Archives of Diseases in Childhood, 1979, **54**, 421–426

Hearing loss in very low birthweight infants treated with neonatal intensive care

SOLOMON J. ABRAMOVICH, SHEILA GREGORY, MARGARET SLEMICK, AND ANN STEWART

Departments of Obstetrics and Paediatrics, and Royal Ear Hospital, University College Hospital and Medical School, London

SUMMARY The hearing of 111 perinatal intensive care survivors of birthweights 1500 g or less was assessed at a mean age of 6½ years (range 4–12). These 111 infants included 86% of the long-term survivors of this birthweight cared for in the newborn unit of University College Hospital, London, during the years 1966–72. All these infants were nursed in commercially available incubators for periods ranging from 2 to 80 days (mean 37) in which the mean noise threshold was 65 dB. Ten (9%) had sensory neural hearing losses, one (1%) infant had a congenital conductive hearing loss, and 21 (19%) infants had exudative otitis media with a mean loss of 25 dB. Apnoeic attacks in the neonatal period were the most significant predictors of hearing loss in these infants ($P<0.05$) and an indirect serum bilirubin level of at least 170 μmol/l (10 mg/100 ml) in the neonatal period had an additive effect ($P<0.05$). There was no evidence that ambient noise had affected the hearing of these very low birthweight infants.

Reports indicate that the incidence of sensory-neural hearing loss among very low birthweight infants is high (4 to 15·9%) (Campanelli *et al.*, 1958; McDonald, 1964). Douek *et al.* (1976) suggested that this might be due to damage of the cochlea by ambient noise from the incubators in which these infants are usually nursed. To support this suggestion they quoted a study by Campanelli *et al.* (1958) which showed that the mean length of stay in incubators for low birthweight infants with sensory neural loss was significantly longer than the mean for low birthweight infants without hearing loss. There were, however, other important differences between the two groups. In particular, the mean birthweight in children with sensory neural hearing loss was only 981 g, whereas the mean for the children without hearing loss was much greater—1718 g ($P<0.05$).

The incidence of potentially lethal or damaging perinatal complications increases as birthweight and gestation decrease. Thus, the infants in the study of Campanelli *et al.* (1958) who had sensory neural hearing loss are much more likely to have suffered brain damaging complications than the heavier infants without hearing loss. Perinatal complications are also likely to influence the length of incubator stay. Thus it is important to investigate the potential role of all these factors in the aetiology of sensory neural hearing loss before attempting to apportion blame.

Ear, Nose and Throat Department, King's College Hospital, London
SOLOMON J. ABRAMOVICH, senior registrar
Department of Obstetrics and Paediatrics, University College Hospital Medical School, London
ANN STEWART, senior lecturer in perinatal medicine
Barnet Educational Psychology Service, London
SHEILA GREGORY, research/educational psychologist
MARGARET SLEMICK, retired audiometrician

Population

During the years 1966–72, 273 infants of birthweights 1500 g or less were admitted to University College Hospital (UCH) for neonatal intensive care. 164 were born in UCH, 99 were referred from other hospitals, and 10 were admitted from home where they had been born unexpectedly. 132 (48%) of these infants survived. 129 of the surviving infants were followed up for more than 5 years, 2 were lost to follow-up, and one child died after a domestic accident at age 4 years.

Methods

The management of the infants during the perinatal period and at follow-up after leaving hospital has been described (Stewart and Reynolds, 1974;

Stewart *et al.*, 1977; Turcan *et al.*, 1977). Methods included a high standard of obstetric care, prompt resuscitation at birth and subsequently, prevention if at all possible of hypoxia, hypothermia, hypoglycaemia, hyperbilirubinaemia, and infection; and the provision of warmth and adequate nutrition.

