

Canadian Hearing Report

Revue canadienne d'audition

CAA Canadian Academy of Audiology
Heard. Understood.
ACA Académie canadienne d'audiologie
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Vol. 8 No. 4
2013

**Development of Pitch
Perception and the
Processing of Simultaneous
Sounds in Infancy**

**Hearing Preservation
in Cochlear Implant Users**

**Don't Upset The Balance:
Why Children Need To
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The word war rages on, is it sensori-neural?

Sensory neural? Sensoryneural? Or sensory-neural?

This may seem like a trivial issue like word recognition score versus speech discrimination score, but the ramifications are more far reaching. Is an issue of hearing related to the cochlea (sensory) or is it neural, or perhaps both? Does a pathology in one area affect the function in another?

Certain tests can demonstrate problems in one area that other tests cannot – we have known this since long before advanced imaging technology has been available. In 1962, Jack Katz came out with the SSW test that demonstrated clearly areas of auditory dysfunction that could not be shown with “routine” audiometry. With the advent of new audiology tests, new imaging techniques, and new paradigms, we are better able now to distinguish between peripheral pathology (cochlear) and central pathology (neural and cortical).

Terms like sensori-neural (or however we end up writing it) demonstrates a certain level of ignorance. We don’t really know whether its sensory or whether its neural,

or both, so like the proverbial waste paper basket, we just dump it all in and use a difficult to spell longer word.

This issue of the *Canadian Hearing Report* begins with a letter to the editor about this very issue. It was written by Fred Martin and John Greer Clark. If the names sound familiar, and they should, these are the two authors behind a very successful audiology text book. In writing their book, Doctors Martin and Clark had to be consistent with the usage of this term (these terms?). Personally I am in the midst of being an associate editor for Jack Katz’s seventh issue of his *Handbook of Clinical Audiology*, and like Doctors Martin and Clark, we needed to settle on a consistent and up-to-date term.

It is no coincidence that we had invited Drs. Martin and Clark to write the Letter to the Editor. This is an issue that has been guest edited by Dr. Lendra Friesen whom many will recognize for her long-standing contributions to Spotlight on Science in previous issues of the *Canadian Hearing Report* (now written by Sheila Moody and Steve Aiken). Lendra has put together a wonderful overview of some of the audiology areas that span the sensory and the neural auditory domains.

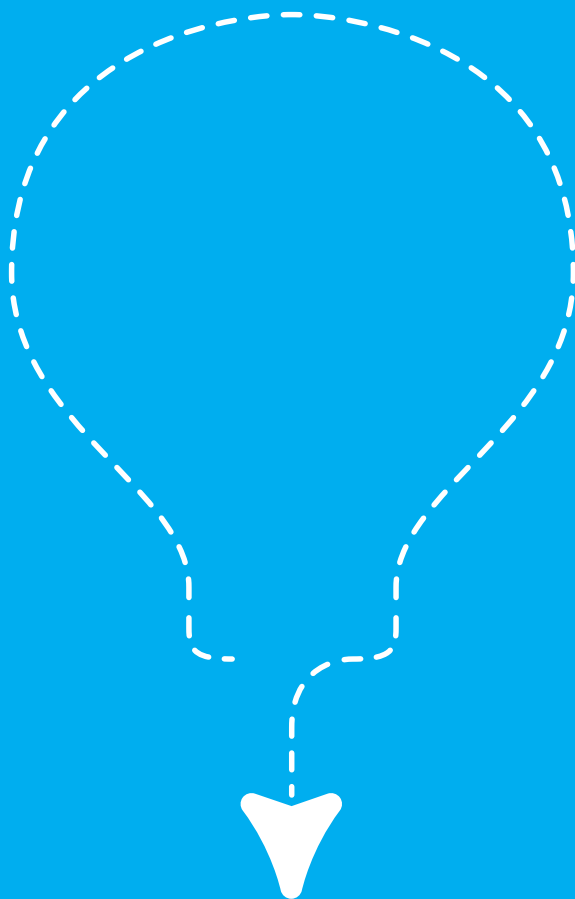
In this issue we have articles by Steve Aiken and Philippe Fournier called “Tinnitus: The Dark Side of Neuro-

plasticity.” Tinnitus is something that is frequently noticed after a peripheral insult but is central in origin, and central in treatment. And, there is no better way to appreciate the role of the central auditory pathways than to examine the topic of binaural hearing which Karen Gordon and Blake Papsin write about “Why Children Need to Hear from Both Ears.” Bernhard Ross writes about “The Auditory Evoked P2 Response Indicates Effects of Aging on Central Auditory Processing,” and Laurel Trainor tackles the topic of the “Development of Pitch Perception and the Processing of Simultaneous Sounds in Infancy.”

I would like to thank Lendra for her past contributions to the *Canadian Hearing Report* as well as putting this issue together as the guest editor. It not only makes my life a bit easier, but more importantly this issue will serve to contribute to the richness of all of our readers’ central academic neurons (or is it peripheral?).

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La guerre des mots est déclenchée, est-ce neuro-sensoriel? Neurosensoriel? Neuro sensoriel? Ce qui semble être un enjeu insignifiant tel la note de reconnaissance des mots versus celle de la discrimination des mots, mais les ramifications sont beaucoup plus profondes. Est-ce un enjeu d'ouïe lié à la cochlée (sensoriel) ou est-ce neuronal, ou peut-être les deux? Est-ce que la pathologie d'un domaine affecte la fonction de l'autre?

Certain tests peuvent montrer des problèmes dans un domaine que d'autres tests ne peuvent pas – Nous le savions bien avant que la technologie avancée de l'imagerie ne soit disponible. En 1962, Jack Katz a inventé le test SSW qui a clairement montré des domaines de dysfonctionnements auditifs qui ne pouvaient être montrés avec l'audiométrie de "routine". Avec l'avènement de nouveaux tests audiolinguistiques, de nouvelles techniques d'imagerie, et nouveaux paradigmes, nous pouvons maintenant mieux distinguer la pathologie périphérique (cochléaire) de la pathologie centrale (neuronale et corticale).

Des termes tel neurosensoriel (peu importe comment on finira par l'écrire) démontre une certaine ignorance. Nous ne savons pas réellement si c'est sensoriel ou neuronal, ou les deux, alors on tasse le tout et on utilise la difficulté pour écrire

l'orthographe d'un mot plus long.

Ce numéro de la *Revue Canadienne d'audition* débute avec une lettre au rédacteur au sujet de cet enjeu même. Lettre rédigée par Fred Martin et John Greer Clark. Si ces noms vous semblent familiers, et ils devraient l'être, ce sont les auteurs d'un manuel d'étude en audiologie à très grand succès. Les docteurs Martin et Clark se devaient d'être consistants avec l'utilisation de ce terme (ces termes?) tout le long de leur travail sur leur manuel. Personnellement, je suis en plein processus d'être rédacteur associé du septième numéro du *Handbook of Clinical Audiology* de Jack Katz, et comme les docteurs Martin et Clark, il fallait qu'on se mette d'accord sur un terme consistant et à jour.

Ce n'est pas une coïncidence que nous ayons invité les docteurs Martin et Clark à écrire une lettre au rédacteur. La rédactrice invitée de ce numéro est Dr. Lendra Friesen que plusieurs vont reconnaître pour ses contributions de longue date à la chronique Spotlight on Science dans des numéros précédents de la *Revue Canadienne d'audition* (maintenant rédigée par Sheila Moody et Steve Aiken). Lendra a mis ensemble un aperçu superbe de certains domaines de l'audiologie qui s'étend sur les domaines auditifs sensoriel et neuronal.

Dans ce numéro, nous avons des articles par Steven Aiken et Philippe Fournier intitulé "Tinnitus: La face obscure de la

plasticité synaptique." Le tinnitus est fréquemment constaté après un accident périphérique mais son origine est centrale, et son traitement est central. Et pour apprécier le rôle des chaînes auditives centrales, rien de mieux que de se pencher sur le sujet de l'ouïe binaurale que Karen Gordon et Blake Papsin examine dans "Pourquoi les enfants ont-ils besoin d'utiliser leurs deux oreilles pour entendre." Bernhard Ross rédige son papier "Le potentiel évoqué auditif P2 indique les effets du vieillissement sur le processus auditif central," et Laurel Trainor s'attaque au sujet du "Développement de la perception des tons et traitement des sons simultanés chez les nourrissons."

Je voudrai remercier Lendra pour ses contributions passées à la *Revue Canadienne d'audition* et aussi pour préparer ce numéro en tant que rédactrice invitée. Non seulement ça facilite un peu ma vie, mais plus important, ce numéro va contribuer à la richesse des neurones académiques centraux de nos lecteurs (ou sont-ils périphériques?).


Marshall Chasin, AuD, M.Sc., Aud(C), Reg. CASLPO

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This issue of the *Canadian Hearing Report* is about the role of the brain and neural plasticity in the field of hearing. We are in an exciting time

where we have the technology to examine changes in the brain that occur with many different populations including those in the stages of development and aging, those undergoing auditory training or auditory deprivation, those receiving different types of stimulation (amplification or electrical as is used with a cochlear implant, or CI), or experiencing various disease processes, as well as many others.

In this day and age we can examine different levels of the brain, beginning with the auditory nerve and moving up to higher centers in the auditory cortex. We can use technology such as auditory

evoked potentials (AEPs), or magnet-encephalography (MEG), to name only a few of the available techniques to measure neural activity.

We are featuring several groups of researchers that are examining different processes in the brain using a variety of techniques and different technology. Although I will not define all the terms in this particular article, they will be defined in each individual paper. Karen Gordon and Blake Papsin investigate the importance of binaural hearing in young children implanted with CIs at The Hospital for Sick Children, using the auditory brainstem response (ABR) and cortical responses. Laurel Trainor, from McMaster University, explores the development of pitch processing in infants using a preconscious discrimination cortical response, the mismatch negativity (MMN). Bernhard Ross, from the Rotman Research Center at Baycrest, examines the effects of aging in elderly adults,

specifically examining the P2 waveform of the cortical P1-N1-P2 response, recorded using magnet-encephalography (MEG) in different experiments. Steve Aiken, from Dalhousie University and Philippe Fournier from the University of Montreal, discuss the relationship between neuroplasticity and tinnitus, a world that is virtually unknown. And finally, our lab at the Sunnybrook Health Sciences Centre, examines hearing preservation in CI users and the underlying neural effects, examining eCAP responses from the level of the auditory nerve, as well as cortical responses.

