OFFICIAL PUBLICATION OF THE CANADIAN ACADEMY OF AUDIOLOGY
PUBLICATION OFFICIELLE DE L'ACADÉMIE CANADIENNE D'AUDIOLOGIE

Canadian Hearing Report

Revue canadienne d'audition

Vol. 8 No. 1
2013

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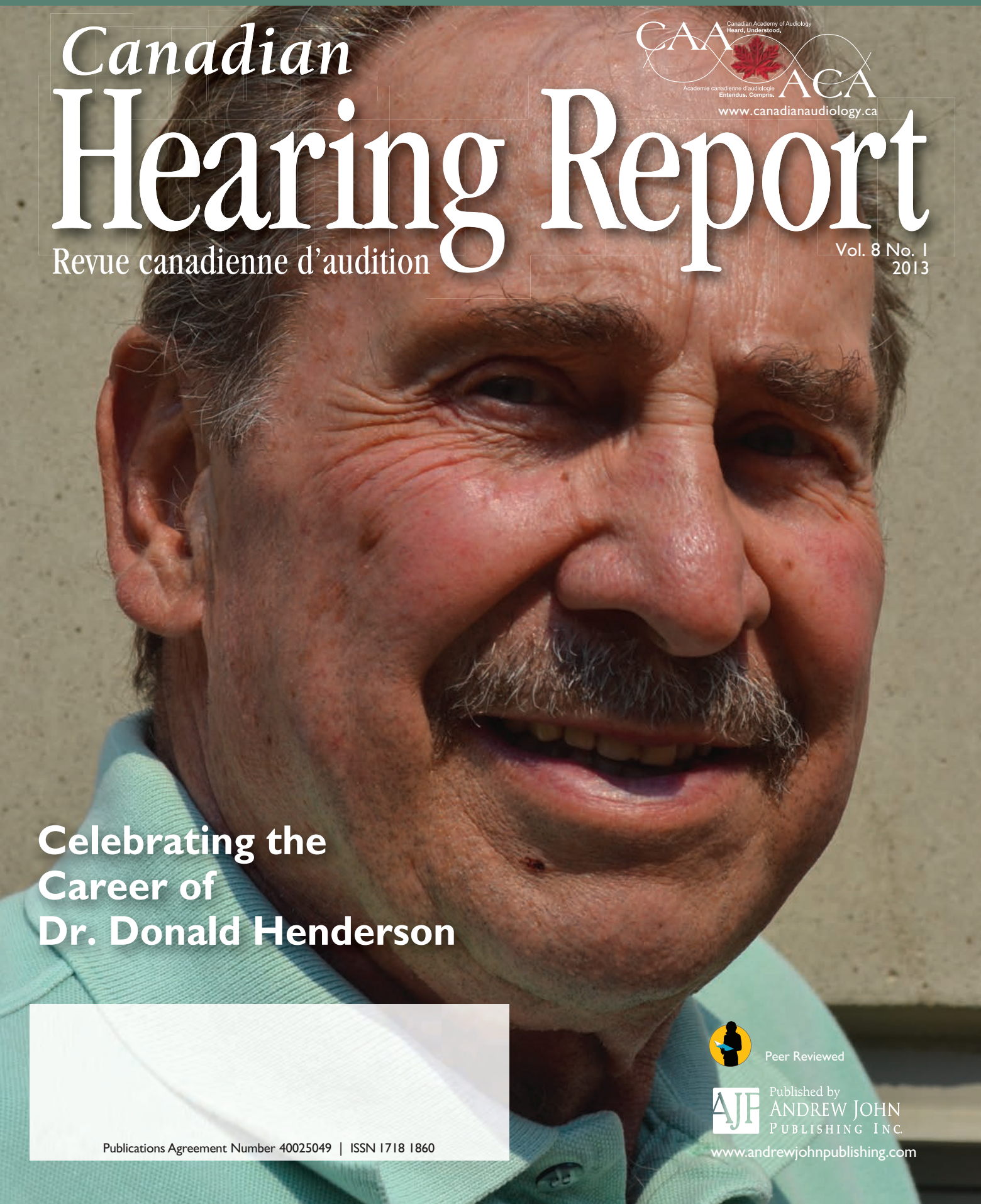

Celebrating the Career of Dr. Donald Henderson

Publications Agreement Number 40025049 | ISSN 1718 1860

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By the time you open this issue of the *Canadian Hearing Report*, Christmas will be long passed and the snow will still probably be on the ground. And, unfortunately, we will all be carrying about 5 kg of unwanted bulge. So don't forget to get out to exercise, or at least shovel the snow. It's not only good for our waistline but also for our hearing. Increased pulmonary blood flow and an improved cardiac system has been related to improved hearing as we age. Presbycusis was once considered to be inevitable but research (Alessio and Hutchinson, *Canadian Hearing Report* 2010;5(6):20–29) demonstrate that 80-year-old couch potatoes have statistically poorer hearing than 80 year olds who are physically fit. Like many areas of audiology, there is a significant degree of variability in this area of study, and variability is one of several themes that run through this issue.

Spotlight on Science is a regular column which is shared by Dr. Sheila Moody at Western University (previously called the University of Western Ontario), and Lendra Friesen and Samidha Joglekar at the Sunnybrook Health Sciences Centre – each sharing alternating duties. This will be Lendra's and Samidha's last column (but Lendra will be back guest editing an issue of the *Canadian Hearing Report* later this year), and we wish both Lendra and Samidha all the best. To try to fill their shoes, Dr. Steve Aiken of Dalhousie University will be sharing writing responsibilities with Sheila Moody. Steve is currently the president of the Canadian Academy of Audiology and has the rare combination of working in a clinical environment, in industry with a hearing aid manufacturer, and in an academic setting so I suspect that his Spotlight on Science column will be broadly based.

This issue's Spotlight on Science column touches on the often annoying feature of the variability of a neural response for those implanted with cochlear implants. Why does one person do well and another person may have a more limited response? Neural variability and some of its indicators are covered in this column. Variability touches on all aspects of audiology and in some areas only about half of the clinical or evoked response variability can be explained.

And speaking of variability in human populations, the topic that always comes to mind is the study of noise and how it affects our auditory system. When one thinks of the research on noise, you can't help but come across the name of Dr. Don Henderson.

Actually, Don Henderson will be honoured at this year's American Academy of Audiology (2013) in Anaheim, California, for a lifetime of work in research in audiology… Well not to brag, but the State University of New York at Buffalo and the *Canadian Hearing Report*, have both beat them to the punch.

On Aril 20, 2012 the Department of Communicative Disorders and Science and the Center for Hearing and Deafness hosted a one-day seminar in honour of Don Henderson's life's work. A little known fact is that Don was born is Hamilton, Ontario, and even less known is that he played one year for the British Columbia Lions in the Canadian Football League. The greater known facts include reams of publications and book chapters in virtually all aspects of our field, concentrating on the effects of noise on our auditory system. In this issue of the *Canadian Hearing Report* we have amassed an impressive list of articles by Don's colleagues and students who attended this one day conference last April.

I won't say anything more about Don at this point because we already have a wonderful introduction to Don and his life's work co-written by Dr. Dick Salvi (recently promoted to distinguished professor status at SUNY at Buffalo) and by Carol Altman (who actually is one of the driving forces behind the department). There are seven articles in total, some of which have been written by people who have presented at the Canadian Academy of Audiology – remember Dr. Eric Bielefeld at our most recent meeting in Ottawa?

I hope you all are having a pleasant winter season and haven't put on too many of those annoying, hearing-loss affecting, kilograms.

Marshall Chasin, AuD, M.Sc., Aud(C), Reg. CASLPO, Editor-in-Chief
marshall.chasin@rogers.com

Canadian Hearing Report 2013;8(1):3.

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Canadian
Hearing Report
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Vol. 8 No 1 • 2013

Official publication of the
Canadian Academy of Audiology

Publication officielle de l'académie
canadienne d'audiologie
www.canadianaudiology.ca

Canadian Hearing Report is published six times annually by Andrew John Publishing Inc. with offices at 115 King Street West, Dundas, On, Canada L9H 1V1.

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Publications Agreement Number 40025049 • ISSN 1718 1860

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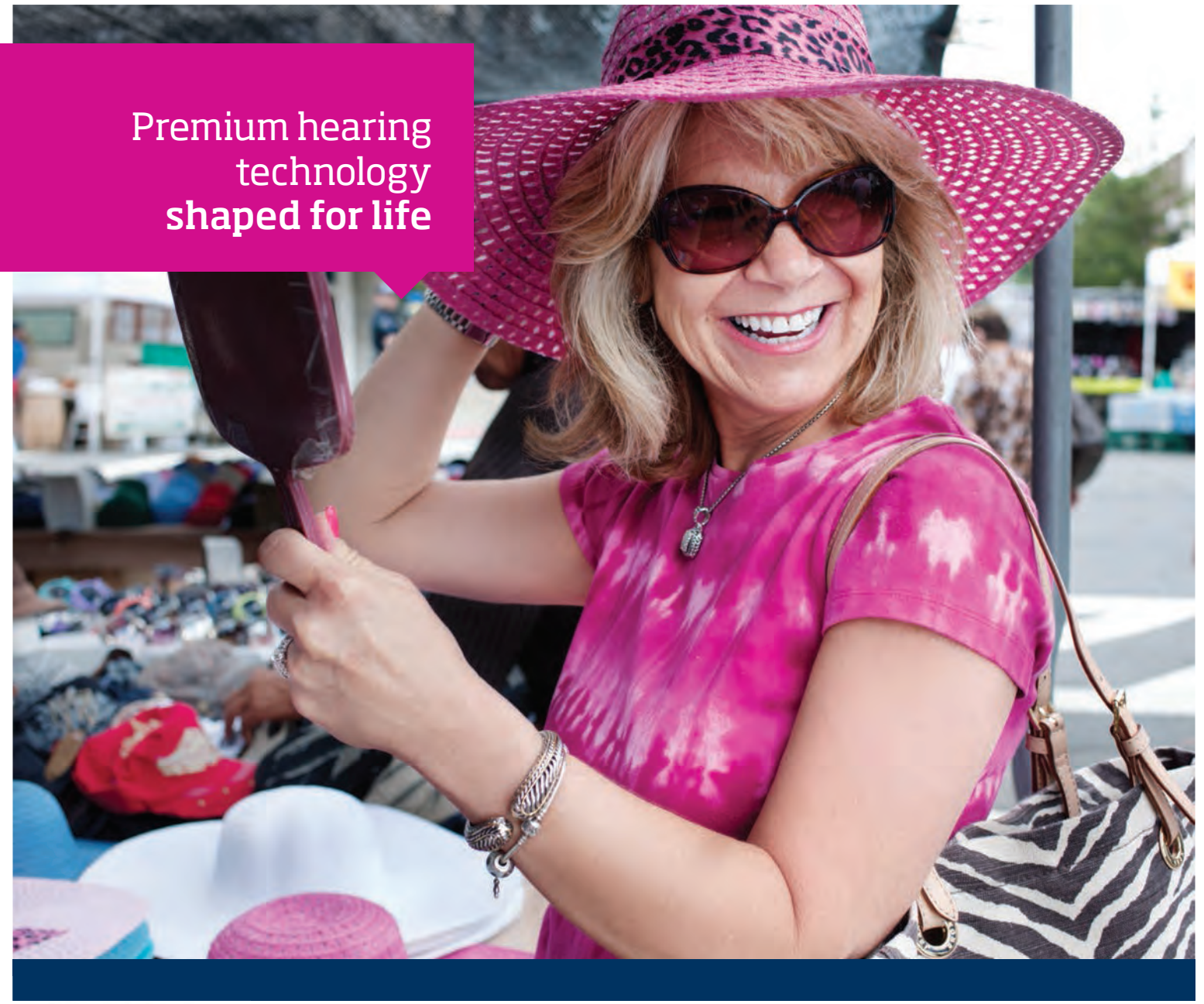

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PEOPLE FIRST

Au moment où vous allez ouvrir ce numéro de *la Revue Canadienne d'audition*, noël serait loin derrière nous et la neige probablement toujours au sol. Malheureusement, nous porterons tous près de 5 Kg de surpoids non voulu. Alors n'oubliez pas de sortir pour faire de l'exercice, ou au moins pelleter la neige. Ce n'est pas seulement bon pour notre tour de taille mais aussi pour notre ouïe. Une augmentation du débit sanguin pulmonaire et un système cardiaque amélioré ont été liés à une ouïe améliorée pendant le processus de vieillissement. La presbyacousie a été une fois considérée inévitable mais la recherche (Alessio and Hutchinson, Revue canadienne d'audition 2010;5 (6):20–29) démontre qu'une personne sédentaire de 80 ans a statistiquement une ouïe de qualité inférieure à celle d'une personne de 80 ans qui fait de l'exercice. Similaire à beaucoup de domaine de l'audiologie, on trouve un certain degré de variation dans ce domaine d'étude, et la variation est un des plusieurs thèmes de ce numéro.

Spotlight on Sciene est une chronique régulière qui est partagée par Dr. Sheila Moody de l'université Western (de son nom précédent the University of Western Ontario), et Lendra Friesen et Samidha Joglekar du Sunnybrook Health Sciences Centre – chacune partageant des taches alternantes. C'est la dernière chronique de Lendra et Samidha (mais Lendra va être de retour en tant que rédactrice invitée de *La Revue Canadienne d'audition* plus tard cette année), et nous souhaitons le meilleur à Lendra et Samidha. Pour essayer d'accéder à leur niveau, Dr. Steve Aiken de Dalhousie University va partager les responsabilités de rédaction avec Sheila Moody. Steve est actuellement le président de l'académie canadienne d'audiologie et a la combinaison rare de travailler dans un environnement clinique, dans l'industrie avec un fabricant d'appareils auditifs, et dans un environnement académique, alors je me doute bien que sa chronique spotlight on sciences aura une base assez large.

Ce numéro de Spotlight on sciences touche la caractéristique souvent contrariante de la variabilité de la réponse neurale pour ceux et celles qui ont un implant cochléaire. Pourquoi certains affichent de bons résultats et d'autres ont une réponse plus limitée ? La variation neurale et certains de ses indicateurs sont traités dans cette chronique. La variation touche tous les aspects de l'audiologie et dans certains domaines, à peu près seulement la moitié de la variation de la réponse clinique ou évoquée peut être expliquée.

En parlant de variation chez les êtres humains, le sujet qui surgit toujours est l'étude du bruit et son effet sur le système auditif. Quand on pense à la recherche sur le bruit, on ne peut ne pas croiser le nom du Dr. Don Henderson.

… En fait, un hommage sera rendu à Don Henderson à la American Academy of Audiology de cette année (2013) à Anaheim, en Californie, pour le travail de toute une vie en recherche en audiologie … Bon, sans être prétentieux, mais the State University of New York à Buffalo et *la Revue Canadienne d'audition*, les a coiffé au Poteau.

Le 20 avril 2012, le département des sciences des troubles de communication et le centre pour l'ouïe et la surdité a organisé un séminaire d'une journée pour honorer le travail de toute une vie de Don Henderson. On ne le sait pas assez mais Don est né à Hamilton, en Ontario, et ce qu'on sait moins encore est qu'il a joué pour une année pour les Lions de la Colombie britannique dans la ligue canadienne de Football. On est plus au courant de la multitude de ses publications et chapitres de livres pratiquement au sujet de tous les aspects de notre domaine, avec une concentration sur les effets du bruit sur notre système auditif. Dans ce numéro de *la Revue Canadienne d'audition*, nous avons amassé une liste impressionnante d'articles de collègues et étudiants de Don qui ont participé à cette journée unique de conférence au mois d'avril dernier.

