3

THE PATHOPHYSIOLOGY OF THE EAR

Peter W.Alberti

Professor *em.* of Otolaryngology　　**Visiting Professor University of Singapore**
University of Toronto　　　　　　　　**Department of Otolaryngology**
Toronto　　　　　　　　　　　　　　**5 Lower Kent Ridge Rd**
CANADA　　　　　　　　　　　　　**SINGAPORE 119074**
　　　　　　　　　　　　　　　　　　palberti@attglobal.net

Things can go wrong with all parts of the ear, the outer, the middle and the inner. In the following sections, the various parts of the ear will be dealt with systematically.

3.1. THE PINNA OR AURICLE

The pinna can be traumatized, either from direct blows or by extremes of temperature. A hard blow on the ear may produce a haemorrhage between the cartilage and its overlying membrane producing what is known as a cauliflower ear. Immediate treatment by drainage of the blood clot produces good cosmetic results. The pinna too may be the subject of frostbite, a particular problem for workers in extreme climates as for example in the natural resource industries or mining in the Arctic or sub-Arctic in winter. The ears should be kept covered in cold weather. The management of frostbite is beyond this text but a warning sign, numbness of the ear, should alert one to warm and cover the ear.

3.2. THE EXTERNAL CANAL

3.2.1. External Otitis

The ear canal is subject to all afflictions of skin, one of the most common of which is infection. The skin is delicate, readily abraded and thus easily inflamed. This may happen when in hot humid conditions, particularly when swimming in infected water producing what is known as swimmer's ear. The infection can be bacterial or fungal, a particular risk in warm, damp conditions.

　　The use of ear muffs particularly in hot weather may produce hot, very humid conditions in the ear canal leaving it susceptible to infection, and similarly insertion and removal of ear plugs may produce inflammation. Although this is surprisingly rare; it does occur particularly in those working with toxic chemicals. These people should take care to wash their hands before inserting or removing ear plugs or preferably use ear muffs. The soft seal of a muff should be kept clean and if reusable plugs are used, they should also be regularly washed. Inflamed or infected ear canals should be treated by a physician.

3.2.2. Obstructing Wax.

One of the more common conditions found is obstruction of the ear canal by a mass of wax. Wax is a mixture of dead skin, oily secretions and sweat and it has varying consistencies from soft to almost rock hard. It normally is extruded on its own but if it is not, it needs careful removal. Contrary to popular belief, wax is not soluble in oil but does disintegrate in water. Gently flushing the ear with water, as for example in a shower, goes a long way towards clearing the ear of wax, or softening it for a physician or nurse to remove. Methods for removal of wax are beyond the scope of this document.

3.2.3. Exostosis

Sometimes there are bony narrowings of the ear canal known as exostoses which are often found in people who have swum a great deal in cold water. They sometimes look like white pearls and are frequently mistaken for cysts. Their only importance is that they may obstruct the view of the tympanic membrane and may be mistaken for pathology. For fuller details of diseases of the pinna and external auditory canal, the reader should consult standard texts or atlases such as Hawke et al (1990).

3.3. THE TYMPANIC MEMBRANE

Perforations of the tympanic membrane may occur as a result of disease or trauma. They usually occur in the pars tensa and may be central or marginal. A central perforation is one which does not reach the edge (annulus) and is usually harmless; a marginal perforation on the other hand which reaches the edge of the membrane and is congruent with the ear canal. This allows the skin of the ear canal to grow into the middle ear space where it desquamates without the debris being able to fall out and may lead to severe disease. Attic perforations are considered as dangerous as marginal perforations. These matters will be dealt with further under the pathology of the tympanic membrane and middle ear.

   Traumatic perforations of the tympanic membrane are not infrequent and may occur as a result of a foreign body being pushed through the membrane, as for example a pencil, hair clip or cotton applicator. Industrially, sparks from welding or brazing may fall into the ear canal and burn a hole in the membrane and finally, intense explosion, such as nearby shells or bombs, particularly in a confined space or accidental exposure to mining and quarrying blasts may perforate the membrane. If the explosion is severe enough , there may be disruption of the ossicular chain, and even a sensory neural hearing loss as well (see for example, Cudennec, 1986; Borchgrevink, 1991). Traumatic perforation of all types usually heal on their own; if they do not, surgical grafting may be necessary.

   Wormald and Browning (1996), give an excellent, simple, logical, well illustrated guide to diseases of the tympanic membrane and middle ear.

3.4. THE MIDDLE EAR

3.4.1. Acute Otitis Media

The most common causes of disease of the middle ear are respiratory infections producing acute or chronic otitis media. The middle ear, being part of the respiratory tract, is subjected to the

same infections as the nose and sinuses and is frequently involved when they become inflamed. The most common is acute otitis media, inflammation of the lining membrane of the middle ear, including the tympanic membrane. If the infection is severe, the middle ear lining, including the tympanic membrane, swells. This produces intense pain and if the swelling is too great then the blood vessels in the ear drum are compressed, local tissue necrosis and the ear drum bursts, letting out pus and relieving the pain. Usually the hole is small and heals quite quickly. It is customary to prescribe an antibiotic although it should be said that about 80% of all acute otitis media resolve spontaneously without treatment.