Our goal is to provide the reader with new insights into what types of auditory neural research are being investigated and the importance of remembering the brain's role in hearing.

Lendra Friesen, MSc, PhD, Guest Editor
Canadian Hearing Report 2013;8(4):9.

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Ce numéro de la *Revue Canadienne d'audition* est au sujet du rôle du cerveau et de la plasticité synaptique dans le domaine de l'ouïe. Nous passons par des temps stimulants, nous avons les techn-

ologies pour examiner les changements au niveau du cerveau qui surviennent pour beaucoup de populations différentes, populations en cours de développement et vieillissement, celles en cours d'éducation auditive ou en privation auditive, celles recevant différents types de stimulation (amplification ou électrique utilisées avec les implants cochléaires, ou CI) ou populations touchées par des maladies variées, et aussi tant d'autres.

Ces temps-ci, on peut examiner les différents niveaux du cerveau, commençant avec le nerf auditif et avançant le long de centres supérieurs dans le cortex auditif. Nous pouvons utiliser des technologies telles les

potentiels auditifs évoqués, ou l'encéphalographie magnétique, pour citer quelques-unes des techniques disponibles pour mesurer l'activité neuronale.

Nous présentons plusieurs groupes de chercheurs qui examinent différents processus du cerveau utilisant une variété de techniques et de technologies différentes. Même si je ne vais pas définir tous les termes dans cet article en particulier, ce sera fait dans chaque article individuel. Karen Gordon et Blake Papsin enquêtent sur l'importance de l'audition binaurale chez des jeunes enfants auxquels on a implanté des implants cochléaires à l'hôpital pour enfants malades, en utilisant la réponse auditive évoquée du tronc cérébral et les réponses corticales. Laurel Trainor, de McMaster University, explore le développement du traitement des tons chez les nourrissons en utilisant une réponse corticale de discrimination préconsciente, la négativité discordante. Bernhard Ross, du centre de recherche Rotman à Baycrest, examine les effets du vieillissement sur les personnes

âgées, spécifiquement l'onde P2 de la réponse corticale P1-N1-P2, enregistrée par encéphalographie magnétique dans différentes expérimentations. Steve Aiken, de Dalhousie University, touche à la relation entre la plasticité synaptique et le tinnitus, un monde qui est virtuellement inconnu. Et finalement, notre laboratoire au centre des sciences de la santé de Synnybrook, examine la préservation de l'ouïe chez les utilisateurs d'implants cochléaires et les effets nerveux sous-jacents, en examinant autant les réponses eCAP au niveau du nerf auditif, que les réponses corticales.

Notre objectif est de fournir au lecteur de nouvelles perspectives au sujet des types de recherche en nerf auditif et l'importance de se rappeler le rôle du cerveau dans l'ouïe.

*Lendra Freisen, Docteur
Réactrice invitée*

Canadian Hearing Report 2013;8(4):10.

Letter to the Editor

ADVOCATING FOR A NEW SPELLING: "SENSORY/NEURAL"

In past years the first terms encountered in university classes and in the literature to describe hearing losses that result from lesions of the cochlea or auditory nerve were "nerve type hearing loss" and "perceptive hearing loss." The former, unfortunately, is still in common parlance even though the majority of losses do not involve the auditory nerve at all. The latter is wholly inaccurate for these lesions are not expected to involve perception.

In the 1960s the term "sensorineural" was coined which was greatly welcomed. This term was designed to imply that the hearing loss was caused by a sensory (inner ear) lesion, a neural (auditory nerve) lesion, or both. The problem with this one-word term is that it does not suggest the separation between these two anatomical sites. Of course, in the 1960s, the profession had only relatively crude site-of-lesion tests for attempted separation of the two subcategories of sensory/neural hearing loss. The ability to accurately separate sensory from neural lesions has increased dramatically.

Since introduction of the term "sensorineural," other spellings have

emerged such as "sensory neural" and "sensory-neural." Beginning with the 11th edition of our book, *Introduction to Audiology*, and continuing to the 12th, which is now in press, we began the use of the spelling "sensory/neural." Our reasoning is based on the fact that the dictionary describes the dash (-) as a "horizontal stroke in writing or printing to mark a pause or break in sense, or to represent omitted letters or words," and the slash as "an oblique stroke (/) in print or writing used between alternatives (e.g., and/or)." This spelling meets the criteria for accurate terminology in audiology.

It is our hope that the profession of audiology will rally around use of a spelling that is a more accurate reflection of the lesion when a clear differentiation between sensory and neural cannot be made. When test results clearly differentiate between these two lesion sites, audiologists can facilitate patient management by distinctly stating that the loss is cochlear (sensory) or neural in nature.

We have long recognized that our profession, despite the zeal with which practitioners accept new scientific procedures, is slow to change in the adoption of new terminology. Consider the continued use of "speech

discrimination score" despite the obviously improved and more accurate "speech recognition score." Another example is "speech reception threshold" rather than "speech recognition threshold" for the very commonly practiced SRT.

We hope that this communication may serve to raise consciousness about the spelling "sensory/neural" as a replacement for previous spellings and that it may serve as an impetus for adoption of its use. We believe that this more-accurate orthography clearly states that a hearing-loss producing lesion may be found in the cochlea, the auditory nerve, or both. It is to our betterment as a profession that a commonality of accurate terms and spellings is used in audiological communication.

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Canadian Hearing Report 2013;8(4):11.



By Calvin Staples, MSc
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I hope this finds you all enjoying your summer. I opted to submit blogs that focus on children and hearing. My wife and I recently welcomed our third child into our family. We have had the great experience of watching our older children care and learn about the development of a newborn. Once we checked off major health concerns such as breathing, 10 fingers, 10 toes, etc, I was quick to be sure he was hearing. Our two older children easily helped to establish the startle. I thought the submissions below would provide some insight into the wonderful world of hearing for children. I know as an audiologist, helping a child hear is at the top of the list in terms of professional reward. I hope you enjoy the blogs.

MUSIC TRAINING HEARING

By Jane Madell

The world is a very noisy place and much of what children learn, they have to learn listening in noise. Hearing in noise requires the ability to hear with both ears. Binaural hearing enables us to focus on the speech signal and ignore the noise. Language is a left-brain activity and the majority of the input to the right ear goes to the left brain. Our primary focus in

working with children with hearing loss (and also children without hearing loss) is to develop language skills. Therefore, the right ear has been considered the more important ear. While the right ear certainly is critical, To hear in noise, we also have to build skills in the left ear, which sends the majority of the information going to the right brain.

MUSIC AND THE BRAIN

Nina Kraus, PhD, director of the Auditory Neuroscience Laboratory at Northwestern University, has studied the effect of musical training on the brain. She has demonstrated that musicians excel in vocabulary, reading, non-verbal reasoning, perception of speech in background noise, auditory memory and attention. She has concluded that musical training causes the brain to undergo neurological changes. For example, string players demonstrate a reorganization of the motor cortex related to the left hand, which performs extensive intricate fingering. There are many examples of brain changes in musicians explicitly involving auditory centers.

MUSIC TRAINING AND CHILDREN

Music training is associated with increased vocabulary, reading and phonologic processing, attention and reasoning skills in children. Kraus reports on the tie in between musicianship and literacy. Learning to read is closely related to phonics and being able to decipher the sounds of language. So musical practice can hone the auditory system providing a channel towards literacy. Her research also demonstrates the relationship between music and hearing in noise.

MUSIC AND CHILDREN WITH LANGUAGE LEARNING DISORDERS

Some of Kraus's work supports music as

a therapy for children with a language learning disorder and difficulties with speech in noise. Her work suggests that music can help children with auditory processing disorders improving listening in noise.

WHAT DOES THIS MEAN FOR CHILDREN WITH HEARING LOSS?

Well, we do not absolutely know. But we do know that, in general, music can help develop the auditory brain. I have always encouraged children with hearing loss to study music because, as a rule, I think music is a good thing. In addition, we know that music is a right-brain activity and improving right-brain functioning improves the ability to hear in noisy situations. Since noise is a difficult problem for children with hearing loss, music training seems like a good idea. If, in addition, it has a significant effect on literacy, it is a win-win situation.

http://hearinghealthmatters.org/hearingandkids/2013/music-training-and-hearing/#utm_source=rss&utm_medium=rss&utm_campaign=music-training-and-hearing

NEW PHONE APP LETS PARENTS TEST THEIR CHILDREN FOR HEARING LOSS AT HOME

By David Kirkwood

For 69 pence (about \$1.15), parents in Great Britain can obtain a phone app that can be used with a smartphone or an iPad to help them assess their children's hearing without taking them to an audiology clinic.

The Early Ears app was developed by British scientists at Aston University in Birmingham, who say that it can offer parents "immediate reassurance" for worries about hearing loss. The app

presents a series of eight images and the children must correctly touch the image that responds to the app's audio instructions—which are broadcast at different volumes.

Robert Morse, PhD, from Aston University's Health Care Clinics, said, "Our Early Ears app provides parents with a simple, but reliable, resource to test their children's hearing. For three decades the McCormick toy test has been used by audiologists to identify common causes of hearing loss in kids. This app brings that test into the home, offering parents immediate reassurance or recommendations to seek expert medical advice if they suspect their child may have a hearing problem."

<http://hearinghealthmatters.org/hearingnewswatch/2013/new-phone-app-lets-parents-test-their-children-for-hearing-loss-at-home/>

STUDY FINDS OBESITY IN ADOLESCENTS IS LINKED TO HEIGHTENED RATE OF HEARING LOSS

By David Kirkwood

Anil K. Lalwani, MD, lead author of a new study showing an association between hearing loss and obesity among adolescents, recommends that obese adolescents "receive regular hearing screening so they can be treated appropriately to avoid cognitive and behavioral issues."

The study, published June 17 in *The Laryngoscope*, found that obesity in adolescents is associated with sensorineural hearing loss across all frequencies. The highest rates were for low-frequency hearing loss – 15.2% among obese adolescents compared with

7.9% in non-obese adolescents.

