Archives of Disease in Childhood, 1981, **56**, 245–252

Hearing, speech, and language in survivors of severe perinatal asphyxia

S W D'SOUZA, ELSPETH McCARTNEY, M NOLAN, AND I G TAYLOR

Department of Child Health, and the Department of Audiology and Education of the Deaf, University of Manchester

SUMMARY Hearing, speech, and language were studied in 26 children who survived severe perinatal asphyxia. The results of hearing tests showed that most children had a favourable outcome. Only 1 child had sensorineural deafness. Hearing loss in 6 others was due to middle-ear disease which resolved after treatment, and on retesting was found to be normal. The study also showed that neither gentamicin treatment nor incubator noise seemed to affect hearing. The results of speech and language assessment were less encouraging and about one-third of the children without serious mental or physical handicap had deficits in speech and language. It is suggested that the quality of life in such children could be improved if these deficits were detected early and adequately treated.

The introduction of intensive care treatment to ill newborn babies has ensured that more of them survive. Among the many developments in this area has been skill in resuscitation and mechanical ventilation, but there remains concern that the quality of life in some survivors may not match the expectations of their parents. There is greater awareness that minor mental and physical disabilities may cause clumsiness, deviant behaviour, or learning difficulties. It is also apparent that in some children with deafness there is a history of various perinatal conditions—such as low birthweight, asphyxia, or jaundice.[1 2]

The prevalence of speech impairment and language retardation in schoolchildren has been reported,[3 4] but the relationship of these to perinatal conditions has not been clearly indicated. Environmental causes operating in later infancy and childhood may be responsible for some such cases but in others the origin is probably multifactorial.

A group of babies who had suffered severe perinatal asphyxia was followed up and we report the results of hearing tests and assessments of speech and language, and compare their neurological status in later childhood.

Methods

Subjects. Twenty-nine babies born at St Mary's Hospital, Manchester, between 1973 and 1976, had received intensive resuscitation for at least 10 minutes before spontaneous regular respiration could be established. The birth scores were 0, or 1, at one minute of age. Details of the mother's antenatal history and labour were obtained from the obstetric case notes. Gestational age, calculated from the first day of the last menstrual period, had been checked by reference to uterine size, fetal biparietal diameter, or bone age. The baby's condition at birth, details of resuscitation, and the subsequent progress in the neonatal period were obtained from the case notes. The clinical progress of each baby had been followed by regular examinations, and by the relevant laboratory or radiological investigations. Information about the number of days each baby had been nursed in an incubator and about gentamicin treatment was obtained from the case notes. After leaving hospital the babies were followed up at an outpatient clinic by one of us (S W D'S). Three babies were lost to the study because their parents had moved to another area and could not be traced. Of the remaining 26 babies 24 were singletons, and 2 were one of twins.

Measurement of incubator noise. The noise levels of 3 types of incubators in common use in the special care baby unit were measured with a Brüel and Kjaer sound-level meter.

Assessment of neurological status in later childhood. Neurological examinations were carried out according to the methods of Paine and Oppé[5] at

an outpatient clinic. Each child was given an overall rating on a 4-point scale according to the presence and severity of neurological abnormality and resulting physical handicap.[6 7] The four categories were:

Category 1 no abnormal neurological findings;
Category 2 neurological findings of slight or doubtful significance;
Category 3 definite disorder but producing little physical handicap or none;
Category 4 definite and obviously handicapping disorder.

Assessment of hearing. Hearing was assessed by the techniques and criteria developed by Ewing[8 9] and comprehensively described by Taylor.[10] Impedance bridge measurements were carried out to assess middle-ear function, by means of a Madsen ZO 70 electroacoustic impedance bridge attached to a Hewlett-Packard 7035B X-Y plotter. Fourteen children were assessed by pure-tone audiometry via a Peters AP5 diagnostic audiometer. Seven children were assessed by the distraction test of hearing.[10] One child was assessed by a co-operative test of hearing[10] and a back-up distraction test of hearing. Three children were assessed by the performance test of hearing.[10] The age of each child determined the type of hearing test used. Impedance bridge measurements were carried out on all children. The ranges of normal values of middle-ear pressure and gradient have been reported by Brooks,[11] and Jerger *et al.*[12] Reference was made to these ranges to determine whether a child had normal middle-ear function.

