PROGRAM and ABSTRACTS

of the

One Hundred Forty-Second
Annual Meeting

AMERICAN OTOLOGICAL SOCIETY, INC.

May 29-30, 2009

Grand Sonoran G

JW Marriott Desert Ridge Resort & Spa
Phoenix, Arizona

OFFICERS
JULY 1, 2008—JUNE 30, 2009

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Boston, MA 02114

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Herman A. Jenkins, MD
John W. House, MD

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)*™. 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.

LEARNING OBJECTIVES

1. A deeper knowledge of potential medical managements of otologic disease such as drug delivery to the inner ear and management of acoustic neuromas.
2. A deeper understanding of the diagnosis and treatment of vestibular disorders.
3. Become knowledgeable about implantable hearing devices such as cochlear implants, BAHA and implantable hearing aids.

DESIRED RESULTS

Attendees should be able to:

1. Discuss current and prospective medical management protocols for otologic disease.
2. Describe the potential limitations in drug delivery to the inner ear.
3. Identify emerging technologies in the medical management of acoustic neuroma.
4. Name two or three contemporary and emerging technologies in the diagnosis and treatment of vestibular disorders.
5. Recognize the applicability and limitations in use of implantable devices for hearing rehabilitation.

All Authors/Presenters signature on the following statements were required on all papers submitted to the American Otological Society. All authors/presenters were 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 persons in a position to control CME content of the American Otological Society's sponsored activities are required 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. Three weeks prior to the AOS meeting, the Council will review the manuscripts to identify a conflict of interest and make a decision if that individual should be the presenter or ask the primary author to select another person who does not have a conflict of interest to present the paper. If a conflict of interest is identified then one of the following mechanisms will be used to resolve it: Individuals may choose to discontinue their relationship, the individual can elect to alter the educational design or format of the presentation, and select someone else to present that portion of the content. 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. 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, (Name of Abstract) , 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)

***FACULTY DISCLOSURES

American Otological Society Council

Joseph B. Nadol, Jr., MD—Boston Medical Prod.(Royalty Agreement, Otologic Surgery)
Lippincott (Book Royalties, Books)
Gyrus (Royalty, Otologic Surgery)

Bruce J. Gantz, MD—Cochlear Corp (Consultant, Cochlear Implants)
Advanced Bionics (Consultant,Cochlear Implants)
Medtronics (Consultant, Instrumentation)
Anspach (Consultant, Otologic Drills)

Clough Shelton, MD—Cochlear Corp
(Research Grant, Cochlear Implant)
Synthesis (Research Grant, CSF Leak)

Herman A. Jenkins, MD—Otologics
(Grant Recipient—Basic Animal Research)

Paul R. Lambert, MD (Nothing to Disclose)
C. Phillip Daspit, MD (Nothing to Disclose)
Antonio De La Cruz, MD (Nothing to Disclose)

John W. House, MD (Nothing to Disclose)

Administrators:

Shirley Gossard (Nothing to Disclose)
Kristen Bordignon (Nothing to Disclose)

2009 Program Advisory Committee

Roberto A. Cueva, MD (Nothing to Disclose)
C. Phillip Daspit, MD (Nothing to Disclose)
Howard W. Francis, MD (Nothing to Disclose)
Bruce J. Gantz, MD (Nothing to Disclose)
Jeffrey P. Harris MD, PhD—Immco Diagnostics (Equity/Consultant)
Otonomy Inc. (Stock/Board of Directors)
S. George Lesinski, MD—Omniguide (Fee+options/Consultant)
Lawrence R. Lustig, MD—Advanced Bionics Corp
(Honorarium Medical Advisory Board)
Michael J. McKenna, MD (Nothing to Disclose)
Saumil N. Merchant, MD (Nothing to Disclose)
Lloyd B. Minor, MD (Nothing to Disclose)
J. Gail Neely, MD (Nothing to Disclose)
Steven A. Telian, MD—Cochlear Americas
(Honorarium/Medical Advisory Board)
David F. Wilson, MD (Nothing to Disclose)

Friday, May 29, 2009, Scientific Session

Moderators

Joseph B. Nadol, Jr., MD (Nothing to Disclose)
Paul R. Lambert, MD (Nothing to Disclose)

Guest of Honor Presentation

Robert J. Ruben, MD (Nothing to Disclose)

Friday, May 29, 2009, Scientific Session

*****Oral Pressentations: Authors/Presenters/Panel Participants Disclosures (listed in order of presentation)**

8:05 am Abstract No. 1
Kelley M. Dodson, MD (Nothing to Disclose)
Alexandros Georgolios, MD (Nothing to Disclose)
Noelle Barr, BS (Nothing to Disclose)
Aristides Sismanis, MD (Nothing to Disclose)
Kathleen Arnos, PhD (Nothing to Disclose)
Virginia Norris, MS (Nothing to Disclose)
Walter Nance, MD, PhD (Nothing to Disclose)
Arti Pandya, MD (Nothing to Disclose)

8:14 am Abstract No. 2
Yu-Lan Mary Ying, MD (Nothing to Disclose)
Carey D. Balaban, PhD (Nothing to Disclose)

8:23 am Abstract No. 3
Jordan Hochman, MD (Nothing to Disclose)
Joseph Chen, MD (Nothing to Disclose)
Julian Nedzelski, MD (Nothing to Disclose
Vincent Lin, MD (Nothing to Disclose)
David Shipp, PhD (Nothing to Disclose)
Tracy Stockley, PhD (Nothing to Disclose)

8:32 am Abstract No. 4
Yukiko Iino, MD, PhD (Nothing to Disclose)
Kozue Kodama, MD (Nothing to Disclose)
Hajime Usubuchi, MD (Nothing to Disclose)
Katzumi Takizawa, MD (Nothing to Disclose)
Takeharu Kanazawa, MD, PhD (Nothing to Disclose)
Yasushi Ota, MD, PhD (Nothing to Disclose)

8:41 am Abstract No. 5
Samuel H. Selesnick, MD (Nothing to Disclose)
Luke A. Donatelli, BA (Nothing to Disclose)
Deya N. Jourdy, MD (Nothing to Disclose)

8:50 am Abstract No. 6
Oliver F. Adunka, MD (Nothing to Disclose)
Harold C. Pillsbury, MD (Nothing to Disclose)
Marcia C. Adunka, AuD (Nothing to Disclose)
Craig A. Buchman, MD—Med El Consultant; Advanced Bionics Consultant; Cochlear Corp Consultant
8:59 am Abstract No. 7
Thomas J. Balkany, MD—Cochlear Americas Consultant; Advanced Bionics Corp Consultant
Matthew Whitley, MD (Nothing to Disclose)
Yisgav Shapira, MD (Nothing to Disclose)
Fred F. Telischi, MEE, MD (Nothing to Disclose)
Simon I. Angeli, MD—Medtronic (Research Support/Otology)
Osmopharm (Research Support Otology)
Adrien A. Eshraghi, MD—Med EL (Research Support)

9:21 Basic Science Lecture No. 1
Scott Plotkin, MD, PhD—No relationship with commerical products or services in presentation

***Disclosures—Oral Presentations (Cont)

10:15 am Abstract No. 8
Joni K. Doherty, MD, PhD (Nothing to Disclose)
Zana Ahmad (Nothing to Disclose)
Carrie Brown, MD (Nothing to Disclose)
Andrew K. Patel, MD (Nothing to Disclose)
Allen F. Ryan, PhD (Nothing to Disclose)

10:24 am Abstract No. 9
Moises A. Arriaga, MD (Nothing to Disclose)
Lydia F. Arriaga, APRF, FNP-C, CNOR, RNFA (Nothing to Disclose)
Daniel Nuss, MD (Nothing to Disclose)
Kelley Scrantz, MD (Nothing to Disclose)
Elizabeth Montgomery, MCD, CCA-A (Nothing to Disclose)
Patti St. John, MCD, CCA-A (Nothing to Disclose)

10:33 am Abstract No. 10
Cameron L. Budenz, MD (Nothing to Disclose)
Susan B. Waltzman, PhD (Nothing to Disclose)
J. Thomas Roland, Jr., MD Advanced Bionics Consultant; Cochlear Americas Consultant

10:42 am Abstract No. 11
David Bakhos, MD (Nothing to Disclose)
Alain Robier, MD (Nothing to Disclose)
Stephane Velut, MD (Nothing to Disclose)
Emmanuel Lescanne, MD, PhD (Nothing to Disclose)

10:56 am Panel No. 1
Michael J. McKenna, MD (Nothing to Disclose)
Jeffrey P. Harris, MD, PhD Immco Diagnostics (Equity/Consultant); Otonomy Inc. (Stock/Board of Directors)
Derald E. Brackmann, MD (Nothing to Disclose)
D. Bradley Welling, MD, PhD (Nothing to Disclose)
Robert K. Jackler, MD (Nothing to Disclose)
Bruce J. Gantz, MD (Nothing to Disclose)

