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EDITORIAL: OTOTOXICITY

Ian J Mackenzie MD MSc

Liverpool School of Tropical Medicine
Pembroke Place
Liverpool L3 5QA
UK

Email: macken34@liv.ac.uk

The majority of illnesses worldwide are treated with medication. Medicines have been used in the treatment of illness for hundreds of years. The medicines of today are sophisticated and their development is at the forefront of science. Medicines have eradicated some diseases and controlled others. The successful conquering of some cancers, previously thought untreatable, show the ingenuity and science that has been part of their development. However, the development of new drugs takes a long time and is a very expensive process.

Side Effects of Medicines

Drugs of all sorts have known side effects which can only be discovered by clinical experimentation and a careful and accurate recording system of any side effects a drug generates. Subtle side effects, like tinnitus and deafness, are not identified until many people are affected and it is reported in the medical literature. It is difficult, particularly in animal experiments, to record tinnitus or deafness as a side effect of a drug, even though the hearing and balance systems are anatomically very delicate and sensitive to toxins.

Teaching and learning at a School for the Deaf in Afghanistan
Photo: SHIP/CBM

Ototoxicity

The term ototoxicity covers any damage to hearing and balance caused by a toxin, as it affects the end organs of the eighth cranial nerve. The toxin can enter the body by ingestion, inhalation or with skin contact. When a patient needs life-saving medication for severe illness, the problem may be whether or not to prescribe certain essential drugs knowing that there is a high chance of leaving the patient with a long term hearing deficit. There is little information on the side effects of herbal and homeopathic medication.

The long term cost to the community of ototoxicity is not known but hearing impairment, deafness, tinnitus or dizziness can cause difficulties with language development and learning in children, and with work communication and performance.

In many instances, these side effects are not present if drugs are prescribed properly. Some drugs may interact with each other and lead to increased toxicity, but these interactions are not well understood. When the cause of deafness is not identified, it is important to review occupation and medication to see if there is any toxin responsible for the deafness. Ototoxicity is more common than often suggested and in most cases is a preventable cause of deafness.

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DEAFNESS CAUSED BY OTOTOXICITY IN DEVELOPING COUNTRIES

Rajan N Patel

Medical Student
Department of Child and Reproductive Health
Liverpool School of Tropical Medicine
Pembroke Place
Liverpool L3 5QA
UK

Email: rajanpatel86@hotmail.com

Table 1: Grades of Hearing Impairment

Grade of impairment	Corresponding audiometric ISO* value
0 - None	25 dB or better
1 – Slight	26-40 dB
2 – Moderate	41-60 dB
3 - Severe	61-80 dB
4 – Profound	81 dB or greater

*International Organization for Standardization

This report attempts to review systematically the current literature on deafness caused by ototoxicity in developing countries and make an appraisal of its current status in different regions of the developing world. This involves critically assessing research and accessing routine electronic literature databases.

Deafness Worldwide

Deafness is a worldwide problem. It is estimated that 1:1000 children are born deaf, while 2:1000 children are born hard of hearing.[1] In 2002, the World Health Organization estimated that 250 million people in the world had a disabling hearing impairment and that two-thirds of these people lived in developing countries.[2] Furthermore, Torrigiani in Geneva outlined that avoidable hearing impairment and deafness are an important public health problem, particularly in low-income countries. Although infectious conditions, such as otitis media, account for the largest proportion of conductive hearing loss, damage to sensori-neural hearing caused by ototoxic medication has been increasingly reported from countries in recent years.[3]

Grades of Hearing Impairment

Deafness can be expressed as a complete loss in the ability to hear from one or both ears. It can also be described as a hearing threshold of 81dB or greater, averaged at frequencies 0.5, 1, 2, 4 kHz.[2] Table 1 provides the different grades of hearing impairment.[2]

Ototoxicity and its Causes

Ototoxicity refers to the harmful effect of a drug, chemical substance or heavy metal on the organ of hearing or balance, which may lead to a hearing impairment, and/or balance problems. Table 2 displays some of these substances.[3]

Table 2: Causes of Hearing Impairment and/or Balance Problems

Aminoglycosides	Gentamicin, streptomycin, kanamycin, amikacin, tobramycin, neomycin, netilmicin, polymyxin-B	
Macrolides	Erythromycin, azithromycin, clarithromycin	
Loop diuretics	Furosemide, bumetanide, ethacrinic acid	
Salicylates		
Antimalarials	Quinine, chloroquine (high dosage)	
Non-steroid anti-inflammatory drugs	Naproxen, indomethacin (no definite findings)	
Anti-neoplastic drugs	Cisplatin, bleomycin, carboplatinum	
Chelating agents	Desferoxamine	
Topical otological preparations	***Antibiotic solutions:***	Neomycin Aminoglycosides Polymyxin-B Chloramphenicol Fosfomycin
	Anti-inflammatory:	Propylene-glycol, hydrocortisone
	Antiseptic:	Chlorohexidine, povidone-iodine (?)
	Acidifying:	2% acetic acid solution (?)
Chemical agents	***Heavy metals:***	Mercury, lead (Industrial pollution, cosmetics).
	Solvents:	Toluene, styrene
	Others:	Arsenic, cobalt, cyanides, benzene, propylene-glycol, potassium bromide

Ototoxicity tends to be thought of in the context of drug administration leading to damage of the cochlea or vestibular portion of the inner ear, causing transitional or permanent sensorineural hearing loss (SNHL) and/or vertigo. Antibiotics, diuretics and anti-malarial pharmaceuticals have been implicated as potentially toxic to both the auditory and vestibular systems. Kanamycin and neomycin are perhaps the most alarming ototoxic drugs at this time.[4] This report will later discuss and evaluate the current status of ototoxicity due to these substances. This will be accomplished by assessing and reviewing different literature written about the use of these chemicals in various regions of the world, and in particular, the developing world.

While aminoglycosides have been largely replaced over the last decades by modern antibiotics with fewer side effects, they remain a mainstay in medicine. In fact, they may be the most commonly used antibiotics worldwide, chiefly due to their use in developing countries. Their high efficacy, coupled with extremely low cost, frequently makes aminoglycoside antibiotics the only affordable drugs. Furthermore, since tuberculosis is on the rise worldwide, particularly in low income countries, aminoglycoside use will not be reduced.[5]

Streptomycin and kanamycin are part of the recommended regimen of the World Health Organization against tuberculosis, and their widespread use makes these antibiotics a major cause of preventable hearing loss in the world today.[5] Given that most drug-induced hearing loss is caused by the prescription of ototoxic drugs, one should assume that preventive measures could be taken effectively. Minja makes reference to the preventability of deafness due to ototoxicity, despite its variety of causes.[1] Suggestions will be made regarding methods and strategies for the prevention of ototoxicity in developing countries.

Another report refers to the extensive use and abuse of aminoglycosides and how they are a major concern.[3] It suggests that the most common cause of hearing impairment from ototoxic damage by drugs is due to injectable aminoglycosides. It is also implied that gentamicin is cheaper than newly available alternatives and, hence, is more widely available. Additionally, WHO recognises that the global resurgence of tuberculosis is leading to greater use of streptomycin.[3] For example, in South Africa, streptomycin and kanamycin form part of the drug regimen administered to multi-drug resistant tuberculosis (MDR-TB) sufferers.[6] One of the aims of this report is to ascertain the extent to which these antibiotics are being abused.