All the children were nursed in standard, commercially available incubators until their weights were >1500 g. Noise levels in these incubators under normal working conditions in the neonatal unit were tested using a Dawe octave band sound level meter. The noise levels in 9 incubators of 4 types which had been in use in UCH for at least 10 years, including the years that the children being studied were born, varied from 58 to 72 dB on all weightings of the octave band sound level meter. The mean threshold was 65 dB sound pressure level. The mean threshold on A weighting was 62 dB, on B weighting 65 dB, on C weighting 67 dB, and D 66 dB. The noise level outside the incubators was 65–75 dB sound pressure level on all weightings.

After leaving hospital the children were seen regularly for clinical and neurological assessments, including otoscopic examination and developmental testing. Psychometric measurements were made at 3½, 5, and 8 years; these included educational and attainment tests, and assessment of motor skills for the older children. Hearing responses were elicited in the course of developmental testing, and children in whom satisfactory responses could not be obtained by age 9 months were referred for detailed testing, using 'distraction' techniques and objective hearing testing, using the posterior auricular muscle (PAM) response method (Fraser *et al.*, 1978), in the audiological unit of the Royal Ear Hospital, UCH.

Pure tone audiometry within the speech frequencies (250–8000 Hz) was tested on all children who were able to co-operate sufficiently to give a reliable response at ages ranging from 4 to 12 years. The function of pure tone audiometry was to compare the hearing of the child with accepted hearing levels for normal subjects (International Standards Organisation). Thus, a mean loss of >20 dB in the speech frequencies was considered to be abnormal. Among children unable to co-operate, usually because of other serious handicaps—such as mental retardation—the hearing threshold was evaluated using free field audiometry, the PAM response method (Fraser *et al.*, 1978), and transtympanic electrocochleography (ECOG) (Beagley *et al.*, 1974).

Results

The hearing threshold was elicited in 111 of the 129 children. Pure tone audiograms were obtained from 107 children at a mean age of 6½ years (range 4–12).

In one very retarded child the threshold was obtained with pure tone audiometry in free field, and 3 children were assessed with PAM or ECOG. No audiogram was obtained from 18 children, comprising 14 who were living abroad and 4 whose parents did not co-operate. All these 18 children had developed normal speech and were considered by their local medical practitioners, teachers, and parents to have normal hearing. In the group of 111 children in whom the hearing threshold was elicited, one child had congenital conductive hearing loss, 21 (19%) had exudative otitis media with mean hearing loss of 26 dB, and 10 (9%) had sensory neural hearing loss. Of these 10 children, 8 had bilateral losses in the speech frequencies (250–8000 Hz) which varied from 35–80 dB (mean 51). Four of the 8 were seriously handicapped by their hearing loss and required special education. The other 4 were able to compensate for the loss with hearing aids and remedial help, and attended normal schools. Two other children had unilateral losses, including one with a loss of 70 dB at 8000 Hz only, but both these children had hearing which was adequate to perceive speech without difficulty.

The distributions of birthweight, period of gestation, sex, single or multiple pregnancy, place of birth, or method of delivery were the same for children with or without sensory neural hearing loss (Tables 1 and 2). Records for length of stay in the incubator were only available for the 56 infants, including 7 with sensory neural hearing loss, who were born in the years 1968–69 and 1971–72. (Records

Table 1 *Perinatal characteristics of 10 children with and of 101 children without sensory neural hearing loss, birthweights≤1500 g*

	Sensory neural hearing loss	
	Present (n=10)	*Absent (n=101)*
Boys	5	43
Girls	5	58
Singletons	5	79
Multiple births	5	22
Born in UCH	5	68
Referred	5	33
Birthweight		
Appropriate for gestational age	9	76
Small for gestational age	1	25
Method of delivery		
Vertex	5	42
Forceps	3	20
Breech	2	19
Caesarean section	0	20

for the other years were destroyed in a flood during the preparation of these data.) There was no significant difference between the length of stay for the children with or without sensory neural hearing loss among the 56 children ($P<0\cdot1$) (Table 2).

