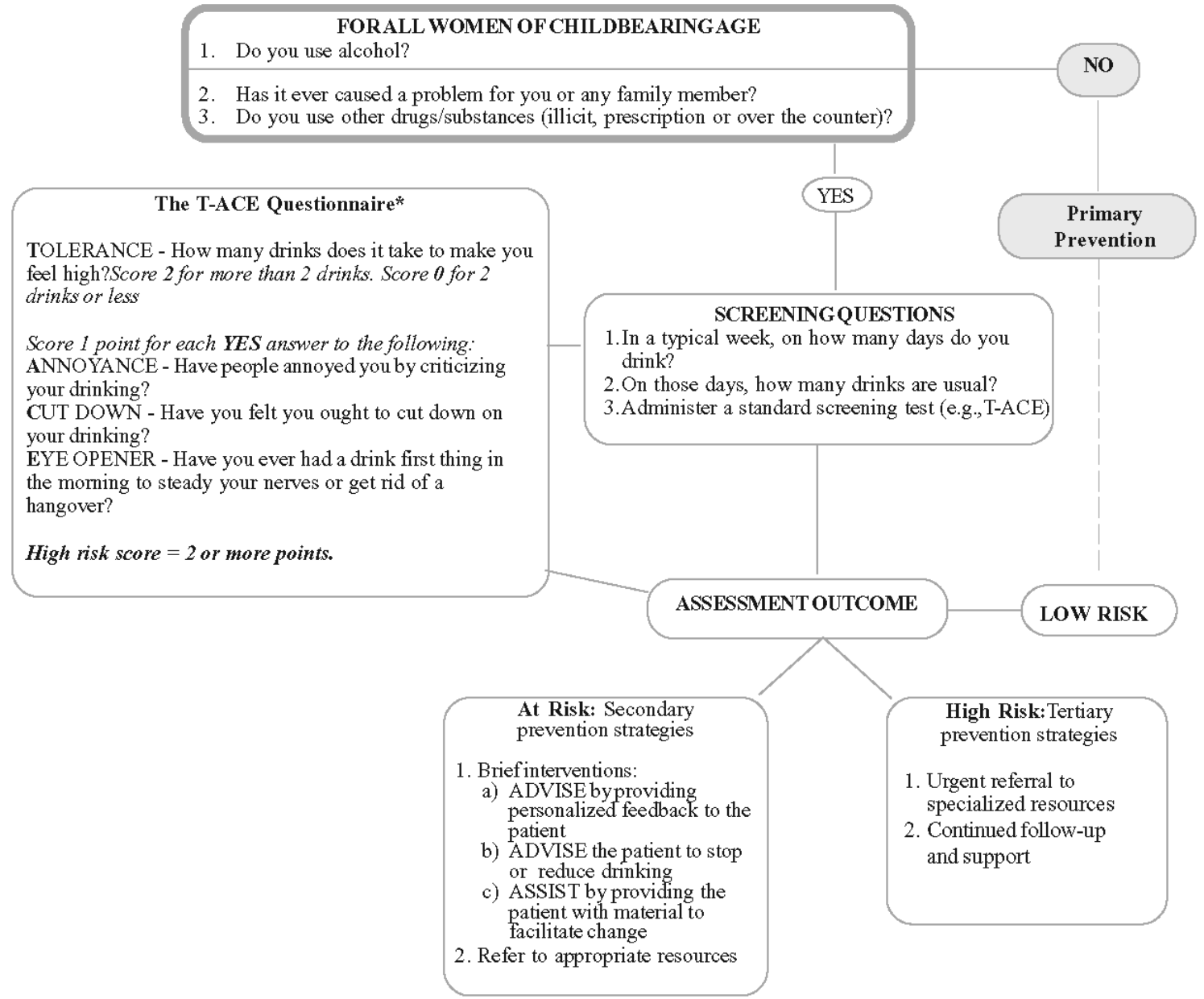
*Prevention of FAS - April 1999*

# ALGORITHM: SCREENING FOR ALCOHOL USE




* The T-ACE questionnaire is an example of one systematic approach to alcohol screening. Reference: Sokol R. et al. The T-ACE Questions, practical prenatal detection of risk drinking. American Journal of Obstetrics and Gynecology, April 1989; 160(4).

