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We welcome any type of article related to audiology and otology. Please send them at editor.chr@andrewjohnpublishing.com

FEEDBACK:
We welcome your views and comments. Please send them to hearingreport@andrewjohnpublishing.com
I am pleased to provide Canadian Hearing Report an overview of some of our recent research that may be of interest to audiology professionals. I have directed the Auditory Science Laboratory at the Hospital for Sick Children (SickKids) for over 30 years, and during that time we have published research on animal models of hearing loss of many types and causes. The list is long: presbyacusis, conductive loss, ototoxic drugs, endolymphatic hydrops, acoustic trauma, chronic hypoxia. Most recently we have been exploring another etiology, hearing loss caused by congenital cytomegalovirus (CMV).

Those involved in pediatric audiology will be particularly interested in CMV induced hearing loss. You will know that it is very poorly diagnosed – suspected often but not definitively proven. The degree of hearing loss resulting from CMV infection ranges from mild to severe/profound. In our cochlear implant program at SickKids, more than 20% of our candidates have severe or profound hearing loss related to CMV infection. The hearing problem can be present at birth, or can develop more slowly over time and manifest after birth. Many CMV infected newborns are asymptomatic and pass hearing screening with inner ear problems becoming apparent much later. One study estimates that 10% of children, 4 years of age, with “idiopathic” hearing loss have had a congenital CMV infection.

This research to study the effects of CMV infection on the inner ear has largely carried out by Dr. Mattia Carraro (as part of his PhD thesis) in collaboration with a team at the University of Utah (led by Dr. Albert Park in the Division of Otolaryngology). Our focus has been on damage to the vasculature of the cochlea. For this Canadian Hearing Report, I will generally describe our findings rather than the full details that are (or will soon be) available in published papers.

We have used a mouse model that shows many similarities to human CMV infection. We inoculate the brain of a newborn mouse with virus. Of course in humans CMV infection typically starts during pregnancy, but because the mouse is born in an immature state its condition at birth (including stage of hearing development) is equivalent to an infant in utero. Furthermore in our mouse model, as with humans, the degree of hearing loss resulting from CMV infection is extremely varied. Animals given identical doses of CMV develop a range of hearing impairments from mild through to profound deafness (as assessed using from auditory brainstem evoked potentials (ABR).

A novel aspect of our research is that we predicted that this viral infection might first affect the blood capillary beds of the cochlea. It turns out that we were correct. In order to investigate any change to blood vessel structure we refined a histological technique called corrosion casting. This involves injecting a liquid polymer into all of the blood vessels. When this plastic polymerizes it create a hard plastic cast of the vessels including arterial supply, capillary beds and veins. To see the casts we corrode away the bone and all soft tissue. In the illustrations of this report, we see the corrosion casts of capillary networks in the cochlea viewed with a scanning electron microscope.

Our experimental protocol is summarized in the box below. Essentially we inject (arrow) the brain of newborn mice with CMV. At 4-6 weeks hearing function is tested with ABR and otoacoustic emissions (DPOAEs) and at 8 weeks we study the cochlear vasculature.

The first signs of vascular damage resulting from CMV infection are seen at the cochlear apex. This is illustrated by Fig. 2 below. Note the lack of stria vascularis capillaries in the highlighted region.

In other subjects CMV causes more extensive damage to the cochlea vasculature. Two examples are illustrated in Figs. 3 and 4 below.

In addition to the CMV related damage to the stria vascularis we also see degeneration of the capillaries that supply the organ of Corti, and the spiral ganglion region of the cochlear modiolus. These are the vessels of the spiral limbus, and in the normal subject they are arranged as shown in the left hand Fig below. On the right-hand panel we can see the effects of CMV infection.

The average hearing loss in the CMV infected subjects compared to controls is shown in the graphs below. These comparisons are made between six control subjects (12 ears) and six CMV infected animals.