Saturday, May 30, 2009, Scientific Session

1:00 pm Abstract No. 12
Gerald R. Popelka, PhD—Sonitus Medical, Inc. (Consultant/ Experimental & Clinical Expertise)

M. Jennifer Derebery, MD—Sonitus (Consultant, Board of Directors)
Nikolas Blevins, MD —Sonitus Medical (Member, Scientific Advisory Board/Stock Ownership)
Michael Murray, MD— Sonitus Consultant
Brian C. J. Moore, PhD— Sonitus Consultant
Robert W. Sweetow, PhD—Sonitus Consultant
Ben Wu, DDS, PhD—Sonitus Medical, Inc. Consultant
Linda Centore RN, PhD (Nothing to Disclose)
Mina Katsis—Sonitus Medical, Inc. (Independent Contractor-Ownership Interest/Tinnitus/SSD)

1:09 pm Abstract No. 13
Laura Hetzler, MD (Nothing to Disclose)
Ryan G. Porter, MD (Nothing to Disclose)
John P. Leonetti, MD (Nothing to Disclose)
Sam Marzo, MD (Nothing to Disclose)

***Disclosures—Oral Presentations (Cont)

1:18 pm Abstract No. 14
Charles A. Mangham Jr., MD (Nothing to Disclose)

1:27 pm Abstract No. 15
Gül Ö. Acar, MD (Nothing to Disclose)
Bassem M. Hanna, MS (Nothing to Disclose)
Dennis S. Poe, MD (Nothing to Disclose)

1:36 pm Abstract No. 16
Michael H. Fritsch, MD (Nothing to Disclose)
Chris Chacko, MD (Nothing to Disclose)
Emily Patterson, PhD (Nothing to Disclose)

1:45 pm Abstract No. 17
Robert F. Labadie, MD, PhD (Nothing to Disclose)
Ramya Balachandran, PhD (Nothing to Disclose)
Jason Mitchell, MS (Nothing to Disclose)
Jack Noble, BS (Nothing to Disclose)
Omid Majdani, MD, PhD (Nothing to Disclose)
Benoit M. Dawant, PhD (Nothing to Disclose)
J. Michael Fitzpatrick, PhD (Nothing to Disclose)

1:54 pm Abstract No. 18
J. Eric Lupo MD, MS (Nothing to Disclose)
Kanthaiah Koka, PhD (Nothing to Disclose)
N. Julian Holland, MD—Otologics LLC (Educational Grant for Lab Expenses—Manufactured Device used in part for experiment)
Herman A. Jenkins, MD—Otologics (Grant Recipient, Mechanisms of Inner Ear Stimulation)
Daniel J. Tollin, PhD (Nothing to Disclose)

2:03 p.m. Abstract No. 19
Eric M. Jaryszak, MD, PhD—Medtronic ENT (Grant Recipient/ Ossicular Prosthesis Research)
Edith Sampson, MS—Medtronic ENT (Grant Support) Sharklet Technologies (Grant Support)
Patrick J. Antonelli, MD—Medtronic ENT (Grant support/Paid Consultant) Alcon Laboratories (Grant Support/Speakers Bureau) Sharklet Technologies (Grant Support)

2:17 pm Basic Science Lecture No. 2
Alec N. Salt, PhD—Otonomy (Compensated member of Scientific Advisory Board) Med-El (Research Grant Recipient (expired) Advanced Bionics (Research Grant Recipient)

3:15 pm Abstract No. 20
Antti A. Aarnisalo, MD, PhD (Nothing to Disclose)
Jeffrey T. Cheng, PhD (Nothing to Disclose)
Michael E. Ravicz, MSc (Nothing to Disclose)
Nesim Hulli, MSc (Nothing to Disclose)
Ellery J. Harrington, MSc (Nothing to Disclose)
Maria S. Hernandez-Montes, PhD (Nothing to Disclose)
Cosme Furlong, PhD (Nothing to Disclose)
John J. Rosowski, PhD (Nothing to Disclose)
Saumil N. Merchant, MD (Nothing to Disclose)

***Disclosures—Oral Presentations (Cont)

3:24 pm Abstract No. 21
Quinton Gopen (Nothing to Disclose)
Dwight Jones, MD (Nothing to Disclose)
Dennis S. Poe, MD (Nothing to Disclose)
Guangwei Zhou, MD, ScD (Nothing to Disclose)

3:33 pm Abstract No. 22
Benjamin T. Crane, MD (Nothing to Disclose)
Lloyd B. Minor, MD (Nothing to Disclose)
John P. Carey, MD (Nothing to Disclose)

3:45 pm Abstract No. 23
Carlos A. Oliveira, MD, PhD (Nothing to Disclose)
Elienai A. Menezes, MD (Nothing to Disclose)
André LL Sampaio, MD (Nothing to Disclose)
Alessandra R. Venosa, MD (Nothing to Disclose)
Pedro Tauil, MD (Nothing to Disclose)

3:51 pm Abstract No. 24
Jay T. Rubinstein, MD, PhD—Cochlear Corp
(Consultant, grant recipient)
Steven Bierer, PhD (Nothing to Disclose)
Albert Fuchs, PhD (Nothing to Disclose)
Chris Kaneko, PhD (Nothing to Disclose)
L. Ling, PhD (Nothing to Disclose)
Kaibao Nie, PhD (Nothing to Disclose)
F. Santos, MD (Nothing to Disclose)
James O. Phillips, PhD (Nothing to Disclose)

4:05 pm Panel No. 2
Lloyd B. Minor, MD (Nothing to Disclose)
John P. Carey, MD (Nothing to Disclose)
Charles C. Della Santina, MD, PhD (Nothing to Disclose)
Scott D. Eggers, MD (Nothing to Disclose)
Timothy E. Hullar, MD (Nothing to Disclose)
Steven D. Rauch, MD (Nothing to Disclose)
Judith A. White, MD, PhD—Micromedical Technologies Course
(Honorarium)

***** 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.

The following competency areas will be addressed through this CME activity/scientific session

1. **Patient Care** that is compassionate, appropriate, and effective for the treatment of health problems and the promotion of health
2. **Medical Knowledge** about established and evolving biomedical, clinical, and cognate (e.g. epidemiological and social-behavioral) sciences and the application of this knowledge to patient care
3. **Practice-Based Learning and Improvement** that involves investigation and evaluation of their own patient care, appraisal and assimilation of scientific evidence, and improvements in patient care
4. **Interpersonal and Communication Skills** that result in effective information exchange and teaming with patients, their families, and other health professionals
5. **Professionalism** as manifested through a commitment to carrying out professional responsibilities, adherence to ethical principles, and sensitivity to a diverse patient population
6. **Systems-Based Practice** as manifested by actions that demonstrate an awareness of and responsiveness to the larger context and system of health care and the ability to effectively call on system resources to provide care that is of optimal value.

Please refer to back of program book outlining the summary of professional practice gaps related to the CME activity. Each abstract is numbered and referred to as Program ID # and referenced to the above competency areas.

Friday, May 29, 2009

7:00 **Business Meeting** *(Restricted to Members)*
Room: Grand Sonoran G

Minutes of the Annual Meeting 2008

Introduction of New Members

Election of Nominating Committee

Report of the Secretary-Treasurer

Report of the Editor-Librarian

7:30 **Scientific Program**
(Open to Registered Members & Non-Members)
Room: Grand Sonoran G

7:30 **Remarks by the President**
Joseph B. Nadol, Jr., MD

7:35 **Presidential Citation**
Michael J. McKenna, MD
Saumil N. Merchant, MD
Jay T. Rubinstein, MD, PhD
Isamu Sando, MD
D. Bradley Welling, MD, PhD

7:40 **Introduction of Guest of Honor**
Robert J. Ruben, MD
Guest of Honor Presentation
The Promise of Otology

8:00 **Discussion**

Hearing Loss: Genetics, Molecular Biology, Surgical and Medical Therapies

Moderators: *Joseph B. Nadol, Jr., MD*
Paul R. Lambert, MD

8:05 **1. Genetic Studies of Unilateral Hearing Loss**
Kelley M. Dodson, MD
Alexandros Georgolios, MD
Noelle Barr, BS
Aristides Sismanis, MD
Kathleen Arnos, PhD
Virginia Norris, MS
Walter Nance, MD, PhD
Arti Pandya, MD

8:14 **2. Regulation of Spiral Ganglion Cell Manganese Superoxide Dismutase (Mn SOD2) Expression after Kanamycin Challenge**
Yu-Lan Mary Ying, MD
Carey D. Balaban, PhD

NOTES

8:23 **3. The Incidence of GJB2 Mutations in Adult Cochlear Implant Candidates with a History of Early Onset Hearing Loss**
Jordan Hochman, MD
Joseph Chen, MD
Julian Nedzelski, MD
Vincent Lin, MD
David Shipp, PhD
Tracy Stockley, PhD