# Letter to the Editor

## Some Observations on the Phenomenology of Thought Disorder; a Neglected Sign in Attention-Deficit Hyperperactivity Disorder

To the Editor:

In a recent article on thought disorder in ADHD & schizophrenia in children Caplan et al (2001), present empirical evidence for the presence of clinically identifiable thought disorder in children with ADHD, compare and contrast its phenomenology with that found in schizophrenic children. They found that whilst the internal distraction of "loose associations" are found primarily in schizophrenic children that" digression from the conversational text to referents in the immediate external surrounding (i.e., exophora) can contribute to the clinician's difficulty with following the topic of conversation in both ADHD and schizophrenia." In contrast to schizophrenic children they found evidence of ADHD children showing an "excess of lexical cohesion" in their speech; a semantical rather than an ideational connection The resultant effect to the listener being that " the connections between the speaker's ideas could appear to be tenuous and idiosyncratic"

The phenomenological differences described between these two important neuropsychiatric disorders are in keeping with earlier observations by the author (Jerome 1996). In this earlier work comparing the phenomenology of thought disorder in ADHD and Bi-polar Disorder the observation was made to further the discussion regarding the differential diagnosis of Bi-polar Illness and ADHD (Wozniak, et al 1995 and Pelletier, et al 1996) by comparing the phenomenology of thought disorder in the two conditions.

Clinical observations in children and adults with ADHD were offered as presumptive evidence of what was thought to represent a new phenomenological description of thought disorder in this condition; "Patients with a diagnosis of ADHD describe experiencing "multiple tracks" of thought. These tracks of thought were experienced simultaneously and sometimes in rapid succession switching between two or more different topics. Sometimes up to five or more tracks of thought ran in parallel. These multiple tracks of thought appeared not to have been previously described in the phenomenological literature. These patterns of thought may be described by an external observer as internal distractibility or difficulty maintaining focus on topic in conversation similar to the exophora described by Caplan et al but distinct in their origin; namely multiple tracts of thought not always observable to the external examiner unless enquired about directly. Whilst sometimes accompanied by rapid cluttered speech patterns the phenomenon will often be described by patients with a normal rate of speech, in comparison to Bipolar Patients where flight of ideas and pressure of thought as well as pressure of speech are present and often occur together." Since the initial report I have since noticed that this phenomenological description is often immediately recognisable by other family members; such as a parent with ADHD as applying to themselves and not recognised at all by the other non-clinical relatives who are unable to identify with what is often an invisible, internal, subjective experience only observable by introspection and not normally described by the patient.

Interestingly, child and adult ADHD patients in whom I observed this phenomenon often describe a reduction in the number of tracks of thought and a consequent reduction in internal distractibility with the use of stimulant medication. The thought disorder is reduced. This particular response to medication would seem quite distinct from that seen in Bi-polar Illness and in Schizophrenia where stimulant medication would be expected to either increase the severity of thought disorder or have no immediate effect.

This ADHD thought disorder pattern seems unrelated to observable mood state and is often noted in patients with ADHD who seem otherwise euthymic. In comparison, Bi-polar Illness would invariably be associated with hypomanic or manic mood change observable in the patient. In the initial report (Jerome 1996) I suggested that this difference in the phenomenology of thought disorder and its response to stimulants might be another useful avenue for empirical research in distinguishing ADHD and Bi-polar Disorders.

Caplan et al's argue that the association with cognitive correlates of thought disorder with IQ, Span of Apprehension accuracy, CPT signal/noise discrimination, and digit span scores in ADHD not seen with schizophrenic thought disorder might argue for the differentially involvement of frontotemporal neural networks giving rise to a different underlying substrate to the thought disorder in Schizophrenia and ADHD; namely "cognitive and attentional deficits in ADHD versus deficits in higher level linguistic processes in Schizophrenia". This argument is supported by the cognitive deficits of filtering and fusion deficits described by the author in ADHD child populations with deficits in speech prosody (Jerome 2000) and not currently described in children with schizophrenia. In order to test this hypothesis it would be instructive to test children with schizophrenic thought disorder to see if they show similar deficits in central audition to children with ADHD.

Dr. Laurence Jerome MBCh.B M.Sc. MRC. PSYCH FRCP(C)

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# Book Reviews

## Attachment Disorganization

***Edited by Judith Solomon and Carol George. The Guilford Press. 1999. 420 pp. $67.50 hard cover. $35.00 soft cover (Canadian).***

Reactive Attachment Disorder (RAD) is under represented in the clinical literature in comparison to the impact it has on the children who suffer from it. All child psychiatrists will see psychopathology along the spectrum of attachment problems. Attachment disorganization in particular brings alarming results. Therefore, this book should be of interest to every clinician who has seen or may see these unfortunate children in their practice.

The book begins with an in-depth introduction to the subject, followed by a series of research articles outlining the outcomes of disorganized attachment. The theory and research of John Bowlby, Mary Ainsworth and other main authors and researchers in the field of attachment, including the editors Solomon and George, provide a foundation for many chapters. This book is about attachment theory and how and why it goes wrong. It is a basic text specifically aimed at understanding "disorganized" attachment behaviour (versus other attachment patterns such as insecure or ambivalent attachment behaviours). It is not a clinical text on Reactive Attachment Disorder (DSM IV) and in fact the diagnosis is never mentioned in the book. Yet, the underpinnings of every diagnostic criteria for RAD are laid down and explored in depth. For example, in comparison to the other types of attachment problems, the disorganized attachment process brought on freezing or stilling of expression, sequential or simultaneous contradictory behaviours and under directed or misdirected behavioural patterns.

