



Risk Factors and Incidence of Hearing Impairment among Children in the Audiology Department of a Children Hospital

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Abstract

Background: The incidence of sensorineural hearing loss is between 1 and 3 per 1000 in healthy neonates and 2 to 4 per 100 in high-risk infants.

Methods: In this study, we assessed the incidence of hearing impairment in normal term (≥ 37 wga) infants (control group), in children with suspicion and/or risk factors of hearing loss, included premature infants (<37 Weeks Gestational Age (WGA) and/or low birth weight <2.5 Kg), in children diagnosed with a specific syndrome and in children with speech disorder, candidate for speech therapy. All premature infants (<37 Weeks Gestational Age (WGA) and/or low birth weight (<2.5 Kg) were treated as infants with risk factors due to their prematurity.

Results: 63% of all tested children were normal term (≥ 37 WGA), 21% were preterm, 4% had at least one other risk factor for hearing loss except prematurity (Ototoxic medications, prolonged respirator care in intensive care unit, high serum bilirubin concentration, family history of hearing loss, craniofacial anomalies, congenital infections), 2% were diagnosed with a specific syndrome and 10% were candidate for speech therapy.

The final diagnosis of hearing impairment was given predominantly in extremely preterm infants born <28 weeks of gestation -19% and in extremely low birth weight preterm infants (<1 Kg) -18%.

Hearing deficit was diagnosed in 9% of children with other risk factors for hearing loss, except prematurity.

Hearing impairment was diagnosed in 2% of syndromic children.

On the contrary, only $<1\%$ among children candidate for speech therapy had hearing loss. It is noteworthy that almost 75% of the children who were candidates for speech therapy were children who belonged to the autism spectrum.

Conclusion: Hearing impairment is a severe consequence of prematurity. Its prevalence is inversely related to the maturity of the baby. Premature infants have many concomitant other risk factors for hearing loss, which influence the occurrence and the intensity of the hearing deficit.

Keywords: Hearing loss; Prematurity; Risk factors; Otoacoustic emissions; Auditory brainstem response

Abbreviations

AN/AD: Auditory Neuropathy-Dysynchrony; ABR: Auditory Brainstem Response; DPOAE: Distortion Product Otoacoustic Emissions; OAEs: Otoacoustic Emissions; PHL: Permanent Bilateral Hearing Loss

What is Known

- The combination of OAEs with A-ABR is considered ideal for early detection and diagnosis of congenital hearing loss and Auditory Neuropathy-Dysynchrony in high risk newborns.
- Both above parameters (age and weight) are of particular importance for severe hearing impairment and it has not been found that one factor prevails over the other.

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What is New

- Other frequent risk factors for hearing impairment in premature infants e.g. congenital infections and family history of hearing loss, although frequently recorded, does not seem to be very significant.
- Children with speech disorder do not seem to suffer from hearing impairment more frequently than children in general population and are mainly children who belong to the autism spectrum.

Introduction

The incidence of sensorineural hearing loss is between 1 and 3 per 1000 in healthy neonates and 2 to 4 per 100 in high-risk infants.

In this study, we assessed the incidence of hearing impairment in normal term (≥ 37 wga) infants (control group), in children with suspicion and/or risk factors of hearing loss, included premature infants (<37 Weeks Gestational Age (WGA) and/or low birth weight <2500 gr), in children diagnosed with a specific syndrome and in children with speech disorder, candidate for speech therapy. All premature infants (<37 Weeks Gestational Age (WGA) and/or low birth weight <2500 g) were treated as infants with risk factors due to their prematurity.

Significant risk factors of hearing loss are high-risk premature infants (<37 weeks gestation), low birth weight (<2500 g), low Apgar score 0 to 4 at 1 min or 0 to 6 at 5 min, prolonged respirator care in intensive care (>7 days), mechanical ventilation for at least 5 days, high serum bilirubin concentration, hyponatremia, family history of hearing loss, craniofacial anomalies-anomalies of pinna, ear canal, ear tags, ear pits and temporal bones, complex congenital anomalies associated with congenital hearing loss, congenital infections (TORCH infections, particularly cytomegalovirus), ototoxic medications including but not limited to aminoglycosides used in multiple courses or in combination with loop diuretics such as furosemide and bacterial meningitis [1,2].