8:32 **4. Eosinophilic Inflammation in the Middle Ear Induces Deterioration of Bone Conduction Hearing Level in Patients with Eosinophilic Otitis Media**
Yukiko Iino, MD, PhD
Kozue Kodama, MD
Hajime Usubuchi, MD
Katzumi Takizawa, MD
Takeharu Kanazawa, MD, PhD
Yasushi Ota, MD, PhD

8:41 **5. Assessment of Variation in the Incidence of Idiopathic Sudden Sensorineural Hearing Loss Throughout the Year**
Samuel H. Selesnick, MD
Luke A. Donatelli, BA
Deya N. Jourdy, MD

8:50 **6. Is EAS Better Than Conventional CI for Speech Perception in Quiet?**
Oliver F. Adunka, MD
Harold C. Pillsbury, MD
Marcia C. Adunka, AuD
Craig A. Buchman, MD

8:59 **7. The Temporalis Pocket Technique for Cochlear Implantation: An Anatomic and Clinical Study**
Thomas J. Balkany, MD
Matthew Whitley, MD
Yisgav Shapira, MD
Fred F. Telischi, MEE, MD
Simon I. Angeli, MD
Adrien A. Eshraghi, MD

9:08 **Discussion**

9:13 **New Clinical Trial Initiatives and Funding Opportunities at NIDCD**
Gordon B. Hughes, MD

9:21 **Basic Science Lecture—1**
The New Frontier: Targeted Therapies for NF2-related Vestibular Schwannomas
Scott Plotkin, MD, PhD

9:41 **Discussion**

NOTES

9:45 **ACCME New Guidelines**
Paul R. Lambert, MD

9:50 **Break with Exhibitors**

10:15 **8. Merlin Knockdown in Human Schwann Cells: Clues to Vestibular Schwannoma Tumorigenesis**
Joni K. Doherty, MD, PhD
Zana Ahmad
Carrie Brown, MD
Andrew K. Patel, MD
Allen F. Ryan, PhD

10:24 **9. Telemedicine-assisted Neurotology in Post-Katrina, Southeast Louisiana**
Moises A. Arriaga, MD
Lydia F. Arriaga, APRF, FNP-C, CNOR, RNFA
Daniel Nuss, MD
Kelley Scrantz, MD
Elizabeth Montgomery, MCD, CCA-A
Patti St. John, MCD, CCA-A

10:33 **10. The Effect of Cochlear Implantation Technology in Sequentially Bilaterally Implanted Adults**
Cameron L. Budenz, MD
Susan B. Waltzman, PhD
J. Thomas Roland, Jr., MD

10:42 **11. Three-Dimensional Modeling of the Bone for Surgical Training**
David Bakhos, MD
Alain Robier, MD
Stephane Velut, MD
Emmanuel Lescanne, MD, PhD

10:51 **Discussion**

10:56 **Panel 1: Management of Acoustic Neuromas: Plotting the Collision Course of Benign Disease and Patient Well Being**
Moderator: *Michael J. McKenna, MD*
Panelists: *Jeffrey P. Harris, MD, PhD*
Derald E. Brackmann, MD
D. Bradley Welling, MD, PhD
Robert K. Jackler, MD
Bruce J. Gantz, MD

11:54 **Discussion**

12:00 **Adjourn**

12:10 **AOS Members Group Photograph (Location to be announced)**

6:30 **President's Reception & Dinner Dance**
Wildflower Foyer/Wildflower AB
(Members and Invited Guests Only)

12:30 **Business Meeting**
(Restricted to Members)
Room: Grand Sonoran G

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
F. Board of Governors

Report of the Audit Committee

Report of the Membership Development Committee

Report of the Nominating Committee

Unfinished Business

New Business

1:00 **Scientific Program**
(Open to Registered Members and Non-Members)
Room: Grand Sonoran G

Surgical Management of Hearing Loss and Complications

Moderators: *Joseph B. Nadol, Jr., MD*
Paul R. Lambert, MD

1:00 **12. Preliminary Evaluation of a Novel Bone Conduction Device for Single Sided Deafness**
Gerald R. Popelka, PhD
M. Jennifer Derebery, MD
Nikolas Blevins, MD
Michael Murray, MD
Brian C. J. Moore, PhD
Robert W. Sweetow, PhD
Ben Wu, DDS, PhD
Linda Centore, RN, PhD
Mina Katsis

1:09 **13. Improved Flap Design in Bone Anchored Hearing Aid Surgery**
Laura Hetzler, MD
Ryan G. Porter, MD
John P. Leonetti, MD
Sam Marzo, MD

1:18 **14. Impact of Incus Necrosis on Revision Stapes Surgery Evaluated by Kaplan-Meier Product-Survival Procedure**
Charles A. Mangham Jr., MD

1:27 **15. Comparison of Stapedotomy Minus Prosthesis (STAMP), Circumferential Stapes Mobilization, and Small Fenestra Stapedotomy for Stapes Fixation**
Gül Ö. Acar, MD
Bassem M. Hanna, MS
Dennis S. Poe, MD

1:36 **16. Operating Room Sound Level Hazards for Patients and Physicians**
Michael H. Fritsch, MD
Chris Chacko, MD
Emily Patterson, PhD

1:45 **17. Clinical Validation Study of Percutaneous Cochlear Access Using Patient-Customized, Microstereotactic Frames**
Robert F. Labadie, MD, PhD
Ramya Balachandran, PhD
Jason Mitchell, MS
Jack Noble, BS
Omid Majdani, MD, PhD
Benoit M. Dawant, PhD
J. Michael Fitzpatrick, PhD

1:54 **18. Prospective Electrophysiological Findings of Round Window Stimulation in a Model of Experimentally-induced Stapes Fixation**
J. Eric Lupo MD, MS
Kanthaiah Koka, PhD
N. Julian Holland, MD
Herman A. Jenkins, MD
Daniel J. Tollin, PhD

2:03 **19. Effect of Ossicular Prosthesis Biofilms on Middle Ear Scarring and Hearing Outcomes**
Eric M. Jaryszak, MD, PhD
Edith Sampson, MS
Patrick J. Antonelli, MD

2:12 **Discussion**

Disorders of Hearing and Balance: Medical and Surgical Interventions

2:17 **Basic Science Lecture 2**
Opportunities and Techniques for Local Drug Delivery to the Inner Ear
Alec N. Salt, PhD

2:40 **Discussion**

2:45 **Break with Exhibitors**

3:15 **20. Middle Ear Mechanics of Cartilage Tympanoplasty Evaluated by Time-Averaged Laser Holography**
Antti A. Aarnisalo, MD, PhD
Jeffrey T. Cheng, PhD
Michael E. Ravicz, MSc
Nesim Hulli, MSc
Ellery J. Harrington, MSc
Maria S. Hernandez-Montes, PhD
Cosme Furlong, PhD
John J. Rosowski, PhD
Saumil N. Merchant, MD

3:24 **21. Posterior Semicircular Canal Dehiscence: First Reported Case Series**
Quinton Gopen
Dwight Jones, MD
Dennis S. Poe, MD
Guangwei Zhou, MD, ScD

3:33 **22. Improvement in Autophony Symptoms after Superior Canal Dehiscence Plugging**
Benjamin T. Crane, MD
Lloyd B. Minor, MD
John P. Carey, MD

3:42 **23. Vestibular-Ocular Reflex (VOR) as Predictor of Cerebral Death in Comatous Patients**
Carlos A. Oliveira, MD, PhD
Elienai A. Menezes, MD
André LL Sampaio, MD
Alessandra R. Venosa, MD
Pedro Tauil, MD

3:51 **24. Prosthestic Implantation of the Semicircular Canals with Preservation of Rotational Sensitivity: A "Hybrid" Vestibular Implant**
Jay T. Rubinstein, MD, PhD
Steven Bierer, PhD
Albert Fuchs, PhD
Chris Kaneko, PhD
L. Ling, PhD
K. Nie, PhD
F. Santos, MD
James O. Phillips, PhD

4:00 **Discussion**

NOTES

4:05 **Panel 2: Diagnosis and Treatment of Vestibular Disorders: Recent Advances and Future Directions**
Moderator: *Lloyd B. Minor, MD*
Panelists: *John P. Carey, MD,*
Charles C. Della Santina, MD, PhD
Scott D. Eggers, MD
Timothy E. Hullar, MD
Steven D. Rauch, MD
Judith A. White, MD, PhD

4:55 **Discussion**

5:00 **Introduction of Incoming AOS President**
Bruce J. Gantz, MD

5:05 **Adjourn**

2009 Program Advisory Committee
Roberto A. Cueva, MD
C. Phillip Daspit, MD
Howard W. Francis, MD
Bruce J. Gantz, MD
Jeffrey P. Harris MD
Lawrence R. Lustig, MD
Michael J. McKenna, MD
Saumil N. Merchant, MD
Lloyd B. Minor, MD
J. Gail Neely, MD
Steven A. Telian, MD
David F. Wilson, MD

COSM 2010
143rd AOS Annual Spring Meeting
April 30-May 1, 2010
Bally's
Las Vegas, NV

Abstract Deadline: October 15, 2009
Abstract submission form
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Genetic Studies of Unilateral Hearing Loss

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Walter Nance, MD, PhD; Arti Pandya, MD

Objective: To characterize unilateral hearing loss (HL) in a National Hereditary Deafness Repository.