The term "disorganization" in the title of the book is important. It is best compared with other examples of disorganized biopsychosocial systems, such as "Schizophrenic disorganization." Here, several domains of function are simultaneously disturbed. The term "disoriented" is sometimes used interchangeably with the term disorganized. It gives the reader a deeper feel for the degree of psychopathology these children suffer. Clinical snapshots in the book give some life to this degree of pathology, as for example:

"In the second reunion, Kate approached her mother with arms outstretched toward her mother. When she was about two feet away from making contact, she moved her arms to the side and abruptly circled away from her mother like a banking airplane. As she moved away, she had a dazed, blank expression on her face."

The early chapters go into great depth in understanding pathological mother-infant interactions, where both frightening and frightened maternal behaviours are important. It reviews the individual and physiological correlates of such interactions in the infant and toddler. Findings include, for example, more intense cardiac responsiveness and higher adrenal cortical activity. These children were easily and intensely alarmed. As young children try desperately to cope with their disorganization, they shift to a controlling behaviour, seen clinically as manipulative, punitive, intimidating, rigid and humiliating behaviours, so disturbing to their caregivers. The emotional basis of the disorganized child is characterized either by under control of verbal abilities, ( resulting in verbal and physical aggression to peers) or over control (resulting in expressionless withdrawal). Both are examples of affective dysregulation common to RAD.

Subsequent chapters review studies of the outcomes of these children in the school-age population. School personnel become very concerned and often initiate a referral when affective dyscontrol contributes to both social and cognitive dysfunction. Communications, motor and learning developmental patterns are also at risk, requiring assessment by speech and language clinicians, occupational therapists and psychometricians for psycho-educational testing. Even with this information, school personnel and parents need guidance that at the core of these problems remains attachment disorganization. Hopefully, a combination of these assessments and a clear clinical formulation will assist in ensuring the long term funding for the treatment and educational planning these children require, and for which we, as physicians, should advocate.

The authors also describe atypical populations affected by attachment disorganization: neurologically impaired children, specific pre-school populations, broken family's and malnourished children. Further chapters review the familiar link between disorganized attachment and adult dissociative disorders. The problem is, of course, that most children who suffer severely disorganized parenting also suffer from abuse and neglect. These children lie along an RAD/PTSD (Post Traumatic Stress Disorder) spectrum. A unique chapter late in the book tries to classify disorganized adult attachment. This is important for child psychiatry, as, more than we possibly realize, this relates to the parents we often see of RAD children. Here, very disorganized parents give way to another generation of disorganized attachment problems. Several decades of studies of deinstitutionalized mentally ill mothers reconfirm a clinical bind we have all seen in which children are at risk whether remaining with or taken from these mothers. More research is needed to address this important issue.

In summary, this is a basic science text, an in-depth orientation to and a series of psychological research studies of disorganized attachment. It is useful to child psychiatrist who desires a better understanding of attachment problems typically seen in the office, and, in particular, the problems of disorganized attachment. It is not a source of clinically useful information regarding the diagnosis or treatment of Reactive Attachment Disorder, but rather a way to understand the underpinnings of its psychopathology and diagnostic criteria.

***Drew Bremness MD FRCPC***

## A Clinical Guide to Sleep Disorders in Children and Adolescents

***Gregory Stores. Cambridge University Press, NY. 2001. 196 pp. $40.00 USA***

"There can be few more striking examples of the gap between clinical need and the provision of clinical services than sleep disorders medicine, especially concerning children."

Stores argue that there is a blind spot in education. Apparent familiarity with sleep and common difficulties leads to neglect of a serious problem. 20-30% of children from infancy to adolescence have sleep problems considered significant by their parents. Within this group of children there is a significant prevalence of serious disorder such as obstructive sleep apnea, narcolepsy and seizures.

The writing style is clinical. Each section gives the central topic and surrounds it with all the information you need to look at and understand the topic. The varieties of each problem are described along with the differential diagnosis. The text is clear and full of clinical information. The approach is comprehensive and broad. The reader learns what is known and what is not. Tables are clear and relevant to the topic. The clinical examples are always interesting and designed to grab one's attention.

The first chapter (General Issues) identifies children at high risk of sleep disorders, and then describes the effects of chronic sleep deprivation on learning, emotions, behaviour and health.

The second chapter reviews physiology and provides structures for assessment and treatment. The emphasis is on thorough assessment, and on professional support and encouragement. The suggested recording forms are useful and understandable by most patients or families.

Chapters follow on sleeplessness, excessive sleep and parasomnias.

The book rounds off with a section on the organization of services and the need for professional education.

Sleep medicine uses acronyms. A good glossary is provided, but I recommend reading the book through first to gain a sense of what each means.