3.4.2. Chronic Serous Otitis Media

Otitis media with effusion, OME, is probably the most common form of sub-acute ear disease found in the developed world. It occurs following otitis media, when the fluid in the ear, formed by the infection, does not drain spontaneously. The tympanic membrane is intact but the middle ear is fluid filled. This puts it at risk for further infection and certainly worsens hearing by about 30 dB. This is most frequently found in children and can interfere with language acquisition and learning.

3.4.3. Chronic Otitis Media

Sometimes the infection does not settle down and a chronic perforation occurs. This may produce a conductive hearing loss because there is not enough area of the tympanic membrane to catch sound. This type of perforation is usually central, and the middle ear lining becomes thickened and chronically inflamed. The ear is at risk for further acute infections, particularly if dirty water enters the ear. The hearing is also reduced, with a conductive loss of about 20 to 50 dB. The perforation usually happens in childhood and is often associated with a malfunction of the Eustachian tube.

3.4.4. Chronic Otitis Media with Cholesteatoma

In the presence of marginal perforations skin from the ear canal can migrate into the middle ear and space and into the attic surrounding the ossicles and into the mastoid. This skin sheds its surface cells which remain in the middle ear space, looking like white pearly material. If this gets wet or infected it swells and can produce a great deal of damage in the ear and surrounding structures such as the brain and the facial nerve, the nerve that supplies the muscles of the face, because it runs through the ear. Its diagnosis and management are outside the scope of this document.

3.5. INDUSTRIALLY RELATED PROBLEMS OF THE EXTERNAL AND MIDDLE EAR

3.5.1. Trauma

3.5.1.1. Direct blows

Blows to the pinna may produce haematoma described previously. Wearing of dirty ear plugs may produce external otitis. Insertion and removal of ear plugs with dirty hands may produce

contact dermatitis of the ear canals. Hard blows to the side of the head may produce perforation of the tympanic membrane, which usually heals spontaneously. All have been described above.

Severe blows to the head may fracture the temporal bone and dislocate and fracture the ossicular chain. This may produce a significant amount of conductive hearing loss although it is usually accompanied also by a sensori-neural loss.

3.5.1.2. Foreign bodies

These may fall into the ear as for example sparks when welding and hot objects may hit the tympanic membrane burning holes in it. These are difficult to close permanently by operation. The writer has seen unfortunate individuals who have fallen into vats containing chemicals, producing chemical external otitis or in whom hot liquids which they had been carrying on their shoulder have spilled into the ear, burning the ear (Frenkiel and Alberti, 1977). The tympanic membrane may also be directly perforated by sharp object stabs in the ear, or by explosions as already described.

3.5.1.3. Barotrauma

 Divers are subject to middle ear haemorrhage and blockage if they cannot clear their ears when descending or ascending. If this occurs, a physicians' opinion should be sought. Care should be taken not to dive with a cold, for this reduces Eustachian tube function and thereby the ability to equalize middle ear pressure.

3.6. THE INNER EAR

At birth the inner ear is fully developed. The cochlea is adult sized and has its total complement of hair cells, supporting cells and nerve fibres. The tissues respond like those elsewhere in the body to trauma and infection by producing an inflammatory response. However, unlike most other tissue in the body, if the damage is severe enough to destroy a mammalian hair cell or nerve fibre, it does not regenerate. One is born with the full complement of hair cells and nerve fibres. Through life they are gradually diminished as a result of a variety of processes including infection, trauma and ageing. By contrast, avian auditory hair cells retain the capacity to regenerate if destroyed by, for example, acoustic trauma; there is much research worldwide to elucidate the mechanism in the hope that it may be applied to the mammalian ear.

3.6.1. Infection

Certain viral infections have a predilection for the ear and wreak havoc with its structure. Pregnant women may contract Rubella (German Measles) and the virus may destroy the developing cochlea leading to a child born deaf, as part of the rubella syndrome. In post-natal life both measles and particularly mumps may infect the inner ear destroying the cochlea, producing total deafness in that ear. This is usually unilateral and almost always happens in childhood. It can be completely prevented by appropriate vaccination. It may occur in as many as one in a hundred children who develop mumps, leaving them with a unilateral hearing loss. If this occurs early it may produce noticeable symptoms, often only being discovered in later in life when the telephone is first used and held to the deaf ear.

3.6.2. Bacterial Infections

Meningitis commonly affects the inner ear because the perilymph, the fluid surrounding the membranous labyrinth is in direct continuity with the cerebral spinal fluid. Meningitis produces an acute inflammatory response of the meninges (the membrane surrounding the brain)and may also produce a similar response in the cochlea destroying it completely. Of those who survive meningitis up to five percent may be deaf (Fortnum and Davis, 1993). This too can usually be prevented by a vaccination. It has become a major problem in the central African and West Asian countries which form part of the meningitis belt (Moore and Broome, 1994).