Je n'en dirai pas plus sur Don parce que cet éditorial sera suivi par une superbe introduction à Don et son travail de toute une vie co-écrit par Dr. Dick Salvi (récemment promu au statut de professeur distingué au SUNY à Buffalo) et par Carol Altman (qui est une des forces dirigeant le département). En somme, sept articles, dont certains ont été rédigés par des personnes qui ont présenté à l'académie canadienne d'audiologie– vous rappelez vous du Dr.Eric Bielefeld à notre dernière réunion à Ottawa?

J'espère que vous passez tous et toutes une bonne saison d'hiver et que vous n'avez pas pris beaucoup de ces kilogrammes tellement irritants affectant la perte auditive.

Marshall Chasin, AuD, M.Sc., Aud(C), Reg. CASLPO
Éditeur en chef
marshall.chasin@rogers.com

Canadian Hearing Report 2013;8(1):7.

What's the Matter with Canada?

By David H. Kirkwood

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People who know me probably wonder why I would ask such a tendentious question. After all, my father, who was born and raised in Ontario, remained a Canadian for most of my childhood years. And, except for my mother and brother, all my Kirkwood relatives still live on the northern side of the border. I also have fond memories of family visits to Toronto, Montreal, and the Gaspé Peninsula.

Professionally, I've also had great experiences with Canada and its people. As an editor, I have observed consistently that manuscripts submitted by Canadian audiologists and hearing instrument specialists are especially well written. I don't know if that's true of Canadians across the board or just among those who go into the hearing care field. In any case, I appreciate it. I should also note that two of our blog's outstanding editors are from Ontario – Marshall Chasin and Gael Hannan.

A HEALTHCARE ISSUE

So, what's my beef with the world's second largest nation? Well, it has to do with healthcare. No, I'm not talking about its vaunted publicly funded health care system, which, despite its detractors in the U.S., even conservative Canadians have grown to cherish during the nearly 30 years it has been in effect.

Canada also has a pretty good record of taking care of citizens with hearing loss. In several provinces, the government covers at least part of the cost of hearing aids for adults as well as children.

The country also does an excellent job of educating hearing instrument specialists. It has several two-year program for that purpose, putting it well ahead of the U.S. in that respect.

That's why it is so shocking that of the ten Canadian provinces, only Ontario and British Columbia require universal newborn hearing screening (UNHS). Quebec is on its way. It passed a law mandating UNHS, but it is not scheduled to be fully implemented until the end of 2013.

Other provinces tend to test only babies at high risk of hearing loss, such as preemies and those suffering serious infections like meningitis. Contrast that within the U.S., where the UNHS movement took off in the 1990s. Now, 95% of babies born here are screened.

THE CASE IS CLEAR

One reason that universal newborn screening is such a no-brainer is its cost effectiveness. True, when you add up the hundreds of screenings, at about $35 a pop, that it takes to identify a single newborn with hearing loss, the price may seem high. But then consider the value of early identification and intervention. It is well established that the earlier a child's disability is addressed, the more likely that child is to learn as well as his or her normal-hearing classmates and to become a successful, productive adult.

Children whose hearing loss is discovered and treated at age 1 or 2 or older are likely to need special education that will cost society far more than the cost of detecting and addressing it early. What's worse is that these children will be far less likely to achieve their full potential. Their lost opportunities are a tragedy both for the children and their families and for society as a whole.

A lot of Canadians are working hard to introduce UNHS in every province and territory of the country. The Canadian Association of Speech-Language Pathologists and Audiologists has adopted position papers to that effect, most recently in 2010. And last January, CASLPA recommended that the national budget provide for universal access to newborn hearing screening throughout Canada.

In 2011, the Canadian Paediatric Society issued a statement calling on all provinces to implement universal newborn hearing screening. One of the leading champions for UNHS in Canada is Hema Patel, MD. A staff pediatrician at Montreal Children's Hospital, she is lead author of the Paediatric Society's position statement. In an Interview with the *Globe & Mail,*

Patel said, "Virtually every developed country has a screening program. It's shameful that Canada doesn't."

She also pointed out to a Montreal CTV affiliate that each day a child unnecessarily lives in silence can result in permanent and possibly irreparable loss of development. She explained, "Hearing is actually not about the ears. Hearing is about the brain and the longer that the child is deprived of that auditory input and the sound that is all around us, the more that it shuts down that development."

The Canadian Hearing Society, which provides services to people with hearing loss, also supports universal newborn hearing screening.

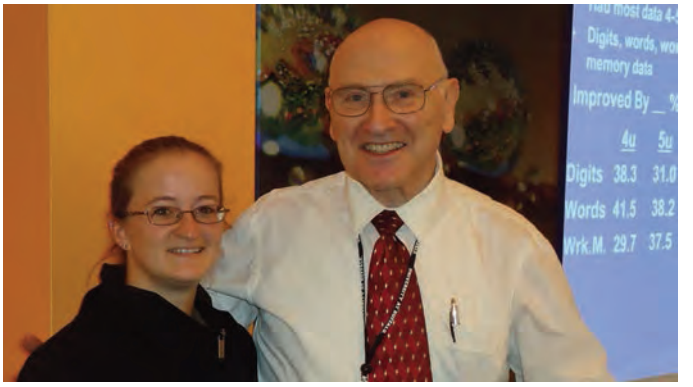
According to the Calgary Herald, advocates for UNHS in Alberta are petitioning their province to get with the program. Twice before, the government in Edmonton has considered and rejected the idea. But this time, there seems to be growing support for this eminently sensible health policy.

It is shocking that in a country that is generally so progressive universal newborn hearing screening is more the exception than the rule. It is high time that Canadians who share our blog's credo that Hearing Health Matters take steps to remedy one of the few things about Canada that need fixing.

Canadian Hearing Report 2013;8(1):8-9.

CAPD Seminar in Buffalo a Big Success!

Organizers of the recent CAPD Seminar in Buffalo Dr. Kim Tillery and Dr. Nancy Stecker flank the legendary Dr. Jack Katz.



Dr. Jack Katz and Taralyn Keizer of Keizer's Hearing Clinic in Sudbury Ontario.

Elliott Berger to Receive National Hearing Conservation Association's Lifetime Achievement Award

Elliott H. Berger, MS, division scientist for 3M's Occupational Health & Environmental Safety Division, will be presented with the National Hearing Conservation Association Lifetime Achievement Award in St. Petersburg, FL, in February, 2013.

This prestigious award represents the highest honour bestowed by the NHCA and is intended to recognize a lifetime of extraordinary accomplishment in the field of hearing loss prevention and in service to NHCA. The NHCA honours Elliott Berger for his significant and lasting contributions to hearing conservation and to NHCA as evidenced by his extensive body of quality work.

For over 35 years, Berger has been a driving force in hearing loss prevention research and training, personal hearing protection product development, and the establishment of national and international standards and regulations. Through his role at 3M (previously E-A-R and Aearo Technologies), Berger has helped direct the industry's approach to the prevention of hearing loss due to occupational and environmental noise. Berger is perhaps most widely known as a respected author and editor of hearing-conservation-related books, book chapters, peer-reviewed papers and articles, and through hundreds of national and international presentations. As chair of the ANSI Working Group on hearing protectors, Berger has laboured tirelessly since 1985 to establish relevant standards that support hearing conservation efforts. He is a past president of NHCA and currently serves as NHCA's historian.

Berger's acceptance of the Lifetime Achievement Award will take place at an awards luncheon during the NHCA's 38th Annual Conference to be held February 21-23, 2013 in St. Petersburg, Florida. Many of Berger's colleagues and associates will be on hand to honour and recognize this prestigious achievement. For more information about the NHCA Conference, The Art of Hearing Conservation, go to http://www.hearingconservation.org.

The Cégep of La Pocatière Hires A Specialist for Its New Audioprosthesis Program

The Cégep of La Pocatière has just proceeded with the hiring of Mr. Daniel Bois, audiologist. With a master's degree in audiology, Mr. Bois has nearly 20 years' experience in private practice and in hospitals. He was a lecturer at the University of Montreal, has published articles, and has lectured extensively on hearing problems and hearing aid technology in North America. He was also recently seen on the T.V. shows *Une pillule, une petite granule* (Télé-Québec) and *Famille 2.0* (Canal V).

Mr. Bois will teach the new Audioprosthesis Program which will receive its first intake of students in the fall semester of 2013. He officially took office on December 3, 2012 and will be primarily responsible for the coordination and the implementation of the new curriculum, devising the course layout and setting up the required installations including the premises and equipment required. The Cégep of La Pocatière will invest almost $700,000 acquiring the proper equipment for this new program.

The Cégep of La Pocatière thus becomes only the second institution in Quebec, and the fifth in Canada, to offer the Diploma of Collegial Studies (DEC) in Audioprosthesis. Managing director of the Cégep, Mr. Claude Harvey said "The announcement in June by Minister of Education, Recreation and Sports (MELS) Michèle Courchesne, was the crowning moment of two years hard work and at the same time proved that our college was able to offer this training in optimal conditions. This news was greeted with much enthusiasm from our college community since it will allow a regional college to train young people in a profession that will hopefully incite them to settle into our region later. There will be an increase in demand for this technology given the ageing of the population."

Some study programs already offered at the Cégep of La Pocatière such as Physical Technology, Nursing and Special Care Education have affinities with the technological, medical and patient-centered approaches to audio-prosthesis.

It is to be noted that due to the shortage of labour in this sector, the job market is very favourable and is in constant progression and that working conditions are also very appealing.

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The CAA has partnered with LMS PROLINK to ensure that members have access to the most comprehensive and cost effective Professional Liability policy in Canada. Plaintiff lawyers are getting more aggressive and are more frequently taking a *"shot-gun"* approach to their lawsuits. There may be circumstances in which your employer's insurance company may *deny* coverage to an employed Audiologist.

The employer may argue that their Audiologist was not acting in the normal scope of practice when the alleged malpractice occurred and therefore is not covered. Under this circumstance, the Audiologist needs to have their own coverage available to protect their personal interests and assets.

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- Reimbursement for legal costs incurred to defend work related criminal charges.
- Sexual abuse therapy counseling fund for the injured patient.

These are some (but not all) of the primary reasons why it is important to protect yourself! The only way to ensure complete malpractice protection is to secure an independent policy offered through the CAA member program.

PROTECTION FOR MEMBERS AND THEIR PATIENTS

Ensuring adequate protection of the public is without question a fundamental part of any health professional's delivery of care. Audiologists provide services to hundreds of thousands of Canadians each year. It is imperative that financial resources, liability protection, and legal representation are accessible in the event a patient is injured due to the negligence of an Audiologist.

Each year a variety of allegations surrounding patient injuries are brought forth that need to be defended. In such instances, not only does the professional need to be provided with legal protection, but in addition, an adequate financial fund *(the limit of insurance)* needs to be available for potential damage rewards to ensure the protection of the public.

For members of the CAA participating within the insurance program, the Insurer agrees to defend lawsuits where the patient alleges they were injured as a result of the actions of an Audiologist.

In the event of a professional liability claim, the insurer has the right to settle or defend a claim in court on behalf of an insured member. In return, members are relieved of all responsibility for securing their own legal counsel and defence. Under all Professional Liability policies it is imperative that members report all claims immediately.

RISING LEGAL COSTS

The average hourly rate charged by a lawyer in Canada is **$362/hour**. The average hourly rate for lawyers specializing in Professional negligence lawsuits is **$400 to $450/hour**. A major benefit of the Professional Liability policy is that it will pay legal expenses, investigation and defence costs, as well as judgments awarded to the injured party.

For More Information Please Contact:

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Does My Baby Have My Hearing Loss?

By Gael Hannan
gdhannan@rogers.com

If you are a parent, what did you worry about when expecting your child, especially the first child? Probably the standard issues: will all the regular bits be in good working order, will it be healthy, and please let it look like my side of the family?

One thing *most* parents don't think about is that their baby might be born deaf. And why would they? Chances are, no one told them to add hearing loss to their worry-list. And, as approximately only three in 1000 babies are born with some degree of hearing loss, the odds are favourable that the child would come into this world with typical hearing.

I didn't worry about it either. I swear to heaven, even with my own hearing loss that wasn't diagnosed until age two and a half, I don't recall thinking about it during pregnancy. Perhaps it was because hearing loss didn't "run in the family," and at the time I didn't know that congenital hearing loss has many causes. A more likely reason was that I was *finally* having a wonderful, viable pregnancy, and in the greater scheme of things, hearing loss just didn't seem to matter.

What I did worry about, however, was how I was going to hear the baby? I was concerned that my hearing might put my child in danger. What if I didn't hear him cry, or burp, or call for me? What if he became lost and I couldn't find him? All those things actually did occur at some point and to varying minor degrees, in Joel's childhood. But one day, as I blubbered about my some hearing-related baby mishap, my husband gave me a most wonderful gift – the reassurance that parenting is 50% luck, regardless of hearing ability.

Recently, my now 17-year-old son roared into the room (teenage boys are incapable of simply *entering*) with a bunch of his baby pictures, saying: "Let's look at them together, Mum." In the pile was one I'd almost forgotten – a sleeping four month-old having an auditory brainstem response (ABR) hearing test.

Because of my severe congenital loss, Joel's pediatrician considered him high risk and he had three hearing tests in his first year of life, which was 1995, a few years before Universal Newborn Hearing Screening was introduced in Ontario.

For the ABR test that measures the brainstem's response to sound, the baby must be asleep so he had to arrive at the hospital. (The fact that I, the mom, was already brutally sleep-deprived was not considered good enough.) This meant keeping the baby up late, getting him up early and not feeding him his breakfast. Up to that point, it was stressful but we got through it.

Then came the drive to SickKids hospital. Have you ever tried to keep a drowsy baby awake? As my husband drove the car as fast as legally possible, I jiggled and wiggled Joel in an attempt to *irritate* him into wakefulness. When he started to slip away into sleepy-land, I started singing, loudly and badly, but his eyes rolled up in his head and he was gone. He perked up at the hospital, but when little electrodes were placed on his head, he conked out again. And *that's* when it hit me.

What if my baby had my hearing loss? There had been no signs in his early months, but seeing him asleep with thingies stuck to his head, the possibility that he might have similar life-long hearing challenges shocked me into tears. But this screening, and subsequent tests confirmed he did not have hearing loss, and we were grateful.

If Joel *had* been born with hearing loss, I know that like other parents who receive the diagnosis, we would have been upset. But the good news is that interventions would have given Joel the best shot at optimal communication, for effective language. If the loss had been severe or profound – and if I knew then what I know and believe now – we would have raised him to communicate in both spoken and signed languages. Through the years, I've watched closely

for signs of hearing loss and had him tested again at age 10. So far, his hearing is "fine" although I have a new worry – noise damage.

Raised by a hard of hearing advocate, my son should, you might think, understand the consequences of unsafe listening practices. When he was younger, he could make the angels (and his parents) weep with the beauty of his classical guitar playing, but now he's happier screeching out chords on an electric guitar and a wicked amplifier. He recently came home from a club party with ringing ears that lasted for two days. It scared the heck out of him – which I was glad to see, hoping it would spur him to wear earplugs in the future.

But for now, I'm grateful for the newborn hearing screening available for today's babies – that is, the ones born in Ontario, BC, and some Atlantic provinces. But what about all the other Canadian babies who don't benefit from this one simple test, unless they are considered high risk?

As hearing health professionals and consumer advocates, we need to ramp up our advocacy to federal and provincial bodies for a national UNHS strategy that is implemented in every province and territory. When an infant's hearing is screened, he or she has just taken an important first step to a good life of language and communication. Don't all our babies deserve that?

Canadian Hearing Report 2013;8(1):15-16.