This report will also consider the many agents within the workplace, particularly within heavy industry, that can potentially bring about chemical trauma to the ear. Examples include xylene, toluene, mercury, tin, lead and carbon monoxide.[4]

The meaning of a developing country is a final point of importance in this introduction. It has been defined by the World Bank Income Groupings, in which the main criterion for classifying economies is gross national income (GNI) per capita. Based on its GNI per capita, every economy is classified as low income, middle income (subdivided into lower middle and upper middle), or high income. Table 3 identifies some of the developing countries that will be discussed in this report, and others that are noteworthy.[7]

Table 3: High Income, Upper Middle Income, Lower Middle Income and Low Income Countries

High Income	Upper Middle Income	Lower Middle Income	Low Income ($765 or less)
Australia	Argentina	Brazil	Bangladesh
France	Barbados	China	Ghana
Germany	Botswana	Colombia	India
Hong Kong	Chile	Indonesia	Kenya
Ireland	Costa Rica	Iran	Malawi
Italy	Czech Republic	Morocco	Nepal
Japan	Latvia	Namibia	Nigeria
Korea	Lebanon	Paraguay	Pakistan
Netherlands	Malaysia	Peru	Sudan
Singapore	Mauritius	Philippines	Tanzania
Switzerland	Mexico	South Africa	Uganda
UK	Oman	Sri Lanka	Zambia
USA	Poland	Thailand	Zimbabwe

To summarise this introduction to ototoxicity-induced deafness, it is important to note that the global magnitude of the problem is not accurately known and that there is a great need for more detailed research on ototoxicity.

Discussion and Results

This report will now analyse and review the literature found. It will discuss the current status of ototoxicity in developing countries by comparing results from clinical studies carried out. It will then assess the disagreements, strengths and weaknesses of various papers. The problems facing people in developing countries will also be considered in depth.

The fact that aminoglycosides and other drugs, such as antimalarials, can produce ototoxicity has been well established in both humans and experimental animals. The ototoxicity can take the form of damage to the auditory system or the vestibular system, or both.[8] In one study, Tange et al showed that malaria patients experienced adverse effects related to ototoxicity induced by quinine: 9 had impaired hearing, 11 tinnitus, 8 had feeling of pressure on the ears and 4 felt giddiness.[9] While malaria may cause deafness, the drugs used in the treatment are potentially ototoxic. Quinine is the drug of choice in the treatment of chloroquine resistant falciparum malaria in the developing world. Minja observed 354 pupils at a School for the Deaf in Dar es Salaam, of which five had become totally deaf following intravenous infusion of quinine.[1] Table 4 displays the distribution of the 354 children according to causes of deafness. Ototoxicity can be seen as a cause in 20 % of cases.[1]

Studies on the ototoxicity of quinine in humans are scarce, however, and there are still some questions about the reversibility of quinine induced hearing loss. Nevertheless, quinine induced ototoxicity in patients and volunteers appears to be largely, if not completely, reversible.[9] The salicylates and diuretics produce

Table 4: Causes of Deafness in Dar-es-Salaam

Causes of deafness	No. of children
Meningitis	76
Ototoxicity	66
Mumps	53
Congenital	36
Otitis media	28
Measles	13
Febrile convulsions	5
Unknown	77

temporary hearing loss that may be reversible, fully or partially, when the patient is taken off the medication.[4]

Conversely, aminoglycosides, such as streptomycin and kanamycin, cause the destruction of outer hair cells and hearing changes are most likely irreversible.[6] These antibiotics alerted the medical community and the public more than any others with regard to the ototoxic side effects of medications. This is despite the fact that at the time of its introduction, streptomycin was the long-sought cure for tuberculosis.[5] Sixty cases treated with streptoduocin and sixty cases treated with streptomycin were studied clinically and by various tests in Kanpur, India to find their ototoxicity. It was established that 25 % of the patients on streptoduocin (mixture of streptomycin and dihydrostreptomycin) developed ototoxicity compared to 10 percent on streptomycin. Table 5 summarises the findings on the incidence of ototoxicity as a result of streptoduocin and streptomycin administration.[10]

It can be noted that the toxicity of streptomycin is almost exclusively directed against the vestibular system, whereas, dihydrostreptomycin (a derivative which is chemically different in only one position of the molecule) can cause irreversible hearing loss.[5,6,10]

In South Africa, streptomycin and kanamycin form part of the drug regimen administered to MDR-TB sufferers. In the Western Cape, the incidence of ototoxicity varies between 0-20% depending on the type of aminoglycoside.[6] As in most developing countries, acquired causes of deafness and hearing impairment are also common in Tanzania. Minja reports that gentamicin and streptomycin, prescribed for treatment of septicaemia and tuberculosis respectively, was a cause of deafness in 18.6% of cases.[1] Other aminoglycosides show varying ototoxic effects. Gatell et al showed that slight or mild auditory toxicity developed in 42.1% of patients given tobramycin and 35.2 percent of those given amikacin.[11]

Despite the lack of data on deafness in developing countries, the ototoxic effect of drugs such as aminoglycosides is clear to see. However, there are disagreements between reported incidences of ototoxicity-induced deafness. For example, reports on the toxicity of streptoduocin have been contradictory.[10]

The discrepancy between the incidence rates can be attributed to the criteria used to define ototoxicity by different writers. Most studies consider ototoxicity to have occurred if at any time after a base-line audiogram has been obtained, an increase occurs in the auditory threshold of 15dB or more.[8] Yet, one study describes criteria for auditory dysfunction as a hearing loss greater than 10dB [10] and others use a ≥20dB change in threshold.[11,12] It is important to note the different definitions for ototoxicity presented by clinical studies in developing countries.

The disagreements between papers can also be accounted for by referring to the patients used in the studies. Screening by questionnaire, otoscopy and tympanometry has been used,[12] whereas Minja relied on the policy of admission to a deaf school in Dar es Salaam.[1] Another study carried out a loudness balance test and a difference limen test (a test of loudness perception) before recruiting,[10] which may have been insufficient. A gold standard screening process recruited patients with normal hearing from a TB-hospital in the Western Cape, after consent was received, and treated them with streptomycin and kanamycin.[6]

In a number of developing countries, it is reported that sub-standard drugs are readily available. After collecting 96 samples of chloroquine from Nigeria and Thailand, the results indicated that 36.5% were sub-standard.[13] Not only does this imply discrepancies in clinical studies, but this may, in itself, be a cause of ototoxicity in developing countries.

Following the industrial revolution, new health hazards appeared, and industrial solvents, chemicals and pollutants became a new category of environmental factors causing hearing loss.[5] For example, in Colombia, environmental causation was found to be a cause of deafness in 33.8% of cases.[14] Most notable among these chemicals, and of concern today, are solvents such as organotoxins or toluene, but also carbon monoxide and a number of lesser-used chemicals which can adversely affect the hearing and balance functions of the inner ear.[5] It is now known that certain organic solvents in industry are ototoxic when inhaled in excess. They may produce brain damage involving the vestibular pathways and the inner ear directly. One must keep in mind agents within the workplace, as well as medications prescribed by health professionals. There is one further area which may be a much greater cause of ototoxic hearing loss than has been recognised up to now - the synergistic action of noise exposure and inhaled volatile organic substances.