Methods

Data collection

We reviewed retrospectively the audiological charts of all children ($n=448$) who were admitted to the Audiology Department of our hospital between 2014 and 2020. All children had none /one or more risk factors for hearing loss, as they have been defined and modified by the American Joint Committee on Infant Hearing during the study years [3-6]. All premature infants (born <37 wga and /or birth weight <2.5 Kg) were treated as infants with risk factors due to their prematurity.

Audiologic evaluation included history taking, otoscopy, tympanogram and screening with TOAEs (Transient Otoacoustic Emissions).

ABR (Auditory Brainstem Response) testing under natural or chloral hydrate-induced sleep were also performed in all neonates and infants with abnormal TOAEs responses and/or had risk factors. Especially preterm infants, with an increased risk for SNHL and auditory neuropathy spectrum disorders, are screened with Auditory Brainstem Response (ABR) which allows objective and accurate assessment of the hearing function, with normal variation according to age due to physiological maturation of the auditory pathway [7]

(Figures 1-6).

Results

We analyzed the incidence of different types and intensity of hearing deficits in relation to gestational age and gestational weight.

For the evaluation of hearing threshold all the examined children had tympanogram type A bilaterally (Figure 5).

The most serious problem—permanent profound sensorineural unilateral and/or bilateral hearing deficit (90 dB) was diagnosed in 12.4% of extremely preterm infants <28 wga, in 5% at very preterm infants 28 to <32 wga and in 0.2% at moderate to late preterm infants 32 to <37 wga) (Figure 1).

Based on gestational weight, the most serious problem-permanent profound sensorineural unilateral and/or bilateral hearing deficit (90 dB) was diagnosed in 17.7% of extremely low birth weight preterm infants (<1 Kg), in 3% at very low birth weight preterm infants (<1.5 Kg), in 0.8% at low birth weight preterm infants (<2.5 Kg) (Figure 1).

The mean time of the final diagnosis in all groups of children was the 120th day of life.

63% of all tested children were normal term (≥ 37 wga), 21% were preterm, 4% had at least one other risk factor for hearing loss except prematurity (ototoxic medications, prolonged respirator care in intensive care unit, high serum bilirubin concentration, family history of hearing loss, craniofacial anomalies, congenital infections), 2% were diagnosed with a specific syndrome and 10% were candidate for speech therapy.

80% of all the examined children were males and 20% females.

Among all preterm ($n=94$), based on gestational age, 71.21% were moderate to late extremely preterm infants <28 wga.

Among all preterm ($n=94$), based on gestational weight 79% were low birth weight preterm infants (<2.5 Kg), 15% very low birth weight preterm infants (<1.5 Kg) and 6% extremely low birth weight preterm infants (<1 Kg).

Hearing deficit was diagnosed in 5.82% of all the examined children.

78% among the children with hearing deficit were males and 22% females.

Hearing deficit was diagnosed in 9% of all preterm infants.

75% of the preterm children were males and 25% females.

Hearing screening was positive for hearing impairment in 19% at extremely preterm infants <28 wga in 5.5% at very preterm infants 28 to <32 wga and in 3% at moderate to late preterm infants 32 to <37 wga.

Hearing screening was positive for hearing impairment in 18% of extremely low birth weight preterm infants (<1 Kg), in 7.5% at very low birth weight preterm infants (<1.5 Kg) and in 2% at low birth weight preterm infants (<2.5 Kg).

Hearing screening was positive for hearing impairment in $<1\%$ in normal term (≥ 37 wga) infants (control group). In most such cases typical and replicable ABR waveforms were elicited at 60 and 40 dBnHL bilaterally (Figure 3).

All above results were statistically significant (p value <0.05).

Thus, the final diagnosis of hearing impairment was given predominantly in extremely preterm infants born <28 weeks of gestation -19% and in extremely low birth weight preterm infants (<1 Kg) -18%.

Hearing deficit was diagnosed in 9% of children with other risk factors for hearing loss, except prematurity.

The most important other risk factors were ototoxic medications, very low birth weight and "treatment in the intensive care unit" (low Apgar score and mechanical ventilation).

Risk factors of hearing deficit were found in 4% of all tested infants. All premature infants (born <37 wga) were treated as infants with risk factors due to their prematurity. At least one other risk factor was identified in 85% of them. Most frequent risk factor was exposure to "ototoxic medications", accounting for 63% in this population. The second and the third most frequent risk factors were "low birth weight <2.5 Kg" - 54% and "treatment in the intensive care unit" - 42%.

Other significant frequent risk factors were congenital infections 5% and family history of hearing loss 3%, however their frequency in normal term (control group) infants was comparable to preterm infants (<37 wga) and was not significant (p value <0.05).

