The Impact of Prenatal Alcohol Exposure on Addiction Treatment

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Fetal alcohol spectrum disorders (FASDs) are conditions caused by prenatal alcohol exposure in amounts sufficient to cause permanent deficits in brain functioning. Extent of damage largely depends on timing, dose, frequency, and pattern of exposure. Timing is especially important because prenatal alcohol exposure during critical periods of gestation can affect brain development in ways that produce varying patterns of neurocognitive deficits and associated adaptive impairments. This article describes some of the more serious neurophysiological and neuropsychological sequelae of prenatal alcohol exposure that contribute to increased risk for substance abuse problems among people with an FASD. We discuss the unique interface between pharmacological treatment and FASD, noting that failure to consider the possibility of FASD in treatment planning may result in treatment failure and/or relapse. Finally, we present a clinical case example and recommend service accommodations to address some of the impairments in FASD that limit substance abuse treatment success.

Key Words: comorbid disorder, fetal alcohol spectrum disorders, fetal alcohol syndrome, substance abuse, treatment

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Since fetal alcohol syndrome (FAS) was first identified in the United States in the early 1970s (Jones and Smith, 1973; Jones et al., 1973), thousands of human and animal studies have demonstrated that alcohol is a teratogen that can interfere with fetal development and cause serious and permanent birth defects. *Fetal alcohol spectrum disorder* (*FASD*) is an umbrella term for medical conditions involving multiple central nervous system (CNS) deficits caused by prenatal alcohol exposure (PAE): FAS, partial FAS, alcohol-related neurodevelopmental disorder, and alcohol-related birth defects. In the past, all conditions under the FASD umbrella that did not involve full FAS diagnostic criteria (ie, emblematic dysmorphic facial characteristics, growth deficit, and CNS abnormalities) were referred to as fetal alcohol effects (FAE) or static encephalopathy, alcohol-exposed. Regardless of diagnosis and terminology, CNS damage in all of these conditions is similar (Streissguth et al., 1996, 2004; Connor et al., 2000; Astley et al., 2009).

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PREVALENCE AND COST

Studies by the Centers for Disease Control and Prevention report prevalence rates of FAS in the United States from 0.2 to 3.0 cases per 1000 live births across various populations, comparable with developmental disabilities such as Down syndrome or spina bifida. (Centers for Disease Control and Prevention, 2004b). Recent epidemiological studies suggest a combined prevalence of FAS and FASD in up to 5% of the US population (May et al., 2009). Given that FASD conditions without the characteristic facial abnormalities make diagnosis quite difficult, it may be that 5% is an underestimate (May et al., 2009).

Fetal alcohol syndrome alone imposes a substantial financial burden on our economy. For example, the 2002 estimated total lifetime cost to care for one individual with FAS is $2 million (approximately $1.6 million for medical care services and $0.4 million for loss of productivity). In 1998, estimated annual cost of FAS in the United States was more than $4 billion (Lupton et al., 2004).

NEUROPHYSIOLOGICAL EFFECTS

A fetus is susceptible to alcohol damage throughout pregnancy. Within minutes after a pregnant woman drinks alcohol, it crosses the placenta and blood-brain barrier, and the blood alcohol level of the fetus equals that of the mother. Alcohol's teratogenic impact on a fetus varies, depending upon the stage of pregnancy, the concentration of alcohol, and the frequency and pattern of exposure. The timing of exposure is especially important as PAE during critical periods of gestation can affect brain development differently, producing varying patterns of neurocognitive deficits (Stratton et al., 1996; Abel, 1998; Bookstein and Kowell, 2010; Lipinski et al., 2012). For example, PAE during the first few weeks of pregnancy, when brain cells are developing and migrating to locations in the embryo where brain structures eventually form, can be quite destructive (Whitty and Sokol, 1996). The brain, which

develops throughout pregnancy, is the organ most vulnerable to the effects of alcohol. Abnormally small brain size, or microcephaly, may be observed in some individuals with PAE. Magnetic resonance imaging studies on individuals with PAE reveal significantly reduced brain volumes in regions such as the cerebellum, basal ganglia, hippocampus, diencephalons, and corpus callosum (Mattson et al., 1996; Swayze et al., 1997; Archibald et al., 2001). Because many women drink alcohol until they learn that they are pregnant, which could be several weeks into the pregnancy, they may unknowingly expose their unborn children to the harmful effects of alcohol. Other factors also influence the extent of prenatal damage, such as maternal health, nutrition, smoking, and other drug use, and maternal and fetal genetics (Paintner et al., 2012). Notably, even low levels of PAE may harm some fetuses, whereas others may be impervious to relatively high levels of exposure (Olson et al., 1997; Sood et al., 2001; Sayal et al., 2007).

