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Neurotoxicology and Teratology 25 (2003) 681–688
NEUROTOXICOLOGY AND TERATOLOGY
www.elsevier.com/locate/neutera

Recognition and management of fetal alcohol syndrome☆

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Accepted 11 July 2003

Abstract

Fetal alcohol syndrome (FAS) is a common cause of developmental disability, neuropsychiatric impairment and birth defects. The disorder is identified by the presence of growth impairment, central nervous system dysfunction, and a characteristic pattern of craniofacial features. The reported prevalence of the disorder varies widely and recent estimates approach 1% of live births. Expression of these features varies by age. People with FAS have high rates of comorbid conditions: attention deficit hyperactivity disorder (40%), mental retardation (15–20%), learning disorders (25%), speech and language disorders (30%), sensory impairment (30%), cerebral palsy (4%), epilepsy (8–10%). Birth defects are common. In the United States, the annual birth cohort of persons with FAS could be as high as 39,000 cases annually. Cause-specific mortality is 6% for patients with FAS. The disorder is expensive to treat and most patients have lifelong impairment. The annual cost of care in the United States would approach US$5.0 billion. Early recognition and entry into appropriate treatment programs appear to improve outcome. Prevention efforts should involve screening for alcohol use prior to pregnancy and at the first prenatal care visit.

Keywords: Fetal alcohol syndrome; Diagnosis; Treatment; Prevention; Prevalence; Comorbidity; Epidemiology

1. Introduction

In 1892, Templeman reported on 258 deaths from Dundee, Scotland due to overlaying of children of alcoholic women who drank during pregnancy [40]. He noted that 46% of the infant deaths due to overlaying occurred between Saturday night and Sunday morning. Sixty-two percent occurred during the winter months, 32% had been born to unmarried mothers (compared to 10.3% of children in the city). The risk was highest from birth to 3 months and disappeared by 9 months. In 1899, Sullivan reported on the role of maternal alcoholism as a cause of infant mortality in 100 incarcerated women [39]. Of the 600 children born to these women, 55.8% were born dead or died before age 2. The rate of infant mortality was doubled in alcoholic mothers. Eighty women had three or more infant deaths. Later born children had an increased risk compared to earlier births (1st borns 33.7%; 6th to 10th born 72%). Sixty-one percent of these alcoholic women had one or both alcoholic parents. In the 299 surviving children born to mothers with a previous child death, the rate of epilepsy was 4.1% (10 to 40 times higher than in the general population of England at that time). Sixty-one years later a physician in France described a pattern of physical and behavioral characteristics of children with prenatal alcohol exposure [25]. This was followed by detailed descriptions of a physical phenotype with a spectrum of impairment producing a distinctive pattern of dysmorphic facial features in some children of mothers who abused alcohol during pregnancy [23,24]. This pattern of abnormal facial features, growth impairment, and neurologic abnormalities resulting from prenatal alcohol exposure was aptly described as fetal alcohol syndrome (FAS) [23,24].

FAS has been reported across the world [4,7]. In the United States, the problem is now a public health problem of sufficient scope to be the focus of a monograph by the

☆ This work was supported by 5KD1SP09199-01, 02, 03 from the Centers for Substance Abuse Prevention; Substance Abuse and Mental Health Services Administration.

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doi:10.1016/j.ntt.2003.07.020

Institute of Medicine and a specific objective in the United States Public Health Service's Healthy 2000 campaign [35]. In this paper we will discuss the mechanism of teratogenicity of ethanol, epidemiology, costs of treatment, diagnosis, intervention strategies, and prevention of FAS and related disorders.

2. Methods

2.1. Teratogenicity of ethanol

Ethanol readily crosses through the placenta and blood alcohol levels in the fetus equal those of the mother in minutes. In the central nervous system, ethanol acts to reduce neural cell progenation, and to increase cell death by apoptosis. This occurs throughout pregnancy. Prenatal alcohol exposure causes massive cell loss from late ethanol exposure in the developing mouse brain [30]. Burd et al. [12] have presented a conceptual model of cognitive and behavioral dysfunction damage to the brain from prenatal alcohol exposure. Several imaging studies utilizing CT and MRI have demonstrated increased rates of structural brain malformations in exposed children [32]. Population-based prevalence studies of CNS abnormalities are not yet available. Current case series data suggest increased rates of midline defects, heterotopias, and damage to sensory systems.