3.6.3. Immunological Diseases

The inner ear is subject to certain diseases, one of the most common of which is Meniere's disease or syndrome. The disease is characterized by episodes of loss of hearing, a sense of fullness in the ear, ringing, nausea and vomiting. To begin with, the hearing loss is transient but ultimately it becomes permanent. The dizziness lasts for two or three hours at a time and the whole episode with repeated attacks may last for a month or six weeks only to recur again several months later. The pathophysiology is almost certainly an immune reaction. This gives rise to an inflammatory response producing too much fluid within the membranous labyrinth which distends and ultimately may rupture. When there is inter-mingling of endolymph and perilymph, hearing loss occurs. If the ruptured membrane heals, the hearing may recover. In time fifteen percent of patients develop the disease in both ears. After many years it ultimately burns out. It rarely leaves the person deaf but it does produce a severe hearing loss. For further details see standard texts or monographs such as Nadol (1989) or Oosterveld (1983).

3.6.4. Sudden Hearing Loss

A sudden inner ear hearing loss, defined as a loss which develops in a matter of seconds to two or three days, is quite common. The person will often notice an increased buzzing in the ear and a loss of hearing and associated with a distortion of sounds. Treatment varies widely throughout the world, ranging from nil to aggressive in hospital management with a variety of medications. The results seem to be similar: more than one-third recover completely, one-third recover somewhat and one-third do not recover. It may be associated with an episode of acute vertigo. The cause of this type of loss is unknown although it is sometimes attributed to excessive pressure change as for example hard nose blowing which may rupture Reissner's membrane or the round window membrane. This is probably conjecture.

For more information about diseases of the inner ear, the reader should consult the standard textbook of Otology in use in their country.

3.6.5. Tinnitus

Ringing in the ears is an extremely common phenomenon found at some time or another in up to one-third of the adult population; twelve percent have it sufficiently severely for them to seek a medical opinion about it. The noises in the ear are of many types, ranging from hearing one's own pulse to buzzes, clanging, clicking, whistling, humming and ringing of which the most common types are buzzing and ringing. Usually tinnitus cannot be heard by the outside observer and is known as subjective tinnitus. It is irritating but usually harmless. People with tinnitus are

often worried that it is a precursor of some serious disorder such as stroke, hypertension or brain tumour. The prevalence of tinnitus in these conditions is no greater than the population at large, so they can be reassured. It is more common in the presence of hearing loss and may be precipitated by an acute traumatic episode such as an explosion or a head injury. Transient tinnitus is a fairly common finding in response to loud sound, often occurring in those who go to a noisy disco. It is also a common finding in occupational hearing loss, found somewhat more frequently in those exposed to impact than to steady state noise. It should be considered as a warning of excessive sound exposure and appropriate precautions taken.

Occasionally the noises may be heard by another person in which case they are known as objective tinnitus. This is caused either by a vascular malformation in which case the sound is a pulsatile one which may be heard by an outside observer through a stethoscope applied to the head or it is due to muscleclonus, usually of the palate in which case there is an audible clicking sound. For further reading about tinnitus in general, the reader is referred to conference proceedings edited by Feldmann (1987) and by Aran and Dauman(1991).

3.7. OCCUPATIONAL CAUSES OF INNER EAR HEARING LOSS

3.7.1. Noise

Excessive exposure to noise is probably the most common cause of preventable hearing loss on a global basis. In general terms, prolonged exposure to sound in excess of 85 dB(A) is potentially hazardous although the important factor is the total amount of sound exposure i.e., both the level and length of exposure are important and the two interrelate (see for example Robinson, 1987, Dobie, 1993). Chapter 4 in this document deals with safe levels of sound exposure both in terms of loudness and duration. Here we will deal with the damage that excessive sound may cause to the inner ear. After exposure to a typical hazardous industrial sound, perhaps in the low nineties (dB(A)) for an 8-hour work day, the ear fatigues and develops a temporary threshold shift (TTS). The hair cells become exhausted from the excessive metabolic stress placed upon them and hearing becomes less acute. This is usually transient and after appropriate rest, recovery ensues. Workers notice this with their car radios: when they leave work they turn the volume up and by the next morning the radio is too loud; those going to discos cannot hear while they are in the disco and cannot hear when they come out but by the next morning their ears too have recovered.

The pathophysiology of noise damage to the ear has been extensively studied in man and animals and much is now known of the mechanism whereby excessive sound exposure damages the ear. Low levels of damaging sound exposure produces TTS, as described in the preceding paragraph. If TTS occurs day after day, the recovery becomes less complete and a permanent threshold shift (PTS) occurs because with persistent exposure to such sounds some hair cells do not recover. First to fail permanently are the outer hair cells (OHCs) in the basilar part of the cochlea, in the area which responds to 4 kHz and the adjacent areas of 3 and 6 kHz. This is where the ear is most sensitive, in part because of the harmonic amplification of the ear canal and in part because of an absolute sensitivity. Once hair cells degenerate they do not recover and a permanent hearing loss develops. Classically therefore, following noise exposure, hearing loss is shown as an audiometric notch, usually maximal at 4 kHz but may also be based anywhere between 3 and 6 kHz. With higher noise exposure for longer periods, the loss extends into adjacent frequencies. If the sound is sufficiently intense, it produces a much more severe TTS which may go on to a more rapidly produced PTS. There is a critical point where moderate TTS

changes to longer term TTS which correlates well with anatomical damage to the OHCs, a process of damage and scarring or repair. The threshold for TTS is somewhere between 78 and 85 dB and the point where it changes from mid-term to long-term is about 140 dB. The spectrum of the sound and the length of exposure are critical.