Study Design: Prospective clinical study

Setting: Tertiary Referral Center

Patients: 34 subjects identified in a National Hereditary Deafness Repository.

Interventions and Main Outcome Measures: Clinical data and family history of (HL) were obtained on enrollment. Candidate deafness genes were screened by SSCP and mutations were confirmed with sequencing.

Results: 34 subjects (19 males, 15 females) with unilateral HL were identified ranging in age from 2 months to 36 years. The mean age at diagnosis was 7 years, affecting the left ear in 62%. The population was 62% Caucasian, 23% African-American, and 15% Hispanic. CT scans were available in 49%, with the majority (69%) read as normal. 19% had enlarged vestibular aqueduct, with one case of unilateral Mondini and one case of unilateral common cavity. Twenty subjects (59%) had a family history of HL, with 26% specifically relating familial unilateral HL. Mutational screening revealed polymorphisms in the PDS, GJB3 (Connexin31), and COCH genes. There were three heterozygous changes in GJB2 (Connexin26), 3 homozygous mutations of GJB3, a novel heterozygous mutation in COCH, and a novel homozygous mutation in TECTA.

Conclusions: Family history and the potential for a genetic etiology should be pursued in unilateral deafness. Although sequence variants were identified in 35% of subjects with unilateral HL, further genetic and gene/environmental interaction studies are necessary to better define the etiologies comprising unilateral HL.

IRB Approval Number: HM10050

2.
Regulation of Spiral Ganglion Cell Manganese Superoxide Dismutase (Mn SOD2) Expression after Kanamycin Challenge

Yu-Lan Mary Ying, MD; Carey D. Balaban, PhD

Objectives: To study the regulation of spiral ganglion cell Mn SOD2 expression during reactive oxygen species (ROS) challenges.

Background: Mn SOD2 is a key metabolic anti-oxidant enzyme of the superoxide dismutase family for detoxifying free radical cascade inside the mitochondria of the cochlea. Variation in Mn SOD2 expression suggests that spiral ganglion cellular response to ROS may vary along the cochlear spiral, with a lower response capacity in the basal turn corresponding to high frequency hearing loss seen in clinical settings.

Study Design: Young adult (4 weeks) C57BL/6 mice used in a 2x2 factorial experimental design, with kanamycin (700 mg/kg, s.c.) or vehicle as the ROS challenge, and dihydroxybenzoate (300 mg/kg, s.c.) or vehicle as the antioxidant treatment for fifteen consecutive days. Mice from treatment group were used for either microdissection of cochlear modiolus for quantitative-PCR (n= 48) or immunohistochemical studies on decalcified temporal bone sections
(n = 36). To quantify immunostaining intensity for Mn SOD2, a pixel count analysis was performed for individual spiral ganglion cells at each cochlear turn.

Results: Compared to the control animal group, Mn SOD2 expression is responsive to kanamycin-induced ROS load by a modest upregulation of gene transcription, (1.6 fold, $p < 0.05$). The immunohistochemical studies revealed an upregulation of spiral ganglion cell Mn SOD2 expression, independent of cochlear location after kanamycin treatment, and that the changes were attenuated by antioxidant treatment. Pixel intensity analysis further corroborated both the kanamycin-induced upregulation and changes in the gradient of Mn SOD2 staining of spiral ganglion cells along the cochlear spiral.

Conclusions: In this study, we conclude that lower baseline MnSOD2 expression in the basal turn of control animals may indicate lower baseline constant ROS exposure, which makes basal turn vulnerable to sudden oxidative challenges. Hence, enhancement of this dynamic Mn SOD2 response capacity is a potential otoprotective strategy in the face of dynamic ROS challenges.

IRB Approval Number: N/A

3.
The Incidence of GJB2 Mutations in Adult Cochlear Implant Candidates with a History of Early Onset Hearing Loss

Jordan Hochman; Joseph Chen, MD; Julian Nedzelski, MD
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Objective: To assess the incidence of GJB2 mutations in a population of adult patients with a history of either early idiopathic (prelingual onset with no family history or obvious etiology) or hereditary progressive (progressive early onset, with familial association) bilateral severe sensorineural hearing loss.

Background: Significant efforts have been applied in defining the epidemiology of Connexin 26 associated hearing impairment in the pediatric population, yet the issue remains ambiguous for adult patients with severe hearing loss. Causation is important as there are implications to prognosis, risk of associated medical manifestations, and for genetic counseling.

Study Design: Case Series

Setting: Tertiary Referral Center

Patients: Adult patients meeting criteria for cochlear implantation with early onset hearing loss were approached for participation (November 2007 onward).

Intervention: Sequencing of the GJB2 gene exons 1 and 2 on DNA isolated from peripheral leukocytes.

Main outcome measure: Presence of GJB2 mutations.

Results: Forty seven patients were analyzed for GJB2 mutations. Five patients (10.6%) had two GJB2 mutations confirming GJB2-related origin of the hearing impairment. Five additional patients had either one known mutation (1 patient), or one (3 patients) or two (1 patients) variants of unclear significance. The vast majority (83%) of patients possessing a GJB2 mutation self report as early onset idiopathic impairment. Four additional patients were found to be monoallelic for Pendred Syndrome associated mutations.

Conclusion: The incidence of GJB2 related hearing impairment (10.6%) in an adult population with early onset idiopathic severe sensorineural hearing loss is significant in this discrete population.

IRB Approval Number: 408-2005

4.
Eosinophilic Inflammation in the Middle Ear Induces Deterioration of Bone Conduction Hearing Level in Patients with Eosinophilic Otitis Media

Yukiko Iino, MD, PhD; Kozue Kodama, MD
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Takeharu Kanazawa, MD, PhD; Yasushi Ota, MD, PhD

Background: Eosinophilic otitis media (EOM) is characterized by the extensive accumulation of eosinophils in the middle ear mucosa and middle ear effusion, and is usually associated with bronchial asthma. EOM patients show gradual deterioration of hearing and sometimes become deaf suddenly. In our previous study, we reported that high tone loss was more frequently found and more sever in EOM patients than in control patients with chronic otitis media. These results suggested that not only bacterial infection but also eosinophilic inflammation in the middle ear may damage the inner ear.
Objective: To determine whether eosinophilic inflammation is really related to the deterioration of the bone conduction hearing level (BCHL).
Patients: Fifty-five ears of 28 patients with EOM associated with bronchial asthma were included in this study.
Outcome measures: The middle ear effusion of each patient was collected and the concentrations of ECP and IgE were measured by fluorescence enzyme immunoassay. The BCHLs at 2000 and 4000 Hz of the patients were correlated with the concentrations of ECP and IgE.
Results: A significantly higher concentration of IgE in the middle ear effusion was detected in ears showing deterioration of BCHL at 2000 and 4000 Hz. Higher concentration of ECP in the middle ear effusion also tended to affect the deterioration of BCHL at 2000 Hz,
Conclusion: Eosinophilic-inflammation-related substances such as ECP and IgE are closely related to inner ear damage. To prevent the deterioration of BCHL in EOM patients, the control of eosinophilic inflammation and bacterial infection is required.

5.

Assessment of Variation in the Incidence of Idiopathic Sudden Sensorineural Hearing Loss Throughout the Year

Samuel H. Selesnick, MD; Luke A. Donatelli, BA
Deya N. Jourdy, MD

Objective: This study was conducted to determine whether the incidence of idiopathic sudden sensorineural hearing loss (ISSHL) varies throughout the year.

Study design: This study is a retrospective case review.

Setting: This study was conducted at a tertiary referral center within a teaching hospital.

Patients: All patients included in the study were given a diagnosis of ISSHL by a physician in the department, which was confirmed by audiometric data. The onset of hearing loss must have occurred within the three-year study period. Patients with intracranial neoplasms, or a history of Meniere's disease, prior ear procedures, chemotherapy, or radiation therapy to the head or neck were excluded from the review. One hundred and six patients met these criteria. The mean age was 53 years (range: 25 to 87), and there were 57 (53.77%) females and 49 (46.23%) males.

Interventions: No interventions were performed.

Main Outcome Measures: Monthly incidence values were grouped across a three-year period to yield total incidence values for each month of the year. A Chi-square test was used to identify uneven incidence distributions throughout the year, and a Rayleigh test was used to detect a peak in incidence at any point.

Results: No evidence was found for an uneven distribution (Chi-square, $p < 0.05$) or for a peak (Rayleigh, $p > 0.10$) in incidence of hearing loss throughout the year.

Conclusion: The results of this study suggest that ISSHL incidence does not vary throughout the year. The implications of these findings with respect to etiology will be discussed.

