The 2011

Libby Harricks

Memorial Oration

Honouring the Deafness Forum's first president & profoundly deaf achiever

Elisabeth Ann Harricks AM 1945 – 1998

Contents

Introduction to the 13th Libby Harricks Memorial Oration

Dr Jenny Rosen AM, Chairman, Libby Harricks Memorial Oration Committee

Libby Harricks was born and educated as a normally hearing person. On developing a profound hearing loss as a young wife and mother, she quickly educated herself with skills to manage her own hearing difficulties, and soon became a committed advocate for hearing impaired people. Libby was a founding member and long term President of SHHH Australia Inc (Self Help for Hard of Hearing People) and in 1993 was elected inaugural President of Deafness Forum of Australia, the national peak body in deafness and related issues. In these voluntary roles, she worked ceaselessly to raise awareness of the need for equal inclusion in life activities for hearing impaired people, travelling widely throughout Australia to lobby for this on their behalf. Libby also served on the Board of Australian Hearing for a number of years and represented hearing impaired people on the Sydney 2000 Olympics Access Committee. In recognition of her work, in 1990 Libby was made a Member of the Order of Australia.

After her death in 1998, Deafness Forum established the Libby Harricks Memorial Oration Series. The aim of the Oration Series is to honour her achievements and to continue her work towards gaining appropriate recognition, awareness, and access, for hearing impaired people. In order to reach further than each Oration audience and indeed to make these important contributions available on an on-going basis, the Orations are published by Deafness Forum of Australia in a Monograph series.

The inaugural Oration entitled *'Hearing Access Now!'* was presented by Emeritus Professor Di Yerbury in Sydney in 1999. Professor Bill Gibson focussed on tinnitus and Meniere's Disease at the international Federation of Hard of Hearing Conference in Sydney in 2000. Senator Margaret Reid spoke on *'The Politics of Deafness'* in Canberra in 2001. In 2002 at the XXVI International Congress of Audiology in Melbourne Professor Paul Mitchell presented *'The Prevalence, Risk Factors and Impacts of Hearing Impairments in an Older Australian Community: The Blue Mountains Study'*, a major

demographic study. At Macquarie University Sydney in 2003, Donna Sorkin addressed progress in disability law and hearing loss from an international perspective.

In 2004 in Brisbane at the 3rd National Deafness Sector Summit, Dr Peter Carter's topic was *'A Sorry Business: Lack of Progress in Aboriginal Hearing Health.'* In the Blue Mountains NSW in 2005, Alex Jones made a major impact with his presentation, the first in Auslan, entitled *'Deafness and Disability Transformed: An Empowering Personal Context.'* Professor Harvey Dillon presented his 2006 paper *'Hearing Loss: The Silent Epidemic'* at the 4th National Deafness Sector Summit in Perth. In Albury in 2007, Rick Osborn impressed the 9th Rural Health Conference with insights relating to *'Hearing and Communication – A Primary Concern in Aged Care.'* At the 5th National Deafness Summit in Canberra in 2008, Professor Robert Cowan spoke on *'Access, Equity and Hearing Loss in Australia in 2008'* and in 2009 Professor Graeme Clark addressed General Practitioners at a Continuing Education Conference in Sydney, on the background and current status of cochlear implants. In 2010 Professor Greg Leigh addressed the 6th National Deafness Summit in Sydney, with the timely topic *'Early Identification of Hearing Loss in Australia; Well Begun is not All Done'.*

Over the years, the Oration Series has developed a well-deserved reputation for carrying forward Libby's commitment to raising awareness of issues relating to hearing impairment, and for furthering the aims of Deafness Forum. This is undoubtedly due to the great contributions of our outstanding Orators who have presented on such a wide range of relevant topics. We are also very gratified that it has been possible to provide the opportunity for audiences across Australia to hear these Orators, as well as to enable continuing availability via the on-going Monograph series. I would like to acknowledge the support of the Libby Harricks Memorial Oration Committee, and also that of the Deafness Forum national secretariat. I am equally very pleased to also acknowledge our Oration sponsors for 2011 Cochlear Ltd, Australian Hearing and Ai-Media. Without such sponsors, neither presentation of the Oration nor preparation of the companion Monograph series would be possible.

This year, we are privileged to welcome as our 13th Orator, Dr Robert Patuzzi. Dr Patuzzi is an academic and researcher at The University of Western Australia. He is an electronics engineer turned neuroscientist, with a Diploma in Science and Maths teaching and a PhD in Physiology. He was part of the team in the early 1980's that discovered the fundamental mechanisms of sensorineural deafness, and presently teaches physiology, molecular biology and audiology at UWA to science, medical and engineering students. He is a world authority on the hearing portion of the inner ear (the cochlea), and more recently on the human balance mechanism (the vestibular system). He has researched hearing function in animals ranging from insects to birds and mammals, the mammalian ear's response to noise trauma and drug therapies, and also the inner ear's salt and water regulation.

Ten years ago he created the Master of Audiology program at UWA, now acting as its Director of Audiology, and in that capacity he has studied many aspects of human hearing and its clinical diagnosis and treatment with his Masters and PhD students. He has been voted a life member of the Neuro-otological Society of Australia, and more recently a Fellow of the Audiological Society of Australia.

Dr Patuzzi is widely published in international journals of hearing research, and serves as a reviewer for international journals and grants. He has been State President of the Australian Deafness Council, and is presently Chair of the Scientific Advisory Panel for Australia's largest hearing research consortium (the Hearing Co-operative Research Centre or HCRC). He has a broader interest in audiological education and appropriate audiological technology, especially aspects of (remote) hearing screening. He recently received WA's Harry Blackmore Award for his community service in promoting community education in hearing issues over the years.

We are indeed fortunate that such an outstanding expert as Rob has been able to accept the invitation to speak to us about noise, its impact on hearing, and how this can be minimised.

Would you please welcome Dr Rob Patuzzi.

MOLECULES, MANAGERS OR MENTORS: HOW CAN WE MINIMIZE NOISE DAMAGE IN THE WORKSITE?