Hearing impairment was diagnosed in 2% of syndromic children.

On the contrary, only $<1\%$ among children candidate for speech therapy had hearing loss. In the rest of those children normal otoacoustic emissions were obtained bilaterally (Figure 2). It is noteworthy that almost 75% of the children who were candidates for speech therapy were children who belonged to the autism spectrum. The frequency of hearing impairment in children with speech disorder was comparable to general population and was not statistically significant (p value >0.01). Thus, hearing impairment in children with speech disorder does not seem to be the etiological factor of their disorder and they are candidate for speech therapy due to other reasons, such as autism, cognitive (intellectual, thinking) or other developmental delays, weak oral muscles, chronic hoarseness, cleft lip or cleft palate, motor planning problems and articulation problems.

Discussion

Permanent bilateral Hearing Loss (PHL) affects 1 to 3/1000 live

births in wellborn infants and 2 to 4/100 infants in the Neonatal Intensive Care Unit (NICU) population [8-10].

In order to achieve effective treatment, congenital or perinatal hearing loss should be recognized within three months of life, with confirmative audiological diagnosis and early intervention before the 6th month of age [11]. Early treatment is essential, as the first year of life is critical for normal development of speech and language, as well as intellectual and emotional growth [7,12,13,]. Children with untreated hearing loss may have delayed or limited speech and language development as they age. They may have difficulty understanding others, learning new words and saying them properly. Children may also have difficulty socializing with other children because of their communication deficits. The sooner the hearing loss is diagnosed and treated, the more likely the child will have better speech and language outcomes, according to ASHA. In some cases, hearing loss may develop at a later age for premature babies. Depending on the factors associated with the prematurity, some children may be at risk for onset, delayed hearing loss.

Premature infants are 50% more likely than normal term infants to develop hearing loss [13,14]. There are two sub-categories of premature infants based on gestational age and based on birth weight. According to gestational age, the premature infants are separated in extremely preterm infants born <28 wga, in very premature infants born between 28 to <32 wga and in moderate to late preterm infants born between 32 to <37 wga. According to birth weight the premature infants are separated in extremely low birth weight <1000 g, very low birth weight <1500 g and in low birth weight <2500 g (Figure 4). Based on gestational age, the prevalence of hearing loss in our group was 19% at extremely preterm infants <28 wga, 5.5% at very preterm infants 28 to <32 wga and 3% at moderate to late preterm infants 32 to <37 wga.

Based on gestational weight, the prevalence of hearing loss in our group was 18% in extremely low birth weight preterm infants (<1 Kgr), 7.5% in very low birth weight preterm infants (<1.5 Kg) and 2% in low birth weight preterm infants (<2.5 Kg).

This latter value is in substantial agreement with the available literature data [15,16].

Marlow et al. [17] referred that preterm children with SNHL required more intensive care in the perinatal period and developed more neurological complications than controls. Among very preterm babies, the coexistence of risk factors for hearing loss may be more

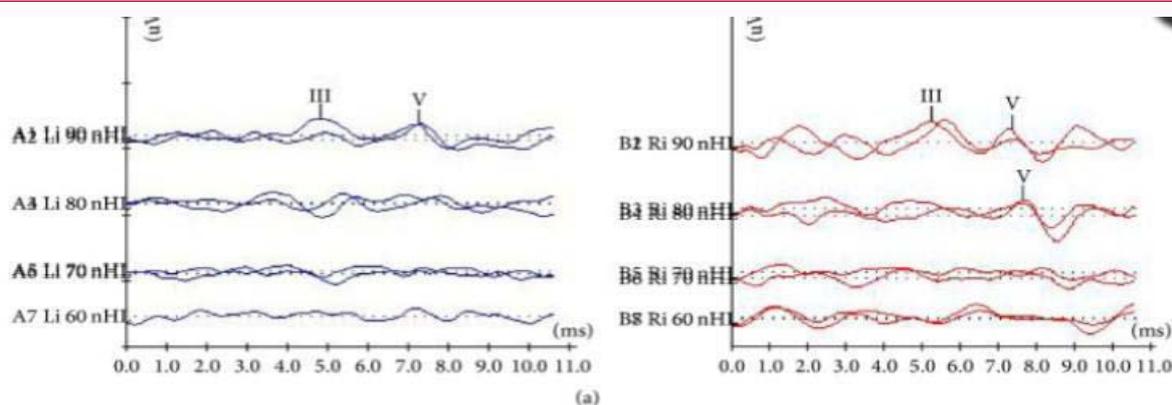


Figure 1: ABR waveforms were obtained at 90 dBnHL on the left and 80 dBnHL on the right ear.