Lalwani, who is vice-chair for research in the Department of Otolaryngology/Head & Neck Surgery at Columbia University Medical Center (CUMC), told the CUMC news office, "This is the first paper to show that obesity is associated with hearing loss in adolescents."

Lalwani, who is also an otolaryngologist at New York-Presbyterian Hospital/Columbia University Medical Center, noted that the results of the study "have several important public health implications." Because it has been found that 80% of adolescents with hearing loss are unaware of their hearing difficulty, he said it is important that those whose obesity puts them in a high-risk category be routinely screened for hearing loss. About 17% of children in the U.S. are obese.

"Furthermore," Lalwani said, "hearing loss should be added to the growing list of the negative health consequences of obesity that affect both children and adults – adding to the impetus to reduce obesity among people of all ages."

Lalwani called for additional research on the adverse consequences of this early hearing loss on social development, academic performance, and behavioral and cognitive function. He also said that more research would be needed to determine the mechanisms involved in hearing loss among obese adolescents. He speculated that obesity-induced inflammation may contribute to hearing loss. Low plasma levels of adiponectin, an anti-inflammatory protein, have been found in obese children, and low levels in obese adults have been associated with high-frequency hearing loss.

The study, whose other authors are

Karin Katz, MD; Ying-Hua Liu, MD, PhD; Sarah Kim, BA; and Michael Weitzman, MD, all from the New York University Langone Medical Center, analyzed data from nearly 1500 adolescents in the National Health and Nutrition Examination Survey, conducted 2005-2006 by the National Center for Health Statistics of the Centers for Disease Control and Prevention.

<http://hearinghealthmatters.org/hearingnewswatch/2013/study-finds-obesity-in-adolescents-is-linked-to-heightened-rate-of-hearing-loss/>

HOW MANY HOURS A DAY DOES A CHILD NEED TO HEAR?

By Jane Madell

We know, for sure, that kids need to hear all day long in order to learn language, and to be ready to read. Typical hearing kids hear 24 hours a day. Children with hearing loss hear only when they have their technology on.

Here is what else we know:

- Typical children hear 46 million words by age 4 years
- Children need 20,000 hours of listening to learn to read. (That would mean listening for 12 hour days for 1,667 days)
- Children with hearing loss require three times the exposure to learn new words and concepts.

SO, WHAT DOES THIS MEAN FOR A CHILD WITH HEARING LOSS?

If a child wears hearing aids 4 hours a day, it will take 6 years for the child to hear what a typical child who does not need hearing aids hears in one year. That means that the child with hearing loss

will have significantly less auditory input, resulting in less language exposure, which will result in poorer language and poorer reading. What can we do to change this outcome? We need to help families to keep hearing aids and other technology on their child's head. For children with hearing loss, time is of the essence. There is no turning back. We cannot make up for lost listening time when the child is older. When children do not receive sufficient auditory exposure, the auditory portion of the brain will shrink and the visual cortex will expand to take over the area usually associated with audition.

WHAT CAN WE DO TO HELP FAMILIES KEEP TECHNOLOGY ON A CHILD'S HEAD?

1. We need to help families understand how important it is to

use technology full time.

2. We need to help families find the appropriate retention devices that will keep the hearing aids on the child's head full time. These may include devices such as Huggies, Ear Gear, Critter Clips, and toupee tape.
3. We need to teach families to make sure that their children's technology is working each and every day. Parents can never assume. They need to check their kids' hearing aids every day.
4. We need to test to be sure that a child is receiving enough benefit from the hearing aids, and can hear both normal and soft speech.

IT IS EVERYONE'S JOB

Making sure technology is working and on is everyone's responsibility. Audio-

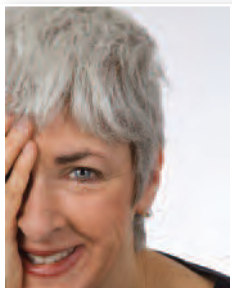
logists need to be sure parents understand why technology is important and how to check technology. Parents need to check technology daily and take action if it is not working. Children need to be taught to pay attention to whether or not technology is working and report problems. Teachers and therapists need to pay attention to whether the child is responding well with technology and, if not, get help. No one person can do this job alone, but if we all work together, we can be certain that we are providing good auditory access to kids with hearing loss.

<http://hearinghealthmatters.org/hearingandkids/2013/how-many-hours-a-day-does-a-child-need-to-hear/>

Canadian Hearing Report 2012;8(3):12-14.

What Hearing Research Tells Me about Me

By Gael Hannan
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Almost every day I read about a new hearing loss study, research or statistics. Most of them sound exciting and others are single-eyebrow-raising, such as surveys announcing how many

of us actually have hearing loss. The stats for the overall population seem to fluctuate a great deal, from the often-quoted but urban myth of 10% to between 16–25%.

Why the big difference – are we perhaps a difficult group to count? Is there perhaps a shortage of *counters*? And, why, when they *do* count, do they use such narrow population criteria, such as Chinese Factory Workers or Swedish Teachers or Seniors in Rural Kentucky, or Large Fishing Families in Newfoundland Outposts, or Teenagers Whose Ears Ring After a Concert? And, to be honest, we don't always put up our hand to be counted. Even when asked on a census, we *may* not tell the truth (“Hmm, that's a ‘no’. I'm not harda-hearing, just don't hear the wife too well anymore, nothing important.”)

Some of the research project titles are beyond me, requiring a triple-PhD to understand what the researcher is saying. Take, for example, this study posted at The Journal@HHTM: An Investigation of the Relationship of ABR Wave V to Na-Pa of the MLR.” Now, I'm sure this is a fine study that will quicken the heartbeats of hearing researchers around the world. But, although I understand

that these medical studies have the potential to someday improve my hearing, I'll wait for the results to be announced in plain English.

Mind you, there has been some interesting, *understandable* stuff coming out recently.

Study #1: People with hearing loss who don't use hearing aids are more tired at night

Surveys conducted of people with significant hearing loss in Italy, UK, France, Germany, Switzerland, Norway, and Japan, showed that 50% of non-hearing aid-users said they often felt mentally exhausted in the evenings, as compared to 30% of those who use hearing aids.

So, it's clear that if you need a hearing aid *and* you use one, there's a good chance you'll be mentally fresh as a daisy come dinner time. But, unfortunately, I fall in the 30% of hearing aid users – I often feel pooped at night. Is that because I'm Canadian? Would I do better hanging out in Germany? Or am I pooped because of something else – age, lack of sleep? Still, this is good stuff to know. My German hearing loss friend will be pleased to learn that after a long day of reading people's lips, chances are she'll still be raring to go!

Study #2: Stress makes exhausted women over-sensitive to sounds

According to a Swedish study, women suffering from stress-related exhaustion exhibit hypersensitivity to sounds when exposed to stress. But this is where I start

to get confused. I need more information. Would this sound sensitivity be *all day* or just in the *evenings*? Are these woman wearing anything in their ears?

Study #3: Red wine may protect against noise-induced hearing loss

A plant compound found in red grapes and red wine, may guard against hearing loss and cognitive decline, according to a study published in the journal *Otolaryngology-Head and Neck Surgery*. Fabulous! This is something I can understand. But I'm hoping that rosé, my personal favourite and which is kind of red-kind of white, may also offer *some* protection. If not, I'm switching to shiraz, merlot or some other horsey-sounding wine.

Study #4: High blood pressure can lead to hearing loss

A Mumbai study showed that people suffering from high blood pressure could also suffer from hearing loss as a result of their medical condition. However, if the hypertension is controlled with the right medication, additional loss of hearing can be prevented.

But guess what? According to *another* study reported by the American Heart Association, non-alcoholic *red wine* can reduce blood pressure, which in turn can help prevent hearing loss. So, if you take Studies # 3 and 4 together, it's clear that we should drink red wine *with* alcohol and *without*. (I'd suggest the fake stuff for breakfast and the real stuff for dinner – which may have the added benefit of helping stress-and-loud-noise issue of Study #2.)

Study #5: The chance of dementia is increased in people with hearing loss.

A 2011 study from the Johns Hopkins School of Medicine found that hearing loss may increase one's chances of developing dementia. In the study, Dr. Frank Lin reported that for every 10 decibels of hearing that is lost, the extra likelihood of development of dementia jumped up by 20%.

This is not good news for me. I have a 75 dB loss, which means I have a 140% chance of developing dementia. (And if my math is wrong on this – please, let me know.) But here's the thing – do I get brownie points for being a hearing aid user and having, as of this minute,

converted to red wine?

Study #6: Stanford University has introduced the “Stanford Initiative to Cure Hearing Loss”

This is a positive research initiative that I'm going to take at face value. They're not saying *maybe* or that there's a “27% chance of.” The title clearly states that these people *are* going to find a cure for hearing loss. And that's why, for now, I'm not reading any further than the title.

To sum up, let's take a look at what we've got here. Let's say we have a woman, late-50ish, severe-to-profound hearing loss who wears her hearing aids all the time, keeps the grey matter bubbling with

activity, drinks fake wine for brekkie and a smooth cab-sauv for din-dins, checks her blood pressure regularly, and works hard to reduce stress in her life.

By adopting all these strategies, would her hearing loss go into reverse? Or would we simply have a healthy, physically fit, and mentally agile woman who knows how to live successfully with hearing loss?

The latter? Oh damn.

Be sure to visit Gael's blog, “The Better Hearing Consumer” at:

<http://hearinghealthmatters.org/>.

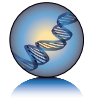
Canadian Hearing Report 2012;8(4):15-16.



John C. Booth, PhD
SEPTEMBER 27, 1942 – JULY 16, 2013

John C. Booth, Professor Emeritus at Western University's School of Communication Sciences and Disorders passed peacefully surrounded by his family on July 16, 2013. John was among the original faculty members of Western's audiology program. As a hearing scientist with expertise in electrophysiology and hearing instrumentation, John was a highly regarded teacher and mentor to Western's audiology students for three decades, supervising many master's students' introduction to the research process and to evidence based practice. He is remembered respectfully and fondly by former students for his knowledge, guidance and support, as well as for a teasing sense of humor signaled by the twinkle in his eye. He was a member of the Canadian Standards Association – Subcommittee on Hearing, the American Auditory Society and the Acoustical Society of America and served on the diagnostic hearing test task force, outcome assessment subgroup for the Ontario College of Physicians and Surgeons. John's administrative service to the School of Communication Sciences and Disorders and to Western University spanned over a decade. He was assistant chair in 1990, then acting chair in 1992. A year later, John was appointed chair of the School of Communication Sciences and Disorders and served for four years. He is remembered best among his colleagues for his dedication to the School, fairness, generosity, mentorship and good humour.