Federal Health Partners October 2012 Meeting

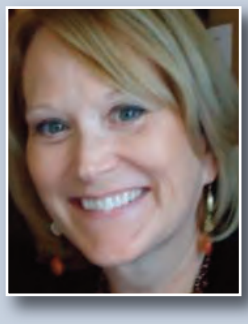
The Federal Healthcare Partnership (FHP) consists of Veterans' Affairs Canada, the Department of National Defense (DND), the Non-insured Health Benefits Branch of Health Canada (NIHB), and the Royal Canadian Mounted Police (RCMP).

Representatives from the CAA meet, together with other audiology professional organizations, with the FHP twice annually 'to maintain open channels of communication with our Third Party Payers to ensure effective, efficient and beneficial hearing healthcare services are being provided to our mutual clients. Minutes of the meeting held in October 2012 in Ottawa are available at http://www.canadianaudiology.ca/assets/docs/CAA_October_2012_FHP_Meeting_Notes.pdf

CAA members are asked to express their issues or concerns so that CAA may continue the dialogue with its partners in hearing healthcare services. Contact us at caa@canadianaudiology.ca.

Screening Measures for Auditory Processing Disorders

By Kim L. Tillery, PhD, CCC-A
kltillery@gmail.com



About the Author
Dr. Kim L. Tillery, professor and chairperson of the Department of Communication Disorders and Sciences at the State University of New York at Fredonia also has a private practice in diagnosing and treating individuals with (C)APD. She has been honoured to present 90 workshops or presentations at national, international, and regional conferences, and authored and co-authored several chapters and journal articles on (C)APD.

While there are questionnaires and screening tests that are used routinely in clinics and schools there are some points to keep in mind. One point is that a questionnaire is only as good as who is answering the questions. A story comes to mind – of a psychologist who administers a well-recognized questionnaire to rate attention behaviours. While the psychologist's comprehensive evaluation revealed a significant attention disorder, the teacher's ratings were found within normal ranges on the attention questionnaire. The psychologist telephoned the teacher to discuss the rating scores and was surprised to hear that the teacher "does not believe in attention disorders."

A second point is that if we need to control for fatigue, noise, and attention during the administration of the APD test battery, then the same precautions need to be applied when screening measures are being administered. These tests should be administered in a quiet room, and in the morning to enhance reliable test results. False positive test results can be related to the test conditions.

There is an advertisement in the ASHA Leader marketing AP screening tools for ages 3 to 59. Thanks to Robert Keith we have new versions of the SCAN: one for adults/adolescents and one for children. The original SCAN (1986) test was considered a screening measure. The newest versions contain three screening measures and several diagnostic tests to be given if the individual fails the screening measure or if there is a referral for a diagnostic evaluation. The screening tool for very young ages is the Auditory Skills Assessment (ASA) (Geffner & Goldman, 2010) measuring discrimination ability in noise, proper understanding of nonsense words, blending phonemes, recognizing rhymes and ability to sequence nonverbal (music) sounds. A third choice is the Differential Screening Test for Processing (DSTP) (Richards and Ferre, 2006) which is the only test available that screens at various levels of auditory and language to identify areas for further evaluation.

PEARSON PRODUCTS

Auditory Skills Assessment for ages 3.6 to 6.11 years

SCAN–3:C Tests for Auditory Processing Disorders in Children (SCAN-3:C) ages 5 to 12 years

SCAN–3:A Tests for Auditory Processing Disorders in Adolescents and Adults (SCAN-3:A)

LINGUISYSTEMS

Differential Screening Test for Processing (DSTP) for ages 6 to 12.11 years

Canadian Hearing Report 2013;8(1):17.

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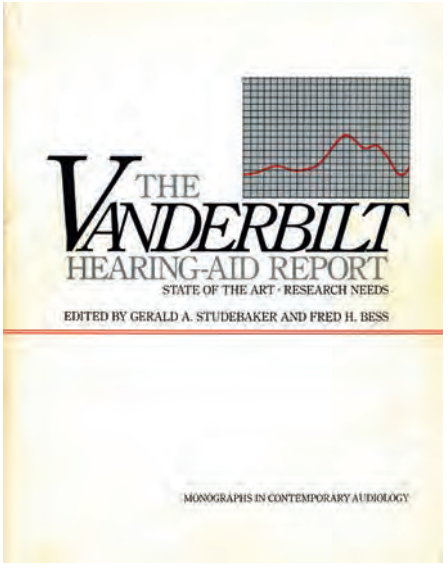
PHONAK
life is on

The Vanderbilt Hearing-Aid Report: State of the art research needs,

Edited By Gerald A Studebaker and Fred H. Bess. Monographs in Contemporary Audiology, 1982.

Reviewed by Marshall Chasin, AuD



This is one of my favourite books on my bookshelf and indeed was quite dusty. I looked at it often from afar and that seemed sufficient to remind me of what was in there and to remind myself of some important technologies and design approaches that permeated the 1980s. Dusting off the front cover, the first paragraph of the Preface gives the rationale for the "Vanderbilt Report":

> *This monograph reports the proceedings of "A Working Conference on Amplification for the Hearing Impaired: Research Needs" held at the Bill Wilkerson Hearing and Speech Center of Vanderbilt University in Nashville, Tennessee on June 7 through 10, 1981.*
>
> *The impetus for the meeting was the belief that progress on how best to design and apply amplification systems for the hearing impaired has been impeded by insufficient communication between researchers working on matters related to different aspects of the problem…."*

Actually, what was not said was even more important… There was a belief that progress… has been impeded by insufficient communication between manufacturers, researchers, and front line clinicians. And I must say that this is indeed the case today. In the 1970s and 1980s manufacturers were scrambling to glean information on how to build a better hearing aid and the front-line clinician was the rock star of the era. If a clinical or research audiologist showed up at a hearing aid manufacturer's facility, everything stopped and the audiologist was bombarded with questions and perspectives on how best to help the hard of hearing clients. In contrast, today it's the other way around. Many manufacturers have well-funded facilities and employ world class researchers that provide in-house solutions to hearing aid design problems. I can see strengths to both approaches, but for obvious reasons I recall a fondness for the early 1980s!

The "*Vanderbilt Report*" as it colloquially became known, brought together those researchers at the time who were at the top of their game. All aspects of the design and the fitting of hearing aids were addressed including new and updated models of normal and pathological auditory systems.

Section I is entitled "Basic Research – Sound Perception by the Hearing Impaired". There are articles on the psychoacoustics of elementary sound, spectral and temporal resolution by the hearing impaired, Spectral considerations in the speech discrimination ability of the hearing impaired, and temporal distortions in noise. The authors involved B. Scharf, M. Florentine, L. Humes, H. Levitt, and A. Nabelek. D. Dirks and S. Gelfand also contributed with well thought out comments on some of these topics.

The next section was a product of its time and technology. Section II is entitled "*Basic Research-Electroacoustic Considerations for Hearing Aid Performance.*" The articles were about research problems in coupler and in situ measurements, and functional gain correlates of electroacoustic performance data. The author list included E. Burnett, D. Preves, R. Cox. Reading this section

provides us with an amazing insight into the issues and audiological hurdles of the early 1980s.

Section III deals with "Engineering Applications – Special Problems." Topics include acoustic feedback control, telephone coupling, transducer and earmold effects, signal and speech processing, the evaluation of compression processing, and a new topic- recent research on multi-band compression. Again, the author list is impressive – S. Lybarger, D. Egolf, M. Killion, E. Libby, J. Lim, R. Schafer, L. Braida, N. Durlach, and E. Villchur. Some of the material in this section is dated but it's nice to see the original thoughts of some of the founders and early thinkers of this field. Acoustic feedback control was limited to simple phase shifting by an all pass filter that was non-linear with respect to phase- a far cry from today's use of phase control, notching filtering, and gain reduction, sometimes all available in one hearing aid product. E. Villchur is known as the father of modern day compression, and although his article is from 1981, it's well worth re-reading even today. (See also Founders of our Profession interview with Eddie Villchur in the *Canadian Hearing Report* 2008:3[1]:19–20.)

The final section (IV) is about the Delivery of Services and this discussion is as valid today as it was 30 years ago. Issues discussed were about hearing aid selection, a basis for selection amplification characteristics, validation of the selection measures and measures of hearing aid fitting success, factors affecting hearing aid use, and my personal favourite, a chapter by Mark Ross on Communication Access. Although Dr. Ross's article was written more than 30 years ago, it is still apropos to today's time. (See also Founders of our Profession interview with Mark Ross in the *Canadian Hearing Report* 2009;4(1):28–30.) Other authors read like the list of rock stars that they were- G. Studebaker, L. Beck, M. Skinner, D. Pasco, J. Miller, G. Popelka, G. McCandless, D. Byrne, B. Walden, E. Owens, M. Osberger, M. Collins, M. Ross, and D. Konkle.

Finally Fred Bess and Wayne Olsen wrapped it up in the final section V "Research Needs." Many of these issues and topics have still not been resolved today, but we are on our way.

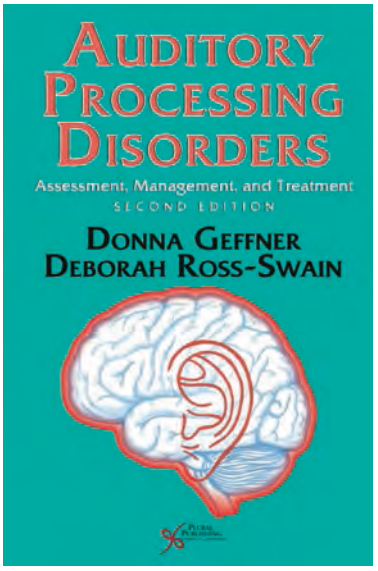
The *Vanderbilt Report* is a conglomeration of the state of the art 30 years ago. Reading through the yellowed pages will provide the reader with insight of where we have been, and even more importantly, where we are going.

Canadian Hearing Report 2013;8(1):19-20.

Congratulations to Gilbert Li,
Winner of the 2012 CAA Moneca Price Humanitarian Award

The Moneca Price Humanitarian award is presented to an audiologist in recognition of extraordinary humanitarian and community service, above and beyond the requirements of employment. The award is to honour the late Moneca Price, who took on significant leadership roles in both the Canadian Academy of Audiology (CAA) and the College of Audiologists and Speech Language Pathologists of Ontario (CASLPO).

This year's winner, **Gilbert Li**, provides free audiology services in under-privileged regions in the Yunnan Province of China. Prior to each visit, Gilbert invests substantial personal time in soliciting donated or refurbished hearing aids and equipment in preparation for his humanitarian work. The organization he volunteers with is called EMAS (Education, Medical Aid and Service). His team has seen over 300 people and dispensed 207 hearing aids – free of charge. The $500 award he received at the 2012 CAA conference in Ottawa was put towards furthering his work on another trip to China in November.



AUDITORY PROCESSING DISORDERS ASSESSMENT, MANAGEMENT, AND TREATMENT

Second Edition
November 2012 | 904 pages | ISBN13: 978-1-59756-495-3
$99.95 / £79.00
Plural Publishing Inc
Edited by Donna Geffner, PhD and Deborah Ross-Swain, EdD

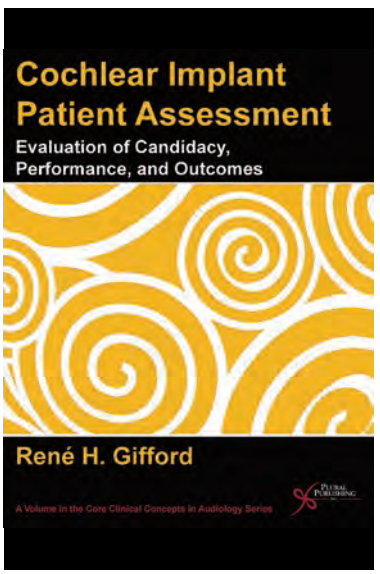
About the Book

This book details the history, definition, behaviours, and co-morbidity of auditory processing disorders before educating the reader on the most current practices for audiological and speech-language assessment of APD, including its impact on literacy and language processing. Practical rehabilitation and management strategies are covered in detail. This book a highly practical book designed specifically for practicing clinicians, both audiologists and speech-language pathologists.

Key Features

New to this edition:

- 5 new chapters written by a Who's Who of luminaries in the field of Audiology includeing Frank Musiek, Gail Chermak, Teri Bellis, Jeff Weihling, Harry Levitt, Chris Oden, Helen Simon, Carla Noack, Al Lotze, Bunnie Schuler, and Leah Light.
- New information from Nina Kraus and associates from the Northwestern Brain Volts lab on the value of using cABR in the diagnosis and measurement of APD in children, as well as a chapter on the effectiveness of musical training to alter that brain's engagement with sound.
- New chapter on case law from one of the USA's leading attorneys in special education, Gary Mayerson
- Updated information on the use of computer-based software programs from Larry Metwedsky.
- Three new appendices: Tips for Parents, Tips for Teachers, and a Study Questions Answer Key.

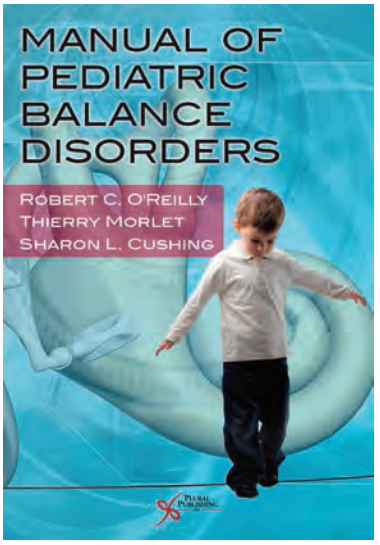


COCHLEAR IMPLANT PATIENT ASSESSMENT EVALUATION OF CANDIDACY, PERFORMANCE, AND OUTCOMES

January 2013 | 200 pages | ISBN13: 978-1-59756-446-5
$59.95 / £48.00
Plural Publishing Inc
By René Gifford, PhD

About the Book

This unique text is designed to provide information on best practices assessment of implant candidacy as well as the postoperative assessment of performance over the long term. This book is written for audiologists, speech-language pathologists and deaf educators to serve as a clinical handbook on the assessment tools and therapeutic intervention that are critical during the pre- and post-implant periods. Given that the numbers of both adult and pediatric cochlear implant recipients continue to increase, more and more clinicians will be expected to gain and maintain a level of experience surrounding the clinical management of this population. Though this book contains useful information for even the most seasoned clinicians, it will serve an especially important role in the education and training of students and clinicians being introduced to cochlear implant clinical practice. Having an experienced audiologist and speech-language pathologist authoring this work unites the inter-disciplinary nature of this practice.



MANUAL OF PEDIATRIC BALANCE DISORDERS

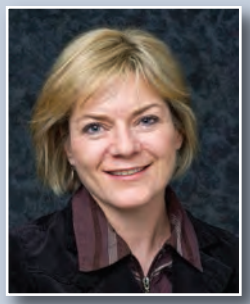
January 2013 | 250 pages | ISBN13: 978-1-59756-452-6
$129.95 / £103.00
Plural Publishing Inc
Robert C. O'Reilly, MD, FACS, Thierry Morlet, PhD, and Sharon L. Cushing, MD, MSc, FRCSC

About the Book

This book is designed to meet the informational needs of pediatric specialists—including medical specialists involved in the care of children with balance disorders—specifically otolaryngologists, neurologists, physiatrists, orthopedists, neurosurgeons, audiologists, and physical therapists.

Appendices cover reference information and cross references to existing Plural Publishing texts. The accompanying DVD includes self-assessment questions for each chapter covering principle teaching points.the educator as well as pragmatic tools for the student and clinician.