Cilia of the OHCs are attached to each other near their tip by linking filaments and each cilium has a little rootlet which passes through the ciliary plate (see Figure 3.1).

If the mechanical disturbance produced by sound is sufficient to fracture the rootlet, or to
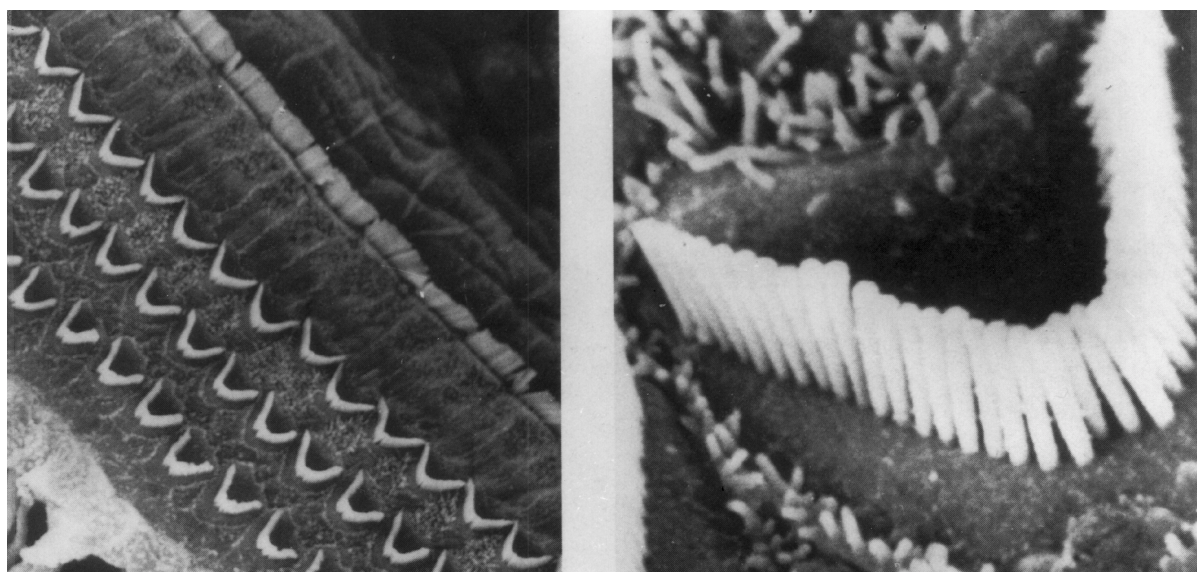

Figure 3.1. **(a) Surface of the normal organ of Corti, guinea-pig, X 1100.**
(b) Close up view of the stereocilia of OHC, X 11000 (from Gao et al, with permission).

disturb the linkages, which frequently are concurrent, the result is a floppy cilium. These either partially recover or are totally destroyed and replaced by phalangeal scarring. By contrast, moderate sound excursion produces much less (and temporary) distortion of the cilia and they recover (see Figure 3.2).

Noise characteristically damages the OHCs of the basilar turn. If sound is intense enough, there is physical disruption of the cochlea and other structures may also be damaged, such as the stria vascularis and the supporting cells. Some time after hair cell death there is also neural degeneration of the first order neurones. Very intense sound has been shown to produce damage to the vestibular epithelium of guinea-pigs but has not been convincingly demonstrated in man.