2.2. Prevalence of FAS

Understanding the epidemiology of FAS is critical in determining the societal impact of the disease, as well as allocating funding for prevention and treatment programs. Published prevalence estimates of FAS and related disorders have been quite variable [7,18,33]. Over 20 prevalence estimates have been completed.

Prevalence estimates of FAS range from 19% of live born infants in one study to as few as one case per 10,000 live born infants [2,7,18]. The most widely used summary prevalence estimate of FAS is 1 to 1.5 cases per 1000 live births [17,18,33]. Much of the variation is due to ascertainment strategies. Studies with population-based screening produce higher prevalence rates than other case finding strategies. Less complete and widely variable manifestations of the syndrome that do not meet the full criteria for FAS are six to eight times more prevalent [17,33]. These related disorders have been described as fetal alcohol effects (FAEs) or more recently as alcohol-related birth defects (ARBDs) and alcohol-related neurodevelopmental disorders (ARNDs). Using a rate of 1.0 cases of FAS and the related disorders (ARND and ARBD) per 1000 live births, the number of new cases in the annual birth cohort for the United States (3.9 million) would be 39,000. The total number of affected people (children and adults) in the United States would exceed 2.6 million.

The cause-specific mortality rate from FAS and related disorders is 6% or about 2100 to 2300 deaths annually in the United States. These prevalence rates establish FAS and related disorders as an important cause of morbidity and mortality.

2.3. Costs of FAS

Annual cost estimates for FAS and related disorders in the United States range from US$74.6 million [6,7,22] to US$9.7 billion dollars per year [15]. Costs like prevalence are strongly influenced by diagnostic thresholds. The most recent cost estimates by Abel use a prevalence rate of 0.33 cases per 1000 live births and an annual cost of US$74.6 million [2,7]. Two other cost studies have produced lifetime cost of care per case in excess of US$1.4 million [15,17]. These costs include neonatal care; management of developmental delays and birth defects; years of special education; decades of developmental disabilities services; costs to the criminal justice system; alcohol and drug abuse treatment; mental health services; health care costs; and a lifetime of supported living costs. A lifetime cost of care of US$1.4 million for each patient with FAS suggests that funding for prevention of FAS and related disorders should be a public health priority [19].

2.4. Maternal risk factors

Current estimates suggest that less than 5% of severely alcoholic women who become pregnant and drink heavily throughout pregnancy will have children with complete FAS [2,3,5]. However, once a woman has a child with FAS, the risk of having another child with FAS may be as high as 75% in each subsequent pregnancy, if the woman continues to drink [1,2]. Thus, multiple risk factors act to form a causal chain in addition to prenatal ethanol exposure to influence risk [3,19]. Abel et al. [2,3] have suggested that risk markers for FAS can be conceptualized as permissive and provocative. "Permissive" risk markers are sociobehavioral risk markers that provide the context within which vulnerability to the teratogenic effect of alcohol is increased. These permissive risk factors create an environment that is said to be "provocative" for FAS, causing cellular susceptibilities to the toxic effects of alcohol. They include: (1) consumption of large amounts of alcohol in a short period of time and chronically elevated blood alcohol levels; (2) low socioeconomic status and malnutrition; (3) cultural and racial factors; (4) smoking; and (5) increased maternal age [2,3,16]. Population-based studies of maternal and paternal risk factors support this model of risk interaction and stratification [1,5,11,16]. Burd et al. utilized a case–control methodology with birth certificate data to identify both maternal and paternal risk markers for a population-based study of FAS (Table 1) [11,16]. Fig. 1 summarizes the data on the FAS family developed from data from North Dakota studies.