Neural Survival and Neural Population: Recent Findings with Electrically Evoked Compound Action Potential (ECAP) Measures in Cochlear Implant Users



Lendra.Friesen@sunnybrook.ca

Samidha.Joglekar@sunnybrook.ca

By Lendra Friesen PhD, and Samidha Joglekar, MCISc (C), Audiologist, Reg. CASLPO
Cochlear Implant Research Program, Sunnybrook Health Sciences Centre

One of the greatest challenges in hearing science research is the task of examining and understanding what produces patient variability in performance. We have all faced clinical situations in which we apply strategies that we expect will optimize patient experience and benefit, using methods that have worked with other patients in the past, only to find that these strategies are not optimal for the patient in question. It is then up to us, as audiologists and researchers, to consider the complex variables that make two patients who are similar in hearing loss type and configuration, duration of hearing impairment, and age, so different in their overall experience with a cochlear implant (CI) or a hearing aid. Research with auditory evoked potentials can allow us to investigate the auditory system at a deeper level and detect changes in the brain that may precede or follow certain behavioural outcomes in patients with hearing loss.[1] A growing area of interest is the exploration of auditory neural survival and correlates of patient performance.[2]

Variability in objective performance and perceived benefit is very common among CI users and can be perplexing for the clinical audiologist. A current area of study related to this issue focuses on investigating both auditory nerve survival and neural population size using electrically evoked compound action potentials (ECAP).[3] To review, the output of the cochlea is a series of action potentials (APs) that are conducted along the thousands of neurons that form the auditory nerve.[1,2] The ECAP is an auditory evoked potential that is electrically elicited from surviving auditory neurons and measured from an intra-cochlear (CI) electrode stimulated with biphasic pulses.[1,3] It is characterized by a single negative peak (N1) that is followed by a less prominent positive potential (P2).[1] Presently, various CI manufacturers have built-in systems and provide software to measure ECAPs using intra-cochlear electrodes for both stimulation and recording.[1,3,4] A major goal of this research is to make it relevant within a clinical framework, with the ambition of developing better cochlear implant fitting methods for the clinical audiologist.[2,3] However, this a long-term goal that depends greatly on our understanding of the complex changes that occur throughout the auditory system as a result of hearing impairment, the aging process, and neuroplasticity, all of which are key factors that generate high variability in patients' perceived, as well as measurable, benefit.[5]

Recent studies in this area of CI research have looked at the ECAP recovery function in relation to rate of stimulation and loudness perception. In brief, to measure the ECAP recovery function two pulses are used and the neural response to the second pulse is measured as a function of the inter-pulse interval.[3] Refractoriness in the neural fibres arises from the first pulse (the masker) and leads to a masking of the ECAP evoked by the second pulse (the probe).[3,6] The masker-probe interval is varied and the ECAP amplitude is measured at each interval in order to plot the recovery function.[3] It is important to remember that the ECAP, as well as the recovery function, are the collective response of numerous neurons.[1,3] Thus, if a given neuron exhibits refractoriness after firing, the capacity of the whole nerve to be excited might not be diminished if there are many other neurons in the region that are not in a refractory state and therefore ready to fire.[1,3]

An interesting study by Botros and

Psarros (2010) examined ECAP recovery functions with the hypothesis that ECAP recovery is heavily influenced both by neural survival as well as the size of the neural population that can be recruited to respond to a pulse-train stimulus. The popular understanding of the recovery function has been that faster recovery indicates a more efficient response to the individual pulses within a sequence.[3] However, psychophysical data have not supported this view and, in fact, the opposite has been observed; faster recovery has been found to lead to poorer psychophysical thresholds.[3] The researchers attempted to investigate this counterintuitive finding by postulating that the size of the neural population available to respond would heavily influence the refractoriness and operating status of the whole nerve. As it is not possible to count neural populations in human subjects, their study relied on a computational model of the cat auditory nerve and human ECAP measurements. Their findings demonstrated that slower ECAP recovery was related to better temporal synchrony with increasing stimulation rate.[3] Based on these findings they propose that the size of the neural population, in addition to neural survival, influences the whole nerve refractoriness: large neural populations operate near threshold and are more susceptible to masking, leading to **slower** ECAP recovery; however, they maintain temporal responsiveness through greater numbers of non-refractory neurons.[3,6] It is known that auditory nerve degeneration advances with duration of hearing impairment and another major finding of this study was that longer durations of hearing loss were associated with faster ECAP recovery.[2–4]

Overall, studies such as the one described highlight the complexity of the auditory system and the changes that occur in the brain when auditory input changes due to hearing loss. Studies that allow us to investigate brain-related changes are our gateway into discovering the underlying causes of patient variability, and ultimately in the long term, can help us provide the best clinical care for our patients.

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Canadian Hearing Report 2013;8(1):23-24.

Dr. Donald Henderson – Canada's Research Maestro of Noise-Induced Hearing Loss and Otoprotection

By Richard Salvi, PhD and Carol Altman
salvi@buffalo.edu

About the Author
Dr. Richard Salvi, (far left) distinguished professor and Carol Altman are with the Department of Communicative Disorders and Sciences and the Center for Hearing and Deafness, University at Buffalo, Buffalo, NY, USA.

Dr. Donald Henderson, Professor, Department of Communicative Disorders and Sciences and Center for Hearing and Deafness, University at Buffalo

On April 20, 2012, the Department of Communicative Disorders and Science and the Center for Hearing and Deafness hosted a one-day Noise-Induced Hearing Loss colloquium to honour Dr. Donald Henderson's numerous contributions to audiology, hearing science, noise-induced hearing loss, otoprotection, and ototoxicity during his 45 year academic and research career. Don was a prolific scientific contributor and author, outstanding teacher and mentor, skilled administrator and he was well known for organizing many high profile international conferences on "hot topics" in the field.

Don was born in Hamilton, Ontario, Canada on October 3, 1938. After graduating from high school, Don attended college at Western Washington State College in Bellingham, WA where he majored in psychology. Unknown to most of his colleagues, Henderson was recruited to Western Washington State on a football scholarship and, more importantly, played professional football for one season with the BC Lions of the Canadian football league. Having found professional football too easy, Henderson decided to pursue an academic career and in 1962 entered the PhD program in sensory psychology at the University of Texas at Austin. After completing his PhD in 1966, Henderson spent two years as a post-doctoral fellow at the prestigious Central Institute for the Deaf in St. Louis, MO where he developed an interest in auditory evoked potentials and noise-induced hearing loss. In 1968, he was appointed assistant professor at the State University of New York Upstate Medical Center in Syracuse, NY. Anticipating the digital revolution, Henderson purchased the first university laboratory computer, a DEC PDP8 with an "unbelievable" 4k of core memory and a teletype and tape reader for output and input. Using this advanced computer hardware and special amplifier, he was one of the early pioneers to assess auditory evoked potentials from humans and primates and to record single neuron discharge patterns from the auditory brainstem, techniques considered cutting edge at this time. In the early 1970s, Henderson teamed up with Dr. Roger Hamernik, a mechanical engineer who had been using a shock tube to study shock waves

Beaune, France.

produced by supersonic aircraft. Henderson, recognizing that blast wave exposure was a major cause of hearing loss among soldiers serving in Vietnam, convinced Hamernik to use his novel shock tube to study blast wave induced hearing loss in animal models. Working together, they published dozens of seminal papers documenting the relationship between the characteristics of the blast wave and the degree of hearing impairment and cochlear pathology. These studies highlighted the importance of peak pressure, spectrum and repetition rate in causing hearing loss. For example, at very high intensities, the blast literally ripped the cochlea apart while at lower intensities metabolic exhaustion played a significant role in hearing loss. Unexpectedly, the addition of moderate intensity background noise during a blast wave exposure was found to significantly exacerbate hearing loss and cochlear pathology. These findings cast doubt on universal application of the equal energy hypothesis to predict noise-induced hearing loss. These studies, along with others, helped guide the development of federal noise regulations.

During his tenure at the Upstate Medical Center, Henderson rapidly advanced to the rank of full professor. In 1980, he moved his research team to the University of Texas at Dallas where he was promoted to the director of the Callier Center. He also served briefly as the acting dean of the School of Human Development. In 1987, Henderson moved to the University of Buffalo (UB) and assumed the position of professor and chair in the Department of Communicative Disorders and Sciences. A few years after arriving at UB, he co-founded the Center for Hearing and Deafness, a multidisciplinary research group that included scientists and clinicians from a wide range of schools, departments and scientific disciplines.

INTERNATIONAL CONFERENCES

In 1975, Henderson organized the first in a long series of international conferences on noise-induced hearing loss, ototoxicity and acquired hearing loss. The proceedings of the first noise conference held at Cazenovia College were published in 1976 by Raven Press. This was followed by international noise conferences in Syracuse, USA, Beaune, France, Gothenburg, Sweden, Trento, Barga and Bari, Italy, Cambridge, England, and Niagara Falls, Canada. Henderson was also responsible for organizing international conferences on auditory plasticity, hair cell regeneration, tinnitus, immunologic diseases of the middle ear, central auditory processing, education of the hearing impaired, and ototoxicity. These conferences, which were scientifically stimulating, brought together leading scientists, administrators and clinicians from around the world in pleasant surroundings that fostered the exchange of ideas and new collaborations. For connoisseurs of elegant dining, the gala banquet in the heart of the wine cellars of Beaune will long be remembered as one of the best social and scientific events of the 20th century. A major accomplishment of these meetings was the compilation of 11 scholarly, widely read, state of the art books, some which are considered classics in the field of noise-induced hearing loss.

SCHOLARSHIP

Over the course of his distinguished career, Henderson authored more than 130 scientific papers published in prestigious journals, 43 book chapters and 11 edited monographs. Henderson has given more than 300 scientific presentations at national meetings,

Dr Chiemi Tanaka and Dr. Don Henderson.

Don and Terri in Italy.

international conferences or university colloquia. Over the course of his academic career, Henderson was able to secure continuous and substantial extramural grant support from numerous federal, state and private agencies including NIH, NIOSH, NIEHS, U.S. Army, NATO NOHR and DRF. Henderson often served as a consultant, reviewer, and advisor to many federal and state agencies. He served on numerous prestigious national and international committees including the National Institute of Occupation and Health (NIOSH), National Institute of Deafness and Other Communication Disorders (NIOSH) and Committee on Hearing and Bioacoustics (CHABA). Henderson was an associate editor for noise and health and the and was also a frequent reviewer for leading journals in audiology, hearing science and neuroscience such as *Ear and Hearing, Hearing Research, Nature Medicine, Journal of the Acoustical Society of America, and Neuroscience* to name a few. In recognition of his many accomplishments, Henderson received the National Research Award in 2006 from Hofstra University for distinguished contributions to the field of research in hearing science. In the same year, he received the Outstanding Hearing Conservationist Award from the National Hearing Conservation Association. Henderson was also granted two patents related to the development of drugs to prevent noise and drug induced hearing loss.

MENTORSHIP

In addition to collaborating with many prominent scientists in the field, Don has also mentored numerous MS, AuD, and PhD students in audiology and psychology. Henderson has a special gift for teaching and making anatomy, physiology, acoustics, clinical pathology and audiology sound easy, interesting and relevant in such courses as Advanced Hearing Science; Anatomy and Physiology of the Auditory System, Industrial Audiology, Introductory Psychology, Medical Audiology, Neural Basis of Communication Disorders, Physiological Psychology and Sensory Psychology. Over the years, he attracted many bright, highly motivated students who worked on research projects in his lab. Although he is best known for his work on noise induced hearing loss, his research interests spanned a broad range of topics including ototoxicity, evoked potentials, acoustic reflexes, cochlea anatomy, ototoxicity and age-related hearing loss. Several of his PhD students, including me, have gone on to hold faculty positions at major universities such as Ohio State University, SUNY Geneseo, West Virginia University, University at Buffalo and Medical University of South Carolina. His last PhD student, Dr. Chiemi Tanaka is a post-doc at Oregon Health Sciences University Hearing loss is a major problem for the armed forces and Don was responsible for training and mentoring 4 PhD audiologists who returned to military duty after completing their academic training. Don's intellectual and scientific achievements will continue to influence our understanding of hearing loss acquired from noise, aging and ototoxic drugs for many years and many of his former students will continue the scholarly work that Don started more than 45 years ago.

Ask him about the latest novels or economic, political, sports and social events and Don will immediately offer an "earful" of thoughtful, provocative and engaging commentary about the world around us. His quick wit, smile and active mind will immediately capture your interest and imagination. Don and his wife, Terri, love to travel, enjoy meeting new people, tasting new cuisines, exploring big cities, small towns and the natural environment. Don and Terri have traveled over most of North America and Europe and in addition, they have visited many parts of Asia, South America and the Far East. The other day, I thought I overheard him discussing the purchase of the Maid of the Mist to take family and friends on a trip around the world. Bon voyage, Don! We'll miss seeing you in the lab, but please stop by from time to time to tell us about your adventures. I see smooth sailing ahead.

Canadian Hearing Report 2013;8(1):25-27.

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oticon PEDIATRICS

New Insights on Cisplatin Ototoxicity

By Dalian Ding, MS, Jingchun He, Dongzhen Yu, Haiyan Jiang, Yongqi Li, Richard Salvi, PhD
dding@buffalo.edu

About the Authors

Dalian Ding (left) is with the Center for Hearing and Deafness, University at Buffalo, Buffalo, NY, USA; the Department of Otorhinolaryngology, Shanghai Sixth Hospital, China; the Department of Otorhinolaryngology, Third Hospital, Sun Yat-sen University, China; and the 4Department of Otorhinolaryngology, Xiangya Hospital of Central South University, China.

Jingchun He and Dongzhen Yu are with the Department of Otorhinolaryngology, Shanghai Sixth Hospital, China.

Haiyan Jiang and Richard Salvi are with the Center for Hearing and Deafness, University at Buffalo, Buffalo, NY, USA.

Yongqi Li is with the Department of Otorhinolaryngology, Third Hospital, Sun Yat-sen University, China.

OTOTOXICITY

Cisplatin and other platinum-based compound are widely used to treat a variety of solid and disseminated forms of cancer. Although cisplatin and related platinum compounds are highly effective anti-tumor agents, their clinical usage is limited by a number of serious side effects. Among these side effects, ototoxicity, neurotoxicity, and nephrotoxicity are the most common.[1–4] While ototoxicity is not life threatening, it can result in severe hearing impairment that can significantly degrade an individual's ability to communicate resulting in social isolation. Cisplatin ototoxicity can be particularly devastating when it occurs in young children because it can impair language development and social development.

CISPLATIN MECHANISMS

The long-term goal of administering anti-cancer drugs such as cisplatin is to block the uncontrolled proliferation and growth of cells that from malignant tumours. When cisplatin enters the cytoplasm of a cell, chloride ions bound to cisplatin are displaced by water molecules; the aquated cisplatin becomes a potent electrophile that forms intra- and interstrand cross links with DNA thereby preventing malignant cells from proliferating further. In addition, cisplatin binds with intracellular glutathione to form a toxic cisplatin-glutathione complex that can kill a malignant or healthy cell.[5] In order for either of these reactions to take place, cisplatin must first be transported from the blood stream into a malignant or healthy cell. How does cisplatin enter a cell and where does it go after it enters?