IRB Approval Number: 0805009815

6.
Is EAS Better Than Conventional CI for Speech Perception in Quiet?

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Marcia C. Adunka, AuD; Craig A. Buchman, MD

Objective: To assess whether combined electric and acoustic stimulation (i.e. EAS) provides a significant hearing-in-quiet advantage over (1) ipsilateral electrical stimulation alone, (2) ipsilateral acoustic stimulation alone, or (3) full-length cochlear implantation without preserved hearing.

Setting: Tertiary care academic referral center

Patients: Two similar groups of cochlear implant candidates with substantial residual hearing.

Intervention: EAS cochlear implantation and hearing preservation (n=10, study group) or conventional CI (n=18, control group) without hearing preservation.

Outcome measures: Status of residual hearing and speech perception data in quiet at 3 and 6 months after fitting.

Results: Preoperatively, the mean CNC word score was 18.5%±6.58 for the EAS group and 18.3%±10.39 for the conventional CI group ($p=0.884$). In the conventional CI group, hearing was not preserved following surgery in any subject while 9 out of the 10 subjects in the EAS group had hearing preservation. Mean CNC word scores at 3 months post-activation using electrical stimulation alone was 50.0% ±11.95 in the EAS group and 45.3%±17.44 in the conventional CI group ($p=0.47$). Between condition comparisons among the EAS subjects revealed that combined stimulation was significantly better than either the electrical or acoustic stimulation condition alone ($p<0.05$). When compared to the conventional CI group, combined stimulation in EAS subjects was again superior ($p<0.05$).

Conclusions: Limited length CI with ipsilateral hearing preservation provides comparable speech perception performance results to conventional CI when electric stimulation alone is used. The addition of ipsilateral acoustic stimulation in ears with preserved residual hearing provides an additional benefit over electrical stimulation alone.

IRB Approval Number: 06-0479

7.

The Temporalis Pocket Technique for Cochlear Implantation: An Anatomic and Clinical Study

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Objective: Cochlear implant (CI) receiver-stimulators (R/S) are generally secured by drilling a bone seat and tie-down holes. However, multiple intracranial complications have been reported highlighting the rare but potentially serious risks of drilling to dura. We report the temporalis pocket technique for securing R/S without drilling.

Study design: Anatomic: Fifty-six sides of 28 whole human cadaver skulls were dissected. In sixteen sides, a cochlear implant dummy was placed in a tight temporal-parietal pocket. Specimens were then dissected in layers with serial photography to determine the limits of the pocket, actual device position and points of peri-cranial fixation. Sites of fixation were then analyzed morphometrically in an additional 40 sides of 20 skulls. Clinical: Retrospective, non-randomized, controlled series of 227 consecutive CI recipients over a 2-year period to determine rates of migration. Patients received a CI using either the temporalis pocket (TJB) or standard technique (bony seat and suture retention holes) (FFT, SIA, AAE) with at least 12-month follow-up.

Results: The temporalis pocket is limited anteriorly by temporalis fascia; inferiorly by lamdoid suture and anteriorly by the ridge of the squamous suture. 171 subjects were implanted using the temporalis pocket technique and 56 using the standard technique. Subjects ranged in age from 7 months to 90 years. There were no migrations or intracranial complications in either group.

Conclusions: The temporalis pocket secures the R/S with anatomically consistent strong points of fixation while precluding dural penetration. This technique prevented migration in all cases. Further testing of this technique is necessary before it is widely adopted.

8.

Merlin Knockdown in Human Schwann Cells: Clues to Vestibular Schwannoma Tumorigenesis

Joni K. Doherty, MD, PhD; Zana Ahmad; Carrie Brown, MD
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Hypothesis: To investigate the molecular progression towards vestibular schwannoma (VS) development, we depleted merlin to model human Schwann cell tumorigenesis in vitro.

Background: NF-related and sporadic VS are associated with loss of functional merlin (schwannomin) in the Schwann cell. Following loss of merlin expression, the steps toward VS tumorigenesis are unknown. Merlin, a putative tumor suppressor protein, interacts with many cellular proteins, regulating their function. Among these are the ErbB family receptors, EGFR and ErbB2, which signal for proliferation and survival through the ERK1/2 and AKT pathways and are potential therapeutic targets for VS treatment.

Methods: Merlin depletion was performed using transfection of small interfering RNA (siRNA) into human Schwann cell primary cultures. Knockdown was confirmed by real-time quantitative PCR (Q-PCR) and Western analysis. To identify the early effects of merlin deficiency on gene expression profiles, microarray analysis was performed to compare normal human Schwann cells and those deficient in merlin. Q-PCR and Western blotting were used to verify results of microarray and examine ErbB expression profiles in knockdowns, as well. Sensitivity to gamma irradiation-induced apoptosis was also assessed.

Results: Merlin knockdowns demonstrated robust upregulation of EGFR and modest upregulation of ErbB2, as well as the stem cell marker, BMI-1. Merlin knockdown conferred relative resistance to gamma irradiation.

Conclusions: Loss of functional merlin promotes a dedifferentiated state, and deregulation of ErbB receptor signaling is a critical step towards VS tumorigenesis. Elucidating the molecular steps involved in schwannoma tumorigenesis after loss of merlin may identify additional molecular targets, such as BMI-1.

9.
Telemedicine-assisted Neurotology in Post-Katrina, Southeast Louisiana

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Elizabeth Montgomery, MCD, CCA-A
Patti St. John, MCD, CCA-A

Objective: This retrospective study evaluates technical requirements, privacy and legal constraints, reimbursement considerations and overall feasibility of a new telemedicine Neurotology patient care delivery model in post-Katrina, Southeast Louisiana.

Method: This study is a retrospective review of the first year of a telemedicine Neurotology practice with limited on-site Neurotology physician availability (three-days monthly) with full-time audiology, full-time specialty-trained nurse practitioner, full-time Neurosurgery and full-time otolaryngology on-site, back-up availability

Result: A combined "store-and-forward" and "real-time" telemedicine delivery model was implemented for a new Neurotology practice. Technical requirements include secure data transfer, real-time video-streaming, high quality video otoscopy and microscopy, infra-red video (IRV) eye movement visualization and recording, remote visualization of radiologic imaging studies, and formalized diagnostic algorithms for patient evaluation. Radiologic imaging studies are available through remote visualization. Patient evaluations occur with the patient in Baton Rouge, Louisiana while the examining neurotologist is linked through a secure, commercially-available communication connection in Pittsburgh, Pennsylvania. Specifically designed consent forms and bi-location licensing and liability insurance coverage was required. Third-party payers were consulted prior to implementation to assure adherence to local reimbursement requirements.

During the first 9 months of operation, 147 patient encounters were accomplished purely through telemedicine with an additional 858 on-site patient visits and 108 operative procedures including 26 neurotologic skull base procedures.

Conclusion: Telemedicine is a viable delivery model for Neurotology care delivery. Planning and implementation of such a model requires systemic considerations of medical, nursing, information systems, legal, reimbursement and management parameters. While the authors' initial motivation for this model is the resource-restricted, post-Katrina health care environment in South Louisiana, this delivery model has wider applicability in otolaryngology, other medical specialties, humanitarian outreach and medical education. Prospective assessment of clinical outcomes and patient satisfaction is ongoing for objective validation of this delivery model.

IRB Approval Number: RC4738 - Allegheny General Hospital

10.
The Effect of Cochlear Implantation Technology in Sequentially Bilaterally Implanted Adults

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Objective: Bilateral sequential cochlear implantation outcomes have been shown to be dependent on many different factors, including duration of deafness, age at implantation, and time between implantations. Newer technology in the second implanted ear may also contribute to outcome. The purpose of this study is to examine the effect of cochlear implant technology on speech recognition outcomes in a population of adult subjects who have undergone bilateral sequential implantation using different technologies in each ear.

Study Design: Retrospective chart review.

Setting: Tertiary referral center.

Patients: Eighteen adults who underwent bilateral sequential cochlear implantation with different technologies and processing strategies in each ear were subjects for this study.

Intervention: Bilateral sequential cochlear implantation.

Outcome Measures: Outcome measures included speech recognition measures at the phoneme, word, and sentence level in quiet and noise. A multivariate analysis was performed to account for factors including length of deafness, residual hearing, time between implantations, and electrode design.

Results: Absolute scores in the second implanted ear were variable and were affected by electrode design, age at implantation and length of deafness in the second implanted ear. All subjects were consistent users of both devices and, on average, the use of different technology in the second implanted ear did not affect subjects' ability to benefit from bilateral implantation despite the variability.

Conclusion: Bilateral sequential implantation with newer and/ or differing technology in the second implanted ear did not reduce the benefits of bilateral stimulation and should not be considered a deterrent to second-sided implantation.

IRB Approval Number: 11281

11.
Three-Dimensional Modeling of the Temporal Bone for Surgical Training

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Introduction: The anatomy of the temporal bone can only be mastered by repeated surgical and anatomic dissections and surgical teaching initiative had a major effect on outcomes. The aim of this study was to investigate the validity of an artificial temporal bone model devoted to surgical training and education.