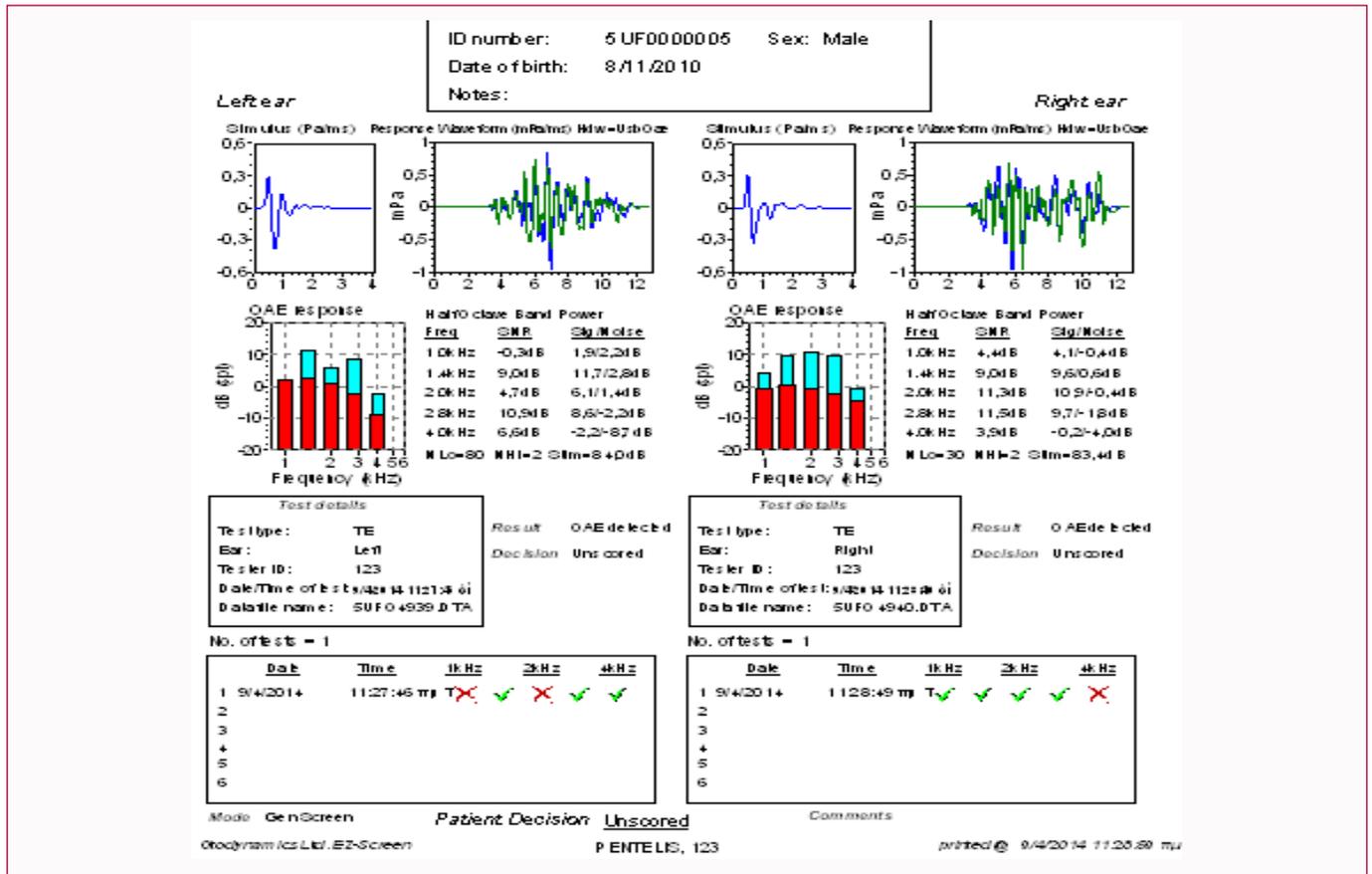


Figure 2: Normal otoacoustic emissions were obtained bilaterally.