Dr. Robert Patuzzi, The Auditory Laboratory, Physiology, The University of Western Australia

Introduction

Like urban Australians, about 1 in 12 adults in Rural Australia have damaged their hearing with loud sound. The damage is almost always occupational noise-induced hearing loss (ONIHL), referred to here simply as 'noise damage'. The damage is slow, subtle and insidious: a bit like gum disease or tooth decay, but without a warning pain, and with no dentist in sight. It is permanent and debilitating, and it all could have been avoided. To put the problem in context, in 2004 the National Occupational Health and Safety Commission presented eight disease categories for particular national focus, and noise damage was third on the list[1]. The problem is particularly bad in some key populations in Rural Australia, with 60–70% of Australian farmers having a measurable hearing loss compared with 27% of the general Australian community[2], and with miners and transport workers among those most at risk of noise damage[3]. Coupled with Rural Australia's isolation, lack of services and support, ongoing problems in Aboriginal ear health, and its high numbers of vulnerable individuals in self-employment or small family businesses[i], rural hearing health is in crisis. One key document summarizing many problems in hearing health is the 2010 Senate report entitled *Hear Us: Inquiry into Hearing Health in Australia*[4]. It draws on another key 2006 document entitled *Listen Hear!*[5], a report from Access Economics on the economic impact of hearing loss in Australia, commissioned by the HEARing Cooperative Research Centre (CRC) and the Victorian Deaf Society. Both documents cover a very broad range of hearing issues, including its economic, social and psychological impact, and I encourage you all to read them. Although there are many facets of the hearing problems in Rural Australia that need discussion, I can only focus today on noise damage, and on some strategies to avoid it, most notably education. If an employer abides by the law, they are largely off the hook, and it is each employee's responsibility to preserve their own

i Because few people sue themselves, legal penalties for employers provide little incentive for hearing conservation in the self-employed.

hearing. Unfortunately the 2010 Senate report has noted that *"While the Office of the Australian Safety and Compensation Council have developed a national standard for the control of OHL*[6] *and this standard has been widely adopted into state regulations7, there is no nationally co-ordinated OHL prevention campaign."* The report also noted that in 2009 the Commonwealth's National Acoustic Laboratories found that *'there are currently no large scale, on-going general hearing health education or awareness programs in Australia'*, and concluded that *'The evidence strongly suggested the need for a nationally coordinated, adequately funded, public education and awareness campaign'*. The report also noted explicitly that there was a lack of awareness about the risks of noise damage among farmers, and that difficult access to screening and support services would hamper an effective prevention program. On that point, a recent 2009 report *'Improving Hearing Health for Farming Families'*[2] suggested that information may pass effectively through local rural networks, including those companies dealing in farm equipment and noise protection. It also suggested that there is some reason for hope, with the minimal interventions that have occurred producing an increase in the use of noise protection, and some reduced noise damage with younger farmers. Whichever education strategy ultimately yields results, I would like to tell you something about my involvement in a ground breaking schools education program in WA. Unfortunately it happened thirty years ago, and its funding was discontinued! Nevertheless, I think we can learn something from it, so I would like to retrace some of our steps from thirty years ago, update some of the information given to those children, and discuss what we did right and what we may have done wrong.

Molecules, Managers and Mentors and the Decibel Danger program

Soon after I started my career in hearing research in WA in the late '70s, I had the opportunity to help deliver a ground-breaking high-school education program called *Decibel Danger*. It covered a very broad range of hearing issues and included a strong warning to the children about noise damage. Originally known as "Boilermakers Deafness", the problem was most often associated with *obviously* loud events (like hammering or explosions), with a tell-tale 'ringing in the ears'. Over time it became clear that the damage mechanisms were far more subtle and more widespread in the community than we first thought, affecting tradesmen, farmers, miners, and even children, not just soldiers and sheet-metal workers. For many years we did not know what caused it, we did not know how to organize the workplace to minimize it, and we did not know how to

educate people to avoid it. I can tell you that thirty years ago we knew little of how our ears worked, apart from the basics of the bones just behind the ear drum, and a bit about the nerves in the inner ear. Now we understand much more, and we realize that noise damage does *not* require one-off cataclysmic events, like explosions or gun-fire, but can involve the slow biochemical 'suicide' of the ear's sensory cells with less dramatic sounds. I will explain that process shortly, when I talk about the *molecules* involved in hearing and deafness. As we have learnt more about hearing and noise damage, attempts have been made to prevent the problem with better education, better work practices, better equipment and better noise-abatement legislation, so I also need to touch on the **management** needed to avoid noise damage. The *Decibel Danger* program also touched on management issues, with talks from noise-abatement officers, and play with sound level meters. The program was revolutionary in many ways. It was privately organized but publically funded. It involved a broad range of contributors, including scientists, clinicians, and noise-abatement officers, but it also included members of the deaf community, talking about the isolation of hearing loss[ii]. This was a big break with anything that had gone before and, in my opinion, it was more effective than anything since. The power of the *Decibel Danger* program came from the emotional bond between its participants, especially between the students and the members of the deaf community, and the actors and musicians who presented the program's mime and music[iii]. The wonderful insight from the program's creator and co-ordinator, Judy White, was that children learn with their heart and soul. Presenting just *facts* about noise damage or, worse still, *fear* about it, may not convince them that the message is worth keeping. They have to form an emotional bond with a subject, largely through their bond with people. I think we all understand that from our own school days: we need **mentors** in the learning process. So I would like to talk today about the *molecules, managers and mentors* required for conserving hearing in rural Australia, and I will start by showing you why the ear is such a marvellous thing. Some of the physiological facts also help understand the management issues, and hopefully they can contribute to the motivation for hearing conservation.

ii Notably Norma Levitzke and Ursula Holcz, who in WA were similar in some ways to Libby Harricks in the east.

iii Alan Blackwood and Kavisha Mazzella provided mime/music, and other contributors included Sharon Weeks, Pam Gunn and Jack Etherington.

Molecules

How the ear works, what we damage with loud sound and what we lose as a consequence.