Figure 3.2(a). Changes in stereocilia, guinea pig, (X 1700) after 30 minutes exposure at 110 dB. Note slight bending and separation at the tips of the stereocilia. The ear had a 25 -30 dB TTS.
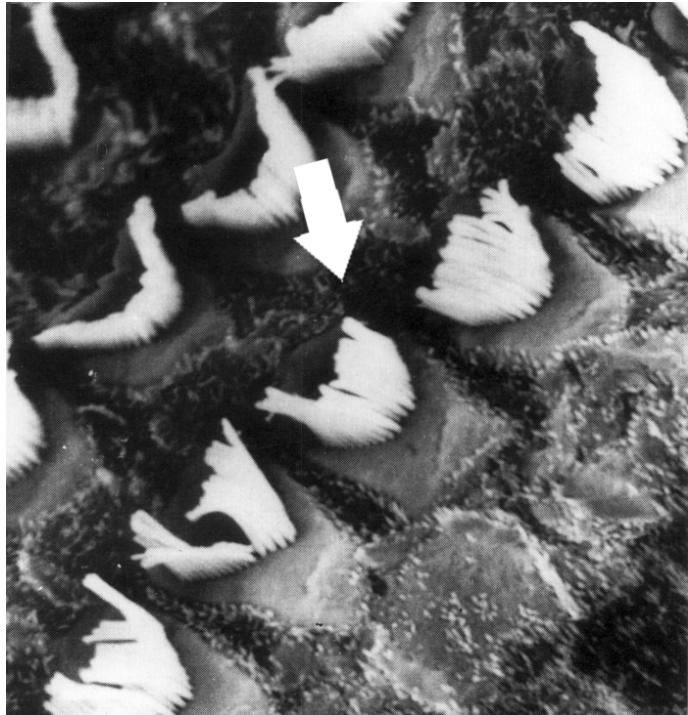
Figure 3.2(c). Changes in stereocilia, guinea pig, (X 1700) of the 110 dB group eighty days after exposure. The hearing was normal and so was the appearance of the stereocilia
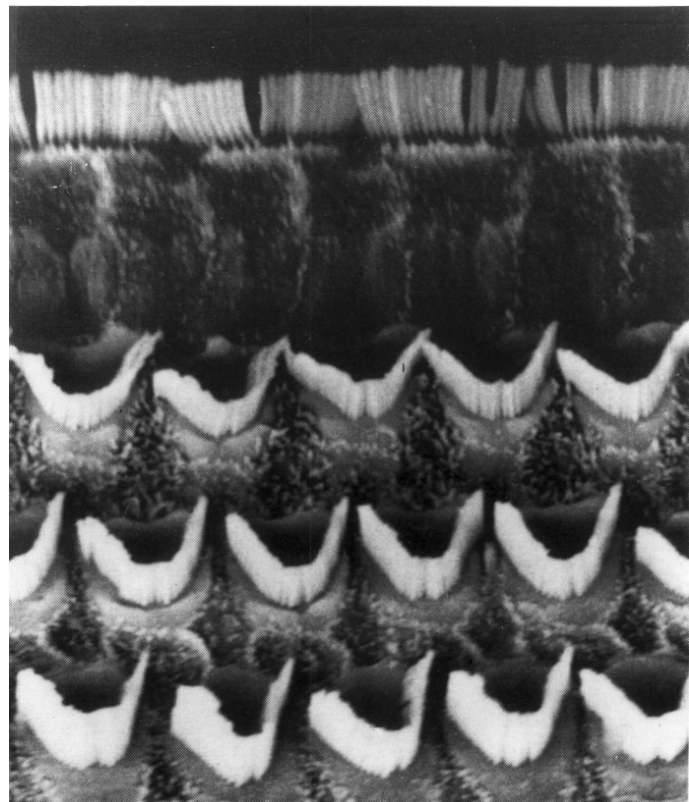
Figure 3.2(b). Changes in stereocilia, guinea pig, (X 1700) after 30 minutes exposure at 120 dB. Note complete collapse at the bases of stereocilia. The ear showed a 45 to 50 dB TTS.

Figure 3.2(d). Changes in stereocilia (X 1700) in the apical surface of the organ of Corti, guinea pig, of the 120 dB group eighty days after exposure. The surface is devoid of both stereocilia and hair cells. (from Gao et al, with permission).

3.7.1.1. Tuning curves

Each auditory nerve fibre is most responsive to a specific frequency, but as the intensity of sound increases it becomes progressively sensitive to adjacent frequencies. With OHC loss (as occurs in NIHL), the most sensitive fine tuned part of the response disappears and the nerve fibres respond at an elevated threshold to a broader band of frequencies. It is generally assumed that the sharp tuning of these curves at low intensity is due to active mechanisms in the OHCs and associated efferent nerve pathways. Their loss may be correlated with the clinical finding of poor sound discrimination, a common complaint in NIHL.

3.7.1.2. Toughening

There is some evidence that prior exposure to none damaging levels of low frequency noise protects the cochlea from damage by subsequent high intensity sound. The mechanism and importance of this phenomenon are not clear (Henderson et al, 1993; Henselman et al, 1994).

Permanent hearing loss from exposure to hazardous noise may happen quite early and an audiometric notch may be noticeable within six months or a year of starting a job in a hazardous level of sound. The international table of risk (ISO 1999E) gives these predictions. There is a great variation in the susceptibility of the ear to the effect of sound also evident in well controlled animal experiments - some people have tough ears and some have tender ears

There are military small arms instructors who over a lifetime have fired hundreds of thousands of rounds and who have little or no hearing loss at the end of it; there are recruits who after one day on the range develop a permanent notch. The international table takes this into account. It is, therefore, very difficult to state with certainty what a safe level of sound may be; a level which is safe for 85% of the population may leave 15% at risk and a level which is safe for the total population is so low that it is impractical to implement. In any event, with continuing hazardous sound exposure, the hearing continues to worsen although the greatest loss occurs in the first ten years and thereafter the rate slows.

