Hearing Impairment in Low Birth Weight Saudi Children

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إن الهدف الرئيسي من هذه الدراسة هو استقصاء نسبة حدوث اعتلال السمع لدى المولودين دون الوزن الطبيعي.

لقد أجريت دراسة عشوائية على (٦٤٢١) طفل سعودي تتراوح أعمارهم من شهرين الى (١٢) اثنا عشر سنة، وقد تم اجراء تقييم سمعي للاطفال الذي كان وزنهم عند الولادة دون الطبيعي ولم يتعرضوا خلال حياتهم الى عوامل أخرى قد تؤدي الى اعتلال السمع.

كان وزن ٦٥٥ طفلاً منهم (١١,٢٪) عند الولادة (أقل من ٢٥٠٠ غرام) وتبين أن (١٧) طفلا من الـ (٦٥٥) (٣,٤٪) مصابون بنقص سمع حسي عصبي ليس له تفسير آخر. وهي نسبة أعلى بكثير من نسبة حدوث الصمم الحسي العصبي عند الاطفال ذوي الوزن الطبيعي عند الولادة (٠,٣٪)، علماً بأن الفئتين متماثلتين بالنسبة للجنس والسن.

وفي هذا البحث نناقش العوامل المحتملة التي تسبب اعتلال السمع الحسي العصبي لدى الاطفال الناقصي الوزن عند الولادة.

The main objective of this study was to investigate the prevalence of hearing impairment associated with low birth weight. A random survey of 6421 Saudi children aged between 2 months and 12 years was carried out. Children of low birth weight not having been exposed to other known causes of hearing impairment were subjected to audiological assessment. In the sample, 655 (11.2%) were of low birth weight (< 2500 grams). Of the low birth weight children tested for hearing, 17 (3.4%) were found to have otherwise unexplained sensorineural hearing loss. This is compared with an incidence of 0.3% sensorineural deafness of unknown cause found in an age- and sex-matched normal birth weight control group. The possible associated factors of sensorineural hearing impairment in LBW children are discussed.

The early detection of hearing impairment in children is of utmost importance because of the necessity to start proper training in speech and language at the youngest possible age. To achieve this goal, some authors have proposed mass screening for hearing of all the newborns.' However, the US Joint Committee on Infant Hearing has opposed mass universal screening of the hearing of neonates as inadequately productive and unnecessarily expensive.* The Joint Committee instead recommended that the emphasis should be placed on testing of high risk infants. The factors that identify those infants who are at risk for hearing impairment include the following:

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1. Family history of childhood hearing impairment.
2. *Intra-uterine* and *perinatal* infections (e.g. rubella, cytomegalovirus, toxoplasmosis, syphilis, herpes simplex).
3. Anatomical malformation of the ear, head and neck.
4. Low birth weight.
5. Hyperbilirubinaemia at a level exceeding the indication for exchange transfusion.
6. Bacterial meningitis.
7. Severe neonatal asphyxia.

The purpose of this study was to assess the prevalence of hearing impairment among LBW Saudi children (factor 4 in the high risk register) to reveal the true dimension of this problem.

Material and Methods

This study was conducted as a part of a comprehensive survey undertaken to assess the epidemiology and aetiology of hearing impairment among a random

sample of Saudi infants and children. The survey was conducted in Riyadh for a 27-month period from May 1988 to September 1990. The sampling design was essentially based on quota sampling using two interlocked quotas, age and sex. The actual sample selection was by a three-stage stratified random sampling of Riyadh, based on administrative areas and roads. The field work was carried out using the areas and roads as sampling frames because Riyadh households are not numbered and there are no names or landmark boundaries between segments except roads. For the study, 229 roads were randomly selected out of 1376. Each road was divided into manageable blocks; and samples of each block were randomly selected. Each child was assigned a code number and rhe questionnaire was subsequently administered to each selected child. The questionnaire included, among many other variables, the age. sex, birth u-eight, and the perinatal history with special emphasis on the presence or absence of the other high risk factors for hearing impairment. We defined low birth weight as <2500 g as suggested by the American Academy of Paediatrics.

Children of low birth weight (LBW) nor having been exposed to any of the other high risk factors were suhjecred to full physical examination and hearing assessment. Hearing evaluation w as done by pure tone audiometry or auditory- brain stem potential response (ABR), depending upon the mental age and the cooperation of the child, The same physical examination and hearing evaluation was carried out on an age- and sex-matched control population of an almost equal number of normal birth weight children with no history of exposure to the known causes of deafness. The type of loss in the ABR was assessed by analysing the abnormalities in the latency level curve extracted from the response pattern.' The amount of sensorineural hearing loss was established by the pure tone bone conduction threshold or by the ABR threshold.