CISPLATIN AND COPPER TRANSPORTER

Copper is essential for life; consequently, cells have developed specialized transport mechanisms to control its uptake, export and compartmentalization. Ctr1, ATP7A, and ATP7B are three copper transporters that play prominent roles in regulating intracellular copper; however they were also recently shown to regulate the movement of cisplatin into and out of cells. Ctr1, located in the cell's membrane, is mainly responsible for transporting copper and cisplatin from the extracellular environment into the cytoplasm.[6] ATP7A and ATP7B are mainly responsible for sequestering intracellular copper into secretory vesicles in order to export copper out the cells.[7] Importantly, Ctr1, ATP7A and ATP7B were detected by in great abundance within the organ of Corti, stria vascularis and spiral ganglion neurons of the cochlea using immunocytochemistry[4,8] and likely play an important role in uptake of cisplatin into the hair cells and spiral ganglion neurons.

CISPLATIN DOSE-RESPONSE

We used organ cultures of the postnatal rat cochlea to study cisplatin ototoxicity in a precise and high controlled. We first determined the relationship between a

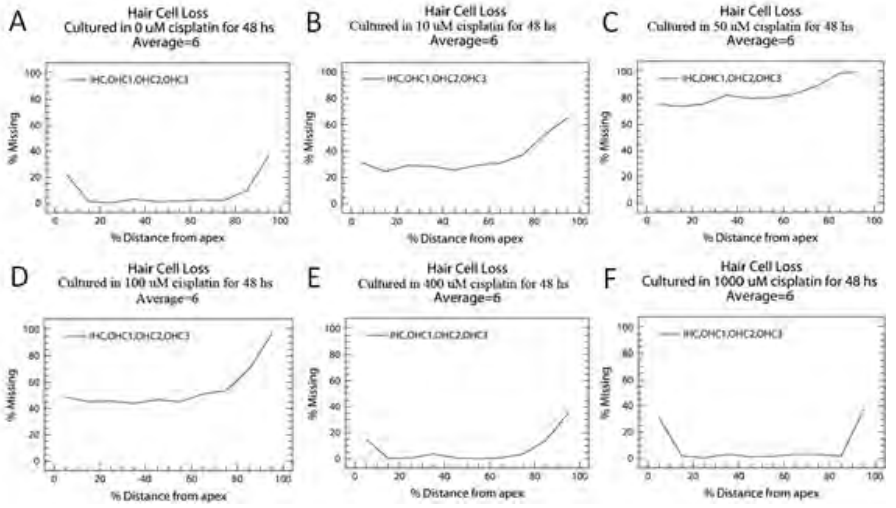

Figure 1. Mean (*n*=6) cochleograms showing the percentage of missing cochlear hair cells as function of percent distance from apex of the cochlea 48h after cisplatin treatments. (A) Control (0 μM) shows small hair cell loss near extreme base and apex due to preparation artifact. (B) 10 μM cisplatin; hair cell loss increases to 30-60%. (C) 50 μM cisplatin, hair cell loss increases to 80-90%. (D) 100 μM cisplatin, hair cell loss decreases to 50-90%. (E and F) 400-1000 μM cisplatin, most hair cells present; results similar to control in panel A.

wide-range of cisplatin concentrations and hair cell loss and obtained very surprising and unexpected results. Consistent with earlier studies, we found that low doses of cisplatin (10–50 μM) produced considerable hair cell damage (Figure 1B and C), but surprisingly hair cell loss began to decrease at higher concentration and little damage was observed at 400 or 1000 μM (Figure 1 D–F). Thus, cisplatin is highly toxic to hair cells at low concentrations, but hair cells become resistant to the drug at high concentration.[4,8]

CISPLATIN UPTAKE

To determine if the decreased toxicity at high concentration was due to decreased cisplatin uptake, we labelled cisplatin with Alexa Fluor 488 to track its uptake into hair cells.[4,8] At low cisplatin concentration, a large amount of cisplatin was observed in damaged outer hair cells (Figure 2A); however, at high concentrations, little cisplatin uptake was observed in normal looking outer and inner hair cells (Figure 2B).

Western blots were used to quantify the presence of Ctr1, ATP7A and ATP7B proteins in normal and cisplatin treated cochlear cultures. Ctr1, which regulates the uptake of cisplatin, was relatively unchanged following treatment with cisplatin. However, ATP7B, which mediates the efflux of cisplatin increased significantly.[4,8] These results suggest that cochlear hair cells dynamically upregulate the expression of ATP7B in order to extrude excess cisplatin and protect against cisplatin damage.

EXTRACELLULAR COPPER INHIBITS CISPLATIN DAMAGE

The uptake of cisplatin through Ctr1 can be competitively inhibited by extracellular copper[9] and therefore should protect hair cells from cisplatin damage. To test this hypothesis, cochlear cultures were treated with cisplatin (10 μM) alone or cisplatin plus copper sulfate (10, 50 or 100 μM). Cisplatin induced hair cell loss was completely prevented by the addition of copper sulfate.[10–12] However, copper sulfate failed to prevent hair cell loss when the cisplatin dose was increase to 50 μM. Thus, the protective effect of copper sulfate in vitro is only protective at low cisplatin concentrations. Additional studies with Western blots showed that the expression of Ctr1 was greatly reduced by copper sulfate, while the expression of ATP7B was significantly increased. These results suggest that the protective effect of copper sulfate may arise from decreased influx and increased efflux of cisplatin.

COPPER SULFATE PREVENTS OTOTOXICITY IN CHINCHILLAS

Carboplatin, a less ototoxic derivative of cisplatin, preferentially damages inner hair cells (IHC) in chinchillas.[13] To determine if extracellular copper can protect cochlea against carboplatin-induced hair cell loss in vivo, we applied 50 μL of copper sulfate (100 μM) on the right round window membrane of the chinchilla cochlea; 50 μl of saline was applied to the left round window membrane. Immediately afterwards, 50 mg/kg (i.p.) of carboplatin was administered to the chinchillas. Ten day after carboplatin treatment, the left and right cochleas were evaluated to determine the degree of hair cell loss. As shown in Figure 3, IHC loss was greatly reduced in the copper sulfate treated cochleae (Figure 3B) compared to saline treated cochleae (Figure 3A). These results indicate that local application of copper sulfate on round window provides significant protection against carboplatin-induced IHC loss presumably by reducing the influx of carboplatin into the hair cells.

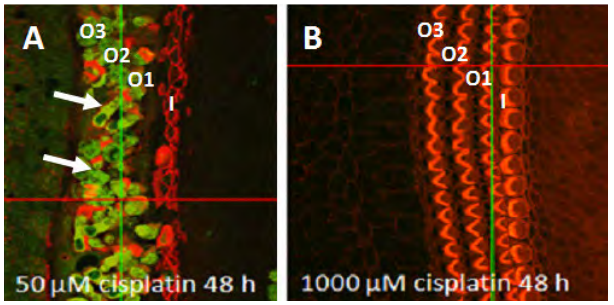

Figure 2. Confocal images of cochlear organ cultures labelled with Alexa Fluor 488-labeled cisplatin (green) and fluorescently-labelled phalloidin (red) that labels the actin in outer hair cells (O1, O2, O3) and inner hair cells (I). (A) 48 h after 50 µM cisplatin exposure, Alexa Fluor 488-cisplatin (green) found in damaged and dying hair cells (arrows). (B) 48 h after 1000 µM cisplatin treatment; hair cells were intact with no Alexa Fluor 488-labeled cisplatin.

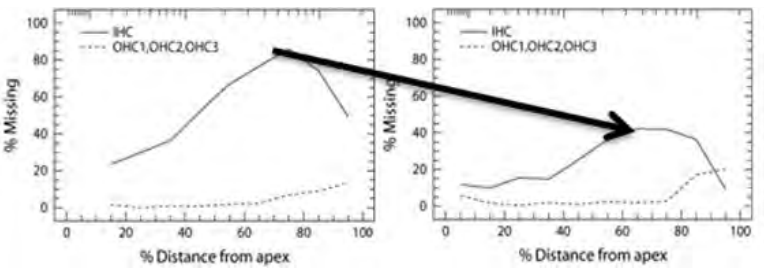

Figure 3. Mean cochleograms (n=5) showing the percentage of missing inner hair cells (IHC) and outer hair cells ($OHC^{1,2,3}$) in the left, saline treated ear (A) and right copper sulfate treated ear (B) 10 days after carboplatin (50 mg/kg, i.p.) treatment. Note that the carboplatin-induced IHC lesion was much less (arrow) in cochleas treated with copper sulfate than saline.

SUMMARY

Many strategies have been considered to prevent the cisplatin ototoxicity. Some strategies involving the use of antioxidants have been shown to provide some protection after cisplatin enters the inner ear and begins to induce damage. The new strategy adopted here provides protection by suppressing the uptake of cisplatin into the hair cells and support cells. Local application drugs to block the uptake of cisplatin or enhance its extrusion from hair cells represent a novel approach to preventing ototoxicity.

ACKNOWLEDGEMENTS

Supported in part by NIH grants R01DC006630, and in part by the Project-sponsored by SRF for ROCS, SEM.

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Canadian Hearing Report 2013;8(1):29-31.

Development of Early Detection of Noise-Induced Hearing Loss with Forward Masking of the Auditory Brainstem Response

By Eric C. Bielefeld, PhD
bielefeld.6@osu.edu

About the Author
Eric C. Bielefeld, PhD, is with the Department of Speech and Hearing Science, The Ohio State University, Columbus, OH.

Noise-induced hearing loss (NIHL) continues to be a significant public health problem for industrialized populations.[1] Currently, prevention of NIHL primarily involves hearing conservation programs built around modifications of the noise source, use of hearing protection devices, and education and counselling on noise and the consequences of hearing loss over the life span.[2,3] Noise standards typically rely on noise exposure measurements to dictate who needs enrolment into a hearing conservation program. Yet the problem remains that even the most conservative standards do not effectively account for all acoustic factors that dictate the potential ototoxicity of noise, and leave a significant percentage of workers vulnerable to hearing loss. Therefore, there exists a need to detect those workers who are experiencing NIHL as early as possible. Many current noise standards define NIHL as a minimum of 10 dB of threshold shift at one or multiple frequencies. The current work was undertaken with the belief that 10 dB or more threshold shift is already too much to allow for the large noise-exposed population, and that even that mild threshold shift has the potential to lead to more severe cochlear degradation over time.[4–6] Early detection of damage, prior to the accumulation of enough cochlear damage to cause significant threshold shift, would be a powerful tool to minimize the incidence of clinically-significant NIHL.

The current studies in the Auditory Physiology Lab at The Ohio State University were undertaken as an extension of work performed by Oxenham and Plack[7] who studied behavioural measures of cochlear compression using forward masking (FWM) growth of masking (GoM) functions. FWM occurs when the masker sound is presented prior to the probe sound, with a silent gap separating the masker and probe. GoM functions chart the masker threshold, the minimum masker level required to mask out the probe, as a function of the probe level. In Oxenahm and Plack,[7] for normal-hearing listeners, in conditions in which the probe and masker frequencies were the same (the on-frequency condition), the GoM function was linear. For example, the masker threshold for a 50 dB SPL 6 kHz sound was 50 dB SPL when the masker was also a 6 kHz sound. When the probe level was increased to 60 dB SPL, the masker threshold increased to 60 dB SPL. But when the masker was one octave below the probe (the off-frequency condition), the GoM function of was highly compressive. In the off-frequency condition, a high masker level was required to mask even lower-intensity probe levels. For example, the 50 dB SPL 6 kHz sound required an 80 or 85 dB SPL masker threshold when the masker was a 3 kHz sound. This compressive non-linear off-frequency GoM function changed significantly in the study participants with sensorineural hearing losses. In those participants, the off-frequency GoM was linear, and closely mirrored the on-frequency condition. The results indicated that the linear off-frequency FWM GoM function reflected a loss of cochlear compression in the participants with sensorineural hearing loss.[7] Since the non-linear nature of the off-frequency FWM GoM functions is attributable to active processing from the outer hair cells, the FWM GoM functions are expected to be sensitive to noise-induced cochlear damage. The current study investigated

changes in FWM GoM functions using on-frequency and off-frequency maskers of 7 and 10 kHz probes in the Sprague-Dawley rat. An animal model was used because a test for early detection of NIHL requires presentation of a noise that will reliably produce a slow, gradual hearing loss, and that animal model allows for cochlear analyses to confirm the extent of damage induced by the noise exposure.

For the initial study, 11 Sprague-Dawley rats' auditory brainstem responses (ABRs) from probe stimuli of 7 and 10 kHz were recorded with and without forward maskers to create FWM GoM functions. The off-frequency forward maskers were one octave below the 7 and 10 kHz probes. The masker threshold was defined as the masker level required to reduce the initial positive wave of the rat's ABR to 50% or less of the unmasked waveform. The rats were then exposed to a hazardous noise (a 5–10 kHz octave band noise at 110 dB SPL combined with 120 dB pSPL impacts presented at a rate of 1/second with a total duration of 120 minutes) that induced permanent threshold shift. After the noise exposures, the FWM GoM functions were measured to assess noise-induced changes in the FWM GoM functions.

Pre-exposure FWM GoM functions showed compressive non-linear functions in the off-frequency conditions, and linear functions in the on-frequency conditions for both the 7 kHz and 10 kHz probes. The hazardous noise induced a 30–40 dB permanent threshold shift in the 5–20 kHz frequency range. The NIHL rendered the off-frequency FWM GoM functions more linear, and made them statistically indistinguishable from the linear on-frequency conditions, which changed little from pre to post noise exposure.

The findings from this initial study demonstrate that FWM GoM functions of the rat ABR behave in a pattern consistent with human behavioural work in both the normal condition prior to any noise exposure, and after cochlear damage from noise. The off-frequency FWM GoM functions displayed compressive non-linearity in the normal-hearing animals, and then appeared linear after significant NIHL that is assumed to have created a large lesion of damage/dead outer hair cells (please see review by Henderson et al.,[8] on noise-induced outer hair cell damage). The results serve to validate ABR FWM patterns as potential research tools for detecting acquired changes in the cochlea, which was important because FWM GoM functions had never been tested in an eletrophysiologic ABR paradigm in an animal model before. Currently, ongoing studies are aimed at determining the most stable and sensitive dependent variable for detecting changes in the ABR. The study reported currently utilized the point of 50% reduction in amplitude as the dependent variable, but initial findings suggest that latency shifts might be more stable and sensitive indicators of cochlear damage than amplitude changes. The other ongoing phase of the study is using a long-term noise at a level that will gradually induce cochlear damage. The rats in this study are being exposed to the combined 5–10 kHz octave band continuous noise with the high-level impacts, but the level had been reduced from 110/120 dB SPL to 96/106 dB SPL. The rats are being exposed to that noise for 4 hours per day, 4 days per week (Tuesday through Friday). They then recover on Saturday and Sunday and are tested on Monday. The off-frequency FWM GoM functions are measured weekly to determine if changes in the FWM functions precede significant changes in threshold. If they do, and if the FWM changes correlate with underlying cochlear pathology, then the test may be a useful measure for detecting early NIHL.

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Canadian Hearing Report 2013;8(1):32-33.

The Biology of Oxidative Stress:

Lessons from the Proteins of the Cochlea – Or, What Every Audiologist Wants To Know about Auditory Biochemistry

By Donald Coling, PhD
dcoling@buffalo.edu

About the Author
Don Coling is a research associate professor, Center for Hearing and Deafness, Department of Communicative Disorders and Sciences, University at Buffalo, the State University of New York.

My essay is written for audiologists and dedicated to my audiology teacher, my colleague, and my friend, Donald Henderson. It was presented as an oral seminar on April 20, 2012 in Buffalo at the Donald Henderson Noise-Induced Hearing Loss Colloquium in honor of Dr. Henderson's retirement from the Center for State University of New York at Buffalo. Although the essay focuses on damage to proteins, we would be remiss not to briefly mention the importance of oxidative damage to lipids and DNA. We can't escape the influence of molecular biology and chemistry on our lives. We don't have to understand the chemistry in detail, but, we've reached a time when we have already begun to treat hearing disorders with chemistry. As hearing professionals, it behooves us to be aware of a few basic ideas in auditory biochemistry. I've tried to present them in a painless and easy-to-read manner that should help give some perspectives in the practice of modern audiology.

