

PROGRAM and ABSTRACTS

of the

One Hundred Forty-First Annual Meeting

AMERICAN OTOLOGICAL SOCIETY, INC.

May 2-3, 2008 Mediterranean Ballroom Salon 4

JW Marriott Grande Lakes Resort Orlando, Florida

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Accreditation Statement: The American Otological Society (AOS) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. The AOS takes responsibility for the content, quality, and scientific integrity of this CME activity.

Credit Statement:

The American Otological Society designates this educational activity for a maximum of 8 AMA PRA Category 1 Credit(s)TM. Physicians should only claim credit commensurate with the extent of their participation in the activity.

Certificate of Attendance will be issued at the close of the meeting upon completion of the questionnaire required by us for the certifying organizations.

AMERICAN OTOLOGICAL SOCIETY, INC. MISSION STATEMENT

The mission of the American Otological Society, Inc., shall be

- to advance and promote medical and surgical otology/ neurotology and lateral skull base surgery in adult and pediatric patients including the rehabilitation of individuals with hearing and balance disorders.
- to encourage, promote, and sponsor research in otology/neurotology and lateral skull base surgery and related disciplines.
- to conduct an annual meeting of the members for the presentation and discussion of scientific papers and the transaction of business affairs of the Society.
- to publish the peer reviewed papers and discussions presented during the scientific program and the proceedings of the business meetings.

EDUCATIONAL MISSION STATEMENT

The Educational Mission of the American Otological Society is to foster dialogue on, and dissemination of information pertaining to advances in the understanding and management of otologic and neurotologic disorders. It is expected that the CME program of the AOS will enhance the competency of the participant in otology/neurotology and lateral skull base surgery.

Goals & Objectives: The overall goal of this course is to provide up-to-date information pertaining to advances in the understanding and management of otologic and neurotologic disorders. The target audiences are otologists, neurotologists, and otolaryngologists with specific interests in otologic and neurotologic disorders.

After attending this meeting, the participants will have a better understanding of:

Implantable Hearing Devices

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Advances in Cochlear Implants

Surgical Treatment of Disorders of Eustachian Tube Function

resulting in more effective patient care to achieve improved hearing.

First Author/Presenter's signature on the following statements were required on all papers submitted to the American Otological Society. The author was advised that the submitted paper becomes the property of *Otology & Neurotology* and cannot be reprinted without permission of the Journal.

FULL DISCLOSURE POLICY STATEMENT

In accordance with the ACCME Essential Areas and Policies. it is the policy of the American Otological Society to ensure balance, independence, objectivity and scientific rigor in all of its educational activities. All faculty participating in the American Otological Society's sponsored activities are expected to disclose to the audience the existence of any significant financial or other relationships with the manufacturer(s) of any commercial product(s) or provider(s) of any commercial service(s) discussed in an educational presentation. The purpose of this form is to identify and resolve all potential conflicts of interests that arise from financial relationships with any commercial or proprietary entity that produces healthcare-related products and/or services relevant to the content you are planning, developing, or presenting for this activity. This includes any financial relationships within the last twelve months, as well as known financial relationships of your spouse or partner. The intent of this policy is not to discourage speakers who have relationships with commercial entities from presenting, but to identify these relationships to the listeners so that they may form their own judgments. It remains for the audience to determine whether the speaker's outside interest may reflect a possible bias in either the exposition or the conclusions presented. Failure to disclose this information on submission forms, or failure to return this disclosure form will result in exclusion from this activity and from future CME activities for up to two years. The American Otological Society is committed to the non-promotional advancement of knowledge and science and to a free exchange of medical education in otology and neurotology.

PUBLICATION STATEMENT

The material in this abstract, (<u>Name of Abstract</u>), has not been submitted for publication, published, nor presented previously at another national or international meeting and is not under any consideration for presentation at another national or international meeting including another COSM society. The penalty for duplicate presentation/publication is prohibition of the author and co-authors from presenting at a COSM society meeting for a period of three years.

Submitting Author's Signature (required)

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Jay Rubinstein, MD Advanced Bionics – Consultant, Research Funding Cochlear Corp – Consultant, Research Funding

P. Ashley Wackym, MD Advanced Bionics – Consulting fees donated to Medical School, Advisory Board Cochlear Americas – Consulting fees donated to Medical School, Advisory Board

John Dornhoffer, MD - Nothing to disclose

John T. McElveen, Jr., MD - Nothing to disclose

Miles Pensak, MD - Nothing to disclose

Debara L. Tucci, MD - Nothing to disclose

***Disclosures—Oral Presentations

Douglas D. Backous, MD Cochlear Corporation- Surgical Advisory Board Medtronic - Neurotechnology- Consultant

Sujana Chandrasekhar, MD Scientific Development & Research, Inc.—Own shares, Board Member, Patent holder for intranasal surfactant

H. Ric Harnsberger, MD Amirsys, Inc—CEO, shareholder

Sunil Puria, PhD Earlens Corp—Chief Scientist

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Joseph B. Roberson, Jr., MD Cochlear Americas- Advisory Board Member, Consultant Advanced Bionics- Advisory Board Member, Consultant Medtronic- Consultant, Business Development Acclarent- Consultant, Business Development

J. Thomas Roland, Jr., MD Advanced Bionics - Advisory Board Cochlear Americas - Advisory Board

Michael Seidman, MD National Institute of Health-NCCAM- Grant (Resveratrol & hearing loss) Visalus Sciences- Director, Product Development Body Language Vitamin Co- CEO (company to be sold) (Design nutritional supplements that have high content of Resveratrol and other nutrients with potential health benefits)

Olivier Sterkers, MD Medtronic – Consultant, Nerve Monitoring

Thomas R. Van De Water, PhD Advanced Bionics Corp - Research grant (otoprotection)

Jack J. Wazen, MD Cochlear Corp - Consultant, Research Yuri Agrawal, MD Charles D. Bluestone, MD Craig A. Buchman MD Chul-Hee, Choi, PhD Daniel H. Coelho, MD Vittorio Colletti, MD Sharon L. Cushing, MD Rose J. Eapen, MD Howard W. Francis, MD Michael H. Fritsch, MD Yoav Hahn, MD Barry Hirsch, MD Yukiko Iino, MD Herman A. Jenkins, MD Shin-ichi Kanemaru MD, PhD Annerose M. Keilmann, Prof. Dr. med. Richard D. Kopke, MD Arvind Kumar, MD Robert F. Labadie, MD, PhD Xi Lin, PhD Fred H. Linthicum Jr., MD Charles M. Luetje, MD Charles A. Mangham, Jr., MD Elias Michaelides, MD Yasuo Mishiro, MD John K. Niparko, MD Joseph B. Nadol, Jr., MD Dennis Poe, MD Richard D. Rabbitt, PhD Masafumi Sakagami, MD, PhD James E. Saunders, MD Majestic Tam, MD

*** American Otological Society

Any presentations, conversations, exhibits, or other meeting communications, including description of the use of drugs or devices, does not imply nor constitute endorsement of any company, product, application or use by the American Otological Society.

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Friday, May 2, 2008

- 12:00 Registration
- 12:30 Business Meeting (Restricted to Members) Room: Mediterranean Ballroom - Salon 4

Minutes of the Annual Meeting 2007

Introduction of New Members

Election of Nominating Committee

Report of the Secretary-Treasurer

Report of the Editor-Librarian

- 1:00 Scientific Program (Open to Non-Members) Room: Mediterranean Ballroom - Salon 4
- 1:00 Remarks by the President Clough Shelton, MD

Presidential Citation *M. Jennifer Derebery, MD James L. Parkin, MD Rodney C. Perkins, MD*

1:10 Introduction of Guest of Honor H. Richard Harnsberger, MD

> Guest of Honor Presentation Decision Support in the 21st Century

Moderators: Clough Shelton, MD Paul R. Lambert, MD

- 1:31 Recurrent Ipsilateral Infranuclear Facial Paralysis Arvind Kumar, MD Richard Wiet, MD
- 1:40 Assessing Stapes Piston Position Using Computed Tomography Yoav Hahn, MD Hilary A. Brodie, MD, PhD
- 1:49 Long-Term Success of Four Piston Stapes Prostheses Evaluated by Product-Survival Procedure Charles A. Mangham, Jr., MD, MS Jacqueline Neel, MS Hannah F. Mangham
- 1:58 Discussion

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2:03 Automatic Identification and 3-D Rendering of Temporal Bone Anatomy Jack H. Noble, BS Benoit M. Dawant, PhD Frank M. Warren III, MD Omid Majdani, MD, PhD Robert F. Labadie, MD, PhD

2:12 Imaging the Human Tympanic Membrane Using Optical Coherence Tomography Majestic Tam, MD James Ridgway, MD Hamid Djalilian, MD Brian JF Wong, MD, PhD

2:21 Biopolymer Released Dexamethasone Prevents Tumor Necrosis Factor-Alpha Induced Loss of Auditory Hair Cells: Implications Toward Development of a Drug Eluting Cochlear Implant Electrode

> Thomas R. Van De Water, PhD Christine T. Dinh, BS Scott Haake, BS Shibing Chen, MD Richard Vivero, MD Kimberly Hoang, BS Adrien A. Eshraghi, MD, MSc Thomas J. Balkany, MD

2:30 Resveratrol, an Extract from Grapes and Red Wine, and Age-Related Hearing Loss

Michael Seidman, MD Ilaaf Darrat Wenxue Tang Uma Bai Hao Jiang Joseph Media Alexander Nakeff Wayne S. Quirk

- 2:39 Discussion
- 2:45 Break with Exhibitors

3:15 Reccurence Rate of Cholesteatoma with Kaplan-Meier Survival Analysis Yasuo Mishiro, MD Masafumi Sakagami, MD, PhD Tadashi Kitahara, MD, PhD Takeshi Kubo, MD

3:24 Current Bacteriology of Suppurative Otitis: Resistant Patterns and Outcomes Analysis James E. Saunders, MD Ryan P. Raju, MD Wayne E. Berryhill, MD Gregory Blakely, MD Johne Boone, BS Nathan Hales, MD

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3:33 Bone Conduction Hearing Level and Clinical Characteristics in Patients with Eosinophilic Otitis Media Associated with Bronchial Asthma Yukiko Iino, MD Kozue Kodama Hajime Usubuchi Rika Otake Takeharu Kanazawa Yasushi Ohta

3:42 Functional Regeneration of the Mastoid Air Cells by In Situ Tissue Engineering for Intractable Otitis Media Shin-ichi Kanemaru, MD, PhD Masaru Yamashita, MD, PhD Hiroo Umeda, MD Harukazu Hiraumi, MD Tatsunori Sakamoto, MD Koichi Omori MD, PhD

Juichi Ito, MD

- 3:51 Atresia Repair: Surgical Results When Performed before Medpor® Microtia Reconstruction Compared to Following Rib Graft Microtia Reconstruction Joseph B. Roberson, Jr., MD John F. Reinisch, MD Tahl Colen, MD
- 4:00 Habitual Sniffing and Eustachian Tube Function in Middle Ear Cholesteatoma Masafumi Sakagami, MD, PhD Shigeto Ota, MD Yasuo Mishiro, MD
- 4:09 Discussion
- 4:14 Panel: Controversies in Pediatric Cochlear Implantation: Meningitis, MR Imaging and Bilateral Implantation Moderator: John K. Niparko, MD Presenters: Craig A. Buchman, MD Howard W. Francis, MD Barry E. Hirsch, MD J. Thomas Roland, MD

Discussants: Thomas J. Balkany, MD Bruce J. Gantz, MD; Gerard O'Donoghue, MD Lorry G. Rubin, MD; Nancy M. Young, MD

- 4:59 Discussion
- 5:04 Adjournment
- 5:10 AOS Members Group Photograph (Location to be announced)

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Saturday, May 3, 2008

7:00 Business Meeting (Restricted to Members) Room: Mediterranean Ballroom - Salon 4

REPORT OF THE

- A. Board of Trustees of the Research Fund
- B. American Board of Otolaryngology
- C. Award of Merit Committee
- D. American College of Surgeons
- E. American Academy of Otolaryngology-HNS

Report of the Audit Committee

Report of the Membership Development Committee

Report of the Nominating Committee

Unfinished Business

New Business

7:30 Scientific Program - (Open to Non-Members) Room: Mediterranean Ballroom - Salon 4

Moderators: Clough Shelton, MD Paul R. Lambert, MD

7:30 Temporal Effects of a Combination of Antioxidant Drugs on the Treatment of Acute Acoustic Trauma Chul-Hee Choi, PhD Kejian Chen, PhD Angelica Vasquez-Weldon, BS Ronald L. Jackson, PhD Robert A. Floyd, PhD Richard D. Kopke, MD

7:39 Spiral Ganglion Cell Loss Is Unrelated to Segmental Cochlear Sensory System Degeneration Fred H. Linthicum, Jr., MD Jose N. Fayad, MD

7:48 Functional Studies Reveal New Mechanisms for Deafness Caused by Connexin Mutations Xi Lin, PhD Wenxue Tang, MD Qing Chang, MD, PhD Shoab Ahmad, PhD Benjamin Stong, MD Grace Leu, MD

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- 7:57 Risk Factors for Hearing Loss in US Adults: Data from the National Health and Nutrition Examination Survey, 2001-2002 Yuri Agrawal, MD John K. Niparko, MD
- 8:06 Discussion
- 8:11 Panel: Update on Disorders and Surgery of the Eustachian Tube Moderator: Dennis S. Poe, MD Presenters: Charles D. Bluestone, MD Sujana Chandrasekhar, MD Elias M. Michaelides, MD
- 8:56 Discussion
- 9:01 EarLens Transducer Behaviors in High Field Strength MRI Scanners Michael H. Fritsch, MD Jonathan P. Fay, PhD
- 9:10 Intra Operative Electromyography and Surgical Observations as Predictive Factors of Facial Nerve Outcome in Vestibular Schwannoma Surgery Olivier Sterkers, MD, PhD Isabelle Bernat, MD Alexis Bozorg Grayeli, MD, PhD Gonzalo Esquia, MD

Zhihua Zhang, MD Michel Kalamarides, MD, PhD

9:19 Vestibular End-Organ and Balance Deficits Following Meningitis and Cochlear Implantation Correlate Poorly with Functional Outcome Sharon L. Cushing, MD Blake C. Papsin, MD Susan I. Blaser, MD Adrian James, MA, BM BCh John A. Rutka, MD Karen A. Gordon, PhD

- 9:28 A Model to Determine the Financial Performance of Cochlear Implant Programs Douglas D. Backous, MD Erin Ressler, MS
- 9:37 Implanting Common Cavity Malformations Using Intraoperative Fluoroscopy Daniel H. Coelho, MD Susan B. Waltzman, PhD J. Thomas Roland, Jr., MD
- 9:46 Discussion

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- 9:51 Break with Exhibitors
- 10:15 Foreign Body Granuloma of the Inner Ear Following Cochlear Implantation. One Possible Cause of a Soft Failure? Joseph B. Nadol, Jr., MD Donald K. Eddington, PhD Barbara J. Burgess

10:24 Hearing-in-Noise Benefits Following Bilateral Simultaneous Cochlear Implantation Continue to Improve Four Years after Implantation Rose J. Eapen, MD Emily Buss, PhD Marcia S. Clark, AuD Harold C. Pillsbury III, MD Craig A. Buchman, MD

10:33 Cochlear Implant and Hearing Aid: A New Approach to Optimize the Fitting in This Bimodal Situation

Annerose M. Keilmann, Prof. Dr. med Andrea Bohnert, MTAF Jan Gosepath, Dr. med. habil Wolf J. Mann, Prof. Dr. dr. h.c. mult.