Assessment of speech and language. Two standardised scales were used: the Reynell Developmental Language Scales (Experimental version) (RDLS)[13] and the Edinburgh Articulation Test (EAT).[14] The RDLS has three sections—one measuring the structure of language, one measuring vocabulary, and one measuring language content. These combine to give a composite score. The EAT was included as a sensitive measure of articulatory difficulty which is often associated with neurological dysfunction.[15] It is suitable for children aged at least three years.

Children were tested using the RDLS Verbal Comprehension Scales, scale A or B as appropriate. Those under 3 years were assessed on the RDLS Expressive Language Scale too, and those over 3 years on the EAT. Case 12 was tested on the Expressive Language Scale which was thought more suitable for his overall level of functioning. Two children (Cases 4 and 22) were not available for testing.

The measures used are not standardised on immigrant children nor on the children of immigrant parents. However, in view of the lack of standardised measures of such children's linguistic abilities, the 2 immigrant children were assessed using the same measures as non-immigrant ones.

Table 1 *Antenatal conditions, intrapartum complications, and mode of delivery*

Case	*Antenatal conditions*	*Intrapartum complications*	*Mode of delivery*
1	Nil	Nil	Breech
2	Nil	Nil	Breech
3	Prolonged (96-hour) rupture of membranes. Vaginal discharge	Fetal distress	Caesarean section
4	Nil	Fetal distress	Caesarean section
5	Raised rhesus antibody titres	Nil	Normal cephalic vaginal
6	39-year-old primipara	Fetal distress	Normal cephalic vaginal
7	Nil	Fetal distress	Caesarean section
8	Twins. Urinary infection	Nil	Breech
9	Nil	Transverse arrest of fetal head	Forceps
10	Antepartum haemorrhage. Raised rhesus antibody titres	Nil	Normal cephalic vaginal
11	Nil	Fetal distress	Caesarean section
12	Diabetes. Hypertension. Antepartum haemorrhage	Nil	Breech
13	Sepsis after membrane rupture at 31 weeks	Nil	Normal cephalic vaginal
14	Nil	Transverse arrest of fetal head	Caesarean section
15	Epilepsy	Nil	Breech
16	Toxaemia	Nil	Breech
17	Hydramnios	Nil	Normal cephalic vaginal
18	Antepartum haemorrhage	Fetal distress	Forceps
19	Nil	Shoulder presentation	Caesarean section
20	Eclamptic fits	Nil	Normal cephalic vaginal
21	Twins. Toxaemia	Failure to progress	Forceps
22	Antepartum haemorrhage	Fetal distress	Forceps
23	Nil	Nil	Normal cephalic vaginal
24	Toxaemia	Fetal distress	Forceps
25	Nil	Failure to progress	Forceps
26	Nil	Transverse arrest of fetal head	Forceps

Results

The babies were born at a gestational age of 32 to 42 weeks, weighing 1420 to 3880 g; 11 (42%) babies were born prematurely (<37 weeks), and the remaining 15 (58%) were born at term (37 to 42 weeks). Using the data of Milner and Richards,[16] 5 (19%) babies were small-for-dates (birthweight <10th centile for gestational age) and the remaining 21 were appropriate-for-gestational-age.

Antenatal conditions, intrapartum complications, and mode of delivery. The antenatal conditions, intrapartum complications, and mode of delivery are shown in Table 1. A history of antenatal conditions was noted in 15 (58%), intrapartum complications in 14 (54%), and abnormal deliveries in 19 (73%) of 26 mothers.