What I am about to summarize in 15 minutes is what I teach university students in hours and weeks, so hold on for the ride. When pressure fluctuations or sound waves travel through the air to the head, they pass the high-tech sonar dish called the *pinna* which gives us directional hearing, and travel down the funnel of the ear canal to the ear drum, vibrating it back and forwards. This wobbling is transferred through the middle ear's lever system, consisting of the three bones in the air-filled cave behind the ear drum. The three bones are the hammer (malleus), the anvil (incus) and the stirrup (stapes). Contrary to popular belief, they only vibrate by atomic dimensions with normal sound, even with intense noises. They only vibrate by more than this during the pressure trauma (*barotrauma*) associated with gas explosions or direct mechanical shock to the head (as occurs in car accidents, sport or fist fights). The bones are never broken by the ongoing loud sound in a factory or on a farm: that only damages the structures in the inner ear, as I will explain. It is also a myth that there is any pain associated with noise damage, or that loud sound causes bleeding of the ear. This is a problem, because people often assume incorrectly that without bleeding or pain they have escaped damage. It is also not true that the two muscles connected to the middle ear bones offer much protection against noise trauma: (a) they only contract with low-frequency sound; (b) they fatigue after about 5 minutes; and (c) for very brief sounds, like a hammer blow, the damaging sound has passed through before the muscles contract. The middle-ear muscles probably evolved to protect us against our own voice when shouting: because we create the sound, the muscles get sufficient warning. The important point here is that our ears did not evolve to withstand constant high-level sound: there was no such thing in the pre-industrial age, and we have no protection against it. This also puts pay to the silly notion that ears toughen with over-exposure, as if they callous in some macho fashion. There may be many evolutionary advantages to high testosterone, but ear protection is not one of them!

In any case, when the stirrup bone wobbles with sound, it pumps in and out on the watery fluid of the coiled inner ear (the snail-like *cochlea*), and the pressure fluctuations produced initiate a wave that travels away from the stirrup bone like a ripple on a pond surface, along a very delicate dividing membrane that splits the cochlea in two, called the

basilar membrane. The sound waves delicately launch from the stirrup bone at the broader high-frequency end (the cochlear base) towards the narrower low-frequency end (the cochlear apex). Because of the membrane's graded properties (stiff at the base and floppy at the apex), different frequencies are spread along the ribbon like a player-piano. The vibration is extraordinary in many ways[iv]. First, the distribution of frequency components is so effective, that a particular note or pitch of sound vibrates an extraordinarily narrow region of the ribbon. Another extraordinary aspect of the vibration is that it is so small, with 1 millionth of a millimetre corresponding to the threshold of hearing. More astoundingly, as we increase the sound level, the vibration does not grow proportionally: a million-fold increase in sound only increases the vibration 100-fold. This explains how the normal ear can cope (briefly at least) with a million-to-one range of sound (from a whisper to a jet engine) without blowing apart. It also explains why we humans had to develop the *decibel* unit for sound level: there are too many zeros in hearing[v]. For those of you who are not familiar with decibels, and therefore are befuddled by noise legislation, let me explain in 60 seconds.

If the softest sound we can hear is defined as 1 unit of sound vibration (pressure fluctuation in air), then we can easily imagine sound levels of 10, 100, 1000, 10000, 100000 and 1000000 (one million) times greater. Because we all hate zeroes, we just agree to write down only the number of zeros, and use the code of 0, 1, 2, 3, 4, 5 and 6 to represent the sound level, a bit like the Richter scale for earthquakes. But then that is a bit *too* coarse to be practical, so we all agree to use 20 units per step instead of 1, and the scale becomes 0, 20, 40, 60, 80, 100 and 120, with 0 as the threshold of hearing, and 120 as a million times more sound pressure, or roughly a jumbo jet on the tarmac at take off. And that is the decibel scale in a nutshell, with 10-fold being 20dB, 100-fold being 40dB, 1000-fold being 60dB, and so on. To give you a yard stick, if you plug your ears firmly with your fingers, giving yourself a *conductive hearing loss*, you will reduce your hearing sensitivity about 200-fold, or by about 45 dB, and that is what it sounds like to a child with their ear drums and bones glued by a middle ear infection[vi]. Sound is soft and muffled, but not distorted as it is with ONIHL, and that is a crucial distinction between these two kinds of hearing loss. Another distinction

iv I was part of the UWA team that first measured normal vibration in a living ear and discovered why we go deaf.

v Australians have a terror of nullius.

vi We still hear with fingers in our ears because the sound bypasses the block by vibrating the skull directly.

is their severity. 45dB is about as bad as a blockage in the outer or middle ear can get[vii]. If the block gets worse, then we just listen to the sound coming directly through our skull to the inner ear, via a process known as *bone conduction*. Unfortunately there is no 45dB limit to the amount of cochlear deafness that can be produced by loud sound: the more the over-exposure to sound, the more decibels we lose, and the loss is permanent and frequency-specific, and often more than 60dB at high frequencies. With a pronounced ONIHL we also lose the more subtle aspects of hearing, like pitch and speech discrimination, and our ability to cope with a wide range of sound level (see below). What we get in return is distorted sound, a warped sense of pitch and music, and sometimes ringing in the ear or *tinnitus* (Latin for 'tinkling'). The hearing loss is not at all like a simple conductive loss with blocked ears, and it is sometimes hard to get that message across.

It took us a long time to figure out why this all occurred, because the structures of the inner ear are so small and delicate (Figure 1), and the surgery for experimentation is so difficult. Perched upon the basilar membrane and detecting its vibration are two types of special cells, called *hair cells* because at the top of each is a cluster of cell protrusions called *stereocilia*, looking like fingers on a glove or hairs (making the hair cells the last of the microscopic Mohicans). One hair cell type, the *inner hair cells*[viii], sit in a single row along the whole length of the basilar membrane, and detect whatever vibration occurs. They communicate this information to the brain by releasing a chemical from their base that stimulates the neural wires into electrical activity. In fact 95% of all of the ear's nerves come from the inner hair cells, so that we effectively hear through them. Adjacent to the inner hair cells are three rows of outer hair cells, with very few neural connections and different shaped hair cuts.

Each hair cell detects its own atomic vibration using a set of microscopic trap doors at the top of each 'hair'. The vibration of the basilar membrane opens and closes the trap doors with each cycle of the sound, which produces a minute electric current by gating the salt flow through them. This then controls the release of neurotransmitter to stimulate the nerve endings attached to each inner hair cell (about 20 per cell or about 10,000 per ear).

vii The 45dB attenuation with fingers in the ears is also much greater than we can reliably get with some ear muffs and ear plugs (often only 15dB at some frequencies), so it is important to buy effective equipment, and even custom-fit gear.

viii Inner hair cells are closer (inner) to the central axis of the cochlear spiral than the outer hair cells.