How to determine susceptibility to noise exposure before the event is elusive. Attempts to correlate TTS after one day's exposure to long term loss have repeatedly failed. TTS at the end of a work shift does mark the upper bound of the PTS produced by the same sound exposure after ten years. However the PTS may be much less. A promising (and fashionable) test is based on changes in oto-acoustic emissions; some investigators have suggested that there is a reduction in these emissions before a change is evident in the pure tone threshold, giving an early warning of incipient damage.

3.7.2. Asymmetric Hearing Loss

Usually if both ears are exposed to the same level of sound, the hearing loss is symmetrical. The left ear may be a little worse because, in general terms, the hearing in the left ear of males is slightly worse than the right by about 4 dB at 4 kHz (Pirila et al, 1991). However, causes for greater asymmetry should be sought. These may be industrial and non-industrial.

Not all industrial noise exposure is equal in both ears. Usually sound level measurements in industry are made at the work site, they are not taken at the worker's head.

There are many processes where the sound is more intense at one side of the head than the other and indeed the head may produce a sound shadow. A classic example is rifle firing where the left ear which is nearest the muzzle in a right-handed person, is exposed to more sound than the right ear, which is protected by the head shadow and the result is a notched hearing loss in the left ear. Use of hard rock drills in mining produces a similar affect and so may the use of heavy electric drills into concrete where there can be up to 8 dB difference in the sound pressure level between the ears which may translate into different hearing losses of the two ears. This is also a common finding in agricultural workers, particularly tractor drivers (especially if there is no cab), who sit with their head turned watching what they are pulling with the leading ear exposed to the exhaust at the front of the equipment. Much further study is required of the noise exposure at the ear as opposed to sound pressure levels at the work site.

3.7.3. Social Noise Exposure

Social noise exposure is also a significant source of acoustic trauma, both from recreational pursuits (Clark, 1991) and from the noise enveloping the cities of the developing world. There are good studies of city sound levels in Asian cities showing sound levels sustained at hazardous levels for many hours of the day(e.g., Bosan, 1995; Chakrabarty, 1997). Exposure to city sound levels may interact with industrial noise exposure and it may be difficult to decide how much of a hearing loss is due to workplace hazards and how much due to recreational or environmental sounds. The factors which are important vary by community. Further discussion is beyond the scope of this chapter.

3.7.4. Progression of Hearing Loss

It has already been mentioned that with prolonged exposure to the same noise, hearing loss continues to worsen. The international standard, ISO 1999, allows one to predict how much hearing loss may be expected for a given noise exposure for varying periods of time. For a given hazardous sound level, the maximal effect is in the first few years, although there is a slow, continuing progression of hearing loss thereafter as long as the noise exposure continues. However, at the same time, all people are subject to the hearing loss of ageing, known as presbyacusis, in which there is a gradual loss of hearing in later years caused in part by hair cell degeneration. Individual variation is great: some people maintain good hearing into old age, others do not (Corso, 1980; Macrea, 1991; Robinson, 1991; Rosenhall, 1990). Tables exist to predict the amount and range of presbyacusis, and they are also incorporated into the noise standard, ISO1999.

The interaction between noise exposure and presbyacusis as causes of hearing loss are important and complex. Are the two additive, or is an ageing ear more or less susceptible to the affects of hazardous noise? It seems that in the earlier years they are additive, but thereafter as the exposure continues and the subject ages, the hearing loss is less than would be expected by simply adding the predicted loss from noise in dB to that predicted from ageing in dB. Many formulae have been devised to attempt to separate the affects of noise from ageing (Dobie, 1992; Robinson, 1987, Bies and Hansen, 1990).

The question is often asked whether hearing loss from noise continues after removal from the noise source. It is overwhelmingly accepted, although not universally, that this does not occur and that any worsening that happens in the months and years after quitting working in a noisy place is due to other causes, almost always presbyacusis.

3.7.5. Trauma

The inner ear can be damaged by direct head injury or by blasts such as explosions, or pressure changes.

3.7.5.1. Head injuries

Head injuries, even those that do not produce unconsciousness, can produce disruption of the cochlea with a sensory-neural hearing loss. This is not necessarily notched as with noise but may be flat. More severe head injury can produce a fracture of the temporal bone leading to disruption of the middle ear, as well as well as to trauma to or a fracture through the cochlea itself, and thus a conductive loss as well as a sensory neural loss; the latter destroying the hearing totally.

3.7.5.2. Explosions

Blast injuries, i.e. ones where the sound levels exceed those normally found in industry, can produce physical disruption to the cochlea. Any sound loud enough to produce more than a 40 dB temporary threshold shift is likely to produce permanent trauma to the cochlea. The cochlea, like all other tissue in the body, responds to trauma with an inflammatory reaction and cells may be repaired, in which case some recovery of hearing takes place or the cells may be so badly damaged that they degenerate and are absorbed, producing hearing loss. In general terms, if the trauma is loud enough to snap the cilia, the cells will not recover (see above). This type of damage occurs with blasting accidents in mining, gas explosions and in the military (Borchgrevink, 1991; Cudennec et al., 1986; Phillips & Zajtchuk, 1989; Roberto et al., 1989).

