

The 2000

# Libby Harricks

## Memorial Oration



Libby Harricks Memorial Oration number 2

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

**Elisabeth Ann Harricks AM** 1945 – 1998

Published by Deafness Forum Limited  
January 2001

Designed by Design Edge

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## 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

The representational base of the Deafness Forum is divided into five Sections:

- a) Hearing Impaired Section - persons with a hearing loss who communicate predominantly orally,
- b) Deaf Section - i.e. the Deaf Community - those persons who consider themselves to be members of that community by virtue of its language (sign language known as Auslan) and culture,
- c) Ear Disorders Section - persons with a chronic ear disorder (such as Tinnitus, Meniere's Disease or Acoustic Neuroma) and
- d) Parents section - parents or legal guardians of persons who are Deaf or hearing impaired,
- e) Service Providers section - service providers to the Deaf and/or hearing impaired communities.

### 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



# The 2000 Libby Harricks Memorial Oration

Delivered by Professor William P R Gibson AM, MD, FRACS, Professor of Otolaryngology, University of Sydney, Royal Prince Alfred Hospital Sydney on Tuesday 4 July 2000



## Recent Advances in the Understanding of Menière's Disease and Tinnitus

It is a great privilege to have been asked to present the second lecture which honours Libby Harricks. I will always remember her as a very elegant lady who took such pleasure in thrusting her microphone under my chin while we were talking. She was always interested in the cochlear implant, but remained cautious. As the first chairperson of the Deafness Forum, she was always anxious that I should not upset the Deaf members who feared the cochlear implant would change their world. Her caution was wise because we now know that the cochlear implant provides little or no benefit for those who were born deaf and have reached adult life using sign rather than speech to communicate. The use of cochlear implants for deaf children who are young and still can develop listening and speech is more contentious, although the results now literally speak for themselves. For adults who have become deafened later in life, the cochlear implant has proved to be of great benefit.

Libby came to see me asking to begin the cochlear implant assessment program in 1997. At the consultation she complained of a persistent cough so I ordered the chest X-ray which showed her breast cancer had spread into her lungs. She had chemotherapy with all the horrid side effects like loss of hair. Optimistically we still talked about the day she would get her cochlear implant but we both knew it would never be. She was amazingly brave and down to earth. When she died it was a terrible loss to us all and it must have been an unbearable loss to David and her family. I attended her funeral a few days after all my hair had been removed for a neurosurgical procedure to remove a bilateral sub-dural haemorrhage. I know she would have enjoyed ribbing me at losing all my hair. My lecture is dedicated to her memory, which remains so vivid today.

## Menière's Disease and Tinnitus

Last year I attended the 200th anniversary of the birth of Prosper Menière. He died of pneumonia in 1862; the year after his article describing Meniere's disease was published. After several posts, he became the director of The Institute of Deaf Mutes in Paris. He was an oralist despite the fact that the Institute had been founded by L'abbé de l' Épée, who introduced the first formal system of sign language. The disciple of L'abbé was Gallaudet who left to found the Gallaudet College in Washington, USA. Prosper Menière's other interest was obviously Menière's disease.

Before Menière's description, the attacks of vertigo associated with Menière's disease were thought to be due to cerebral apoplexy - or a 'stroke'. It is easy to understand this as many people when they suffer their first attack of vertigo are convinced they are having a 'stroke'. The attacks of vertigo cause a sensation similar to stepping off a child's roundabout with a feeling the world is spinning, which can last for several hours. The affected person is nauseated and usually vomits- sometimes even suffers diarrhoea. The attacks occur in clusters with variable periods of remission between each cluster. The condition is associated with a feeling of fullness and tinnitus in the affected ear. Initially (stage 1) the hearing remains normal between attacks. In many cases the attacks will cease eventually and a long period of remission occurs. About 50 per cent of people progress to stage 2. In stage 2, the hearing is constantly affected but fluctuates in level. Eventually, in stage 3, the hearing becomes very poor. The severity of the vertigo is worse at the onset (stage 1) and gradually the attacks become less severe as the disease progresses. At stage 3, the attacks of vertigo are usually very mild or non-existent.