Much of this was known many years ago, but for decades we did not understand the function of the other type of hair cell, the *outer hair cells*. Their mysterious role was only discovered slowly. First, in the early 1960's, children were saved from pneumonia by large doses of a new wonder antibiotic called *kanamycin*. They survived pneumonia, but were deafened in the process. Later checks showed that their inner hair cells had survived the drug, but their outer hair cells had not. Outer hair cells were clearly essential for hearing, but we did not know why. They seemed to have little to do with the ear's neural communication (they get only 5% of each ear's 10,000 nerve fibres). Then in the late '70s researchers noted a few important things in rapid succession. First, most of us have soft whistles coming from at least one of our ears (*otoacoustic emissions*), a bit like the squealing of a poorly adjusted microphone or hearing aid. Next, others noted that there were echoes coming from the cochlea that were larger than could be explained by an ear that was just pushed around passively by sound. Something strange must be occurring in our ears, but we did not know what. Then we had a breakthrough at UWA. We successfully performed the microsurgery in guinea pigs to place a radioactive speck of metal on the basilar membrane, so that we could measure its atomic movement *in a living animal*. What we found was that the vibration was 1000-times bigger than had previously been reported, because earlier researchers had caused deafness before they measured it, due to poor surgical technique. An additional observation we made was even more crucial: in our experiments it was the collapse of the vibration over time that explained completely the drop in neural sensitivity and the loss of frequency discrimination (the deafness). We had discovered that what used to be called *nerve deafness* was not nerve deafness at all. Most often the hearing loss was caused by a loss of the vibration stimulus to the inner hair cells controlling the nerves. The inner hair cells and nerves *per se* were often fine. The mystery then centred on why the vibration was so good in a normal ear. There should be too much friction with the inner ear fluid for the vibration to be so sensitive (ever tried playing tennis under water?). Finally, some colleagues from the US noted an odd thing. When outer hair cells are separated from the inner ear and are placed in a dish of salt water, they twitch when given a small electric shock, unlike the inner hair cells. It turned out that outer hair cells not only detect vibration using their stereocilia, they produce it, using their cell walls. Each is lined with a scaffolding or cyclone wire fence made from a unique protein called *prestin*, and it changes shape with the sound-evoked electrical current through the cells. We finally understood! Inner hair cells

detect vibration and stimulate nerves, but outer hair cells help inner hair cells by improving the vibration that stimulates them. Outer hair cells twitch to cancel friction, like a child on a swing. They are similar to the power steering in your car, or can be viewed as the ear's inbuilt hearing aids. When they are intact, our hearing is 60dB (1000-fold) better than we could expect. If damaged in any way, including with loud sound, they cannot help the vibration, which collapses giving deafness (try driving your car down hill with the ignition and power steering off).

What happens with loud sound to produce deafness or a NIHL?

What changes in the cochlea after loud sound to cause the outer hair cells to malfunction? For moderate over-exposure we suffer a temporary threshold shift or TTS, because the outer hair cells' molecular trap doors twist out of shape for a day or so before recovering. With more sound, ONIHL is produced because the hair cells cells die. Whether the disruption to outer hair cells is temporary or permanent, they are unable to cancel friction, and the vibration becomes much smaller and less localized along the length of the basilar membrane for a particular frequency of sound. With those changes, once amplified to be heard, a particular musical note or frequency component in speech stimulates too many neurones along the inner ear's length. Because of this excessive spread of neural excitation, we lose part of our frequency discrimination and mis-hear musical notes. In some cases the notes can move up in pitch by half an octave (about four white keys on a piano), which is devastating for a music lover, especially when one ear is damaged but the other is not[ix]. This results in two *different* notes being heard in the left and right ears (so-called *diplacusis* or two-toned hearing). There is also a loss of the cochlea's ability to squeeze a million-to-one range of sound level into a mere 100-to-one range of vibration amplitude. This means that if we use a hearing aid to amplify the soft sounds, we will hear an uncomfortable distorted sound. What once was heard as delicate fingers on the inner ear's player piano ends up as bombastic elbows, slamming wildly on the keyboard. This phenomenon is known as (*hyper)recruitment*, because the nerves are inappropriately recruited into firing in the damaged ear, when they would not in the normal ear.

The good news is that we now understand all of these issues, and can partially compensate for some of the changes with sophisticated and well-fitted hearing aids. The bad news is that the damage is permanent.

ix This often happens to farmers who get right ear damage from continuously looking back in loud tractors, or with rifle fire where right-handers get damage to their left ear that is closer to the muzzle.