Condition at birth. The condition at birth had been assessed by a paediatrician present at delivery in 19 babies, and in the remaining 7 by an obstetrician or a midwife. All babies were apnoeic at birth, and showed no signs of recovery when mucus was extracted from the mouth or nasal passages. They were therefore, resuscitated by administering intermittent positive pressure ventilation via an endotracheal tube. In 9 babies 5–15 ml of 8·4% (mass/vol) dextrose was administered through an umbilical venous catheter before the baby was transferred to the special care baby unit. In 2 babies spontaneous regular respiration was established at 12 and 14 minutes; in 17 babies spontaneous regular respiration was established between 15 and 30 minutes after birth; and the remaining 7 babies took longer than 30 minutes to establish spontaneous regular respiration. These 7 babies were ventilated until between 35 minutes and 24 hours of age.

Two babies (Cases 11 and 24; Table 2) had no vital signs at birth. After receiving external cardiac massage the heart beat in each was audible by age 5 minutes.

Clinical conditions in the neonatal period. The clinical conditions in the neonatal period are summarised in

Table 2 *Clinical conditions in the neonatal period*

Case	*Sex*	*Gestational age (weeks)*	*Birthweight (g)*	*Onset of spontaneous regular respiration (min)*	*Clinical conditions*
1	M	32	2340	>30	Hypocalcaemia. Hypothermia. Meningitis. Maximum serum bilirubin 170 μmol/l
2	F	33	1420	15	Respiratory distress. Apnoea. Umbilical hernia. Maximum serum bilirubin 162 μmol/l
3	M	35	2400	25	Pneumonia. Septicaemia. Maximum serum bilirubin 178 μmol/l
4	M	38	2920	12	Convulsions. Inguinal hernia. Maximum serum bilirubin 182 μmol/l
5	F	37	3000	20	Rhesus haemolytic disease. Exchange transfusions. Maximum serum bilirubin 156 μmol/l
6	F	40	2540	10	Convulsions. Congenital absence of right hand. Maximum serum bilirubin 95 μmol/l
7	M	33	2500	15	Respiratory distress. Pneumonia. Maximum serum bilirubin 202 μmol/l
8	F	38	1600	15	Twin 2. Convulsions. Maximum serum bilirubin 212 μmol/l
9	F	40	3480	15	Respiratory distress. Apnoea. Thrombocytopenia. Hypocalcaemia. Maximum serum bilirubin 116 μmol/l
10	F	33	1700	>30	Hypothermia. Exchange transfusion for hyperbilirubinaemia. Maximum serum bilirubin 320 μmol/l
11	M	42	3880	23	Convulsions. Apnoea. Maximum serum bilirubin 194 μmol/l
12	M	34	2760	10	Respiratory distress. Convulsions. Hypoglycaemia. Maximum serum bilirubin 274 μmol/l
13	M	31	1290	>30	Respiratory distress. Apnoea. Septicaemia. Maximum serum bilirubin 186 μmol/l
14	F	38	3200	>30	Pneumonia. Hypothermia. Maximum serum bilirubin 138 μmol/l
15	M	38	2600	15	Respiratory distress. Maximum serum bilirubin 258 μmol/l
16	M	39	2600	15	Convulsions. Apnoea. Maximum serum bilirubin 178 μmol/l
17	F	42	3290	20	Deviant neurological signs. Maximum serum bilirubin 122 μmol/l
18	M	37	3050	>30	Convulsions. Hypothermia. Cephalhaematoma. Maximum serum bilirubin 136 μmol/l
19	M	34	2610	18	Respiratory distress. Patent ductus arteriosus. Maximum serum bilirubin 128 μmol/l
20	M	32	1930	>30	Convulsions. Respiratory distress. Maximum serum bilirubin 133 μmol/l
21	M	37	2830	20	Twin 2. Deviant neurological signs. Cephalhaematoma. Maximum serum bilirubin 236 μmol/l
22	M	33	2750	15	Deviant neurological signs. Maximum serum bilirubin 196 μmol/l
23	M	42	3450	15	Respiratory distress. Hypothermia. Maximum serum bilirubin 198 μmol/l
24	M	40	2840	>30	Deviant neurological signs. Maximum serum bilirubin 117 μmol/l
25	F	34	1990	15	Respiratory distress. Apnoea. Maximum serum bilirubin 240 μmol/l
26	M	37	3720	20	Convulsions. Maximum serum bilirubin 160 μmol/l

Conversion: SI to traditional units—bilirubin: 1 μmol/l ≈ 0·058 mg/100 ml.