Stores met his goal of an interdisciplinary approach, useful to generalists and specialists.

This is a book to keep close and use often. It will add to the management of each child with a sleep problem. It is a sleep consultant in a book. I have had this guide on my desk for 2 months and used it regularly. There is more than accurate, thoughtful and clear information. In daily use, it directs one to the complexity of each case a significant strength of this book. I will keep it close until the next edition arrives.

***David Aldridge MB FRCPC***

## Blackbird: A Childhood Lost and Found.

***Jennifer Lauck. Simon & Schuster Inc., 2000. 406 pp. (Paperback). $21.50 (Canadian)***

## Still Waters.

***Jennifer Lauck. Simon & Schuster Inc., 2001. 419 pp. (Hardcover). $36.50 (Canadian)***

When we find a person whose childhood and adolescence is marked by parental deaths, step family ridicule, deceitful and demanding relatives, abandonment to a commune, multiple placements and constant degradation from the only sib, we expect an adult with multiple problems with interpersonal relationships and self concept. That is the clinician in us.

When that adult has a successful career, a rewarding spousal relationship, a loving child and a book on the New York Times best seller list, we fall back on our clinical lore about resiliency. If, however, we live and read only wearing our clinical hats, we will miss books like these.

Blackbird (age five to twelve) and Still Waters (thirteen to adulthood) are Jennifer Lauck's autobiographical books with all the above and more. We will all have seen children with more disasters in their life but, more telling, we will have seen more children with less who cannot function, let alone succeed, in life. How did she do this and how does she bring this experience home to us?

When her only, and hated, sibling turns out to be different than her childhood image and when the promise of his future turns tragic, the seeds of change are sown in Jennifer. Later failed relationships and inner confusion bring the recognition that all is not well with her world. She begins the search for understanding. While this leads her to a supportive therapist, it is clear that the intense will to survive, shown during life's earlier forced changes is now transformed into a more arduous inner journey. Using her journalistic research skills, she travels throughout several states to revisit her past and collect the interviews and documentation that culminate in writing the narrative that enables her to rediscover herself and her strengths. The unfailing support of her husband and a child, who reintroduces her to unconditional love, complete the journey to a fulfilling adulthood for her.

Jennifer's life is captured literally and figuratively by the many recurring details that hold significance and meaning for her. The story of Snow White enters the book over the years, each time with renewed understanding as she grows. Early photographs in her parent's wedding album bring reassurance, history and awaken old connections. Her pink trunk holds it all together on the inside and the family table, chairs and princess bed bring the external support and presence of her family.

The compelling power and page turning fascination of Jennifer Lauck's books come from more than the events. She is an accomplished author whose first person singular account from inside the eyes of a child makes the books a unique and absorbing experience. She brings us back to a childhood world of sights, smells, gestures, thinking and emotions by finding her voice as five year old preliterate Jennifer. We grow with her and experience her presence so directly that there is no need for the observations and interpretations of the adult or the clinician. She is remarkably capable of conveying the unknowing and naive acceptance of a child, the perception of secrets of the adult world and the slow, insidious development of mistrust of adult relationships. We arrive before we know we are there.

Yes, it is a first hand account of resiliency (and the factors are there), and it was that hint that peaked my interest in the books but is was her power as a writer that drew me far past my clinical world. Jennifer's journey is a testament to the human spirit and the will to survive. With the tragedies that our world brings to our children on a daily basis around this planet, we need more Jennifers. Write on! And to you, reader, buy these books, read on!

***Wade Junek MD FRCPC***

## Developmental Disability and Behaviour

***Edited by Christopher Gillberg and Gregory O'Brien. Cambridge University Press, NY. 2000. 189 pp. $64.95 USA.***

This is an English multi-authored book with two North American contributions, one from Roger Freeman and Maryke Groenveld of Vancouver,B.C, Canada, and the other from James C. Harris of Johns Hopkins University, Baltimore, USA. As is often the situation with multi-authored books the content is rather uneven, and it is a case of the 'parts being greater than the whole.'

The editors set the tone for the book in their introduction by posing questions such as:

"To what extent do we find different types of behavioural problems in children with different types of disability?"

or

" Do we find any substantial differences in behaviour according to whether disability is congenital or of early onset, or acquired later in childhood?"

Individual chapters that were particularly enlightening, with carefully clarified clinical insights and a series of practical recommendations.included:

[a] The Behaviour of Children and Youth With Visual And Hearing Disability by Freeman and Groenveld. This chapter captured the importance of the loss of a major sense on the individual's development and behaviour. It also established clinical distinctions between the loss of a major sense at birth, or later onset loss of the sense. The chapter included carefully described clinical terminology and concluded with a practical list of recommendations.

