

Unfortunately, at least in the near future, such systems are unlikely to be set up in many developing countries, partly because of lack of financial and human resources but largely because of lack of political will to face this problem. Creating this political will is the responsibility of the international obstetric community.

OUR PROPOSAL FOR IMPROVING MATERNAL HEALTH

At international meetings of obstetricians, where developing countries are well represented it is depressing to find that the emphasis is almost entirely on the high-technology subspecialties and that sessions on the social issues are usually attended by a small minority of physicians who are already knowledgeable about these problems.

Despite publications about the proportion of maternal deaths in hospitals due to haemorrhage, eclampsia, and so on, little thought seems to be given to the deaths from these causes that occur outside the medical-care system. The obstetric community should lead a review of the problem, in conjunction with government health departments and donor agency officials. Improvement of maternal care in developing countries is a long-term undertaking that will require national and international health planning to provide the necessary facilities, personnel, and supplies.

UNICEF has taken the lead in promoting effective primary health care for infants and young children; UNFPA in promoting family planning and population programmes for both demographic and maternal/child health purposes; and WHO in promoting overall primary health care. The World Bank has been a leader in describing the ways in which population growth hinders socioeconomic development. Some years ago the Bank established a department of population, health, and nutrition, to which it has committed sizeable resources, but programme and project developments have, in general, been difficult. In part this is because the Bank's programme is based on loans, an approach that has been successful for large capital projects such as road construction, industrial development, and dam building, but less so for social programmes such as population and health.

We suggest that the Bank makes maternity care one of its priorities. A programme for the prevention of maternal deaths could be built around the building of maternity centres in rural areas, the recruitment and training of staff for the centres, and the provision of supplies and drugs. The programme could be phased so that governments would take over these expenses in time. Loans for these purposes should be seen as an acceptable long-term investment in improving the health of women. In addition, because women receiving maternity care are an appropriate group to whom contraceptive information and services can be offered, our proposal provides an opportunity for the Bank to work toward its goal of reduced population growth rates. Leadership by the Bank, with its enormous resources, could, as an integral part of maternity care, considerably reduce maternal morbidity and mortality and perinatal mortality, and encourage contraceptive practice. In the socialist/communist countries of Cuba and the People's Republic of China, most women have ready access to maternity care, with effective referral systems for higher-level care. This has been accomplished through the allocation of the necessary resources. Such a system is not beyond the means of most countries, but it requires a dramatic shift in priorities.

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Occasional Survey

NATURAL HISTORY OF THE FETAL ALCOHOL SYNDROME: A 10-YEAR FOLLOW-UP OF ELEVEN PATIENTS

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Summary Of the eleven children who were the first to be diagnosed as having the fetal alcohol syndrome ten years ago, two are now dead, one is lost to follow-up, and the remaining eight continue to be growth deficient and dysmorphic. With menarche, which occurred with normal timing, the female patients developed increased body fat. The mothers were all severe chronic alcoholics. Four of the eight known survivors are of borderline intelligence and have needed some remedial teaching. The other four are severely handicapped intellectually and need complete supervision outside the home. The degree of growth deficiency and intellectual handicap was directly

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TABLE I—DEMOGRAPHIC AND GROWTH DATA

Patient number/ Sex	Race	Alive in 1983		Birth data					Follow-up data			
		Child	Mother	Gest age (wk)	Length (cm)	Weight (g)	HC (cm)	Breech	Age (yr)	Height (cm)	Weight (g)	HC (cm)
1/F*	Am Ind	-	?	40	45.0	1850	32.0	Yes
2/F	Am Ind	+	+	40	44.5	2500	31	No	10.7	130.0	26.36	50
3/F	Am Ind	+	+	38	47.0	2500	31	No	14.7	143.5	45.00	not taken
4/F	Black	+	-	36	42	1600	29	No	14.2	143.7	46.93	50
5/M†	Black	?	?	38	43	1673	..	Yes
6/M	Black	+	+	34	38	1550	30.5	No	13.9	145.1	31.14	50.6
7/F	White	+	+	44	45.7	2345	32	Yes	14.4	142.5	35.57	48.5
8/M	White	+	+	37	43.2	2250	35	No	13.2	145.2	32.39	53
9/M	Am Ind	+	-	38	43	2020	29	Yes	10.1	119.3	19.5	47.8
10/F*	Am Ind	-	?	32	38.5	1300	27	No
11/F	Am Ind	+	-	7 mo	..	964	..	Yes	14.0	129.5	25.85	47

*Deceased; †lost to follow-up; + = alive, - = died, ? = unknown.
HC = head circumference.

related to the extent of craniofacial abnormalities. New features of the syndrome include dental malalignments, malocclusions, and eustachian tube dysfunction, which may relate embryologically to midface hypoplasia.