This study is the first to reveal that the initial effect of CMV infection is on the cochlear vasculature, specifically the stria vascularis. It is well known that the strial
Fig 1. The blood capillary networks of the normal (un-infected) mouse cochlea. Corrosion cast specimen Fig. using scanning electron microscopy. On the outer wall of the cochlea there are two separate vascular beds. The outer vessels (vertically aligned here) make up the spiral ligament, and inside (behind) are the horizontally aligned capillaries of the stria vascularis. Scale bar=xxx.

Fig 2. Degeneration of stria vascularis in the apical region of the cochlea after CMV infection.

Fig 3. Extensive damage to cochlear vasculature caused by CMV infection.

EXPERIMENTAL PROTOCOL
Balb/c mice inoculated 3 days postnatally with murine cytomegalovirus
(right) intra-cerebral injection (2000 pfu in 1 ul)
At 4 weeks, auditory function tested with ABR and OAE measurements
At 8 weeks, vascular damage evaluated using partial corrosion cast and SEM microscopy
mechanisms are important for maintaining the endolymphatic (endocochlear) potential (EP) that, in turn, powers haircell transduction mechanisms. We suggest that early stage vascular damage can cause changes to the EP that will manifest as a mild to moderate hearing loss. If the extent of vascular damage is not extensive, there is a possibility of some recovery, as is sometimes the case with other causes of hearing loss that temporarily cause strial dysfunction (e.g. ototoxic diuretics).
such as Lasix). Importantly we have noted in our experiments some signs of regeneration (angiogenesis) of strial capillaries after CMV damage.

What does this mean for clinical application? There are still many aspects of CMV related hearing loss that we do not fully understand, indeed many more questions than answers. For example how does the virus migrate from the brain to the inner ear? How can we detect this inner ear involvement early on? How exactly does CMV damage to the strial vessels? Is there involvement of reactive oxygen species (ROS) in causing vessel damage? Can this be prevented? After degeneration of strial capillaries, is there a way of promoting a regeneration of the vasculature? These are all questions that we might address in the future using an animal model such as we have now described here.

REFERENCES
Hearing Loss in Infants

Mădălina Georgescu

“Blindness separates humans from things,
Deafness separates humans from humans.”

Helen Keller

Congenital deafness, together with other sensorial impairments has an important negative impact on child’s development. The severity of the hearing impairment, especially if not habilitated through surgery or hearing aids, shapes the child in many aspects. Regarding hearing aspect, a child can be anywhere between deaf and mute and bad pronunciation. But this deficit is not alone – it is associated with integration difficulties in kindergarten and at school, limited academic development, frustration and lack of self-esteem.

Acquired bilateral hearing loss does not influence dramatically speech, but has an important influence on hearing impaired person’s social life, since communication difficulties leads to isolation and emotional disturbances. Almost 10% of the population has some degree of hearing loss and needs appropriate treatment and/or rehabilitation.

Due to this large number of persons with hearing loss, hearing loss is a public health issue which requires specific health politics in order to allow access to each patient to standard medical services. This can be offered through following:

- Mandatory universal new-born hearing screening
- Follow-up program for hearing impaired children up to school age
- Hearing screening in preschool and school-age children
- National register of hearing impaired persons
- Educational programs in Audiology, to have enough trained audiologist for the large number of patients with permanent hearing loss

Two main categories of childhood hearing loss are considered – prelingually and post lingually hearing loss.

Prelingually hearing loss is mostly congenital, being the most frequently congenital deficiency (1-3% of alive new borns). Deafness is an invisible handicap and for this reason active detection through new born hearing screening programs should be promoted and implemented. New born hearing screening programs are the only solution for early detection of hearing loss. Infants who do not pass the screening test should be referred to an audiological diagnostic centre for certain diagnosis of hearing loss and quantification of the impairment.