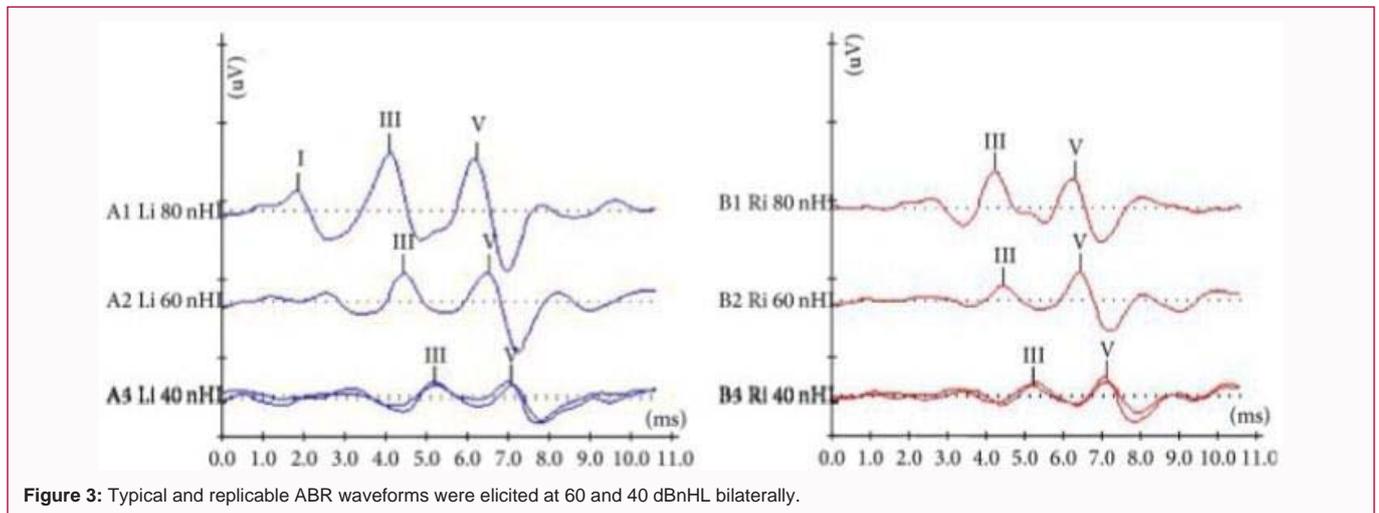


Figure 3: Typical and replicable ABR waveforms were elicited at 60 and 40 dBnHL bilaterally.

important than the individual factors themselves.

Ira Bergman et al. [18] concluded in their study that bilateral hearing loss occurred in 9.7% of infants who survived despite very low birth weight (<1500 g), 16.7% of infants who survived neonatal seizures, and 28.6% of infants who survived both low birth weight and neonatal seizures. All neonates in the above study received treatment in a single neonatal intensive care unit between 1976 and 1980. Twenty-two of 36 hearing-impaired children were normal physically and mentally, with IQ scores of >85. Significant neonatal predictors of hearing loss in high-risk premature infants (<37 weeks gestation), as

determined by multivariable testing, were prolonged respirator care, high serum bilirubin concentration, and hyponatremia. Exchange transfusions were associated with a decreased risk of hearing loss.

Bacterial meningitis is a significant cause of late sensorineural hearing loss. Because the cochlea can ossify after meningitis, making it difficult or impossible to place a cochlear implant, children with severe sensorineural hearing loss due to meningitis, should be considered for short cochlear implant placement (within 6 months of illness).

This study confirms the frequency of hearing loss among surviving

sub-categories of premature infants:
BASED ON GESTATIONAL AGE;

- i. extremely preterm (<28 weeks)
- ii. very preterm (28 to <32 weeks)
- iii. moderate to late preterm (32 to <37 weeks).

BASED ON WEIGHT;

- i. Low birth weight <2500g
- ii. Very low birth weight <1500g
- iii. Extremely low birth weight <1000g

Figure 4: Sub-categories of premature infants.