- 10:42 Objective Measures of Cochlear Stimulation through the Round Window Herman A. Jenkins, MD James R. Easter, MS, ME Brian M. Conn, BS, MBA James F. Kasic, MS, MBA
- 10:51 Basic Science Lecture: Pathological Semicircular Canal Afferent Signals Transmitted to the Brain During Benign Positional Vertigo and Their Biomechanical Origins Richard D. Rabbitt, PhD
- 11:11 Discussion
- 11:16 The Round Window Implant: The Last Chance for Hearing Restoration in Mixed Hearing Losses Vittorio Colletti, MD Marco Carner, MD Sheila Veronese, PE Liliana Colletti, PhD

11:25 The EarLens System: An Innovative Sound Transduction Method Rodney Perkins, MD Jonathan P. Fay, PhD Michael T. Murray, MD Lisa Olson, MS Sunil Puria, PhD

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- 11:34 Hearing Restoration: Improved Multi-talker Speech Understanding Sunil Puria, PhD Andy Vermiglio, MS Jonathan Fay, PhD Sig Soli, PhD
- 11:43 Vibrant Soundbridge Implantable Hearing Device: Long- and Short-term Results Charles M. Luetje, MD Sandra A. Brown, MA, CCC-A Robert D. Cullen, MD
- 11:52 Successes and Complications of the BAHA System Jack J. Wazen, MD Dayton Young, MD
- 12:01 Discussion

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- 12:06 Introduction of Incoming AOS President Joseph B. Nadol, Jr., MD
- 6:30 **President's Reception & Dinner Dance** (Members and Invited Guests Only)

2008 Program Advisory Committee

Patrick J. Antonelli, MD Douglas D. Backous, MD M. Jennifer Derebery, MD John L. Dornhoffer, MD John T. McElveen, Jr., MD Myles L. Pensak, MD Peter S. Roland, MD Jay T. Rubinstein, MD, PhD Debara L. Tucci, MD P. Ashley Wackym, MD

COSM 2009 142nd AOS Annual Spring Meeting May 29-30, 2009 JW Marriott Desert Ridge Resort & Spa Phoenix, Arizona

Abstract Deadline: October 15, 2008

Abstract submission form Website—www.americanotologicalsociety.org E-Mail– segossard@aol.com

Journal Requirements/Instructions to Authors/Presenters The journal of OTOLOGY & NEUROTOLOGY no longer accepts paper manuscripts. All manuscripts must be submitted online two weeks prior to the annual meeting, via the journal's website: https://www.editorialmanager.com/ on/. Instructions for registering, submitting a manuscript, and the author guidelines can all be found on the Editorial Manager site: https://www.editorialmanager.com/on/.

One copy of the manuscript (.pdf format) is to be submitted electronically to the AOS Administrative Office.

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Recurrent Ipsilateral Infranuclear Facial Paralysis

Arvind Kumar, MD; Richard Wiet, MD

Objective: The purpose of this paper is to underscore the importance of an exhaustive investigation of all patients with recurrent ipsilateral infranuclear facial paralysis.

Study Design: Retrospective case review.

Setting: Tertiary referral center.

Patients: The names of the patients included in this study were extracted from our database of unilateral facial paralyses. Four patients with ipsilateral, recurrent facial paralysis were identified. All were women and their ages ranged from 22 to 55 years. The number of recurrences ranged from 2-18. The interval between attacks ranged from one month to one year. The facial paralysis was complete in three out of the four patients at the time of presentation. The fourth patient, at the time of presentation, had normal facial function.

Intervention: All four of these patients were re-imaged with fine cut CT scans in axial, coronal and sagittal planes, and MRI scans with special pulse sequences. This despite the fact that both studies had been completed previously.

Main outcome measures: In all four patients a specific lesion was diagnosed and three patients have undergone surgery.

Results: The diagnoses reached were glomus tumor at the geniculate ganglion, mucoepidermoid tumor involving the facial nerve in the mastoid segment and in the parotid schwannoma of the facial nerve at the tympanic segment and probable viral infection of the tympanic and mastoid segments. This last patient is awaiting surgery.

Conclusions: "All that palsies is not Bell's"

2. Assessing Stapes Piston Position Using Computed Tomography

Yoav Hahn, MD; Hilary A. Brodie, MD, PhD

Hypothesis: Temporal bone computed tomography scanning in the postoperative stapedotomy patient is inaccurate in assessing stapes piston position within the vestibule.

Background: Post-stapedotomy patients that have persistent vertigo often undergo CT scanning to assess the position of the stapes piston within the vestibule in order to rule out overly deep insertion. Vertigo is a recognized complication of the deep piston and CT evaluation is often recommended. The accuracy of CT scan in this setting is undetermined.

Methods: Stapedotomy was performed on 12 cadaver ears and stainless steel McGee pistons were placed. The cadaver heads were then scanned using a fine cut temporal bone protocol. Temporal bone dissection was performed with microscopic measurement of the piston intrusion into the vestibule with two independent measures. These values were compared with depth of intrusion measured on CT scan by four independent measures.

Results: The CT scan measurement of intra-vestibular piston penetration was 0.25 mm to 1.4 mm longer than the anatomic measurement (p<0.01). Two independent anatomic measures were performed during cadaveric dissection and were identical on 66% of the measures and only 0.1 mm different on 33% of the measures.

Conclusions: High-resolution temporal bone CT scan is not a valid study to assess intra-vestibular piston measurement after stapedotomy. CT scan consistently overestimated the penetration of the piston within the vestibule and can lead to inaccurate assessment of piston position within the vestibule.

Long-Term Success of Four Piston Stapes Prostheses Evaluated by Product-Survival Procedure

Charles A. Mangham Jr., MD, MS Jacqueline Neel, MS; Hannah F. Mangham

Objective: To determine if modifications to a first-generation stapes prosthesis have improved long-term hearing results.

Study design: Retrospective chart review.

Setting: Private practice.

Patients: 283 ears in 255 consecutive patients who had primary stapes surgery during 1990-2000 and in 2005.

Intervention: The devices were examined sequentially in the following order: first-generation Teflon-wire (52), Teflon-ribbon (30), double-bend Teflon-ribbon (168) and titanium CliP® piston (33).

Main outcome measures: AAO-HNS guidelines including four frequency pure-tone average (PTA), success rate (gap < 10 dB), and Kaplan-Meier product-survival procedure.

Results: Mean 1-year PTA air-bone gap and success rate (84 to 97%) were similar for the four devices. The Kaplan-Meier procedure was used to examine both short and long-term success. The three Teflon pistons had greater long-term success than the CliP® piston. Although the Teflon-ribbon pistons had a lower failure rate than the Teflon-wire in the first 5 years, all three of the Teflon pistons accumulated 7-13% additional failures by the 15th year. Of the surgical failures, 85% of the patients with Teflon pistons had an unstable connection to the incus.

Conclusions: All four of the piston designs had 1-year results that met common standards for adequate success, but accumulated additional prosthesis-related failures over time. Superiority of two the Teflon-ribbon pistons in the first 5 years after surgery may be explained by their ability to delay but not eliminate incus necrosis. Suggestions for a better incus connection will be presented

IBR#: 06-0065

4.

Automatic Identification and 3-D Rendering of Temporal Bone Anatomy

Jack H. Noble, BS; Benoit M. Dawant, PhD Frank M. Warren III, MD; Omid Majdani, MD, PhD Robert F. Labadie, MD, PhD

Hypothesis: Using atlas-based registration, vital anatomy of the middle ear can be automatically identified in CT scans and used to create 3-D renderings.

Background: While difficult to master, surgeons compile 2-D data from CT scans to envision 3-D anatomy. Computer programs exist which can render 3-D surfaces but are limited in that middle ear structures, e.g. the facial nerve, can only be rendered after being manually identified. Herein, we present results from novel computer algorithms which automatically identify temporal bone anatomy from unknown CT scans based on an atlas of a "normal" patient and mathematical models of the structures of interest.

Methods: Using registration and segmentation methods, the facial nerve, chorda tympani, cochlea, ossicles, and external ear canal were automatically identified from temporal bone CT scans of 14 patients. To measure the accuracy of the identification algorithms, the structures were then manually identified. Error was quantified by measuring the distance from each false positive voxel to the manually identified structure (FP) and from each false negative voxel to the automatically identified structure (FN).

Results: For the 14 CT scans; the average maximum error distances for FP and FN (\pm standard deviation) were calculated to be 0.68 \pm 0.06mm and 0.86 \pm 0.12mm for the facial nerve.

Conclusions: The automated anatomy identification algorithms were extremely accurate with maximum error no greater than 4 voxels. As will be seen from demonstrative figures, this information is useful in understanding spatial relationships of middle ear anatomy.

Supported by: NIH (NIDCD) R01DC008408 IBR#: 060028

Imaging the Human Tympanic Membrane Using Optical Coherence Tomography

Majestic Tam, MD; James Ridgway, MD Hamid Djalilian, MD; Brian JF Wong, MD, PhD

Objective: Optical coherence tomography (OCT) is a diagnostic imaging modality that employs low coherence light with interferometry to produce high-resolution cross-sectional images of living tissues. Using this technology we have characterized the human tympanic membrane in the office clinic setting in a non-invasive manner.

Study Design: Prospective clinical trial.

Setting: Tertiary care center, Hospital

Patients: Inclusion criteria: Newborn and older, infant/ children/minor accompanied by parent or guardian

Exclusion criteria: subject unable to understand or carry out instruction or refusal to participate.

Materials and Methods: Ten healthy adult subjects were evaluated in preoperative and postoperative states. Each subject underwent direct microscopic examination prior to OCT imaging to ensure optimal visualization and imaging of the tympanic membrane and associated subsites including the annulus fibrosis, pars tensa, pars flaccida and umbo. The OCT probe was introduced into the ear canal under direct visualization to ensure precise placement of the probe. In addition, any visible pathologies or abnormalities were imaged.

Results: Systematic imaging of the tympanic membrane was performed with characterization of the epithelial and collagenous layers. The overall membrane thickness was clearly demonstrated and quantified as well as the membrane relationship with underlying bony structures.

Conclusion: The ability to non-invasively study otologic microstructures in the clinical setting is essential in the treatment of diseases of the ear. OCT could potentially offer the otolaryngologist the imaging capability to characterize pathologies such as cholesteatoma, lesions of the external canal, and chronic otitis media, in addition to patients who have undergone mastoidectomy, tympanoplasty and other otologic surgical procedures. This imaging modality holds the promise to becoming a valuable technology in the characterization of the tympanic membrane and associated otologic structures.

Biopolymer Released Dexamethasone Prevents Tumor Necrosis Factor-Alpha Induced Loss of Auditory Hair Cells: Implications Toward Development of A Drug Eluting Cochlear Implant Electrode

Thomas R. Van De Water, PhD; T. Dinh, BS Scott Haake, BS; Shibing Chen, MD Richard Vivero, MD; Kimberly Hoang, BS Adrien A. Eshraghi, MD, MSc; Thomas J. Balkany, MD

Hypothesis: Dexamethasone bioreleased from a polymer will retain its activity and protect hair cells from the ototoxicity of a traumaassociated cytokine (i.e. TNF-alpha).

Background: Drug elution technology has been successfully used for cardiac pacemaker leads and circulatory system stents. Insertion of an electrode array into a laboratory animal's scala tympani can result in a progressive loss of hearing while local perfusion with dexamethasone immediately following electrode insertion can protect against this electrode trauma-induced hearing loss.

Material and Methods: Three-day-old rat organ of Corti explants were used to test the ability of SIBS biorelease dexamethasone to protect auditory hair cells from an ototoxic level of TNF-alpha (i.e. 1 ug/ml). FITC-phalloidin stained organ of Corti explants from the experiments were analyzed for hair cell integrity. Hair cell density counts were made for all turns of the cochlear cultures. Total RNA was extracted from a separate series of control, TNF-alpha challenged and TNF-alpha challenged and biorelease dexamethasone treated explants with a Qiagen RNeasy Kit at 0, 24 and 48 hr time points. Gene expression studies with real time RT-PCR focused on selected anti- and pro-apoptotic molecules. All data was statistically analyzed with a p value of < 0.05 considered significant.