Women at highest risk of producing a child with FASD seem to be those who binge drink. Binge drinking, which involves alcohol intake in an amount that brings blood alcohol concentration to 0.08 g percent or higher, equates to 4 or more drinks in a 2-hour period for most women (National Institute of Alcohol Abuse and Alcoholism, 2004). Such a drinking pattern constitutes a particularly dangerous pattern of exposure (Stratton et al., 1996) because high blood alcohol concentrations produce an intensely toxic amniotic environment (Burd et al., 2012; Paintner et al., 2012). In contrast, the toxic impact of low amounts of exposure is unclear and controversial (Stratton et al., 1996).

NEUROPSYCHOLOGICAL EFFECTS

Most children and adults with FASD have IQs of 70 or above and so are not typically classified as having an intellectual disability or mental retardation. However, learning problems and functional deficits are common in this population, even among people with average IQs. Because of, in large part, significant executive function deficits, people with FASD often perform more poorly on tests of adaptive behavior and in real-life situations than would be expected from their IQ test scores (Streissguth et al., 2004). Deficits in executive functioning (eg, sequencing information and behavior, effective planning, organizing behavior, focusing on and completing goal-directed activity, response inhibition, flexible problem solving, generalizing from experience) are found in most individuals with FASD whether or not they have the dysmorphic facial characteristics of FAS (Mattson et al., 1999; Connor et al., 2000; Rasmussen, 2005; Riley et al., 2009). Because such skills are critical for appropriate decision making and self-regulation, deficiencies can substantially impair day-to-day functioning, adaptive behavior, and social interactions, posing major challenges for individuals with FASD, their families, and treatment providers. This is especially true for those who do not have the facial features of FAS, because others assume that if there are no visible facial abnormalities, there is no brain damage and the person's behavior is entirely within volitional control.

DIAGNOSIS

Fetal alcohol spectrum disorder involves medical diagnoses that are typically made by trained physicians in conjunction with multidisciplinary teams that assess specific domains of CNS functioning (Bertrand et al., 2004; Brown et al., 2012). Fetal alcohol syndrome is characterized by PAE and 3 specific abnormalities: (1) dysmorphic facial characteristics (ie, smooth philtrum, thin upper lip, and short palpebral fissures); (2) prenatal and postnatal growth deficiency in height or weight ($\leq$10th percentile); and (3) CNS dysfunction, including structural, neurological, and/or functional brain abnormalities (which may include microcephaly, mental retardation, and attention deficit disorder) (Bertrand et al., 2004). The facial features and growth deficits in individuals exposed prenatally to alcohol may attenuate over time. Partial FAS involves only 1 or 2 of the facial abnormalities; alcohol-related neurodevelopmental disorder does not require the presence of any dysmorphic facial characteristics. However, CNS abnormalities are present in all the 3 conditions. The most opportune time to make an FASD diagnosis is between the ages of 10 months and 10 years because the physical features related to the disorder are most observable during this time. Diagnosing FASD in adulthood can be problematic in the absence of reliable informants that can provide PAE history. Because identifying FASD is challenging when there are no obvious physical abnormalities, FASD conditions are often referred to as "hidden disabilities." In fact, many affected individuals are unaware of their conditions (Bertrand et al., 2004), even though they may know from life experiences that functioning successfully on a day-to-day basis is challenging.

Ideally, an FASD diagnosis can explain to an affected individual and those with whom he or she interacts why his or her behavior and functioning are impaired. Diagnosis may result in more realistic expectations for the future and the adoption of compensatory strategies. Thus, formal diagnosis can be an important first step in acquiring appropriate treatment and services (eg, social security disability benefits, substance abuse and mental health treatment, specialized vocational programs) that might ameliorate adverse life outcomes common to individuals with this permanent birth defect (Streissguth et al., 1996).