As with occupational noise, many developing countries have little or no legislation to prevent critical exposure to toxic substances. Regulations that do exist are poorly enforced and implemented, and many workers remain ignorant of such problems.[4]

A large proportion of hearing impairment related to ototoxic drugs results from their inappropriate or indiscriminate use by health care providers.[3] In Cambodia,

Table 5 : Ototoxicity after Streptoduocin and Streptomycin Treatment

Group	Total number of patients	Auditory toxicity	Vestibular toxicity	Auditory and vestibular toxicity	Total
Streptoduocin	60	10	-	5	15 (25%)
Streptomycin	60	-	6	-	6 (10%)

as in other developing countries, there is a disturbing tendency for misuse of antibiotics by certain practitioners - for example, the use of antibiotics to prevent infections rather than treat established disease, treatment of untreatable infections, treatment of infections of undetermined origin, without adequate biological knowledge, and frequently improper dosage. Not only does this malpractice encourage increased microbial resistance, but it also raises the potential for ototoxic effects from those drugs that are dangerous to the ear.[3,4] In Tanzania, however, these drugs are controlled and strictly available on prescription only, although one study notes that situations arise when the use of drugs (gentamicin and streptomycin) is required in the absence of any substitute.[1]

Health care professionals are not only to blame. Health authorities, in general, can also be put to shame. To date, out of 122 institutions in the Western Cape, South Africa, where aminoglycoside treatment is provided to TB sufferers, ototoxicity monitoring takes place at only one.[6] The injudicious use of drugs with ototoxic side-effects can also be attributed to self-diagnosis and self-medication. The easy availability of these drugs 'over the counter' and without a physician's prescription favours self-medication with potentially harmful drugs.[3]

The ototoxic potential of drugs should be stressed during training of staff, with regular refresher courses to update relevant knowledge.[3] This approach is already demonstrated in Dar es Salaam where all health workers are taught about the potential hazard of using these drugs during pregnancy and in treating trivial infections.[1]

It is well known that the use of aminoglycoside antibiotics carries a risk of damage to the cochlea. In spite of the introduction of new classes of antibiotics, the aminoglycosides still remain primary agents of choice in treating serious gram-negative infections.[12] Gatell et al also refer to the fact that despite the introduction of new cephalosporins and penicillins, aminoglycosides still have their place amongst treatment options.[11] Their low-cost to developing countries is the reason for this. Along with their effectiveness against gram-negative bacteria, this advantage has led to the persistence of aminoglycoside use, especially in countries like South Africa.[6] In some developing countries, the government infrastructure is grossly deficient, unable to provide the high quality, high volume health care services which can cope with the many ototoxicity-related health problems.[4]

Hearing loss due to ototoxicity is generally irreversible but avoidable in most instances, given proper preventive action through controlled use of drugs in the health care system and by consumers.[3] Minja's findings indicate that most (75.8%) of the causes of acquired deafness are preventable through immunisation, early diagnosis and proper treatment of ear infections and avoidance of prescribing ototoxic drugs.[1] The World Health Organization reports that there are no restrictions in most developing countries limiting the availability of drugs causing ototoxicity.[3]

In one study, deafness due to ototoxicity is substantial, yet preventable at primary and secondary levels of health care. The alarming rate of deafness due to the use of ototoxic drugs calls for a deliberate policy to create awareness among prescribers and the public to avoid these drugs as much as possible.[1] In China, aminoglycosides are available with or without prescription; in India, there are strict rules for their delivery, but regulations are not enforced.[3] Legislation should be introduced in countries where it does not yet exist, and, where legislation exists, it should be strictly enforced.

Conclusion

The conclusion to this report considers future problems facing people in low-income countries and summarises what needs to be done to resolve them. It refers to the limitations and controversies in some of the studies carried out in developing countries.

The global magnitude of the problem of hearing impairment or deafness due to ototoxicity is not accurately known, but it is probably responsible for 3-4% of all deafness in children in developing countries.[3] Childhood deafness has two serious consequences; delayed speech and language development, leading to the need for special education. These problems are worse in low-income countries where economic difficulty, human and material resources to enable early diagnosis and appropriate rehabilitation are lacking.[1]

Thus, we need to explore more efficient ways of monitoring, in order to do more with limited resources. Only then will ototoxicity be detected early and the negative side-effects avoided or alleviated.[6] Encouragingly, Schacht and Hawkins believe there is real hope that ototoxicity can be conquered. Simple over-the-counter supplements and medications will become part of an inexpensive pharmacological protection to render drug-induced hearing loss a medical concern of the past.[5]

The lack of general knowledge, however, about the risk of ototoxic damage and insufficient public education on ototoxicity is a great obstacle to preventive action. The aim of public education should be to provide individuals with information about the use of medicines in an appropriate way.[3]

In reading ototoxicity-related scientific papers, the existence of limitations and controversies has become apparent. For example, one study reports that for the first 10 courses of aminoglycosides, the therapeutic benefit could be considered to outweigh the risk of cochleotoxicity.[12] The result of this high-dose therapy is contradictory and not in keeping with many other studies.

One particular drawback is the general lack of concern or ignorance towards ototoxicity-induced deafness in developing countries. Small doses of quinine, for example, can cause tinnitus in susceptible persons. However, because of the lack of clinical significance, the interest in the ototoxicity of quinine has been subdued.[9]

In summary, it is important to realise that this report and the studies cited represent only a fraction of the true extent of deafness caused by ototoxicity in developing countries. More research is needed to find out if there is any substance that could reduce damage from ototoxic drugs during their administration. More importantly, national surveillance systems are needed in most developing countries to set up a monitoring system for ototoxic damage.[3]

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SOLVENT EXPOSURE AT THE WORKPLACE: WORKERS' HEARING IN JEOPARDY

Adrian Fuente BSc

Doctoral Student
Division of Speech and Hearing Sciences
The University of Hong Kong
5F Prince Philip Dental Hospital
34 Hospital Road
Hong Kong

Email: afuente@hkusua.hku.hk

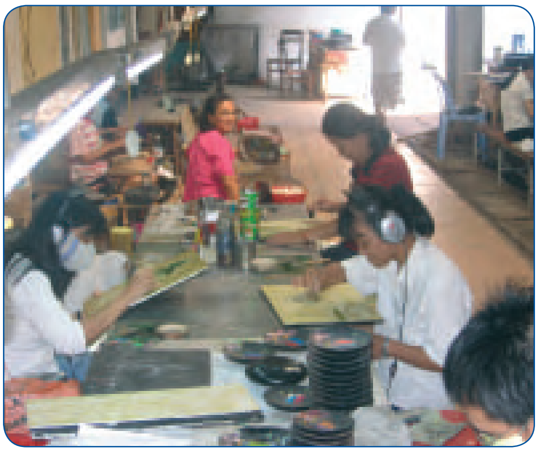
Modern industry no doubt brings enormous benefits to our society. New industrial techniques accelerate and improve production. New machinery improves the efficiency of manufacture, often creating better quality products, at a better price for consumers. In developed countries, the use of high technology machinery, as well as the introduction of less toxic raw materials, has allowed workers to have less contact with hazardous chemicals. However, in developing countries newer technology is not always available and non-toxic chemicals are not always used, due to economic factors.

Since the start of the industrial revolution, many raw materials have been identified as dangerous for human health. Organic solvents fall within this category. It has been widely demonstrated that solvents may adversely affect the central and peripheral nervous system and other body structures. More recently, the ototoxic properties of solvents have also been uncovered by a number of different research groups.[1,2] Despite this new scientific knowledge, audiologists, industrial hygienists and occupational safety and health professionals have been focused on noise as the main agent capable of inducing hearing loss in the workplace. In developed and some developing countries, workers exposed to solvents receive epidemiological surveillance programmes focused on the effects of these chemicals on the central nervous system. Currently, in most countries not much attention is paid to the ototoxic properties of solvents. This is surprising, considering the diverse range of solvents in daily use.