Hearing screening must be universal, to cover all new born, since 50% of children with congenital hearing loss has no risk factors for hearing loss. In 2007, Join Committee on Infant Hearing defined risk factors for hearing loss:

- Prenatal period
  - Hereditary aetiology
  - Genetic disorders (Connexine 26 mutation)
  - Pregnancy evolution
  - Maternal infections during pregnancy or delivery (Toxoplasmosis, Syphilis, HIV, Hepatitis B, Rubella, CMV, Herpes simplex, and others)

- Neonatal period
  - Intoxications (drugs, alcohol)
  - Birth condition (hypoxia)
  - Prematurity (less 37 weeks)
  - Low birth weight (less 1500 g)
  - Cardio-respiratory distress (mechanical ventilation more than seven days)
  - NICU admission more than five days
  - Hyperbilirubinemia
  - Syndrome associated with hearing loss (Pendred, Usher, Waardenburg, neurofibromatosis)
  - Physical problems of the head, face, ears, or neck (cleft lip/ palate, ear pits/tags, atresia, and others)
  - Ototoxic medications given in the neonatal period (one or more aminoglycosides antibiotics, loop diuretics associated with aminoglycosides antibiotics)
  - Infections - bacterial meningitis and other infections (mumps, encephalitis, viral labyrinthitis)

New born hearing screening is the cheapest birth screening. It is a non-invasive, simple, short method. Appropriate medical device is needed and 2 to 3 instructed persons – coordinator physician and maternal-ward nurses.

“Carol Davila” University of Medicine and Pharmacy Bucharest, Romania
Post lingually hearing loss defines hearing loss with onset after speech development. It is an acquired hearing loss, most frequently during small childhood. Incidence of this type of hearing loss is 10 times larger (3-5% of 3 to 5 years old children) than the incidence of congenital hearing loss, but its severity is smaller than the severity of the congenital hearing loss. The later one is characterised by bilateral deafness in most cases. Bilateral hearing loss, even mild one, impedes on school progress of hearing impaired children, induces greater tiredness for school activities and affects children’s social relations with their school mates.

Appropriate management of hearing impaired child includes early detection of hearing loss associated with early appropriate treatment. For permanent bilateral hearing loss, conventional or implantable hearing aids are the only solution for auditory habilitation of the deaf child. Quality of speech and language measures the benefit of the hearing aid. Early treatment of the hearing loss with specific speech therapy leads to correct speech and language development, like normal hearing children one.

This achievement is the result of cerebral neuroplasticity property (cortical remapping), a process in which cortical areas modifies through experience. This “compliance” of the brain is correlated with learning processes through of adding or removal of connections. Cortical plasticity is time-dependent, with maximum capacity in the first one and a half-two years of life. This opportunity window cannot be missed for best management of the deaf child.

Late auditory habilitation has limited benefits on child’s pronunciation skills or even worse, no benefit, if cochlear implantation is provided after age of six. The child will still be mute and deaf if no auditory stimulation was provided until this age. In this case, cochlear implant will deliver information in an auditory cortex already organised, but took over by visual system and stimulation of the auditory pathway will not finalise in audition as final sensation.

For infants, the standard health services include hearing screening test until age of one month, hearing loss diagnosis until age of three-month-old and treatment onset until age of six month. This is the best strategy we should aim to help efficiently children with congenital or first month acquired hearing loss.
Efficacy of Implantable Devices for Conductive and Mixed Hearing Loss

By Ad Snik*

INTRODUCTION
Nowadays, several types of conventional and implantable amplification options are available for patients with conductive or mixed hearing loss. Table 1 presents an overview. Implantable devices are being developed because the conventional devices (behind-the-ear (BTE) devices and conventional bone conductors applied with headband or soft band) might fail for various reasons [1]. Besides implantable bone-conduction devices (percutaneous bone-conduction implants, passive and active transcutaneous bone-conduction implants), middle-ear implants can be applied with their actuator coupled to one of the cochlear windows. Such implantable devices have often been launched with enthusiasm while well-documented scientific and clinical data were not available. So, the ‘market’ (implant centres) had to find out whether or not these devices were more effective than existing devices and whether or not these implants were stable over time. This ‘let-the-market-decide’ is time consuming; mostly the search is not carried out systematically and it might result in non-optimally treated patients.