Results: TNF-alpha exposure caused a highly significant (i.e. p < 0.001) loss of hair cells from the explants, whereas explants exposed to the same level of TNF-alpha but protected with biorelease dexamethasone showed a highly significant level of hair cells survival (p < 0.001) when compared to the TNF-alpha only explants. Analysis of the expression levels of genes that encode for proapoptotic and anti-apoptotic molecules demonstrate that TNF-alpha exposed explants up regulate pro-apoptotic genes (e.g. Bax) and TNF-alpha cultures treated with bioreleased dexamethasone down regulated pro-apoptotic genes (e.g. Bax) and up regulated anti-apoptotic genes (e.g. Bac).

Conclusion: Biorelease dexamethasone retains it otoprotective activity against TNF-alpha induced hair cell loss in vitro. Biorelease dexamethasone treatment protects against TNF-alpha induced hair cell loss by regulating genes that encode for apoptosis related molecules. Drug eluting electrode arrays represent a promising technology and dexamethasone is an excellent candidate drug for this application.

Acknowledgements: Research Supported by Advanced Bionics, Valencia, CA Univ. Miami ACUC protocol # 05-101

7.

Resveratrol, an Extract from Grapes and Red Wine, and Age-related Hearing Loss

Michael Seidman, MD; Ilaaf Darrat; Wenxue Tang; Uma Bai; Hao Jiang; Joseph Media; Alexander Nakeff; Wayne S. Quirk

Objective: To evaluate the protective effects of resveratrol, on age-related hearing loss and to attempt to understand the biochemistry underlying the protective effects.

Study design: 36 Harlan Fisher rats (9 months of age) were randomized to four groups that were implanted with either 43 ug/kg/day subcutaneous time release resveratrol pellet (Group 1) (replaced every 3 months); 430 ug/kg/day pellet (Group 2); 4300 ug/kg/day pellet (Group 3); or a time releasing subcutaneous placebo pellet (Group 4).

Methods: Subjects were treated for one year with one of three doses of resveratrol or placebo. Auditory measurements were obtained at baseline and every three months. Western blot analysis was performed on cochlear hair cells at 21 months and studied for Cycloxygenase-2 (Cox-2) and 5-Lipoxygenase (5-LOX) expression (two biomarkers for inflammation and aging).

Results: Auditory measurements at baseline demonstrated that all subjects had normal hearing (15-25 dB range from 3-18 kHz). Placebo subjects demonstrated progressive sensorineural hearing loss consistent with earlier studies. There was a statistically significant attenuation in the hearing loss in the two highest doses of resveratrol (p<0.005). Decreases in expression of Cox-2 was observed in all groups (for 43 ug: p<0.005, 430 ug: p<0.000, 4300 ug: p<0.000, as compared to the control group). Decreases in 5-Lox expression was observed at the lowest (p<0.001) and highest (p<0.000) doses of resveratrol. There was no significant decrease at the mid dose of resveratrol (430 ug).

Conclusions: Oxidative stress is associated with most medical disorders and appears to be a key mediator in the aging process. Resveratrol contained in grapes and wine has multiple biological actions. It is a powerful antioxidant, an inhibitor of inflammation and suppresses Cox-2 and 5-LOX expression, both of which are upregulated in aging. Earlier studies from our laboratory have shown a protective effect against noise induced hearing loss and the present study demonstrates its ability to protect against age-related hearing loss.

Acknowledgements: NCCAM R-21AT001067-01A2 California Table Grape Association

8. Reccurence Rate of Cholesteatoma with Kaplan-Meier Survival Analysis

Yasuo Mishiro, MD; Masafumi Sakagami, MD, PhD Tadashi Kitaharam MD; Takeshi Kubo, MD

Objectives: To calculate the rate of cholesteatoma recurrence using Kaplan-Meier survival analysis

Study Design: Retrospective Study.

Setting: Tertiary referral medical center.

Patients: Three hundred twenty-five patients with cholesteatoma underwent surgery by the same surgeon between 1987 and 2002. The mean follow-up period was 5.96 years with a range of 2 months to 18.9 years.

Methods: The rate of recurrence in these 325 cholesteatoma cases was calculated using Kaplan-Meier survival analysis.

Results: Cholesteatoma recurred in 39 ears. Two recurrences were classified as residual cholesteatoma and the remaining 37 were classified as recurrent cholesteatoma. The rate of cholesteatoma recurrence was 0% at one year, 2.9% at 2 years, 7.2% at 3 years, 9.3% at 4 years, 11.1% at 5 years, 13.3% at 6 years, 13.3% at 7 years, 16.1% at 8 years, 17.1% at 9 years and 18.3% at 10 years. Rate of cholesteatoma recurrence after canal wall down procedure (2.4% at 5 years and 2.4% at 10 years) was significantly better than that after canal wall up/canal wall reconstruction procedure (16.0% at 5 years and 25.1% at 10 years) (p<0.01, Log Rank test).

Conclusions: The rate of cholesteatoma recurrence deteriorated during long-term follow-up, suggesting that reporting the postoperative outcomes for cholesteatoma with follow-up periods shorter than 2 years is meaningless. Kaplan-Meier survival analysis should be used for discussing recurrence of cholesteatoma.

Current Bacteriology of Suppurative Otitis: Resistant Patterns and Outcomes Analysis

James E. Saunders, MD; Ryan P. Raju, MD Wayne E. Berryhill, MD; Gregory Blakely, MD Johne Boone, BS; Nathan Hales, MD

Objective: Rising antibiotic resistance presents a challenging problem in the management of otolaryngological infections, but few studies have investigated the role of resistant bacteria in the management of the draining ear. In a recent study at our institution, resistant bacteria and Methicillin Resistant Staphylococcus Aureus (MRSA) isolates were noted in 50% and 7.8% of infections, respectively. This study examines specific resistance patterns, clinical outcomes and the role of cultures in the management of these patients.

Study design: Retrospective study

Setting: Academic Otology Practice

Patients: 170 Patients with suppurative otitis, children and adults

Interventions: Diagnostic and Therapeutic

Main Outcome Measures: Information regarding the cultured organisms, antibiotic sensitivity, clinical diagnosis, prior treatment, treatment efficacy, and outcomes were analyzed.

Results: MRSA isolates were resistant to trimethoprimsulfamethoxazole and clindamycin in 33% and 60% of MRSA infections, respectively. Overall, highly resistant bacteria (sensitive to intravenous antibiotics only) were seen in 5.7% of infections. Quinilone resistance was present in 5.3% of infections. Patients using topical quinilone drops prior to culture were significantly more likely to have a quiniloneresistant infection (p=0.011). There was a significant relationship between the presence of resistant bacteria and prior treatment for otitis externa, but not for any other diagnosis category. In 28% of infections, specific treatment changes were based on the cultural results.

Conclusions: We conclude that outpatient cultures play an integral role in the management of suppurative otitis, and should be performed in refractory otorrhea. Our results suggest that liberal use of topical quinolone therapy is associated with increased bacterial resistance.

Acknowledgements: Oklahoma University Department of Microbiology

Bone Conduction Hearing Level and Clinical Characteristics in Patients with Eosinophilic Otitis Media Associated with Bronchial Asthma

Yukiko Iino, MD; Kozue Kodama; Hajime Usubuchi Rika Otake; Takeharu Kanazawa; Yasushi Ohta

Objective: Eosinophilic otitis media (EOM) is a new middle ear disease entity with extensive accumulation of eosinophils in the middle ear mucosa and middle ear effusion (MEE), and is usually associated with bronchial asthma. Patients with EOM show gradual deterioration of hearing and sometimes become deaf suddenly. However, there have been no systemic studies of bone conduction hearing levels of patients with EOM.

Study design: Retrospective case review.

Setting: Tertiary referral center.

Patients: Thirty-four patients with EOM associated with bronchial asthma diagnosed by histological or cytological studies of MEE or middle ear mucosa.

Main outcome measures: The clinical characteristics of the patients were evaluated by blood test, eosinophil cationic protein (ECP) level of MEE, bacterial and fungal culture of MEE, condition of middle ear mucosa and temporal bone CT scan. The relationship of bone conduction hearing level of speech range and 4000 Hz, and clinical characteristics in each patient was analyzed.

Results: Most of the ears showed high tone hearing loss, and bone conduction hearing level more than 40dB at 4000Hz was found in 37% of the ears. Two patients became profound deafness unilaterally during the course. Two deaf ears had granulation tissue formation in the mesotypanum, and were infected by Psuedomonas aeruginosa. The high levels of ECP were detected from all the patients, but there was no correlation between the bone conduction hearing levels and ECP levels of MEE.

Conclusions: High tone hearing loss and profound hearing loss were frequently associated with EOM, suggesting inflammatory products of the middle ear invade to the inner ear via the round window to cause inner ear damage. To prevent deterioration of bone conduction hearing level, control of eosinophilic granulation tissue formation and bacterial infection is mandatory

Functional Regeneration of the Mastoid Air Cells By In Situ Tissue Engineering for Intractable Otitis Media

Shin-ichi Kanemaru MD, PhD; Masaru Yamashita MD, PhD Hiroo Umeda MD; Harukazu Hiraumi MD Tatsunori Sakamoto MD; Koichi Omori MD, PhD Juichi Ito MD

Objective: To assess whether regenerated MACs have gas exchange functions or not by nitrous oxide gas study with a new device. In order to a complete recovery from intractable otitis media, regeneration of the mastoid air cells' (MACs') gas exchange functions is thought to be necessary. Our previous study showed that MACs' structure was able to be regenerate.

Study design: Pilot study

Setting: University hospital and general hospital.

Patients: Intractable otitis media

Intervention: Therapeutic.

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Material and Method: Collagen coated hydroxyapatite of honeycomb-like structure was used as an artificial pneumatic bone (APB). At the 1st-stage tympanoplasty, APB was implanted into the newly opened mastoid cavity of 36 patients. At the 2nd-stage operation, the changes of the middle ear pressure were measured of 10 in 12 patients whose MACs' structure and aeration in the mastoid cavity were identified by the images of CT scan. (Group I) For control, the same study was performed to the 5 patients with the good developed MACs who underwent the cochlear implant or the facial nerve decompression. (Group II) Final assessments of the regeneration of MACs were performed 12 months after the 2nd-stage operation.

Results: In all 10 patients in group I, gas exchange functions were regenerated to varying degrees. In the final assessment, MACs were regenerated in 21 of 36 patients.

Conclusions: This study indicated that MACs were able to regenerate not only morphologically, but also physiologically by implantation of APB into the newly opened mastoid cavity. The abilities of gas exchange function of regenerated MACs were inferior to those of normal MACs.

Atresia Repair: Surgical Results When Performed before Medpor® Microtia Reconstruction Compared to Following Rib Graft Microtia Reconstruction

Joseph B. Roberson, Jr., MD John F. Reinisch, MD; Tahl Colen, MD

Objective: To compare results of atresia repair before and after microtia reconstruction.

Study Design: Retrospective case review

Setting: Tertiary referral center, ambulatory surgery center and clinic

Patients: Congenital aural atresia with or without microtia

Intervention: Atresia repair prior to Medpor® microtia reconstruction (ARM) vs. atresia repair following microtia reconstruction with autogenous rib (ARR) vs. atresia reconstruction without microtia (AR).

Main Outcome Measures: Surgical outcomes, pre- and postoperative audiometry results, complications

Results: Data from the three groups are as follows: ARM - 31 patients with average age 4.5 yrs (range 2.5 - 9.3 yrs), ARR - 28 patients with average age 18.4 yrs (range 6.9-61), AR - 11 patients with average age 19.8 yrs (range 5.5 - 59 yrs). Preoperative CT grading utilizing the Jahrsdoerfer scale demonstrated results for the three groups of ARM 7.4 (range 5-9), ARR 7.7 (range 6-9), and AR 8.5 (range 8-9). Postoperative PTA2 Average for each group J-scale 8-10 were ARM 28, ARR 32, AR 29. Postoperative PTA2 Average for each group J-scale 7 or less were ARM 42, ARR 41, AR (no patients). Surgical complications of infection and facial nerve injury were not seen in any group. Re-stenosis, need for middle ear reconstruction, and need for surgical revision was higher in the ARR group. One patient in the ARM group suffered a sensorineural hearing loss. No patient receiving Medpor® microtia reconstruction suffered a complication due to the presence of the ear canal prior to microtia reconstruction.

Conclusions: Early results of Atresia Repair prior to Medpor® Mictrotia reconstruction compare favorably to results achieved with Atresia Repair following Microtia Reconstruction with autogenous rib cartilage and with Atresia Repair without Microtia repair and to previously reported expert results in terms of hearing outcome and complication rates. Since restoration of binaural hearing has been shown to be advantageous for development and auditory function, timing of Atresia repair should be considered prior to Microtia reconstruction on an individual case basis - provided preoperative CT evaluation shows an adequate chance of surgical success. Long term follow up to determine stability of surgical results compared to other techniques should be carried out to aid in this change of treatment protocol.

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Habitual Sniffing and Eustachian Tube Function in Middle Ear Cholesteatoma

Masafumi Sakagami, MD, PhD; Shigeto Ota, MD Yasuo Mishiro, MD

Objective: To investigate the incidence of habitual sniffing and function of the Eustachian tube in middle ear cholesteatoma

Study design: Prospective study

Setting: University hospital

Patients: Eighty consecutive cases of primary acquired cholesteatoma were treated surgically between July 2005 and May 2007, and they consisted of 58 pars flaccida type (72.5%), 20 pars tensa type (25.0%) and 2 unclassified large type (2.5%). As a contrast, 131 consecutive cases of chronic otitis media (COM) were also examined.