Menière's great contributions were to give the first accurate description of the disease and to attribute the disease to the inner ear. Only a few years earlier, another Frenchman, Flourens, had shown that the ear was involved in maintaining balance as well as being a hearing mechanism. Flourens described his experiments with pigeons when he destroyed the pigeon's ear with a red-hot wire and then watched the hapless birds unable to control their balance. Menière was the first to apply this basic research to a clinical disease.

The treatment of Menière's disease in the late 19th century consisted of purging and leeches placed behind the ear. In the early 20th century was based on a strong belief that the circulation of the inner ear was faulty. Nicotinic acid was prescribed by doctors which caused their Menière's patient to flush like beacons, and a popular surgery was cervical sympathectomy which dilated the blood vessels in the ear but also dilated the pupil of the eye and caused the upper eyelid to droop. Menière's sufferers, today, are fortunate to be able to receive betahistidine (Serc®).

The next breakthrough was when endolymphatic hydrops was shown to occur in Menière's disease. The membranous inner ear or labyrinth (figure 1) is located within the otic capsule, which is the hardest bone of the skull. The membranous labyrinth has two main parts: the cochlea, which relates to hearing; and the semicircular canals, which relate to balance. The fluid surrounding the membranous labyrinth is called perilymph. The fluid of the membranous labyrinth is endolymph, which is rich in potassium. Too much endolymph causes a ballooning of the membranous labyrinth and is called 'endolymphatic hydrops' (figure 2).

The discovery of endolymphatic hydrops in ears affected by Menière's disease is usually attributed to Hallpike and Cairns who published their findings in 1938. Charles Hallpike was a brilliant Englishman who undertook the preparation of the specimens. 'Cairns' was Sir Hugh Cairns an Australian neurosurgeon who was interested greatly by the disease. Actually, in the same year a Japanese surgeon, Kumagawa also published a case in a German magazine in Japanese. It was not the optimal place to publish in those days and the article was unnoticed in the Western world for many years.

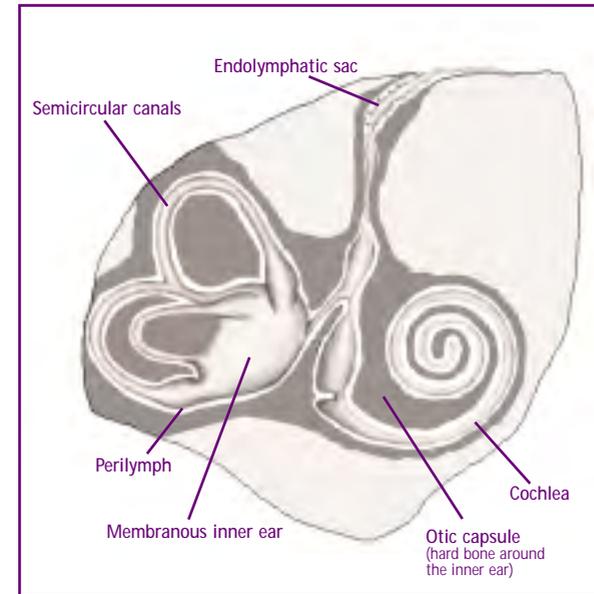


figure 1

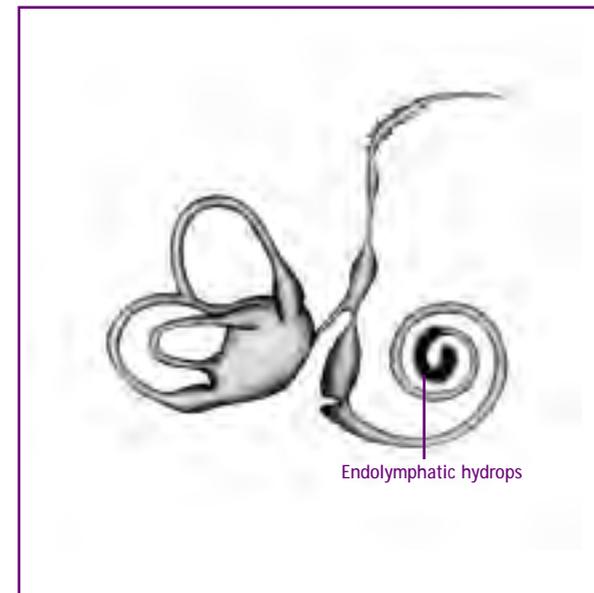


figure 2



The relationship of endolymphatic hydrops to Menière's disease remains a contentious issue. In the past animal experiments have been performed. It was shown that removal of the endolymphatic sac in guinea pigs caused endolymphatic hydrops to occur after 2-3 weeks. However none of the animals ever suffered from attacks of vertigo. In human autopsies, ears that had clearly been affected by early (stage 1) Menière's disease rarely show any evidence of endolymphatic hydrops although the majority of stage 2 and stage 3 ears do show hydrops. Thus the question was asked, 'Is endolymphatic hydrops only the consequence of the disease rather than the cause?'