3.7.5.3. Baro-trauma

Extreme pressure changes can cause temporary and permanent damage to the ear. The changes associated with flying are the best known, such as pain on ascent and particularly on descent, caused by inadequate function of the Eustachian tube, the small passage which connects the middle ear to the nose. Most readers will be familiar with a stuffy sensation in the ears when riding a high speed elevator in a tall building. Similar problems occur with industrial elevators in mines, where workers may descend several hundreds of metres at high speed to reach the active mining site. Ear pressure equalization problems are a common complaint amongst workers in the deeper gold and nickel mines.

The greatest hazard to the ear related to pressure comes from working in higher than normal atmospheric pressure such as in some tunnelling operations and in diving. When a person is exposed to higher than normal pressures the blood gasses equilibrate with the surrounding gas and greater amounts are absorbed into the body. If the ambient pressure suddenly returns to normal, the gas dissolved in the body tissues comes out of solution, and particularly nitrogen, forms bubbles in body tissues. These are painful and may produce damage by preventing oxygen reaching the tissues. The condition is known as "The Bends" and can affect the ear, producing permanent damage to the cochlea, and with it, varying degrees of hearing loss (Al-Masri et al, 1993; Molvaer et al, 1990; Talmi et al, 1991).

It is also suggested that exposure to high noise levels while exposed to high pressure may be

more hazardous to the hearing than the same noise at ambient pressure.

3.7.6. Complex Interactions

 It is becoming clear that noise is not the only industrial hazard to hearing; exposure to certain chemicals such as toluene and trichlorethylene can produce hearing loss (Boettcher et al, 1992; Franks et al, 1996); as can interaction with certain medicines (Aran et al, 1992). More important, the interaction between noise and the chemicals may produce more hearing loss than expected; i.e., they act synergistically (Johnson et al, 1995; Morata et al, 1995). The same is true of those subject to vibration induced white hand, as may occur in the forestry industry; they develop more hearing loss from the same exposure than fellow workers whose hands do not turn white (Iki 1996).

REFERENCES

Al-Masri, M., Martin, A.and Nedwell, J., (1993). Underwater hearing and occupational noise exposure limits. In: Noise and Man '93 - Noise as a public health Problem. *Proc. 6th Int. Cong.* Vol. 2. ed M.Vallet. Inst. Nat. de Recherche sur les Transports et leur securite. Arcueil Cedex, France.

Aran, J-M, Dauman, R.,(1991) eds, *Tinnitus 91*. Kugler Pub., New York.

Aran, J. M., Hiel, H., Hayashida, T., Erie, P., Auroussean, C., Guilhaume, A., and Dulon, D. (1992). Noise, aminoglycosides and diuretics. In *Noise induced hearing loss*, ed. Dancer, A., Henderson, D., Salvi, R.J. &Hamernick, R.P. St Louis: Mosby Year Book.

Bies, D.A. and Hansen, C.H. (1990). An Alternative Mathematical Description of the Relationship Between Noise and Hearing Loss. *J. Acoust. Soc. Am.* **88**.

Boettcher, F.A., Gratton, M.A., Bancroft, B.R. & Spongr, V. (1992). Interaction of noise and other agents: recent advances. In *Noise induced hearing loss*, (ed. Dancer, A. L., Henderson, D., Salvi, R.J. & Hamernick, R. P). St Louis: Mosby Year Book.

Borchgrevink, H.M. (ed), (1991). Effects of noise and blasts. *Scan Audiol.* Suppl. **34**

Bosan, A., Zaidi, S.H. and Noneel, T. (1995). The problem of noise. *Pakistan J .Otolaryng.*, **11**, 128-131.

Chakrabarty, D., Snatra, S.C., Mukherjee, A., Roy, B. and Das, P. (1997). Status of road traffic noise in Calcutta metropolis, India. *J. Acoust. Soc. Am.* **101**, 943-949.

Clark, W.W. (1991). Noise exposure from leisure activities: A review. *J Acoust. Soc. Am.*, **91**, 175-181.

Corso, J.F. (1980). Age correction factor in noise induced hearing loss: a quantitative model. *Audiology*, **19**, 221-232.

Cudennec, Y.F., Lory, D., Poncet, D.L., Christau, R., et al, (1986). Les lesions auriculaires par blast - Aspects actuel et etude de 200 cas. *Ann.Oto-Laryng.*, (Paris), **103**, 335-341.

Dobie, R.A. (1992). The relative contributions of occupational noise and ageing in individual loss of hearing loss. *Ear, Hearing*, **13**,19-27.

Dobie, R.A. (1993): *Medical-Legal Evaluation of Hearing Loss*. New York, Van Nostrand Reinhold.

Feldmann, H., *Proc. 111 Int Tinnitus Seminar*, Harsch, Karlsruhe.

Fortnum, H., and Davis, A. (1993). Hearing impairment in children after bacterial meningitis; incidence and resource implications. *Brit. J.Audiol.*, 43 -52.

Franks, J.R., and Morata, T.C. (1996). Ototoxic effects of chemicals alone or in concert with noise: A Review of Human Studies. *Scientific Basis of Noise-Induced Hearing Loss* **35**, 437-446, Thieme, New York.