Solvents and Their Effects

Solvents are now widely used in industrial processes such as in automotive and aviation fuels, plastics industries, as a thinner for paints, lacquers, coatings, and dyes - in the manufacture of artificial leather, detergents, medicines, perfumes, fabric and paper coatings, photogravure inks, spray surface coatings and insect repellents (Table 1). In many occupational settings, workers are often exposed to a combination of solvents and other hazardous agents such as noise.[3]

Focusing on the ototoxic properties of solvents, studies have demonstrated that solvents such as toluene, styrene, and xylene may induce damage on the peripheral auditory system (the cochlea). This means that these chemicals may



A factory in Vietnam

Photo: Adrian Fuente

Table 1: Solvents: Effects on Hearing and Recommendations to Avoid Hearing Loss

Industrial processes where solvents are used
Toluene: printing, rubber manufacture, wood stains and varnishes, and footwear manufacture.
Styrene: pulp and paper manufacture and in plastics, resins, coatings, and paint manufacture.
Xylene: paint manufacture, paint stripping, paper coating, pesticide manufacture, pharmaceuticals manufacture and printing.
Effects of solvents on hearing
Peripheral auditory system: adverse effects on hearing thresholds in a wider range than 4000-6000 Hz.
Central auditory system: difficulties in discriminating speech, especially in the presence of background noise.
Synergism between solvents and noise
Recommendations to avoid solvent-induced hearing loss
✓ Alert both employers and employees to the hazardous effects of solvents on hearing.
✓ Replacement of solvents with less toxic compounds such as water.
✓ Implementation of hearing conservation programmes for all workers exposed to solvents, independent of the noise level in the workplace.

adversely affect hearing thresholds. The audiometric pattern of solvent-induced hearing loss is not necessarily the same as that seen in noise-induced hearing loss. The latter typically shows a 'noise notch' around 4000-6000 Hz, while in solvent-induced hearing loss a wider range of high frequencies may be affected. Other studies have demonstrated that solvents may also induce damage to the central structures of the auditory system. This means that an affected person may not necessarily have poor hearing thresholds but he or she may encounter difficulties in understanding what others say, especially in the presence of background noise. The effect of solvents on the central auditory system may be related to the ability that solvents have to penetrate into lipid structures.

Hearing Health Care and Solvents

The multiple effects of solvents on the auditory system make it difficult to assess and identify workers with solvent-induced hearing loss, especially when hearing health care professionals are unaware of the problem. This scenario becomes more complex still when workers are also exposed to other oto- or neurotoxic agents such as noise. Noise has been extensively recognised as an agent that may induce hearing loss when exposures are above certain limits (85 dBA TWA*). A number of studies have suggested a synergistic effect on human hearing when noise and solvent exposure occur together. In other words, the combined effect of solvents and noise may induce auditory damage more severe than the effect that each of these agents may have by itself. Unfortunately, this combination of agents frequently occurs in factories, especially in developing countries. The high intensity of noise produced by non-renovated machinery plus the presence of solvents in the environment, together with the absence of protective equipment or measures to diminish solvent and noise exposure, is commonly seen. Workers may not be exposed necessarily to high intensities of noise, but when solvents are also present, an adverse effect on hearing may still be induced. Hearing conservation programmes should be conducted for all workers exposed to solvents and noise even when the latter is less than 85 dBA TWA. This type of programme should include not only the early detection of hearing loss induced by solvents but also implement the necessary measures to avoid workers being exposed to high intensities of noise and to solvents above the recommended levels.

The Situation in Asia

At present, there is little consideration of the ototoxic properties of solvents. Many countries still use solvents without control. In Asia, for instance, many factories use high concentrations of solvents in different industrial processes. The footwear industry is a good example. Asia is the dominant producing region in the world. Its contribution to world production of shoes has steadily increased from 51% in 1985 to 63% in 1993, and 77% in 1999, with China by far the first in the world. In China alone, millions of footwear industry workers are likely to be affected.[4] Factories of many famous brands have moved to Asia due to the cheaper costs of labour. However, from the occupational safety and health point of view, the factories are not an improvement. Many of these factories do not control the levels of solvents that they use, and the environmental concentrations of these chemicals at workplaces may be totally unknown. Even worse, regulations concerning the use of ventilation systems and the provision of masks, gloves or other personal protective equipment do not exist in many of these factories. In addition, highly hazardous solvents such as benzene are still being used in many factories of Asian countries. All this makes such factories potentially hazardous for workers. Taking into account this scenario, plus the fact that solvent-induced hearing loss is a relatively recently discovered pathology and, therefore, not widely known among health care professionals, even for those who specialise in occupational medicine, the idea of regular hearing conservation programmes in workers exposed to solvents is still a new one in countries such as China. Recently, new guidelines and standards have emerged in some non-Asian countries that consider the inclusion of workers exposed to ototoxic agents in hearing conservation programmes. These regulations should be considered as a reference by legislators in Asian countries for implementing similar programmes.

**TWA means 'time-weighted average':*
- The average exposure to a contaminant or condition (such as noise) to which workers may be exposed without adverse effect over a period such as in an 8-hour day or 40-hour week. See http://www.answers.com/topic/time-weighted-average

What We Can Do

It is within the scope of health care professionals to alert both employers and employees about the hazardous effects of solvents and so help them to avoid solvent-related diseases (Table1). It is the role of the scientific community to work towards the introduction of new non-toxic materials which have similar or better properties than toxic solvents, and, thus, help employers to adapt their factories to use these new raw materials. One example is the possibility of replacing solvents such as toluene or benzene with water in some industrial processes. It is the responsibility of hearing health care professionals to be aware of the adverse effects of solvents on hearing, especially when they co-exist with noise. Hearing conservation programmes should be implemented for all workers exposed to solvents only, noise only or solvents combined with noise. Research so far has consistently identified solvents as oto- and neurotoxic agents, it is now the responsibility of all of us - researchers, clinicians, workers, and employers - to take the necessary actions to avoid solvent-induced hearing loss in workers.

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OTOTOXICITY: A CANADIAN VIEW

Peter W Alberti
MD MB PhD FRCSC FRCS

Professor Emeritus
Toronto
Canada

Ototoxicity continues to be a significant cause of hearing loss and vestibular dysfunction throughout the world. Just when one thinks the problem has been virtually eliminated, another cause appears.

Ototoxicity in Canada

The present causes of ototoxicity in Canada include some long standing ones, such as aminoglycosides, although the injudicious and inappropriate systemic use is rare. However, the systemic or local absorption of topically applied aminoglycosides continues to produce hearing loss and imbalance. They may be given as topical ointment or ear drops.

Until quite recently, the most commonly used ear drop in Canada was a gentamicin steroid mix, usually safe in the presence of a disease thickened middle ear mucosa, and if given for a short time, but potentially toxic with longer use, especially in the presence of a thin middle ear mucosa. This was sufficiently serious that Health and Welfare Canada put out a warning notice in 2002 about the hazards of prolonged use. They are being replaced by safer ciprofloxacin, which is, however, up to four times as expensive. Gentamicin containing ointments may be used to treat burns or infected skin sites around catheters, such as are used for intra-peritoneal dialysis. Any renal compromise potentiates the ototoxic effects of aminoglycosides because they will not be excreted, leading to cumulative high (and toxic) blood levels. Quite a few elderly patients on temporary renal dialysis recover from their renal shutdown only to find themselves ataxic, and even with bobbing oscillopsia. (Oscillopsia - the sensation that viewed objects are moving or wavering back and forth).

Immigrants from China and Eastern Europe

Canada is a land of immigrants, no more so at present than Chinese. It is not well recognised that some Chinese suffer from an (m)RNA transmitted hereditary susceptibility to the ototoxic affects of aminoglycosides. If it has not already done so, it will inevitably lead to more unexpected vestibular disturbance and hearing loss. A small but steady number of immigrants with ototoxic hearing loss were treated with streptomycin or gentamicin in Eastern Europe for long periods - for infections which in Canada would have been treated by safer (but costlier) antibiotics. Likewise, immigrants arrive with hearing loss caused by quinine and other antimalarial treatment, either those cured of cerebral malaria, or long time regular consumers, often retired regular soldiers.