FASD AND SUBSTANCE ABUSE

People with FASD are at high risk for developing substance abuse problems. In a landmark study on adverse life outcomes among 415 people with FAS and FAE, Streissguth et al. (1996, 2004) found that 29% of individuals age 12 to 20.9 years and 46% of adults age 21 and older had alcohol or drug problems (defined as ever having alcohol or drug abuse problems or ever having been in alcohol or drug abuse treatment). In contrast, results from the 1992 National Epidemiologic Survey on Alcohol and Related Conditions found an adult lifetime prevalence rate of 18.2% for alcohol-use disorders in the United States (Vergés et al., 2011) and 10.3% for drug-use disorders (Compton et al., 2007). Given the well-established association between substance use disorders and mood and anxiety disorders (Grant et al., 2004; Wolitzky-Taylor et al., 2011), these high prevalence rates in FASD may stem from a disproportionate amount of Axis I disorders in this population (Brown et al., 2012). For example, although a nationwide prevalence study in 2001 to 2002 found mood disorders in 9.21% and anxiety disorders in 11.08% of the general adult population (Grant

et al., 2004), much higher rates of mood disorders were found in an FASD sample: 44% had major depressive disorder and 20% had either bipolar disorder or an anxiety disorder (Famy et al., 1998). In another study, Streissguth and colleagues (1996) found that more than 90% of adults and adolescents with FASD had comorbid mental health problems. Slightly more than half of the adults in this study had depression problems, 29% had psychotic symptoms, 43% had histories involving suicide threats, and 23% had made actual suicide attempts. A study that compared 2 groups of children (alcohol exposed and nonexposed) not only found high rates of mental illness in the PAE group but also found that exposure history significantly discriminated between groups for depression, anxiety, psychosis, and hyperactivity (Roebuck et al., 1999).

Streissguth et al. (2004) found that living in a stable and nurturing home for most of the childhood strongly protected against later alcohol and drug problems. This protection may have something to do with the capacity of protective caregivers to shelter their alcohol-affected children from negative peer influences. Because many individuals with FASD lack social skills and tend to mimic others (Brown et al., 2011), they are especially prone to use alcohol and drugs if they see this behavior in peers. Alternatively, if children with FASD are raised in protective caregiving environments where substance use is not modeled, they tend to abstain. Specific type of FASD diagnosis seems important. Streissguth and colleagues (1996) found that individuals diagnosed with FAS rather than FAE had significantly reduced odds of developing alcohol and drug problems. This outcome may be because of visibility: because of their medical diagnosis, dysmorphic facial characteristics, and lower IQs, youth with FAS are usually better able to qualify for developmental disability services than those without the full syndrome and also may live in more protective environments (Streissguth et al., 1996, 2004).

PAE AND DEVELOPMENT OF SUBSTANCE ABUSE DISORDERS

Prenatal alcohol exposure is consistently found to be a significant risk factor for adolescent and adult alcohol use and alcohol-related problems. In one study, Baer and colleagues (1998) examined the relative importance of PAE and family history of alcoholism in predicting alcohol problems in a large sample of 14-year-olds whose mothers had enrolled in a population-based longitudinal study during the index pregnancy. Results showed that PAE was more predictive of adolescent alcohol use and its negative consequences than family history of alcohol problems, even after adjustment for family history and other prenatal and environmental covariates. Baer et al. (2003) conducted a second study based on the same cohort and reported significant positive associations between alcohol exposure in midpregnancy and alcohol problems at the age of 21 years.

In another study, Yates et al. (1998) studied the effects of PAE on adult nicotine, alcohol, and drug dependence in a sample of adoptees. Twenty-one adoptees whose mothers drank during pregnancy were compared with 102 adoptee controls who did not have PAE. The latter were matched to the PAE subjects by adoption agency, age, sex, and maternal age at the time of birth. The study found higher symptom counts for alcohol, drug, and nicotine dependence in the PAE group, and the effect of PAE remained after controlling for sex, mothers' alcohol dependence diagnosis, birth weight, gestational age, and other environmental variables.

Alati et al. (2006) conducted a 21-year follow-up study among more than 2000 subjects enrolled in a population-based birth cohort study and found evidence for the specific contribution of PAE to both early and late onset of alcohol disorders at the age of 21 years. ("Early" onset was defined as meeting criteria for an alcohol abuse disorder between the ages of 13 and 17 years; "late" onset involved diagnosis between the ages of 18 and 21 years.) In particular, children of mothers who consumed 3 or more glasses of alcohol during any drinking occasion in early pregnancy had approximately 3 times the odds of early- and late-onset alcohol disorders than those whose mothers drank no more than 2 glasses. The authors suggested that their findings supported a biological component in the origin of alcohol disorders and recommended that greater attention be given to the physiological "programming" role that PAE may play in the development of such disorders.