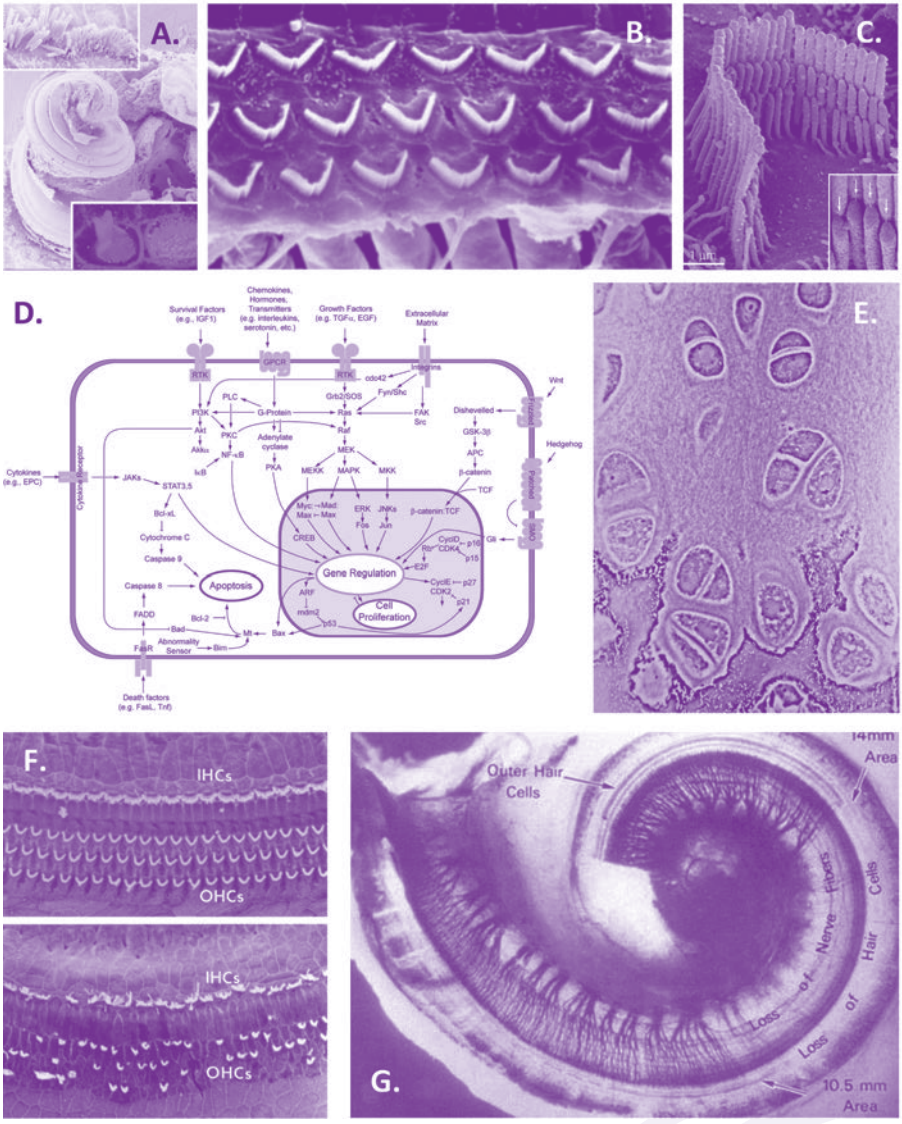

Figure 1. **A.** The spiral-shaped cochlea is separated in two along its length by the elegantly vibrating basilar membrane. Its vibration is detected by thousands of hair cells in neat rows along the spiralling basilar membrane. **B.** The three rows of outer hair cells shown here with their 'W' shaped hair bundles twitch in time with the sound vibration as it passes on the basilar membrane, cancelling friction and increasing it 1000-fold (60dB). **C.** Their twitching is synchronized by a small electric current caused by the opening and closing of molecular trap doors at the tip of each hair bundle that produces a pulsating salt flow. This electrical signal controls twitching in outer hair cells, and the release of chemical to control nerves in the single row of adjacent inner hair cells, but somehow **(D)** it also can trigger the complex chain of chemical events that leads to hair cell suicide (apoptosis), and ends with the cells breaking apart into **(E)** 'apoptotic bodies' that are eaten by white blood cells. **F.** The four rows of hair cells (upper) degenerate (lower) with ONIHL, first the outer hair cells and then **(G)** inner hair cells and their attached nerves, leaving even worse ONIHL.

We do not yet understand the link between the temporary changes producing temporary hearing loss and the more permanent cell death. We do know that within each cell is a system of chemical messengers, checks and balances, and that when given the appropriate signal, key chemicals cause the disintegration of essential components in the cell. This sequence of events and the ultimate cell death is known as *apo-(p)tosis*[x], which can occur usefully in other places in the body[xi]. It may be hard to accept it as you look around the room, but we are all born with far too many neurones for our own good. Part of the process of maturation as an infant is the systematic culling of unnecessary neurones, leaving the fully functioning nervous system. It is a bit like neural sculpture, or brain witling. As another example, in utero apoptosis also creates the gaps between our fingers and toes. Ultimately we need a balance: too much apoptosis and we waste away, too little and we have unbridled cell proliferation, or cancer. The problem with loud sound is that it somehow encourages apoptosis of the hair cells, which never happens in a normal ear. We are given one set of hair cells at birth, and that is our lot for life... so far at least. Chickens *can* replace their damaged hair cells, so some researchers are trying to 'chickenise' human ears by fooling human hair cells to regrow. This is very hopeful, because uncontrolled growth would lead to an inner ear tumour or very abnormal hearing. Overall, it is not yet clear how the electrical and chemical changes that accompany loud sound over a long time trigger apoptosis, but we are beginning to understand how it might be avoided, perhaps using drugs known to block apoptosis in other tissue. Some favourites recently are aspirin and the anti-oxidants (of course). In the end, noise abatement legislation has a 140dB peak limit on transient sounds to avoid barotrauma, and a time-intensity trade-off provision to avoid apoptosis.

x John Kerr at the University of Queensland is recognized as an early influential researcher of apoptosis, publishing a 1972 article in the British Journal of Cancer that first used 'apoptosis' in this context, which translates from Greek as the "dropping off" of petals or leaves from plants.

xi Although many factors trigger apoptosis, there is only one way that it unfolds. After a cell receives the suicide command, its parts (organelles) degrade systematically under the influence of 'activated proteolytic caspases'. These are a bit like the enzyme or catalyst in BioAd stain remover. As the caspase enzymes act to dissolve the cell's protein structures, the cell shrinks and becomes rounder with the breakdown its internal skeleton; its cytoplasm (soup) appears dense, because organelles tightly pack together when the protein motors keeping them apart malfunction; DNA clumps into patches against the nuclear envelope (pyknosis), and both soon break up (karyorrhexis); the cell membrane then protrudes or 'blebs' like the fingers on a glove, and finally the cell breaks into large pieces called the 'apoptotic bodies', which are eaten quickly by nearby white blood cells. This overall process is so fast that it is difficult to see it in action.

Managers

Managing the worksite, the people and the equipment

There are many in this audience who are far better managers than I will ever be, so I will simply mention a few key points in the management of noise. First, the cause of noise damage is well known. It's NOISE! So anything that decreases exposure to loud sounds is a help. Because we cannot engineer a silent workplace, we will always need equipment and processes for noise-reduction, and legislation to encourage it, but we also need co-operation. As for the processes and management strategies to reduce noise exposure, it is best to follow what is known as *the hierarchy of control*, as summarised in Table 1.