Table 2. The maximum serum bilirubin levels ranged from 95 to 320 μmol/l (5·5 to 18·7 mg/100 ml), the mean ± SE being 180 ± 10 μmol/l.

Nursing in incubators and gentamicin treatment. All 26 babies had been nursed in incubators in the special care baby unit, and most of them had received penicillin and gentamicin (Table 3). The duration of nursing in an incubator ranged from 2 to 27 days, the mean (± SE) duration in an incubator being 8·8 ± 1·1 days. A combination of gentamicin and penicillin had been administered to 15 (58%) babies for a period of 5 to 11 days; the mean (± SE) duration of antibiotic treatment was 7·8 ± 0·5 days.

Incubator noise levels. Three types of incubators in use in the special care baby unit were studied. None of the incubators showed C weighted levels exceeding 67 dB in the normal running condition. The C weighting on a sound level meter shows a uniform response from 20 to 20 000 Hz and is the appropriate range of frequencies to include responses at low frequencies as well as high. Investigation of the noise showed a maximum at a frequency of 90 Hz. The sounding of incubator alarms raised the measurement to 78 dB C, inside the incubator at 2 kHz.

Neurological findings in later childhood. After their discharge from hospital all 26 babies were followed up for between 2 and 5 years. The neurological findings are shown in Table 4. Sixteen babies had no neurological abnormalities at follow-up. Six had slight, or doubtful, abnormalities—such as squints, breathholding attacks, hyperactive behaviour, or a head circumference >97th centile[17] between 1 month and 10 months postnatally. Two children showed clear abnormalities but no handicap, one child had increased extensor tone in the lower limbs and walked clumsily, and one child had sensorineural deafness. These 2 children had been making average progress in normal schools. Two children were severely handicapped: one had microcephaly, spastic quadriplegia, severe mental retardation, and epilepsy. Computerised tomography showed dilated brain ventricles and cortical atrophy. The other child had spastic quadriparesis and athetosis; he has been attending a special school.

Table 3 *Duration of nursing in an incubator and duration of gentamicin treatment*

Case	*Duration (days)*	
	In an incubator	*Treatment with gentamicin*
1	6	11
2	17	7
3	8	11
4	7	Nil
5	2	Nil
6	4	9
7	7	11
8	8	Nil
9	7	Nil
10	17	7
11	7	Nil
12	17	Nil
13	8	7
14	8	7
15	8	10
16	4	Nil
17	2	Nil
18	11	Nil
19	11	10
20	5	6
21	7	Nil
22	13	Nil
23	8	5
24	4	5
25	6	5
26	27	7

Hearing assessment. On initial assessment 19 of the children were found to have normal hearing. Six (Cases 5, 7, 8, 10, 22, and 23) were found to have mild depressions in hearing together with abnormal middle-ear function as shown by impedance bridge testing. One child (Case 24) had a pronounced depression in hearing with normal middle-ear function. His audiogram showed a bilateral symmetrical sensorineural loss of hearing (Figure). Hearing was found to be normal up to 500 Hz in both ears with a subsequent loss in the higher frequencies of a sensorineural type. He has been reviewed over a period of 18 months and his audiograms have been consistent.

The 6 children showing a mild depression in hearing with abnormal middle-ear function were followed up after the initial assessment. Two

Table 4 *Neurological findings in later (2–5 years) childhood*

	Total patients (n=26)		*Cases*
	No	*%*	
No abnormal neurological findings	16	61	1,2,5,6,7,9,10,13,15, 16,17,18,19,20,26
Neurological findings of slight or doubtful significance	6	23	3,12,14,21,22,23
Definite disorder but producing little physical handicap, or none	2	8	8*,24†
Definite and obviously handicapping disorder	2	8	4‡,11§

*Increased extensor tone in the lower limbs and clumsy gait.
†Sensorineural deafness.
‡Microcephaly, spastic quadriplegia, severe mental retardation, and epilepsy.
§Spastic quadriparesis and athetosis.