In research involving various methodologies, genetic factors have long been found to contribute to the risk of substance abuse disorders (eg, Cadoret et al., 1986; Pickens et al., 1991; Harford et al., 1992; Kendler et al., 1992, 2007; Noble, 1993). However, this association has not yet been studied among people with FASD. Thus, a significant research challenge lies in untangling the independent contributions of PAE and genetic factors (eg, family history of alcoholism) in the development of substance abuse disorders because offspring in alcoholic families are likely to have been exposed to alcohol in utero.

PAE, EARLY AGE AT ONSET OF ALCOHOL USE, AND DEVELOPMENT OF LATER ALCOHOL PROBLEMS

People with FASD may be at increased risk for substance abuse disorders because of young age at first alcohol and drug use. In their study of adverse life outcomes among people with FAS or FAE, Streissguth et al. (1996) did not assess age of onset for alcohol use, but they did find that 19% of adolescents and adults with alcohol or drug problems reported onset of such problems before the age of 12 years, and 63% reported onset of substance abuse problems between the ages 12 and 20 years. In general, studies illustrate a link between early onset drinking and elevated risk for later alcohol abuse and dependence (Grant et al., 2001a,b; McGue et al., 2001; Hingson et al., 2006; Warner et al., 2007; Pitkanen et al., 2008; Buchmann et al., 2009; Blomeyer, 2011). Related research also indicates that youth who begin drinking early may develop biological reward mechanisms associated with alcohol use or may learn to use alcohol as a coping strategy to offset social deficits (Sinha, 2001; Dawson et al., 2007).

SUBSTANCE ABUSE TREATMENT AND FASD

Only about half of the approximately 2.6 million people who enter substance abuse treatment in the United States each year complete treatment (National Institute on Drug Abuse, 2011; Substance Abuse and Mental Health Services Administration, 2012). Completion rates are even lower in outpatient

treatment settings. For example, in 2005, only 32% of women in outpatient treatment completed the treatment. Of those who did not complete treatment, 29% dropped out and 11% were terminated by the facility (Substance Abuse and Mental Health Services Administration, Office of Applied Studies, 2009). Given that FASD may affect 5% of the population (May et al., 2009), we suggest that one reason for this substantial treatment failure in the general population may be undiagnosed neurocognitive deficits associated with PAE-associated learning disabilities. Review of the literature finds no information about treatment participation or completion rates for people with PAE or FASD. Similarly, there is limited research regarding treatment interventions for adults with neurocognitive impairments, including those with intellectual disability or mental retardation (Burgard et al., 2000). Because nearly half of adults with FASD have substance abuse problems (Streissguth et al., 1996), it is likely that many adults with PAE enter treatment each year with neurocognitive deficits that impair their ability to learn and benefit from treatment. In an initial study that examined this hypothesis, treatment failure rates among female patients were significantly higher for those with FASD and PAE than those without PAE (Grant, Brown, Graham, and Ernst, 2012, manuscript under review). As this initial study implies, if undiagnosed cognitive deficits and associated learning problems do in fact play a significant role in treatment failure, it will be important for treatment programs to screen for such factors during intake and modify therapeutic approaches accordingly.

In Table 1, we recommend treatment modifications that have been found to support success for patients with PAE/FASD. The treatment interventions we recommend are based on scientific understanding of areas of the brain affected by PAE, the behavioral manifestations of that brain damage, and the authors' clinical experience in interviewing and treating individuals with FASD. We have found these interventions effective in improving functioning over the short term. In Table 2, we also suggest strategies that we have not found to be effective with this population.

TREATMENT FOR PATIENTS WITH COMORBID DISORDERS

People with undiagnosed FASD often receive diagnoses (and treatment) for one set of symptoms, whereas other symptoms are missed or ignored (Brown et al., 2012). However, failure to consider underlying medical conditions such as FASD can result in medical errors with serious consequences, especially when psychopharmacology is part of treatment planning for ancillary problems such as mood disorders, pain, sleep deprivation, or posttraumatic stress disorder. Patients may enter treatment with medications already prescribed for various problems and receive additional medications during treatment if they are diagnosed with comorbid medical and mental health conditions. Although medication may be a helpful adjunct to treatment for patients with comorbid disorders, pharmacological intervention should be considered only after comprehensive multigenerational psychiatric assessment that includes PAE history. Individuals with undiagnosed FASD are prone to have heart or other organ problems, which requires special precautions as some psychopharmacological medications (eg, tricyclic antidepressants) can cause adverse reactions (Burd et al., 2003), as can frequently prescribed α adrenergic agonists such as guanfacine and clonidine (Sallee, 2010). Although risk of kidney damage from PAE is lower than expected, if lithium is prescribed for mood control, the patient must be closely monitored for toxic effects on the kidneys (Centers for Disease Control and Prevention, 2004a). In other words, although pharmacotherapy that targets comorbid conditions can be helpful for individuals with PAE, it also can cause adverse reactions in this population (Brown et al., 2012).