5.2.4 MANAGING EXPOSURE THROUGH EFFECTIVE USE OF THE HIERARCHY OF CONTROL

The hierarchy of control covers a number of potential activities:

- Elimination (**Do you have to use that noisy thing 'round here?**)
- Substitution (**For gords sake, use the new one, it's a lot quieter, ...**)
- Engineering controls (isolation, engineering control at source, engineering control in transmission)
  (**...and if you do use it, stick it at the back of the shed, put the muffler back on, and chuck a box over the top.**)
- Administrative Controls (**Don't go anywhere near that thing...**)
- PPE – Hearing Protection (**...and if you do, wear ear plugs.**)

Table 1. The 'hierarchy of control', as listed in a document from the Government's Safe Work Australia[3] entitled *'WorkRelated Noise-Induced Hearing Loss in Australia*'. I have taken the liberty of translating their five descriptors into RuralSpeak in bold.

Unfortunately some managers jump straight to issuing hearing protection, forgetting that elimination and substitution should be their starting point. I once worked as a sheet metal worker in a factory where the management failed to change the bearings in a belt grinder for months, even when the workers complained of the noise. The 'Elimination' in this case was simple maintenance. They certainly never issued hearing protection. If they had, it would have been important to choose the right type. It needs to be high quality to attenuate the sound significantly, and there often needs to be a range of equipment to match the task at hand, or the working conditions on the day. A great deal of gear has been

issued, never to be used, because it was too heavy, too hot, too sweaty or too obstructive of other equipment, like safety helmets or communication gear. Good management in this case is presumably getting good advice and listening to workers when they tell you what works and what does not. As for 'administrative controls' we mean the work protocol, perhaps preventing staff from entering areas during noisy work, or rostering to share exposure more equitably across staff, or studying the worksite well to determine what is really the problem. We recently monitored sound levels in detail for staff in entertainment venues, and discovered that the loudest sounds did not actually come from the music, but came from the patrons shouting into the ears of the staff so that they could be heard above the music. A close assessment of your own work conditions should be instructive. Overall, the aim of the game is to avoid intense one-off exposures (they must be less than 140dB at their peak to avoid barotrauma), and reduce all staff cumulative noise doses to below an equivalent of 85dB over a 40 hour working week to avoid apoptosis. Based on epidemiological data and the push-and-shove of industrial relations (and perhaps limited arithmetic capacity in the legal profession), every 5dB increase in noise dose requires a halving of exposure time[xii]. By this rule, an 8 hour equivalent dose must be reduced to only 2 hours per day if the sound level rises from 85dB to 95dB (a two-fold time factor per 5dB), and to 1/2 hour if it rises to 105dB, and so on.

There are, of course, many other aspects of the management of ONIHL, including hearing screening, staff training and worksite enculturation, but in my remaining time I would like to talk about people's motivation to avoid noise damage.

xii Earlier, it was assumed that equal damage was done by equal sound energy. The maximum allowed dose was set at 90dB for a 40 hour week, and the exposure time was halved for each 3dB rise in sound level (ensuring equal delivered energy). This "equal energy principle" was wrong: intermittent sound is less damaging than continuous sound of the same energy; sounds with multiple frequencies or noise produce less damage than a pure tone of the same energy; and sound to both ears produces less damage than sound to a single ear because of nerves that cross from one ear to the other. This failure of the 'equal energy principle' partly explains the change from a 3dB rule to a 5dB rule. The legislation still takes no account of the complexity of the noise damage process, because such laws would be unusable.

Mentors and Motivation

Where there is a will, there is a way ...but where there is a way, there is not always a will...

In the end, whether self-employed or not, the biggest problem in reducing noise damage appears to be people: they do not always behave in their own interests. As an example, let me tell you a tale of three 'chippies'. My sister married the son of a carpenter, who became a carpenter, and she gave birth to a carpenter (apparently it is genetic). All three generations of carpenters have given themselves a pronounced hearing loss at work. How can that happen, especially with me in the family? First, all of them are self-employed, as are many people in Rural Australia, and legislation that penalizes employers would seem powerless to change the behaviour of the self-employed: people do not sue themselves. So what could have stopped my carpenters from damaging their ears? Had we educated them sufficiently to avoid the problem? In the case of the two oldest, I think the answer is a clear 'no'. None of their beliefs was formed with the help of a formal education process, but evolved on the job with few good examples (and possibly many bad ones). They had mentors, but none who were interested in hearing conservation (and possibly some who believed in testosterone toughened ears). Unfortunately, there is another problem. When a worker becomes aware that they have a hearing problem, they may give up too easily, assuming that it is pointless to continue hearing conservation, or even start[xiii]. This is never so. The loss of hair cells and neurones is gradual. Although outer hair cells are damaged first, dropping vibration and changing perception as described earlier, some inner hair cells remain, providing some degraded hearing. If people do feel disempowered, and abandon hearing protection too early, they risk losing their inner hair cells and neurones too, so nothing is heard at those frequencies and no hearing aid can help. It is *never* too late to save some hearing.

Although our ignorance of noise damage may have been an excuse many years ago, I think it is not now. As a community I think we failed my youngest carpenter, with little excuse. By the time he reached high school

xiii Of course, some use the 'hopelessness' of their situation to justify their own laziness in using noise protection, or comfort themselves with the machismo of their deafness, or take solace in their conformity to their trade 'identity'. Whichever, it is never too late to save some hearing.

we knew a lot more about hearing and noise damage, but the *Decibel Danger* program had ended: thirty years earlier we could not convince the Education Department that the ongoing funding was worthwhile, despite there being only one full-time staff member. Over the years I had suggested to him that he wear ear protection, but I was not with him at his worksite, and uncles, whatever their profession, may not have the necessary authority. Clearly young workers need to have good information, but in the end they have to think that hearing protection is worth it. This is the crucial point: people continuously make cost-benefit analyses in real-time, so we need to understand the (il)logic of their decision making?