[b] Recent Developments in the Neuropsychological Assessment of Attention and Executive Function in Children with Learning Disabilities by Thomas P.Kelly, University of Newcastle. This was a dense but fascinating analysis of the reasons behind the behaviours that we see in attentional problems and executive function disorders. These disorders form clinical substrates underpinning many complex disturbing and often aggressive behaviours.

[c] Ambulatory Electronic Monitoring in Sleep Disordered Children with Learning Disabilities. by David Bramble of Nottingham University. Here, the practical use of a neuropsychological clinical technique is described in detail. As well, its application to clinical assessment is ably demonstrated.

[d] Multimodal Interventions for Developmental Neuropsychiatric Disorders by Harris. This chapter was a very complete overview of clinical management unique to developmental disabilities. James Harris showed the strengths of the developmental perspective in case formulation: the use of developmental tasks as treatment goals, a present / future orientation in considering the impact of the past on present functioning and an emphasis on the 'best fit' of a person in the family, to name but a few.

Unfortunately the chapter on medication was a bit thin, and did not address the use of atypical antipsychotics, GABA agents such as gabapentin or toparimate, buspirone or buproprion among others. The two-page suggested algorithm was somewhat unwieldy. However, Michael Prendergast from Norththumberland did describe a clinical truism in eloquent terms. When commenting on the fluctuating symptoms of subacute confusional states resulting from medication, he stated "Fluctuation in consciousness with lucid intervals is the neuropsychiatry equivalent of a raised erythrocyte sedimentation rate."

Finally, the chapter on Psychotherapy with Disabled Young People by Val Hawes and Sheila Hollins of St George's Hospital, London, gives many clear clinical examples that have validity on both sides of the water.

Overall this is a timely addition to the increasing literature providing a fine understanding, with management recommendations, for developmental disability in children.

***Kieran D. O'Malley MD FRCPC***

## Innovations in Adolescent Substance Abuse Interventions

***Edited by Eric F. Wagner and Holly B. Waldron. Bergman Press, Elseiver Science Inc. New York, N.Y. 2001. 400 pp. $75.00 USA***

This book purports to provide a "state of the art," empirically tested approach to assessing, preventing, and treating adolescent substance abuse problems. The editors argue that previous texts have not been empirically based or provided an array of treatment approaches. This is a multi-authored text with 16 chapters predominantly written by American psychologists and covers the area of assessment, primary and secondary prevention, and treatment programs. There is one chapter by a physician on psychopharmacological therapies. There are 35 contributors overall.

This book reflects the current state of Substance Use Disorder assessment and treatment in the USA. The approach is primarily psychosocial in origin with little integration of biological or medical approaches. A single chapter by a physician reflects this dichotomy.

In Canada there is an absence of formal training in Substance Use Disorder (SUDS) in most residency training programs, particularly in the area of child and adolescent psychiatry. The approach to managing concurrent disorders is still in its infancy and there is a dearth of training opportunities for child and adolescent psychiatrists who wish to learn more about this important area of clinical practice.

This book provides a good overview of the current situation in the USA. The single chapter on psychopharmacology is well written but lacks in-depth detail and provides little clinical information that would be helpful to routine everyday practice. Interestingly, parts of the section on the management of Attention-Deficit/Hyperactivity Disorder (ADHD) mentions the use of pemoline, still used in the USA despite enough concerns about liver toxicity which has led to a removal from the Canadian market. The section on motivational interviewing, an important aspect of assessment and engagement with adolescents and the complex problems of SUDS, is well written.

This is a multi-authored text and there is a lack of "a single voice" as one reads the book. One result of this is some inevitable repetition of background information. The lack of a biopsychosocial perspective with minimal mention of "Dual" or "Concurrent" diagnoses would tend to restrict the use of this book to specialized libraries rather than a book for the practising specialist or resident-in-training.

***Laurence Jerome MD FRCPC***

## Handbook of Attachment.

***J. Cassidy, P.R. Shaver (eds), The Guilford Press, New York. 1999. Hardcover. $144.50 USA***

If handbook means "a small reference book providing specific information or instruction", then this book does not fit the definition (1). Rather, it is a large, very complete review of the field of attachment. It also is a heartfelt tribute to Bowlby and Ainsworth.

Early in Bowlby's career, he became convinced that disruption in the mother/child relationship was seminal in the development of psychopathology, rather than the current psychoanalytic thinking that emphasized secondary drives and internal phantasy as causal.

In 1944, he published his study of "44 Juvenile Thieves" that explicated his thesis about early life relationships and started an explosion of thought, study and publications (2). Meeting up with Mary Salter Ainsworth was fortuitous and led to an extremely fertile collaboration with her naturalistic studies in Uganda, continuing in Baltimore, providing a strong research base for the theory.

The postulation of inherent motivation to establish a relationship or bond, continued to put him at odds with the psychoanalytic establishment. He stressed the importance of the affectional bonds and care-taking system and the uniqueness of the symmetrical relationship between child and mother, with the mother as solution to threat. His theorizing culminated in the publishing of the trilogy of Attachment and Loss, and the rest is history (3). As the theory is now so well accepted, it is hard to understand the strong early objections and how revolutionary his theory was at the time.