If comorbidity involves severe mental illness such as bipolar disorder, suicidality, and psychosis, which disproportionately affect individuals with FASD (Streissguth et al., 1996; Famy et al., 1998; Spohr et al., 2007; Huggins et al., 2008), substance abuse treatment can be quite challenging. An evidence-based intervention model that has shown promise in patients with substance abuse problems and serious mental illness is the Assertive Community Treatment (ACT). According to Offord and colleagues (2012), the ACT involves comprehensive community psychiatric treatment and 24/7 support services provided by a multidisciplinary team of professionals (eg, social work, rehabilitation, counseling, nursing, psychiatry). Interventions typically extend far beyond substance abuse treatment to include case management, initial and ongoing assessment, psychiatric care, employment and housing assistance, family support and education, and other services important for living successfully in the community. Clients best served by the ACT are those with persistent mental illness and functional impairments who have avoided or not responded well to traditional outpatient mental health services and often have coexisting problems such as homelessness, substance abuse problems, or involvement with the judicial system. In other words, intervention programs built around this model can be a safety net for people who would probably not be successful in standard treatment programs. The ACT treatment guidelines (Stein and Santos, 1998), which focus on structure, service linkage, and support for treatment follow-through, seem particularly well-suited to patients with FASD.

Treatment settings do not routinely screen for PAE. We suggest that such screening, along with ancillary screening for cognitive impairments, be conducted during treatment intake for patients who present with learning and behavioral problems. Results of such screening should be used to determine whether follow-up testing and multidisciplinary FASD assessment are needed, to identify functional impediments that might compromise treatment success, and to individualize treatment planning. Methodologies designed to screen for PAE in adolescents and adults typically involve informant-based protocols that ask friends and family members to rate or score their loved ones on behavioral factors correlated with FASD (eg, Streissguth et al., 1998). Presumably, the more items endorsed on such screens, the greater the likelihood of FASD. To our knowledge, there are no behavioral screens based on self-report. Generally, whereas informant-based information from unimpaired individuals is likely to be more accurate than self-reported data, most treatment programs do not involve informants in the intake process for various reasons. Thus, self-reported information may be the only available data at time of treatment. Given this situation, the authors have

TABLE 1. Functional Deficit Treatment Recommendations: Characteristics of FASD and Recommendations for Accommodating Impairments and Modifying Treatment Methodologies