INTRODUCTION

IN 1973, we described eleven children with a common pattern of altered morphogenesis and central nervous system function whose mothers were chronic alcoholics who continued to drink heavily during pregnancy.^{1,2} Since then the fetal alcohol syndrome has been identified in children from every racial group³ and in many countries;⁴⁻⁶ the teratogenicity of alcohol has been confirmed in laboratory studies involving many different species of animals;⁷ and a dose-response curve for prenatal alcohol exposure has been established.^{8,9} However, the long-term outcome and the course of the disorder have not been reported. In this paper we describe how those eleven children have developed physically and mentally over the past 10 years.

CLINICAL REPORTS

Patients 1 to 8 (table 1) are presented in the same order as when they were described by Jones et al,¹ and subjects 9 to 11 are the three children described by Jones and Smith.² The medical conditions observed in these eleven children over the past 10 years are summarised in table II, and their growth curves and intelligence quotient (IQ) scores are shown in fig 1.

Patient 1

She drowned in the bath at age 3½ years. She had been adopted at age 15 months and was an easy child to manage. Her grade 4/6 systolic murmur, interpreted as representing a ventricular septal defect, had disappeared at 15 months. She had been treated by the use of a cast and surgically for a congenitally dislocated hip and had received treatment repeatedly for ear infections.

Patient 2

This short, thin, prepubertal 10½-year-old girl has lived in a stable, adoptive home since she was 3. She is in the 5th grade, attends regular classes at school, and receives supplementary lessons for reading. Her performance is normal for her grade. She is responsible for some household chores and the shopping and her social interactions are age-appropriate.

Her craniofacial features have not changed much over the past 10 years. Her palpebral fissures remain short (right 2.5 cm, left 2.4 cm), while her inner canthal distance (3.0 cm) is normal. Her nose is not disproportionately short, but the alae nasi are narrow, she has a teardrop-shaped philtrum, and her flat midface and normal jaw size have resulted in a mild class III malocclusion. She continues to have a grade 2/6 heart murmur due to an atrial septal defect, and she also has the abnormal palmar crease pattern commonly found in the fetal alcohol syndrome.^{1,10}

The patient has been in good general health apart from chronic serous otitis media which, despite regular follow-up

TABLE II—MEDICAL DISORDERS

Disorder	Number	Disorder	Number
<i>Intra-oral</i>	8/8	<i>Other</i>	
Class III malocclusion	5/7	Breech	5/11
Poor dental alignment	3/8	Weak suck	10/11
Cleft palate	2/11	Long hospital stay after delivery	8/9
<i>Ears</i>	5/9	Failure to thrive	6/9
Chronic otitis media	5/9	<i>Behavioural</i>	
Permanent hearing loss	3/8	Past hyperactivity	5/9
<i>Eyes</i>	4/8	Present hyperactivity	1/8
Optic nerve hypoplasia	2/8	Attention deficit	7/8
Ptosis	2/8	<i>Environmental</i>	
Strabismus	4/9	Neglect	5/9
Severe myopia	2/8	Physical abuse	2/8
<i>Cardiac murmurs*</i>	8/11	Suspected sexual abuse	1/8
VSD	6/11		
ASD	1/11		
PDA	1/11		
<i>Skeletal</i>	9/11		
Congenital dislocated hip	3/9		
Progressive thoracic scoliosis	1/8		
Radio-ulnar synostosis	3/9		
Flexion contractures	1/9		
Bilateral halluces valgus	2/9		
Minor hand anomalies	6/9		

*Interpreted as ventricular septal defect (VSD), atrial septal defect (ASD), or patent ductus arteriosus (PDA).

The denominator 8 represents the 8 long-term follow-up patients (nos 2, 3, 4, 6, 7, 8, 9, and 11).

The denominator 9 also includes patient 1, who was followed-up until her death at age 3½ years.

The denominator 11 is applicable only for birth and neonatal data, available on all patients.

The denominator 7 indicates that patient 3 was not examined for malocclusion.