Written by colleagues Elizabeth Skarakis-Doyle and Susan Stanton



Dealing with Otitis Media: New Guidelines for Tympanostomy Tubes

By Steve Aiken, PhD
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About the Author

Steve Aiken, PhD, is President of the Canadian Academy of Audiology and associate professor, audiology, surgery, and psychology and neuroscience at Dalhousie University.

There have long been concerns about the impact of persistent or recurrent otitis media with effusion (OME) on the development of speech, language and auditory processing.¹⁻³ Fluid in the middle ear cavity can reduce the efficiency of the ossicular chain as a conductor of sound energy to the cochlea, thereby diminishing sensitivity to important acoustic information, perhaps during critical periods of auditory development and language acquisition.⁴ Studies have shown deficits in auditory processing in children after the resolution of OME, such as poorer binaural unmasking (i.e., a smaller binaural masking level difference)^{5,6} comodulation masking release⁷ and speech perception with temporally complex maskers.⁸ Similarly, there are reports of speech and language deficits in children with histories of OME.⁹⁻¹² These and similar findings provide a theoretical justification for the use of tympanostomy tubes.

However, most studies showing significant effects of OME have been conducted on young children within a

few years following the episode(s). Evidence for long-term effects of OME on auditory processing, speech and language are more difficult to find apart from smaller studies,¹⁰ and meta-analyses of the literature have concluded that long-term effects of OME are either small or insignificant.¹ Moreover, randomized control trials have not shown a significant impact of tympanostomy tubes on speech, language and hearing outcomes,^{13,14} making the case for tubes much less compelling.

Interestingly, these equivocal results may be because many studies have failed to determine whether the children with OME had significant hearing loss.^{1,15} In a study of young children (1–3 years old),¹⁶ children with frequent bilateral OME had average thresholds that were only 6–7 dB worse than the children without OME, with mean thresholds that ranged from 18 to 20 dB HL. The proportion with significant hearing loss (i.e., thresholds poorer than the 95th percentile from the non-OME distribution, between 15.5 and 21.5 dB

HL) ranged from 40 to 60%. In other words, many of the children with bilateral OME had normal hearing thresholds and most showed only small threshold shifts. Other studies have found similar results.¹⁷ When we focus on studies that measured the impact of OME coupled with significant conductive hearing loss, the evidence for communicative impact is much more compelling.¹⁵ For example, Zumach and colleagues¹⁸ found speech perception to be impaired in 7 year old children with a history of OME and significant conductive hearing loss. In fact, OME with threshold elevation has even been found to be associated with an increased risk of tinnitus in later life.¹⁹

Clearly, the decision of whether or not to treat OME with tympanostomy tubes, at least as far as these functional outcomes are concerned, should be made on the basis of information about hearing status. Not surprisingly, this is one of the main recommendations of the new clinical practice guideline for the use of tympanostomy tubes in children, just published by the American Academy of Otolaryngology–Head and Neck Surgery.²⁰ The guideline was developed by an inter-professional panel comprised of otolaryngologists, an otologist/neurologist, pediatricians, physicians, an audiologist, a speech-language pathologist and consumer advocates, and includes 12 specific action statements – some of which are quite relevant for audiologists.

The first recommendation is that tubes not be offered for OME with less than 3 months duration (Action Statement #1). One study of 2,565 children found that only 10% of children with middle ear effusion had fluid present after 3 months,²¹ so surgical interventions are unnecessary in most cases. However, once OME has persisted for at least three months, only a quarter of the cases resolve within 6 months and only a third within a year.²² Children with Type B tympanograms are also far less likely to experience spontaneous resolution, with only a quarter to a third resolving to a type A tympanogram by 6 months.²²

For those children with chronic OME (i.e., OME persisting longer than 3 months), the guideline recommends that clinicians obtain an age-appropriate hearing test (Action Statement #2). The guideline allows for the use of simple audiometric screening for children aged 4 or older, but recommends a comprehensive audiological evaluation for children who do not pass the screening, as well as for all children with chronic OME between 6 months and 4 years old. The guideline recommends offering tympanostomy tubes only to the children with chronic OME and hearing difficulties (Action Statement #3). Otherwise the recommendation is to monitor the situation for the development of hearing loss, structural abnormalities of the tympanic membrane or middle ear, or resolution of the effusion, at 3 or 6 month intervals (Action Statement #5).

There are two important exceptions to the hearing difficulty requirement, and one exception to the requirement for OME to persist for 3 months. First, tubes are suggested as an acceptable option for children with chronic OME without hearing difficulties but with other associated symptoms (vestibular

difficulties, poor school performance, behavioural problems, ear discomfort, or reduced quality of life; Action Statement #4). Second, tubes are suggested as an acceptable option for children at increased risk for speech, language and learning problems because of other factors such as Down syndrome, cleft palate, or permanent hearing loss, *in any case where the OME is unlikely to resolve quickly* (e.g., chronic OME or OME of any duration with a Type B tympanogram; Action Statement #9). The guideline thus recommends that clinicians determine if a child is at risk for speech, language and learning problems in cases of recurrent acute otitis media or OME of any duration (Action Statement #8).

The need for comprehensive audiometric assessment and for determining which children are at risk for speech, language and learning problems establishes a clear role for audiologists (and for speech-language pathologists) in informing decisions regarding tympanostomy tubes – one that has not always been recognized. For example, a 2004 survey in the US found that pediatricians disagreed, on average, with the statement that “I will send a child for audiological testing after 3 months of middle ear effusion.”²³ Hopefully this new guideline will help promote a better approach to the management of ear disease that affirms the importance of audiological assessment and inter-professional cooperation. The complete guideline can be downloaded free-of-charge from the following web address:

http://www.entnet.org/guide_lines/guidelines.cfm.

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- Canadian Hearing Report 2012;8(4):17–19.

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Hearing Preservation in Cochlear Implant Users

By Lendra Friesen, MSc, PhD and Samidha Joglekar, MCISc (C), Audiologist, Reg. CASLPO

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Lendra Friesen, (left) is an associate scientist at Sunnybrook Health Sciences Centre, director of research, cochlear implant program, otolaryngology, Sunnybrook Health Sciences Centre, and assistant professor, otolaryngology, University of Toronto.

Samidha Joglekar is a graduate of the MCISc Audiology program at the University of Western Ontario. She has been an audiologist at Sunnybrook Health Sciences Centre for the last three years. She currently splits her time between adult clinical audiology and cochlear implant research with the Friesen CI Lab.

The field of cochlear implants (CI) is constantly changing with new processing strategies, smaller hardware, cordless devices, longer and thinner electrode arrays, different surgical techniques, and revised candidacy criteria. One of the newer areas of interest relates to a combination of these factors: (1) candidacy criteria, (2) the electrode array, and (3) surgical techniques. This area of CI interest is currently called hearing preservation. Hearing preservation involves carefully inserting a longer, thinner, more flexible electrode array into either the round window, or into a very small oval window cochleostomy. The use of this type of electrode array and surgery are thought to preserve more of the auditory neural structures, as observed in animal studies.¹ The end result hoped for is that hearing will also be preserved. Thus, the candidacy criteria when using this technique have been changed to include

individuals who have measureable auditory thresholds (better than 90 dB at 250, 500, and 1000 Hz). This often includes even a mild loss at some of these frequencies. It is hoped that using this surgical technique will preserve low-frequency hearing so that it can perhaps be aided, but also in the hopes that it will better preserve all auditory neural structures, so that electrical hearing is also improved.

Along with this new method of implanting devices come questions related to whether neural structures are actually better preserved so that speech understanding or music perception is improved, and how different areas in the central auditory system are responding to electrical stimulation with this combination of new techniques. In the CI research community and especially in our center, we are trying to seek out the answers to some of these questions.

An individual with a CI is said to have hearing preservation if after their implantation they have low-frequency hearing thresholds under 90 dB at 250, 500, and 1000 Hz. Recent studies have demonstrated that low-frequency hearing preservation is initially possible with implanted patients.^{2,3} Also, at least one study has shown promising results in aided speech discrimination in these individuals.⁴ In our clinic we aren't finding differences between the two groups when we test speech understanding in quiet and in a +5 SNR with the CI only. Despite these initial findings, the clinical outcomes of using this technique are still relatively unknown. Perhaps even more importantly, the underlying neural structural survival and function with this type of stimulation are unknown. If we examine these patients using different types of tests, we might discover some fundamental differences.

One important thing to remember is that before we can even measure auditory thresholds, the underlying neural structures need to be functioning, beginning at the level of the auditory nerve and moving up to the higher centres in the auditory cortex. At the level of the auditory nerve, we can measure electrically evoked compound action potentials, commonly referred to as eCAP. This is a measure of how groups of auditory neurons are functioning. This type of testing can be measured through the CI manufacturer's clinical interface, used to measure the patient's threshold and comfort levels on a routine basis. Measurements such as amplitude growth can be completed, where the maximum amount of current required to evoke a prominent N1 waveform, followed by a less prominent P2 waveform are determined. The slope of the amplitude growth has been significantly correlated with speech performance.⁵ Recovery threshold is another factor of eCAP that can be measured. Here, two pulses are presented with increasing interstimulus intervals and the shortest interstimulus interval after which the second pulse is presented where an N1 and P2 waveform are observed is termed the recovery threshold. This interstimulus interval represents the refractory time required from many neurons (although it might not be similar for each individual neuron) to evoke an eCAP for a particular condition. One would assume that shorter intervals should in theory represent better or more efficient neural function; however, the opposite has been found. In fact, slower eCAP recovery was related to better temporal synchrony.⁶ How does this translate to speech understanding? Are these results better in individuals with hearing preservation? These answers are yet to be determined.