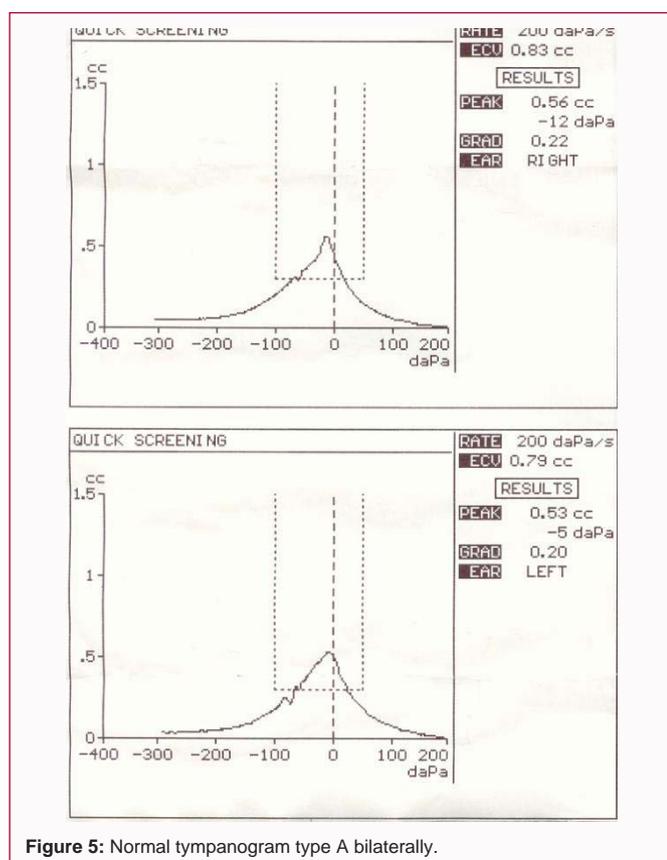


Figure 5: Normal tympanogram type A bilaterally.

VLBW premature infants and/or among extremely preterm infants born <37 weeks and highlights the fact that 90% of these children are otherwise neurologically and intellectually intact. Prevention of neonatal hearing loss can only be achieved by knowledge of its cause.

The most common causes of neurologic disabilities in the preterm infant, hypoxic-ischemic insults and intracranial hemorrhage are unlikely causes of isolated hearing loss. Kernicterus can produce hearing loss as its only manifestation, and this retrospective study demonstrates an association between hyperbilirubinemia and hearing loss in premature infants. Prospective studies, however, are necessary to demonstrate whether hyperbilirubinemia is an important, preventable, cause of hearing loss in the premature infants.

It is known that only 50% of children with sensorineural hearing loss are detected by the high-risk group. The other hearing-impaired

children never had any of the known risk factors. For this reason it is necessary to conduct screening tests for all newborns.

Related to the molecular genetics of hearing loss, it is estimated that in the developed world more than 50% of childhood hearing loss is due to genetic causes. Since 1996, 40 new genes associated with non-syndromic hearing loss have been identified, as well as a number of genes associated with syndromic hearing loss. In addition, more than 120 gene positions related to non-syndromic hearing loss have been identified and more than 400 separate syndromes associated with hearing loss have been recorded. Congenital hearing loss is inherited in the autosomal recessive (80%), autosomal dominant (15%), sex-linked (2% to 3%) and mitochondrial (1% to 2%) manner. Most deaf children are born from parents with normal hearing. Hereditary hearing loss varies in expression and penetration, passing on to one or more generations (Figure 6, Table 1).

Non-Syndromic Hearing Loss (NSHL) accounts for about 70% of genetic hearing loss. Autosomal Recessive Non-Syndromic Hearing Loss (ARNSHL) is characterized by severe to very severe prelingual hearing loss. It is estimated that more than 100 genes are responsible for Non-Syndromic Hearing Loss (NSHL) (Figure 6, Table 1).

In the chromosomal region 13q12-13 is located the gene GJB2 encoding Connexin 26 (Cx26), which is responsible for the recycling of potassium ions.

It is estimated that mutations in the GJB2 gene are responsible for more than 50% of cases of autosomal Non-Syndromic Hearing Loss (NSHL) phenotypes and for 20% of all pediatric prelingual hearing loss. The main mutation (35delG) occurs with a frequency of 2% to 4% in the general population. In Greece the frequency is 3.5%. That is, almost 1 in 28 adults is heterozygous for this mutation. Based on these findings, there is a strong interest in screening for Cx26 mutations (35delG) as a cause of congenital hearing loss and a special screening kit is now commercially available (Figure 6, Table 1).

In cases of bilateral severe Non-Syndromic Sensorineural Hearing Loss (NSHL), the presence of the 35delG mutation was investigated due to its large impact on the general population in Greece. Thus in our study, neonates with bilateral profound sensorineural hearing loss referred for genetic testing in order to detect early the frequent mutation in the gene GJB2 which encodes the protein Connexin 26 (Cx26) in the family. Only in 20% cases of infants with bilateral severe sensorineural hearing loss who referred for genetic screening was detected this specific mutation.