The incidences of perinatal complications in the children with or without sensory neural hearing loss are shown in Table 3. Two complications—apnoeic spells in the neonatal period that were sufficiently severe to require treatment with mechanical ventilation, and a serum bilirubin level of at least 170 μmol/l in the neonatal period were present more often in children with sensory neural hearing loss than in those without. However, the difference was only significant in the children who had apnoeic spells ($P<0\cdot025$). The mean maximum serum bilirubin level among the 6 jaundiced children with sensory neural hearing loss was greater than the mean among the 28 jaundiced children without hearing loss, but this difference was also not significant ($P<0\cdot2$).

Only one (10%) of the 10 children with sensory neural hearing loss had cerebral palsy. This incidence did not differ significantly from that for the 101 (4%) children without hearing loss.

All the children in the study had a psychological assessment at $3\frac{1}{2}$ years. To accommodate the varying skills of such children, one of three different tests was chosen. It was, therefore, not possible to calculate mean scores, but the distribution of the SD grouping was significantly lower in the 10 children with sensory neural hearing loss than in those without ($P<0\cdot05$). 55 of the children were aged at least 8 years and were assessed on the (WISC) IQ scale. There were highly significant differences between the means of the performance, verbal, and full-scale scores in this test for the 6 children with sensory neural hearing loss and for the 49 without such loss ($P<0\cdot001$) (Table 2). This indicated an overall depressed level of functioning for the children with sensory neural hearing loss and suggested that these children may have suffered additional cerebral

Table 2 *Neonatal and developmental characteristics of 10 children with and of 101 children without sensory neural hearing loss, birthweights ⩽1500 g*

	Sensory neural hearing loss		*Significance of difference*
	Present (n=10)	*Absent (n=101)*	
Mean birthweight (g)	1199	1266	NS
Mean gestation (weeks)	29·8	30·8	NS
Mean pH within 2 hours of birth	7·20	7·24 (n=74)	NS
Mean serum bilirubin level for infants with a value >170 μmol/l	272	238 (n=29)	NS
Mean duration of incubator care (days) (1968–69 and 1971–72 only)	45·4 (n=7)	36 (n=49)	NS
Mean full-scale IQ(WISC)	75± 8·7 (n=6)	97±19·6 (n=49)	P<0·001
Verbal scale	74± 7·8 (n=6)	98±16 (n=46)	P<0·001
Performance scale	80±13·2 (n=6)	101±12·6 (n=46)	P<0·001

Table 3 *Neonatal illnesses and drug treatment in 10 children with and 101 children without sensory neural hearing loss, birthweights ⩽1500 g*

	Sensory neural hearing loss		*Significance of difference*
	Present (n=10)	*Absent (n=101)*	
Intrapartum hypoxia			
Heart rate <100 at birth	2	27	NS
pH <7·2 (within 2 h)	3(n=5)	28(n=74)	NS
Negative base excess >15 mmol/l (within 2 h)	0(n=5)	6(n=75)	NS
Neonatal illness			
Hyaline membrane disease	4	35	NS
Apnoea needing intubation ± ventilation	6	22	P<0·025
Bilirubin >170 μmol/l	6	29	P<0·1
'Dextrostix' <25	1	14	NS
Infections			
Rubella, CMV (Intrauterine)	1 0	2 0	NS
Meningitis, Septicaemia (Postpartum)	1* 0	1 1	NS
Ototoxic drugs			
Streptomycin (1966–68)	2(n=4)	11(n=38)	NS
Kanamycin (1967–71)	2(n=5)	20(n=63)	NS
Gentamicin (1971–72)	1(n=1)	9(n=30)	NS

*Aged 2 years.