Cortical auditory evoked potentials

(CAEPs) reflect neural activity related to stimulus processing in the auditory cortex. In particular, the N1-P2 complex (responses at 100–200 ms after stimulus onset) is known to be sensitive to hearing thresholds.⁷ Larger-amplitude and shorter-latency N1 and P2 waveforms have been observed in individuals with better hearing thresholds. Another question we are interested in is whether individuals with hearing preservation have CAEPs that are larger in amplitude and shorter in latency than those without. If their neural structures are better preserved than in individuals without hearing preservation, this is something we would anticipate on finding. However, this doesn't appear to be the case. The exact reasons for this are still unknown.

Another area of interest in this group of patients involves the administration of pre-surgical steroids. Steroids are anti-inflammatory agents and it is thought that inflammation is reduced when they are used. Even though a less traumatic surgery is being implemented with the hearing preservation patients, literature suggests that even greater levels of protection may be achieved through pharmacologic hair cell protection, before, during, or after implantation.⁸ Therefore, the use of steroids, especially in these patients where more neural structures are already better preserved might have an even greater impact on patient speech understanding, eCAPs and CAEPs. However, questions surrounding the use of pre-operative steroids still need to be answered. For example, the optimal methods of delivery (oral vs. trans-tympanic) and duration pre-operatively are unknown. Also, further examination is needed into the specific effects of steroids particularly on patients who have hearing preservation.

One of the questions that is arising in the process of examining individuals with hearing preservation and those without is that perhaps we aren't correctly defining hearing preservation. It might be that the less traumatic surgery and more flexible electrode array are preserving so many more of the neural structures that a few measurable low-frequency thresholds aren't really a good indicator of hearing preservation. Perhaps we should be using some other measure to define this group. Exactly what is not yet known. This question is always at the back of researchers' minds as they move forward in this area.

Regardless of all these questions, we are in an exciting age where we are not only helping individuals to hear better with the use of CIs, but we are also learning more about hearing, auditory structural preservation, and its effect on the brain's ability to adapt in the process. Exactly how individuals with hearing preservation differ from those without is not jumping out at us with the types of testing implemented. However, we are still only examining a few of the factors involved in the auditory deprivation, cochlear implantation, and then the novel electrical stimulation used with a CI.

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Don't Upset the Balance: Why Children Need to Hear from Both Ears

By Karen A. Gordon, PhD and Blake C. Papsin, MD, MSc, FRCS, FACS, FAAP

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The cochlear implant program at The Hospital for Sick Children has been providing bilateral cochlear implants to children with severe to profound hearing loss in both ears. In the following article we review why we feel it is important to provide these children with two rather than one cochlear implant and why this should be done with minimal delay when possible.

THE IMPORTANCE OF BINAURAL HEARING IN CHILDHOOD

Binaural hearing is so integral a part of normal listening that we rarely pay attention to the fact that sound waves from one source are entering each of our ears separately. The integration of these two independent inputs in the auditory pathways is so effective that we focus on

the single auditory image of the sound we are listening to. The first point of integration in the ascending pathways is in the superior olivary complex in the brainstem. We perceive where the sound is coming from along the horizontal plane in space using subtle differences in timing and level of the inputs reaching each ear. These cues are carefully coded in the brainstem using a combination of excitatory and inhibitory inputs.¹

Binaural/spatial hearing is particularly important for children who are rarely alone and often in groups (child care, classrooms, playgrounds, etc.). These situations require a child to listen to sound from all directions and frequently to multiple sounds and talkers at once. A child with unilateral deafness loses access

to binaural/spatial hearing^{1,2} and thus has more difficulties hearing in these types of noisy environments.¹ This has negative implications for learning and, indeed, educational outcomes in children with unilateral deafness are poorer than normal.³⁻⁵ Perhaps these are some reasons why these children rate their quality of life at levels similar to children with bilateral hearing loss.⁶ With this in mind, why were children who are deaf in both ears traditionally provided with only a unilateral cochlear implant?

UNILATERAL COCHLEAR IMPLANTS RESTORE HEARING BUT SET UP ASYMMETRIC AUDITORY DEVELOPMENT

Cochlear implants have given children who are deaf access to sound.⁷ Early fears

about the risks of the surgery and the uncertainty about improvements to the device or possible treatments for deafness kept the procedure limited to one ear so that the other ear would be unaffected. The primary aim of cochlear implantation in children is to limit effects of bilateral deafness including cross-modal reorganization and delays in speech and language development.^{7,8} This aim can be met by identifying the hearing loss as soon as possible after onset (congenital in many children) and implanting shortly thereafter even if only in one ear.^{7,8} In practice, we routinely provide cochlear implants to children who were deaf from infancy at very young ages (i.e., < 2 years).

We have used both electrophysiological and behavioural measures to track auditory development after early unilateral cochlear implant use and find development is promoted throughout the auditory pathways and often at very similar rates of maturation to normal hearing children. Responses from the auditory brainstem can be largely mature after 1 year of unilateral implant use^{9,10} and cortical responses show a progressive change in morphology with the mature P1-N1-P2 wave amplitude peaks emerging after ~12 years of unilateral implant use.¹¹ Children implanted early develop spoken communication skills¹² and often go to school with their normal hearing peers. Many graduates of our cochlear implant program have gone to university and have become professional athletes, academics, health care professionals, lawyers, administrators, and business people.¹³ With all of this success it is hard to remember that this development has been achieved with an abnormal input coming from only one ear. On the other hand, we must acknowledge the incredible work and effort that is put into this development by the parents/caregivers, teachers,

therapists, audiologists, otolaryngologists, and, of course, the implant users themselves.

Bilateral implantation was first suggested as a way of making hearing easier for individuals with deafness in both ears. Our concern was that the plasticity of the pathways from the non-implanted ear might be compromised either because the pathways were changed by the development we promoted with the first implant and/or because the ear opposite to the implant had been deprived of input for too long. It was clear that the pathways from the non-implanted side remained immature relative to the implanted side; auditory brainstem response latencies were prolonged once that second ear was implanted in children who had had unilateral implant experience^{14,15} and cortical responses on the newly implanted side were abnormal.^{16,17} Further studies revealed that unilateral implant use exceeding 1.5 years in children allowed an abnormal strengthening of activity to both contralateral and ipsilateral auditory cortices from the stimulated ear.¹⁸ This was not reversed by a period of bilateral implant use of up to 4 years.¹⁸ The abnormal cortical findings were correlated with poorer speech perception scores which suggested that we may be able to achieve better hearing if more symmetric and normal patterns of auditory activity could be promoted.¹⁸

STIMULATING BOTH EARS WITH LIMITED DELAY ALLOWS SYMMETRIC DEVELOPMENT OF BILATERAL AUDITORY PATHWAYS

We asked whether we could avoid asymmetric development of the auditory system by limiting the duration of deafness in children and providing bilateral implants simultaneously. To answer this, we have been studying almost 200 children who have received

bilateral implants in the same surgery. We have found that responses from the auditory brainstem change in tandem on both sides with no significant differences in latencies between sides at any time measured.^{14,19} Cortical responses are similar between the ears and, in young children, appear to have oscillating characteristics similar to those described in the normal hearing brain.²⁰ Moreover, bilateral stimulation without delay appears to protect the auditory cortex from the abnormal changes found after unilateral implant use; responses from children in this group were not significantly different from normal.¹⁸

Behavioural measures support the electrophysiological findings and show that children receiving bilateral implants simultaneously are able to detect and recognize speech in quiet and noise equally on both sides.²¹ They also show larger benefits of listening with bilateral compared to unilateral implants than do children implanted sequentially.²² These findings have supported our clinical practice of providing bilateral cochlear implants to children with limited delay when appropriate and possible.

WHY SEQUENTIAL BILATERAL IMPLANTATION IS STILL WORTHWHILE

The advantages of simultaneous bilateral implantation should not be interpreted to mean that there is no sense in providing bilateral implants sequentially. Although we showed that there are changes to the developing bilateral auditory pathways with unilateral implant use, it does not mean that binaural function has been concurrently eliminated. We were somewhat amazed to find that, despite the asymmetric development promoted with a unilateral implant, binaural integration was retained in the auditory brainstem of children who were bilaterally implanted

(simultaneously and sequentially).¹⁴ Moreover, there was evidence that some degree of tonotopic organization persists and that the auditory brainstem remains able to code interaural level differences.¹⁴ This means that there is potential to restore binaural processing in children who are deaf even when implants are provided sequentially.

Behavioural data also show that bilateral cochlear implants benefit children even when provided sequentially. Children receiving bilateral implants sequentially and simultaneously were able to use spatial cues to improve their detection of speech in noise.²³ Speech detection thresholds improved when the noise source was moved away from the speech source by 90 degrees either to the left or right. These improvements were more modest for children implanted sequentially than for children implanted simultaneously and showed an asymmetry (less improvement when noise was moved to the first implanted side than the second) but were still significantly better than nothing. This meant that they were receiving some benefit from their two implants.

Results like these might explain why most of the >150 children who have received a second implant sequentially in our cochlear implant program still choose to use both their cochlear implants. In ongoing work, we are examining how we might optimize how they hear with their bilateral devices.

FUTURE DIRECTIONS FOR IMPROVING BILATERAL COCHLEAR IMPLANT USE IN CHILDREN

We have a number of studies ongoing and planned to further improve bilateral cochlear implant use in children. We are interested in how to better program the current levels delivered by the two

implants so that binaural level cues are accurately delivered to the auditory system. In addition, we seek to better match pitch and timing of input from the two independent devices. We are also embarking on a new project in which we will attempt to reduce asymmetries in the auditory system after unilateral implant use by removing input from the first implanted ear to strengthen pathways from the second implanted ear. We will be asking children to do this for regular periods throughout the day.

The benefits of bilateral implantation in children are now well documented and increase as the delay to implantation decreases (at least in the early years of bilateral implant use). Ongoing work seeks to capitalize on this success and further improve hearing for children who receive bilateral cochlear implants.

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Tinnitus: The Dark Side of Neuroplasticity

By Steve Aiken, PhD, and Philippe Fournier, MScS

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Neuroplasticity should be the *least* surprising feature of the human brain. After all, we are amazingly flexible and adaptive creatures who are always learning new things, navigating complex social environments, and having new experiences. Like rivers and snowflakes, no two thoughts are precisely the same. If the brain didn't have the capacity to change, it's hard to imagine how this would even be possible.