Material and method: A helical computed tomography (CT) scan was used to acquire high resolution data of cadaveric temporal bone. DICOM data were converted in .stl files after data processing. Cadaveric temporal bones were prototyped using stereolithography. The validation of the prototype needed several steps. First of all we have studied on CT scan the positional relationship between the facial nerve and other structures of the cadaveric temporal bones and prototyped bones. Otoendoscopy of the middle ear and the internal acoustic canal, and visualisation of anatomic landmarks during temporal bone drilling of the cadaveric temporal bones and prototyped bones were also performed.
Results: Seven normal CT scan of cadaveric temporal bone were selected to make prototyped bone using stereolithography. Measurements of volume and distance showed no significant difference between prototypes and cadaver temporal bones. Classical mastoid surgical procedures were performed in the anatomy department: exposing sigmoid sinus, facial nerve, labyrinth, dura mater, jugular bulb, and internal carotid artery. Two simulations of implantable middle ear prosthesis were made successfully.

Conclusion: These prototypes made using stereolithography appear as a good anatomic model for surgical training. This model could be also interesting for surgical planning in congenital ear anomalies before middle ear prosthesis implantation.

12.
Preliminary Evaluation of A Novel Bone Conduction Device for Single Sided Deafness

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Auditory deficits from single sided deafness (SSD) can be reduced with a device that uses a microphone on the deaf side and signal delivery to the opposite, normal hearing ear. Signal delivery to the normal ear by air conduction has only limited effectiveness, while delivery by bone conduction is much more effective. In spite of this, the existing bone conduction approaches have at least three substantial limitations: transducer coupling problems, narrow bandwidth, and suboptimal microphone location.

Described here is an entirely new device that addresses these limitations. It employs a unique bone conduction location (a removable oral appliance that delivers the signal via a molar surface), a transducer that potentially has a wider bandwidth (piezoelectric vs electrodynamic), and a microphone location (deep in the ear canal of the deaf ear) that capitalizes on the spatial sound qualities of the external ear without interfering with auditory function in the normal ear.

The purpose of this paper is to assess this entirely new approach. Measurements in the laboratory and on normal human subjects are presented including assessments of oral comfort (tolerance), oral health (tooth abrasion, tooth movement, heat, etc), safety (biocompatibility, etc), dynamic range (range of force levels required for auditory perception), tactile comfort (maximum force levels that do not evoke oral vibratory sensations), bandwidth (frequency response), calibration (to account for individual skull impedance differences) and other factors. The results indicate that this new and innovative approach can mitigate current coupling and bandwidth issues while enhancing spatial hearing ability in SSD patients.

IRB Approval Number: IRB approval # 07014-02, IRC, Corte Madera, CA

13.
Improved Flap Design in Bone Anchored Hearing Aid Surgery

Laura Hetzler, MD; Ryan G. Porter, MD
John P. Leonetti, MD; Sam Marzo, MD

Objective: To determine if fewer revisions or severe skin reactions were encountered using standard dermatome created skin grafts versus thinned "C" or "L"- shaped postauricular flaps created at the time of Bone Anchored Hearing Aid (BAHA) surgery or using a previous incision site.

Study Design and Methods: Retrospective chart review of all BAHA implantations from 2003-2008.

Setting: Tertiary referral center, ambulatory surgery

Patients: One hundred twenty-eight primary BAHAs were performed on 127 patients with one patient undergoing bilateral implantation. Of these, 110 cases (86.6%) were ultimately included.

Interventions: BAHA

Main Outcome Measures: Revision and wound complication rates.

Results: Ninety subjects (81.8%) underwent dermatome created flaps and 20 subjects (18.1%) underwent "C" or "L" shaped flaps de novo or using prior postauricular incision sites. Twenty-four of 90 (26.6%) dermatome patients required revisions compared to 1/20 (5%) in the flap group ($p<0.05$). Regarding Grade 3 skin reaction, 23/67 were in the dermatome group and 1/20 in the flap group ($p<0.05$). Prior surgery was not associated with increased revision rates or skin reactions, ($p>0.05$).

Conclusion: Bulkier yet sufficiently thinned flaps are superior to traditional dermatome created skin grafts when performing BAHA implantation. Our data set includes all BAHA surgeries performed at our institution allowing the revision rate to appear elevated in our dermatome group consistent with a learning curve. Regardless, we feel strongly that our results using previous or "C" or "L" shaped incisions result in less severe skin reactions and require fewer return trips to the operating room.

IRB Approval Number: LU# 201041

14.
Impact of Incus Necrosis on Revision Stapes Surgery Evaluated by Kaplan-Meier Product-Survival Procedure

Charles A. Mangham, Jr., MD

Objective: To determine the effect of incus necrosis on the success of revision stapes surgery.
Study Design: Retrospective chart review.
Patients: Two hundred-five ears in 152 consecutive patients who had revision stapes surgery using crimpable pistons from 1989 to 2007.
Intervention: The status of the incus was determined at revision surgery in all 205 ears. There were 62 ears with no incus damage and 143 ears with a range of incus damage from mild notching to complete necrosis. Based on surgical judgment, 51 ears had a reconstruction from the malleus to an oval window fenestra and 92 ears had a reconstruction from the damaged incus.
Main Outcome Measures: American Academy of Otolaryngology-Head and Neck Surgery guidelines including 4-frequency pure-tone average, success rate (gap < 10 dB), and Kaplan-Meier product-survival procedure.
Results: When success was defined as an air-bone gap of 10 dB or less at one-year, patients with a reconstruction to a normal incus, necrotic incus, or malleus all had similar success rates ranging from 54 to 62%. Success over time was significantly poorer for patients with a reconstruction to a necrotic incus (17% at 20 years) compared with reconstruction to a normal incus (51%) or malleus (42%; $p < 0.05$).
Conclusions: Incus necrosis bodes poorly for reconstruction from the incus using crimpable pistons. Reconstruction from the malleus provided a more stable long-term reconstruction, but is less desirable anatomically. Characteristics of better prostheses to connect to the necrotic incus and to the malleus will be presented.

IRB Approval Number:
Approved by Chairman of Swedish Medical Center IRB

15.
Comparison of Stapedotomy Minus Prosthesis (STAMP), Circumferential Stapes Mobilization, and Small Fenestra Stapedotomy for Stapes Fixation

Gül Ö. Acar, MD; Bassem M. Hanna, MS; Dennis S. Poe, MD

Educational Objective: To be able to discuss the surgical results of 3 techniques: stapedotomy minus prosthesis (STAMP), circumferential stapes mobilization, and small fenestra stapedotomy for stapes fixation

Objectives: To compare the outcomes of three surgical techniques for primary stapes fixation: stapedotomy minus prosthesis (STAMP), circumferential stapes mobilization (CSM) and small fenestra stapedotomy (SFS).

Study Design: Retrospective review of 277 primary cases operated for stapes fixation from 1997 to 2007.

Methods:

SETTING: Tertiary academic center.

PATIENTS: Consecutive adult and pediatric cases operated for conductive hearing loss due to stapes fixation.

INTERVENTIONS: STAMP was performed for otosclerosis limited to the anterior footplate, CSM was done for congenital stapes fixation, SFS was done for more extensive otosclerosis or anatomic contraindications to STAMP/CSM.

MAIN OUTCOME MEASURES: Pure-tone audiometry was performed preoperatively, and postoperatively at 4, 12 months, and most recent long term results.

Results: 196 ears in 160 patients met criteria for the study. 149 ears (76.02%) underwent SFS, 25 (12.76%) STAMP, and 22 (11.22%) CSM. There was statistically significant improvement in average Air Conduction(AC) thresholds and air-bone gap(ABG) for all techniques ($p<0.05$). Mean ABG for STAMP closed from 27.88 to 2.88 dB (SD, 5.02 dB) and for CSM from 32.46 to 15.92 dB (SD, 12.30 dB). Average AC thresholds of STAMP cases was better than CSM or SFS cases, which were comparable ($p=0.0013$). AC and Bone Conduction(BC) thresholds at 4 and 8 kHz were significantly better than for CSM or SFS ($p<0.05$).

Conclusions: Excellent hearing results were achieved with all techniques and STAMP showed better outcomes, especially in high frequencies. CSM is a good option for children and patients for whom it is desirable to avoid a footplate fenestration or prosthesis.

IRB # M08-02-0076

16.
Operating Room Sound Level Hazards for Patients and Physicians

Michael H. Fritsch, MD; Chris Chacko, MD
Emily Patterson, PhD

Hypothesis: Exposure to certain new surgical instruments and operating room devices during procedures can cause hearing damage.

Background: Surgical instruments and other equipment generate significant sound levels during routine usage. Both patients and physicians are exposed to these levels during the operative case. Many cases last for hours. The noise loads during the cases are cumulative. OSHA and NIOSH standards are inconsistent in their appraisals of potential damage. Implications of the newer powered- instruments are not widely recognized.