Methods: A questionnaire survey about symptoms of patulous tube (autophonia, ear block) and habitual sniffing to alleviate ear symptoms was conducted. Eustachian tube function was examined with sonotubometry, tubo-tympano aerodynamic graph, and inflation/deflation test.

Results: Habitual sniffing was significantly higher in cholesteatoma (25/80, 31.3%) than in COM (6/130, 4.6%) (p<0.001). Eustachian tube function in cholesteatoma showed the open type (33.8%), stenotic type (41.3%) and normal type (25.0%), while that in COM 14.5%, 23.7%, and 61.8%, respectively. The incidence of open type is significantly higher in cholesteatoma than in COM (p<0.01). The existence of diseases on the contralateral side was significantly higher in patients with habitual sniffing (20/25, 80.0%) than in those without habitual sniffing (24/55, 43.6%) (p<0.01). After canal wall up procedures (n=33), 13 patients with habitual sniffing showed retraction in 3(23.1%), recurrence in 1(7.7%), and good outcome in 9(69.2%), while 20 patients without habitual sniffing showed in retraction in 1(5.0%), recurrence in none (0%), and good outcome in 19(95.0%).

Conclusions: Habitual sniffing is closely related to the pathogenesis of middle ear cholesteatoma.

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Temporal Effects of a Combination of Antioxidant Drugs on the Treatment of Acute Acoustic Trauma

Chul-Hee Choi, PhD; Kejian Chen, PhD Angelica Vasquez-Weldon, BS; Ronald L. Jackson, PhD Robert A. Floyd, PhD; Richard D. Kopke, MD

Background: Acute acoustic trauma (AAT) results in oxidative stress to the cochlea leading to overproduction of cellular reactive oxygen, nitrogen, and free radical species. Oxidative stress occurs shortly after noise exposure and extends to 7-10 days after noise exposure.

Objective: This study tested the temporal effects of a combination of hydroxylated alpha-phenyl-tert-butylnitrone (4-OHPBN) and N-acetyl-L-cysteine (NAC) and acetyl-L-carnitine (ALCAR) on the treatment of AAT.

Methods: Thirty-six chinchillas were exposed to a 105 dB narrow-band noise centered at 4 kHz for 6 hours and received the following treatments: 1) carrier solution only; 2-6) 4-OHPBN (20mg/kg) + NAC (50mg/kg) + ALCAR (20mg/kg) 4 hours with 3-day treatment, 24 and 48 hours after noise exposure with 3- and 10-day treatment. The drug combination was intraperitoneally injected 4, 24, and 48 hours after noise exposure twice daily for the next two and nine/eight days while carrier solutions were injected 24 hours after noise exposure with the same method twice daily for the next nine days. Auditory brainstem response threshold shifts were analyzed with ANOVA.

Results: The drug combination administered 4 hours after noise exposure reduced the threshold shifts up to 90% while those administered 24 and 48 hours after noise exposure decreased the threshold shifts up to 50%. There was no significant difference between 9-day treatment after 24 hours and 8-day treatment after 48 hours. There was a non-statistical trend for greater hearing preservation with 10-day verses 3day treatment.

Conclusion: These results demonstrate that the early administration of antioxidant drugs after noise exposure can effectively treat AAT. A significant reduction in the threshold shift was still observed when the administration was initiated 24 or even 48 hours after noise exposure.

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Spiral Ganglion Cell Loss Is Unrelated to Segmental Cochlear Sensory System Degeneration

Fred H. Linthicum Jr., MD; Jose N. Fayad MD

Hypothesis: Contrary to what occurs in animals, neuron loss in the human spiral ganglion is not in proportion to hair or supporting cell loss.

Background: Reports in the literature, based on animal experiments, have suggested that the poor performance of cochlear implant patients implanted many years after the onset of deafness is due to a degeneration of spiral ganglion cells as a result of the loss of hair cell input from the organ of Corti. This supposition does not apply to humans.

Methods: Four temporal bones, from an archival collection of 1,448 temporal bones, were found that had a total loss of hair and supporting cells limited to the basal segment of the cochlea, a hearing loss of 10 or more years, and an audiogram within 4 years of death. Cochlear reconstructions were carried out to demonstrate the relative populations of hair and supporting cells, peripheral processes (dendrites), spiral ganglion cells, and the amount of stria vascularis.

Results: The total loss of hair and supporting cells of the organ of Corti in the base of the cochlea is not accompanied by a proportional loss of spiral ganglion cells in the modiolar base.

Conclusions: A long-term loss of hearing, 10 to 28 years, in frequencies above 2kHz, and corresponding hair cell loss does not result in a consequent loss of spiral ganglion cells in humans in contrast to what has been reported in animals. These findings suggest that the poor performance of cochlear implant patients after prolonged deafness is not due to loss of ganglion cells as suggested by animal studies.

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Functional Studies Reveal New Mechanisms for Deafness Caused by Connexin Mutations

Xi Lin, PhD; Wenxue Tang, MD; Qing Chang, MD, PhD Shoab Ahmad, PhD; Benjamin Stong, MD; Grace Leu, MD

Gap junctions (GJ) are membrane channels facilitating exchanges of intercellular biochemical and electrochemical signals. Connexins (Cxs) are a family of 20 protein subunits constituting the building blocks of GJs. Twelve compatible Cx subunits assemble together to form GJs. The importance of Cxs in hearing has been revealed by genetic studies showing that at least four Cx subtypes (Cxs 26, 30, 31 & 43) are essential for normal cochlear functions in human. Mutations in these Cxs account for a significant portion (20-50%) of prelingual non-syndromic deafness.

Most GJs in the cochlea are assembled heteromerically from Cx26 and Cx30. The two Cxs display almost identical cellular expression patterns, both in the developing and in matured cochlea. From birth to postnatal day 10 (P10), GJs in the lateral wall were found to be concentrated between basal cells and fibrocytes surrounding the stria vascularis. More cells in the cochlea were connected by GJs after P12 when hearing starts to mature in mice. Null mutations of either Cx26 or Cx30, which don't eliminate GJs in the cochlea, eliminate the endolymphatic potential (EP) and cause severe hearing loss at early development stages. Hair cell (HC) death could not be the direct cause of deafness in Cx mutant mice since HC loss doesn't parallel the degree of hearing loss. Using a nonhydrolyzable fluorescent analogue of D-glucose (2-NBDG) to study GJ-mediated biochemical coupling, we found that both the rate and extent of the dye diffusion were significantly decreased in Cx30-/- mice. Compromise in GJ-mediated intercellular diffusion of glucose analogue suggested that biochemical coupling important for metabolic activities in the cochlea is affected in the mutant mice, which may be responsible for dysfunction of mitochondria suggested by our microarray and proteomics data. Our studies also identified a possible direct link between the loss of EP in the mutant mice and a significant reduction in expressions of KCNO1 and KCNE1 before the establishment of EP (e.g., P10), since both K+ channels are known to be required for generating EP.

Conclusions: These new data suggested that deficits in gap junction (GJ) facilitated biochemical coupling in early postnatal cochlear development, which is essential for the full expression of ion channels/transporter required for establishing EP, is directly responsible for deafness found in the Cx mutant mice.

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Risk Factors for Hearing Loss in US Adults: Data from the National Health and Nutrition Examination Survey, 2001-2002

Yuri Agrawal, MD; John K. Niparko, MD

Objective: To relate noise exposure (occupational, recreational, firearm) and cardiovascular risk factors (hypertension, smoking, diabetes) to frequency-specific audiometric thresholds among US adults, while assessing synergistic interactions between these exposures.

Design: National cross-sectional survey.

Setting/Participants: US adults aged 20-69 who participated in the 2001-2002 National Health and Nutrition Examination Survey (N=2046).

Main Outcome Measures: Air conduction thresholds at 0.5 to 8 kHz (dB) in the poorer hearing ear. Multivariate models adjusted for age, sex and race/ethnicity.

Results: Exposure to firearm noise was significantly associated with high frequency (4-8 kHz) hearing loss (HL), whereas smoking and diabetes were associated with significantly increased hearing thresholds across the frequency range (0.5 to 8 kHz). A significant interaction was observed between exposure to firearm noise and diabetes, such that firearm noise was associated with a mean 26 dB hearing loss in diabetic participants compared to a mean 2.5 dB hearing loss in non-diabetics at 2 kHz. We also observed significant interactions between firearm noise exposure and smoking.

Conclusions: Noise exposure was associated with highfrequency HL whereas cardiovascular risk was associated with both high- and low-frequency HL. The frequency-specific effects of these exposures may offer insight into mechanisms of cochlear damage. We also demonstrated an interaction between cardiovascular risk and noise exposures, possibly as a result of cochlear vulnerability due to microvascular insufficiency. Such significant interactions provide proof of principle that certain pre-existing medical conditions can potentiate the effect of noise exposure on hearing. Data-based stratification of risk should guide our counseling of patients regarding HL.

IBR#: NA

EarLens Transducer Behaviors in High-Field Strength MRI Scanners

Michael H. Fritsch, MD; Jonathan P. Fay, PhD

Objective: Identify the problems and patient safety issues associated with a magnet-containing medical device, when it is secured to the human tympanic membrane (TM) and exposed to MRI magnetic fields.

Study design: Cadaver human temporal bone (TB) and Forcemeasurement devices studies.

Setting: University Medical Center.

Patients/ Interventions: NA.

Main outcome measures: Measuring the translational and torque forces acting on the implant and also, defining the clinical safety correlations based on the TB study findings and actual force recordings.

Results: Testing of the EarLens in 1.5, 3.0, and 9.4 Tesla MRI fields revealed movement and displacement of the prosthesis in the TB study. Progressively larger torque and translational forces were recorded with larger MRI magnets. The maximum translational and torque forces recorded were at the entrance to the 9.4T MRI.

Conclusions: The EarLens is attached to the TM during patient use. The appearance of this device on the market is near-term. The inevitable need for patients using the device to receive MRI scans makes understanding of the device's behavior in MRI magnetic fields, and safety countermeasures, crucial.

Intra Operative Electromyography and Surgical Observations as Predictive Factors of Facial Nerve Outcome in Vestibular Schwannoma Surgery

Olivier Sterkers, MD, PhD; Isabelle Bernat, MD Alexis Bozorg Grayeli, MD, PhD; Gonzalo Esquia, MD Zhihua Zhang, MD; Michel Kalamarides, MD, PhD

Objective: To evaluate the facial nerve position in the cerebellopontine angle (CPA), the tumor adhesion, and several electromyographic parameters as predictive factors of facial nerve outcome after vestibular schwannoma (VS) surgery.

Study design: Between November 2005 and August 2007, 155 patients operated on for solitary VS were included in this prospective study.

Setting: Tertiary referral center

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Patients: The population comprised 85 females and 70 males with a mean age of 50 years (range: 16-83). Tumor size was assessed as stage 1 (intracanalicular)in 12%, stage 2 (<16 mm in CPA) in 46%, 3 (16-30 mm in CPA) in 27% and 4 (> 30 mm) in15%.

Interventions: Patients were operated on through translabyrinthine (84%), retrosigmoid (13%) or middle cranial fossa (3%) approache and monitored by a four-channel facial EMG (NIM response, Xomed Medtronics, France) intra operatively.

Main outcome measures: Facial nerve position (1: anterior and inferior to the tumor in the CPA, 2: anterior to the tumor, 3: anterior and superior), tumor adhesion (weak, intermediate and strong), stimulation threshold of the facial nerve in mA at the brainstem after dissection, response amplitude of the mentalis muscle (μ V) to supramaximal stimulation of the nerve at the brainstem (2 mA) before and after tumor dissection, immediate postoperative facial nerve function (day 8, House and Brackman classification).

Results: Facial nerve position in the CPA influenced the outcome (51% of grade 1 and 2 facial in position 1, 39% in position 2, and 11% in position 3, P< 0.001, Chi-2 test), and tumor adhesion influenced the immediate outcome (43% for weak, 33% for intermediate and 24% in strong adhesion group, P< 0.005, Chi-2 test). Combination of the following intra operative electromyographic parameters predicted a good facial nerve outcome (grades 1 2) in all cases: mentalis response to supramaximal stimulation after tumor removal > 800 μ V with a after/before tumor dissection > 0.8, and a stimulation threshold at brainstem after tumor removal < 0.04 mA.

Conclusion: Intra operative observations and combination of several electromyographic parameters have a high predictive value of immediate facial function outcome.

Vestibular End-Organ and Balance Deficits Following Meningitis and Cochlear Implantation Correlate Poorly with Functional Outcome

Sharon L. Cushing, MD; Blake C. Papsin, MD Susan I. Blaser MD; Adrian James MA, BM BCh John A. Rutka MD; Karen A. Gordon PhD

Objective: Assess the impact of vestibular and balance function in meningitis-induced profound sensorineural hearing loss (SNHL).

Study Design: Prospective cohort study.

Setting: Tertiary pediatric referral centre.

Patients: Ten pediatric unilateral cochlear implant users with profound sensorineural hearing loss (SNHL) from bacterial meningitis.

Intervention: Vestibular function was measured by caloric, high frequency rotation (0.25–5Hz) and vestibular evoked myogenic potential(VEMP) testing. Static and dynamic balance were assessed by the Bruininsk-Oseretsky Test of Motor Proficiency 2 (BOT2) Balance Subset. Labyrinthine ossification on computed tomography (CT) was graded pre-implant, with follow-up imaging in a subset (n=6).

Main outcome measure(s): BOT2 score, nystagmus (velocity, direction) following caloric stimulus, VOR gain during rotation, VEMP waveform and graded ossification on CT.