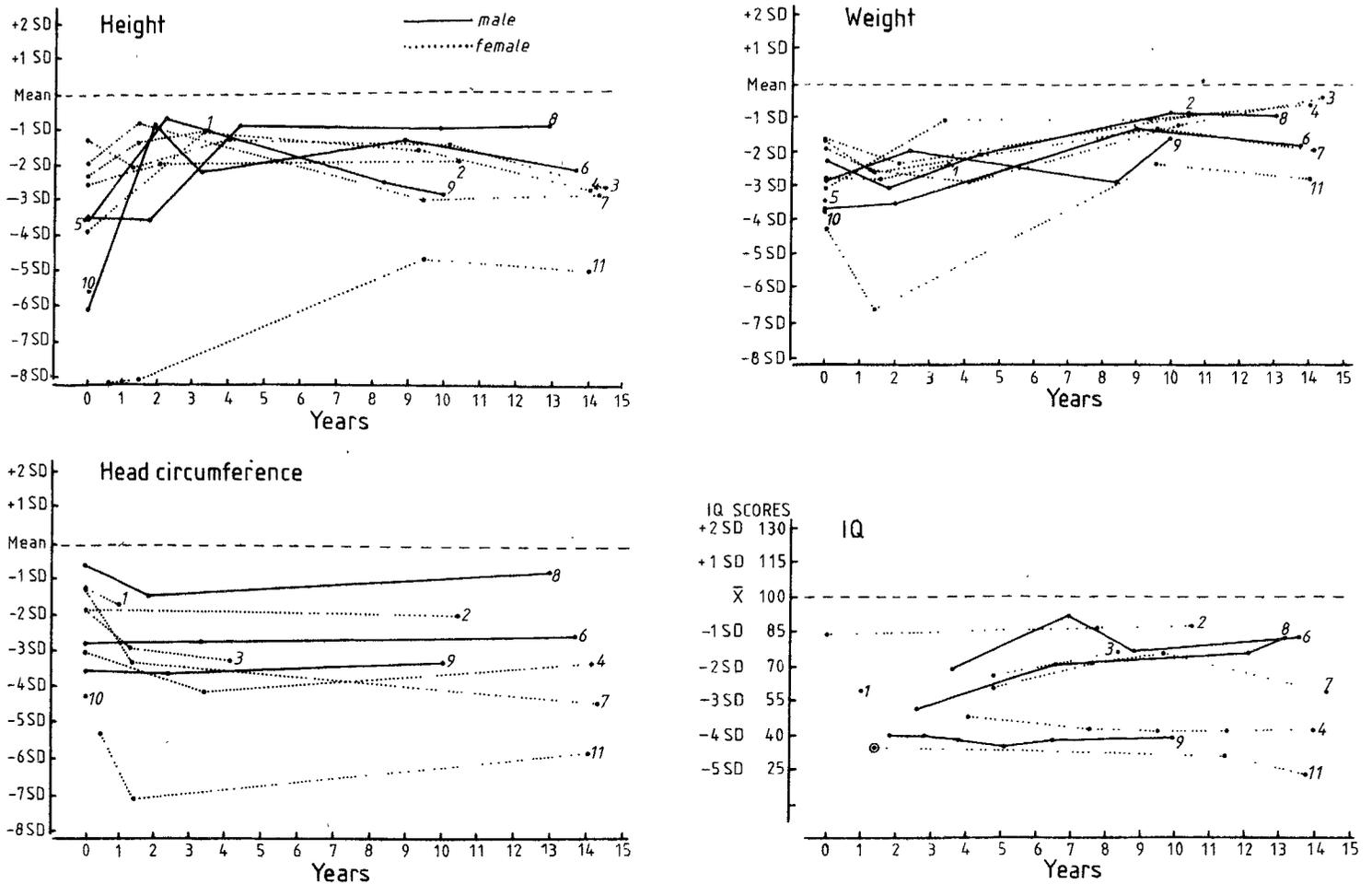


Fig 1—Growth and IQ curves with age.

Records taken at five ages are plotted when available. IQ scores are derived from individual age-appropriate tests of general intelligence and mental development, including the Wechsler intelligence scale for children (revised), the Wechsler preschool and primary scale of intelligence, the Stanford-Binet intelligence scale form L-M, and the Bayley scales of mental development. The Stanford-Binet scores reported in ref 1 have been revised according to norms published since then. The first IQ point for patient 11 is circled because it was measured from the Vineland social maturity scale and clinical observation.

and treatment with tympanic ventilatory tubes, has left her with a fixed 80 decibel conductive hearing loss in the left ear. She is undergoing standard orthodontic treatment for malocclusion.