FASD Characteristic	Treatment Recommendation
Impaired executive functioning:	Establish, teach, and model structure and consistency:
Inability to organize stored information, plan future activities, regulate and sequence behavior, make good judgments, and think flexibility when faced with unexpected circumstances.	Be consistent with appointment times, locations, and providers. Schedule multiple brief sessions per week rather than sessions that occur only once a week. Plan changes in routines/transitions carefully, modeling for patient advance risk planning and problem-solving strategies. Teach patient how to set appointment reminders on his or her cell phone. Designate person who will ensure that patient gets to appointments. Designate a mentor/treatment buddy who will respond to patient's calls for advice and support. Role-play likely high-risk situations and methods to handle them. Use a positive, immediate, clear reinforcement system—*not punishment*. If consequences must be implemented, ensure they are immediate, short term, and related to the action that prompted the consequence. Along with telling patients they are not allowed to do something, also describe what they are allowed to do. Limit changes to the treatment plan. Eliminate interventions that may overwhelm patients with PAE with impossible challenges that lead to shutting down or acting out (eg, expecting that such patients will be able to attend to verbal dialog in group treatment, process that information, wait his or her turn to speak, and respond appropriately). Teach patient to keep and use a calendar for daily planning. Teach patient to review and assess his or her own behavior on a daily basis.
Impaired ability to think abstractly:	Remember the client's *functional* age and abilities, which are lower than chronological age, and set reasonable goals:
Difficulty understanding abstract concepts such as time, space, money, quantities, cause and effect, idiomatic expressions, humor, and sarcasm.	Use literal, concrete terms when asking a patient to do something (eg, do not say "take a hike"). Do not use metaphors or similes. If joking, tell the patient with FASD you are joking; when a peer is joking, tell the patient with FASD know that the peer is joking. Teach the patient to generalize and do not assume a lesson learned in one context will transfer to another. Help the patient understand that he/she thinks literally and therefore needs to check out the meaning of what others are saying and doing before reacting. Use positive reinforcement systems rather than reward and consequence systems because these do not help the persons change their behavior. If consequences are necessary for misconduct, make them brief, immediate, and directly related to what just occurred. Evaluate the patient's ability to manage money and assess the need for a representative payee. Assess the possible need for guardianship for safety.
Impaired verbal receptive language processing:	Learn client's "unique" language patterns and present information strategically:
Inability to accurately process and understand incoming verbal information in social situations (poor receptive language skills), although verbal expression is adequate and the client may be very social and talkative.	Use multiple sensory modes. Use simple step-by-step instructions (written and with illustrations, if possible). Use short, concrete sentences and examples when communicating with client (no abstract ideas/concepts). Verify client's understanding (ie, ask "what does this rule or instruction mean?" and "how would you follow this rule or complete this task?"). When a rule is broken, respond with "I know it's hard to remember everything we are asking you to remember. How can I help you remember that rule when you need to?" Role-play and ask client to demonstrate skills (ie, do not rely solely on verbal responses). Revisit important points during each session. Remember the client's functional age in terms of communication and vocabulary. Role-play how to decide whether something involves "risky" behavior and what to say when friends suggest doing something risky. Carefully evaluate the potential effectiveness of group treatment, because this treatment modality is most often completely verbal. If group treatment is used, have the patient sit next to the facilitator for support and follow up each group session with 1:1 review of the important topics covered that day and their relevance to the patient's situation. If group treatment is used, assess frequently to see whether this modality complicates recovery or whether individual therapy should be used instead.
Social impairments:	Frame treatment expectations and requirements with the perspective that coping capacity and interpersonal skills are likely to be childlike:

(*continues*)

TABLE 1. Functional Deficit Treatment Recommendations: Characteristics of FASD and Recommendations for Accommodating Impairments and Modifying Treatment Methodologies (*Continued*)

FASD Characteristic	Treatment Recommendation
Difficulty reading and responding to social cues and body language.	Role-play various social situations and how the patient might best respond.
	Role-play social skills and have patient practice with multiple people.
	Point out misinterpretations of words, body language, and actions of others when they occur.
	Remind patient that other patients having a bad day has nothing to do with him/her.
	Do not interpret lack of eye contact as lack of motivation.
Impaired working, short-term memory:	Communicate concretely and repetitively:
Difficulty remembering information and following sequential verbal instructions.	Communicate rules, instructions, and directions in concrete terms that convey only one idea/instruction at a time (ie, do not state rules or give instructions that involve 2 or more actions/thoughts/steps).
	Provide support to ensure task completion, modeling appropriate actions.
	Review rules, instructions, and directions regularly and repeatedly.
	Teach patient to always carry a notebook and ask providers to write down instructions, appointment times, etc.
	Use a mentor/treatment buddy to remind patient of important activities.
	Monitor retention of important treatment principles by having patient regularly describe those principles in his or her own words.
Impaired coping ability:	Help patient implement/improve effective coping strategies while reducing/eliminating ineffective strategies:
Inability to respond appropriately to stress or pressure due that may manifest as overreaction or shut-down.	Teach staff how to recognize signs that the patient is becoming stressed, including how to intervene appropriately.
	Teach patient to identify when he/she is getting upset and practice use of effective coping technique(s).
	Model and have patient practice multiple relaxation techniques, retaining 1 or 2 strategies that are effective.
	Help patient find extracurricular activities that are calming, fun, easy to access (art, crafts, music, swimming, and walking).
	Help patient identify 1 or 2 activities that can trigger physical release when emotions become overwhelming (eg, ice cubes on face or wrists, running in place).
	Praise the patient every time he/she uses coping techniques.
Impaired sensory integration:	Reduce all distracting stimuli in the environment to the extent possible:
Difficulty modulating incoming stimuli (eg, noise, lights, smells, tastes, tactile sensations).	Prefer individual over group therapy in treatment planning.
	Teach patient to keep living area uncluttered and basic.
	Keep treatment areas uncluttered and basic.
	Find ways to reduce auditory distractions during treatment.
	Avoid fluorescent lights.