On the other hand, it's a bit surprising when we find plasticity at more "foundational" levels of the nervous system like the brainstem. We all know that the brainstem matures far earlier than the cortex – one of the reasons we rely on the auditory brainstem response to estimate infant hearing thresholds – so how can the adult brainstem be neuroplastic?

A decade ago, Craig Formby and colleagues¹ showed that the perceived loudness of a sound is significantly increased after only a few weeks of auditory deprivation (i.e., wearing an

earplug). Of course, this could be because of an up-regulation of activity in the lower brainstem (i.e., a gain increase) or a change in the way that the information is processed at higher levels of the auditory system. Munro and Blount² decided to test this by exploiting the relationship between loudness perception and acoustic reflex thresholds,³ since the acoustic reflex is mediated by the brainstem. They had people wear earplugs in one ear for a week, and measured acoustic reflex thresholds at the start of the study, immediately after the earplug use, and one week following earplug use. They found that reflex thresholds decreased by 5–7 dB in the plugged ear immediately after the week of earplug use, but returned to normal a week later. In other words, a mere seven days of earplug use produced a physiological change – an up-regulation of activity in the brainstem! Evidence for this type of neuroplasticity, called "homeostatic regulation," has also been found in animal models: rodent studies have found increased firing rates in the cochlear nucleus after acoustic trauma.^{4,5}

Could this be the source of tinnitus? Roland Schaette and colleagues suggest that it could be.⁶ Auditory deprivation from earplug use (or hearing loss) translates into less sound-induced movement of the basilar membrane, less inner hair cell depolarization and less auditory nerve firing. But remember that the auditory nerve never sits quietly – it is always spontaneously firing at some rate – it just increases its firing rate and becomes more synchronized when excited by the inner hair cells. If the output of the auditory nerve is "amplified" by an up-regulation of activity in the cochlear nucleus, this would also "amplify" the spontaneous firing of the auditory nerve, perhaps to the point where it is detected as sound. This would be similar to the increase in microphone noise that you get when you raise the gain in a hearing aid. Gain has consequences! In support of this idea, tinnitus has been experimentally induced in normal participants by isolating them in a soundproof booth^{7,8} and by having them wear specialized musician's earplugs for a one-week period.⁹

Of course there is no principled reason why the up-regulation could only occur in the cochlear nucleus. In fact, increased spontaneous firing rates associated with induced tinnitus have also been found in the inferior colliculus in mice¹⁰ and in the auditory cortex in cats.¹¹ If Schaette's hypothesis is correct – that tinnitus is at least partly caused by an up-regulation of activity in the cochlear nucleus – we should see this gain increase occurring in people experiencing tinnitus. Fortunately we have a way of testing this in humans, by using the auditory brainstem response. The primary sources of wave I (the distal auditory nerve) and V (the lateral lemniscus) are on either side of the cochlear nucleus, so a gain increase in the cochlear nucleus should result in an increase in the amplitude of wave V relative to the amplitude of wave I. Is there any evidence for this? Yes: Gu and colleagues¹² found reduced wave I amplitudes and enhanced wave V amplitudes in subjects with tinnitus than in non-tinnitus controls (matched for age, sex, and hearing thresholds). Likewise, Kehrle and colleagues¹³ and Schaette and McAlpine¹⁴ found significantly higher wave V/I amplitude ratios in normal-hearing subjects with tinnitus than in normal-hearing subjects without tinnitus.

It's interesting to note that tinnitus is strongly associated with hyperacusis.¹⁵ The results of Munro and Blount² suggest

that deprivation-induced loudness changes are mediated by an up-regulation of activity in the brainstem, so homeostatic neuroplasticity in the cochlear nucleus might play a role in both tinnitus and hyperacusis. The dark side of neuroplasticity may hide more secrets!

These studies suggest that brainstem neuroplasticity plays a significant role in the problem of tinnitus and possibly hyperacusis, and hint at a way to prevent these problems: compensate for hearing loss or reduced auditory nerve activity so that neuroplastic compensation does not occur in the first place. This is certainly not the end of the story, but it's an interesting chapter.

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The Auditory Evoked P2 Response Indicates Effects of Aging On Central Auditory Processing

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ABSTRACT

Auditory communication deficits in aging are evident in complex listening situations and include impairment in central auditory processing in addition to sensation loss. In several experimental studies we found that the auditory evoked P2 response was reduced and prolonged in older adults whereas earlier N1 and P1 responses were consistent with young adults. We interpret the findings that in aging even complex sound stimuli are represented precisely at the level of the auditory cortex, whereas the following interpretation of sound information and binding, which are required for object formation, is less effective and hence, perception is impaired.

Elderly people often experience difficulties in speech comprehension in a noisy environment.¹ The speech-in-noise deficits are correlated with age-related hearing loss, suggesting that hearing loss is a main cause of the deficits.² Although, hearing aids compensate for sensation loss, speech understanding in noise improves often less than expected.³ One reason is that hearing impairment is more complicated than just a loss in sensation; rather it includes limitation of the loudness range and impairment of temporal and spectral acuities. Degraded temporal acuity strongly affects speech comprehension^{4,5} and is often measured as gap detection performance, which again decreases with sensitivity loss at high frequencies.⁶

However, even when accounting for the effects of hearing loss, older listeners require a wider gap for detection^{7,8} particularly under complex listening conditions. Older adults detect a short gap as well as young listeners, when the gap is presented in isolation, but they perform poorly when the gap occurs in vicinity of other sound changes.⁹ Such findings have been interpreted as supporting the hypothesis that aging affects central auditory processing.^{9,10} Moreover, hearing loss occurs gradually over time and may result in acquired neuroplastic changes in the central auditory system.¹¹ This mini review summarizes some results from our recent studies, which suggest that aging affects central auditory processing at higher level

than it has been previously thought.

One main function of central auditory processing is to enhance the representation of spectro-temporal acoustical patterns and then binding the sound elements into a higher-order representation of an auditory object.¹² This processing hierarchy can also be described as a first level of interpreting the sound, which for example leads to perception of speech objects. Neuroimaging showed that anterior temporal brain areas responded specifically to sound categories produced by animals, human, or musical instruments, suggesting that an auditory object is most completely represented in anterior auditory regions.¹³

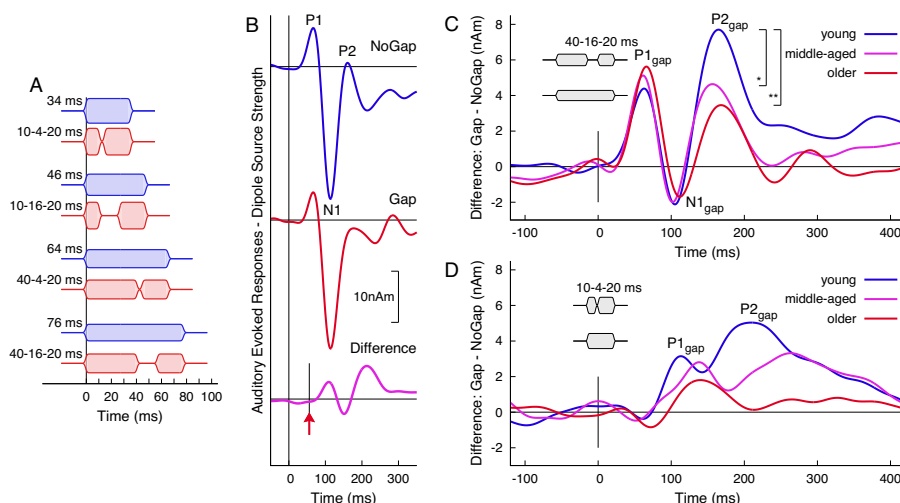


Figure 1. Auditory evoked responses in gap detection. (A) Stimuli were random sequences of short 1000-Hz tones, either continuous or containing a gap. Gap durations were 4 ms or 16 ms. The duration of leading marker was 10 ms or 40 ms, whereas the trailing marker was always 20 ms. Amplitudes were adjusted for equal sound energy. Stimuli were presented binaural at 80 dB SPL with randomized inter-stimulus interval of 120 to 320 ms. (B) Grand averaged response waveforms to stimuli with 76 ms duration without a gap and with a 16-ms gap after a 40-ms leading marker. The difference waveform illustrates a P1-N1-P2 like response following the onset of the trailing marker, which is indicated by the red arrow. (C) Grand averaged gap responses for the young, middle-aged and older participants. (Time zero relates to the onset of the trailing marker). Whereas the P1gap and N1gap responses were consistent across age groups, the P2 gap amplitude was significantly reduced in the middle-aged and older compared to the young participants. (D) Gap responses in the most difficult condition of a 4-ms gap following a 10-ms leading marker. The prominent difference between the groups was a decline in the P2gap response with increasing age.

Auditory evoked responses recorded with electroencephalography (EEG) and magneto-encephalography (MEG) can serve as objective indicators for central auditory processing. Most related work has focused on the P1-N1-P2 waves of the auditory evoked responses at 50 ms, 100 ms, and 200 ms latency, which may indicate processing steps along the hierarchy of auditory processing. However, specifically the functional significance of the P2 response is widely unknown. Historically, the N1 and P2 have been seen as biphasic waves of a single response. Thus, in some early ERP studies the amplitude has been measured as the difference between the negative peak of the N1 and the positive peak of the P2 wave. However, several studies found that N1 and P2 amplitudes

depended differentially on variation of experimental parameters.¹⁴⁻¹⁶ Crowley and Colrain¹⁷ examined different scalp topographies, effects of brain lesions, and the effects of age, sleep, and attention on the N1 and P2 amplitudes and concluded functional independence of both responses. Moreover, the N1 and P2 waves consist of multiple distinct components responding differentially to exogenous and endogenous events.¹⁸ Considering which step of auditory processing occurs at the latency interval around 200 ms may provide a further hint about the functional meaning of the P2 wave. Jääskeläinen²⁰ suggested that the earlier N1 component serves as a gating mechanism that transfers incoming sensory information to further analysis of the auditory object in more anterior brain

regions. There is also evidence that the N1 reflects the stage of sensory coding of stimulus onset as well as acoustic changes (e.g., VOT).^{21,22} At 200 ms, this early sensory processing has been completed, an auditory object is established, and can be accessed for perception.²³ Therefore one focus of interest in our studies was on the functional relevance of the P2 response with 200-ms latency and how it changed with increasing age.