From systematic reviews of the literature it has been concluded repeatedly that evidence level of most published studies is weak and the overall result is not convincing [3,4]. So, the question remains: do we have good evidence to choose between hearing solutions for a given patient? Consensus is lacking while the devices are not equivalent in terms auditory capacity and efficacy, invasiveness and complexity of implant surgery, stability over time, MRI compatibility, costs, etc. [5].

THE USE OF A WEBSITE
As a first step to obtain consensus, objective data was gathered and published on a website; the website was developed by the author for professionals (http://www.snikimplants.nl). Subjective (questionnaire) data were not taken into account because such data is easily biased [6], especially when applying new technology [7].

The website format is chosen as websites are easily accessible and they can be updated e.g. when new information on the efficacy of implantable devices becomes available.

The first objective measure discussed on the website is the capacity of the different types of implantable devices. Following [8], the maximum output (MPO) was measured while the devices were programmed in linear amplification mode [9,5]. Next, the MPO was used to define inclusion criterion for the application of each device in terms of the maximum auditory capacity and efficacy.

Table 1. Types of conventional and implantable devices for conductive and mixed hearing loss.

<table>
<thead>
<tr>
<th>Device for conductive or mixed hearing loss</th>
<th>Manufacturer</th>
<th>Indication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Behind-the-ear</td>
<td>Several</td>
<td>Dry ear, normal ear canal and air-bone gap &lt;40 dBHL*</td>
</tr>
<tr>
<td>Conventional bone-conduction device (BCD)</td>
<td>All BCDs applied with softband or steel headband</td>
<td>Running ear or aural atresia</td>
</tr>
<tr>
<td>Percutaneous bone-conduction implant</td>
<td>Baha (Cochlear) Ponto (Oticon)</td>
<td>Idem, also used as CROS device in single-sided deafness</td>
</tr>
<tr>
<td>Transcutaneous bone-conduction implant</td>
<td>Baha Attract (Cochlear) Sophono (Medtronic)</td>
<td>Running ear or aural atresia</td>
</tr>
<tr>
<td>Active transcutaneous bone-conduction implant</td>
<td>Bonebridge (Med-El)</td>
<td>Idem, also used as CROS device</td>
</tr>
<tr>
<td>Middle ear implant</td>
<td>Vibrant Soundbridge (Med-El) and MET (Cochlear)</td>
<td>Infection-free middle ear; no severe middle ear anomalies</td>
</tr>
</tbody>
</table>

*de Wolf et al. [2]

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allowable sensorineural hearing loss component.

Table 2 presents the mean MPO, expressed in dB HL, of all the implantable devices that were introduced in Table 1. As Table 2 shows, the MPO of most devices is limited, obviously below loudness discomfort levels (LDL) of patients; considered at the cochlear level, LDL levels will be found between 90 and 110 dB HL [10]. Note that all the introduced implantable devices stimulate the cochlea, bypassing the impaired middle ear. The ‘dynamic range of hearing’ is by definition the difference between the cochlear thresholds and LDL. When using a device with limited MPO, the upper part of this ‘dynamic range of hearing’ (LDL-MPO) cannot be addressed. So, the higher the MPO of the device is, the wider the aided ‘dynamic range of hearing. Only if the MPO level coincides with LDL, full use can be made of the patient’s ‘dynamic range of hearing. For proper application of today’s implantable devices with their limited MPO, some compromise is needed concerning a just acceptable ‘dynamic range of hearing. The suggested, rather arbitrary compromise is the following: a specific device should only be applied if the dynamic range is at least 35 dB (width of the ‘speech area’ or ‘speech banana; [5] while the ‘lost’ dynamic range (LDL-MPO) is less than 1/3 of the total ‘dynamic range of hearing’ (MPO - cochlear threshold; named the 2/3 rule [5]. Using such a compromise, the implantable devices can be categorized; the maximum allowable cochlear hearing loss component can now be calculated, see Table 2, last column.