A theory was suggested by Dr Harold Schuknect to explain the disease. He suggested that if the endolymphatic duct became blocked, endolymph was unable to drain out of the ear adequately resulting in hydrops. This would lead to a rupture of a delicate membrane in the inner ear (Reissner's membrane) with mixing of the potassium rich endolymph fluid with the sodium rich surrounding perilymph fluid. This mixing of the inner ear fluids paralysed the nerve connections in the ear causing the attack of vertigo. The ear then sorted itself out and the rupture rapidly repaired itself terminating the attack. Based on the rupture theory, endolymphatic sac surgery was designed to unblock the endolymphatic duct and to drain the excess endolymph from the inner ear; thus preventing the attacks of vertigo. It is moderately successful but many patients do suffer a recurrence of the attacks after several months or even years.

Schuknect was such a great man and such a leading figure in otology that theory became the accepted. Nevertheless, over the last 25 years, further basic science has been undertaken which seriously challenges these concepts.

In 1977, Gibson, Moffat and Ramsden were the first to describe electrophysiological changes in human ears affected by Menière's disease. I studied electrocochleography in Bordeaux in 1972 and then undertook my MD thesis at Guy's Hospital in London. Later, with my colleagues, I went to show that the presence of endolymphatic hydrops caused a distortion of the electrical potentials that could be obtained from the inner ear. The main change was an increase in the summing potential that was

produced when the basilar membrane on which the hair cells lie, vibrates asymmetrically. I developed a method of comparing the amplitude of the action potential and the summing potential (SP/AP ratio) which was soon adopted as the main method for using electrocochleography for the diagnosis of Menière's disease. The transtympanic test involves placing a needle electrode through the tympanic membrane and this proved unpopular in the USA where an electrode is placed in the ear canal. Unfortunately external ear canal or extratympanic electrocochleography rarely provides a clear-cut result and it is only possible to use click sounds to test the ear. I have shown that sounds (or stimuli) composed of bursts of pure tone are about twice as accurate as clicks in diagnosing endolymphatic hydrops. Currently about 80% of ears affected by Menière's disease show the presence of abnormal tone burst summing potentials on transtympanic testing.

If electrocochleography shows the presence of endolymphatic hydrops, the relationship of endolymphatic hydrops to the attacks of vertigo can be shown. Electrocochleography reveals that endolymphatic hydrops builds up before attacks, gradually reduces during the attack and then rebuilds at a variable rate after the attack. At least 24 hours is needed to refill the ear before the next attack of vertigo can occur.

Dr Alex Salt has undertaken some very important work on how endolymph is produced and how it circulates. He has shown that in guinea pigs, the composition of endolymph is maintained by the stria vascularis. This is a big mass of blood vessels, which run along the entire cochlea. The stria vascularis controls the influx of water and modifies the ionic content. Normally any excess endolymph is reabsorbed back into the stria vascularis (radial flow). Only under exceptional circumstances does the endolymph move longitudinally to the endolymphatic sac. Humans can maintain the balance of endolymph by the radial mechanisms alone and rarely have to call upon the endolymphatic sac.



Another outstanding contribution has been from workers in Scandinavia who have described the ultrastructure of the endolymphatic sac. The endolymphatic sac has been shown to have a structure like a sponge and does not have an empty centre like a sac. The endolymphatic sac secretes various substances. One of the substances, called glycoprotein is highly hydrophilic and probably attracts endolymph into the endolymphatic sac when it is secreted. Thus the longitudinal flow of endolymph to the sac is an active process. The endolymphatic sac becomes damaged in stage 3 Menière's disease, perhaps because of excessive glycoprotein activity.