SOURCES OF OXIDATIVE METABOLITES

Radiation, toxic chemicals, and hyper-activity, can induce oxidative stress characterized by the production or accumulation of reactive oxygen species (ROS). ROS is the name given to oxygen containing molecules that have a particularly high chemical reactivity. The most frequently studied ROS are superoxide and nitric oxide. Superoxide is produced when oxygen gains an electron from cellular enzymes in mitochondria, peroxisomes, nuclei and cytoplasm. Nitric oxide is produced when nitrogen is removed from the amino acid arginine in the cytoplasm by nitric oxide synthases. ROS are used at low levels as physiological signalling molecules before being degraded by antioxidant enzymes. ROS are effective signalling molecules because their reactive electron structure and short lifetime allows them to quickly combine with nearby cellular proteins, change the protein's surface charge and shape, and thereby change its function by affecting the strength of interactions with other proteins that depends on surface charge and shape. If the level of ROS is low, cells can rapidly degrade the signal and activate an antioxidant response in preparation for higher levels of ROS – a state of preparedness. However, if the level of oxidative metabolites is too high, ROS can combine with each other and with other molecules to form a variety of metabolites, giving rise to irreversible oxidative damage to proteins, lipids, and nucleic acids.

The title of this essay, the "Biology of Oxidative Stress," is an enormous topic that has produced over 200,000 research articles indexed by the National Library of Medicine over the last 60+ years. This article will focus on only one aspect in this large arena and that is oxidative damage to proteins in the cochlea. We are particularly interested in damage to outer hair cells because they are typically the most vulnerable to environmental insults to the inner ear.

ROS AND HEARING LOSS

Several lines of evidence have underscored the importance of ROS in cochlear pathologies. Purturbations that generate hearing loss include ionizing radiation and heat, toxic chemicals, hyperactivity from noise and loud music,

and aging. Each of these perturbants is known to induce oxidative damage. ROS have been detected at early time points after cochlear damage.[1,2] and have been shown to induce direct pathological effects on outer hair cell morphology.[3,4] Endogenous cochlear antioxidant enzymes are induced by noise exposure.[5] The transcription factor Nrf2 which regulates antioxidant enzyme gene induction has been linked to cisplatin ototoxicity[6] and to age-related hearing loss and gentamycin ototoxicity.[7] Finally, several studies have shown that supplemental doses of small molecule antioxidants and compounds that mimic antioxidant enzymes can protect from hearing loss induced by ototoxic drugs,[8–14] hypoxia-reperfusion,[15] and noise.[16–20] This list is not exhaustive, but accompanies some of the ground breaking manuscripts. Several excellent reviews have been published that give a more comprehensive assessment of the role of ROS in hearing loss.[21–28]

METABOLITES AND SMALL MOLECULES IN REDOX IMBALANCE

Small molecules can contribute to both oxidative stress and redox homeostasis. The best example is glutathione, the most abundant cellular antioxidant. Glutathione is like a mini-protein. It contains three amino acids, the building block of proteins and is used as a reducing agent. That means it can donate an electron in reactions to reduce oxidizing agents. In doing so, glutathione itself becomes oxidized. So in a redox reaction, a reducing agent becomes oxidized and an oxidizing agent becomes reduced. The protective effect comes from the fact that cells can tolerate oxidized glutathione better than oxidizing agents. For example, hydrogen peroxide, a metabolite of superoxide, can be detoxified by the enzyme glutathione peroxidase. In the reaction, the oxidizing agent, hydrogen peroxide, is converted to water and oxygen while two molecules of the reducing agent, glutatione are joined to form oxidized glutathione. Glutathione reductase then restores the cell to its normal state by reducing oxidized glutathione. If the peroxide is not detoxified it can metabolize noncatalytically to more harmful oxidizing agents and do irreversible damage by reacting with proteins, DNA and lipids. So glutathione peroxidase is just one example of detoxifying enzymes that borrow an electron from glutathione to lower the reactivity of ROS. In addition to glutathione, there are a variety of other small molecules that affect redox balance. These involve dietary intake of vitamins, glucose, fatty acids, lipoproteins, and heavy metals.[29] The chemistry is complicated, but the take home message is clear. Eat plenty of glutathione. You can get lots of it in broccoli and asparagus. Get lots of vitamins. These are small molecule electron and proton carriers needed by our enzymes that our bodies can't make. But plants can, so eat lots of vegetables. You'll have better hearing when you're old. Share this chemistry lesson with your patients – young and old.

LIPID OXIDATION

If the cellular redox state is upset such that antioxidant defense enzymes like glutathione peroxide and small anti-oxidants like glutathione and vitamins E and A are unable to detoxify ROS fast enough, then macromolecules become damaged. One consequence is lipid oxidation. Lipids are the molecules used to build membranes. Cells may be thought of as little chemical plants each with its own specialized chemistry. The chemistry within cells is water-loving or hydrophilic meaning that it occurs in the aqueous cytoplasm or on membranes facing an aqueous environment. Specialized chemistries are separated by barriers made of hydrophobic lipid molecules that form the plasma membrane around cells and intracellular compartments within cells. These lipid membranes are like the outside and inside walls of our homes. When the lipids get oxidized, the membranes leak and the specialized chemistries in separate compartments get mixed together. A small leak can be repaired. A massive leak will signal cell death. Those with some background in cell biology are referred to.[30–32] A little dose of redox chemistry practiced at the dinner table can help audiologists and their patients here too.

DNA DAMAGE FROM OXIDATIVE STRESS

DNA damage oxidative stress has very serious consequences. Thousands of DNA modifications from oxidative damage occur in every cell daily and lead to mutations, epigenetic and oncogenic reprogramming and programmed cell death. A good example is damage to skin cells from ultraviolet radiation leading to melanomas. While cochlear outer hair cells can not be damaged by sun exposure, they have an exceptionally high metabolic rate are subject to enhanced vulnerability from noise exposure and exposure to a variety of ototoxic drugs. As we get older, we've found that the neurons of the cochlear spiral ganglion are particularly susceptible to oxidative deletion of DNA within their mitochondria.[33] Of particular importance to hearing is the ototoxic side effect of the cancer drug cisplatin. Treatment of many cancers depends on the ability of cisplatin to generate ROS and on its ability to form DNA adducts. Cisplatin-DNA adducts signal DNA repair in most cells of the body. High doses of cisplatin, however, can overwhelm DNA repair mechanisms resulting in the initiation of programmed cell death. This mechanism evolved to

protect organisms from retaining cells with damaged DNA. It is exploited in treatment of tumours with cisplatin at the expense of negative side effect in particularly vulnerable cells like kidney tubule cells and cochlear outer hair cells.[14,26,27] Since cochlear hair cells cannot regenerate from basal precursors like olfactory receptors, DNA damage repair mechanisms are particularly important to cochlear function. Recently attempts have begun to employ agents to prevent DNA damage as otoprotectants.[34]

REVERSIBLE PROTEIN OXIDATION AND COCHLEAR FUNCTION

To date, there is very little direct evidence for the physiological role of protein oxidation in normal cochlear function. However, evidence for the presence of reversible protein oxidation in the cochlea suggests that protein oxidation will no doubt play important physiological roles in redox signalling and pathological roles from oxidative damage.

Two types of oxidative processes that occur in every cell under normal physiological conditions are disulfide formation and S-nitrosylation. Interestingly, both involve the amino acid cysteine found in glutathione. As in glutathione, the sulfhydryl groups of cysteine found in proteins can also oxidize to from a disulfide –S-S- linkage between proteins or between two cysteines on a single protein. The linkage formed causes proteins to maintain a given shape until the disulfide bond is reduced. You can imagine it to be like a pair of handcuffs. With this analogy, you can imagine an enzyme whose hands are tied might no longer be able to do the same activities as in the reduced state when the cuffs are removed. Nitrosylation also involves a modification of the amino acid cysteine where nitrogen and oxygen from nitric oxide are covalently joined to the SH group to form an S-ntrosothiol (SNO). This can happen to cysteines in small tripeptides like glutathione or polypeptides (proteins). Nitrosylation can be used in signalling to alter enzyme or ion channel activity and may serve as a means of delayed release of nitric oxide. Besides modification of the amino acide cysteine, dissulfide formation and nitrosylation share one other important aspect. They can be enzymatically reversed. That means the functional changes they induce in proteins can be reversed and cell function can return to the initial state of reducing conditions. Like other aspects of cell signalling, high levels of oxidative stress can overwhelm cells. Thus, dysregulation of disulfide formation and nitrosylation has been associated with stroke, Parkinson's and Alzheimer's diseases, amyotrophic lateral sclerosis and cancer. Though both are undoubtedly important to hearing function, evidence supporting a connection is just beginning to emerge.

A recent publication suggests that S-nitrosylation may also be important in cisplatin ototoxicity. Cisplatin was found to induce elevations in the levels of S-nitrosylation in certain cochlear proteins and reduced levels in others.[35] Ongoing research is aimed at identifying these proteins and discerning their role in hearing. Glutathionylation of cochlear proteins is elevated in the aging cochlea.[36] Glutathionylation, the covalent attachment of glutathione to protein sulfhydrils is an emerging field in protein regulation and numerous labs have reported crosstalk between nitrosylation and glutationylation.[37]

Glucose regulated protein 58 (Grp58) is a stress-induced[38] endoplasmic reticulum protein[39] whose cochlear expression was altered by cisplatin.[40] Pdia3 is important in the formation of dissulfide bonds[41] and in protection from prion-induced apoptosis.[42] The enzyme can be phosphorylated by src family kinases[43] and src inhibitors have been show to protect the cochlea from hearing loss induced by noise[44] and cisplatin.[45] The alteration in Pdia3 cochlear expression[40] is consistent with protein phosphorylation, thus it is of great interest to determine whether Pdia3 phosphorylation is inhibited by src kinase inhibitors.

OXIDATIVE DAMAGE TO PROTEINS – IRREVERSIBLE OXIDATION OF COCHLEAR PROTEINS

Pathological damage to proteins occurs when modified proteins cannot be repaired or efficiently removed by proteolysis and replaced by protein synthesis. Oxidized proteins are recognized by cellular proteolytic machinery, but, in some cases, oxidized proteins can accumulate and even inhibit the proteolysis of other damaged proteins.[46] In addition to modifying amino acid constituents of proteins, oxidation can lead to dysfunction through protein cleavage and by pathological cross linking. The chemistry of protein oxidation is diverse and reviewed in detail elsewhere.[45–47] What every audiologist will want to know is found right here.

Irreversible protein oxidation includes cleavage, crosslinking, carbonylation, oxidized lipid-protein adduct formatin and nitration. The first three processes, though common, have simply not been studied in the cochlea. The fourth has been reported describing 50-60 kDa 4-hydroxynonenal-protein adduct in mouse cochlea following noise exposure.[48] The fifth, nitration, is the process by which the amino acid tyrosine is irreversibly modified by ROS-

mediated addition of a nitro group. Modification of proteins by nitration can be benign with no effect on function. Alternatively, protein tyrosine nitration can lead to either gain or loss of function. Unlike nitrosylation of cysteine, nitration is irreversible and often taken as a marker of oxidative damage.

The presence of nitrated proteins in the cochlea was first reported by immunocytochemistry using antibodies against nitrotyrosine in lipopolysaccharide-induced damage to guinea pig cochlea[49] and has been used as a marker of cochlear oxidative damage in several subsequent studies.[36,48,50–64] The major nitrated protein of cochlea was first reported to be a 76 kDa protein whose nitration was induced by cisplatin.[65] This protein has recently been identified as Lmo4[66] a transcriptional regulator associated with neuronal survival.[67] Ongoing research will determine the mechanism by which Lmo4 contributes to survival of outer hair cells and by which nitration of Lmo4 contributes to outer hair cell loss. Future studies will determine whether Lmo4 is critical to the survival other cochlear cells and whether Lmo4 nitration is an important factor in the pathology of other tissues.

Lmo4 is likely one of several key outer hair cell proteins damaged by oxidative stress. Investigations of stress-induced carbonylation, glutathionylation and proteolysis will identify other damaged proteins and help piece together the puzzles of the initiation of cochlear pathologies.

DRUG DISCOVERY FOR NOVEL TREATMENTS OF OXIDATIVE DAMAGE TO THE COCHLEA

We have already mentioned some of the pioneering work with antioxidant intervention to treat hearing loss. Donald Henderson, the person we honour in this issue, has pioneered a novel treatment for hearing loss. The Henderson Lab has shown that inhibitors of the protein kinase src can provide protection from noise induced hearing loss[44,68–70] and hearing loss from the cancer drug cisplatin.[45] Src regulation by phosphorylation is well characterized, but src is also known to be activated in states of elevated oxidation by the formation of dissulfide bonds between catalytic and regulatory domains.[71–73] When activated in lung endothelial cells, src phosphorylation of the regulatory p47phox subunit of NADPH oxidase signal translocation to the plasma membrane and elevated production of superoxide by NADPH oxidase.[74] This can represent a type of feed forward amplification where small increases in ROS can avalanche into even high levels of oxidative stress. The situation can be amplified further at the transcriptional level. For example, cisplatin leads to the induction of Nox3, an NADPH oxidase that is highly expressed in the cochlea.[75,76]

HENDERSON LABORATORY CONTRIBUTIONS TO OXIDATIVE STRESS AND HEARING

The person we honoured in Buffalo has not ignored these issues by any means. Don Henderson has contributed to hearing research with 5 manuscripts on toxic chemicals, 16 on aging, and 96 on noise-induced hearing loss. He is well known for the books and special journal issues that resulted from the symposia on noise-induced hearing loss that he organized with Roger Hamernik, Richard Salvi and a host of others,[77–85] books related to hearing,[86–88] and over 40 book chapters. Although his background is in physiological psychology, with Bo Hua Hu, Henderson contributed one of the earliest papers on apoptosis in noise-induced hearing loss which pointed to the importance of programmed cell death in outer hair cell pathology.[89] In another pioneering paper, he and Dr. Hu explored the role of mitochondria in programmed cell death.[90] Henderson made many contributions that outlined the importance of the induction of antioxidant enzymes by noise[5,24,91] and by aging.[92,93] Over the latter part of his career Henderson made significant strides in developing therapeutic strategies for treating hearing loss induced by noise and drugs.[44,45,68–70,82,83,94–103] Among his most recent efforts has been the discovery and development of otoprotection using inhibitors of the src kinase family.[45,68–70] We are indebted to Donald Henderson for these and many other contributions to hearing research.

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Please go to http://andrewjohnpublishing.com/documents/CollingReferencesforCHR.pdf for a complete list of references for this article.

Canadian Hearing Report 2013;8(1):34-37.

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The Relationship between Hearing Loss and Auditory Hair Cell Loss in Rats

By Guang-Di Chen, PhD
gchen7@buffalo.edu

About the Author
Guang-Di Chen, PhD, is with the Center for Hearing and Deafness, The State University of New York at Buffalo.

Intense noise and numerous ototoxic agents may target and damage cochlear hair cells leading to hearing loss. Auditory hair cells in mammals, including humans, cannot be regenerated; thus, hearing loss due to hair cell death may never be restored. We have found that many auditory hair cells may still survive in the cochlea under severe hearing loss. If a certain number of dysfunctional auditory hair cells survive in the cochlea, treatment of hearing loss may become possible in the future. Therefore, it is important to know the relationship between hearing loss and hair cell loss under different circumstances. This report presents data showing surviving hair cells in the cochlea of rats exposed to intense noise and styrene, an industrial solvent.