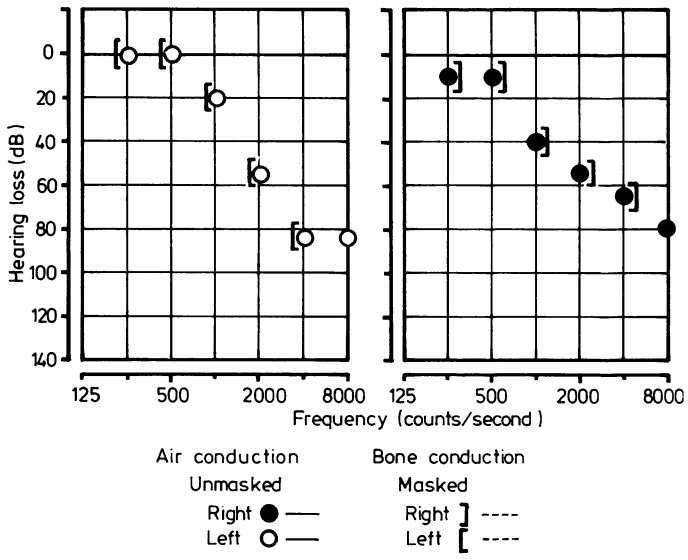

Figure (*Case 24.*) *Audiogram of the child with bilateral sensorineural deafness.*

(Cases 8 and 10) were reviewed 2 months later and were found to have normal hearing with normal middle-ear function. These children were subsequently retested 6 months later and were found to be hearing normally. They were therefore, suffering from a minor middle-ear problem at the time of initial assessment which subsequently cleared without treatment.

Two others (Cases 5 and 22) were prescribed a course of decongestants after the initial assessment. These children returned after the treatment and were found to have normal hearing with normal middle-ear function. Hearing was tested again 6 months later and was found to be normal.

The remaining 2 (Cases 7 and 23) children were also prescribed a course of decongestants but showed no improvement in hearing or middle-ear function. Arrangements were therefore, made for surgical intervention to clear the serous otitis media. In view of the results of the tests of hearing and of middle-ear function, it was concluded that no sensorineural element was responsible for the hearing loss. Thus, only one child had sensorineural hearing loss. In 6 others deafness was due to middle-ear dysfunction.

Table 5 *Details of the language-delayed children*

Case	*Age at test‡*	*Reynell Developmental Language Scales*				*EAT score†*	*Comments*
		Verbal Comprehension		*Expressive language*			
		Score†	*Age level‡*	*Score†*	*Age level‡*		
1	2·1	−1·2	1·7	−1·9	1·6		Expressive language delay—no known reason
3*	2·2	−2·1	1·6	−2·3	1·4/5		Delayed language development associated with arrested hydrocephalus
4	See text						Gross physical handicap—no speech
7	2·3	−2·2	1·5	−1·2	1·10		Poor verbal comprehension: hearing depressed on date of test
11	3·0	0·8 (scale B)	3·5	BN			Cerebral palsy: no speech due to gross physical handicap
12*	5·1	BN	3·1	−2·9	2·11		Language delay in association with delayed mental development
15	1·10	−1·1	1·6	−1·4	1·3		Expressive language delay—no known reason
23	4·2	−0·4	4·0			6	Arrested hydrocephalus. Markedly defective speech, unintelligible to therapist. Conductive hearing loss. Hearing depressed on date of test
24	4·9	−1·8	3·9			72	Deviant fricatives, with sensorineural hearing loss. Verbal comprehension depressed
26	4·3	1·1	5·0/2			72	Dental/s/ sound, and reduced consonant clusters

BN = below norms—that is, <−3 SD.
*Immigrant child, †standard score, ‡in years and months.
RDLS standard score (mean ± SD) = 0·0 ± 1, EAT standard score (mean ± SD) = 100 ± 15.