Table 4 *Neonatal and developmental details of 10 children with sensory neural hearing loss, birthweights ≤1500 g*

Case	Pregnancy	Sex	Birthweight (g)	Gestation (weeks)	Birthweight/ gestational age	Intrapartum hypoxia	Neonatal hypoxia	Bilirubinaemia (μmol/l)	Infection	Incubator care (days)	Cerebral palsy	Visual defect	IQ Verbal	IQ Performance	IQ Full scale	Attainment
'Handicapped' and requiring special education																
1	Twin	M	1500	31	AGA	Apex beat <100 pH <7·2	Apnoea	187	Nil	49	Nil	Nil	65	76	67*	Slow learner
2	Single	M	910	26	AGA	pH <7·2	Nil	289	Nil	46	Nil	Nil	—	—	110†	Appropriate
3	Twin	F	1220	35	SGA	Nil	Apnoea (MV)	289	Rubella	34	'Clumsy'	Nil	—	—	100†	Appropriate
4	Quad	M	1310	30	AGA	Nil	Apnoea (MV)	153	Nil	49	Nil	RLF	—	—	100†	?Slow learner
'Compensated' by the use of hearing aids and remedial help in normal schools																
5	Single	F	1435	32	AGA	Nil	Apnoea	187	Nil	—	'Clumsy'	Nil	71	72	70*	Learning difficulties
6	Single	F	1446	32	AGA	Nil	Nil	340	Nil	—	Nil	Nil	69	75	69	Slow learner
7	Single	F	850	26	AGA	pH <7·2	Apnoea (BE = −23)	Nil	Nil	—	Nil	Nil	72	92	80*	Learning difficulties
8	Single	M	1090	28	AGA	Nil	Apnoea (MV)	Nil	Nil	64	Yes	Nil	85	65	73	Appropriate
Unilateral loss, hearing adequate for the perception of speech																
9	Twin	M	1030	28	AGA	Apex beat <100	Nil	Nil	Nil	41	Nil	Nil	82	100	90*	Learning difficulties
10	Twin	F	1200	30	AGA	Nil	Nil	349 (Rh affected)	Meningitis (aged 2 years)	35	Nil	Squint	—	—	94‡	Appropriate

*WISC at 8 years; †provisional score on Merrill-Palmer scale; ‡Stanford Binet at 3½ years. AGA = appropriate for gestational age, SGA = small for gestational age, RLF = retrolental fibroplasia, MV = mechanical ventilation.

insults. This was also suggested by the finding that 7 of the 10 children with sensory neural hearing loss were receiving extra help in school for such difficulties as distractability, overall delay, and manual control, as well as those due to their hearing loss. Data on the 10 children with sensory neural hearing loss are summarised in Table 4.

To investigate the aetiology of sensory neural hearing loss in this group of children, analysis of variance with stepwise regression was carried out using 29 social, obstetric, and perinatal variables. 25 (22%) of the 29 variables contributed significantly to the sensory neural hearing loss. The most important contribution (5% of the variance) was made by apnoeic spells occurring in the newborn period; this variable was the only one that made a significant independent contribution ($P<0{\cdot}05$). Four other variables—mechanical ventilation, jaundice (defined as a serum bilirubin $\geqslant$ 170 μmol/l), duration of incubator stay, and method of feeding—made significant additional contributions. However the treatment variables—mechanical ventilation, duration of incubator stay, and method of feeding—were significantly correlated with many of the illness variables. In order to try to separate the effects of the methods themselves and the conditions for which they were given, the variables were grouped into those concerning the antenatal period, perinatal illness, or treatment, and the regression was repeated. On this occasion, only 10 of the 11 variables concerning perinatal illness proved to be significant predictors of sensory neural hearing loss, and together these contributed 12% of the variance. As in the first regression, apnoeic spells occurring in the neonatal period was the most important variable, and the only one to make a significant independent contribution to the variance. Jaundice was the only variable to make a significant addition. Thus, it appears that perinatal illnesses, particularly those known or likely to have caused hypoxia in the newborn period including apnoea, were the most important factors associated with sensory neural hearing loss in this group of very low birthweight infants; jaundice may have had an additive effect.