GENERAL METHODS

MEG was recorded in groups of young (mean age of 25 years), middle-aged (50 years), and older (70 years) adults. Participants had normal hearing for their age. Commonly, thresholds were elevated in the older participants above 2,000 Hz increasingly toward higher frequencies. All auditory stimuli were at low spectral frequencies and sensation was equally well for the participants. The experimental procedures were designed for eliciting sound onset responses and specific responses to temporal gaps, interaural time relation, and pre-voicing time of a speech sound, respectively. This design allowed comparing within each group the responses to sensation of the stimuli and higher order central processing related to performance in temporal acuity, sound localization, and learning. Cortical source were consistently found anterior to the N1 source.

GAP DETECTION

In a gap detection study, we presented brief tone bursts, either continuous or containing a gap, in random order.²⁴ Generally, a gap was more difficult to detect if it was short and even more if it occurred immediately after the sound onset, such as if the leading marker was short. Specifically for elderly listeners gap detection with a short leading marker is challenging.²⁵ The stimuli are shown in order of gap detection difficulty with the

THE AUDITORY EVOKED P2 RESPONSE INDICATES EFFECTS OF AGING ON CENTRAL AUDITORY PROCESSING

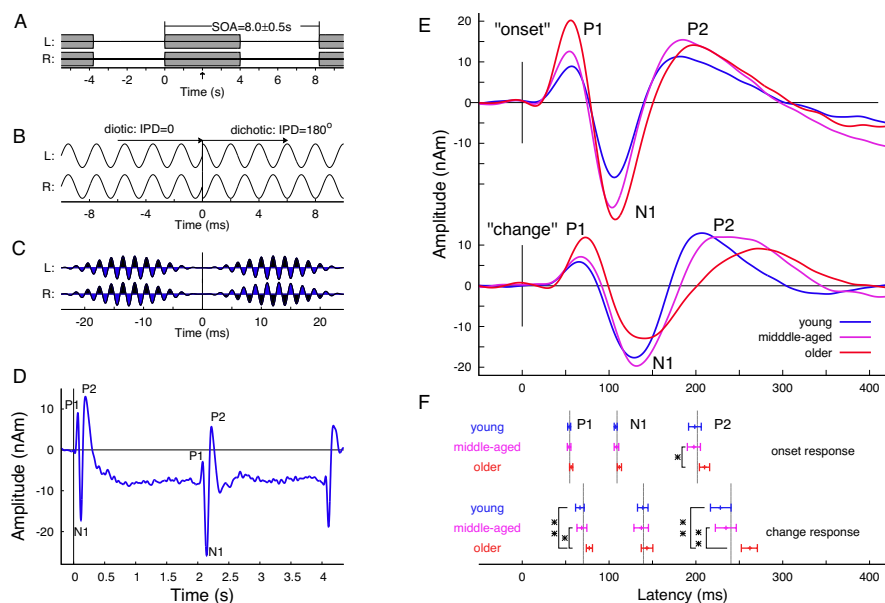


Figure 2. Auditory evoked responses to changes in inter-aural phase difference (IPD). (A) Stimuli were tone bursts of 4-s duration, presented with stimulus-onset asynchrony (SOA) of 7.5 s to 8.5 s. (B) At midpoint of the tone, the inter-aural phase relation was changed from same to opposite polarity. (C) The sounds were amplitude modulated at 40 Hz. The inter-aural phase shift occurred at a minimum of the amplitude modulation to avoid a click sensation. The stimuli induced the perception of a change in spaciousness of the sound, originating either from a focal source in the center of the head or from diffuse sources in space. (D) The grand averaged response to a 500-Hz stimulus in the young group was characterized by the P1-N1-P2 onset response, a sustained negative shift, a P1-N1-P2 change response, and an offset response. (E) Comparison of onset responses and change responses between the age groups. P1-N1-P2 amplitudes even increased with age and latencies were generally similar across the groups. For the IPD change response an increase in P2 latency becomes evident. (F) Analysis of peak latencies showed consistent latencies for the onset responses, except a slightly increased P2 latency in the older group. The change response N1-latencies were consistent across groups, whereas P1 latency increased slightly. Most prominent effect was the P2-latency increase across the age groups, considerably larger than for the onset P2.

most difficult one at top of Figure 1A.

The onsets of both types of stimuli elicited series of P1-N1-P2 responses. For the gap stimuli, the onset of the trailing marker elicited a second series of P1-N1-P2 responses. Subtraction of the responses to continuous sounds from the gap responses diminished the onset responses and revealed the response to the trailing marker in the gap stimuli (Figure 1B). Under the assumption that detection of the trailing marker is critical for gap detection, the difference wave was interpreted as an objective indicator for gap detection.

The main findings with respect to aging were that the P1 and N1 waves of the gap response were consistent across the age groups (Figure 1C). For the stimulus with longest duration the P1 amplitude showed even a tendency to increase with age, and this P1 increase was even more clearly expressed for the onset response. As a difference between age groups, the P2 amplitude was significantly reduced in the middle-aged and older participants compared to the young (Figure 1C). The contrast between age groups was even larger for the most difficult stimulus, which elicited a clear P2 wave in the young and a smaller and delayed P2 wave

in the middle-aged group, whereas the P2 response was practically absent in the elderly (Figure 1D). The P2 results were paralleled by gamma band oscillations, which were consistent across age groups at early latencies, however were considerably reduced in the 150 ms to 200 ms latency range.

PROCESSING OF INTER-AURAL TIME DIFFERENCES

Binaural hearing for sound localization is one hallmark for central auditory processing because it requires that the sounds, received with both ears, will be combined at multiple levels along the auditory pathway. Interaural time or phase differences are used for spatial localization of low frequency sounds. Moreover, listeners can tolerate more noise if the target speech sound appears with a phase difference at both ears.²⁶ We used in our study binaurally presented tones, which were either same at both ears, such as in phase, or of opposite phase (Figure 2A–C). The stimuli elicited an onset response related to hearing the sound and a change response, which is an objective indicator for detection of the interaural phase or time difference (Figure 2D).²⁷ Whereas the main finding was that young people can detect changes in ITD in sound with frequencies up to 1500 Hz, middle-aged and older people can do this at lower frequencies only, the data gave insight in how the onset and change responses varied with increasing age.²⁷ The P1 onset response increased significantly with age. The N1 was largest in the elderly however the N1 increase did not reach significance because of between-subject variability. P2 amplitudes were not different in the onset responses (Figure 2E). The change response showed a slightly increase in the P1, otherwise consistent amplitudes across the age groups. However the P2 latency was significantly delayed in the elderly compared to middle-aged and

younger listeners (Figure 2F). The simultaneous recording of onset and change responses showed that the P2 delay in the older listeners was specific to detection of the interaural time difference but not to hearing the sound.

SOUND FAMILIARITY AND LEARNING

Changes in the acoustic environment require continuous adaptation of central processing for optimal auditory perception. Such capability of neural plasticity and reorganization has been shown even in the adult brain. In a study about the neural mechanism of auditory learning, we trained participants identifying a new speech sound, which was a modification of the syllable /ba/ by increasing the pre-voicing time, a feature, which is not used in English language (Figure 3A). We recorded the MEG at two succeeding days before and one day after six days with one hour of identification training each (Figure 2B). Main findings were that the P2 amplitude increased significantly, and the P2 increase was already evident between the two pre-training sessions, whereas P1 and N1 amplitudes were not modified between repeated recordings (Figure 3C). The initial P2 increase was interpreted as reflecting familiarization with the stimulus,²⁸ whereas the later P2 increase was correlated with improvement of behavioural performance during training.²⁹ Comparison of the evoked responses between age groups showed similar sizes of P1-N1-P2 responses. The P1 and N1 amplitude increased with age, which was significant for the P1. The main difference between groups was that the amount of P2 increase was smaller in the middle-aged and even more in the older participants compared to the younger (Figure 3C).

CONCLUSIONS

In a series of studies we recorded

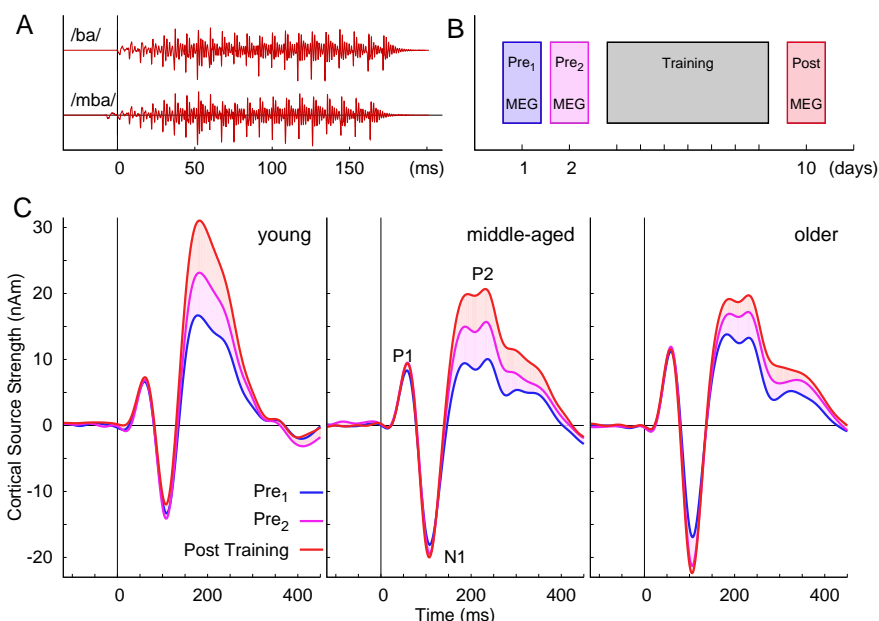


Figure 3. Auditory evoked responses in training for identification of voice-onset time differences. (A) Stimuli were versions of the syllable /ba/ containing different amounts of pre-voicing and then labeled /mba/. (B) Time course of the experimental procedure. MEG recordings were performed two days before and one day after six days of identification training as well as after the training (C) Grand averaged response waveforms for the three MEG recordings in the three age groups. Whereas the P1 and N1 responses were of same amplitude in repeated recordings, the P2 amplitude increased between the pre-training sessions as effect of familiarization and after the training. P1 and N1 amplitudes increased across age groups. The experience and training induced P2 amplitude increase was smallest in the older participants.

neuromagnetic auditory evoked responses, which were specific to central auditory processing and observed commonly, that P1 and N1 responses were preserved or even enhanced in aging, whereas the P2 response was decreased and prolonged in older adults compared to young and these effects were often evident in middle-aged participants. We interpret these results that the representation of the sensory input is less affected in older adults; however, it seems that they have difficulties to access and interpret the sensory information, thus binding for object formation is less efficient and perception is impaired in aging. Such interpretation is consistent with the common complaint of elderly people in a complex listening situation such as in noise or reverberating environments: “I can hear you, but I

cannot understand what has been said.” Thus aging seems to affect central auditory processing beyond the stage where perception is successfully completed. The findings are important to understand the neural mechanism underlying communication deficits in aging. Specifically this is important for developing new training strategies. Although we found that the effects of training and familiarization with sound are reduced in aging, it has to be investigated to which extend training could overcome the aging related decline in central auditory processing.

