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From the President

It is with great sadness and shock that I write this President’s Message due to the tragic death of Andrew Wearne at the end of last month. We have lost Australia’s best – and probably one of the world’s top five – railway noise and vibration acoustic engineers. I attended his memorial service along with approximately 800 others and there was overwhelming praise and recognition for Andrew’s achievements from friends, colleagues and clients.

I was quite surprised how much this loss affected me personally, given that I only knew Andrew as a fellow professional and didn’t know him socially. Although we had worked together on a number of projects over the last 13 or so years and others in our office had been working more closely with him recently, his name was always mentioned with the utmost respect.

I believe the real reason behind my feelings was the fact that Andrew was always so polite, easy to deal with and a true professional that you subconsciously knew what he would have been like as a husband, father, team mate and friend, hence you actually felt that you knew him well. I know that many other acousticians were similarly shocked and it was wonderful to see so many of his peers attend the memorial service which was a celebration of his life and achievements.

It is tragic circumstances like this that make you pause for a moment and take stock of your life. Do I enjoy my job? Do I have the right work / life balance? Am I contributing to society making the world a better place? The memorial service certainly indicated the great contribution Andrew has made through his skills as an engineer and also his dedication to friends in his local Berowra community. I trust that we can all reflect on the way we behave both professionally and socially to achieve the right outcomes. Hopefully this involves compromise and resolution rather than argument and conflict.

In our Articles of Association, the Acoustical Society is termed a “learned” society. Today we are a little less “learned”. Andrew will be truly missed. The family has requested any donations be sent to African Enterprise www.africanenterprise.com.au

Looking to the future, it would appear from recent email correspondence that the first joint Australian and New Zealand Conference this November in Christchurch is shaping up to be one of the best ever in terms of likely attendance and number of papers. It really is the only opportunity each year to get together with your peers from around the region to share ideas and knowledge.

On a more local stage, the need to have regular get-togethers in your own State at technical meetings is also important so I would encourage you all to support these events by both offering to present and supporting your peers.

Neil Gross

From the Guest Editor

Since the last issue of Acoustics Australia directed at basic mechanisms in 1993, biological research has drawn over a billion of dollars of funding and is poised to make several huge advances. Hearing science has increasingly embraced other disciplines, such as genetics and molecular biology, which at first glance might seem to have little connection with acoustics. Accordingly, the Australian authors were challenged to condense into just a few pages those developments which may soon have application to acousticians and clinicians, i.e. the articles had to relate to sound parameters, signal processing, engineering concepts and auditory psychophysics.

Accordingly the articles are presented in a “top-down” order, from the psychophysics of sound localisation using Head Related Transfer Functions (Carlile). Next, Irvine and colleagues review the concept of plasticity of ‘wiring’ of the neural connections in the auditory cortex and how that has applicability to the fitting of cochlear implants. Further down the brainstorm, Mulders reviews the extensive neural organisation with particular emphasis on the signals which descend to the cochlea to either control the mechanical processing of sound via the outer hair cells, or the excitability of primary neurons carrying the frequency analysis upward.

The next two articles are about the genetics of hearing loss (Dahl and colleagues) and the source of biological energy which drives cochlear processes (Pickles). These authors ask why deafness occurs; and what syndromes relate to malformations of molecular structures which allow ionic currents to flow. The charged ions must be pumped using energy provided by mitochondria. These two papers also contain the basis of mechanisms for characterising individual susceptibility to hearing loss. Basic hearing science now regards the effects of noise trauma as just another of the toxic influences which cause hair cells to die. Hair cell death occurs by two separate processes and, whereas we might once have had a simple mental connection between temporary threshold shift as an indicator for permanent loss, we now have a new branch of science.

Traditionally, reviews of cochlear mechanics discuss how the frequency analysis in terms of tuning curves for each frequency and the important nonlinear processes which occur due to outer hair cell activity. The sixth article (Sen) models two-tone interactions at the mechanical level and explains the upward spread of masking which started out being the basis of mpeg compression technology, but has deep associations with distortion product emissions, combination tones, and critical bands. Increasingly, analysis of cochlear mechanics suggests that the outer hair cells providing the frequency analysis are also involved with regulation. The last article (LePage) reviews direct mechanical evidence for an internal automatic volume control system which optimises cochlear performance during trauma and aging of the ear. The new insights into structure and function lead to some possible explanations of the relationship between sound level and duration – Dixon Ward’s equal energy relation, and suggest new approaches to hearing loss prevention based on actual mechanisms.

This special issue thus has particular significance because it draws attention to the exciting cross-fertilisation now taking place between the worlds of psychoacoustics and physiological acoustics. The next decade may well reveal precise explanations of well-known psychophysical phenomena and issues of individual susceptibility.