These maximum values can be used when counselling patients. Longevity is directly related to such values. To illustrate this, assume that the progression in hearing loss is known. Then longevity can be assessed. Fig. 1 shows an example, taken from [5] chapter 3. The data suggest that in this case (OTSC 7 patients) the percutaneous bone conductor and the Vibrant Soundbridge can be used life long. Another important and rather objective measure is implant stability. A straightforward measure dealing with stability is the number of revision surgeries related to follow-up. According to the Swiss national database, on the average, revision surgery in patients with a cochlear implant occurs once in 30 years of follow-up, personal communication. Only for the percutaneous Baha, long-term stability data have been published. Using the adults’ results published by [11] a similar revision rate was calculated. Preliminary data showed that the revision rate for the percutaneous Baha is improving owing to new implant technology and surgical approaches while the preliminary revision rate for middle ear implant applications is still lagging behind [5], chapter 4. Definitely, more data on stability issues should be published.

Table 2. The mean MPO determined objectively of the mentioned devices. The maximum allowable sensorineural hearing loss component for proper application, according to the 2/3 rule, is presented in the third column.

<table>
<thead>
<tr>
<th>Device</th>
<th>MPO dB HL</th>
<th>SNHL component:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sophono Alpha 1-2</td>
<td>53</td>
<td>&lt;5</td>
</tr>
<tr>
<td>Baha Attract with BP110</td>
<td>63</td>
<td>15</td>
</tr>
<tr>
<td>Bonebridge</td>
<td>67</td>
<td>20-25</td>
</tr>
<tr>
<td>Baha/Ponto standard</td>
<td>67-69</td>
<td>25-30</td>
</tr>
<tr>
<td>BP110, Ponto power</td>
<td>74-76</td>
<td>35-40</td>
</tr>
<tr>
<td>Cordelle, Ponto plus</td>
<td>78-80</td>
<td>50</td>
</tr>
<tr>
<td>VSB</td>
<td>85</td>
<td>50-55</td>
</tr>
</tbody>
</table>

Patient with otosclerosis OTSC7

<table>
<thead>
<tr>
<th>Age</th>
<th>SNHL</th>
<th>Sophono, etc</th>
<th>Baha*</th>
<th>VSB</th>
<th>Bonebridge</th>
</tr>
</thead>
<tbody>
<tr>
<td>30 yrs</td>
<td>24dB</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>40</td>
<td>28</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>50</td>
<td>33</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>60</td>
<td>36</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>70</td>
<td>42e</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>80</td>
<td>47e</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Fig 1. Deterioration of the mean sensorineural hearing loss component of patients with otosclerosis type OTSC7 [12]. The second row presents the mean sensorineural hearing loss component (row labelled SNHL) as a function of age (row labelled Age). In the next rows (labelled Sophono, Baha, VSB, Bonebridge) the red line indicates whether the indicated device can be used, based on the maximum allowable sensorineural hearing loss component, taken from Table 2. VSB stands for Vibrant Soundbridge.
In summary, in order to categorize the capacity and stability of hearing devices for conductive and mixed hearing loss, a website for professionals was developed based on new data and published objective data. Based on comments by professionals in the field, the website has been updated several times (for the history of the website, see Appendix 3). The analyses presented on the website can be considered as a starting point for professionals counselling patients.

During recent years, the role of the patient in the selection of rehabilitation options becomes more and more acknowledged. ‘Patient-centred-health-care’ should be based upon specific outcome measures as should be defined together with patients [13]. Next, such outcome measures should be systematically studied and reviewed to optimise counselling of patients.