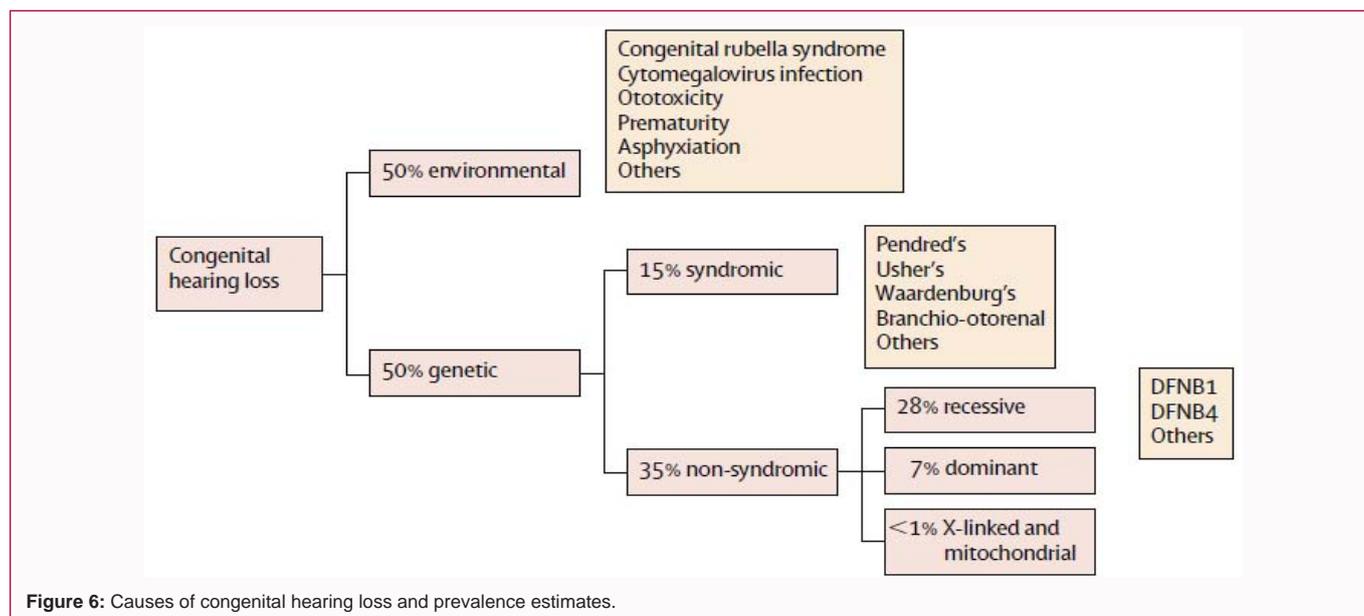


Figure 6: Causes of congenital hearing loss and prevalence estimates.

Table 1: Known causes of UHL or MBHL and prevalence estimates.

Connexin	Accounts for 30% to 40% of all genetic hearing loss; 1% to 16% of these cases are estimated to result in MBHL.
Mitochondrial	Accounts for less than 1% of prelingual deafness and 5% of postlingual, nonsyndromic hearing loss; 21% of these cases are MBHL.
Sudden	Three percent to 5% of all cases occur in children; as many as 98% have UHL.
Auditory neuropathy	Accounts for less than 1% of all pediatric hearing loss; approximately 33% of cases have borderline normal, MBHL, and there have been few reports of UHL.
Mumps	Accounts for approximately 2% of childhood hearing loss; 80% to 95% of these cases are UHL.
Congenital CMV	Thirteen percent to 24% of all children with asymptomatic CMV and up to 40% of children with symptomatic CMV will have SNHL; 17% of the asymptomatic and 12% of the symptomatic cases are MBHL; 52% of the asymptomatic cases and 33% of the symptomatic cases are UHL.
Meningitis	Ten percent of all children with bacterial meningitis are left with SNHL; 4% to 30% of these are UHL; 14% are MBHL.
Prematurity	Five percent of premature infants have MBHL or UHL.

Note: MBHL: Mild Bilateral Hearing Loss; SNHL: Sensorineural Hearing Loss; UHL: Unilateral Hearing Loss

All infants with prelingual severe sensorineural hearing loss confirmed by repeated audiological procedures (at least two ABR before the age of one year old) are candidate for cochlear implantation. It is known that among children with atypical ABR waveforms, a complete restoration to normal ABR waveforms was noted within few months in follow up. This phenomenon may reflect maturational central nervous system changes after the resolution of harmful factors.