Medications

Aminoglycosides are still used to treat severe infections such as caused by motor vehicle accidents or gunshot wounds. The writer has seen a patient with bobbing oscillopsia which was the result of treatment by gentamicin for multiple bowel perforations caused by bullets. Here, although the risk was known, life saving therapy took precedence over saving the sense of balance.

New medications may bring their own problems. This was seen with cisplatin, a useful but initially unrecognised ototoxic agent; its very success in treating malignant disease led to the discovery of long term side effects of the therapy. This still may not be recognised and the patients may suffer unnecessarily from a hearing loss which could be rehabilitated with a hearing aid.

Noise and Organic Solvents

There is one further area which may be a much greater cause of ototoxic hearing loss than has been recognised up to now - the synergistic action of noise exposure and inhaled volatile organic solvents. It is now known that certain organic solvents used in industry are ototoxic when inhaled to excess. They may produce brain damage involving the vestibular pathways and they may involve the inner ear directly. The most used chemical is toluene, but workers in the petrochemical industry are also at risk, as are those using some of the adhesives which have replaced riveting in the aerospace industry. It has been shown that toluene, certainly, and other solvents may act synergistically with noise, so that safe levels of one may still lead to hearing loss if the worker is exposed to both simultaneously, as often happens. The loss is similar to that produced by excessive noise exposure, but greater than would be expected from the noise dose alone. ❑

MOLECULAR DIAGNOSIS OF MITOCHONDRIAL GENES: EARLY DETECTION AND PREVENTION OF AMINOGLYCOSIDE OTOTOXITY

Min-Xin Guan PhD

Division of Human Genetics and Center for Hearing and Deafness Research
Cincinnati Children's Hospital Medical Center
Cincinnati, Ohio 45229
USA

E-mail: min-xin.guan@chmcc.org

ABSTRACT

Aminoglycosides such as gentamicin, streptomycin, kanamycin and tobramycin are clinically important drugs. The use of these drugs can frequently lead to irreversible hearing loss. Aminoglycoside ototoxicity accounts for a significant portion of deafness. In familial cases of ototoxic deafness, the aminoglycoside hypersensitivity is often maternally transmitted, suggesting that the mutation(s) in mitochondrial DNA is the molecular basis of this disorder. Mutational analysis has led to the identification of several ototoxic mutations in mitochondrial 12S rRNA. In particular, the A1555G and C1494T mutations account for significant cases of aminoglycoside ototoxicity. The A1555G or C1494T mutation creates the binding sites of the highly conserved A-site of 12S rRNA and make the secondary structure of this RNA more closely resemble the corresponding region of bacterial 16S rRNA. Thus, these mutations facilitate the binding of aminoglycosides, thereby accounting for the fact that the exposure to aminoglycosides can induce or worsen hearing loss in individuals carrying these mutations. Therefore, these data have been providing valuable information and technology to predict which individuals are at risk of ototoxicity, to improve the safety of aminoglycoside antibiotic therapy, and eventually to decrease the incidence of deafness.

Hearing Loss; Gene Aberrations and Ototoxic Drugs

Hearing loss is one of the most common human sufferings, affecting one in 1000 newborns.[1] Hearing loss can be caused by gene aberrations or environmental factors, including ototoxic drugs such as aminoglycoside antibiotics.[2] These antibiotics, such as gentamicin, streptomycin, kanamycin and tobramycin, are clinically important drugs. They are particularly active against aerobic, gram-negative bacteria and act synergistically against certain gram-positive organisms. In developed countries, these drugs are mainly used in the treatment of hospitalised patients with aerobic gram-negative bacterial infections, particularly in patients with chronic infections such as cystic fibrosis or tuberculosis. However, in developing countries, aminoglycosides are more routinely used, even for relatively minor infections. The use of these drugs can frequently lead to toxicity, which involves the renal, auditory and vestibular systems.[3,4,5] The renal impairment is usually reversible, but the auditory and vestibular ototoxicity is frequently irreversible. Although all of the aminoglycosides are capable of affecting cochlear and vestibular functions, some (streptomycin and gentamicin) produce predominately vestibular damage, while others (neomycin and kanamycin) cause mainly cochlear damage. Tobramycin affects both equally.[4]

Aminoglycosides; Dosage and Age of Patients

In the United States, almost 4 million courses of aminoglycosides are administrated annually.[6] It is estimated that at least 2-5% of patients treated with these antibiotics develop clinically significant hearing loss.[7,8] The problem of ototoxic side effects is more acute in developing countries, where highly effective and low cost drugs such as aminoglycosides are often prescribed without adequate monitoring. Due to the widespread use of these antibiotics, 20-30% of two cohorts of Chinese deaf populations could be due to the administration of various aminoglycosides.[9,10] The type and doses of aminoglycoside medication, the length of treatment, and age at the time of drug administration may relate to the severity of hearing impairments in some subjects. At very high dose of these drugs, most individuals will exhibit toxicity. By contrast, some patients developed aminoglycoside-induced hearing loss after treatment with conventional doses, even one dose of a drug, over a short period.

Maternal Transmission and Mutations

In familial cases of ototoxic deafness, the aminoglycoside hypersensitivity is often maternally transmitted.[9,10] In these families, a woman carrying the trait will have inherited the trait, but only a female can pass the trait on to the subsequent generation. The maternal transmission of deafness suggested that mutation(s) (changes in the gene) in mitochondrial DNA (mtDNA) could be the molecular basis for this susceptibility.[5,11] Mutational analyses of the mitochondrial genome of families with maternally transmitted aminoglycoside ototoxicity have led to the identification of several ototoxic mtDNA mutations, especially the A1555G and C1494T mutations in the 12S ribosomal RNA (rRNA).[11,12,13] Both A1555G and C1494T mutations are located at the highly conserved aminoacyl-tRNA binding site (A-site) of the small ribosomal sub-unit, which is an important locus of action for aminoglycosides.[14,15] In human 12S rRNA, the 1494C and 1555G bases are in apposition to each other but do not form a base pair. However, if the C at position 1494 is mutated to T, or the A at position 1555 is mutated to a G, then a base pair is formed extending the adjacent stem by one nucleotide and making the mitochondrial ribosome more bacteria-like (Figure 1). This new G-C or A-U pair in 12S rRNA creates a binding site for aminoglycosides, which facilitates the binding of these drugs.[12,13,16] In fact, the human cell lines carrying the A1555G or C1494T mutation exhibited the sensitivity to aminoglycosides.[12,17]