Methods: Bruel and Kjaer sound meter spectral recordings for 20 major instruments from 5 surgical specialties were obtained at the ear levels for the patient and the surgeon between 32 Hz and 20 kHz.

Results: Routinely used instruments generated sound levels as high as 116 dB. Patient and operator exposures differed. There was unilateral dominant exposure. Many instruments had levels that became hazardous well within the length of an average surgical procedure. The OSHA and NIOSH systems gave contradicting results when applied to individual instruments and types of cases. Background noise, especially in its intermittent form, was also of significant nature.

Conclusions: Instrument noise levels for average length surgical cases exceeded OSHA and NIOSH recommendations for hearing safety. Specialties such as Otolaryngology, Orthopedics, and Neurosurgery use instruments, which regularly exceed limits. General operating room noise also contributes to overall personnel exposures. Innovative counter measures are discussed.

17.
Clinical Validation Study of Percutaneous Cochlear Access Using Patient-Customized Microstereotactic Frames

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Jason Mitchell, MS; Jack Noble, BS
Omid Majdani, MD, PhD; Benoit M. Dawant, PhD
J. Michael Fitzpatrick, PhD

Objective: Percutaneous cochlear implant (PCI) surgery consists of drilling a single trough from the lateral skull to the cochlea avoiding vital anatomy. To accomplish PCI we utilize a patient-customized, microstereotactic frame, which we call a "microtable" because it consists of a small tabletop sitting on legs. The orientation of the legs controls the alignment of the tabletop such that it is perpendicular to a specified trajectory.

Study design: Prospective

Setting: Tertiary referral center.

Patients: Eight patients (eleven ears) undergoing traditional CI surgery.

Intervention(s): With IRB approval, each patient had three fiducial markers implanted in bone surrounding the ear. Temporal bone CT scans were obtained and the markers were localized as was vital anatomy. A linear trajectory from the lateral skull through the facial recess to the cochlea was planned. A microtable was fabricated to follow the specified trajectory.

Main outcome measure(s): After mastoidectomy and posterior tympanotomy, accuracy of trajectories was validated by mounting microtables on bone-implanted markers and then passing sham drill bits (1 mm) across the facial recess to the cochlea. Distance from the drill to vital anatomy was measured.

Results: Microtables were constructed on a CNC milling machine in under 6 minutes each. Successful access across the facial recess to the cochlea was achieved in all 11 cases. The mean ± standard deviation from the drill to the facial nerve was 1.10±0.33mm and from the chorda tympani was 1.27±0.38mm.

Conclusions: These results demonstrate the feasibility of PCI access using customized, microstereotactic frames.

IRB Approval Number: 060028

18.
Prospective Electrophysiological Findings of Round Window Stimulation in a Model of Experimentally-induced Stapes Fixation

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Daniel J. Tollin, PhD

Hypothesis: Mechanical stimulation of the round window with a middle ear implant (MEI) with and without experimentally induced stapes fixation (SF) results in equivalent electrophysiological measures of cochlear microphonic (CM), compound action potential (CAP) and auditory brainstem response (ABR).

Background: Where normal oval window (OW) functionality is mitigated such as in cases of obliterative otosclerosis or aural atresia, stimulation of the round window (RW) provides an alternate pathway to stimulate structures of the inner ear.

Methods: Measurements of the CM, CAP and ABR were made in 6 chinchillas with application of the MEI to the RW with and without experimentally induced SF using pure tone stimuli (250 Hz to 20 kHz) presented at differing intensities (-20 to 90 dB SPL vs. 0.01 mV to 3.16 V).

Results: Waveform morphologies of the CM, CAP and ABR were similar between RW stimulation with and without SF. The CM thresholds ranged from 0.3 to 10 mV in RW stimulation while thresholds increased to 0.5 to 29 mV when the stapes was fixed. Although the thresholds changed with SF, the sensitivities of the CM and the amplitude dynamic range were identical. The CAP in both conditions demonstrated equivalent decreasing amplitudes and increasing latency with decreasing intensity (dB SPL vs. dB mV) and increased thresholds. ABR waveforms were preserved but with increased thresholds.

Conclusions: Mechanical stimulation of the RW in a model of SF generates inputs to the cochlear that are functionally similar in character to normal acoustic and RW mechanical inputs but with increased thresholds. With further study, MEIs may provide a surgical option for correction of OW fixation.

19.
Effect of Ossicular Prosthesis Biofilms on Middle Ear Scarring and Hearing Outcomes

Eric M. Jaryszak, MD, PhD; Edith Sampson, MS
Patrick J. Antonelli, MD

Objectives: Microbial biofilms have been associated with poor outcomes with a variety of biomedical implants, however, this relationship has not been established with middle ear implants. The purpose of this study was to determine if biofilms are present on ossicular chain reconstruction prostheses (ORPs) in patients undergoing revision tympanoplasty and if their presence correlates with middle ear scarring or hearing outcomes.
Study Design: Prospective and blinded.
Setting: Tertiary referral center
Patients: Patients undergoing revision tympanoplasty with prior ORP placement were enrolled.
Intervention/Main outcome measure: ORPs associated with poor hearing and residual or recurrent disease were collected, cultured, and examined using scanning electron microscopy. Audiometric thresholds and middle ear scarring scores were recorded.
Results: Twelve patients were included in the study. 25% of prostheses were culture positive. 67% had microscopic evidence of biofilm. No difference was found between the middle ear scarring scores ($P = 0.31$) and hearing outcomes ($P = 0.11$) of biofilm and non-biofilm prostheses. There was no correlation between middle ear scarring and degree of conductive hearing loss ($R2 = 0.04$, $P = 0.54$).
Conclusions: Biofilms are commonly found on ORPs at the time of revision tympanoplasty. The interaction between biofilms and the host environment is complex. Similarly, many factors besides biofilms may impact middle ear scarring and hearing.

IRB Approval Number: 243-2006

20.
Middle Ear Mechanics of Cartilage Tympanoplasty Evaluated by Time-Averaged Laser Holography

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Goals: To assess the effects of thickness and position of cartilage used to reconstruct the tympanic membrane (TM) using a novel technique, time-averaged laser holography.

Background: Cartilage is commonly used in TM reconstruction to prevent formation of retraction pockets. The thickness, position and shape of the cartilage graft may adversely affect TM motion (and hearing). We sought to systematically investigate these parameters in an experimental setting.

Methods: Computer-assisted opto-electronic laser holography was used in 5 human cadaveric temporal bones to describe the sound-induced TM motion for 500 Hz-12 kHz. Stapes velocity was also measured with a laser Doppler vibrometer. Baseline (control) measurements were made with the TM intact. Then a 0.5 mm or 1 mm thick oval piece of conchal cartilage was placed on the medial TM surface in the postero-superior quadrant. The cartilage graft was rotated so that it was either in contact with the bony annulus or not.

Results: At frequencies below 4 kHz, the holographic fringe patterns were similar in all experimental and control conditions. Above 4 kHz, TM motion was reduced, especially over the grafted TM, with greater effects seen for the 1 mm thick cartilage. Contact of the cartilage with bone of the external canal made no difference in TM motion or stapes velocity.

Conclusions: Laser holography is a promising technique to investigate middle ear mechanics after tympanoplasty. During cartilage tympanoplasty, having the cartilage extend a little beyond the bony annulus does not adversely affect middle ear sound transmission. Such positioning may prevent postoperative TM retraction.

Support / Acknowledgements: Work supported by NIDCD and a donation from L. Mittal

21.
Posterior Semicircular Canal Dehiscence: First Reported Case Series

Quinton Gopen; Dwight Jones, MD; Dennis S. Poe, MD
Guangwei Zhou, MD, ScD

Objective: To identify clinical, audiologic, and vestibular characteristics of posterior semicircular canal dehiscence (PCD)

Study design: Retrospective case review.

Setting: Tertiary referral center.

Patients: Ten patients, 5 pediatric and 5 adult, with PCD.

Interventions: Patients identified with suspicious clinical history and examination, confirmed by high resolution CT, air and bone audiometry, tympanometry, acoustic reflexes, and abnormally low VEMP thresholds.

Results: Clinical presentation consisted of vestibular and auditory symptoms. None of the patients were asymptomatic. The most common type of hearing loss was a mixed sloping hearing loss. However, two patients had purely conductive hearing loss, one of which had a negative surgical exploration with findings of a normal ossicular chain. One patient had a profound sensorineural hearing loss. Vestibular symptoms were more common in the adult patients than pediatric patients with chronic disequilibrium the most common complaint. Vestibular testing, when performed, demonstrated reduced peripheral vestibular function. VEMP testing confirmed dehiscence with the characteristic response to lower amplitude stimuli than seen in normal patients

Conclusion: Superior semicircular canal dehiscence syndrome, or Minor's syndrome, discovered in 1997 is a well accepted clinical etiology producing variable amounts of hearing loss and vestibular symptoms. Although radiographic studies have shown an approximately equal incidence of dehiscence in both the superior and posterior semicircular canals, clinical reports of posterior canal dehiscence to date have been limited to a few single case reports. This review represents the first case series for PCD.