Patient 3

She is a short, stocky, sexually-mature 14-year old girl who lives with her natural mother and several siblings. She is in the 7th grade but failing in examinations and not attending school regularly. Although she used to be cooperative and engaging, she has become suspicious, guarded, sullen, and a poor mixer at school. Family ties remain very strong, but the family is socially isolated and very poor yet steadfastly refuses help. The patient refused to be examined medically and psychologically but growth data were obtained.

Patient 4

She is a short, overweight, sexually mature 14-year-old girl (fig 2) who has lived in a stable adoptive home since she was 4½. She attends classes for the retarded and reads and spells at 2nd and 3rd grade level. She is a happy, outgoing, talkative child who has no inhibitions with strangers and is too immature to be left alone. She is no longer hyperactive but continues to have a short attention span.

The patient's facial features have not changed. Her palpebral fissures are short (2.2 cm bilaterally) but her inner canthal distance is normal (3.1 cm). The visual abnormalities include ptosis, strabismus, nystagmus, severe myopia, and

bilateral optic nerve hypoplasia. She has a moderate class III malocclusion secondary to a flat midface and prominent mandible. She continues to have prominent lateral palatine ridges. Her external ears are prominent and of an odd shape. Her skeletal anomalies include bilateral radio-ulnar synostosis, brachydactyly, and camptodactyly of the fourth and fifth digits of both hands, and clinodactyly of the third and fourth digits of the left hand; an aberrant palmar crease pattern bilaterally; and bilateral syndactyly of the second and third toes. A cardiac murmur compatible with a patent ductus arteriosus was audible until she was 5. Since infancy, she has been in reasonable health although she has needed careful ophthalmological follow-up. Orthodontia with possible oral surgery is indicated.

Patient 5

This boy, who was 17 months old when first described, has been lost to follow-up for the past 10 years.

Patient 6

This short, thin pre-pubertal 13½-year-old boy has always lived with his natural parents and siblings. He is in the 8th grade and attends regular classes, with remedial teaching for mathematics and English. He is quiet, well-behaved, cooperative, and no longer hyperactive.

He has most of the facial features of the fetal alcohol syndrome except for a normal midface, which tends to make him look less typical than others with the syndrome. His palpebral fissures are short (2.1 cm bilaterally) while his



Fig 2—Patient 4 at ages (a) 3 yr 9 mo and (b, c) 14 yr 2 mo.

Note the persistence across ages of the short palpebral fissures, hypoplastic philtrum, strabismus, and ptosis; the increased growth of the nose and mandible; and the short, stocky stature often associated with puberty in girls with FAS.



Fig 3—Patient 8 at ages (a) 2 yr 6 mo, and (b, c) at 12 yr 2 mo.

Note the short palpebral fissures, epicanthal folds, flat midface, hypoplastic philtrum, and thin upper vermillion border. Note also the short, lean pre-pubertal stature characteristic of young adolescent boys with FAS.

inner canthal distance is normal (3.3 cm). He has the typical frontonasal anomalies including epicanthal folds, short nasal tip, and hypoplastic philtral furrows. Although he has no malocclusion, his mouth is wide, his anterior teeth are irregularly shaped and poorly aligned, and his bite is open. His external ears are prominent and oddly shaped. He has bilateral radioulnar synostosis, camptodactyly of the fifth digits of both hands, and small toenails. He has been in reasonably good health although he is seeing an ophthalmologist for exotropia, has chronic allergic rhinitis and sinusitis, and is receiving orthodontic treatment.

Patient 7

She is a 14½-year-old short, microcephalic, sexually-mature female who is quiet and compliant. She has lived in a stable adoptive family since she was 6. She has attended special classes for the retarded all her life and, academically, she is at the first to second grade level. Because of her immaturity and lack of inhibition with strangers, she is not allowed to go out alone.

Except for normal palpebral fissure size (2.6 cm bilaterally), she has most of the facial features of the fetal alcohol syndrome. Her inner canthal distance is 2.7 cm. She has mild epicanthal folds, a smooth philtrum, and a thin upper lip. Her flat midface and her prognathism have produced a moderate class III dental malocclusion. She has had bilateral congenital hip dislocations and thoracic scoliosis with a 64° curvature to the left which required thoracolumbar fusion. Her nails are small and she has bilateral clinodactyly of first and fifth fingers and bilateral halluces valgus. The multiple haemangiomas present in infancy have resolved, as has the systolic murmur. She has had chronic serous otitis media and still has a mild conductive hearing loss despite regular care and the placement of tympanic ventilatory tubes.