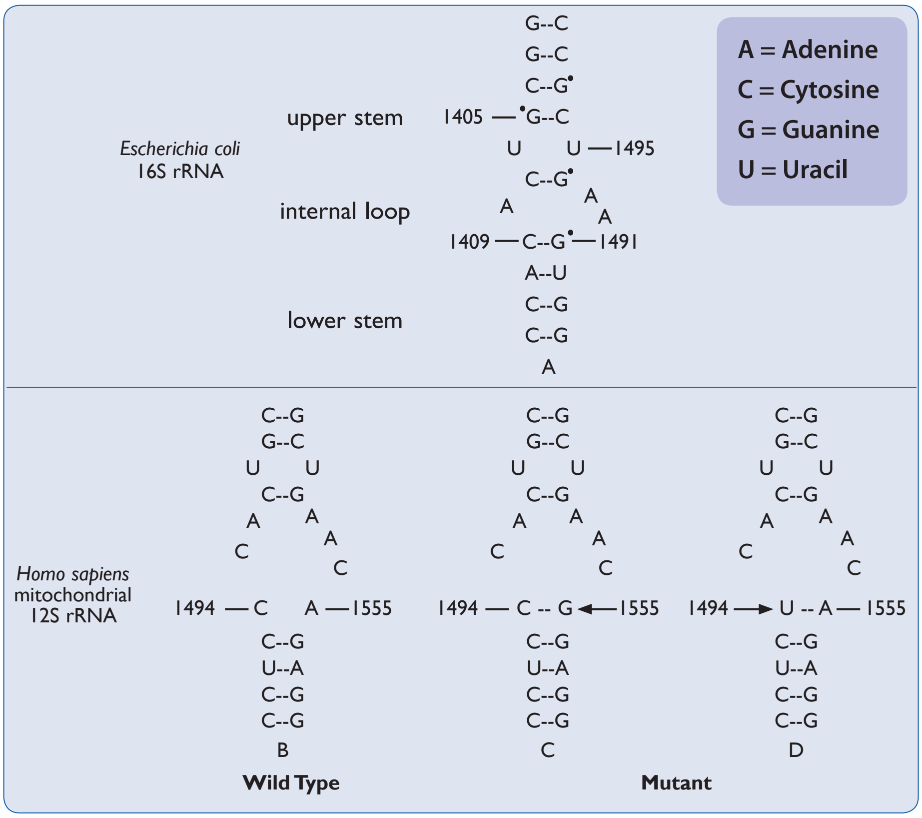

Fig. 1: The sites of the A1555G and C1494T mutations in the decoding region of mitochondrial 12S rRNA.

Mutations, Aminoglycosides and Hearing Loss

The A1555G mutation has been found in many families and sporadic cases worldwide,[12,18,19,20] while the C1494T mutation has been reported in Chinese and Spanish families.[13,21] These mutations account for approximately 20% of deafness patients with a history of exposure to aminoglycosides.[11,22,23] In the absence of exposure to aminoglycosides, the A1555G or C1494T mutation also produces a non-syndromic hearing loss.[13,18] Matrilineal relatives among families or within families carrying these mutations exhibited variable penetrance and expressivity, including severity and age of onset in hearing impairment, ranging from profound congenital deafness, to severe and moderate progressive hearing loss of later onset, to completely normal hearing.[12,13,18,19,20] In some families carrying these ototoxic mutations, only one or a few matrilineal relatives suffered from deafness, while the majority of members in these families exhibited normal hearing.[20] The incomplete penetrance of hearing loss and the mild biochemical defects indicated that the A1555G or C1494T mutation itself is insufficient to produce the deafness phenotype.[11] Therefore, other modifier factors, such as aminoglycosides, are required for the phenotypic manifestation of the A1555G or C1494T mutation. Aminoglycosides, which are concentrated in the perilymph and endolymph of the inner ear,[24] can worsen mitochondrial dysfunctions in cochlear cells in susceptible subjects carrying the A1555G or C1494T mutation. As a consequence, exposure to aminoglycosides leads to tissue specific defects in those cells, thereby inducing or worsening hearing loss in individuals carrying these ototoxic mtDNA mutations. In particular, those children, under ten years old, carrying these ototoxic mtDNA mutations will develop severe or profound hearing loss if given these drugs, even at conventional doses.

Mutations in mitochondrial 12S rRNA are one of the molecular bases for aminoglycoside ototoxicity. Two ototoxic mitochondrial 12S rRNA mutations account for approximately 20% of cases with aminoglycoside ototoxicity.[11,21,23] These data have significant clinical and social impacts. However, the ototoxicity associated with these mutations can be preventable through a combination of evaluating family history and molecular analysis of 12S rRNA gene in susceptible individuals. Every individual, prior to an administration of aminoglycosides, should be examined for a family history. If a member(s) of a family suffered from aminoglycoside-induced deafness, others should be screened for 12S rRNA mutations. Those, who are positive for 12S rRNA mutations, should be warned that they are at risk of aminoglycoside ototoxicity and avoid the use of these drugs. Therefore, genetic and molecular approaches can help us predict which individuals are at risk of ototoxicity, improve the safety of aminoglycoside antibiotic therapy, and eventually decrease the incidence of deafness.

Acknowledgement

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THE NEED OF A PROGRAMME FOR THE PREVENTION OF HEARING IMPAIRMENT IN BENI STATE, BOLIVIA

Diego J Santana-Hernández MD

Foundation Totai
Casilla 158
Trinidad-Beni
Bolivia

E-mail: santanadj@coteautri.net.bo

Dr Diego Santana-Hernández is an ENT Surgeon and General Practitioner. Since 2000 he has worked as a missionary doctor in Bolivia, commended and supported together with his wife Joanne by Carrubbers Christian Centre in Edinburgh, Scotland. He presently chairs Foundation Totai, a community orientated charity.

Introduction

In the Bolivian Amazonian prairies, Beni State has a surface area of 213,564 km^2 (approximately half the size of Spain). It has a dispersed population of 406,982 inhabitants, of which 89,613 live in Trinidad, capital of Beni.[1] Bolivia is the poorest country in South America (64% of the people live below the poverty line). In 2004, GNP per capita was $US 1051 (871€). Direct foreign investment exceeds public investment. $US 6.5 per person per year is provided for health care.[1]

Life expectancy is 62.9 years (men: 61.3 years; women: 64.5 years). Bolivia has the lowest number of deliveries attended by health professionals and the highest maternal mortality in South America: 234 for every 100,000 live births. (In the year 2000, the World Health Organization reported an average of 20 per 100,000 live births in the developed countries). Hospital early neonatal mortality (during first 7 days of life) and infant mortality (under 1 year) are 10 and 54 per 1000 live births, respectively.[1]

Justification

1. Absence of programmes for promotion of ear and hearing health

In a preliminary population based survey, out of a sample group of 658 school children (age 7 to 18), 105 presented with ear or hearing problems (16%). Impacted wax in the ear canal was the main finding, followed by chronic otitis media.

2. Absence of programmes for the prevention of hearing impairment

There are no population based studies to determine either incidence or prevalence of hearing impairment in Bolivia, nor is there a register of people with hearing disabilities. The World Health Organization, based upon investigations carried out in countries with similar characteristics, estimate that 10% of the population suffer some type of disability.[2] The Japanese International Cooperation Agency (JICA), according to investigations carried out in Bolivia in 1998, established that 9.13% of the people with disability studied suffer a disabling hearing loss according to the WHO definition.[3]

Correlating those figures to the population in Beni (0.913%), we estimate a prevalence of 3,716 persons with a disabiling hearing disability. In 2004, 8,268 births were registered in the State (out of 13,528 expected by the Instituto Nacional