The handbook is divided into six sections comprising thirty six chapters with an epilogue by Mary Main. Each chapter reviews theory from Bowlby, which can be repetitious, current research and theory, data available and then suggested directions for further work and development.

The first section of five chapters gives an overview of Attachment Theory. Section II speaks to Biological Perspectives (six chapters). Section III and IV cover Attachment Theory in Infancy and Childhood (three chapters) and Adolescence and Adulthood (six chapters). These are followed by Clinical Applications of Attachment Theory and Research (seven chapters) and a final section on Emerging Topics and Perspectives (nine chapters) with the afore mentioned epilogue by Mary Main.

This is an amazingly rich book with great depth of discussion. Although not a book which is read cover to cover, it is an incredibly thorough reference always bringing new knowledge and insight.

As I went through the book, I was struck by the extensive development of the field and its many applications. The findings are complex and at times contradictory, giving much food for thought. I particularly liked the sections on Intergenerational Transmission explaining how psychopathology can travel through families. I liked also the chapters that show that attachment theory is not just a theory of infancy and has application to adolescents and adults and hence is a life span model. A weakness I found is that there was not much on the application of attachment theory to therapy. The model of the therapist providing a secure base from which the client/patient can explore and extend his or her world view is apt. Attachment theory gives a solid explanation of the predisposing factors leading to difficulties, but does not suggest how the therapist can facilitate the development of the therapeutic context as a secure base and inform what happens in and out of the therapeutic hour.

In summary I would recommend this book highly, (even though it is costly) as an important book to have in one's library and one that will not quicky go out of date, remaining a valuable resource to go back to again and again, each time gaining new insight.

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***Elsa Broder, M.D. F.R.C.P.(C)***

## Male Victims of Sexual Assault.

***Gillian C. Mezey, Michael B. King. Oxford University Press. Don Mills, ON Canada. 2000. 161 pp. Hardcover. $63.95 Canadian***

Male Victims of Sexual Assault is an addition to the sparse literature on male victims of sexual assault.

The introductory chapter reviews some of the epidemiological research to date and presents evidence that challenges previous notions regarding male victims of sexual assault. It provides an overview of the area, with a strong focus on adults rather than children.

The second chapter discusses the "concept of homophobia" and discusses "legal homophobia." The author describes "homosexuals" as a "minority whose life-style and intimate concerns are so different from those of the family-oriented majority," suggesting that the gay population is not family-oriented. The closing comments serve only to dichotomize society according to homo- or heterosexuality, which can only lead to more homophobia.

"Male children and adolescents as victims: a review of current knowledge" is the most useful chapter for child and adolescent psychiatrists and, thankfully, the longest chapter in the book. The authors present the growing data and literature in the field and confront myths about the sexual abuse of boys. Rates of abuse remain unclear and are most likely under-reported. Abuse by women, other children and family members is discussed. Short and long term effects of abuse are reviewed. Only a small portion of the chapter is dedicated to treatment, which may reflect the limited research in this area.

Other chapters discuss sexual assault in locked environments and surviving the trauma of sexual assault. These chapters focus on adults. "Cultural and historical aspects of male sexual assault" provides an interesting but cursory overview of a variety of historical and cultural perspectives about sexuality with some attention paid to the topic of sexual assault. The seventh chapter discusses legal issues regarding male victims of sexual assault in the British legal system and highlights discrepancies between laws based on the gender of the victim.

The final chapter summarizes treatment issues. Like most of the book, the focus is on adult victims. The book will provide some useful information for clinicians who work with male victims of sexual assault but does not have a strong focus on treatment issues. The chapter on homophobia should have been heavily edited or tossed out.

***I. A. Mian M.D.***

## The Biology of the Autistic Syndromes, 3rd Edition.

***Christopher Gillberg and Mary Coleman. Cambridge University Press, NY. 2000. 330 pp. $95.00 USA***

The Biology of the Autistic Syndromes is a timely book, in that the numbers of autistic children appear to be on the rise. It seems likely that many more pediatricians and child psychiatrists will need to develop an understanding of the diagnostic features and known organic associations of these disorders as the need for diagnostic and treatment services increases. Parents of autistic children are often well educated, and generally well informed through parents' and advocacy groups. As a result of this, they expect a higher level of expertise from the specialists with whom they consult.