Discussion

The incidence of sensory neural hearing loss was 9% (10 children) in this group of 111 children of birthweights 1500 g or less. All these children had survived after admission to the neonatal unit of UCH during the years 1966–72, when modern methods of perinatal management were being developed. Among the 10 affected children only 4 (4% of the whole group) were seriously handicapped by hearing impairment; 4 (4%) had moderate losses for which they were able to compensate by wearing hearing aids and receiving extra help within normal schools; and 2 (2%) had hearing which was adequate for perceiving speech without difficulty (Table 4). These figures are the same as those reported by Stennert *et al.* (1977) for a group of children of birthweights 2500 g or less, born 2 years earlier. Reports of other recent follow-up studies of low birthweight infants do not give sufficient data to allow comparison, although Davies and Stewart (1975) reviewing reports of such children suggested that the incidence of moderate to severe hearing impairment among recent very low birthweight survivors may be less than 2%.

Earlier workers reported incidences of about 10–12% of sensory neural hearing loss or moderate to severe deafness in prospective studies of very low birthweight infants (Drillien, 1961; Lubchenco *et al.*, 1963). Other reports gave incidences of 4–15% (Campanelli *et al.*, 1958; McDonald, 1964) for babies of short gestations and very low birthweights, but these figures were either based on selective assessments or were derived from selected groups of deaf children. Thus it is difficult to know if they represent maximum or minimum estimates.

These reports all concerned children who were born at least 20 years ago when little was known or understood about the hazards which low birthweight infants have to overcome in order to survive. Survival rates were low and the incidence of serious handicaps among the survivors was high, suggesting that many had sustained brain damage. What is more, few were nursed in incubators (Douglas and Gear, 1976), and of these, only those born during the 1950s were likely to have been exposed to noise from incubator motors. Incubators in general use before that date usually were heated by manually filled hot water tanks (Hey, 1974). In contrast, all intensive care survivors are likely to have been nursed in potentially noisy motor-driven incubators. In spite of this, the incidence of sensory neural hearing loss among these recent survivors was not greater than among the earlier born infants.

Schulte *et al.* (1977) and Stennert *et al.* (1977) have shown, among low birthweight infants (<2500 g), that 'nonoptimal' perinatal conditions and duration of incubator care both correlate with later hearing defects, but they were unable to separate the two factors. They concluded that noise levels of currently used incubators do not cause sensory neural hearing loss in otherwise healthy preterm infants, regardless of the duration of incubator care, but they were unable to ascertain whether incubator noise may contribute to the excess of hearing deficits noted in infants in whom the perinatal period was 'nonoptimal'.

None of the audiograms of the children with sensory neural hearing loss in this study showed a notch at 4000 Hz which is considered to be characteristic of damage to the cochlea due to long-term noise exposure (Chadwick, 1971). All the audiograms either had a gradual fall of threshold towards the high frequencies, or an abrupt loss at 8000 Hz only. Losses of this kind are generally associated on clinical grounds with anoxic insults or jaundice (Fisch and Osborn, 1954; Fisch, 1955; Fenwick, 1975).

Earlier workers (Johnsen, 1952; McDonald, 1964) suggested that anoxia was probably responsible for the sensory neural hearing loss they observed among low birthweight infants. In our own study, in addition to audiograms typical of this type of insult, or jaundice, perinatal illnesses, particularly those likely to have caused hypoxia in the neonatal period, significantly predicted sensory neural hearing loss; and a serum bilirubin level >170 μmol/l added significantly to the prediction.

This study shows that sensory neural hearing loss in very low birthweight infants is probably caused by hypoxia in the neonatal period and that jaundice may have an additive effect. We found no evidence to support the suggestion that ambient noise caused by incubator motors contributed to sensory neural hearing loss in these infants all of whom were nursed in commercially available incubators. However, as monitoring devices using noise signals are being used with increasing frequency in the management of such newborn babies, attention should be paid to ambient noise levels to ensure that potentially damaging levels are not exceeded.

We thank the doctors, nurses, and members of the audiological staff in the Paediatric Department and Royal Ear Hospital of University College and other hospitals for help, in particular Mr Graham Fraser, Mr Jonathan Hazell, and Professor E. O. R. Reynolds; also Miss Anthea Drake for rescuing the incubator treatment records, and Miss Wendy Galbraith for persuading us to embark on this study.