The latest discovery is by Rask Andersen from Sweden who has described pressure receptors on the round window membrane. It is thought these pressure receptors may be involved in the feeling of aural fullness felt by many Menière's sufferers and have a role in precipitating the attacks.

Since 1991, I have been removing the endolymphatic sac in ears affected by Menière's disease. This is a highly controversial move, as the conventional surgeons still believe they can cure Menière's disease by draining the endolymphatic sac. The finding has been that removing the endolymphatic sac is at least as effective as any drainage procedure and less likely to result in a late recurrence of symptoms. The main problem in humans is that it is difficult to remove as in some cases much of the active portion of the endolymphatic sac is buried within bone and cannot be safely removed. Thus the best that can be accomplished is a partial removal of the endolymphatic sac.

So I would like to conclude the Menière's portion of this oration by presenting my theory which explains Menière's disease which was based on all the facts that I have described previously.

## The Drainage Theory

The drainage theory is based on a new concept of endolymph physiology. Figure 1 showed the shape of the membranous inner ear, which contains the endolymph. Normally, the volume of endolymph is maintained by radial flow or transportation of fluid in and out of the blood vessels inside the membranous labyrinth (stria vascularis). In normal ears, daily situations arise when too much endolymph exists and usually increased radial blood flow clears this excess. Rarely in humans, when radial flow cannot cope, the endolymphatic sac can act by secreting a hydrophilic protein (glycoprotein) which attracts endolymph into the lumen of the endolymphatic sac (longitudinal flow) where it is degraded and absorbed.

The endolymphatic sac can also act when debris is trapped within the membranous labyrinth, perhaps as a result of a virus infection. Somehow the endolymphatic sac senses the presence of the viral debris and secretes a hormone, called by Danish researchers - 'Saccin'. Saccin increases endolymph production causing some hydrops. Then the endolymphatic sac secretes glycoprotein and attracts the extra fluid, shifting the viral debris into the endolymphatic sac lumen.

What a clever endolymphatic sac!

So what happens in ears affected by Menière's disease? Firstly I believe that a narrow bony vestibular aqueduct is a pre-requisite for the disorder. This may account for the differences in the prevalence of the disease among different races. Menière's is most common Scandinavian countries and very rare in black races. Blue eyed, fair-haired people seem the most likely to suffer from Menière's disease.

First there may be a preliminary stage when an episode occurs in such an ear causing debris to accumulate within the membranous labyrinth. The endolymphatic sac acts in the usual way to clear this debris but the debris gets trapped in the narrow duct leading towards the endolymphatic sac. The ear reacts with the secretion of more endolymph and more glycoprotein until finally the obstruction is overcome and the debris and excess fluid shifts. This movement of fluid causes an attack of the vertigo (figure 3).