In psychology there are many models of human behaviour, but one that fits hearing conservation well is *The Theory of Planned Behaviour*. Put simply, it assumes that there are three interacting components to an individual's planned behaviour: (a) their behavioural beliefs (*'wearing ear plugs will protect my hearing'*) and their attitude towards that belief (*'...but a real carpenter has damaged ears'*); (b) their normative beliefs (*'most people wear ear plugs'*) and their personal norm (*'...but I am a rebel with tougher ears than most'*); and (c) their control beliefs (*'there are plenty of earplugs available on site'*) and their perceived ability to use that control (*'...but I can't wear them because people will think I look stupid'*). Combined, these components generate an individual's intention to behave in a certain way, but depending on circumstances they may or may not be able to carry out that intention (*'I want to wear my earmuffs, but Trevor has taken off with the truck and the gear'*).

In the case of my nephew, he believes that noise is damaging and that ear protectors work (his behavioural beliefs), and he has ear protection of various kinds, but he does not use them routinely. What, if anything, is going through his head when he does not? I know that there may be an issue in his normative beliefs (real carpenters are tough, do not wear ear protection, and many good ones are deaf, like his father and grandfather). I also know that there is a practical problem putting ear muffs on and taking them off as required, and that they can be hot and sweaty. So although he believes that the behaviour is good for him, his attitude to doing it is not good. When analysed at this level with the

Theory of Planned Behaviour, what prevents an *individual* from achieving hearing protection becomes clearer. I would like to emphasise this point: each individual is different, so it is possible that more than one education strategy is needed, even within a single cohort (e.g. carpenters)[xiv]. In the end, it is clear that my nephew knows what to do (unlike his father and grandfather), but like so many others in his position, he does not judge the protective behaviour to be worthwhile *at the time*. At that instant, before starting the power saw or striking a nail, the costs outweigh the benefits: it is just *too* hot, *too* sweaty and *too* inconvenient to use the protection, especially when the noise damage and loss of hearing is so slow and insidious. Perhaps a personal dosimeter and sound level alarm would be useful, as would a workplace culture of taking the time to use the protective equipment. Whichever technology might help, *he needs good mentors*, and that includes good teachers in the education system, and sufficient information and resources to keep them effective.

On the matter of customizing any formal education strategy, I suspect that preaching the message of doom and gloom to young people will fail. It did not work with the rock revolution, so why should it work now. Most MP3 users are at an age where non-conformity is normal and healthy. Rather than telling them what not to do, they need to be motivated by the joy of hearing and the benefits it brings. People prefer to be given reasons for doing the right thing. I would certainly caution against scare tactics in hearing conservation, especially for young people, and I would especially warn against a proselytizing tone. Although there is some evidence that recreational music devices have the potential to damage hearing, it is patchy, the exposure is limited to a particular age group, there is some evidence of improved behaviour already, and the cumulative exposures are unlikely to come close to the over-exposures possible at work. Research into the possible damage should continue, and limits on peak levels are essential, but preaching the gospel of MP3 temperance runs the risk of alienating young people, just when they need positive mentors, not nagging nannies. There are better battles to fight (and win) in the war on noise damage.

xiv I encourage you all to view a superb internet talk by the journalist/writer Malcom Gladwell about segmentation in markets and the work of Howard Mosckowitz on spaghetti sauce at http://www.ted.com/talks/malcolm_gladwell_on_spaghetti_sauce.html

Closing remarks

While the incidence of ONIHL is bad, is it increasing or decreasing? It is certainly true that no-one is aghast at how rapidly things are improving. One indicator is discussed in the Senate Committee report: the number of compensation claims over the last few years (Table 2). At first glance, things appear to be getting better, but the numbers may be deceptive. First, there is a long delay between the behaviour and conditions in the workplace and the completion of a law suit for ONIHL compensation. Part of the drop is certainly due to the retirement of older and more-severely noise-damaged workers, and may also reflect changes in work practices some (many) years ago. Second, the large drop from 1998 to 1999 (6,156 to 4,305) was partly due to the redefinition of a compensatable hearing loss in some jurisdictions, so that twice the hearing loss is required for a payout. Third, there has been a progressive casualization of the workforce, distributing responsibility across more employers, making it increasingly difficult to mount a compensation case, especially by uninformed workers. Finally, we need to remember that improved legislation can encourage less law suits, because compliant employers are protected legally, but less law suits and more equipment does not guarantee less damage. Staff need to use the equipment provided and follow directions, but with widespread self-employment in the rural sector, education and motivation would seem to be the key (not legislation with penalties for employers).

TABLE 2-2: PAID WORKERS COMPENSATION CLAIMS FOR OHL, 1998-2003		
YEAR HEARING COMPENSATION CLAIMS		
1998	6,156	
1999	4,305	abrupt '98-'99 drop partly due to a rise in legal requirements: 10% loss rather than 5% loss
2000	4,213	
2001	3,973	otherwise ongoing improvement with drop in compensation claims
2002	3,811	
2003	3,041	
Source: National Data Set for Compensation Based Statistics, OASCC special data request.		

Table 2. Change in Workers Compensation Claims for ONIHL from 1998 to 2003. As noted in the text, the drop may be deceptive.

I would like to end by noting that the children we talked to in *Decibel Danger* have grown, had families, and now work in responsible positions. Did our message have any impact on them? We do not know. What we do know is that we were surprised for years after by greetings in supermarkets, that generally went *"Hey, how are you? Remember me from..... You talked to us about the ear and deafness"*. We guess that something must have stuck, but we do not know what, nor how it helped them. Our *assumption* at the time was that if we told them the truth about loud sound in an interesting way, and explained the consequences of hearing loss, they *would* protect their hearing, but we have no data to prove it. Thirty years ago we knew very little about the physiology of hearing and noise damage, and equally little about the psychology of motivation. Any new education initiative to address our present problem would be advised to plan its strategy very carefully, using market segmentation and recent models of motivation, and it should have an outcomes assessment. How that could be done is unclear. I believe that the WA *Decibel Danger* program was well ahead of its time, and could act as a model for *at least* high-school education across Australia, including rural areas, but a lot of work is needed.). Again, I encourage you to download and read the Senate Committee on hearing impairment and its sad impact on individuals and their family and friends, and its economic impact on them and society generally. The report is a credit to the Senate Committee, and to the political process that spawned it. I hope that you have learnt something new about the Molecules, Managers and Mentors of hearing and noise damage, and that you look for opportunities to improve matters. Thank you for my opportunity to talk to you.

Endnotes

1 work-related musculoskeletal disorders; mental disorders, noise-induced hearing loss; respiratory diseases; occupational cancers; contact dermatitis; infectious and parasitic diseases, and cardiovascular disease.