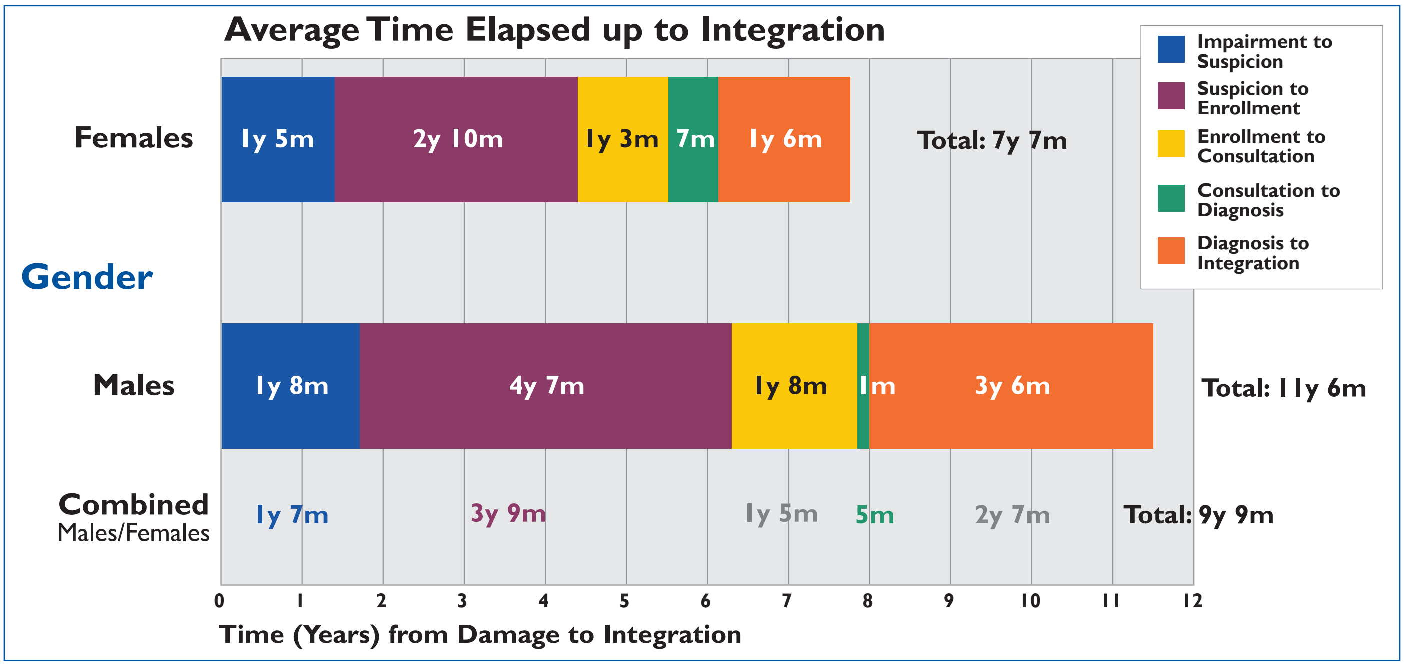

Fig. 1: Representation of the reality of stage sequence in the process of diagnosis and integration

Fig. 2: Community hearing testing in Cobija
Photo: Diego Santana

de Estadística[1]), and, of these, 2473 were hospital births in Trinidad alone. Our locally determined aetiology for hearing impairment in Trinidad (under 18 years old) is: 36% acquired, 50% congenital and 14% perinatal. Due to moderate hearing loss being largely undiagnosed in our population of reference, we have been unable to gather enough data to give a realistic estimated incidence for moderate hearing loss.

3. Absence of programmes for early detection of deafness

Early diagnosis of a hearing impairment in Trinidad-Beni (before 2 years) occurs in 7.8% of cases, and late diagnosis in 92.2%. In developed countries, 50% cases of deafness are diagnosed before age 3 years.[4] Average age at diagnosis in Trinidad is ***9 years 1 month (Females: 8 years; Males: 10 years, 4 months)***, children with a profound hearing loss being diagnosed earlier than those whose loss is severe.[5]

Study

A retrospective closed analytical study of 64 students attending the only School for the Deaf in Trinidad was performed, aiming to study the aetiology of hearing impairment and reasons for the delayed diagnosis of deafness. All the students had a chronic hearing impairment greater than 60 dB HL average for the better ear. Both aetiology and the diagnostic process (timing and investigations) were examined. In order to better evaluate such delay in reaching a diagnosis, the period from the moment when hearing impairment occurred until final integration into society took place, was divided into 5 stages:

1. Hearing impairment occurred (day of delivery in congenital cases) to moment when family suspected hearing impairment (HIO-FSHI)
2. Family suspicion of hearing impairment until medical consultation was requested (FSHI-MCR)
3. Medical consultation to definitive diagnosis of hearing impairment (MCR-DDHI)
4. Definitive diagnosis to enrollment in special school (or special support group) (DDHI-ESS)
5. Enrollment in special school to 'integration' into society (ESS-IS)

Findings

The generally accepted aetiological proportions for congenital deafness are: 50% hereditary, 25% non-hereditary and 25% idiopathic.[6,7] These differ from our study: the hereditary group is relatively smaller (31%); non-hereditary (34%) and idiopathic (34%) are greater. This finding is not unique to our population and is to be expected in an environment with a high prevalence of infectious diseases and limited methods for establishing a diagnosis.[5]

The high proportion of prematurity/low birth weight and foetal complications such as severe hypoxia in perinatal cases (89% of total), and of meningitis in acquired cases (39% of medical causes), is described by other authors, however, their relative proportion is elevated in our population. The same is true of acquired hearing impairment due to trauma, where 26% of our series contrast with the significantly lower figures of developed countries (not greater than 9%). ***Thirty-six percent (36%) of acquired hearing impairment in a mainly infant population, highlights the need to carry out programmes to educate and enable the population to prevent hearing impairment and related systemic illnesses.***[5]

Reality in Beni

In our study, average ***total time elapsed*** from the moment when hearing damage occurred until 'integration into society' (school or work) of the hearing impaired person took place is: ***9 years and 9 months.*** A significant difference exists between genders: ***males: 11 years 6 months; females: 7 years, 7 months***[5] (Figure1).

The average ***time elapsed by stage*** is:

1. HIO-FSHI (Impairment to suspicion): **1 year 7 months**
2. FSHI-MCR (Suspicion to consultation): **5 years 2 months**
3. MCR-DDHI (Consultation to diagnosis): **-1 year 10 months**
4. DDHI-ESS (Diagnosis to schooling): **1 year 5 months**
5. ESS-IS (School to integration): **2 years 7 months.**

Interpretation

There is a significant and important delay for congenital hearing impairment in stage 1: an average ***2 years 4 months*** passed before relatives suspect it (compared to 2 months, 2 weeks in acquired cases). However, the most significant delay in diagnosis happens in stage 2, ***from family suspicion to requesting specialist consultation: average 5 years, 2 months***, with a significant difference between genders: males: 6 years 3 months; females: 4 years 1 month. The negative symbol of stage 3 indicates that the norm is to enter special schooling *before* medical or audiological evaluation takes place. For a clearer interpretation of the sequence see Figure 1.

Frequently, the request for consultation takes place at the schools (special or main stream); this fact and eventuality delays definite diagnosis due to lack of referral routes to health services. As defined by Flores and Berruecos,[8] ***'It is important to distinguish between identification and diagnosis, the latter being the one which leads to the appropriate therapeutic and rehabilitation programmes.'***

We agree with other authors[9] that the ***best solution to reduce time*** elapsed from moment of hearing damage to specialist consultation (stages 1 and 2) is to ***establish systems of universal screening at the health institutions.*** This action is limited in our environment, as the Public Health Insurance, for both the mother throughout pregnancy until 6 months after delivery and for the child up to 5 years of age, is hugely underused and lacks basic provision.

Fig. 3: Donated hearing instrument (Guayaramerín)
Photo: Joanne Santana

Prevention of Hearing Impairment: Bolivia

Present Setting

Currently there are two special Schools of/for the Deaf in Beni: 'Arca Maranata' in Riberalta and IDEPPSO-Beni, Trinidad. In 2005, the first School enrolled 37 students and the second 50. This highlights the situation of the estimated 1000 children of school age with hearing disability, in the Beni, who are neither identified nor registered.

Seventy-seven percent (77%) of our study group are under 19 years old, comparable to 75% of disabled people attending rehabilitation institutions in Bolivia reported by JICA. According to M. Guevara,[10] in Bolivia, only ***1.6% of those with hearing disability are integrated*** into education and the labour market, which represents 6% of the total disabled population (or with learning difficulties) successfully integrated in the country.

Programme

The setting of a programme for the prevention of hearing impairment, with activities for primary, secondary and tertiary prevention, seems to be the way forward to alleviate the burden of hearing disability in Beni and Bolivia.