Table 1
Maternal and paternal risk factor for FAS from 132 cases of FAS/FAE from North Dakota (adapted from Bagheri et al. [10])

Variable	Case/control	Mean (difference) case/control	P
Mother's age (years)	132/660	27.4/25.4 (2.0)	<0.001
Father's age (years)	43/215	32.0/27.8 (4.2)	<0.001
Weight gain in pregnancy (lbs)	33/165	22.1/30.4 (8.3)	<0.001
Gestation (weeks)	101/505	38.7/40.0 (1.3)	<0.001
Month prenatal care began	123/615	3.4/2.7 (0.7)	<0.001
Number of prenatal visits	123/615	5.5/9.7 (4.2)	<0.001
Mother's education	125/625	10.6/13.0 (2.4)	<0.001
Father's education	32/160	10.8/13.2 (2.6)	<0.001

2.5. Screening

For many disorders, the first step in diagnosis is the identification of a high-risk group. This is often accomplished by screening or surveillance strategies. Screening strategies for FAS–ARND have been discussed in detail [13,14]. Four screening tools for FAS have been reported [8,14,20,38]. The tool by Streissguth et al. [38] has been primarily utilized in adolescents and adults. The screening tool from Astley and Clarren [8] has normative data from a clinic sample. A recent screening strategy for screening in a juvenile justice system has been reported [20]. This study found that 23.7% of the children entering the juvenile justice system in British Columbia met criteria for FAS or a related disorder. A tool for FAS screening is included as Appendix A [14]. This tool has acceptable epidemiologic performance characteristics for use in clinical and community settings, takes about 10 min to complete, and can be used with children from 4 to 14 years of age [14].

2.6. Diagnosis

A wide range of signs of FAS and related disorders needs to be considered [9,17,18,36]. These are widely variable and age dependent. Diagnostic accuracy and