ACKNOWLEDGEMENT

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Development of Pitch Perception and the Processing of Simultaneous Sounds in Infancy

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Pitch conveys information about melodic and harmonic structure in music, and it is critical for the perception of syllable stress, phrase prosody and emotional communication in speech. Pitch also provides cues for the identification of objects in the environment. Thus, processing pitch is important for infants who need to learn about what is in their environment and to communicate through language and music.

Sounds with pitch typically have energy at a fundamental frequency (F0) and at harmonics occurring at integer multiples of F0. In this case, the perceived pitch corresponds to the frequency of F0 (e.g., a sound with energy at 100, 200, 300, 400, 500 Hz has a perceived pitch of 100 Hz). However, removal of energy at F0 does not alter the pitch (e.g., a sound with energy at 200, 300, 400, 500 Hz has a perceived pitch of 100 Hz) because adults use spectral and temporal structure conveyed by the harmonic as well as that of the fundamental in order to determine the pitch. This is called

perceiving the pitch of the missing fundamental. Although sound frequency is represented subcortically, converging evidence suggests that the integration of harmonics into a representation of pitch does not occur until auditory cortex.¹

The auditory cortex of very young infants is quite immature,²⁻⁵ raising the possibility that although they can process frequency, they might not be able to perceive pitch. We tested this by measuring electrophysiological (EEG) event-related potential (ERP) responses in infants between 3 and 7 months of age.⁶ Specifically, on every trial we presented two complex tones in succession. On *standard* trials, each tone had 10 harmonics, randomly chosen from the first 15 harmonics, but always including F0. The F0s of the two tones (and the frequencies of their harmonics) varied randomly from trial to trial, with the constraints that F0, and therefore also the perceived pitch, always increased from the first to the second tone, and the frequency of each harmonic present also increased from the first to the second

tone. Occasional *deviant* trials were presented interspersed with standard trials. Deviant trials were the same as standard trials in all respects except that the harmonics of the second tone were chosen such that they were at integer multiples of a missing F0 that was lower than the (present) F0 of the first tone. Thus, if infants were able to integrate the harmonics into a percept with pitch, they would perceive the pitch going down from the first to the second tone on deviant trials. But if they were unable to integrate the harmonics into a pitch percept, they would perceive each frequency component as going up from the first to the second tone, and deviant trials would not sound any different than standard trials.

We made use of the fact that when occasional deviant trials are included in a sequence of standard trials, the evoked ERP response from the brain contains a component called the Mismatch Negativity (MMN) that reflects the brain's "surprise" at hearing the deviance.^{7,8} MMN is an "automatic" response in that

it does not require attention to be elicited, and it can be measured in infants. We found a robust MMN response to the deviant trials in 4-month-olds, 7-month-olds and adults, but not in 3-month-olds. This indicates that a cortical representation for pitch emerges between 3 and 4 months of age.

The flip side of the ability to integrate harmonics into a pitch percept is the ability to segregate harmonics from different sounds that occur at the same time. This is part of what is known as Auditory Scene Analysis,⁹ the ability to determine *what* auditory (sounding) objects are present in the environment and where they are located. When there are multiple auditory objects in an environment, the sound waves they produce add in the air and reach the ear as a single complex sound wave. One cue that the auditory system uses in order to segregate the frequency components that belong to one sound from those belonging to a second sound is harmonicity. In other words, frequency components that form harmonic relations (i.e., are integer multiples of a common F0) are grouped together as coming from one auditory object and are segregated from other frequency components which are interpreted as coming from a different auditory object. We hypothesized that if the ability to form a cortical representation of the pitch of the missing fundamental is not present until 4 months of age, a similar developmental trajectory would be expected for the ability to use

harmonicity cues in auditory scene analysis. We made use of the fact that, in adults, if one harmonic of a complex tone (we used tones with F0 and five harmonics) is mistuned (we mistuned the third harmonic by 8%), the mistuned harmonic is no longer integrated with the rest of the harmonics into a single auditory object with pitch at F0, but “pops out” as a separate tone. In this case, adults hear two sounds, one with pitch at F0 and the other with the pitch of the third (mistuned) harmonic.

Again we measured EEG, but this time we looked for an Object-Related Negativity (ORN) response that has been shown previously to be present when two auditory objects are perceived but not when one is perceived.¹⁰ On 50% of trials we presented the tone with harmonic in tune, and on 50% of trials we present the tone with the third harmonic mistuned. If infants perceived the mistuned harmonic as a separate auditory object, we expected an ORN response on mistuned trials. We found no evidence of an object-related response at 2 months of age, but a significant object related response was present at 4 months of age.¹¹

We conclude that the ability to integrate harmonics into a single percept with pitch and the ability to use harmonicity to perceive two simultaneous auditory objects both emerge around 3 or 4 months of age and depend on cortical maturation. Thus there is rapid development of the pitch perception

underlying speech, music and auditory scene analysis during the first months after birth.

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Things That Make You Go Hum

By Marshall Chasin, AuD

Taos, New Mexico is the home to the Taos hum. It's not a sports team although that would be a great name. It is, however, the name of an indie band, but that's not the topic of this article. The Taos hum is a constant low frequency hum that many of its residents can hear, day or night.

It's not very loud so people can't typically hear it during rush hour. Generally it is better heard inside a house (or when it's very quiet), earplugs don't affect it, and some report that it's more of a "feeling" than an actual sound. Similar hums have been reported elsewhere around the world and have a name associated with their location – Auckland hum in New Zealand, Bristol hum in England, and the Kokomo hum in Indiana but, so far no humming in Canada. The interesting thing is nobody seems to be able to find the source.

When first popularized by the American media in the early 1990s, scientists tried to measure the sound and there are some published reports of the various spectra. It tends to have most of the energy around 55–60 Hz (near the bottom of the piano keyboard). The Taos hum was the subject of television shows such as *Unsolved Mysteries* and the *X-Files*.

Based on what is known about acoustics and about how our hearing mechanism works, it is possible that most of the energy of the Taos hum is below our range of hearing but that certain of its higher pitched harmonics (which are still very low frequency) are the ones that are being perceived. Such a low frequency sound would easily be masked by environmental city noise so that the

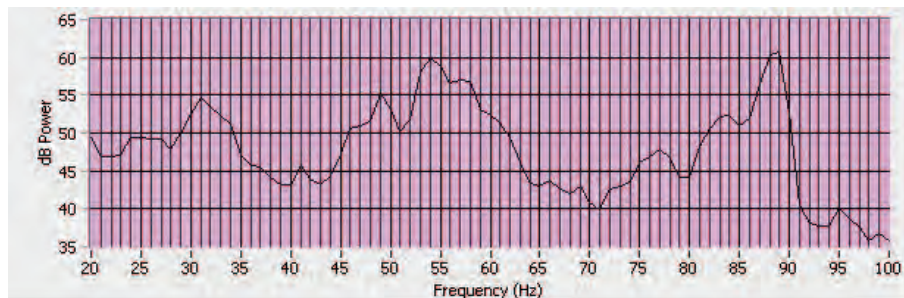


Figure 1. The power spectrum of the Auckland hum

comment that it is heard better inside houses or in quiet locations, makes sense. Also, since earplugs have minimal effect in the very low frequency region, it also makes sense that the Taos hum would not be affected by earplugs. All of this sounds strange, but its straight forward science and not really the subject of the *X-Files*. Low frequencies have long wavelengths and unless an obstruction such as a wall of a house or an earplug is about half of the wavelength, then it has minimal effect. The wavelength of 50 Hz is about 6 meters so obstructions less than 3 meters thick (such as earplugs) would have no real effect on the attenuation of sounds.

The power spectrum of the Auckland hum is shown in Figure 1. This is from Tom Moir of the Massey University in Auckland, New Zealand. There are two important features of this graphical display: (1) The first resonance (peak) is at about 30 Hz, the second at about 55 Hz and the third at about 88 Hz. The second and third resonances are roughly double and triple the values of the first peak at about 30 Hz. This suggests that these sounds are harmonics of a system that has a half wavelength characteristic. If it was just noise of a poorly designed measurement system, there would not be

any such regularities in the response. One criticism of these power spectrum measurements is that they are false, but the regularity in the pattern of peaks would suggest otherwise. (2.) Despite the fact that the power spectrum has peaks on the order of 60 dB SPL, these sounds are near our threshold of hearing, since the minimal audible field correction for such low frequencies are on the order of 40–50 dB, so these peaks are roughly 10 dB SL.

Some candidates for potential sources of the various hums include power lines, ocean currents, dynamic geological structures, or spontaneous otoacoustic emissions. The oceanic and geologic etiologies are possible as are spontaneous otoacoustic emissions. My personal favourite is space aliens. To date there is no real evidence to support the power line etiology since the hum is just as audible during power outages and the power spectrum has a peak at 30 Hz which is one half what would be expected from a "60 Hz buzz" power line (50 Hz in Europe and Australia). However, if space aliens use a 30 Hz alternating current in their equipment, this would be evidence of an extra-terrestrial source.

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