figure 3

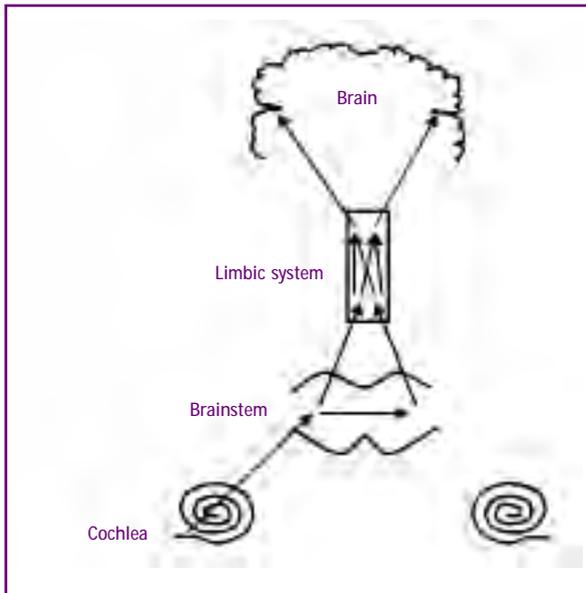


figure 4

In some people the ear can return back to normal and perhaps they will never have another attack, but others will develop Menière's disease as the excess endolymph fails to clear completely. In a Menière ear a delicate balance exists. The endolymph volume can be controlled by drainage radially to the blood vessels, but if an excess of endolymph does develop, the extra pressure (less than 1cm of water pressure) can initiate sudden longitudinal flow and cause an attack of vertigo. For example: the person has a Chinese banquet and the extra salt increases endolymph volume and when a critical level is reached. The critical level triggers the pressure receptors causing the mechanism for longitudinal flow to occur. The endolymph passes to the endolymphatic sac like sand passing through an hourglass, with an attack of vertigo lasting for one hour or even more. The person recovers for a while. Next the person has a visit from the daughter with an unsuitable boyfriend, and the stress causes endolymphatic secretion and yet another attack of Meniere's results. As the disease progresses the endolymphatic sac becomes damaged and the attacks of vertigo become less intense as the ear fails to clear more and more of the excess endolymph. Eventually, the endolymphatic sac is damaged and ceases to function and the attacks of vertigo end, but the hearing has become irreversibly damaged because of the endolymphatic hydrops. Rather like the eye being damaged by the persistent presence of glaucoma.

Medical treatment may be aimed at reducing the intensity of the vertigo attacks (Stemetil®), or at preventing the endolymphatic hydrops increasing. Simple measures like salt restriction and diuretics can reduce the likelihood that the endolymph volume will increase and trigger an attack of vertigo. Vasodilators, such as Serc®, may increase the capacity of the stria vascularis to increase radial flow. Cinnarizine (Stugeron®) acts both as a vestibular sedative as well as having some effect on the radial flow of endolymph.



Surgical treatment can be aimed at the endolymphatic sac. Removing part of the endolymphatic sac<sup>10</sup> or damaging it by a so called 'drainage operation' weakens the drainage function of the endolymphatic sac and reduces or stops the attacks of vertigo. Labyrinthectomy removes all the function of the ear, both the hearing and balance functions, and should only be undertaken as the last resort. Even removing all the balance function in the affected ear is a drastic measure as the development of Menière's disease in the remaining ear may result in loss of postural control. Removal of the balance function from the ear can be achieved by a vestibular nerve section or more simply by gentamycin infusions.

The latest idea is to reduce the endolymph using bursts of hyperpressure. In Scandinavia, a machine (Meniett) has been developed which is inserted into the affected ear for five minutes, three times each day when the ear is actively affected by Menière's disease. When the ear becomes inactive, the machine is used daily for five minutes. These pulses of hyperpressure increase radial flow removing excess endolymph without the need for medication.

The initial results are reputed to be excellent and an extensive clinical trial is under way in Europe. In Australia, the cost of the machine may be as much as \$5000 and, presently, neither Medicare nor private health funds will cover the cost.

### Tinnitus

Tinnitus is one of the symptoms of Menière's disease. It may also be the result of many other causes and tinnitus is one of the commonest afflictions in humans. Over 10% of the population, including myself, have some constant tinnitus. In fact the figure is even higher as most people can notice some tinnitus if they are placed in a totally quiet environment. Tinnitus may therefore be almost a normal phenomena -what is not normal is when perception increases so that it becomes upsetting and bothersome.

The mechanism for tinnitus is now becoming clearer due to work by Pavel Jastreboff and Jonathan Hazell. While I agree with most that they have written, I do differ, as I believe that in 90 percent of tinnitus sufferers, the initial problem began in the ear rather than the brain. The following account is therefore, my own view, which has been heavily influenced by Jastreboff and Hazell.

Sound received by the ear vibrates the eardrum and the vibrations are transmitted through the bones of the middle ear to the fluid of the inner ear. The inner ear is composed of the cochlea, which is involved in hearing, and the semicircular canals, which are involved with, balance (figure one). In the cochlea, the movement of the fluid leads to activation of the hair cells, which connect to the hearing nerve. Activation of the hair cells leads to electrical impulses travelling along the hearing nerve. The information from both ears is combined in the brainstem and passes on to the cortex of the brain where sound perception occurs. Then the electrical signals pass to different areas of the cortex of the brain where association or meaning is given to the sound and 'hearing occurs'. If a person has tinnitus, then an electrical signal is passing to the cortex of the brain despite the absence of sound in the environment. Tinnitus is generated inside the person's own body rather than by outside sound.

The source of tinnitus is most usually a defect within the ear; usually a clump of haircells which are damaged. This sound then passes up the brainstem to the auditory cortex where it is perceived. On its way up the brainstem, the sound passes through an amplifier called 'the limbic system'. The limbic system is rather like an amplifier on the television aerial, which increases the signal in poor reception areas.