REFERENCES
5. http://www.snikimplants.nl
Research Evidence Supporting Progressive Tinnitus Management

By James A. Henry

Tinnitus is the perception of sound that has no source outside of the head. Tinnitus is most typically associated with exposure to loud noise, which can also cause hearing loss [1,2]. A direct correlation exists between degree of hearing loss and prevalence of tinnitus—the likelihood of incurring tinnitus increases with a greater degree of hearing loss [3]. In general, tinnitus can occur as the result of noise damage, blast exposures, head and neck trauma or pathology, drugs or medications, and other medical conditions (e.g., acoustic neuroma, cardiovascular and cerebrovascular disease, hyper- and hypothyroidism) [4,5].

Evidence-based research should guide the clinical management of tinnitus. Randomized controlled trials (RCTs) that are properly conducted are the most important source for providing such evidence [6]. Recently, evidence-based guidelines for tinnitus management became available from the American Academy of Otolaryngology – Head & Neck Surgery Foundation (AAO-HNSF) [7]. Developing their Clinical Practice Guideline (CPG) relied mostly on searching the peer-reviewed literature and identifying relevant RCTs. The AAO-HNSF assembled a 23-member committee to develop the guidelines, which underwent external peer review prior to publication. The AAO-HNSF tinnitus CPG is currently the most comprehensive guide to providing evidence-based clinical services for tinnitus.

The AAO-HNSF CPG recommends: (1) a case history and physical exam by an otolaryngologist; (2) a comprehensive audiologic exam if: the tinnitus is “persistent” (i.e., present for at least 6 months), unilateral, or accompanied by hearing difficulties; (3) determining if the tinnitus is bothersome or no bothersome. For patients with persistent, bothersome tinnitus, the CPG recommends: (1) provide information about realistic treatment options; (2) perform a hearing aid evaluation as appropriate; and (3) suggest treatment with Cognitive Behavioral Therapy (CBT).

The CPG acknowledges the value of “sound therapy” for tinnitus (and there are many forms of sound therapy); however, they only recommend sound therapy as “optional” due to the relative paucity of RCTs verifying its clinical effectiveness. Sound therapy is an essential component of treatment with Progressive Tinnitus Management (PTM). The approach with PTM, however, is to inform patients about how sound can be used therapeutically and how to determine which type of sound might be effective in each tinnitus-problem situation that is experienced [8-18]. PTM does not advocate any particular type of sound or sound-delivery device. The objective is to empower patients so that they can make informed decisions regarding the use of sound as therapy. This information is combined with CBT, which is provided as part of the intervention with PTM.

At the National Center for Rehabilitative Auditory Research (NCRAR) tinnitus research has been ongoing since it was established in 1997. This research has consistently focused on developing and testing components of tinnitus clinical management. Numerous clinical trials have helped to identify procedures that are most effective for clinical application. The culmination of this research has been the development of PTM.

PTM is a stepped-care program for all patients who report tinnitus (Fig. 1). Each step involves assessment and/or intervention to identify and address needs related to hearing loss, tinnitus, and reduced tolerance to sound (hyperacusis). Throughout the various levels of PTM, as needs are identified, the patient and clinician collaboratively decide on the next appropriate course of action. The degree of services received by patients align with their individual needs.

Beyond the initial referral level (Level 1 Referral), the first PTM step (Level 2 Audiologic Evaluation) is a traditional audiologic evaluation with the addition of a 10-item survey to assess the functional effects of tinnitus and to screen for hyperacusis [19]. In rare cases hyperacusis may need to be resolved before hearing problems or tinnitus can be addressed. Patients who are hearing aid candidates are fit with hearing aids or combination devices (amplification and sound generator combined in one unit) to address their hearing loss, which often mitigates bothersome tinnitus [20,21]. After hearing loss and hyperacusis needs have been addressed, patients who require assistance for bothersome tinnitus are offered Level 3 Skills Education.

Level 3 Skills Education is normally provided as five weekly meetings (in group
or individual settings)—two taught by an Audiologist and three taught by a Mental Health (MH) Provider who has expertise in CBT. During the meetings, patients learn different strategies for using sound and CBT-based coping skills to improve their quality of life [11]. The intended outcomes of learning and using the skills that are taught include reduced distress from tinnitus and improved confidence in the ability to self-manage tinnitus.