Results: Caloric function was abnormal in all 6 children tested with 5/6 demonstrating bilateral areflexia and 1/6 unilateral areflexia. VOR gain was abnormal in 6/7 children and VEMP normal bilaterally in 4/6. No significant differences existed between the implanted and non-implanted side. BOT2 scores were significantly poorer in children with meningitis than normal hearing controls (P<.01) or congenital SNHL (P=.01). Imaging suggested labyrinthine ossification in all cases, however degree and location was variable.

Conclusions: Balance is compromised in children with SNHL from meningitis. While vestibular loss appears well compensated at low frequency, deficits become apparent during high frequency rotation. Saccular function appears less susceptible to damage from meningitis. Degree and location of ossification was variable and correlated poorly with differences in vestibular function. Despite profound dysfunction, subjective limitations in balance or functional abilities were not reported.

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A Model to Determine the Financial Performance of Cochlear Implant Programs

Douglas D. Backous, MD; Erin Ressler, MS

Objective: This investigation developed a model to determine the financial status of cochlear implant programs.

Study design: Prospective descriptive study.

Setting: Tertiary cochlear implant program.

Patients: 20 patients, over 18 years old, were value stream mapped while moving through the stages of cochlear implant candidacy determination and device activation. No patient identifiers or specific patient data was included.

Interventions: Cochlear implantation and device programming.

Main outcome measures: The content of each visit type with timings at each stage were evaluated.

Results: Of the total patient volume 8% of patients were in initial consultation, 6%end-consultation, 5% initial activations and 30% in early follow-up, and 51% in annual programming recalls. The charges for each appointment type, concession rates for third party payers, case mix, fixed and variable overhead were placed in an excel-based matrix to calculate the revenues generated per year. The model was also accurate for predicting the impact of patient volume changes or changes in FTE utilization on the clinic profitability. The model was adaptable to a private office setting as well.

Conclusions: This interactive model is transferable to CI programs in varied settings, can determine current program financial status, and can be used in making decisions to expand space or personnel with varied patient volumes.

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Implanting Common Cavity Malformations Using Intraoperative Fluoroscopy

Daniel H. Coelho, MD; Susan B. Waltzman, PhD J. Thomas Roland, Jr., MD

Objective: At the end of the presentation, the listener should be able to summarize the etiology of the common cavity cochlear malformation and the implications for implantation. Successful techniques of intraoperative fluoroscopy will be discussed. Furthermore, clinical and audiometric outcomes will be compared between those patients who did and did not undergo fluoroscopically assisted implantation.

Study design: Retrospective case review

Setting: Tertiary referral center

Patients: Patients with bilateral severe-profound SNHL with common cavity cochlear malformations who received cochlear implants at our institution.

Intervention: Therapeutic

Main outcome measure: Proper positioning of electrodes in the common cavity, postoperative audiometric outcomes

Results: 17 common cavities were implanted in 12 patients. 5 were performed without intraoperative fluoroscopic guidance. Those implanted with fluoroscopic guidance had lower rates of electrode malposition (extracochlear, intrameatal, kinking, bending) and likely lower rates of insertional trauma to the adjacent malformed neuroepithelial elements. Speech performance also appears to be improved in the fluoroscopically assisted group.

Conclusions: Intraoperative fluoroscopy is a useful and beneficial adjunct in the implantation of the common cavity patient.

IBR#: 07-396

Foreign Body Granuloma of the Inner Ear Following Cochlear Implantation: One Possible Cause of a Soft Failure?

Joseph B. Nadol, Jr., MD; Donald K. Eddington, PhD Barbara J. Burgess

Objective: Evaluate the intracochlear biologic response to cochlear implant

Study Design: Retrospective study of human temporal bone specimens

Setting: Tertiary referral center patients.

Patients: Specimens from patients who in life had undergone cochlear implantation

Interventions: Cochlear implantation

Main outcome measure: Review of the light microscopic histopathology of the biologic response within the inner ear near the cochlear implant track.

Results: The sentinel case is that of a 71-year-old man who had undergone a cochlear implantation of the right ear 13 years before death and of the left ear on two occasions, 12 and 11 years before death. In the right ear there was evidence of a postoperative "soft failure," that is deteriorating function as measured audiometrically without clear evidence of device malfunction. Thus, eight months following implantation he had a CID sentence score of 78% which fell to 18% one year after implantation. His short-term ability to use the telephone with the device was lost over the same period of time and new onset of stimulation of the facial nerve required elimination of some electrodes from the simulation map. Because of poor performance using the right cochlear implant, he underwent contralateral left cochlear implantation. Performance on the left side was consistently poor and he began to develop facial nerve stimulation on the left as well. Because of this poor performance without evidence of device failure, the implant was explanted and reimplanted with no improvement in function. At each of the three cochlear implant procedures all electrodes were inserted within the cochlea. In both temporal bones there was an intense foreign body giant cell, necrotizing granulomatous reaction apparently to the device. It was most intense along the track of the device, both within the mastoid and in the inner ear. Osteolysis was induced in the otic capsule, including between the otic capsule and the geniculate ganglion. There was severe degeneration of the organ of Corti and spiral ganglion cells. In temporal bone specimens from other patients who in life had undergone cochlear implantation evidence of cellular immune response in the form of foreign body giant cells and monocytic cell infiltrate was present in over 50% of specimens.

Conclusion: It is hypothesized that this reaction represents a foreign body response to the cochlear implant. Such an "allergic" response has been previously reported in the literature. Although the majority of soft failures of cochlear implant are remedied by a revision procedure, this is not always the case, and in rare cases such a granulomatous response may be responsible for deteriorating audiometric results.

Acknowledgements: With the support of NIH/NIDCD grant # RO1 DC000152-27

IBR#: IRB approval #01-03-014X

Hearing-in-Noise Benefits Following Bilateral Simultaneous Cochlear Implantation Continue to Improve Four Years after Implantation

Rose J. Eapen, MD; Emily Buss PhD; Marcia S. Clark AuD Harold C. Pillsbury III MD; Craig A. Buchman MD

Objective: The purpose of this study was to quantify the benefits of simultaneous bilateral cochlear implantation over four years of use, with special emphasis on the ability to use spatial cues to improve understanding of speech in noise.

Design: We present data from a prospective study of nine patients who were bilaterally implanted with the MED-EL COMBI 40+ at the University of North Carolina, Chapel Hill (UNC-CH). These patients were all post-lingually deafened, had a short preoperative duration of deafness and the implantation was performed in a single-stage surgery. Patients underwent evaluation preoperatively and each year for four years after surgery. Speech perception was assessed with CNC words presented in quiet and CUNY sentences presented in noise. Speech stimuli were presented directly to the implants via direct audio input. Head related transfer functions were used to simulate spatial displacement of CUNY and noise sources: speech was presented at midline (0°) and the noise masker was presented at either side or midline (-90°, 0° or +90°).

Results: Binaural benefits related to the head shadow and summation were demonstrated soon after implantation and remained robust over the course of the study. Squelch was not observed until one year following implantation and continued to increase in magnitude over the four-year study period.

Conclusions: While the head shadow and summation effects may be evident early after implantation, benefits associated with squelch continue to increase over a protracted time period post implantation. Outcome data on this cohort suggest continued integration of signals from both ears resulting in improved performance for both CNC words in quiet and CUNY sentences in noise.

Acknowledgements: The study was supported, in part, by MedEl Corp

IBR#: 01-SURG-503

Cochlear Implant and Hearing Aid: A New Approach to Optimize the Fitting in This Bimodal Situation

Annerose M. Keilmann, Prof. Dr. med. Andrea Bohnert, MTAF; Jan Gosepath, Dr. med. habil. Wolf J. Mann, Prof. Dr. dr. h.c. mult.

Objective: With extending indications for cochlear implants (CI) more and more children and adults with residual hearing on the contralateral side become candidates for CI-surgery. The major benefits of regular binaural hearing are spatial hearing, localization, and signal source discrimination in quiet as well as in noisy surroundings. In most reports, hearing aid fitting was carried out without balancing both devices. Only Ching et al. (2006) adjusted the hearing aid using the NAL-procedure and a 3-step loudness balancing procedure.

Study design: prospective clinical study

Setting: Tertiary referral center, ambulatory

Patients and interventions: Nine children and 7 adults with residual hearing on the non-operated side were binaurally fitted. Our procedure was based on the DSL [i/o]- method (DSL = desired sensation level, Seewald et al., 1992) A loudness-scaling was used for adjusting the loudness perception monaurally as well as for balancing the loudness of both devices. Speech audiometry in quiet and in noise was conducted both monaurally and in the bimodal mode. The fitting was modified according to the respective test results.

Main outcome measures: practicability of the procedure, amelioration of the patients speech perception

Results: In some younger children loudness scaling or speech audiometry was impossible to perform. In 9 children and 6 adults, a measurable gain or a subjective improvement of speech perception was achieved. One adult patient was unable to conduct the loudness scaling with the CI, and her situation could not be improved.

Conclusion: A structured bimodal fitting using loudness scaling for both, the cochlear implant and the hearing aid results in a subjective and often objective amelioration of the patient's hearing and speech perception.

Objective Measures of Cochlear Stimulation through the Round Window

Herman A. Jenkins, MD; James R. Easter, MS, ME Brian M. Conn, BS, MBA; James F. Kasic, MS, MBA

Hypothesis: Mechanical displacement of the round window membrane is a feasible alternative for cochlear stimulation.

Background: Conductive hearing loss arises from a broad variety of causes. Reconstruction of diseased or injured ossicles has been the tradition for restoration of function. Direct mechanical stimulation of the cochlea offers the potential for hearing restoration in situations in which ossicular reconstruction is not feasible or has met with failure previously.

Methods: The cochleae of human temporal bones were subjected to vibratory stimuli through the round window, and the resultant mechanical responses measured. Vibration was applied by a spherical probe tip in contact with the round window membrane, both with and without an intervening layer of fascia. Reflected stapedial velocity, measured with a laser vibrometer, was used as a proxy for sensation level in response to stimulation.

Results: Stapes velocity was comparable to levels observed for acoustic stimulation in normal middle ears. Transmission efficiency was lower than for direct mechanical stimulation of the ossicles. Insertion of a layer of fascia between probe tip and membrane did not affect transmission efficiency at its peak frequency, but did improve transmission at lower frequencies by approximately 15 dB.

Conclusions: Objective measures of cochlear response to direct stimulation through the round window suggest that this approach is a viable therapeutic approach for conductive and mixed hearing loss.

The Round Window Implant: The Last Chance for Hearing Restoration in Mixed Hearing Losses

Vittorio Colletti, MD; Marco Carner, MD Sheila Veronese, PE; Liliana Colletti, PhD

Objective: The indications of the Vibrant Med-El Soundbridge (VMSB), presently limited to the patients with sensorineural hearing loss (SNHL) and normal middle ear function, have been extended to include patients with mixed hearing loss due to congenital or acquired ossicular chain defects. The FMT of the VMSB placed onto the RW, round window implant (RWI), has allowed optimal amplification in patients with moderate-tosevere mixed hearing losses who, at present, do not have good options for adequate functional rehabilitation.

Study design: retrospective case review.

Setting: tertiary referral center.

Patients: Twenty-six patients were unsuitable candidates for air and bone conductive hearing aids (BCHAs and ACHAs) and osseointegrative implants (BAHAs) and were treated with RWI. The patients were subdivided in three groups in relation to the etiologies and the performed surgeries. The first group includes 14 adults with unsuccessful OPLs (9 canal-wall-up (CWU) and 5 canal-wall-down (CWD) tympanoplasty); the second group includes 8 adult patients with CSOM, operated on dry radical or modified RCs and the third group includes, 4 patients, 2 children aged 1 and 10 year-old, and 2 adults with severe congenital malformations of the auricle combined with atresia of the outer ear canal and malformations of the ossicular chain.

Intervention(s): RWI surgeries will be illustrated in detail for each group.

Results. Significant improvements were observed in pure-tone threshold and speech understanding after surgery and at different follow-ups (12 -24 months) with outcomes very similar to stapes surgery and with no complications.

Conclusions. The post-operative results suggest that RWI offers a viable treatment option for patients with severe mixed hearing losses regardless of the etiology of hearing loss and previous surgeries. The RWI bypasses the normal conductive pathway to the cochlea by delivering vibratory energy directly to the cochlea via the RW and allows to compensate for the conductive and sensorineural component. No extrusion or dislocation have been so far observed. This approach is indicated and ideal for patients with: a) bilateral conductive mixed hearing loss for surgical failures of OPLs (cw up/down

techniques; stapedoplasty revision surgery for incus necrosis; b) radical and modified radical mastoidectomies; c) external and/or middle ear malformations; d) tympanomastoid obliteration procedures; e) patients with SNHL obtaining limited benefit from MEI.

The EarLens System: An Innovative Sound Transduction Method

Rodney Perkins, MD; Jonathan P. Fay, PhD Michael T. Murray, MD; Lisa Olson, MS; Sunil Puria, PhD

Problem addressed: The EarLensTM is an innovative method of sound transduction. A thin silicone platform with an embedded magnet is placed on the eardrum. Vibrating the EarLens with a magnetic field creates the perception of sound. The unique characteristics of the EarLens system address the most common problem experienced by the hearing impaired: discerning a target speaker in a multi-talker environment.

Methods and Measures: Sixteen subjects (normal to moderate hearing impairment) wore the EarLens over a six month period. The subjects were monitored for any adverse reaction to the EarLens. Four key functional characteristics of the EarLens system were measured: insertion loss, frequency response, maximum output, and feedback thresholds.