Although the subject of the autistic syndromes is approached predominantly from a biological standpoint, the book's coverage encompasses far more than one would assume from the title, ranging from diagnostic approaches, through epidemiology and cognitive profiles, to a description of the clinical course in both children and adults. It provides an up to date and thorough review of the current state of understanding of autism and related disorders. From a Canadian point of view, one would have hoped for a more detailed discussion of the work of Peter Szatmari, which is so relevant both clinically and in our understanding of the cognitive development of children and adults with these disorders. However, many other ways of conceptualizing autism are reviewed, and the result is several informative and thought provoking introductory chapters describing the diagnostic and developmental issues. Indeed, the majority of the information required to answer the questions of parents, and our own intellectual curiosity, is well covered.

As would be expected from the title, the organic aspects of autism are thoroughly described, from biological and genetic, to disease related etiologies. Autistic disorders are considered to be a common end point of many neurological processes. The discussion of 'double syndromes' (dual/multiple diagnosis that includes autism as one of the diagnoses) is interesting in that it sheds some light on the neurological disturbances that can lead to autistic symptoms and thus suggests many potential areas for future research. Other chapters list the current findings in genetics, endocrinology, etc. Unfortunately, no clear pattern seems to arise from the current body of knowledge, but several topics for further investigation become apparent and each chapter has an extensive list of references to guide further reading.

A suggested list of biological investigations is provided, a useful starting point for those attempting to devise their own list of relevant and cost effective tests during the process of establishing services for this group of children. Unfortunately the usefulness of this list is limited by the lack of discussion as to the clinical relevance and treatment implications of the various tests. As autistic children are not likely to be as cooperative as one would hope, and may be traumatized in the process of providing blood or even a 24 hour urine sample, this analysis would have been useful. As a profession, we also have to face the realities of a medical system that is severely underfunded and in which we have to be more aware of cost benefit ratios than was, perhaps, the case in the past. A brief discussion of these factors would have been useful.

The Biology of the Autistic Syndromes should be available in all medical libraries, and any individuals involved in research into the causes of autism or development of clinical services will find it a useful reference that will enable them to be aware of the findings outside their own body of knowledge. No doubt many parents will also turn to it in their attempts to explain the challenges that their children face and in their desperation to find some relief for one of the most challenging and heartbreaking disabilities.

***Pippa Moss MB FRCPC***

## Treating Abused Adolescents.

***Eliana Gil. The Guilford Press, NY. 1996. 228 pp. Paperback. $40.00. USA***

This is one of a several books that Dr Gil has written or co-authored on abuse issues.

*Treating Abused Adolescents* consists of 7 chapters and an after word. The overview (Chapter 1) reminds the reader about the developmental tasks of the adolescent period, the types of abuse that youth may suffer, and the range of responses to that abuse the clinician may see. Chapter 2 provides an in-depth review of the normal developmental tasks of adolescences and how abuse may interfere with attainment of these tasks. Chapter 3 reviews the differences between working with the youth who are currently being abused or was abused in the recent past and the youth who have a long history of cumulative abuse. Chapter 4 gives a concise and practical framework to the assessment process and the formulation of treatment goals. Chapter 5 provides practical suggestions for carrying out the treatment goals: acknowledging the abuse, addressing the impact of the abuse and creating a way for the individual either to limit the impact of the abuse in current life or to move beyond the abuse. Chapter 6 provides general principles for working with adolescents who have been abused. This chapter should have been much earlier in the book, as the principles need to guide the assessment and setting of treatment goals as well as the process of treatment. Chapter 7 reviews the need to provide treatment through a variety of modalities (individual, group, and family). While this information could have been included in Chapter 5, processing the trauma, it is important to have it highlighted in a separate chapter. Each of these modalities, when used at the right moment, provides specific and powerful benefit to the overall treatment plan. Providing the same information in another modality may not have the same impact. Some of this information is repeated in other parts of the book to highlight a specific situation. The Afterword reminds us that every therapist will have both successes and failures when working with adolescents who have been abused and both outcomes should be reviewed as part of ongoing learning.

Dr Gil writes in a clear and concise manner that engages the reader. She provides useful clinical examples and, at times, provides the transcribed dialogue of an actual treatment session demonstrating how she worked with the individual. Repeatedly, she reminds us that working with people who have been abused is complex, challenging and, ultimately, rewarding. Acts of abuse may have vastly different effects on different individuals. The need for flexibility is highlighted by the observations that techniques that are very effective with one person may be ineffective or even contraindicated in another. The need to address and treat co-morbid conditions, such as depression, could have more emphasis. Dr Gil gives reminders that the therapist must always be aware of transference and countertransference issues and manage them directly.

Mandatory reporting of abuse is superficially addressed by providing the definitions of abuse from the US Department of Health and Human Services. Every therapist who works with youth must be fully conversant with the specific requirements for reporting abuse in the jurisdiction in which they work. More could have been said directly about the issues that arise when mandatory abuse reporting occurs during the course of treatment.

This book is very useful both for the therapist beginning work with people who have been abused, and for the experienced therapist as a reminder of the significant and important issues for treatment.