In a retrospective study of the audiological records of high risk neonates with atypical ABR waveforms suggestive of hearing loss, a complete restoration of all atypical waveforms to normal was noted within few months [19]. According to the findings of this study, regular follow up is of paramount importance in order to determine the final ABR threshold and morphology. The potential of restoration of electrophysiological threshold points should be refrained from any invasive methods of rehabilitation (e.g. cochlear implantation) until the definitive ABR threshold confirmation.

Both Otoacoustic Emissions (OAE) and Auditory Brainstem Responses (ABR) are tests widely used in neonatal hearing screening. This study aimed to investigate the differences and clinical value of Distortion Product Otoacoustic Emissions (DPOAE) and ABR in hearing screening of high-risk neonates. The ABR test appears to be more reliable for hearing screening in high-risk neonates. It is

suggested that hearing screening for high-risk neonates should be conducted with ABR first, followed by OAE after failure on ABR. This is because the incidence of AN/AD was found at 25% in this high-risk population for sensorineural hearing loss and OAE cannot detect the Auditory Neuropathy-Dysynchrony [20].

OAEs are a simple, short-term, effective, non-invasive method that is applied from the first hours of life. The sensitivity of the method and its specificity remain quite low (50% to 85%). It does not specify the type of hearing loss and the exact hearing threshold. OAEs are not recorded in newborns with middle ear pathology. It does not control frequencies higher than 6 kHz and lower than 1 kHz.

ABR is a perplexible method of hearing loss detection. The sensitivity of the method is reported to be 100% and the specificity 97% to 98%. The cost and considerable time required for preparation have prevented their widespread spread. Moreover it is an invasive method, because usually requires suppression of the newborn. ABR cannot be used as the only method of evaluation of hearing loss. There are anatomical and clinical restrictions. They are produced after stimulation of the cochlear region 2 kHz to 4 kHz, not from lower frequencies. They are a start-up response, possibly due to a subset of sensitive neurons in the auditory system. The acoustic structures above the brain stem are not involved at all: They do not provide any information about the functionality of the auditory cortex. ABR are

normal in low frequency hearing loss, absent in normal hearing at low frequencies, absent in diseases that degenerate nerve conduction, normal in severe lesions of the cortex. Moreover ABR are not related to the perception of volume and to speech comprehension.

Thus the combination of OAE and ABR is necessary in non-diagnostic cases. The aim of the various protocols in screening of neonatal hearing loss is to increase as much as possible both the sensitivity and the specificity of the standard screening test methods. This is achieved by reduction to the minimum of false positive results (<3%) (i.e. the percentage of newborns without hearing loss who do not pass the test and by zeroing (0%) of false negative results (i.e. the percentage of newborns with hearing loss who pass the hearing test).

It is noteworthy that almost 75% of the children who were candidates for speech therapy were children who belonged to the autism spectrum. The frequency of hearing impairment in children with speech disorder was comparable to general population. Thus, children with speech disorder, does not seem to suffer from hearing loss and they are candidate for speech therapy due to other reasons. This is not consistent with the literature till date [21].

Conclusions

1. The diagnosis of sensorineural hearing loss should be done as early as possible. This can only be achieved through unified tracking and recording protocols for all births nation widely. Diagnosis is achieved by crossing all available techniques (Typanometry, OAEs, ABR).

2. Although OAEs appear to be the most appropriate screening test method for normal full-term neonates, the combination of OAEs with A-ABR is considered ideal for early detection and diagnosis of congenital hearing loss and Auditory Neuropathy-Dysynchrony in high risk newborns.

3. Monitoring of high risk infants for at least the first 3 years of life is necessary.

4. In the investigation of the bilateral prelingual hearing loss the 35delG mutation should be detected due to its large impact on the general population.

5. Hearing impairment is a severe consequence of prematurity and its prevalence is inversely related to the maturity of the baby based on gestation age and/or birth weight. Both above parameters (age and weight) are of particular importance and it has not been found that one factor prevails over the other.

6. Premature infants have many concomitant risk factors for hearing impairment which influence the occurrence and the intensity of hearing deficit. The most important other risk factors were ototoxic medications, very low birth weight and "treatment in the intensive care unit (low Apgar score and mechanical ventilation).

7. Other frequent risk factors e.g. congenital infections and family history of hearing loss, although frequently recorded, does not seem to be very significant.

8. Children with speech disorder do not seem to suffer from hearing impairment more frequently than children in general population. They are candidate for speech therapy due to other reasons.

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