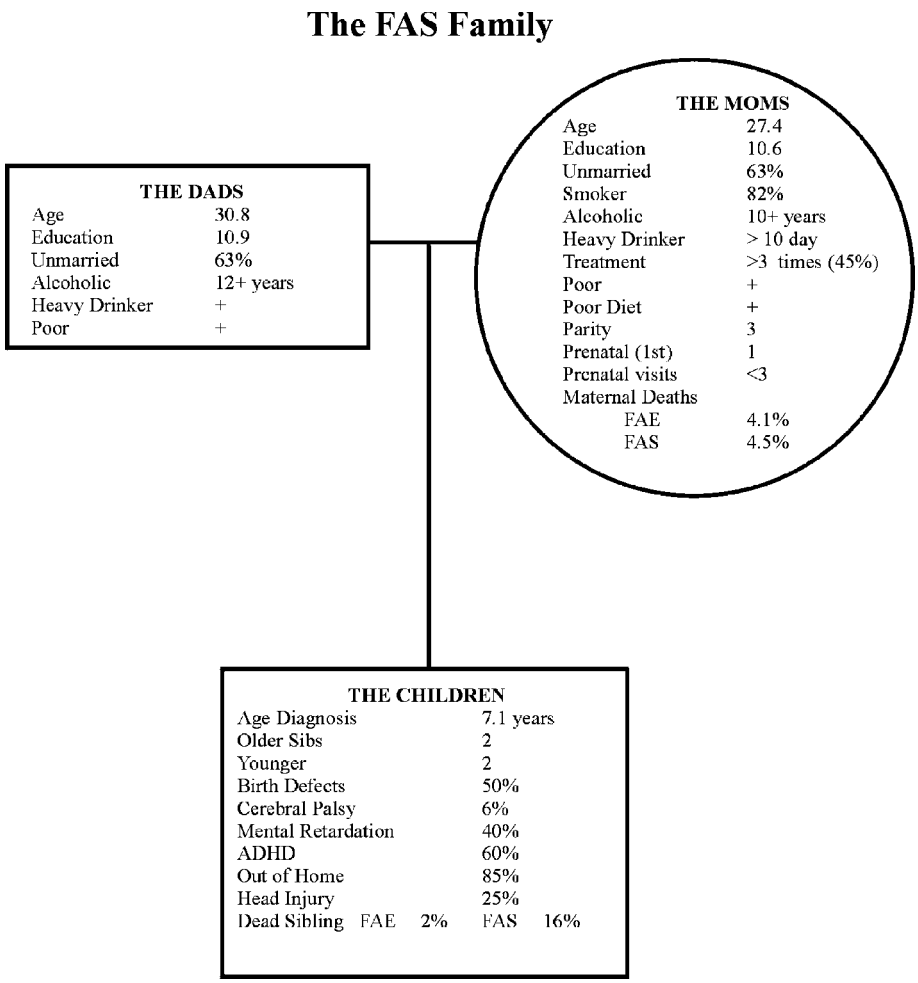

Fig. 1. A summary family pedigree of the characteristics of a child with FAS and the parents. The pedigree was developed using data from The North Dakota FAS Registry.

Table 2
Secondary disabilities in 400 adolescents and adults with FAS and FAE [37]

1. Mental health problems: 90%
2. Disrupted school experience (suspended or expelled from school to dropping out of school): 60%
3. Trouble with the law: 60%
4. Confinement: Includes inpatient treatment for mental health problems, alcohol/drug problems, or incarcerated for a crime: 50%
5. Inappropriate sexual behavior: 50%
6. Alcohol/drug problems: 30%

Protective factors

1. Living in a stable and nurturant home for over 72% of life
2. Being diagnosed before the age of 6 years
3. Never having experienced violence against oneself
4. Staying in each living situation for an average of more than 2.8 years
5. Experiencing a good quality home from age 8 to 12 years
6. Having applied for and been found eligible for Division of Developmental Disabilities services
7. Having a diagnosis of FAS
8. Having basic needs met for at least 13% of life

reliability are also age dependent [18,26]. Accurate diagnosis of FAS in newborns is difficult [18] as is diagnosis in adults [36,37]. Several different diagnostic schema have been utilized and all have emphasized the triad of: (1) growth impairment; (2) facial anomalies; and (3) neurobehavioral dysfunction [9,15,35]. Since most children are adopted or in foster care at the time of diagnosis, an accurate prenatal exposure history is often very difficult to obtain. It is also uncommon to have reliable data on the cumulative alcohol exposure during pregnancy. This dilemma is reflected in the recent diagnostic schema for FAS and related conditions recommended by the Institute of Medicine [35].

Prenatal alcohol exposure should be considered as an etiologic factor for any child with cognitive, behavioral disorders, sensory impairment, epilepsy, or structural malformations of the brain where no other etiology is apparent. Consideration of FAS or ARND should be high for any child with a developmental disorder who is adopted, in foster care, or when the mother has alcoholism.

The cutoff for growth impairment is commonly the third percentile for height, weight, and head circumference. The facial features of FAS provide much of the specificity in the diagnosis of FAS but not ARND and are also among the most variable of the signs of FAS across development.

An early and accurate diagnosis allows the physician and treatment team to better communicate with the parent, caregivers, and the patient and to describe the pathophysiology and etiology of the child's presenting problems. This enhances formulation of a deficit-based treatment program and is essential to the development of an anticipatory model of intervention. Considerable effort is currently underway to identify the behavioral phenotype of FAS and the related disorders.

A differential diagnosis may include conditions that feature growth retardation and facial anomalies, or those that share some cognitive and behavioral signs. Examples of these are the following: William's syndrome, Noonan's syndrome, Dubowitz's syndrome, Bloom's syndrome, fragile X syndrome, and Turner's syndrome. Detailed diagnostic protocols for FAS and related disorders are available [9,15].

2.7. Management

Appropriate developmental and medical management of patients with FAS differs by the age of the affected person, the cognitive status of the patient, the patient's birth defects and neuropsychiatric comorbidities [17,36,37]. A comprehensive assessment of a child will also examine the current alcohol use patterns in the caregivers. This is an important consideration for children with FAS of any age in the care of their biological parents or their relatives. The risk for FAS and related disorders will be very high in these children. The predictive risk of FAS or a related disorder is increased over 700 times for the younger siblings of a child diagnosed with FAS [1,2]. Thus, assessment of all maternal side siblings of the proband is important, even if they are in adoptive or foster care.

