

Most cases of tinnitus are caused by changes in central auditory structures consequent on hearing impairment expressed in the audiogram or more sensitive measures. Compensation for these impairments requires knowledge of how the impairments are expressed, tools for their assessment, and, in our approach, interface of the tools with therapeutic devices that aim to normalize impaired hearing function in affected individuals. Baseline population data are required to make individual measurements informative. We are adapting computer-based, subject-driven tools that were designed initially to measure psychoacoustic properties relevant to understanding the mechanism of tinnitus (Roberts et al., 2008), for use in tinnitus assessment in the clinic. Measurements of peripheral hearing function and of loudness growth for individual patients are subsequently used by an adaptive hearing aid algorithm to restore a more normal pattern of auditory neural activity for individuals experiencing tinnitus with or without audiometric threshold shift. Collaborators in the ongoing project are S. Hébert (Université de Montréal), D. Purcell (University of Western Ontario), and L. Parra (City College of New York).

1. Current Tools and Platform*



Powermate Adaptor (Griffin Technologies)

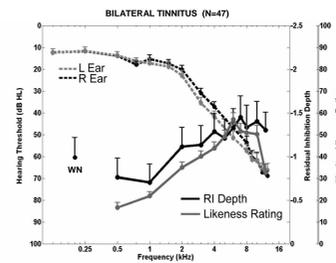


Figure 1

Version 1 2008:

Version 1 tools (described by Roberts et al., 2008) consisted of a “Familiarization Program” that introduced the subject to the test environment, a “Tinnitus Tester”, and a “Residual Inhibition Tester”. The tools provided a controlled environment for subject-driven assessments of properties of tinnitus relevant to understanding the neural basis of tinnitus sounds including tinnitus ear, tinnitus bandwidth, temporal variability (steady or pulsing), tinnitus loudness matching, tinnitus frequency spectrum, and residual inhibition for band limited maskers differing in center frequency (see Figure 1).

Version 2 2010:

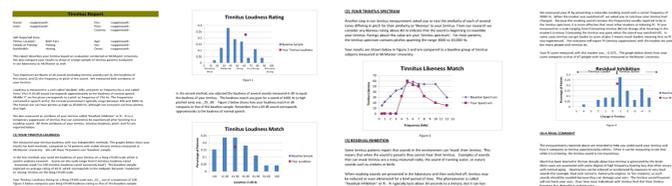
This version added the following:

- (1) An embedded training program to familiarize the subject with the test environment and give experience with variable loudness (pitch constant) and variable pitch (loudness constant) upgraded from version 1;
- (2) Internal checks anticipating common subject errors and re-start options for the clinical user;
- (3) A new, brief test for residual inhibition;
- (4) The option of printing a hard copy report of the patient’s results suitable for use by clinicians or the patient.

The report (example below) explains the measurements that were taken, references the patient’s results to baseline data, and presents some general information about tinnitus. It enables the patient to “see” their tinnitus in relation to the experience of other others with tinnitus, and may itself have therapeutic value (a question under study);

- (5) Software and hard copy report are available in English or French language versions.

Version 2 is being used by the Montreal Tinnitus Clinic which is providing additional baseline data. (<http://www.mytinnitus.ca/>)



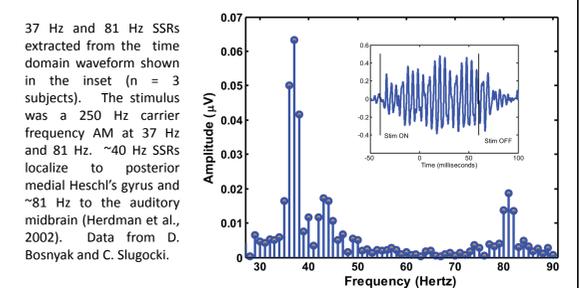
Platform Concept:

Our concept is an eventual “Microsoft Office” for tinnitus, adding new modules in a common platform, upgrading existing ones, and providing baseline data for each to support standardized tinnitus assessment. In addition to clinical utility the approach may provide a framework for evaluation of therapeutic interventions. One module under development assesses hearing thresholds to 20 kHz to obviate the need for separate conventional audiometric testing. Another module assesses loudness growth (see next panel).

2. Loudness Growth

Schaette and McAlpine (2011) have shown that wave 1 of the ABR evoked by 90-100 dB clicks is reduced in tinnitus compared to control subjects (both with normal audiograms), but wave 5 is not. These results suggest that following cochlear damage to high threshold auditory nerve fibers (this damage not expressed in the audiogram; Kajawa and Liberman, 2009), homeostatic mechanisms preserve net excitation expressed in subcortical auditory structures. However, loudness growth measured behaviorally in tinnitus patients with normal audiograms (Fournier and Hébert, 2011) and cortically in an animal model of tinnitus (Engineer et al., 2010) is *increased* compared to controls, implying increased gain in auditory pathways which may be consequent on loss of inhibition and/or map reorganization in central auditory structures. These reports underscore the importance of measuring loudness growth in tinnitus patients, which may reveal hearing impairment not reflected by other methods. Such measures also may have application to treatment interventions in hearing loss, tinnitus, and hyperacusis (next box).