Patient 8

This short thin prepubertal 13-year-old boy (fig 3) lives with his natural father. Although he is finding schoolwork increasingly difficult he attends regular classes, but receives supplementary lessons. His hyperactivity has gradually diminished, but he continues to have an attentional deficit. Because of refusal to attend school for 3 months, he is repeating his 7th grade, but his academic performance is 1–4 years lower. He is friendly, uninhibited, and self-sufficient; he is also a loner and immature.

His facial features remain typical of the fetal alcohol syndrome. His palpebral fissures are very short (1.9 cm on the right, 2.0 cm on the left), while his inner canthal distance is 3.7 cm. He has a unilateral strabismus, epicanthal folds, a hypoplastic philtrum, a thin upper lip, and mild class III malocclusion secondary to midface hypoplasia. The skeletal and dental anomalies include aberrant palmar creases, bilateral camptodactyly of the fifth fingers, and bilateral halluces valgus. Since the age of 1 year he has been healthy, except for having had to undergo a hernia repair and an orchiopexy at the ages of 3½ and 6 months, respectively.

Patient 9

He is a 10½-year-old boy who is extremely thin and short (fig 4). He is hyperactive to the point of being destructive, and outgoing and friendly to the point of being intrusive. He has always attended classes for trainable mentally retarded. He cannot read or write. He has received excellent care from his foster home. Seizures triggered by both high-pitched noises and hyperventilation and followed by postictal lethargy were first noted at 8 months but had stopped by the time he was 4. A grade 2/6 systolic murmur thought to be secondary to a ventricular septal defect also disappeared when he was 4. He has had frequent viral illnesses and diarrhoea, as well as persistent otitis media, for which he has been given tympanic ventilatory tubes, and his hearing is now normal. He is being prepared for orthodontic treatment. He has worn glasses since the age of 3, is photophobic and virtually blind in the right eye, and has optic nerve hypoplasia.

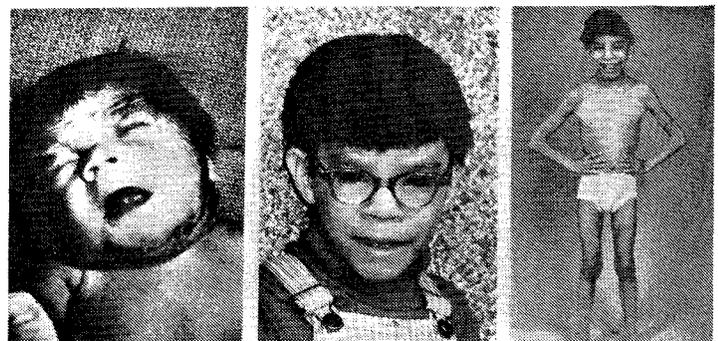


Fig 4—Patient 9 photographed at (a) birth, (b) 5 years, and (c) 8 years.

Note the short palpebral fissures, epicanthal folds, short upturned nose, long and hypoplastic philtrum, thin upper lip vermillion, flat midface, hirsutism, and characteristic emaciated appearance of the pre-pubescent FAS child with minimal subcutaneous fat. (Photograph c from Streissguth et al, CIBA Foundation Monograph 105. London: Pitman, 1984.)

He has retained the typical facial features of the fetal alcohol syndrome. His palpebral fissures are 2.5 cm long while his inner canthal distance is 3.0 cm. Midline anomalies include epicanthal folds, a hypoplastic philtrum, and a thin upper lip. He has midface hypoplasia and micrognathia. The dental alignment is poor but there is no malocclusion. His mild skeletal anomalies include radioulnar synostosis and mild hip flexion contractures, camptodactyly of his fifth fingers, small toenails, and the presence of thirteen ribs bilaterally.

Patient 10

This patient died at age 5 days, after multiple apnoeic episodes; necropsy findings have been reported elsewhere.^{11,12}

Patient 11

She is a very short, sexually mature but mentally retarded 14-year-old girl who has lived in a stable foster home since the age of 8. She is a quiet girl, is highly distractible but not hyperactive, and is totally trusting with strangers. She is obedient and compliant but too immature to be left alone.