Appropriate management nearly always involves a multidisciplinary team effort with ongoing programming and support continuing over the person's lifetime. One of the most important components in management of all patients with FAS is the prevention of secondary disabilities [37]. Table 2 lists common secondary disabilities, the estimated rates at which they occur, and the protective factors for secondary disabilities [37].

In Table 3 we provide several general considerations for the development of a clinical management schema for patients with impairments resulting from prenatal alcohol exposure. This effort should be conceptualized as an ongoing effort to match developmental needs with the chronological age of the patient to interventions.

Table 4 lists specific guidelines for management of FAS by chronological age groups. In addition to access to services, the most frequent management problem we encounter is the premature discontinuation of supportive services for people who are making behavioral progress. Persons

Table 3
Major components to be considered in development of a treatment program for persons with FAS and related disorders

- Start early—avoid abuse
- Develop a 10-year plan
- Avoid multiple foster homes
- Treat in the community where the child will live
- Make a place in the community
- Avoid problem peers
- Use legal system

Table 4
A developmental management model for children and adults with FAS and related disorders

Newborn management:
1. Diagnostic assessment of both physical and neurologic anomalies, with resuscitation as needed.
2. Early involvement of child protective services.
3. Referral to the Department of Human Services for initiation of infant stimulation.
4. Prevention of secondary disabilities, specific to age group, abuse, vision/hearing deficits.
5. Referral of mother to substance abuse treatment program.
6. Sensory evaluation (vision and hearing).

Childhood management:
1. Head Start Program with special learning environment, addressing specific learning disabilities.
2. Annual developmental assessment.
3. Referral for services to address neurologic deficits and psychosocial delays.
4. Prevention of secondary disabilities.
5. Monitoring social skill development.

Adolescent management:
1. Prevention of secondary disabilities, specifically drug and alcohol use. Prevention of school failure, social problems, and institutionalization.
2. Vocational training, preparation for future employment.
3. Continuation of developmental assessments.
4. Develop long-term plan (10 years).
5. Social skill development and monitor peer group.

Adult management:
1. Employment services (vocational rehabilitation).
2. Social services and long-term support.
3. Monitor for substance abuse.
4. Enhance awareness in adult service systems: mental health, substance abuse, corrections.

with FAS and the related disorders typically require very long-term services and usually will not maintain behavioral, vocational, or self-care gains without ongoing support [17,37]. This is especially noticeable in adolescents and adults who are asked to make a wide variety of social and vocational decisions. Inadequate services often lead to loss of employment or increased involvement with the criminal justice system. Impairments of the executive functions of the frontal lobes may produce this deficit in judgment, especially in social situations. The impairments most prominent in FAS and related disorders are listed in Table 5 adapted from Grafman and Litvan [21].

2.8. Prevention

Current research has emphasized a prenatal ethanol exposure "plus" as a causal chain for FAS. Research has begun to focus on a wider range of important maternal, paternal, prenatal, and postnatal risk factors [3,5,10,16,27,31,34]. Incorporating a broader understanding of the lives of women at highest risk for FAS and ARND has been an important step in the development of prevention programs [2,10,19,28]. Simultaneous community-based application of three levels of prevention are needed [35].

2.8.1. Universal (primary) prevention

Universal prevention is aimed at the complete abstinence of alcohol use prior to and during pregnancy, to include the male partner and other social support groups. This would obviously guarantee the primary prevention of FAS and related disorders. Universal prevention is best incorporated with public education, through the media and literature. An important resource of universal prevention is the primary care physician, who provides routine alcohol use and abuse screening and patient education about the risks of drinking during pregnancy for all women, prior to conception, beginning at early adolescence.