Results: The EarLens remained in place and was well tolerated. The EarLens system had sufficient output to treat 60 dB of hearing impairment up to 8 kHz in 84% of the population and up to 11.2 kHz in 50% of the population. The average insertion loss was less than 3 dB. Even with the microphone within the ear canal and without feedback canceling software, the feedback gain margin was better than 10 dB.

Conclusion: The EarLens system has a wide effective bandwidth and little feedback which opens up possibilities for a unique hearing system. The EarLens system allows an intra-canal microphone to be used while maintaining an open canal. This configuration will provide the high frequency pinna diffraction cues (>6kHz) to facilitate better hearing in multi-talker environments.

Acknowledgements: Supported in part by a Phase I SBIR grant from the NIDCD of NIH.

IRB#: 20061405

29. Hearing Restoration: Improved Multi-talker Speech Understanding

Sunil Puria, PhD; Andy Vermiglio, MS Jonathan Fay, PhD; Sig Soli, PhD

Hypothesis: Conventional acoustic hearing devices for sensory-neural hearing impairment are typically limited to frequencies below 4-5 kHz. Some directional cues used by the brain to spatially separate sources of speech are at frequencies above 5 kHz. This study tests the hypothesis that when hearing restoration is extended to 10 kHz, subjects perform better in environments where many people are speaking simultaneously (multi-talker speech).

Methods: The speech reception threshold (SRT) of target speech was measured with interfering speech in front (0 degrees) and bilaterally symmetric locations (± 30 degrees). Measurements were made with male speech low-pass filtered at: 4, 6, 8, and 10 kHz. All testing on normal (n=13) and hearing impaired (n=6) subjects was conducted using virtual acoustics with headphones.

Results: For the normal hearing subjects, speech recognition improved by 16%, 21%, and 26% for bandwidths of 6, 8 and 10 kHz, respectively, when compared to 4kHz. The differences are statistically significant (p<0.02). For the hearing impaired subjects, the corresponding improvements are 14%, 19% and 16%, with a trend towards improvement with increasing bandwidth. Most of the improvements are due to spatial release from masking.

Conclusions: The results indicate that providing the high frequency directional cues allows the brain of both normalhearing and hearing-impaired subjects to segregate different sources of speech and perform better in multi-talker situations. This study suggests that future hearing devices that provide a wide bandwidth response may enhance speech discrimination in multi-talker environments.

Acknowledgements: Work supported in part a grant from the NIDCD of NIH.

IBR#: 06-019

Vibrant Soundbridge Implantable Hearing Device: Long- and Short-term Results

Charles M. Luetje, MD; Sandra A. Brown, MA, CCC-A Robert D. Cullen, MD

Objective: To evaluate the long- and short-term hearing results of the Vibrant Soundbridge

Study Design: Retrospective descriptive study of a consecutive series of patients implanted by a single surgeon from 1996-present.

Setting: Independent tertiary referral otologic center.

Patients: Thirty-one patients with 34 ears implanted who met the criteria for and elected to receive Vibrant Soundbridge implantation. Nineteen men and 12 women were implanted at a mean age of 55.6 years. FDA-study patients and non-study patients were included.

Intervention: Preoperative evaluation, Vibrant Soundbridge implantation, subsequent programming, and diagnostic testing.

Main Outcome Measures: Long- and short-term gain from the Vibrant Soundbridge as measured by conventional audiometry and long-term benefit as measured by the Abbreviated Profile of Hearing Aid Benefit [APHAB].

Results: In all 31 patients, there was no postoperative change in residual hearing at the 2-month initial programming. Seventeen patients [18 ears] were available for follow-up testing with a mean follow-up period of 7.4 years. Of the remaining 14 patients, four patients were unavailable due to geographic circumstances, five have not committed to device testing, and five were lost to follow-up. Initial and long-term gain in hearing as well as benefit obtained with the Vibrant Soundbridge will be described.

Conclusion: Direct drive hearing with the Vibrant Soundbridge is beneficial and provides sustained audiometric gain. Lack of access to local audiological support as well as direct and indirect device costs significantly impact device use and patient follow-up. Without support, some patients may choose to become non-users, select conventional hearing aid amplification, and exhibit apathy regarding hearing improvement.

Successes and Complications of the BAHA System

Jack J. Wazen, MD; Dayton Young, MD

Objective: The purpose of this study is to review the outcomes of the BAHA procedure and to review the success and complication rates. The types of complications are analyzed with suggestions of prevention and management.

Study design: This is a retrospective study.

Setting: Tertiary care. Chart review, and patient questionnaire.

Patients: All adult patients with conductive, mixed, and sensorineural hearing loss age 18-85 implanted between July 2004 and July 2007 were reviewed. Two hundred patients were analyzed, equally divided between males and females.

Intervention: Auditory rehabilitation with the titanium retained auditory implant (BAHA system)

Main outcome measures: 1) Titanium retention and implant loss.2) soft tissue reactions and complications. 3) patient satisfaction with the device. 4) revision surgery.

Results: All patients reviewed were successfully implanted with no incidence of implant loss or bony complications. There was no incidence of bony infection, osteitis, osteomyelitis, meningitis, or brain abscess. The soft tissue reactions were graded from I-III. Only one patient needed a revision due to thick hypertrophied scars around the implant site. Two patients needed the evacuation of a hematoma in the office without loss of the graft. Minor soft tissue inflammation at the implant interface occured in 8% of the patients requiring only topical care. One patient reported dissatisfaction with the device. The rest reported high satisfaction and continue regular use of the device

Coclusions: The BAHA system is a safe and effective system in the rehabilitation of patients with conductive or mixed hearing losses and with single sided deafness. The high success rate , patient satisfaction rate, and predictable auditory outcome place the BAHA system among the top choices for auditory rehabilitation

IBR#: pending

Interim Progress Report for the American Otological Society Research Grants

Research Progress Report Yu-Lan Mary Ying, MD

The objective of this project is to understand the molecular responses of mitochondrial Manganese Superoxide Dismutase (Mn SOD2) in spiral ganglion cells to oxidative stress. The action of Mn SOD2 is a critical step in the signaling cascade that activates mitochondrial Uncoupling Proteins (UCPs), which reduces ROS production by decreasing the protonmotive force across the mitochondrial membrane. Preliminary studies showed that Mn SOD2 expression in spiral ganglion cell is lower at the cochlear base than at the apex. We hypothesize the base to apex gradient in spiral ganglion cell Mn SOD2 expression reflects responses to different ROS load across the cochlear spiral.

To determine if Mn SOD expression in spiral ganglion cells is regulated in response to exogenous ROS load and antioxidant treatment, we employed an adult wild type C57 BL/6J mouse kanamycin administration model for acute ROS exposure. A two-by-two factorial design assesses effects of kanamycin (700 mg/kg) and antioxidant 2,3-dihydroxybenzoate (DHB) (300 mg/kg) treatments versus control vehicles (saline (S) and sodium bicarbonate (NaHCO₃)).

Three sets of mice (12 mice per set with 3 mice divided into each four treatment groups; total number = 36 mice) have been treated for 15 consecutive days of twice daily subcutaneous injections of the respective drug treatment. A comparison of pre- and post-treatment evoked auditory brain stem responses (ABRs) showed marginal kanamycin treatment effect at 16K Hz (21.0 ± 6.5 dB threshold shift from pre-treatment, mean from left and right ears, p = 0.06 relative to the control group of saline and sodium bicarbonate ($1.3 \pm$ 7.2 dB shift)). The effect was attenuated by concurrent DHB treatment (10.6 ± 7.2 dB shift). No significant ABR threshold shifts appeared for click or 4 Hz stimuli. These data confirm expectations for the animal model.

Each mouse was perfused transcardially with PLP fixative. Eight micron thick temporal bone sections in the axial plane are currently been prepared on slides for immunohistochemistry study of Mn SOD2 to determine if its expression in spiral ganglia varies across the four treatment groups, in response to exogenous ROS load and antioxidant treatment conditions.

Another four sets of mice (12 mice per set with 3 mice divided into each four treatment groups; total number = 48 mice) underwent similar drug treatment protocol for molecular biology studies. The cochleae of these mice were

microdissected to harvest the spiral ganglion cells, organ of corti and stria vascularis for real-time quantitative PCR assays. The kidney and liver (i.e., positive and negative tissue controls) from each mouse were harvested for comparative studies since aminoglycoside is known to affect the kidney but not the liver. RNA extraction has been performed on the kidney and liver samples from all four treated mouse groups.

This project provides a starting-point in developing a working model that will help us better understand the kinetics of Mn SOD2 activity and its downstream effect on other protective enzymes (i.e., UCPs) in the free radical cascade. Furthermore, we will explore the preliminary observation of Mn SOD2 expression as a function of regional differences in the cochlea. Delayed Mn SOD2 generation may be a factor in the relative vulnerability of the cochlear base to ROS damage.

Progress Report for Vincent Y.W. Lin AOS Research Training Fellow

The specific goal of this grant is to characterize *in vivo* the extent to which direct transdifferentiation as the sole mechanism for hair cell regeneration leads to functional recovery of hearing after drug-induced hair cell loss. During the past 18 months, I have developed a novel *in vivo* model to study direct transdifferentiation using cytarabine (Ara-C). Ara-C is a DNA chelator and inhibitor of DNA polymerase that also causes mitotic cells to undergo apoptosis. Characterization studies using a dose of 0.5% Ara-C delivered at a rate of 0.5 uL/hr effectively significantly halted mitotic division in lesioned avian basilar papillae while keeping systemic toxicity minimal. I have also confirmed the presence of hair cell regeneration via direct transdifferentiation in birds that were allowed to survive 28 days after a single subcutaneous gentamicin injection.

Immunocytohistochemical (ICC) analyses of the basilar papillae of these birds demonstrate near-complete regeneration of the basilar papillae, little bromodeoxyuridine (BrdU) labeling and excellent morphology of regenerated hair cells. These results confirm that with our *in vivo* model, the regenerative response is limited predominantly to direct transdifferentiation.

The next phase of our study was to test for functional hearing recovery by measuring auditory evoked brainstem responses (ABR). Groups of birds were surgically implanted with either an Alzet osmotic pump containing Ara-C or saline (control). Birds were lesioned with a single gentamicin injection. ABR measurements were recorded every seven days. There was no statistical difference in the ABR thresholds in both groups prior to pump implantation. After gentamicin lesioning, there was an expected drop in hearing thresholds. Hearing levels post-gentamicin was not statistically significant between both groups. Our preliminary results suggest that in comparison with birds implanted with a saline pump. Ara-C treated birds tend to recover more slowly and less completely at the tested time points. The onset of hearing recovery in both groups appears to be delayed by several weeks after known morphological recovery. I will continue to accrue data to reduce our variability of our data. ICC analysis on the basilar papillae of implanted birds will also be performed to correlate with our ABR data.

Identification of a Genetic Contribution to Meniérè's Disease – Progress Report PI: Richard Smith

The specific aims of this proposal are to identify major genetic associations in Ménière's disease (MD) by completing candidate gene association studies and a genome-wide association (GWA) study using the Affymetrix Genome-Wide SNP Array 6.0 (1 million SNP chip).

Our candidate gene association studies have focused on a number of genes including *KCNE1* and *KCNE3* reportedly associated with MD in Japanese subjects (Doi et al. 2005). Consistent with this possibility, these two genes encode potassium channels that are expressed in the stria vascularis and endolymphatic sac, respectively. Their role in ion transport suggests that they may be important in inner ear homeostasis.

To establish whether SNPs in these two genes are associated with MD in a Caucasian population, we sequenced the coding regions and intronexon boundaries of both genes in 180 persons with MD and compared results to 180 age-matched controls. Neither of the two SNPs reported by Doi et al was significantly associated with MD in Caucasians (KCNE1, p= 0.64661773; KCNE3, p= 0.9226549). We then looked at eight additional SNPs in KCNE1 and nine additional SNPs in KCNE3, and none of these SNPs was associated with the Caucasian MD phenotype. Interestingly, comparison of allele frequencies between the Japanese MD population and our MD population revealed no significant differences between groups (KCNE1, p= 0.72; KCNE3, p=0.40), suggesting that the significant differences reported in the Japanese study arose from their control population. Consistent with this possibility, we found allele frequencies between the two control populations to be significantly different (KCNE1, p<0.0001; KCNE3, p=0.0008). Furthermore, comparison of the KCNE3 SNP allele frequency between the control population used by Doi et al and the HapMap JPT population is highly significant (p<0.0001). These results suggest that the association reported between SNPs in KCNE1 and KCNE3 and MD in the Japanese population may reflect substructure within the control Our data show that SNPs in KCNE1 and KCNE3 are not group. associated with MD in Caucasians.

Our second goal is to complete a genome-wide association study using the Affymetrix Genome-Wide SNP Array 6.0. While a GWA study is not hypothesis driven in the sense that candidate genes are not selected for screening, no presumptions are made regarding the pathogenesis of MD - the entire genome is screened to identify major genetic associations. We have made arrangements to collaborate with TGen on this effort and plan to complete analysis of ~250 study subjects and controls in early January, 2008. We are typing each subject on the Affymetrix 6.0 Array as opposed to using a pooling strategy as the former approach allows us to compare all data across cohorts as additional subjects are recruited into the study.

In the short term, we expect the results of these studies to identify many of the major genes that play a role in the etiology of MD. In the long term, our goal is to develop better treatments for MD based on the genetics and pathophysiology of this disease.

I would like to gratefully acknowledge the collaborations associated with this research. In particular J.P. Carey, C.C. Della Santina, B.J. Gantz, M. Hansen, H. Najmabadi, N.B. Smith, D. Stephan, E.M. Stone, L.B. Minor, and G. Van Camp have made this research possible. C.A. Campbell is focusing on Ménière's disease for her graduate thesis in the Genetics PhD Program at the University of Iowa. I am extremely grateful for the generous support provided by the Research Fund of the American Otological Society.