She is in good general health but severely growth deficient. Midline anomalies include marked epicanthal folds, a hypoplastic philtrum, and a thin upper lip. Her midface is flat and she has some prognathism and a class III dental malocclusion. Her ears are prominent and have anomalous convolutions. Her skeletal anomalies include radioulnar synostosis, palmar crease anomalies, bilateral clinodactyly of the fifth digits, mild flexion contractures at the elbow and knee, and abduction contractures at the hips. Multiple small haemangiomas are still present on the trunk and there are striae atrophicae of unknown cause on the inner aspects of both legs. A systolic heart murmur, compatible with a ventricular septal defect, resolved spontaneously by the age of 10. Her cleft palate was closed at an operation when she was 3. She has also undergone surgery for the reduction of her mandible and is receiving orthodontic treatment.

DISCUSSION

Although we do not know how representative the eleven children we have followed up are of all those with the fetal alcohol syndrome, some of the features they have in common may be helpful in the planning for the future of affected individuals.

Growth

All the children remained growth deficient with respect to height, weight, and head circumference (fig 1). Although most of them showed some catch-up linear growth during the first years of life, weight and head circumference decreased relative to the norms during this time in most children. Thereafter, length and head circumference remained relatively constant with respect to the norms, while eight of the nine children on whom follow-up data were available showed some catch-up in weight with increasing age. The slow growth of the head after delivery may explain why in some infants the syndrome is diagnosed at 9–12 months of age instead of at birth.

The measurement which best summarises the appearance of children with the fetal alcohol syndrome is the weight for height age (table III). Initially the children were strikingly underweight for their height, with weight for height ages

TABLE III—WEIGHT FOR HEIGHT AGE AT THREE DIFFERENT AGES

Patient no	Preschool (1.5–5 yr)	Pre-pubertal		Puberty (13–14 yr)
		9–10 yr	13–14 yr	
<i>Girls</i>				
11	7.5%	5%	..	62%*
4	15%	37%	..	85%*
7	8%	50%	..	50%*
3	5%	15%	..	82%*
2	15%	38%
<i>Boys</i>				
9	3.5%	17%
8	3.5%	10%	20%	..
6	<1%	10%	17%	..

Height age represents chronological age on the growth charts at which the child's actual height fell at the 50th percentile (growth charts from National Centre for Health Statistics, National Institutes for Health).

Weight for height age is the percentile at which the child's actual weight fell when plotted against the height age.

Girls and boys have been rank-ordered separately by severity of dysmorphism.

*Menarche attained.

of from less than the 1st to the 15th percentile during the preschool years. However, at puberty the four females had weight for height ages ranging from the 50th to the 85th percentile (eg, fig 2). Neither of the boys who were between 13 and 14 years of age had reached puberty, and both continued to be underweight for height (eg, fig 3). The characteristic emaciated appearance in the young child (see fig 4) may not remain a salient feature in the affected adolescent, at least for females. This may help explain why new patients are rarely recognised during adolescence and adulthood.

Pattern of Malformation

In the eight children for whom follow-up data are available, including the three who had undergone pubertal changes, the craniofacial features did not change over the 10 years, especially the short palpebral fissures, hypoplastic philtrum, thin vermilion border of the upper lip, and flat midface. However, the noses have taken on a new contour, with prominent growth of the nasal bridge, and subtle morphological changes in the nasal tip and/or ala nasi. The mandibles become relatively prognathic. Although the patients continue to resemble each other, the changes in the nose and mandible alter the overall facial phenotype substantially and may again explain why the syndrome tends not to be recognised in adolescents and adults.

Cardiac anomalies, which consisted of an atrial septal defect in one patient, patent ductus arteriosus in one patient, and grade 3/4 systolic murmur interpreted as representing a ventricular septal defect in six patients, have all resolved spontaneously or have become insignificant. Congenital hip dislocation has been managed successfully by the use of casts and/or abduction splints in two patients and by surgery in the third. Except for one case of severe scoliosis that had to be corrected by spinal laminectomy the other skeletal or joint anomalies, such as radioulnar synostosis, camptodactyly, and/or clinodactyly, have not had to be corrected by surgery and (with one exception) have not interfered with performance. The short palpebral fissures are thought to be secondary to decreased growth of the eyes. Frank microphthalmia observed at necropsy in patient 10 as well as optic nerve hypoplasia noted in two other children at their most recent follow-up visit tend to support this interpretation

TABLE IV—IQ AND PERFORMANCE IN RELATION TO SEVERITY OF DISORDER

Patient	FAS diagnosis		Age	IQ	
	1976	1983	1983	1973	1983
<i>Severely impaired</i>					
11*	Severe	Severe	14	30	20
9*	Severe	Severe	10	43	40
4*	Severe	Severe	14	47	48
7†	Severe	Mod/severe	14	60	57
<i>Mildly to moderately impaired</i>					
8‡	Mod/severe	Mod/severe	13	50	80
3‡	Moderate	..	(8)	66	(76)
6‡	Moderate	Moderate	13	68	81
2‡	..	Mod/severe	10	83	86
Mean	56	61