2.8.2. Selective (secondary) prevention

Selective prevention is aimed at decreasing the duration and level of the maternal drinking. This is accomplished by: (1) identifying the high-risk drinkers through screening in

Table 5
Neuropsychological functions and impairments in patients with FAS and related disorders

Functional area of impairment	Behavioral manifestation
Attention—concentration	Distracted—difficulty concentrating
Predictive planning	Unable to perform routine activities routinely
Adaptive planning	Cannot continue on with ongoing activity after interruption
Short-term planning	Unable to carry out activity after pause
Reasoning	Unable to adjust to new demand or change in activity—cannot figure things out
Social skills	Does not utilizes age-appropriate social skills—stranger identification, play or dating, eating, social boundaries
Thematic understanding	Understands situation or event (TV, social activity, or story)
Inhibition	Repeats activities or problem-solving approaches that have not worked for patient in past (stereotyped approaches)
Motivation	Patient is difficult to reward or motivate

The development of these skills is influenced by age and IQ. The manifestation of these problems frequently change in severity and may be situational strengths or impairments, since the generalization of these skills is often impaired [21].

Table 6
Table modeling cost of prevention by risk level using alcohol treatment as the intervention

Treatment 50% effective					
Alcohol use and other risk factors	Risk of FAS (%)	Women treated	Women quit[a]	Cases prevented	Cost per case prevented
Daily alcohol use	0.01	20,100	10,000	1	US$100,000,000
Heavy drinkers, middle class, nonsmokers	0.29	690	344	1	US$3,450,000
Heavy drinkers, low income, smokers, poor diet	4.3	47	23	1	US$235,000
Women who have had a previous child with FAS	75.0	3	1	1	US$15,000

Additional information and more detailed modeling data are available at: http://www.online-clinic.com on FAS Exposure Model.
[a] Quit after 1 year.

various health settings, social service settings, and work place programs; and (2) by appropriate treatment to reduce or eliminate alcohol use. A useful and brief screening tool is available [29]. The TWEAK is a five-item screening tool which has been effective in identifying problem drinkers during pregnancy [29]. Every woman seeking prenatal care should be screened. Women with alcohol abuse during pregnancy should be referred for assessment and treatment.

2.8.3. Indicated (tertiary) prevention

Indicated prevention focuses on reducing the complications, accompanying impairments, and resulting disabilities of a child born with FAS and assisting the mother in changing her substance abuse patterns and/or prevention of future pregnancies.

Table 6 provides a model of a risk stratified approach to prevention with costs of prevention per case [19]. In addition to reducing or eliminating prenatal alcohol exposure, a range of other risk factor reductions is possible. It is important to remember that only a small portion of alcoholic women will give birth to a child with FAS, therefore effective strategies should prioritize identification of: (1) women with a previous child with FAS; and (2) women with a history of excessive drinking, smoking, a poor diet, and low socioeconomic status.

Since FAS is typically an alcohol exposure "plus" syndrome, the opportunity to reduce or eliminate other risk factors from the causal chain for FAS should be more widely utilized. These include enhancing a woman's diet, reducing smoking, reducing physical and emotional abuse, and enhancing a woman's current living status. Each of these components offers an opportunity to improve the outcome of current and future pregnancies. Developing the capacity for effective prevention and treatment of FAS is a long-term process and for most communities will require considerable capacity building and funding [19].

3. Conclusions

Developmental assessment clinics will likely encounter many children with FAS and ARND. They are in a unique position to provide resources on brain function, assessment of resulting impairments and to provide direction on appropriate intervention activities. Their leadership in diagnosis, management, and prevention activities will enhance patient outcomes. Each state needs to have an FAS task force to implement appropriate identification, treatment, and prevention strategies for FAS and related disorders. This would include screening to identify at-risk women and a system to link mothers with diagnosed children in order to identify them for immediate substance abuse treatment. Communities also need to develop and advocate for funding of appropriate regional treatment programs for these extremely high-risk women. Many of these women will have other children and effective treatment programs will need to be long term and to have the capacity to include their children in the treatment programs. This system also needs to emphasize early identification of affected children, early entry into treatment, prevention of secondary disabilities, and development of a specialized service delivery system for affected adults.