In animals, the limbic system can have a significant role. For example, when a deer is endangered by a predator, the hearing is sharpened so that it can hear a twig snap and run from danger. In man, the limbic system is the mechanism that makes us startle to an unexpected sound. If we walk alone through a graveyard at night, our limbic system becomes very active and when a friend suddenly shouts 'boo', we jump four feet into the air. We also may change friends!



In tinnitus the limbic system can become very active increasing the perception of tinnitus, and sound, from the ear to bothersome levels and preventing adaptation from occurring. What is adaptation? It is when the brain gets 'used to' ceases to register a stimulus. Adaptation of a constant sound occurs when it ceases to be important. For example we can visit a friend who has a noisy fridge and be amazed that she does not seem to hear it at all. If people get used to noisy fridges why cannot others get used to their tinnitus? It is because the limbic system prevents adaptation occurring. The limbic system works when the person is threatened or frightened of the noise.

Many people when they first develop tinnitus fear the worst. They believe they have a brain tumour or will become deaf. The doctor's role is to discover the cause, which hopefully will be benign. For example, the commonest cause is noise exposure that has damaged a clump of haircells in the ear. It is very rare to find a brain tumour, and now an MRI examination can give 100% reassurance that no tumour is present.

Unfortunately, the cause of tinnitus is rarely treatable by surgery. A few may have otosclerosis and a stapedectomy operation will help. For most the hearing problem affects the hair cells and no surgery can be performed. Why not destroy the ear? Some severe tinnitus sufferers plead to have the hearing removed completely. The simple answer is that this does not work. It has been tried in the past very unsuccessfully. The problem is the limbic system. The limbic system is controlled by orderly sound passing through the mechanism. If no sounds pass through the mechanism, it develops its own output, which is perceived as tinnitus by the brain. Thus tinnitus has two components: one is generated in the ear itself and the other is generated in the limbic system. Both components need to be addressed to abolish tinnitus and if the ear is destroyed after a few days, the limbic system activity will recreate the tinnitus.

So what treatment is available? Firstly, do visit your ENT surgeon and discover the cause so that any fears can be allayed. In some cases, the cause will be excessive noise and apart from avoiding any future excessive noise no specific medical treatment is available. Research to find methods of restoring hair cells is progressing but not available yet. A few may have a cause of hearing loss which can be alleviated by surgery, or hearing loss which is aggravated by a blocked middle ear helped by realigning the jaw or treating a stuffy nose.

If the ENT surgeon has ruled out any serious cause and has tried medical treatment that is appropriate but without success, the next professional to visit is an audiologist skilled in treating tinnitus. The audiologist will measure the hearing loss and may suggest a hearing aid. The hearing aid will restore hearing to the damaged ear and send orderly signals to the brain suppressing any random activity generated by the limbic system. This will allow gradual adaptation of the tinnitus to occur. For very worried people, the audiologist or a clinical psychologist may help to speed adaptation by reassurance and explanation of the mechanisms involved. This treatment, now called 'tinnitus retraining therapy' can help over 90 per cent of severe tinnitus sufferers.

Finally, tinnitus sufferers do have to be careful of charlatans. Because no drug has been developed which instantaneously 'cures' tinnitus, it is easy for the sufferers to be attracted to people offering so-called 'cures'. Herbal remedies rarely do harm but surgical or invasive therapies can make matters worse. Please always check with your family doctor or ENT surgeon before undertaking any radical treatments. If these treatments fail, the tinnitus can get worse. The Australian Tinnitus Association has a lot of information on tinnitus, which serves as a useful guide to sufferers and to the professionals involved.

Libby Harricks was a wise woman. She accepted her hearing loss and used her experience to help thousands of other people. I hope that Meniere's sufferers and tinnitus sufferers will be able to follow her example and help others by increasing the knowledge of these two disorders. It has been my privilege to speak on this special occasion to honour the memory of such a great woman. Thank you for listening.

## 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", Turrumurra 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 program is supported by the Libby Harricks Memorial Fund of the Deafness Forum of Australia. Donations to this fund are tax deductible. Please see enclosed donation form for full details.

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

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