Hope

For such a programme to see the light, it will be necessary to join the efforts of resident health professionals and external aid agencies. To integrate it into the National Health Service will take some extra help. By their fruit you will recognise them. [11]

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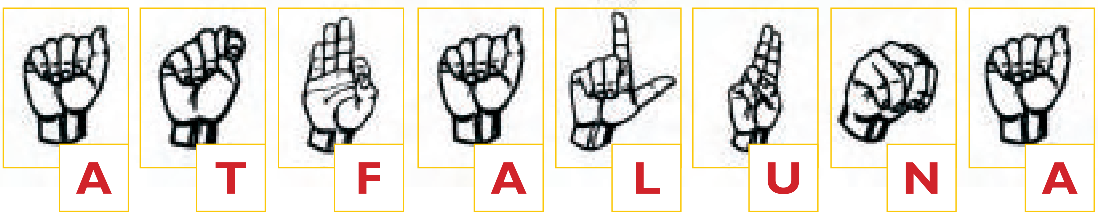
Atfaluna News Update

ATFALUNA NEWS UPDATE, JANUARY 2006



Atfaluna Society for Deaf Children
PO Box 1296 – 72 Philisteen St
Gaza City
The Gaza Strip
The Palestinian Authority

Email: atfaluna@atfaluna.net

Very Dear Friends

It's a warm and sunny morning here in the Gaza Strip, and already there are subtle signs of spring in the air. The clear blue skies made for a perfect day to be outdoors, but few cars and even fewer people are on the streets. It's post-election Palestine and the mood is one of uncertainty and apprehension. Years of military occupation and political unrest have instilled in most an uncanny sense of when it's just best to stay indoors and wait it out.

The children are back to school today following their three-week mid-year break. They are clearly happy to be back where their friends, teachers, and staff members 'speak' their native tongue, Palestinian Sign Language. This morning the playground was a sea of hands of all sizes…hands whose eloquent movements so expressive and so meaningful in context and concept, that stories of thousands of words are expressed in only a few minutes. I watch as two three-year-olds hug each other and say how much they've missed each other and it's clear that they mean it.

How good it feels to see the children hold their heads up high in surroundings where disability has always carried with it social stigma that cannot be erased overnight. I feel proud of the children, their parents, their teachers. I feel proud of what we have been able to achieve through the help and partnerships with our good friends and supporters the world over.

One of the exciting new programmes being implemented at Atfaluna this year is a 4-year training course to teach young deaf women and men to be teaching assistants. Both practical and theoretical training takes place at Atfaluna where the participants assist experienced teachers of the deaf in the classroom, learn to prepare lesson plans and teaching resources, and acquire the skills needed to assist in classroom management.

Fedwa, now 19 years old, was one of the first students to attend the Atfaluna School when it was established in 1992. Always an outstanding student, Fedwa is an excellent role model for other deaf children. A natural comedienne and talented actor, Fedwa is active in the Atfaluna Deaf Theatre Group where her favourite role is the mean stepmother in the group's Cinderella production. One of her favourite pastimes is baking traditional sweets which she learned in an Atfaluna culinary arts training course. Fedwa had thought of starting a home-based sweets business with her mother, but decided that helping other deaf children was her responsibility. She is excited about becoming an assistant teacher one day and hopes to be able to help deaf children to understand more easily things that she had difficulty grasping as a student.

On behalf of everyone here at Atfaluna, I would like to thank you most sincerely for all your support to the children in our care…children like Fedwa who would never have had a chance in life without your encouragement and support.

Sincerely

Geraldine (Gerry) Shawa
Executive Director

Editor's Comment

Although this letter from Geraldine Shawa of the **Atfaluna Society for Deaf Children** was received many months ago, its warm and encouraging news of a very significant programme is considered important to share with our readers.

❑

Abstracts

Combined effects of noise and mixed solvents exposure on the hearing function among workers in the aviation industry

Kim J, Park H, Ha E, Jung T, Paik N, Yang S

Department of Preventive Medicine
College of Medicine
Ewha Womans University
911-1, Mok-6-dong
Yangcheon-ku
Seoul 158-060
Korea

This study aims to evaluate the effect of occupational exposure to noise and organic solvents on hearing loss in the aviation industry. The study population comprised 542 male workers, who worked in avionics jobs in Kimhae, Korea, who kept records of work environment evaluations and medical examinations. The Cumulative Exposure Index (CEI) was constructed to assess the lifetime cumulative exposure of the workers, and pure tone audiometry (PTA) data of the workers from the biannual medical surveillance was used to assess hearing loss. The prevalence of hearing loss found in the group exposed to noise and mixed solvents simultaneously (54.9%) was higher than those in the other groups (6.0% in the unexposed, 17.1% in the noise-only, and 27.8% in the exposed to only solvents mixture). The relative risks, adjusted for age, were estimated to be 4.3 (95% CI 1.7-10.8) for the noise only group, 8.1 (95% CI 2.0-32.5) for the noise and solvents group, and 2.6 (95% CI 0.6-10.3) for the solvents-mixture group. These suggest that chronic exposure to mixed solvents had a toxic effect on the auditory system. This raises the issue of whether hearing conversation regulations should be applied to all workers exposed to solvents.

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Toxic solvents in car paints increase the risk of hearing loss associated with occupational exposure to moderate noise intensity

El-Shazly A

Misr University for Science and Technology
College of Medicine
Department of Otolaryngology
Cairo
Egypt

E-mail: amrel_shazly@hotmail.com

Solvents in car paints are a recognised source of occupational toxicity. In particular, they can cause DNA damage and occupational rhinobronchitis. However, little is known about their toxic effect in noise-induced hearing loss (NIHL) in humans. In this study, a 160 pure tone audiometric test was performed in workers in two independent factories to investigate whether toxic solvents in car paints can result in noise-induced hearing loss in workers exposed to moderate noise levels of less that 85 decibels (dB). It is shown that toxic solvents in car paints increase the risk associated with moderate noise exposure of less than 85 dB, with levels of NIHL being similar to those in workers exposed only to loud noises between 92.5 dB and 107 dB. Tinnitus and spells of dizziness were associated symptoms in all workers with NIHL, and asthma was an associated disease in workers with NIHL exposed to car paints and moderate noise simultaneously. These results may indicate that toxic solvents in car paints act in synergism with moderate noise exposure, damaging the cochlear hair cells. The results also constitute firm grounds for monitoring the hearing of these workers and adherence to strict regulations about wearing special gowns and filtered masks during working hours to protect against this preventable occupational disease.

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Organic solvents and hearing loss: The challenge for audiology

Fuente A, McPherson B

Centre for Communication Disorders
The University of Hong Kong
Hong Kong
China
E-mail: afuente@hkusua.hku.hk

Organic solvents have been reported to adversely affect human health, including hearing health. Animal models have demonstrated that solvents may induce auditory damage, especially to the outer hair cells. Research on workers exposed to solvents has suggested that these chemicals may also induce auditory damage through effects on the central auditory pathways. Studies conducted with both animals and humans demonstrate that the hearing frequencies affected by solvent exposure are different to those affected by noise, and that solvents may interact synergistically with noise. The present article aims to review the contemporary literature of solvent-induced hearing loss, and consider the implications of solvent-induced auditory damage for clinical audiologists. Possible audiological tests that may be used when auditory damage due to solvent exposure is suspected are discussed.

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COMMUNITY EAR AND HEARING HEALTH

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- To promote ear and hearing health in developing countries

Objectives

- To facilitate continuing education for all levels of health worker, particularly in developing countries
- To provide a forum for the exchange of ideas, experience and information in order to encourage improvements in the delivery of ear and hearing health care and rehabilitation.

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