Severity of disorder 1976 based on severity of dysmorphic characteristics and growth deficiency but without knowledge of IQ scores.¹⁶ Patients nos 2 and 11 were not included in the 1976 follow-up but the earlier severity rating was made retrospectively from the records. Patient no 3 was last seen at age 14 but the last available IQ exam was at age 8. Patient 2, who had a hearing loss, was also given the Leiter international intelligence scale; on this test for deaf children she received an IQ score of 88.

*Indicates the patient was receiving education for the trainable mentally retarded; †by self-contained special education classes; ‡by regular and special teaching.

and are consistent with the work of Stromland.¹³ Exotropia and/or myopia were noted in four other patients.

Dysharmonic growth of the midface and mandible has led to relative prognathism with class III malocclusion in five children, while dental malalignments were seen in all patients. Chronic serous otitis media, probably secondary to eustachian tube dysfunction associated with maxillary hypoplasia, required medical and surgical procedures in four children, three of whom had had permanent conductive hearing loss.

The children have been in reasonably good health since infancy. Although immunological deficiency in some children with the fetal alcohol syndrome has been reported,¹⁴ none of the patients we followed up had a high frequency of chronic or recurrent infections. However, they were not assessed immunologically.

Performance

None of the eight children followed up had normal intellectual development (see table IV and fig 1); four were mildly and four seriously handicapped. The four mildly handicapped children had IQ scores in the borderline to dull-normal range, were taught in a combination of regular and remedial classes, and had an academic achievement that was at least commensurate with their intellectual functioning. Two of these four had repeated one grade and their performance was at or below grade level. The remaining two had a performance that was several grades below their placement and were having increasing difficulty with schoolwork. All four of these mildly handicapped children lead normal lives within their own social circle and seem independent, resourceful, and self-sufficient. The discrepancy between their independence and their intellectual skills may later cause social and academic problems and further follow-up seems warranted. Two of these four children, both of whom live with their natural families, already have social/emotional problems and attend school irregularly.

The four seriously handicapped children are mildly to moderately retarded intellectually. They are taught in classes for the mentally retarded. Two of them can read and write at a primary level while the other two can barely write their own names. Although all the seriously handicapped children were hyperactive when young, the three who have reached adolescence have lost their hyperactivity. All continue to have attentional deficits and are friendly, outgoing, socially engaging, and uninhibited. The youngest boy (patient 9) is difficult to control but the other three (patients 4, 7, and 11) are compliant and obedient. All the seriously handicapped children need constant surveillance and do not appropriately distinguish strangers from friends. The severity of the disorder, in terms of physical abnormalities, during the preschool years was highly predictive of the IQ scores at the last follow-up (table IV).

Environment

The degree to which postnatal environmental factors influenced the development of these children is difficult to assess. The first 2 years of life were difficult for seven of these children: six lived intermittently with natural mothers who continued to drink, or were in temporary custody; the seventh was in hospital. The child who was raised throughout life in a stable foster home is among the most retarded. The four most severely handicapped children (whose mothers were unable or unwilling to care for them) were brought up in conventional stable families for at least the past 5 years. Two of the four mildly handicapped children remain with their natural mothers, one is with his natural father, and one has been adopted. The effect of environment on intellectual development is thus not obvious in our sample, although better social and emotional development seemed to be related to stable home environments.

The single factor that stands out most in the backgrounds of the four seriously handicapped children (patients 4, 7, 9, and 11) is that three of them had mothers who were so severely alcoholic that they died of alcohol-related causes within 6 years of the birth of these children (table I). Child neglect has been documented at least once during the early years in five of the children followed up. Two seem to have been the victims of physical abuse (one in a natural home and one in a foster home) and one has been suspected of being abused sexually. For those who remained with their natural mothers, the first years in their alcoholic families were tumultuous. In addition, these children were often difficult to care for during their early years because of their failure to thrive, feeding difficulties, hyperactivity, and intrusive nature. On the other hand, of the three who were in state custody, one died in an apparent drowning accident in an adoptive home and another spent the first 1½ years in a hospital and then had four different foster homes in 7 years before joining a stable household at the age of 8.

CONCLUSIONS

Our 10-year follow-up of children with the fetal-alcohol syndrome whose mothers were severe chronic alcoholics has suggested two factors which may help in giving a prognosis for individuals with this disorder.