Frenkiel S and Alberti PW: (1977). Traumatic thermal injuries of the middle ear. *J. Otolaryngol.*, **6**, l7-22.

Gao, W.W., King, D.L., Zheng ,X.Y., Ruan, F.M. and Liu, Y.J. (1992). Comparison in the changes in the stereocilia between temporary and permanent threshold shift. *Hear. Res*. **62**, 27-41.

Hawke, M., Keene, M., and Alberti, P.W., *Clinical Otoscopy*, 2nd ed, Churchill Livingstone, London.

Henderson, D., and Subramaniam, M. (1993). Toughening: acoustic parameters. In *Noise and Man, '93. Noise as a public health problem - Proc. 6th Int. Congress*, Nice, France. Vol. 2, (ed. Vallet,M., Inst. Nat. RecherchÈ sur les transports et leur securite(INRETS), Arcueil Cedex, France).

Henselman, L.W., Henderson, D., Subramaniam, M. and Sallustio, V.F.G.(1994). The effect of 'conditioning' exposure on hearing loss from impulse noise. *Hear. Res..* **78**, 1 - 10.

Iki, M., Kurumatami, N., Hirata, K., Moriyama, T., Itoh, J., and Arai, T. (1986). An association between vibration induced white finger and hearing loss in forestry workers. *Scan. J. Work Environ. Health*, **12**, 365-370.

Johnson, A., and Nylen, P. (1995). Effects of industrial solvents on hearing. *Occupational Medicine* **10**, 623-640.

Macrea, J.H. (1991). Presbyacusis and noise induced permanent threshold shift. *J. Acoust. Soc. Am.*, **90**, 2513-2516.

Molvaer, O.I. and Albrektsen, G., (1990). Hearing deterioration in professional divers: an epidemiologic study. *Undersea Biomedical Research.*,**17**, 231 - 246.

Moore, P.S. and Broome, C.V., (1994). Cerebrospinal meningitis epidemics. *Sci. Am.*, Nov., 38 - 45.

Morata, T.C. and Dunn, D. E. (1995). Occupational Hearing Loss. *Occupational Medicine*: *State of the Art Reviews*, **10**, No. 3, Philadelphia, Hanleyand Belfus.

Nadol, J.B. Jr., (ed), (1989). *Meniere's Disease*, Kugler and Ghendini. Berkeley.

Oosterveld, W.J. (ed), (1983). *Meniere's Disease.* Wiley and Sons, Chichester

Phillips, Y.Y. and Zajtchuk, J.T., (1989). Blast injuries of the ears in military operations. *Ann. Otol. Rhinol. Laryngol.* **98**, Suppl 140, 3-4.

Pirila, T., Jounio-Ervasti, J., and Sorri, M. (1991). Hearing asymmetry in left and right handed persons in a random population. *Scan. Audiol*. **20**, 223-226.

Roberto, M., Hammernik and R.P., Turrentine, G.A., (1989). Damage of the auditory system associated with acute blast trauma. *Ann. Otol. Rhinol. Laryngol*. **98**, Suppl. 140, 23-34.

Robinson, D.W. (1987). *Noise Exposure and Hearing: A New Look At The Experimental Data*. Health and Safety Executive Research report, 1/1987. H.M.S.O.

Robinson, D.W. (1991). Relation between hearing threshold level and its component parts. *Brit. J. Audiol.* **25**, 93-103.

Rosenhall, U., Pedersen, K., & Svanborg, A. (1990). Presbyacusis and noise induced hearing loss. *Ear, Hearing*, **11**, 257-263.

Saunders, J.C. (1991). The structural and functional consequences of acoustic injury in the cochlea and peripheral auditory system: A five year update. *J Acoust. Soc. Am.* **90**, 136-146.

Talmi, Y.P., Finkelstein, Y., Zohar, Y. (1991). Decompression sickness induced hearing loss. *Scan Audiol*. **20**, 25 -28.

Wormald, P.J. and Browning, G.G. (1996) *Otoscopy, a structured approach*. Arnold, London.

INTERNATIONAL STANDARD

Title of the following standard related to or referred to in this chapter one will find together with information on availability in chapter 12:

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FURTHER READING

Axelsson, A., Borchgrevink, H., Hamernik, R.P., Hellstrom, P.A., Henderson, D. and Salvi, R.J. (1996): *Scientific Basis of Noise-Induced Hearing Loss*. New York, Thieme.

Liberman, M.C. (1987). Chronic ultra structural changes in acoustic trauma: serial section reconstruction of stereocilia and cuticular plates. Hearing Research, **26**, 25-88.

Liberman, M.C. and Dodds, L. W. (1987). Acute ultra structural changes in acoustic trauma: serial section of stereocilia and cuticular plates. Hearing Research, **26**, 45-64.

Saunders, J.C. (1991). The structural and functional consequences of acoustic injury in the cochlea and peripheral auditory system: A five year update. J Acoust. Soc. Am, **90**, 136-146.