The relatively few patients who are still significantly bothered by their tinnitus following Level 3 are advised to undergo a Level 4 Interdisciplinary Evaluation. Level 4 provides an in-depth assessment conducted by an Audiologist and a Psychologist leading to an informed and collaborative decision as to whether to initiate Level 5 Individualized Support. Level 5 involves personalized and ongoing meetings with the Audiologist and/or the Psychologist to incorporate the skills taught at Level 3 into daily life, with modifications as needed to meet the needs and interests of the individual being served.

Whereas the AAO-HNSF CPG recommends a medical exam for every patient, PTM provides referral criteria as part of the assessment during Levels 1 and 2 [10]. Clinicians must also be attentive for unaddressed MH conditions, and to refer for MH screening if such conditions are suspected. Consistent with the AAO-HNSF CPG, medications should not be used specifically for tinnitus, although they would be appropriate if prescribed by a physician for MH symptoms.

Cumulative evidence for PTM consists of: (1) over 20 years of research involving 25 funded projects; (2) clinical implementation at Audiology clinics—PTM is being utilized in one form or another by over 100 clinics; (3) a proof-of-concept study evaluating telephone-based PTM [14]; and (4) two RCTs of PTM that were recently completed (and which are described briefly below).

The first RCT was a two-site study conducted at the Memphis, Tennessee and West Haven, Connecticut Veterans Affairs (VA) hospitals. The purpose was to evaluate the effectiveness of PTM Level 3 Skills Education compared to Wait List Control (WLC) [22]. Three hundred military Veterans (150 at each VA) with bothersome tinnitus who desired treatment were enrolled as participants. Results suggest that PTM is effective at reducing tinnitus-related functional distress when embedded into VA clinical settings. Although effect sizes were modest, they provide evidence of the effectiveness of PTM when it is provided in a clinical setting.

The second RCT of PTM (briefly described in [8] full publication in preparation) followed our pilot study that suggested efficacy of telephone-based PTM [14]. For the RCT, telephone-based PTM Skills Education was evaluated for efficacy compared to WLC. Participants (N=205) were both Veterans and non-Veterans with bothersome tinnitus who were enrolled from around the country. The intervention protocol consisted of five telephone sessions—three with a Psychologist and two with an Audiologist (to correspond with the five sessions that are normally offered in-clinic) in addition to two follow-up calls. Outcomes were assessed at baseline and at 3, 6, 9, and 12 months, using the Tinnitus Functional Index (TFI; [23] as the primary outcome instrument and the Tinnitus Handicap Inventory (THI) [24] as the secondary outcome instrument. At 6 months, improvement on the TFI was about 20 points greater for the tele-PTM group relative to the control group, and the improvement was sustained for another 6 months. The TFI and THI change scores were strongly and linearly related (Pearson’s correlation=0.69; p<0.0001), emphasizing the similarity between these two outcome instruments.

The TFI contains eight subscales: Auditory, Cognitive, Emotional, Intrusive, Quality of Life (QOL), Relaxation, Sense of Control, and Sleep. All but one of the subscales contains three items—the QOL subscale contains four items. This...
second RCT provided data showing substantial differences between subscales, ranging from a 13.2-point reduction for the Auditory subscale to a 26.7-point reduction for the Relaxation subscale [8]. These subscale data reveal that the telephone intervention had the largest effect on the Relaxation domain and the smallest effect on the auditory domain. Much more can be said about the subscale data – the takeaway point is that subscale scores can be informative as to which functional areas are most affected by a person's tinnitus, and which are most impacted by the intervention.

These recent RCTs support the clinical utilization of PTM as an evidence-based method of tinnitus management. Results of these studies were not available when the AAO-HNSF CPG was developed. PTM is mostly consistent with recommendations of the AAO-HNSF CPG, and provides specific methodology for the clinical management of tinnitus by Audiologists and MH Providers.

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5. from http://www.tinnitusarchive.org/