New tools currently under development and testing are measuring loudness growth behaviorally and electrophysiologically in tinnitus subjects and in age and threshold matched controls. Loudness growth measured by ABR wave 1 (reflecting output from the cochlea) is compared to loudness growth measured by 37 Hz and 81 Hz steady-state responses (SSRs) evoked by a simultaneous 37/81 Hz AM sound (reflecting activations in cortical and subcortical auditory structures, respectively). The ratio of loudness-growth slope for frequencies in the tinnitus frequency region (5 kHz and 8 kHz) to 250 Hz in the range of normal thresholds may have diagnostic value for hearing impairment. The measurements may also reveal at which levels of central auditory pathways increased gain is occurring.

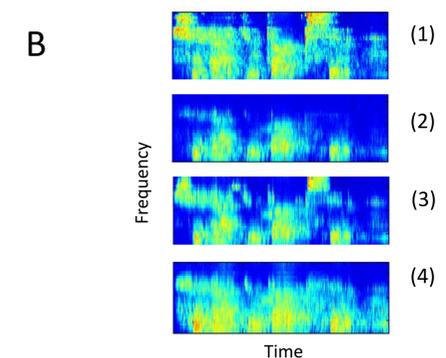
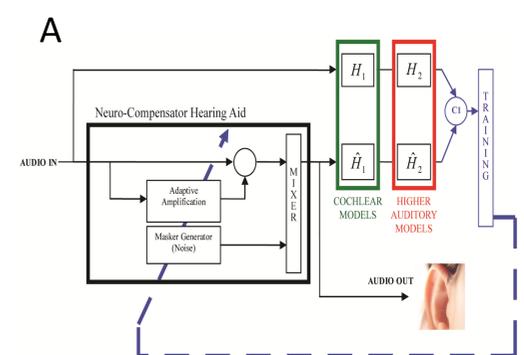


3. NeuroCompensator

Hearing impairment sets into motion processes that may eventuate in tinnitus and (in many if not most cases) increased loudness growth. While the prospect of tertiary treatments for tinnitus is alluring albeit presently unsolved, restoration of normative activity in auditory pathways after cochlear damage and changes in central gain is an alternative approach that may be beneficial in alleviating tinnitus and preventing entrenchment of pathological functional and structural adaptations in early detected cases. In our approach individual measurements of cochlear function (thresholds, DPOAE) and central gain changes (loudness growth) are used by a novel hearing aid algorithm that employs machine learning to normalize the pattern of activity in auditory pathways following cochlear damage and altered central gain (Cochlear and Higher Auditory Models respectively, see panel A). The aim is to produce a more normal pattern of neural activity in central auditory structures in individuals suffering tinnitus, hyperacusis, or hearing loss.

In its present implementation the algorithm (called the NeuroCompensator) employs a biologically realistic model of the auditory periphery (Zilany & Bruce et al. 2009) to generate patterns of auditory nerve activity from the normal and damaged cochlea (Cochlear Models in panel A). Machine learning adjusts the amplification profile so that sound delivered to the damaged ear (\hat{H}_1 , panel A) generates a neural output approximating that of the normal ear (H_1 ; see the neurograms of panel B). The NeuroCompensator algorithm has been patented by Haykin et al. (2006) and licensed by VitaSound Audio Inc. for implementation in audiology clinics. At present the return rate by purchasers has been reported to be half that of conventional wide dynamic range compression algorithms. Laboratory assessments of hearing improvement are presently underway.

In a next implementation models of central auditory processing (H_2, \hat{H}_2 , in panel A) based on Parra and Perlmutter (2007) and Chrostowski et al. (2011) will be added to the Neurocompensator and used to generate a pattern of auditory nerve activity yielding normal loudness growth in individuals with tinnitus and cochlear damage. Benefits for tinnitus, improved hearing in noise, and hyperacusis will be assessed. This approach may also have value in optimizing residual inhibition in tinnitus subjects, which while showing a clear central tendency (Figure 1) is highly variable.



Neurograms of auditory nerve activity generated by a spoken sentence (spike rates proportional to warm colors). (1) Healthy cochlea; (2) Impaired cochlea; (3) Impaired cochlea after NeuroCompensation – high frequency edges are restored and acoustic segregation preserved; (4) Impaired cochlea after conventional wide dynamic range compression.

References: Chrostowski M, Yang L, Wilson HR, Bruce IC & Becker S (2011) *J Comput Neurosci* 30(2):279–299; CT Engineer, CA Perez, YH Chen, RS Carraway, AC Reed, JA Shetake, VJ, KQ Chang & MP Kilgard (2010) *Nature Neuroscience* 11: 603-608; Fournier P & Hébert S (2011) *In Proc 5th Int Tinnitus Research Initiative Conf, Buffalo, NY*; Haykin, S., Becker, S., Bruce, I., Bondy, J., Trainor, L., and Racine, R. J. (2006) *Binaural adaptive hearing aid*. United States Patent No. 7,149,320; Herdman AT, Lins O, Van Roon P, Stapells DR, Scherg M & Picton TW (2002) *Brain Topography* 15(2):69–86; Kujawa SG & Liberman MC (2009) *J Neurosci* 29(45):14077–14085; Parra LC and Pearlmutt B (2007) *J Acoust Soc Am* 121(3):1632–1641; Roberts LE, Moffat G, Baumann, M, Ward LM, and Bosnyak DJ. (2008) *J Association Research Otolaryngology*, 9:417-435. Schaette R & McAlpine D (2011) *J Neurosci* 31(38):13452–13457; Zilany MSA, Bruce IC, Nelson PC & Carney LH (2009) *J Acoust Soc Am* 126(5):2390–2412. **Acknowledgements:** NSERC and CIHR of Canada; Tinnitus Research Initiative; American Tinnitus Association