Assessment of speech and language. On the RDLS Verbal Comprehension and Expressive Language scales, a standard score of −1·3 was taken to be the lower limit of the normal range. This eliminated scores below the 10th centile. The EAT test manual directs that a less stringent criterion be adopted, and that scores below 1 standard deviation from the mean—that is, standard scores below 85—be considered outside normal limits.

Using these criteria, 15 of the 24 children fell within normal limits on all tests. Scores of the remaining 9 children are shown in Table 5, together with comments on the nature of their difficulty.

Two children were not available for testing. One (Case 4) is a severely handicapped child with no speech. Although his parents bring him to an outpatient clinic, they did not show interest in having him participate in formal speech and language assessment. The other child (Case 22) is an Egyptian who had left with his parents for Egypt. He was able to converse in English and in the Egyptian language. At age $3\frac{1}{4}$ years he could recite nursery rhymes, and count to 10, in English.

Five (Cases 1, 3, 7, 12, and 15) of the 24 children tested had delayed language development, 3 (Cases 23, 24, and 26) had speech defects, and 1 (Case 11) child had no speech due to gross physical handicap.

Discussion

Estimates of hearing loss in Britain vary from 1·4 to 49 per 1000 children.[1] In this study, the incidence of sensorineural deafness was 1 in 26, and in a previous study[7] it was 1 in 29 cases. Thus, the incidence of sensorineural deafness in survivors after severe perinatal asphyxia is 4%. The patient reported by Thomson *et al.*[7] had bilateral deafness with a 50 dB loss from 400 to 8000 Hz. She had made average progress in a normal school without the use of a hearing aid. Our patient had bilateral hearing loss at frequencies greater than 500 Hz (Figure). He has been fitted with a hearing aid and is receiving a normal education.

The possibility that perinatal asphyxia causes sensorineural deafness finds some support from observations made at necropsy. Hall[18] reported lesions in the dorsal and ventral cochlear nuclei and in the cochlea. The babies we studied were probably in terminal apnoea at birth, and assuming that the effects of asphyxia after birth were minimised by skilful resuscitation, subsequent neurological sequelae—including sensorineural deafness—may be related to the severity of intrapartum asphyxia. Thus, of the 2 babies with an absent heart beat and without any other vital signs at birth, one (Case 24) had sensorineural deafness in later childhood, and the other (Case 11) had spastic quadriparesis and athetosis. Brain damage in each was associated with acute circulatory failure. A patient with sensorineural deafness and a history of perinatal cardiac arrest has been reported.[19]

It is unlikely that sensorineural deafness in Case 24 is due to bilirubin toxicity. The maximum serum bilirubin level was 194 μmol/l (11·3 mg/100 ml) in the neonatal period. This level is lower than in most of the other children. We are also aware that in clinical practice term babies can tolerate circulating levels of bilirubin greater than 194 μmol/l without showing signs of acute toxicity.

We examined the frequency of sensorineural deafness in gentamicin-treated patients, and in cases that had not received treatment, but there was no appreciable difference. The drug had been administered in accordance with the regimen described by Milner *et al.*,[20] 3 mg/kg intramuscularly initially followed by 2 mg/kg 8 hourly, but serum gentamicin levels had not been measured. Milner *et al.*[20] reported a mean (± SE) gentamicin level of 5·0 ± 0·5 μg/ml 1 hour after the first injection, 1·9 ± 4 μg/ml just before the second injection, and on the third day 1·9 ± 0·3 μg/ml just before an injection, and 5·1 ± 0·5 μg/ml 1 hour after injection. In one study, on the long-term effects of gentamicin administered to newborn babies, Elfving *et al.*[21] followed up 28 babies for 2–$3\frac{3}{4}$ years. Two children had vestibular dysfunction, but because of the complicated clinical histories it is difficult to evaluate the cause and effect relationship.