***Greta Toni Swart MD FRCPC***

# CONFERENCE WATCH

The American Academy of Child & Adolescent Psychiatry, *50th Anniversary Meeting,* October 14-19th, 2003 Miami Beach, Florida, Fontainebleau Hilton.

Canadian Psychiatric Association, *53rd Annual Conference,* October 30-November 2, 2003 Halifax, Nova Scotia, World Trade & Convention Centre.

Canadian Academy of Child & Adolescent Psychiatry, *23rd Annual Meeting*, November 2- 5th, Halifax, Nova Scotia, Delta Barrington, Delta Halifax, World Trade & Convention Centre.

The Editorial staff wish to invite its members and readers to forward listings for upcoming conferences and meetings to be advertised in the Canadian Child & Adolescent Psychiatry Review "Conference Watch".
Please submit listings to:

Tena Roebuck, *Editorial Assistant*
tena.roebuck@iwk.nshealth.ca or
IWK Health Centre, Maritime Psychiatry, 5850 University Ave, Halifax, NS B3J 3G9
Tel: (902) 470-8377 Fax: (902) 470-8937

**FULL TIME ACADEMIC CHILD AND ADOLESCENT PSYCHIATRISTS - EATING DISORDER PROGRAM AND DAY TREATMENT PROGRAM**

London Health Sciences Centre and the University of Western Ontario, Division of Child Psychiatry, London, Ontario, Canada have an excellent opportunity for 2 full time academic Child and Adolescent psychiatrists in a newly developed Eating Disorder Program and a newly developed Day Treatment Program. London is a city of over 330,000 people and is located two hours from Detroit, Buffalo, and Toronto.

The Eating Disorder Program consists of inpatient beds, day treatment program, outpatient services and outreach for children and adolescents in South Western Ontario, a region with a population over 1 million people. The successful applicant would be involved with clinical service, education and research.

The Day Treatment Program consists of 10 places for children and adolescents 17 years and under. The following diagnoses will be treated: mood disorders, anxiety disorders, early psychosis, and psychosomatic illnesses. The successful applicant would be involved with clinical service, education, and research.

Successful candidates will be licensed to practice (or eligible for a license) in Ontario. The Division of Child Psychiatry at the University of Western Ontario is an expanding and stimulating environment. The positions offer excellent benefits and remuneration is commensurate with academic rank.

Interested applicants should send a curriculum vitae and letter describing their qualifications and interests to:

Margaret Steele, MD, FRCP(C)
Chair, Division of Child Psychiatry
University of Western Ontario
Physician Lead, Child and Adolescent Centre
London Health Sciences Centre
346 South Street, Room 102D
London, Ontario, Canada N6A 4G5

# INSTRUCTIONS TO AUTHORS AND CONTRIBUTORS

Manuscripts or material are welcome on any topic relevant to child and adolescent mental health, including child and adolescent psychiatry, child psychology, pediatrics, pharmacology, adult and geriatric psychiatry, internal medicine, or any other branch of medicine, psychology, sociology and occupational health relevant to child and adolescent mental health practitioners.

**Original Articles and Reviews (maximum 3000 words including tables and references)**
Descriptive and qualitative articles, quantitative survey or research articles, review articles, case reports and letters to the editor will be considered. There will be a peer review process for submissions, with the principal author being informed of the results of the review within six weeks of submission. Ordinarily, only original articles will be considered. However, reproduction of published material will be considered in extraordinary situations, where the material has a unique value to the membership of the Canadian Academy of Child Psychiatry. Reproduction of material will only occur after explicit permission has been obtained from the primary author and publisher.

**Case Reports (maximum 1000 words including references if any) and**
**Letters to the Editor (maximum 300 words including references if any)**

**Format of Submissions**
All submissions should be made *both* with a hard copy and on a floppy disc using an MS Word compatible format, even when an e-mail submission has been made. Submissions should be in Arial or Times New Roman, *at least 11 point font, with double-line spacing*. Titles and sub-titles should be in bold. The names of *all* the authors, their relevant qualifications and affiliations, the address, telephone number, fax number and e-mail address of the first, or senior author should be clearly indicated below the main title. References should be as follows in the text of the article: Gorman et al (1998) or …….(Gorman et al, 1998). References should be as follows in the reference list at the end of the article: in alphabetical order, in chronological order where there is more than one reference for the same author or authors, and, when there is more than one reference in the same year for the same author or authors the following notation should be used: Gorman et al (1998a, and 1998b). It is the responsibility of the authors to ensure that their work has received ethics approval where required. If an author wishes to submit their article(s) which has/have been published elsewhere, they should obtain and submit the written permission for republication of this work from the publisher who holds the primary copyright.

*CONFERENCE AND WORKSHOP ANNOUNCEMENTS WILL BE INSERTED FREE OF CHARGE*
*FOR ADVERTISEMENT RATES PLEASE CONTACT EDITORIAL ASSISTANT*

All submissions should be sent to

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## NOTES