METHODS

The first group of rats (n = 61) were exposed to an intense noise (10–20 kHz, 100–115 dB SPL) for 2–4 hours in a chamber with normal oxygen level (21%) or lower oxygen supply (18% and 10%) or higher carbon monoxide (CO) concentration (up to 0.15%). The second group of rats (n = 50) were exposed to styrene by oral gavage (0–800 mg/kg once per day for 5 days per week for 3–24 weeks). Hearing loss was assessed 4 weeks after exposure by measuring threshold shift of cochlear compound action potential (CAP) as described in our previous reports.[1,2] Hair cells were stained and counted as described in our previous reports.[3,4]

RESULTS AND DISCUSSION

Hearing Loss Induced By Intense Noise or Combination of Noise and Hypoxia/CO

Intense noise may cause a complete loss of cochlear amplification, which leads to a CAP threshold shift of about 40 dB, without hair cell loss. Figure 1A presents an example showing a linear CAP input/output function at 12 kHz (i.e. 1 dB decrease of stimulation causes 1 dB of reduction of CAP amplitude) obtained in a rat at post-4-week of noise exposure (see red filled circles). In normal cochlea, reduction of CAP is smaller than the decrease of stimulation (open circles) indicating that the response in the normal cochlea is amplified. Since outer hair cells (OHCs) are known to contribute to the cochlear amplification, the loss of cochlear amplification after noise exposure should indicate damage of OHCs. However, all OHCs in the corresponding region in the cochlea look intact (see Figure 1B).

Our data from 61 noise-exposed rats revealed that (1) in the apical and middle turns there was no significant hair cell loss until a hearing loss of 40–50 dB was reached; (2) in the basal turn (75–100% from the apex or >30 kHz), OHC loss increased monotonically with hearing loss. The surviving OHCs must be dysfunctional. Restoration of the dysfunctional OHCs may open an avenue to hearing rescue at least in patients with noise trauma.

Hearing Loss Induced By Styrene

Styrene is extensively used in industry. Ototoxicity of styrene in workplace is still examined by pure tone audiometry. However, we have found that this kind of ototoxic agents may cause a certain level of cochlear damage with little or no effect on hearing threshold. In other words, styrene exposure which has been accepted as a safe level may already be ototoxic. Figure 2 presents an example showing a severe styrene-induced OHC loss (A) with no reduction of CAP amplitude at the corresponding frequency (B). The rat was exposed to

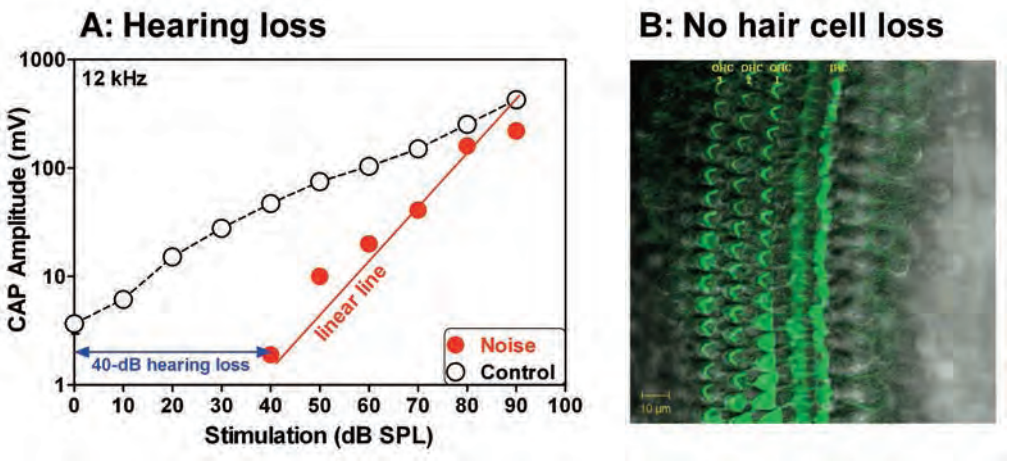

Figure 1. Noise-induced hearing loss may occur without hair cell loss.

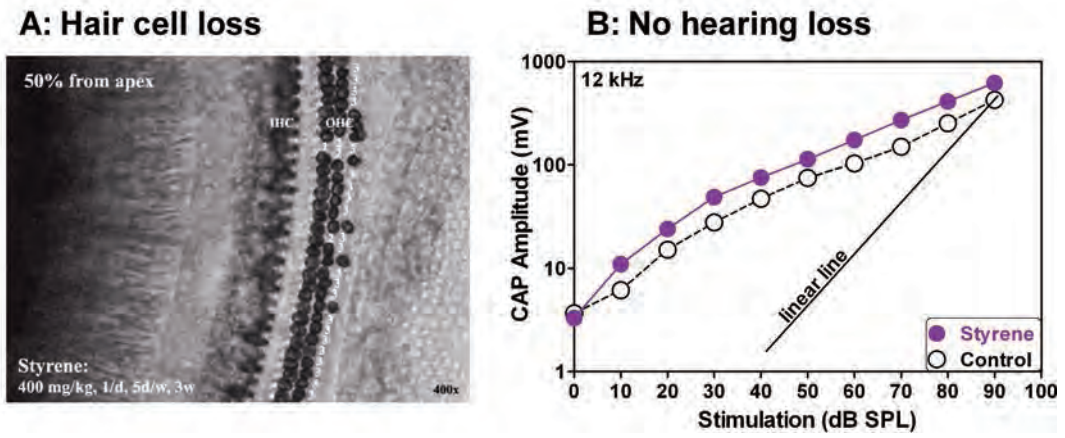

Figure 2. Hair cell loss up to 1/3 after styrene exposure may not result in a functional loss.

styrene at a level of 400 mg/kg per day for 5 days per week for 3 weeks. Almost all OHCs in the third row in the middle turn were missing (marked by "3" in Figure 2A), but CAP input/output functions (see purple filled circles in Figure 2B) at the corresponding frequencies were similar to the control (open circles) indicating a normal cochlear amplification with only 2 rows of OHCs remained. Our data from 50 styrene-exposed rats showed that: (1) loss of OHCs in the third row resulted in little or no hearing loss; (2) further loss of OHCs in the second row caused an OHC-loss dependent hearing loss; (3) after loss of OHCs in the third and second rows, cochlear amplification was completely gone leading to a maximal hearing loss of 40 dB. Different from noise-induced hearing loss, hearing loss caused by styrene or other industrial solvents may never be restored.

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Canadian Hearing Report 2013;8(1):39-40.

Occupational Hearing Loss in the 21st Century

By Rickie R. Davis, PhD
Rrd1@cdc.gov

About the Author
Rickie R. Davis, is captain, US Public Health Service, Hearing Loss Prevention Team, Engineering and Physical Hazards Branch, National Institute for Occupational Safety and Health, Cincinnati, OH.

The link between loud noise and hearing loss has been known for centuries[1]) Certain trades, including blacksmiths, were plagued by noise-induced hearing loss (NIHL). The industrial revolution with water and steam powered engines made NIHL even more common place.

In the United States in 1935 the Walsh-Healey Public Contracts Act empowered the Executive Branch of the government with legal authority in American industries to regulate worker safety and health when doing business with the government.[2] This act provided the legislation that enabled the first U.S. occupational safety and health regulations.

With the end of World War II thousands of U.S. veterans returned home with NIHL and tinnitus from firearms and munitions used in battle and training. Recognizing this handicap, the U.S. military services became the first organizations to deploy hearing conservation programs.

In 1969 the Walsh-Healey Act was amended to include a noise standard.[3] Employees of contractors with US government contracts had noise exposure limited to a maximum A-weighted permissible exposure level (PEL) of 90 dBA over 8 hours. The 5 dB "exchange rate" was incorporated – for every 5 dB increase in exposure level over 90 dBA, the allowable exposure time was reduced by half. A one-sentence description of a hearing conservation program included noise monitoring, hearing protection and audiometry.

In 1970, the Occupational Safety and Health Act was passed by Congress and signed by President Nixon.[4] Under the act a variety of existing standards, including those promulgated under the authority of the Walsh-Healey Act became applicable to all U.S. industries. It also provided language which established the Occupational Safety and Health Administration (OSHA) (regulatory agency) and the National Institute for Occupational Safety and Health (NIOSH) (research and training agency).

In 1972 based on scientific evidence NIOSH recommended that the permissible exposure level (PEL) be lowered to 85 dBA pending an extensive feasibility study.[5] In 1974 OSHA issued a proposed regulation adopting an action level of 85 dBA and a PEL of 90 dBA.[6] At the same time OSHA outlined requirements for a hearing conservation program including noise monitoring, audiometry and hearing protection devices.

In 1983 the Hearing Conservation Amendment replaced the one-sentence description of a hearing conservation program to include more detail including: noise measurement, engineering and administrative controls, audiometric testing, hearing protection, training, record keeping, and program evaluation.[7]

In the ensuing 30 years or so, industry for the most part has not become quieter. We have learned that wearing hearing protection effectively, comfortably, and consistently is problematic. We have also found that occupational audiometry as currently practiced does not prevent hearing loss but only documents loss. Currently noise-induced hearing loss is epidemic in the U.S. population.[8]

In 2006 the NIOSH Hearing Loss Research Program was peer reviewed by a blue ribbon panel of the National Academies of Science, Institute of Medicine.[9] Generally, the report was complementary but NIOSH was taken to

task for not having any programs in surveillance (i.e., collecting real-world information). Since that time NIOSH has undertaken efforts to utilize existing audiometric databases to enhance occupational hearing loss surveillance. Some of those efforts have recently resulted in research publications.

The U.S. National Health Interview Survey is an annual telephone survey of 103,000 randomly selected respondents representing the adult population of the United States. Tak and Calvert[8] reviewed the 1997 through 2003 responses to the question "Which statement describes your hearing (without a hearing aid)? Good, a little trouble, a lot of trouble, or deaf." In addition, respondents were asked employment information. A total of 11.4% of respondents reported hearing difficulty. Of those, 22% could be attributed to occupational exposure. The greatest number of workers reporting difficulty with hearing was in the construction industry with an estimated 400,000 workers affected. The greatest prevalence was in the railroad industry with 34.8% of respondents reporting difficulty hearing.

Utilizing data from the nationally representative U.S. National Health and Nutrition Examination Survey (NHANES) Tak, Davis and Calvert[10] analyzed the question "At your current job, are you currently exposed to loud noise?"; "By loud noise I mean noise so loud that you have to speak in a raised voice to be heard." Among those reporting loud noise exposure, a follow up question then asked "In this (current) job, do you ever wear hearing protection devices?" Exposure to loud noise was reported by 17.2% of the respondents. This represents an estimated 22 million workers in the U.S. Males had a higher prevalence of noise exposure, 26.3%, than females, 6.7%. The industries with the highest prevalence of noise exposure were mining (75%), lumber (55%), plastics (48%), utilities (46%) and maintenance (45%). Of those workers reporting noise exposure but non-use of hearing protection devices construction was the highest with an estimated 1.4 million non-users. Nationally 34% of noise exposed workers report non-use of hearing protection devices.

In 2008 an intramural NIOSH project was funded to collect occupational audiograms from regional and national industrial audiometric providers. These providers generally provide mobile audiometric services to companies that do not have in-house capabilities. A number of studies of this database have been initiated and are in various stages of being analyzed, prepared, and published. There are a number of limitations to these data. We are very excited at the prospect that multi-year audiograms are available for thousands of workers in a multitude of different industries. Analysis of these audiograms and industries may lead to new insights and better protection of workers.

Hearing protection devices (i.e., ear plugs, ear muffs and canal caps) are not simple, foolproof solutions to worker noise exposure. Workers complain about discomfort, impaired communication, missed safety signals, and inconvenience. Hearing protection that is not worn in noise is worthless. In addition, we have found that proper training is vital to obtaining useful attenuation levels.[11] Video or printed training is better than no training. The gold standard of one instructor to one worker is best[12] but seldom implemented in the occupational setting.

A responsible company would not hand a worker a respirator and wish them good luck. A respirator, as personal protective equipment, requires training in care and use. But it also requires fit testing to be sure that it protects the worker correctly. Over the past 10 years or so technology has advanced to allow for individual fit-testing of ear plugs in a non-laboratory environment. A number of companies have developed and sold commercial systems. NIOSH has developed a simple system called HPD Well-Fit which utilizes custom software on a standard personal computer with a mouse and custom earphones. It is currently being field tested.

For about 20 years the NIOSH Hearing Loss Prevention Team has been the technical advisor to the Environmental Protection Agency (EPA) for hearing protector labelling. In the United States the EPA defines and approves the Noise Reduction Rating label which is required on every box of hearing protection devices rated for occupational wear. Our group has suggested that rather than a single number, Noise Reduction Ratings be provided as a range of numbers based on population statistics. This will provide the hearing conservationist a better tool for fitting a hearing protector to a worker than the current "buy to the highest number" option.

The NIOSH Hearing Loss Prevention Team is also interested in hearing protector comfort and communication. These are two of the reasons workers rebel against hearing protector wear.[13] In addition we have been involved in studies of impulsive noise, how it affects the ear[14] and how the hearing protector functions in impulsive noise.[15]

How do we reduce occupational hearing loss going forward? An important component is to reduce or eliminate noise in the workplace. Charles Hayden a member of the NIOSH Hearing Loss Prevention Team has been developing a

program to encourage construction companies to Buy Quiet[16] when purchasing equipment. The NIOSH website already has more than 160 powered hand tools listed with their sound power levels wwwn.cdc.gov/niosh-sound-vibration/). These levels were tested by NIOSH at the University of Cincinnati. Currently, Hayden is developing a website which will allow large and small construction companies to enter their tool and equipment inventories. Utilizing a database backend, the website will determine noise levels for those tools. As tools and equipment wear out and are replaced, the companies can search the database for quieter solutions. This not only benefits the workers using the tools and equipment but also benefits the neighbours in proximity of the building site.

Beginning in 2007 Thais Morata of the NIOSH Hearing Loss Prevention Team was given support to start an award program designed to recognize companies doing an exceptional job in hearing loss prevention. This award was founded in partnership with the National Hearing Conservation Association. The awards are called the Safe-in-Sound Excellence in Hearing Loss Prevention award. Nominations are accepted via a webpage (www.safeinsound.us) during a certain timeframe. Finalist nominees are site-visited. Award winners are recognized at the annual conference of the National Hearing Conservation Association. We have found that past winners appreciate the handsome physical award but also receive even greater support from their management for their noise control and hearing conservation efforts going forward.[17] The goal of the award project is to learn from, and disseminate these real-world success strategies.

Ultimately, reducing occupational hearing loss will require technology advancements, improved surveillance, personal training, motivation, regulation and enforcement. There are environments where hearing protectors cannot safely be implemented. There are environments where noise cannot be reduced to safe levels. Using multiple strategies and collaborative efforts these and many other problems in today's noisy workplaces can be addressed and occupational hearing loss can be reduced or eliminated.

DISCLAIMER

The findings and conclusions in this report are those of the authors and do not necessarily represent the views of NIOSH. Internal accession numbers: DART 12-122; EPHB 345-05p2.

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Canadian Guidelines on Auditory Processing Disorder for Children and Adults / Lignes directrices canadiennes relatives au trouble de traitement auditif chez les enfants et les adultes

The Canadian Interorganizational Steering Group for Speech Language Pathology and Audiology (CISG) released these documents in French and English in the Fall of 2012 . http://www.canadianaudiology.ca/professional.html

The reports are an expert reflection on the state of practice, and an attempt to provide a conceptual framework from which clinicians and other health professionals can approach the problem as there is a limited amount of evidence base available.