Serological tests for rubella had not been carried out in early infancy since there was no history of maternal rubella during pregnancy, or clinical evidence of congenital rubella in any of our patients. In the one child with sensorineural deafness urine had been cultured for rubella virus after the results of audiometric testing were known, but no viruses were cultured and the serological test was negative.

We investigated the possibility of familial deafness in the patient with sensorineural deafness. Information obtained from parents, grandparents, and 1st-degree relatives did not point to any familial reason for the deafness.

There is evidence from experiments on animals that incubator noise has a damaging effect on the sensory cells in the cochlea[22] and it has been suggested that incubator noise may to some extent determine the higher incidence of deafness in children of low birthweights. The highest noise level in the incubators we studied, 78 dB C, is comparable to that reported by Douek *et al.*[22] All 26 babies in our study had been nursed in incubators but we found no

significant relationship between hearing loss and duration in the incubator in premature or term babies.

Any decision as to the limits of normality in speech and language must in the end be an arbitrary one. Various workers have selected different cut-off points to suit their own purposes. Randall *et al.*,[23] in a study of language development in 3-year-old children in Barnet, used a standard score of −2·0 or less to define a group of severely language-retarded children. Only 2 such (non-immigrant) children were found in their sample of 160 children. Since only 2·5% of a normally distributed population could occur below a standard score of −2·0 the incidence reported by Randall *et al.*[23] is not surprising.

The 10th centile has also been used as the point separating 'normal' from 'deviant' functioning, as in a study of Buckinghamshire schoolchildren by Shepherd *et al.*[24] We accepted this cut-off point in view of the small numbers in our study in order to discuss the types of language difficulties found. This applies to the RDLS Verbal Comprehension and Expressive Language scales; the EAT has its own internally defined cut-off point.

As regards the causes of speech and language deficits, Cases 3 and 12 were children of immigrant parents. This means that the scores may be of doubtful significance for them since immigrant children were excluded from the standardisation sample of the RDLS, as it was thought that their language development would probably be atypical for an English standardisation. In the Randall *et al.*[23] study many such children scored poorly on the RDLS, especially if English was not spoken at home. There are no norms for test results on non-standard English speakers. Case 12 also had delayed motor development and was hyperkinetic. He had been seen by an educational psychologist at age 4 years, and it was reported that a score of 26 was obtained on the Merrill Palmer Scale of Mental Tests. This places him at the 1st centile—that is, in the subnormal range of ability.

It is possible to implicate hearing losses in 3 children with speech and language deficits, and in a further 2 children such deficits were due to severe mental and physical disabilities. Cases 7 and 24 had hearing losses which might account for their language difficulties. Case 23 with articulation difficulty had long-standing middle-ear disease but he also appears to have hydrocephalus; his head circumference had been >97th centile between 1 and 10 months postnatally. The problem with his speech is on the output side (comprehension is within normal limits) but both the above factors could be important, interacting to give poor control of movements allied to reduced auditory experience. Cases 4 and 11 had severe degrees of physical and mental handicap, sufficient to account for lack of speech.

In the remaining 3 cases there is no obvious reason for speech and language deficits. Case 26 had a dental /s/ sound and reduced consonant clusters. These defects fell into the 'immature' or 'almost mature' categories, which may be part of a normal developmental process and could be expected to disappear in time. The remaining 2 (Cases 1 and 15) children were outside the defined normal limit on the Expressive Language scale only, but there was no obvious reason for this.

Most children surviving severe perinatal asphyxia do so without severe physical or mental handicaps,[7 25 26] but we have also shown that about one-third of those surviving without handicaps had deficits in speech and language. Such deficits may not be apparent to parents, or to those seeing children regularly at local welfare clinics. The quality of life in such children might be improved if speech and language deficits were detected at an early age and adequately treated.

References

1 Dinnage R. *The handicapped child. Research review.* Vol. 2. *Visual impairment, hearing impairment, speech disorders, and other physical handicaps.* London: Longman, 1972.