A small number of women have a large proportion of children with FAS and the related disorders. Prevention will be difficult, as the necessary strategies for identification and referral for appropriate treatment are not often utilized. As a result, many women who would benefit from treatment–prevention efforts are missed. The substance abuse–FAS prevention link should include: (1) identifying and maintaining contact with the women most at risk; (2) education, both public and professional; (3) increasing the accessibility of long-term intensive treatment programs; and (4) helping these women alter their living environments and peers after substance abuse treatment [2,19,27]. An initial emphasis on the biological mothers of affected children has the greatest potential for prevention by reducing subsequent affected children in these high-risk women. Planning a very long-term system of care should decrease costs and improve outcomes for affected persons. The cost of inaction is high and the prevention of only a few cases at lifetime cost of care of US$1.4 million per case would easily fund many of the required activities.

Appendix A

FAS SCREENING FORM[39]

Name _____ DOB ___/___/___ AGE _____ SEX (circle one) F M
DATE OF EXAM __/__/__

CHILD'S R ACE (circle one)
1) white
2) NA
3) other

HEIGHT_____	INCHES	<5%	Y____	N____	10
WEIGHT_____	POUNDS	<5%	Y____	N____	10
HEAD CIR._____	CM	<5%	Y____	N____	10

HEAD AND FACE	EARS STICK OUT (Protruding Auricles)	Y____ N____	4
	SKIN FOLDS NEAR INNER EYE (Epicanthal Folds)	Y____ N____	5
	DROOPING OF EYELIDS (Ptosis)	Y____ N____	4
	CROSS-EYES, ONE OR BOTH EYES (Strabismus)	Y____ N____	3
	FLAT MIDFACE/CHEEKS (Hypoplastic Maxilla)	Y____ N____	7
	FLAT/LOW NOSE BETWEEN EYES (Low Nasal Bridge)	Y____ N____	2
	UPTURNED NOSE	Y____ N____	5
	GROOVE BETWEEN LIP & NOSE ABSENT OR SHALLOW (Flat Philtrum)	Y____ N____	5
	THIN UPPER LIP	Y____ N____	4
	CLEFT LIP OR CLEFT OF ROOF OF MOUTH (present or repaired)	Y____ N____	4
NECK AND BACK	SHORT, BROAD NECK	Y____ N____	4
	CURVATURE OF THE SPINE (Scoliosis)	Y____ N____	1
	SPINA BIFIDA (History of Neural Tube Defect)	Y____ N____	
ARMS AND HANDS	FINGERS, ELBOWS (Limited Joint Mobility)	Y____ N____	4
	PERMANENTLY CURVED, SMALL FINGERS, ESPECIALLY PINKIES (Clinomicrodactyly)	Y____ N____	1
	DEEP OR ACCENTUATED PALMAR CREASES	Y____ N____	4
	SMALL NAILS/NAIL BEDS) (Hypoplastic Nails)	Y____ N____	1
	TREMULOUS, POOR FINGER AGILITY (Fine Motor Dysfunction)	Y____ N____	1
CHEST	SUNKEN CHEST (Pectus Excavatum)	Y____ N____	3
	CHEST STICKS OUT (Pectus Carinatum) OPTIONAL	Y____ N____	1
	HISTORY OF HEART MURMUR OR ANY HEART DEFECT	Y____ N____	4
SKIN	RAISED RED BIRTHMARKS (Capillary Hemangiomas)	Y____ N____	4
	GREATER THAN NORMAL BODY HAIR, HAIR ALSO ON FOREHEAD AND BACK (Hirsutism)	Y____ N____	1
DEVEL-OPME NT	MILD TO MODERATE MENTAL RETARDATION < 70)	Y____ N____	10
	SPEECH AND LANGUAGE DELAYS	Y____ N____	2
	HEARING PROBLEMS	Y____ N____	1
	VISION PROBLEMS	Y____ N____	1
	ATTENTION CONCENTRATION PROBLEMS	Y____ N____	2
	HYPERACTIVITY	Y____ N____	5

COMMENTS:

SCORE TOTAL_____
Refer if 20 or above

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