Results

The total number of Saudi children entered into this study was 6421, of whom 55% were boys and 45% girls. Their ages ranged from 2 monrhs to 12 years with a mean of 56.67 months.

The number of children excluded because their birth weight could nor be determined was 558. These children were mostly delivered at home. The birth' weight distribution of rhe remaining 5862 children is illustrated in Table 1. This showed that LBW children accounted for 655 (11.2%) of all children with known birth weights. Thereafter a further 76 were excluded because they had been exposed to one or more of the other high risk factors for hearing impairment. The rest of the LBW children were referred for audiological assessment but 81 were unable to report because of various reasons. Consequently, 498 children (281 boys and 217 girls) were subiected to hearing evaluation; 377 bv ABR and 121 by pure tone audiometry.

Of the 5208 normal and over weight children, 500 (280 boys and 220 girls) were chosen as an age- and sex-matched control population. They had not been exposed to factors known to cause deafness. Hearing evaluation was performed by ABR on 382 and by pure tone audiometry on 118 children.

Table 1
Birth weight distribution of children with known birth weight

Birth weight (grams)	No.	%
<2500	655	11.2
2500–3000	3345	57.1
3001–3500	1214	20.7
3501 or more	649	11.1
TOTAL	5863	100

Table 2
The age distribution of the children

Age (months)	Low birth weight children) NO. (%)	Normal weight children NO. (%)
<12	77 (15.5,	75 (15)
13–24	65 (13.1)	64 (12.6)
25–36	51 (10.2)	50 (10)
37–48	50 (10)	48 (9.6)
49–60	48 (9.6)	52 (10.4)
61–72	53 (10.6)	53 (10.6)
73–84	28 (5.6)	30 (6)
85–96	30 (6)	30 (6)
97–144	96 (19.3)	98 (19.6)
TOTAL	496 (100)	500 (100)

Table 3
Results of hearing assessment

Finding	Law birth No. (%)	Control population No. (%)
Normal hearing	427 (85.7)	444 (88.8)
Conductive hearing loss due to secretory otitis media	42 (8.5)	40 (8)
Conductive hearing loss due to chronic suppurative otitis media	12 (2.4)	13 (2.6)
Sensorineural hearing loss	17 (3.4)	3 (0.6)
TOTAL	498 (100)	500 (100)

The age distribution of the children is shown in Table 2 and the results of the hearing assessments of both groups are illustrated in Table 3.

The number of children found to have 20 dB or more sensorineural hearing impairment in the LBW group was 17 which represented 3.4% of the total LBW children tested for hearing. Only three cases (0.6%) of unexplained sensorineural hearing loss were detected among the control group (χ^2_1 10.06 $p<0.01$). All the cases of sensorineural hearing loss were bilateral. The degree of hearing loss is shown in Table 4.

Table 4
Degree of the sensorineural hearing loss

Degree of hearing impairment	Low birth weight	Control children
Severe to profound loss	5	2
Moderate loss	7	1
Mild loss	5	0
TOTAL	17	3

Unfortunately, information about the use of incubators was generally not clear for most of the children and hence was not of any value.

Discussion

In this study, the incidence of sensorineural hearing loss in LBW children (2500 g or less) was 3.4% which is much higher than the 0.3% incidence found in the group of control population. The difference is statistical significant, using Fisher's exact test pi 0.001. This study's prevalence rate is in line with the reported incidences of 2-15.9% sensorineural hearing loss among LBW children.[4-8] The reported incidences are much higher (10–100 times) than in normal weight neonates.* Van Zanten et al. (1988) reviewed the literature and found that prematurity accounted for between 10 and 30% of total high risk group.* The variation in the published prevalence of sensorineural hearing loss of the LBW children was probably due to the variation in the selection criteria and to the methods used for hearing evaluation. Abramovich et *al.* (1979) found an incidence of 9% (10 children) in a group of 111 children of birth weight 1500 g or less evaluated mainly by pure tone audiometry.[6] Anagnostakis *et* al. (1982) reported almost the same incidence (9%) in 98 infants of birth weights of 1800 g or less evaluated by pure tone and impedance audiometry.[7] Using ABR in evaluating the hearing of 108 preterm infants with birth weights between 800 and 2000 g Van Zanten et al. (1988) reported an incidence of 2% of moderate to severe cochlear loss.[8]