The Canadian Academy of Audiology (CAA) served as project liaison for this work for the CISG (made up of CAA, Canadian Association of Speech-Language Pathologists and Audiologists, The Canadian Alliance of Audiology and Speech-Language pathology Regulators).

We wish to acknowledge the enormous, substantially voluntary effort of the author committee detailed below. Special thanks go to Dr. Pam Millett who chaired the committee, wrote large portions of the material, found speech- language pathology reviewers and edited the document following review by dozens of members of the CISG organizations and a copywriter. We would also like to acknowledge the contribution of Dr. Benoit Jutras who reviewed and edited the French translation for subject matter quality. Congratulations and deep gratitude go to the author committee:

Dr. Pam Millet, lead author and the chair of the authorship committee, is an associate professor at York University. She teaches in both the Graduate Program and the Teacher of the Deaf and Hard of Hearing Education Program. Her research interests include the benefit of sound-field amplification in classrooms, hearing accessibility, the development of language and phonological awareness, literacy outcomes in children with cochlear implants and technology in education. Dr. Millet has extensive experience as an educational audiologist and is a consultant with school boards in the Toronto area.

Dr. Benoît Jutras is an associate professor at the School of Speech-Language Pathology and Audiology, University of Montreal and researcher at the Research Centre, Pediatric CHU Sainte-Justine, Montreal. Dr. Jutras' research interests include auditory processing, comorbidity of auditory processing disorder with other disorders/impairments, related interventions, as well as dichotic listening and cortical evoked responses.

Greg Noel is vice-president and director of audiology of the Nova Scotia Hearing and Speech Centres, and a lecturer in the School of Human Communication Disorders at Dalhousie University. He has extensive teaching and clinical experience in the area of auditory processing disorder.

Dr. Kathy Pichora-Fuller is a full professor in the Department of Psychology and Scientist at The Human Communication Laboratory, University of Toronto. She is also an adjunct scientist at the Toronto Rehabilitation Institute and an adjunct professor in the Faculty of Graduate Studies at the University of British Columbia. Prior to joining the University of Toronto in 2002, Dr. Pichora Fuller was a faculty member at the School of Audiology and Speech Sciences and director of the Institute for Hearing Accessibility Research at the University of British Columbia. She has also worked as a clinical audiologist at Mount Sinai Hospital in Toronto. Dr. Pichora-Fuller's research interests include the effects of aging on auditory and cognitive processing as well as audiological rehabilitation for older adults.

Charlene Watson is a clinical audiologist at the Richmond Road Diagnostic & Treatment Centre, in Calgary, Alberta. She has been active in promoting awareness of auditory processing disorders in children at the Alberta Family & Community Resource Centre, and has been involved in research on brainstem response measures and auditory processing assessment tools.

Dr. Arden Nelson is an educational audiologist at ABC Children's Audiology & Hearing Services and Connect Hearing in Winnipeg, Manitoba. She has studied cortical evoked responses in individuals with known lesions and specializes in providing assessments and rehabilitation for individuals with balance deficits and auditory processing disorder.

Noise-Induced Cochlear Damage:
Changes In Cell Adhesion Contributes to Sensory Cell Death

By Bo Hua Hu, PhD
bhu@buffalo.edu

About the Author
Bo Hua Hu is with the Center for Hearing and Deafness, State University of New York at Buffalo. Buffalo, NY.

Noise-induced hearing loss is a common cause of acquired sensory hearing loss in the adult population. Functionally, acoustic overstimulation compromises hearing sensitivity and reduces the temporal and frequency resolutions. Pathologically, acoustic overexposure damages cochlear structures and causes sensory cell death. Because sensory cells in the mammalian cochlea are unable to regenerate once they die, loss of these cells results in permanent hearing loss. To prevent such loss, it is essential to understand the biological and molecular mechanisms of sensory cell degeneration, so that effective treatments can be developed.

Recent studies have shown that sensory cell degeneration is a complex molecular process, involving multiple signaling pathways. Among these pathways, molecular events that are associated with apoptotic cell death have received a great attention. Apoptosis is an active type of cell degeneration that requires a sustained energy supply during the early phase of cell death. In the cochlea, apoptosis is triggered by exposure to a high level of noise exposure and occurs primarily at the initial phases of cochlear damage.[1] Apoptosis has been linked to both intrinsic and extrinsic signaling pathways.[2] However, how these signaling pathways are activated is not clear.

Cell-cell junctions are an important element for maintenance of the structural integrity of tissues. In the cochlea, cell adhesion is organized through tight, gap, and adherens junctions, and through desmosomes and focal adhesions. These junctions play an essential role in the maintenance of structural integrity, cellular function, and signal transduction of sensory cells and supporting cells.

Cell-cell junctions are also a major target of acoustic overstimulation. Morphological analyses of cochlear structures have revealed structural defects in cell junctions,[3,4] such as the detachment of sensory cells from their anchorage and the splitting of the reticular lamina at cell-cell junctions (Figure 1). A recent observation from our laboratory has shown that even at the situation in which the general structure of the organ of Corti is preserved, an increase in the permeability to macromolecules can take place in the intracellular junctions.[5] These observations led us to hypothesize that damage to cell junctions can serve as a trigger event for the generation of acute sensory cell apoptosis.

To investigate the molecular changes associated with cell junction disruption, we recently examined the expression pattern of cell adhesion-related genes in the cochlear sensory epithelium.[6] Under the normal condition, multiple adhesion-related genes are constitutively expressed in the cochlear sensory epithelium. The highest-expressed genes include *Ctnnb1, Catna1, Thbs1*, and *Lamb2*. Interestingly, the expression levels of many genes differ between the apical and basal sections of the organ of Corti. This difference may contribute to the difference in the noise susceptibility of the sensory cells between these two sections of the organ of Corti.

Following exposure to a broadband noise at 120 dB SPL (sound pressure level) for 2 hours, we found a significant change in the expression levels of multiple adhesion genes. Importantly, these expression changes occurred in a time-dependent fashion. Immediately

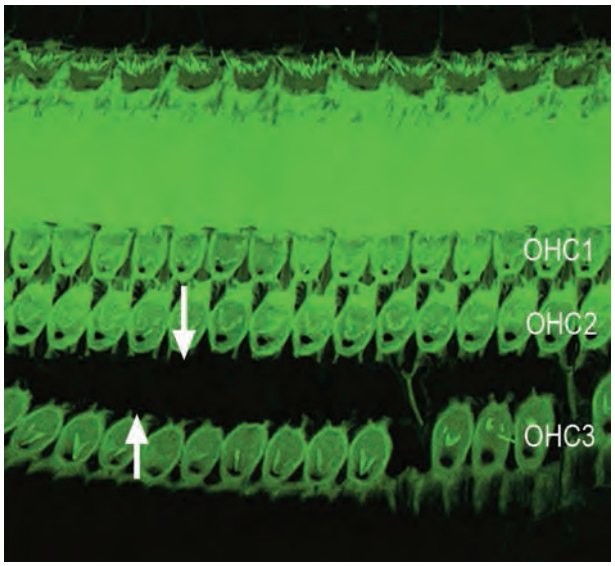

Figure 1. Separation of the reticular lamina between the second and the third row of outer hair cells *(arrows)* following exposure to an impulse noise. OHC1, OHC2 and OHC3 indicate the first, the second and the third row of outer hair cells.

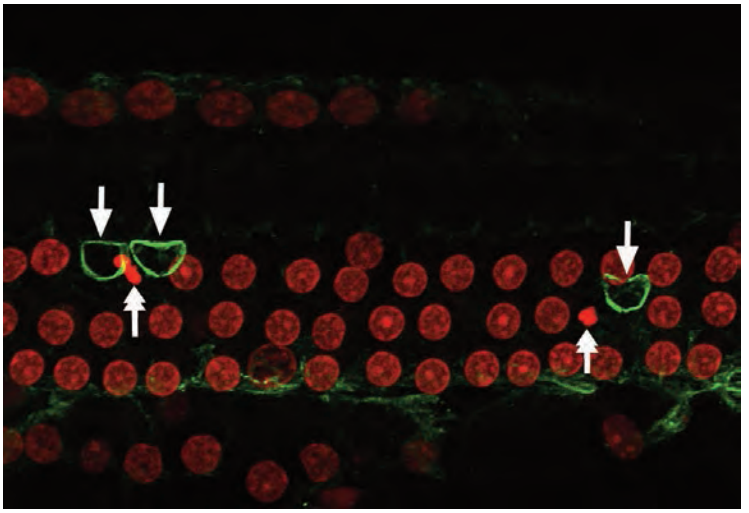

Figure 2. Increase in E-cadherin immunoreactivity *(green fluorescence, arrows)* in the hair cells exhibiting condensed nuclei *(red fluorescence, double-arrows)* in a rat cochlea following acoustic stress.

after the noise exposure, up-regulation of gene expression was the dominant change. As the time elapsed after the noise exposure, downregulation of gene expression became evident This dynamic nature of gene expression change is likely to be caused by the shift of the triggering factors for cochlear injury. The early damage is associated with direct mechanical stress that occurs during the period of acoustic overstimulation, whereas the secondary damage is associated with subsequent cellular events, which include metabolic disruption, oxidative stress, and ion imbalance.

The changes in the expression levels of adhesion genes are also related to the level of hearing loss. Our correlation analysis revealed that the expression levels of several genes, including *Sgce, Sell, Itga5, Selp,* and *Cntn1*, were related, either positively or negatively, to the level of the threshold shift of the auditory brainstem response. These genes may contribute to the individual variation in the magnitude of cochlear damage after acoustic trauma.

The changes in the expression patterns of adhesion-related genes were found to be spatially correlated with the apoptotic activity of hair cells. We observed an increased immunoreactivity of E-cadherin, an adhesion protein, in the circumferential rings of the hair cells that exhibit an apoptotic nuclear morphology, suggesting that the change in E-cadherin expression is associated with apoptotic degeneration (Figure 2). Interestingly, certain hair cells with increased E-cadherin immunoreactivity have a relatively-normal nuclear morphology, suggesting that the E-cadherin change is an early event of apoptosis.

Molecular mechanisms responsible for adhesion disruption are not clear. Matrix metalloproteinases (MMPs) are a group of endopeptidases that participate in the degradation of all components of the extracellular matrix, including the molecules responsible for cell-cell junctions. These enzymes include collagenases, gelatinases, stromelysins, matrilysins, and other proteinases, each with specialized cellular compartmentalization and substrate specificity. We therefore examined the involvement of these proteins in noise-induced cochlear damage.[7] Using a RNA-sequencing technique, we identified multiple MMP and related gene products, indicating that MMPs are constitutively expressed in the cochlea. Following exposure to a broadband noise at 120 dB SPL, the expression pattern of certain MMP genes was altered. For example, the expression of MMP7, which was undetectable under the physiological condition, became detectable in the cochlear sensory epithelium. Moreover, the expression level of intrinsic MMP inhibitors (tissue inhibitors of metalloproteinases) was altered after noise exposure. These observations suggest that MMPs are implicated in cochlear responses to acoustic overstimulation. Thus far, the biological

significance of MMPs in noise-induced adhesion disruption is not clear. In non-cochlear tissues, MMPs have been shown to regulate cell adhesion.[8,9] It is likely that, in noise-induced cochlear damage, MMPs also participate in the regulation of adhesion molecules and affect the function of cell-cell junctions.

In summary, noise-induced sensory cell damage is a complex degenerative process. Our observations support the role of cell adhesion molecules and MMPs in this process. Understanding the interplay between these molecules can shed new light on the biological mechanisms responsible for sensory cell degeneration, which in turn, will assist our efforts to develop novel therapeutic targets for the prevention of noise-induced hearing loss.

DISCLAIMER

The research was supported by NIDCD1R01 DC010154-01.

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Early Age Conductive Hearing Loss Impairs Sound Tolerance and Auditory Processing

By Wei Sun, PhD, Yuguang Niu, Senthilvelan Manohar, Anand Kumaraguru and Brian Allman, PhD

About the Author
Wei Sun (pictured) is with the Center for Hearing & Deafness, Department of Communicative Disorders and Sciences, the State University of New York at Buffalo, Buffalo, NY.

Otitis media is a common illness diagnosed in children. Chronic otitis media at early age, especially in infants, can induce recurrent conductive hearing loss which may adversely affect language acquisition, learning and social interactions.[1] Recent clinical studies have found that children who reported hearing loss often experienced hyperacusis and tinnitus (~37%), suggesting that mild hearing loss during early age might be an associated risk factor for hyperacusis and tinnitus in children.[2] Given that sensory input from the cochlea is crucial for the functional development of the central auditory system,[3,4] we hypothesized that early age hearing loss would affect the normal development of the central auditory system and disrupt sound perception and tolerance. In the present study, we tested this hypothesis using Sprague Dawley rats as an animal model.

During light anesthesia, we surgically perforated the tympanic membranes of rats at postnatal 16 days (note that a rat's ear canals do not open until postnatal 12 days). After surgery, the rats developed a temporary conductive hearing loss for about 2–3 weeks until the trauma of the ear drum had healed. Surprisingly, several weeks after the tympanic membrane perforation, more than 85% of the rats (n = 23) developed a susceptibility to audiogenic seizure, which was characterized by wild running followed by erratic leaping and clonic convulsion during exposure to loud sound (120 dB SPL white noise, < 1 minute. The susceptibility of audiogenic seizure lasted to adulthood; long after hearing loss had recovered. Furthermore, compared to age-matched controls, rats with early age tympanic membrane damage also showed enhanced acoustic startle responses. These results suggest that early age hearing loss can increase sound sensitivity and reduce sound tolerance. By varying the timing of the tympanic membrane surgery, we found that audiogenic seizures could also be induced when tympanic membrane perforation occurred at postnatal 30 days, but not at postnatal 45 days; findings which suggest that hearing loss in early life results in a greater impairment of sound perception and tolerance than hearing loss in adulthood.

We further evaluated the changes in auditory processing induced by early age conductive hearing loss by comparing, within a given animal, the auditory brainstem responses (ABRs) evoked during sound presentation to the tympanic membrane damaged ear versus undamaged (control) ear. Although the ABR threshold showed no significant difference between the control ears versus the tympanic membrane damaged ears two months after the surgery, the ABR interwave latencies of waves of I to V in the tympanic membrane damaged ears were significantly shorter than the ears without tympanic membrane damage. In addition, using immunostaining for c-Fos, an immediate gene whose activation can be used to indicate neural activity, we found a stronger staining in the inferior colliculus in the tympanic membrane damaged rats after exposure to loud sound compared to the rats without tympanic membrane damage. These results suggest that early age hearing loss may cause hyperexcitability and increase the neural signal conduction in the central auditory system.

In a follow-up series of experiments, we

found that the incidence of audiogenic seizure was suppressed by treatment of vigabatrin, an antiepileptic drug that inhibits the catabolism of the gamma-aminobutyric acid (GABA), the major inhibitory neurotransmitter in the brain. Acute injections (250 mg/kg) or oral intake (60 mg/kg/day for 7 days) temporarily abolished audiogenic seizure in rats with early age tympanic membrane perforation. Vigabatrin treatment also caused prolonged ABR latency and reduced the peak amplitude of the ABR responses. Collectively, these results suggest that early age hearing loss may reduce GABAergic inhibition, leading to hyperexcitability and increased susceptibility to audiogenic seizure. Our results support our hypothesis that early age hearing loss affects the normal development of the central auditory system and disrupts sound perception and tolerance. It is possible that our findings may be related to hyperacusis seen in children with recurrent otitis media.

ACKNOWLEDGEMENT

The project was supported by Action of Hearing Loss foundation.

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