Eric LePage, guest editor
LISTENING TO THE WORLD AROUND US

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Our perception of auditory space depends on the integration of a number of acoustic cues to the locations of sound sources. The binaural cues to location arise as a consequence of the two ears being separated by an acoustically dense head which results in differences in the time of arrival and level of the sound at each ear. The outer ears also filter the sound in a directionally dependent manner providing the spectral cues to a sound’s location. Real world listening involves separating out multiple concurrent sound sources and differences in their spatial locations provide a means by which auditory spatial attention can be focused on one sound of interest and other masking sounds are ignored. Recent work has demonstrated that spatial release from masking is more effective when the target and maskers are speech sounds and that this involves both bottom up perceptual processes and top-down cognitive processes. This work indicates that preservation of the spatial cues is essential for the effective use of hearing aids implicating both binaural and in-the-ear fitting strategies.

PLASTICITY IN THE ADULT CENTRAL AUDITORY SYSTEM

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The central auditory system retains into adulthood a remarkable capacity for plastic changes in the response characteristics of single neurons and the functional organization of groups of neurons. The most dramatic examples of this plasticity are provided by changes in frequency selectivity and organization as a consequence of altered experience. These forms of plasticity are likely to contribute to the improvements exhibited by cochlear implant users in the post-implantation period.

EFFERENT CONTROL OF HEARING

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The auditory system comprises both ascending (afferent) and descending (efferent) pathways. The efferent pathways, which originate in a variety of higher brain centres, are capable of altering the activity in the afferent pathways. By modulating cochlear neural output and central auditory neural circuits, these efferent pathways could play an important role in key auditory processing such as optimising the detection of acoustic signals of interest in the presence of competing background noises. The present paper focuses on the final limb of the efferent pathways, the olivocochlear system, which projects directly to the cochlea. It will describe its proposed role in normal hearing and show how dysfunction of this efferent system could contribute to generation of tinnitus and to deterioration in the detection and processing of signals such as speech, especially in non-optimum listening environments.

GENETIC ASPECTS OF HEARING LOSS

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Genes mediate the workings of cells, organs and organisms. Since normal hearing is dependent on highly specialised structures and cellular functions it is not surprising that many genes—as well as environmental factors—affect this complex process. A number of genes have been identified to date that have added to our knowledge of the molecular aspects of hearing. Mutations, or changes, in these genes cause deafness or hearing impairment demonstrating that these genes are essential for normal hearing function. Despite the advances we have made in the discovery of “deafness” genes, little is known about the genes that determine susceptibility to noise-induced deafness, ototoxic hearing loss or early onset presbyacusis. Increasing our knowledge of the genetic aspects of hearing loss will lead to improved genetic counselling and will help the development of novel cell-based, gene or drug therapies.

MITOCHONDRIA, CELL DEATH, AND DEAFNESS: WILL IT BE POSSIBLE TO PREVENT PRESBYACUSIS?

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Mitochondria are energy-producing structures within cells, using oxidation to produce the energy-rich compound ATP (adenosine triphosphate) which drives the cell’s energy-consuming reactions. Mitochondria are also triggers of programmed cell death, called apoptosis. These two important aspects of cell function are linked: when energy production by the mitochondria fails, a set of biochemical reactions is initiated which lead to destruction of the cell. Some cells types are particularly vulnerable, including certain cells of the inner ear (e.g. outer hair cells and cells of the stria vasularis), leading to sensorineural deafness. It is argued here that this response may be an evolutionary maladaptation, that cell death may be sometimes be triggered unnecessarily, and therefore that some forms of sensorineural hearing loss such as that arising in old age might be preventable.

FUNCTIONALITY OF COCHLEAR MICROMECHANICS -- AS ELUCIDATED BY UPWARD SPREAD OF MASKING AND TWO TONE SUPPRESSION

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The effect of increasing the level of a low frequency masker on higher frequency probes can be observed in three paradigms: psychophysically (termed Upward Spread of Masking or USM), mechanically on the basilar membrane (Basilar Membrane Two Tone Suppression or BM-2TS) as well as neuro-physiologically at the auditory neurons (Neural Two Tone Suppression or Neural-2TS). This paper reviews various experimental USM, Neural-2TS and BM-2TS data with the aim of shedding light into the underlying physiological mechanisms in the cochlea.

A REVIEW OF MECHANICAL EVIDENCE FOR A SERVO-LOOP IN THE MAMMALIAN COCHLEA

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The outer hair cells in the cochlea are recognised as the active mechanical elements in the normal operation of the cochlear amplifier. Yet the functions of their two motor mechanisms are still not clear. Increasingly, the outer hair cells are also being implicated the control elements in homeostasis – normal regulation of cochlear activity by the descending neural pathway. This review targets articles with mechanical data and suggests new clues as to structure and function in terms of a mechanical-feedback loop for dc-stabilisation. The literature relevant to such an idea is reviewed and directly leads to clues underlying the notion of a time-intensity trade-off for noise exposure, the cause of Ménière’s disease and the upward spread of masking.