Optimization of Local Drug Delivery to the Cochlea Progress Report PI: Anthony Mikulec, MD

The goal of this study is to develop procedures to optimize drug entry into the cochlea following local application to the round window membrane. We hypothesize that round window membrane (RWM) permeability can be manipulated to allow drugs to more easily and reliably enter the fluid spaces of the inner ear. Currently used local treatment methods for inner ear disorders use medication concentrations and delivery protocols dictated mostly by convenience and anecdotal experience. Agents delivered to the human cochlea through the round window include gentamicin for the treatment of Meniere's syndrome and steroids for the treatment of sudden sensorineural hearing loss, Meniere's syndrome, and tinnitus.

During the first year of the study, round window membrane permeability was evaluated in the guinea pig using the ionic marker TMPA (trimethyphenylammonium). The rate of entry of TMPA into scala tympani was quantified using TMPA-selective microelectrodes sealed into the scala. Marker entry was quantified before and after experimental treatments, permitting the effect of treatments to be assessed in individual animals and minimizing the influence of interanimal variations in RWM permeability. Marker was applied by RW irrigation using a syringe pump at a continuous rate of 5 µL/min. Maintaining a constant middle ear concentration by irrigation allowed changes of TMPA entry rate to be interpreted in terms of RWM permeability alterations. Round window treatments were chosen based on deviations from physiologic values of current clinically utilized drugs. Osmolarity variations and benzyl alcohol concentration (a preservative in some drug preparations) were recreated in TMPA-containing solutions irrigated across the RWM and permeability changes were assessed. The effect of applying suction near the RWM (#3 French) was also similarly evaluated. Results show that suction applied near the RWM results in an approximate doubling of round window permeability, while the presence of benzyl alcohol increases permeability by a factor of about 1.5. Osmolarity changes increased RWM permeability to a small extent. The increased RWM permeability seen with benzyl alcohol and suction have immediate applicability for clinical protocols.

During the second year of the study we have investigated drug entry into the cochlea when the entire middle ear was filled with drug solution, a technique that is widely used. Perilymph drug levels were measured with an apical sampling method, in which ten individual 1 µL samples are taken from an apical cochleostomy. For both TMPA marker and gentamicin, we have found very high concentrations near the apex, demonstrating that drugs can enter perilymph through the thin bony capsule of the guinea pig. This discovery effects the interpretation of numerous studies examining the extent of cochlear distribution of drugs as well as the applicability of animal experiments to humans. In addition, we have shown that the concentration of drug in the bulla following bolus application is variable, with drug clearance from the middle ear significantly influencing intracochlear concentrations. Presently, we are evaluating the performance of wick based systems for drug delivery to the round window.

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1890	O.D. Pomeroy, MD	1973	Ben H. Senturia, MD
1891-94		1974	Wesley H. Bradley, MD
1895-99		1975	Lester A. Brown, MD
1900-02		1976	Victor Goodhill, MD
1903-05 1906-07	·····,·	1977	Harold Schuknecht, MD
1908-07	C.J. Kipp, MD	1978	Clair M. Kos, MD
1908	C.J. Kipp, MIJ	1979	G. Dekle Taylor, MD
1909-10	Frederick L. Jack, MD Edward B. Dench, MD	1980	Eugene Derlacki, MD
1913-14	J.F.McKernon, MD	1981	Richard J. Bellucci, MD
1915-14	C.W. Richardson, MD	1982 1983	J. Brown Farrior, MD
1915-10	C.R. Holes, MD	1985	Jack V. Hough, MD Cary N. Moon, Jr., MD
1918	Norval H. Pierce, MD	1985	Francis A. Sooy, MD
1919	Ewing W. Day, MD	1986	Brian F. McCabe, MD
1920	Robert Lewis, MD	1987	Harold G. Tabb, MD
1921	W.P. Eagleton, MD	1988	Rihard R. Gacek, MD
1922	H.S. Birket, MD	1989	D. hane Cody, MD
1923	G. Shambaugh, Sr., MD	1990	H.A. Ted Bailey, Jr., MD
1924	John B. Rae, MD	1991	William F. House, MD
1925	E.A. Crockett, MD	1992	Mihael Glasscock, III, MD
1926	Thomas J. Harris, MD	1993	Mansfield F.W. Smith, MD
1927	Arthur B. Duel, MD	1994	Robert I. Kohut, MD
1928	M.A. Goldstein, MD	1995	Robert A. Jahrsdoerfer, MD
1929	J.G. Wilson, MD	1996	Derald E. Brackmann, MD
1930	S. Mac C. Smith, MD	1997	Joseph C. Farmer, Jr., MD
1931	D.H. Waler, MD	1998	Charles M. Luetje, MD
1932	L.W. Dean, MD	1999	Gregory J. Matz, MD
1933 1934	G.I. Tobey, Jr., MD	2000	C. Gary Jackson, MD
1934	John R. Page, MD	2001	A. Julianna Gulya, MD
1935	Samuel J. Crowe, MD F.R. Packard, MD	2002	Richard A. Chole, MD PhD
1930	E.P. Fowler, MD	2003	Horst R. Konrad, MD
1938	Harris P. Mosher, MD	2004	Jeffrey P. Harris, MD, PhD
1939	Isidore Friesner, MD	2005 2006	Sam E. Kinney, MD
1940	Horace Newhart, MD	2000	John K. Niparko, MD Antonio De La Cruz, MD
1941	George M. Coates, MD	2007	
1942	L. M. Seydell, MD		
1943-44	W.C. Bowers, MD		
1945-46	Gordon Berry, MD		
1947	William E. Grove, MD		
1948	B. J. McMahon, MD		
1949	Marvin F. Jones, MD		
1950	Philip E. Meltzer, MD		
1951	Kenneth M. Day, MD		
1952	Gordon D. Hoople, MD		
1953	A.C. Furstenberg, MD		
1954 1955	Frederick T. Hill, MD		
1955 1956	D.E.S. Wishart, MD		
1956 1957	William J McNally, MD		
1957	John R. Lindsay, MD Dean M. Lierle, MD		
1959	Moses H. Lurie, MD		
1960	Robert C. Martin, MD		
1961	Henry L. Williams, MD		
1962	Lawrence R. Boies, MD		
1963	Joseph A. Sullivan, MD		
1964	Theodore E. Walsh MD		

I

PAST SECRETARY-TREASURERS OF AMERICAN OTOLOGICAL SOCIETY

1868-1870	C. E. Ryder, MD
1870-1879	J. O. Green, MD
1879-1898	J. J. B. Vermyne, MD
1898-1907	Frederick L. Jack, MD
1907-1912	James F. McKernon, MD
1912-1917	John B. Rae, MD
1917-1919	George E. Shambaugh, MD
1919-1925	Thomas J. Harris, MD
1925-1927	D. Harold Walker, MD
1927-1940	Thomas J. Harris, MD
1940-1945	Isidore S. Friesner, MD
1945-1950	Gordon D. Hoople, MD
1950-1955	John R. Lindsay, MD
1955-1960	Lawrence R. Boies, MD
1960-1965	James A. Moore, MD
1965-1972	Wesley H. Bradley, MD
1972-1977	G. Dekle Taylor, MD
1977-1982	Cary N. Moon, Jr., MD
1982-1987	D. Thane Cody, MD
1987-1992	Robert I. Kohut, MD
1992-1997	Gregory J. Matz, MD
1997-2002	Horst R. Konrad, MD
2002-2007	Clough Shelton, MD
2007-	Paul R. Lambert, MD

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ACTIVE MEMBERS

Ronald G. Amedee, MD (Active 1995) New Orleans, LA

Patrick J. Antonelli, MD (Active 2001) Gainesville, FL

Edward Applebaum, MD (Active 1985) Chicago, IL

Mosies A. Arriaga, MD (Active 2002) Pittsburgh, PA

H. Alexander Arts, MD (Active 2001) Ann Arbor, MI

Douglas D. Backous, MD (Active 2006) Seattle, WA

Thomas J. Balkany, MD (Active 1991) Miami, FL

David M. Barrs, MD (Active 1997) Phoenix, AZ

Loren J. Bartels, MD (Active 1992) Tampa, FL

Carol A. Bauer, MD (Active 2006) Springfield, IL

Charles W. Beatty, MD (Active 1995) Rochester, MN

James E. Benecke, Jr., MD (Active 2006) St. Louis, MO

Brian Blakley, MD (Active 1996) Canada

Derald E. Brackmann, MD (Active 1979) Los Angeles, CA

Hilary A. Brodie, MD, PhD (Active 2001) Davis, CA

Patrick Brookhouser, MD (Active 1988) Omaha, NE

Craig A. Buchman, MD (Active 2005) Chapel Hill, NC

Rinaldo F. Canalis, MD (Active 1991) Santa Monica, CA

John P. Carey, MD (Active 2006) Baltimore, MD

Stephen P. Cass, MD (Active 2000) Denver, CO

Margaretha L. Casselbrant, MD (Active 2001) Pittsburgh, PA Sujana S. Chandrasekhar, MD (Active 2004) New York, NY

Douglas A. Chen, MD (Active 2008) Pittsburgh, PA

Steven Wan Cheung, MD (Active 2006) San Francisco, CA

Richard A. Chole, MD, PhD (Active 1984) St. Louis, MO

Daniel Choo, MD (Active 2008) Cincinnati, OH

Newton J. Coker, MD (Active 1991) Santa Fe, NM

Roberto A. Cueva, MD (Active 2005) San Diego, CA

C. Phillip Daspit, MD (Active 1995) Phoenix, AZ

Antonio De La Cruz, MD (Active 1991) Los Angeles, CA

M. Jennifer Derebery, MD (Active 2002) Los Angeles, CA

John R.E. Dickins, MD (Active 1991) Little Rock, AR

John L. Dornhoffer, MD (Active 2004) Little Rock, AR

Karen Jo Doyle, MD, PhD (Active 2002) Sacramento, CA

Larry G. Duckert, MD (Active 1988) Seattle, WA

Thomas L. Eby, MD (Active 1995) Jackson, MS

Hussam K. El-Kashlan, MD (Active 2006) Ann Arbor, MI

John R. Emmett, MD (Active 1990) Memphis, TN

John M. Epley, MD (Active 2001) Portland, OR

Joseph C. Farmer, Jr., MD (Active 1984) Durham, NC

Jay B. Farrior, III, MD (Active 1990) Tampa, FL

Jose N. Fayad, MD (Active 2007) Los Angeles, CA

Joseph G. Feghali, MD, FACS (Active 2002) Bronx, NY

Howard W. Francis, MD (Active 2003) Baltimore, MD Rick Friedman, MD, PhD (Active 2001) Los Angeles, CA

Michael H. Fritsch, MD (Active 2003) Indianapolis, IN

Bruce J. Gantz, MD (Active 1987) Iowa City, IA

Gerard J. Gianoli, MD (Active 2007) Baton Rouge, LA

Joel A. Goebel, MD (Active 1995) St. Louis, MO

Robert A. Goldenberg, MD (Active 1989) Dayton, OH

Richard L. Goode, MD (Active 1990) Stanford, CA

Marcos V. Goycoolea, MD, PhD (Active 1992) Las Condes, Santiago

J. Douglas Green, Jr., MD, MS (Active 2008) Jacksonville, FL

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Thomas J. Haberkamp, MD (Active 1997) Chicago, IL

Paul E. Hammerschlag, MD (Active 2001) New York, NY

Jeffrey P. Harris, MD, PhD (Active 1988) San Diego, CA

Barry E. Hirsch, MD (Active 1996) Pittsburgh, PA

Michael E. Hoffer, MD (Active 2003) San Diego, CA

Ronald A. Hoffman, MD (Active 1992) New York, NY

Karl L. Horn, MD (Active 2001) Albuquerque, NM

John W. House, MD (Active 1984) Los Angeles, CA

Gordon B. Hughes, MD (Active 1987) Cleveland, OH

Robert K. Jackler, MD (Active 1992) Stanford, CA

Carol A. Jackson, MD (Active 1994) Newport Beach, CA

Anthony Jahn, MD (Active 1992) Roseland, NJ

Herman A. Jenkins, MD (Active 1987) Aurora, CO

Timothy K. Jung, MD (Active 1990) Riverside, CA Bradley W. Kesser, MD (Active 2008) Charlottesville, VA

Barry P. Kimberley, MD (Active 2001) Minneapolis, MN

Sam E. Kinney, MD (Active 1981) Moreland Hills, OH

Richard D. Kopke, MD (Active 2005) Oklahoma City, OK

Arvind Kumar, MD (Active 1993) Hinsdale, IL

Anil K. Lalwani, MD (Active 1999) New York, NY

Paul R. Lambert, MD (Active 1995) Charleston, SC

John P. Leonetti, MD (Active 1995) Maywood, IL

S. George Lesinski, MD (Active 1993) Cincinnati, OH

Samuel C. Levine, MD (Active 1999) Minneapolis, MN

Christopher J. Linstrom, MD (Active 2003) New York, NY

Charles M. Luetje, MD (Active 1991) Kansas City, MO

Lawrence R. Lustig, MD (Active 2006) San Francisco, CA

Charles A. Mangham, Jr., MD (Active 1987) Seattle, WA

Robert H. Mathog, MD (Active 1985) Detroit, MI

Douglas E. Mattox, MD (Active 1992) Atlanta, GA

John T. McElveen, Jr., MD (Active 1997) Raleigh, NC

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Michael J. McKenna, MD (Active 1999) Boston, MA

Cliff A. Megerian, MD (Active 2006) Cleveland, OH

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Terrence P. Murphy, MD (Active 2002) Atlanta, GA

Joseph B. Nadol, Jr., MD (Active 1988) Boston, MA

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John K. Niparko, MD (Active 1995) Baltimore, MD