2 http://www.rrh.org.au/publishedarticles/article_print_1350.pdf

3 http://www.safeworkaustralia.gov.au/AboutSafeWorkAustralia/WhatWeDo/Publications/Documents/418/WorkRelated_Noise_Induced_Hearing.pdf

4 http://www.aph.gov.au/senate/committee/clac_ctte/hearing_health/report/report.pdf

5 http://www.audiology.asn.au/pdf/ListenHearFinal.pdf

6 http://www.nohsc.gov.au/OHSLegalObligations/NationalStandards/NOISE.htm

7 http://www.workplace.gov.au/workplace/Category/Publications/WorkplaceRelations/WorkplaceRelationsMinistersCouncil-ComparativePerformanceMonitoringReports.htm

About the Deafness Forum

Introduction

Deafness Forum is the peak body for deafness in Australia. Established in early 1993 at the instigation of the Federal government, the Deafness Forum now represents all interests and viewpoints of the Deaf and hearing impaired communities of Australia (including those people who have a chronic disorder of the ear and those who are DeafBlind).

Structure

Deafness Forum is divided into four classes.

Consumer means an adult who is Deaf or has a hearing impairment or has a chronic ear disorder; or a parent of such a person.

- **Deaf** refers to people who see themselves as members of the Auslan using Deaf community by virtue of its language (Auslan) and culture.
- **Hearing Impairment** refers to a hearing loss. People with a hearing impairment (or who are hard of hearing) may communicate orally (sometimes described as 'oral deaf') or may use a sign language or other communication methods.
- **Chronic Ear Disorder** refers to such disorders of the ear as tinnitus, Meniere's Disease, Acoustic Neuroma, hyperacusis and recruitment. People with some such ear disorders may also have a hearing impairment.

All Consumers are entitled to describe themselves using whatever terminologies they prefer, and are asked to do so at the time of joining and each time they renew membership.

Consumer Association means an incorporated Association of, or for, consumers (as defined above).

Service Providers also include various other occupations that provide services to consumers who are Deaf, have a hearing impairment or have a chronic disorder of the ear.

Service Provider Association means an incorporated organisation, which has (as its principal purpose) the provision of services that promote the wellbeing of consumers (as defined above).

Objectives

The Deafness Forum exists to improve the quality of life for Australians who are Deaf, have a hearing impairment or have a chronic disorder of the ear by:

- advocating for government policy change and development
- making input into policy and legislation
- generating public awareness
- providing a forum for information sharing and
- creating better understanding between all areas of deafness.

Community Involvement

The Deafness Forum is consumer driven and represents the interests and concerns of the entire deafness sector, including:

- the Deaf community
- people who have a hearing impairment
- people who have a chronic ear disorder
- the DeafBlind community
- parents who have Deaf or hearing impaired children in their families

Libby's Story

Libby's story is one of courage and triumph over adversity by utilising the knowledge of her own severe hearing loss to help others.

Libby started to lose her hearing following a bad dose of flu in the English winter soon after her marriage in 1969. Having returned to Australia in 1970 she began to find difficulty in understanding conversation and instructions, particularly on the telephone which was very important in her profession of pharmacy.

In spite of advice to the contrary, Libby tried hearing aids and found they helped. Had she heeded the negative advice, Libby believed she might never have embarked on the road to self-help, which so enriched her own life and that of many others. She thought her two boys quickly learnt to sleep through the night and her friends remarked they had loud voices, which was the boys' mechanism for coping with a deaf mother!

The more the doctors said nothing could be done to help, the more Libby looked towards self help and so she learnt to lip read, a tool she relied on heavily in her quest to help others.

Libby's will to win led her, with the help of others, to get involved with the setting up of a support group, which became SHHH – Self Help for Hard of Hearing people. The American founder, Rocky Stone, was invited to Australia in 1982 and did a lecture tour entitled "The Hurt That Does Not Show" which cemented the bonds between the US and Australian groups and helped the local SHHH develop.

Libby, with others, then began SHHH News, a quarterly publication, and with Bill Taylor set up the first Hearing Information and Resource Centre at "Hillview", Turramurra with support from Hornsby/Kuringai Hospital. This centre provided reliable information on, and demonstrated, assistive listening devices for hearing impaired people. Through this interest, Libby became an enthusiastic user of technology and with her handbag full of electronic aids was enabled to join in a full social life with family and public.

Libby became President of SHHH in 1986 and began to develop her role as an advocate for hearing impaired people generally.

She became involved in ACCESS 2000, under the Australian Deafness Council, and a member of the Disability Council of NSW. Her horizons broadened further as Vice President of the Australian Deafness Council and then as the first, and two terms, President of the newly formed national peak body in deafness, the Deafness Forum of Australia. In this latter role Libby made a huge contribution to bring together all the different organisations into a central body, and actively lobbied on behalf of Deaf and hearing impaired at the highest level – the archetype of a successful achiever despite her profound hearing loss.

For her work on behalf of hearing impaired people Libby was made a Member of the Order of Australia in 1990. Later she was appointed by the Government to the Board of Australian Hearing Services and was asked to represent the needs of hearing impaired on the Olympic Access Committee.

Unfortunately, Libby faced another hurdle when she was diagnosed with breast cancer in 1995. Following surgery, she continued her family and volunteer work with undiminished vigour. She would wickedly show off her wig at public functions after her chemotherapy, and talked openly of her "mean disease". She died peacefully on 1 August 1998 and was honoured by hundreds who attended her Thanksgiving Service on 6 August.

In her own words, Libby related her outlook:

"I look back over these years since I became hearing impaired and realise that any efforts that I have made have been returned to me threefold. I have found talents I never knew I had, I have gained so much from the many people I have met and worked with to improve life for people with disabilities and through self help I have turned the potential negative of a profound hearing loss into a positive sense of purpose and direction in my life".

The Libby Harricks Memorial Oration

The Libby Harricks Memorial Oration program is supported by the Libby Harricks Memorial Fund of the Deafness Forum of Australia. Donations to this fund are tax deductible.

Donations should be made payable to Deafness Forum. Additional donation forms and general information regarding deafness can be obtained from:

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