Of great predictive significance is the extent and severity of the pattern of malformation. Severity of the pattern of malformation and growth deficiency was correlated with degree of intellectual impairment in an earlier study,¹⁵ which included some of the patients described in this paper. In the present paper the four children with the most striking

craniofacial abnormalities (patients 4, 7, 9, and 11) have the most severe degree of microcephaly, the shortest stature, and the lowest intellectual function. Three of these four infants presented in a breech position, compared with none of the four moderately affected children.

The most predictive factor in the backgrounds of the four most severely handicapped children was the severity of maternal alcoholism. Three of them had mothers who died of alcohol-related causes less than 6 years after their births, which suggests that biological factors associated with the terminal stages of alcoholism may have contributed to the severe handicap. The quality of the later home environment did not counterbalance the severity of the prenatal insult. Two of the four severely affected children lived in the most stable foster homes, which suggests that the prognosis for the most severely affected children depends primarily on the extent and severity of the prenatal exposure to alcohol. However, improvement in social and emotional development was noted in patients whose home environments became stable, either because of reduction in maternal drinking, departure of the alcoholic mother, or placement of the child in a stable foster or adoptive home. The high frequency of abuse and neglect which characterised the first year of life for those who remained with alcoholic mothers who continued to drink indicates the need for intervention from birth.

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Round the World

From our Correspondents

United States

MALPRACTICE AGAIN

ON the day that several thousand physicians gathered from all over New York State to demand action over the medical malpractice crisis in the State, a new type of malpractice suit got underway in California. There, the plaintiffs are the parents of a young man who committed suicide in a state of depression, not having been cured by the ministers of the church of which he had become a member. On behalf of the pastors, it is stated that "the root of all depression is sin" and can be treated with advice based only on the Bible. Unhappily in this case the treatment was not successful, perhaps because the counselling of the church concerned reinforces feelings of guilt, which may already have reached a pathological degree. The outcome of the case, one in a growing number of such cases against churches and pastors, will be of interest.

If religious malpractice has not as yet reached a crisis, medical malpractice, at least in some States, has done so. Some ten years ago, most commercial insurance companies, faced with large increases both in the number of cases and in the size of awards, declined to give coverage. To cope with this crisis, the medical profession itself arranged mutual malpractice insurance coverage, and there are now some forty organisations, many of which face an uncertain future, especially since claims and costs have risen dramatically. In New York, the Medical Liability Insurance Company (MLIC), which insures about 35% of the doctors in the State, now finds itself short of over \$750 million to pay future claims, despite a 55% rise in premiums. But what is making the situation more difficult is that the reinsurance companies, through which the costs can in part be "laid off", are also leaving the field, some backing out completely, others refusing to back certain groups within the medical profession. Some new insurance groups are confining cover to those medical practitioners with the smallest risks. As those concerned draw graphs predicting the future they arrive at quite horrifying figures. Something can be done to ease matters by changing from "occurrence" policies to "claims made" policies, the latter covering only claims made in the year in which the policy is in force. But this is only a small step.

Governor Cuomo of New York has suggested various legislative proposals, but it is very doubtful whether either he or the members of the legislature have anything like a full grasp of the problems. A decision to limit attorneys' fees would effect only a small saving. A ceiling could be put on damages for pain and suffering, but would not this infringe our constitutional rights? Would it not mostly concern the more severely injured? Perhaps all large awards should be terminated with the death of the injured party.

The spokesmen for the medical profession seem to be no more aware of the real problems and their ramifications than the legislators, and perhaps a lot less. What is alarming is the unwillingness of many physicians, and their organisations, to come up with the real solutions. For the plain fact is that there is a lot of real malpractice—some would say a horrifying amount. Incompetence, carelessness, laziness, indifference, and frank inhumanity are all too common. Practice and prescribing over the telephone all too often lead to disaster. Most problems are due to a small minority of individuals, but the profession and the hospitals have done little to rid themselves of these, often repeat, offenders. For years the profession has been denounced for the slackness and uselessness of its medical disciplinary actions. If, at last, the American Medical Association has started a drive to force incompetent physicians out of practice it is about 40 years too late.

But this is only part of the trouble. There are the notorious delays in settling suits (now more than eight years in New York), and there is a tendency in bad cases to add interest from the time of injury. Whence these delays? It is clear that the MLIC must have an enormous number of outstanding claims if its reserves are \$115 billion and it is still \$750 million short. Why are indefensible claims