FASD, fetal alcohol spectrum disorder.

developed an easily administered self-report screen for assessing behavioral characteristics and life history events associated with FASD. A pilot study reporting preliminary findings indicates promising results with good levels of sensitivity (71%) and specificity (79%) (Grant, Novick Brown, Graham, Whitney, and Dubovsky, 2012, manuscript under review.)

CLINICAL CASE EXAMPLE—"JANE"

Since 1991, the Parent-Child Assistance Program (PCAP), a 3-year home visitation case management program in the Pacific Northwest and Canada, has offered supportive relapse prevention services to pregnant and parenting women in recovery (Grant et al., 2005, 2007). The overriding goal of the program is to prevent FASD in their offspring. To date, close to 3000 women have participated in the PCAP. The case study that follows involves one of the women enrolled in PCAP ("Jane"), a young white client in her mid-20s.

Like many women who have taken part in the PCAP neuropsychological evaluation process, Jane was generally aware that there was something about the way her brain worked that prevented her from being able to accomplish goals and be successful in life. Like many of her program peers, she reported a long history of alcohol and polysubstance abuse and multigenerational alcoholism in her family. Jane hoped that the results of the neuropsychological evaluation would help her gain a better understanding of "what is going on in my head." Asked to describe her current cognitive complaints, she reported significant problems across a number of domains, including attention and concentration, learning and memory, expressive and receptive language, and arithmetic skills. Jane also reported a tendency to become easily overwhelmed when given information or instructions because of a lifelong inability to organize her thoughts in a coherent way. She added that the struggles with daily life often left her feeling anxious and depressed.

During a clinical interview before neuropsychological testing, Jane reported that she had always "struggled a lot" in school. She remembered having problems focusing in first grade and said that she had difficulty understanding course material and "keeping up with things" by the time she reached third grade. After an individualized education plan was finally developed for her, she was placed in a series of special education classes. Unable to cope with the challenges of junior high,

TABLE 2. Strategies to Avoid When Treating Patients With PAE/FASD

DO NOT read such patients a list of rules and regulations at intake or hand them a written copy of rules and regulations without verifying they understand what they are hearing/reading.
DO NOT give multiple instructions or directions.
DO NOT expect such patients to think about and implement treatment concepts/ideas on their own.
DO NOT use something that a patient enjoys to reward good behavior and remove it for negative behavior
DO NOT implement delayed or long-term consequences.
DO NOT use treatment methodologies that rely solely on verbal communication (eg, motivational interviewing, insight-oriented psychotherapy) or unstructured milieus (eg, wilderness programs).
DO NOT assume that group treatment will be effective.
DO NOT conclude when patients shut down or fail to make progress that they are unmotivated.
DO NOT overload the treatment environment with distracting stimuli and many people.
DO NOT change schedules, appointment days, or appointment times if possible.
DO NOT have multiple treatment plans with multiple goals.
DO NOT assume that lack of progress, shutting down, isolation, or emotional outbursts are under the patient's volitional control.

FASD, fetal alcohol spectrum disorder; PAE, prenatal alcohol exposure.

Jane eventually left school in the middle of seventh grade to be home schooled by a close relative. Later, efforts to complete high school classes ended unsuccessfully at the age of 16 years when she dropped out of school in frustration. Since then, she has made 2 attempts to complete a general education diploma but abruptly stopped soon after starting the preparation courses because she was unable to understand the course work.

During her early adult years, Jane held a number of entry-level jobs including grocery bagging and stocking and light janitorial work. She was not able to sustain a job for more than a few months primarily because of difficulties understanding verbal and written instructions and occasional social problems with coworkers. Jane reported that although she took pride in her work and enjoyed working, she was currently unemployed and worried about her ability to support herself.

Responding to questions about her psychiatric history, Jane disclosed that she had struggled with daily depression and anxiety for many years. She also described occasional panic attacks. She reported a relatively unremarkable medical history and knew of no pre-l or postnatal events that had impacted her early development. However, she did recall relatives mentioning that her mother drank alcohol throughout the pregnancy with her. Jane's own history was positive for significant alcohol and polydrug abuse, beginning at the age of 12 years, which had resulted in several arrests and a jail sentence for drug possession.