Dennis G Pappas, Jr., MD (Active 2004) Birmingham, AL

Blake C. Papsin, MD (Active 2005) Toronto, Ontario, CANADA

Simon C. Parisier, MD (Active 1982) New York, NY

Lorne S. Parnes, MD (Active 2000) London, Ontario, CANADA

Steven M. Parnes, MD (Active 2002) Albany, NY

Myles L. Pensak, MD (Active 1992) Cincinnati, OH

Harold C. Pillsbury, MD (Active 1988) Chapel Hill, NC

Dennis S. Poe, MD (Active 1995) Boston, MA

G. Mark Pyle, MD (Active 2003) Madison, WI

Steven D. Rauch, MD (Active 2004) Watertown, MA

J. Thomas Roland, Jr., MD (Active 2005) New York, NY

Peter S. Roland, MD (Active 1992) Dallas, TX

Seth Rosenberg, MD (Active 2001) Sarasota, FL

Richard M. Rosenfeld, MD, MPH (Active 2004) Brooklyn, NY Allan M. Rubin, MD, PhD (Active 1997) Sylvania, OH Jay T. Rubinstein, MD, PhD (Active 2002) Seattle, WA Michael J. Ruckenstein, MD (Active 2003) Philadelphia, PA Leonard P. Rybak, MD, PhD (Active 1989) Springfield, IL Clarence T. Sasaki, MD (Active 1992) New Haven, CT Robert T. Sataloff, MD (Active 1990) Philadelphia, PA James E. Saunders, MD (Active 2008) Lebanon, NH Mitchell K. Schwaber, MD (Active 1993) Nashville, TN Michael D. Seidman, MD (Active 2001) West Bloomfield, MI Samuel H. Selesnick, MD (Active 1999) New York, NY Clough Shelton, MD (Active 1995) Salt Lake City, UT Herbert Silverstein, MD (Active 1973) Sarasota, FL Aristides Sismanis, MD (Active 1993) Richmond, VA Peter G. Smith, MD (Active 1988) St. Louis, MO Eric E. Smouha, MD (Active 2004) New York, NY Steven A. Telian, MD (Active 1997) Ann Arbor, MI Fred F. Telischi, MD (Active 2002) Miami, FL Norman Wendell Todd, Jr., MD (Active 1996) Atlanta, GA Debara L. Tucci, MD (Active 2000) Durham, NC Jeffrey T. Vrabec, MD (Active 2004) Houston, TX P. Ashley Wackym, MD (Active 1997) Milwaukee, WI Jack J. Wazen, MD (Active 1993) Sarasota, FL Peter C. Weber, MD (Active 2002) Cleveland, OH

D. Bradley Welling, MD, PhD (Active 1998) Columbus, OH

Stephen J. Wetmore, MD (Active 2001) Morgantown, WV

Richard J. Wiet, MD (Active 1987) Hinsdale, IL

David F. Wilson, MD (Active 1992) Portland, OR

Nancy M. Young, MD (Active 2007) Wilmette, IL

SENIOR MEMBERS

Kedar Adour, MD (Senior 1999 (1988)) San Francisco, CA

Professor P. W. Alberti, MD (Senior 2004 (1982)) Toronto, Ontario, Canada

Bobby R. Alford, MD (Senior 1997 (1970)) Houston, TX

Beverly Armstrong, MD (Senior 1988 (1960)) Charlotte, NC

H.A. Ted Bailey, Jr., MD (Senior 1994 (1969)) Little Rock, AR

F. Owen Black, MD (Senior 2006 (1983)) Portland, OR

Charles D. Bluestone, MD (Senior 2005 (1977)) Pittsburgh, PA

Roger Boles, MD (Senior 1999 (1982)) Woodside, CA

Wesley H. Bradley, MD (Senior 1988 (1961)) Glenmont, NY

Seymour J. Brockman, MD (Senior 1988 (1964)) Beverly Hills, CA

Richard A. Buckingham, MD (Senior 1994 (1969)) Wilmette, IL

Robert W. Cantrell, MD (Senior 2000 (1979) Charlottesville, VA

Francis I. Catlin, MD (Senior 1996 (1975)) Houston, TX

Jack D. Clemis, MD (Senior 2004 (1976)) Wilmette, IL

Noel L. Cohen, MD (Senior 2006 (1985)) New York, NY

D. Thane Cody, MD (Senior 1992 (1969)) Jacksonville, FL

James M. Cole, MD (Senior 1990 (1966)) Danville, PA

Wesley E. Compere, MD (Senior 1989 (1968)) LeMesa, CA James A. Crabtree, MD (Senior 1995 (1972)) San Marino, CA Vijay S. Dayal, MD (Senior 2001 (1975)) Chicago, IL Robert A. Dobie, MD (Senior 2005 (1985)) Sacramento, CA James A. Donaldson, MD (Senior 1994 (1974)) Richmond, WA Patrick J. Doyle, MD (Senior 1996 (1987)) Vancouver, BC Joseph G. Druss, MD (Senior 1971 (1939)) New York, NY Arndt J. Duvall III, MD (Senior 1993 (1971)) Minneapolis, MN Abraham Eviatar, MD (Senior 1999 (1981)) Scarsdale, NY George W. Facer, MD (Senior 2007 (1994)) Bonita Springs, FL John M. Fredrickson, MD (Senior 2002 (1978)) Albuquerque, NM Richard R. Gacek, MD (Senior 1998 (1969)) Worcester, MA L. Gale Gardner, Jr., MD (Senior 2004 (1983)) Shreveport, LA George A. Gates, MD (Senior 2005 (1987)) Boerne, TX Michael Glasscock III, MD (Senior 1997 (1973)) Austin, TX Malcolm D. Graham, MD (Senior 2001 (1979)) Atlanta, GA Irwin Harris, MD (Senior 1993 (1970)) Los Angeles, CA Cecil W.J. Hart, MD (Senior 2001 (1992)) Palm Springs, CA David A. Hilding, MD (Senior 1990 (1972)) Salt Lake City, UT Albert Hohmann, MD (Senior 1990 (1970)) New Brighton, MN Jack V.D. Hough, MD (Senior 1990 (1960)) Oklahoma City, OK William F. House, MD (Senior 1995 (1964)) Aurora, OR Robert A. Jahrsdoerfer, MD (Senior 2001 (1982)) Afton, VA Donald B. Kamerer, MD (Senior 2004 (1988)) Pittsburgh, PA

Athanasios Katsarkas, MD (Senior 2004 (1991)) Montreal, Qc, CANADA Robert I. Kohut, MD (Senior 1998 (1976)) Woodleaf, NC Fred H. Linthicum, Jr., MD (Senior 1991 (1967)) Los Angeles, CA William H. Lippy, MD (Senior 1999 (1988)) Warren, OH Ward B. Litton, MD (Senior 1995 (1969)) Bonita Springs, FL H. Edward Maddox III, MD (Senior 1996 (1970)) Houston, TX Richard E. Marcus, MD (Senior 1987 (1975)) Winnetka, IL Gregory J. Matz, MD (Senior 2002 (1979)) Chicago, IL William L. Meyerhoff, MD (Senior 2002 (1981)) Dallas, TX Eugene N. Myers, MD (Senior 1994 (1974)) Pittsburgh, PA George T. Nager, MD (Senior 1994 (1968)) Baltimore, MD Michael M. Paparella, MD (Senior 2000 (1968)) Minneapolis, MN Dennis Pappas, MD (Senior 2005 (1985)) Birmingham, AL James J. Pappas, MD (Senior 2002 (1983)) Little Rock, AR Claude L. Pennington, MD (Senior 1993 (1973)) Macon, GA Shokri Radpour, MD (Senior 1998 (1989)) Noblesville, IN J. H. Thomas Rambo, MD (Senior 1983 (1958)) New York, NY Frank N. Ritter, MD (Senior 1993 (1972)) Ann Arbor, MI Max L. Ronis, MD (Senior 1997 (1972)) Philadelphia, PA Robert J. Ruben, MD (Senior 1996 (1974)) Bronx, NY Wallace Rubin, MD (Senior 1992 (1967)) Metairie, LA Richard L. Ruggles, MD (Senior 1993 (1967)) Cleveland, OH Joseph Sataloff, MD (Senior 1994 (1960)) Philadelphia, PA

William H. Saunders, MD (Senior 1996 (1972)) Columbus, OH

Arnold G. Schuring, MD (Senior 2006 (1990)) Warren, OH

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George T. Singleton, MD (Senior 2007 (1972)) Gainesville, FL

J. Brydon Smith, MD (Senior 1980 (1958)) Willowdale ON M2L 2B4, CANADA

Mansfield F.W. Smith, MD (Senior 2000 (1973)) Davis, CA

James B. Snow, Jr., MD (Senior 1993 (1973)) West Grove, PA

Gershon Jerry Spector, MD (Senior 2007(1979)) St. Louis, MO

Malcolm H. Stroud, MD (Senior 1990 (1967)) Dallas, TX

G. Dekle Taylor, MD (Senior 1985 (1965)) Jacksonville, FL

Paul H. Ward, MD (Senior 1994 (1972)) Los Angeles, CA

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Joe C. Adams, PhD (Associate 2001) Boston, MA

James F. Battey, Jr., MD, PhD (Associate 2001) Bethesda, MD

Ricardo F. Bento, MD, PhD (Associate 2004) Sao Paulo, BRASIL

Karen I. Berliner, PhD (Associate 1995) Marina del Rey, CA

Barbara A. Bohne, PhD (Senior Associate 2006 (1979)) St. Louis, MO

Robert A. Butler, PhD (Senior Associate 2006 (1978)) Chicago, IL

Mohamed A. Hamid, MD, PhD (Associate 1992) Cleveland, OH

Maureen T. Hannley, PhD (Associate 1992) Durham, NC Raul Hinojosa, MD (Senior Associate 2006 (1989)) Chicago, IL

Vincente Honrubia, MD (Senior Associate 2006 (1972)) Los Angeles, CA

Makoto Igarashi, MD (Senior Associate 2006 (1973)) Tokyo 102, JAPAN

Salvatore J. Iurato, MD (Senior Associate 2006 (1994) Bari, ITALY

Pawel J. Jastreboff, PhD (Associate 1997) Ellicott, MD

Walter H. Johnson, PhD (Senior Associate 2006 (1960)) Toronto ONT M4G 3E2, CANADA

Lars-Goran Johnsson, MD (Senior Associate 2006 (1979)) FINLAND

Steven K. Juhn, MD (Senior Associate 2006 (1980)) Minneapolis, MN

Paul R. Kileny, PhD (Associate 1994) Ann Arbor, MI

Robert S. Kimura, PhD (Senior Associate 2006 (1978)) Weston, MA

David J. Lim, MD (Senior Associate 2006 (1973)) Los Angeles, CA

Brenda Lonsbury-Martin, PhD (Associate 1997) Loma Linda, CA

Michael Merzenich, PhD (Senior Associate 2006 (1986)) San Francisco, CA

Josef M. Miller, PhD (Senior Associate 2006 (1979)) Ann Arbor, MI

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Isamu Sando, MD (Senior Associate 2006 (1975))

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Thomas R. Van De Water, PhD (Associate 1987) Miami, FL

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Sabina Regina Wullstein, MD (Senior Associate 2006 (1999)) D- 97074, Wurzburg GERMANY

Joseph J. Zwislocki, ScD (Senior Associate 2006 (1984)) Syracuse, NY

CORRESPONDING MEMBERS

Marcus D. Atlas, MBBS, FRACS (Corresponding 2005) Nedlands, WESTERN AUSTRALIA

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Vicente G. Diamante, MD (Corresponding 2000) ARGENTINA

Bernard Gil Fraysse, MD (Corresponding 1999) FRANCE

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Thomas E. Linder, MD (Corresponding 2001) SWITZERLAND

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EMERITUS MEMBERS

Warren Y. Adkins, MD (Emeritus 2001 (1987)) Mt. Pleasant, SC

Sean R. Althaus, MD (Emeritus 2004 (1987)) Georgetwon, TX

B. Hill Britton, MD (Emeritus 2000 (1978)) Las Cruces, NM

Lee A. Harker, MD (Emeritus 2006 (1987)) Omaha, NE

C. Gary Jackson, MD (Emeritus 2007 (1990)) Brentwood, TN

Robert J. Keim, MD (Emeritus 1997 (1987)) Oklahoma City, OK

Nelson Y.S. Kiang, PhD (Emeritus 2006 (1969)) Boston, MA

Horst R. Konrad, MD (Emeritus 2005 (1991)) Springfield, IL

K. J. Lee, MD (Emeritus 2006 (1997)) New Haven, CT

Roger C. Lindeman, MD (Emeritus 2001 (1987)) Mercer Island, WA

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Ralph A. Nelson, MD (Emeritus 2004 (1995)) Manchester, WA

James L. Parkin, MD (Emeritus 1997 (1986)) Salt Lake City, UT

Leonard R. Proctor, MD (Emeritus 1997 (1989)) Lutherville, MD

HONORARY MEMBERS

Pedro Albernaz, (Honorary 1993) Miami, FL

Aziz Belal, MD (Honorary 1993) Alexandria, EGYPT

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Graeme M. Clark, PhD (Honorary 2002) AUSTRALIA

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Jerome C. Goldstein, MD (Honorary 1992) Lake Worth, FL

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Michel Portmann, (Honorary 1983) Bordeaux 33000, FRANCE

Naoaki Yanagihara, MD (Honorary 2008) Matsyama, JAPAN

Members Deceased Since Last Spring Meeting

Brian McCabe, MD (Active 1965; Senior 1997) Iowa City, IA Date of Death: 10/7/2007

Mendell Robinson, MD (Active 1969; Senior 1991) Rehoboth, MA Date of Death: 9/29/2007