S.G. and A.S. are supported by a grant from the Department of Health and Social Security.

References

Beagley, H. A., Hutton, J. N. T., and Hayes, R. A. (1974). Clinical electrocochleography; a review of 106 cases. *Journal of Laryngology and Otology*, **88**, 993–1000.

Campanelli, P. A., Pollock, F. J., and Henner, R. (1958). An oto-audiological evaluation of forty-four premature children. *Archives of Otolaryngology*, **67**, 609–615.

Chadwick, D. (1971). Noise and the ear. In *Scott-Brown's Diseases of the Ear, Nose, and Throat. The Ear*, volume 2, pp. 475–593. Edited by J. Ballantyne and J. Groves. Butterworth: London.

Davies, P. A., and Stewart, A. L. (1975). Low birth weight infants: neurological sequelae and later intelligence. *British Medical Bulletin*, **31**, 85–91.

Douek, E., Bannister, L. H., Dodson, H. C., Ashcroft, P., and Humphries, K. N. (1976). Effects of incubator noise on the cochlea of the newborn. *Lancet*, **2**, 1110–1113.

Douglas, J. W. B., and Gear, R. (1976). Children of low birth weight in the 1946 cohort. *Archives of Disease in Childhood*, **51**, 820–827.

Drillien, C. M. (1961). The incidence of mental and physical handicaps in school age children of very low birth weight. *Pediatrics*, **27**, 452–464.

Fenwick, J. D. (1975). Neonatal jaundice as a cause of deafness. *Journal of Laryngology and Otology*, **89**, 925–932.

Fisch, L. (1955). The aetiology of congenital deafness and audiometric patterns. *Journal of Laryngology and Otology*, **69**, 479–493.

Fisch, L., and Osborn, D. A. (1954). Congenital deafness and haemolytic disease of the newborn. *Archives of Disease in Childhood*, **29**, 309–316.

Fraser, J. G., Conway, M. J., Keen, M. H., and Hazell, J. W. P. (1978). The post auricular myogenic response: a new instrument which simplifies its detection by machine scoring. *Journal of Laryngology and Otology*, **92**, 293–303.

Hey, E. M. (1974). Keeping babies warm. *Nursing Mirror*, No. 5, 62–67.

Johnsen, S. (1952). Natal causes of perceptive deafness. *Acta oto-laryngologica*, **42**, 51–63.

Lubchenco, L. O., Horner, F. A., Reed, L. H., Hix, I. E., Jr, Metcalf, D., Cohig, R., Elliott, H. C., and Bourg, M. (1963). Sequelae of premature birth. *American Journal of Diseases of Children*, **106**, 101–115.

McDonald, A. D. (1964). Deafness in children of very low birth weight. *Archives of Disease in Childhood*, **39**, 272–277.

Schulte, F. J., Stennert, E., and Vollrath, M. (1977). The ontogeny of sensory perception in pre-term infants (abstract). *Pediatric Research*, **11**, 1027.

Stennert, E., Schulte, P. J., and Vollrath, M. (1977). Incubator noise and hearing loss. *Early Human Development*, **1**, 113–115.

Stewart, A. L., and Reynolds, E. O. R. (1974). Improved prognosis for infants of very low birth weight. *Pediatrics*, **54**, 727–735.

Stewart, A. L., Turcan, D. M., Rawlings, G., and Reynolds, E. O. R. (1977). Prognosis for infants weighing 1000 g or less at birth. *Archives of Disease in Childhood*, **52**, 97–104.

Turcan, D. M., Rawlings, G., and Stewart, A. (1977). School performance at 8 years of infants who weighed $\leqslant$1500 g (abstract). *Pediatric Research*, **11**, 1025.

Correspondence to Dr Ann Stewart, Department of Paediatrics, University College Hospital Medical School, The Rayne Institute, 5 University Street, London WCIE 6JF.

Received 13 September 1978