At the time of her interview, Jane had been taking part in an intensive and highly structured inpatient substance abuse treatment program specifically designed for parenting women. This was her second experience with inpatient treatment. She had dropped out of treatment the previous year when she became frustrated over her inability to progress in treatment like her peers. She noted that despite some challenges in her current treatment, she was in compliance with program requirements and found staff to be helpful and supportive.

After completing her clinical interview, Jane was administered 4 hours of neuropsychological testing involving a test battery that included measures sensitive to the functional effects of PAE. Testing confirmed that executive function deficits significantly impaired her ability to effectively organize and structure her environment and cope with stress, which led in turn to a chronic state of feeling overwhelmed with life's challenges and associated mood disorders (eg, anxiety and depression). Given her confirmed PAE, evidence of cognitive problems in childhood before the development of competing causes (eg, her own substance abuse and pre- and postnatal adversities that do not involve PAE), and neuropsychological test results consistent with diagnostic impairments, she was diagnosed with alcohol-related neurodevelopmental disorder. After testing was completed, feedback conferences were held with Jane, her mother, and treatment staff. Discussion focused on cognitive strengths and weaknesses, and recommendations were provided regarding optimal teaching/learning strategies. After treatment plan modifications that incorporated these recommendations, Jane was able to complete her course of inpatient treatment successfully.

After 6 months, Jane had enrolled in an intensive outpatient treatment program near her home. Although she had been able to avoid relapse for 6 months, she was struggling with life and self-esteem issues and starting to feel overwhelmed again. Asked what had changed for Jane since leaving inpatient treatment, her PCAP case manager reported that Jane's local outpatient program was unable to provide the kind of structure, routine, and focus she needed to succeed. Environmental factors also interfered: many of Jane's family members continued to drink heavily while holding her to an abstinence standard. In addition, her friends frequently tried to sabotage her efforts to be successful. Meanwhile, she continued her lifelong pattern of seeking approval and acceptance from others and consequently remained vulnerable to people who took advantage of her gentle and trusting nature by temporarily offering her the affirmation she was seeking.

In Jane's situation, confirmed PAE represented a substantial vulnerability factor that set her on a developmental trajectory that was different from those without exposure. In particular, information gathered during her clinical interview found long-standing issues with cognition, learning, and many aspects of daily functioning. Neurological testing confirmed that executive function deficits significantly impaired her ability to effectively organize and structure her environment and cope with stress, which led in turn to a chronic state of feeling overwhelmed with life's challenges and associated mood disorders (eg, anxiety and depression).

It was clear to all that Jane's level of functioning improved when she was in a structured inpatient environment, which removed many of the unpredictable elements present in the "real world." However, her cognitive impairments were not addressed in the outpatient treatment program because of staff's unfamiliarity with FASD. After observing similar unsuccessful treatment outcomes in a number of women with PAE/suspected FASD, the PCAP intervention in which Jane was enrolled began offering other clients with a history of PAE an opportunity to participate in comprehensive neuropsychological evaluation to assess cognitive functioning and learning

styles so as to individualize service planning. To date, 20 women have been evaluated, and eventually their 3-year PCAP outcome data will be compared with approximately 60 PCAP clients with PAE who have not had neuropsychological testing but have had histories of social problems and adult behaviors consistent with FASD.

There were a number of risk factors or red flags in Jane's history that suggested FASD long before she entered treatment. For example, well before she began experiencing alcohol and drug problems, her history involved poor academic performance, low self-esteem, troublesome peer and family relationships, and intergenerational substance abuse that made it likely she would begin using alcohol and drugs at an early age (Hawkins et al., 1992; Jacob and Leonard, 1994). In her case, those early risk factors went largely unattended, but Jane's recent experience teaches us the importance of paying attention to such risk factors, especially those that could mean serious impediments to treatment progress. In particular, when clients like Jane report "learning problems" in school, such information should set in motion an assessment process to determine the basis for those learning problems.

To achieve optimal outcomes in treating individuals with FASD—diagnosed or not-–it will be important first of all for providers to screen for and detect cognitive and functional impairments. For clients who have such disabilities, "one size fits all" treatment program modalities will need revisions. Importantly, if someone has already failed previous treatment, we should adopt the perspective that it is not the *patient* who failed but rather the *treatment program* that failed to individualize the patient's treatment plan and provide services in a manner that supported his or her potential for success (Dubovsky, 2002). Although we recognize that basic programmatic changes are a major undertaking, developing individualized services for clients with significant cognitive impairments is likely to be the difference between treatment failure and a real chance for success.

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