The ABR recording as a method of determining the threshold of hearing especially in the preterm infants has its limitations. ABR lacks the frequency selectivity and lacks sufficient precision in threshold determination.9 Also it has been reported that the ABR threshold increases linearly with decreasing age after conception in infants born prematurely but who are otherwise normal. This is assumed to be because of immaturity of the brainstem.[10]

The cause of the deafness in LBW infants is speculative. Pathological evidence of the nature of the lesion has never been satisfactory because deafness by itself is not fatal and because postmortem changes in the cochlea begin very early. Several factors have been suggested to account for this sensorineural hearing loss e.g. the 'immaturity' of these babies, the ambient noise of the incubators in which infants were nursed, the use of ototoxic drugs, and the presence of some perinatal complications (e.g. hypoxia, acidosis, hyperbilirubinaemia) which are more frequently encountered among LBW infants.

The potential harmful effect of incubator noise has been studied, but the role of this noise as causative factor for deafness is debatable. Falk et *al.* (1974) found the cochlea of newborn guinea pigs is more susceptible to high-intensity noise than that of adult guinea pigs.[11] Douek *et* a/. (1976) demonstrated considerable loss of outer hair cells of cochlea of newborn guinea pigs after subjecting them to incubator noise while no such change were found in adult guinea pigs.'" Also, animal experiments have shown that noise trauma may aggravate the effect of anoxia or antibiotics." Bess et al. (1979) drew attention to the synergic effect of the steady-state incubator noise and the relatively intense impulse noise produced by life supporting machines and by striking the side of the incubator (manoeuvre used by some nurses and physician to stimulate breathing in apnoeic infants).[14] On the contrary to the above, some authors suggested that the ambient noise caused by the incubator *motors* is not contributing to the sensorineural hearing loss in LBW infants.[6,7] Abramovich et al. (1982) found no difference in the incidence of sensorineural hearing loss among infants nursed in motor driven incubators currently in use and those nursed before the 1950s when incubators were heated by manually filled hot water tanks.[6] Also there was no correlation noticed between the incidence of sensorineural hearing loss and the duration of incubator stay.[7] Abramovich *et* al. (1979) and Anagnostakis et al. (1982) in their studies of LBW infants showed that none of the audiograms of the children with sensorineural hearing loss showed a notch at 4000 Hz which is considered to be characteristic of damage to the cochlea due to long-term noise exposure.[6,7]

The possibility that perinatal asphyxia causes sensorineural deafness finds some support from observations made at necropsy and from some clinical studies. Hall (1964) reported lesions in the dorsal and ventral cochlear nuclei and in the cochlea after terminal asphyxia at birth.[15] In addition, a history of apnoeic spells was found

much more commonly in children with hearing impairment than in healthy children.[6,7,16] It was also noticed that all the audiograms of LBW with sensorineural hearing loss had either a gradual fall of threshold toward high frequencies or an abrupt loss at 8000 Hz only, and losses of this kind are generally associated on clinical grounds with anoxic insult or jaundice.[6]

Other factors which have been incriminated as causes of sensorineural hearing loss in infants of LBW are acidosis, hypothermia, and a low serum albumin concentration. These factors are believed to increase the neurotoxicity of bilirubin, either by increasing the unbound portion of bilirubin or by increasing its access to the brain."

In consideration of this problem Schulte & Stennert (1978) found no relationship of hearing loss with gestational age, hyperbilirubinaemia, drugs, or the duration of incubator care.[18] There was, however, a strong correlation between hearing loss and perinatal non-optional score, i.e. the sum of perinatal risk factors. They also concluded that noise levels of currently used incubators do not cause sensorineural hearing loss in otherwise healthy preterm infants but they were unable to ascertain whether incubator noise might contribute to the excess of hearing deficits noted in infants in whom the perinatal period was 'non-optimal'.

This present study showed a high frequency of sensorineural hearing loss among LBW children. The causes of this hearing impairment is still controversial but it is logical to assume that improvement of obstetric and neonatal care will diminish the incidence of such a hearing impairment. It is the otologist's responsibility to identify these infants with hearing impairment as early as possible by screening LBW infants and considering them as being at a high risk. The authors recommend ABR screening for all premature neonates as soon as it becomes possible, with a follow-up examination for those judged to have impaired hearing at the initial examination.8 Meanwhile it should be known that ABR determines a gross level of hearing loss, whilst a slight degree of cochlear damage may remain undetected.[9]

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