2 Fraser G R. *The causes of profound deafness in childhood.* Baltimore: Johns Hopkins University Press, 1976.

3 Butler N R, Peckham C, Sheridan M. Speech defects in children aged 7 years: a national study. *Br Med J* 1973; **i:** 253–7.

4 Sheridan M D, Peckham C. Follow-up at 11 years of children who had marked speech defects at 7 years. *Child Care Health Dev* 1975; **1:** 157–66.

5 Paine R S, Oppé T E. *Neurological examination of children.* Clinics in Developmental Medicine No 20–21. London: Heinemann, 1966.

6 Rutter M, Graham P, Yule W. *A neuropsychiatric study in childhood.* Clinics in Development Medicine No 35–36. London: Heinemann, 1970.

7 Thomson A J, Searle M, Russell G. Quality of survival after severe birth asphyxia. *Arch Dis Child* 1977; **52:** 620–6.

8 Ewing A W G. *Educational guidance and the deaf child.* Manchester: Manchester University Press, 1957.

9 Ewing A W G, Ewing I R. *New opportunities for deaf children.* Manchester: Manchester University Press, 1958.

10 Taylor I G. *The neurological mechanisms of hearing and speech in children.* Manchester: Manchester University Press, 1964.

11 Brooks D N. The use of the electroacoustic impedance bridge in the assessment of middle ear function. *Int Audiology* 1969; **8:** 563–9.

12 Jerger J, Jerger S, Maudlin L. Studies in impedance audiometry. I. Normal and sensori-neural ears. *Arch Otolaryngol* 1972; **96:** 513–23.

13 Reynell J. *Reynell developmental language scales,* experimental edition. Windsor, Bucks: NFER Publishing Company, 1969.

[14] Anthony A, Bogle D, Ingram T T S, McIsaac M W. *The Edinburgh articulation test.* Edinburgh: Livingstone, 1971.

[15] Milisen R. The incidence of speech disorders. In: Travis L E, ed. *Handbook of speech pathology and audiology.* New York: Appleton-Century-Crofts, 1971: 624–33.

[16] Milner R D G, Richards B. An analysis of birthweight by gestational age of infants born in England and Wales: 1967 to 1971. *J Obstet Gynaecol Br Commonw* 1974; **81:** 956–67.

[17] Westropp C K, Barber C R. Growth of the skull in young children. I. Standards of head circumference. *J Neurol Neurosurg Psychiatry* 1956; **19:** 52–4.

[18] Hall J G. The cochlea and the cochlear nuclei in neonatal asphyxia. *Acta Otolaryngol [Suppl] (Stockh)* 1964; **194:** 1–93.

[19] Steiner H, Neligan G. Perinatal cardiac arrest. Quality of the survivors. *Arch Dis Child* 1975; **50:** 696–702.

[20] Milner R D G, Ross J, Froud D J R, Davis J A. Clinical pharmacology of gentamicin in the newborn infant. *Arch Dis Child* 1972; **47:** 927–32.

[21] Elfving J, Pettay O, Raivio M. A follow-up study on the cochlear, vestibular, and renal function in children treated with gentamicin in the newborn period. *Chemotherapy* 1973; **18:** 141–53.

[22] Douek E, Dodson H C, Bannister L H, Ashcroft P, Humphries K N. Effects of incubator noise on the cochlea of the newborn. *Lancet* 1976; **ii:** 1110–3.

[23] Randall D, Reynell J, Curwen M. A study of language development in a sample of 3-year-old children. *Br J Disord Commun* 1974; **9:** 3–16.

[24] Shepherd M, Oppenheim B, Mitchell S. *Childhood behaviour and mental health.* London: University of London Press, 1971.

[25] Scott H. Outcome of very severe birth asphyxia. *Arch Dis Child* 1976; **51:** 712–6.

[26] D'Souza S W, Richards B. Neurological sequelae in newborn babies after perinatal asphyxia. *Arch Dis Child* 1978; **53:** 564–9.

Correspondence to Dr S W D'Souza, Department of Child Health, St Mary's Hospital, Hathersage Road, Manchester M13 0JH.

Received 12 February 1980