IRB Approval Number: None required

22.
Improvement in Autophony Symptoms after Superior Canal Dehiscence Plugging

Benjamin T. Crane, MD; Lloyd B. Minor, MD
John P. Carey, MD

Objective: Autophony, or the unusually loud or disturbing sound of a patient's own voice, can be a prominent and disabling symptom of superior canal dehiscence (SCD) syndrome. The current study measures autophony symptoms before and after SCD plugging, to quantify the benefits of surgery.

Study Design: Patients undergoing SCD plugging during the past year completed a questionnaire prior to and three months after surgery. The questionnaire consisted of 26 statements to assess the disability caused by the sound of the patient's own voice. Patients graded each item on a scale from zero (never) to four (almost always) to how often they noted a symptom or experience. Typical statements included "hearing my voice has interfered with my ability to work", and "hearing my voice has caused me to avoid social situations." An autophony index (AI) was generated with a range from 0 (asymptomatic) to 104 (most severe)

Setting: Tertiary referral center

Patients: Eleven adults with SCD.

Intervention: SCD plugging via a middle fossa approach.

Outcome Measures: Change in AI.

Results: Pre-operatively the mean AI was 41 ± 30 (mean ± SD, range 0 – 86, one patient had no autophony symptoms). Post-op AI decreased to 15 ± 29, a significant ($p = 0.01$) decline. Of the 10 patients with pre-operative autophony, 7 had complete post-operative resolution. In two remaining patients the AI decreased but did not resolve, one of these had bilateral SCD with contra-lateral autophony. In one patient with co-existing palulous Eustachian tube AI increased after SCD plugging.

Conclusions: In patients with autophony symptoms, SCD plugging improved symptoms in 90% of patients.

IRB Approval Number: Exempt

23.
Vestibular-Ocular Reflex (VOR) as Predictor of Cerebral Death in Comatous Patients

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The diagnosis of cerebral death became critically important because of organ transplantations. Controversies still exist regarding this diagnosis.

Objective: To establish the role of VOR measured by a caloric test in predicting cerebral death in comatous patients.

Study design: Prospective case control.

Setting: Tertiary referral center. Methods: Sixty comatous patients (Glasgow index 8 or less) were studied: 49 male, 11 female, 7 to 83 years old. Twenty eight patients had head trauma, 18 had cerebral vascular accidents and 14 had medullary trauma, politraumatism, meningitis, hydrocephaly, cardiorespiratory arrest, hypovolemic or septic shock. Caloric test was done at bedside(saline irrigation at 0°C for 1 minute) and considered present and normal when both eyes deviated towards the stimulated side (19 patients, group 1), irregular unconjugated eye movements were considered VOR present but altered (11 patients, group 2). Absence of eye movement (30 patients, group 3) meant absent VOR.

Results: Group 1 had total recovery from coma in 42%, partial recovery in 37% and cerebral death in 21%. Group 2 figures were 9%, 18% and 73% respectively. Group 3 had 100% cerebral death. Group 1 was statistically different from groups 2 and 3 (Fischer exact test, X2 test).

Conclusion: Absent VOR predicts 100% of cerebral death and present unaltered VOR predicts only 21% of cerebral death in comatous patients.
Key words: vestibulo-ocular reflex, coma, cerebral death.

IRB Approval Number: 026/2007

24.
Prosthestic Implantation of the Semicircular Canals with Preservation of Rotational Sensitivity: A "Hybrid" Vestibular Implant

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C. Kaneko, PhD; L. Ling, PhD; K. Nie, PhD
F. Santos, MD; J.O. Phillips, PhD

Hypothesis:
It is possible to implant a stimulating electrode array in the semicircular canals without damaging rotational sensitivity.

Background:
A number of groups are attempting to develop a neural prosthesis to ameliorate abnormal vestibular function. Animal studies demonstrate that electrodes near the canal ampullae can produce electrically-evoked eye movements. The target condition is typically bilateral vestibular hypofunction. Such a device could potentially be more widely useful clinically, and would have a simpler road-map to regulatory approval if it produced minimal or no damage to the native vestibular system.

Methods:
An electrode array was designed for insertion into the bony semicircular canal adjacent to the membranous canal. It is sufficiently narrow so as to not compress the membranous canal. The arrays were linked to a Nucleus Freedom receiver/ stimulator. Five behaviorally-trained rhesus macaques had arrays placed in both lateral and posterior semicircular canals using a transmastoid approach and "soft-surgical" procedures similar to Hybrid cochlear implant surgery. Postoperative VOR was measured in a rotary chair.

Results:
All animals had minimal postoperative vestibular signs and were eating within hours of surgery. Four animals had no gain, phase or symmetry changes from .01 Hz to 2 Hz. The fifth animal had normal VOR, but was tested over a smaller frequency range.

Conclusions:
It is possible to implant the vestibular system with stimulating electrodes without loss of rotational sensitivity. If robust electrically-evoked vestibular responses can be reliably obtained, it will be feasible to consider treatment of a variety of vestibular disorders using prosthetic electrical stimulation.

Supported by HHS-N-260-2006-00005-C and Cochlear Corporation

American Otological Society Clinician-Scientist Award
Differentiation of Inner Ear Stem Cells
Progress Report: PI: Alan Cheng, MD

The main goal of our project is to characterize the fate of cells differentiated from murine cochlear stem cells *in vitro* and *in ovo*. We have found evidence that only a small subset of cochlear supporting cells has stem cell properties. Efforts during this past year have focused on isolating this subpopulation of supporting cells.

Wnt signaling has been found to regulate and maintain stem cells in the central nervous system, mammary glands, gastrointestinal system, as well as the hematopoietic system. Using a reporter mouse for the Wnt pathway (Axin2-lacZ), we have found active Wnt signaling in the neonatal cochlea. This activity decreases rapidly over the first 2 weeks of life, correlating with the drop in the number of sphere-forming stem cells (Oshima et al., JARO 2007). Cochleae harvested from the heterozygous Axin2-lacZ reporter mouse are dissociated and fluorescently labeled. These single cells are then analyzed and sorted via a fluorescent activated cell sorter into Axin2-positive and -negative groups. Sorted cells are then cultured in non-adherent conditions enriched by sphere-promoting growth factors (EGF, IGF, FGF). When compared to Axin2-negative cells, Axin2-positive cells form significantly more clonal spheres (166.5 ± 9.2 vs. 40 ± 2.8, $p<0.05$), suggesting that Axin2-positive cells are an enriched population of cells with proliferative capacity. Wnt activation has been shown to promote sphere propagation but not primary sphere formation by neural stem cells. Whether it has similar effects on cochlear stem cells are currently being examined.

When cultured in adherent conditions deprived of growth factors for 7-14 days, Axin2-positive cells differentiate into myo7a-positive hair cell-like cells as well as p27kip1-positive supporting cell-like cells. We plan on further characterizing these putative axin2-positive stem cells (as defined by self renewal, multipotency) *in vitro* and *in ovo* by injection into embryonic chicken otic vesicles. These results will be submitted for presentation in the upcoming 7th Molecular Biology of Hearing and Deafness in Boston, MA. We anticipate the proposed research to be completed within the next 18 months.

AOS Research Grant
An Integrative Genomic Approach to Discovering Otosclerosis Genes
Progress Report: PI: Marci M. Lesperance, MD

The goal of this research is to identify the first gene underlying nonsyndromic otosclerosis and to begin to define pathophysiologic mechanisms by integrating the gene into functional pathways. We have recruited Family 52, a large family in which autosomal dominant otosclerosis segregates. To date, a genome scan with approximately 6,000 single nucleotide polymorphisms (SNPs) has been performed using Illumina HumanLinkage-12 chips. Because penetrance in this family is reduced, also characteristic of otosclerosis, initial analysis was performed using DNAs from affected members and their interconnected relatives only. The genotype data was analyzed using MERLIN software to calculate multipoint logarithm-of-odds (LOD) scores. Preliminary analysis identified a novel locus on chromosome 8 with a multipoint LOD score of 2.889. The remainder of the genome generated negative LOD scores, allowing exclusion of linkage to the 7 previously reported otosclerosis loci. The chromosome 8 locus will be confirmed by genotyping short tandem repeat markers to more readily construct haplotypes and identify key recombination breakpoints. Additional family members will also be included once their affection status is confirmed by follow-up clinical evaluation. We hypothesize that gene-gene interactions and/or environmental factors may influence which individuals carrying an otosclerosis susceptibility allele will develop clinical otosclerosis versus subclinical disease or even possibly predominantly sensorineural hearing loss. This complexity may explain the difficulty in identifying otosclerosis genes to date. Creation of lymphoblastoid cell lines from individuals with surgically confirmed otosclerosis and/or conductive hearing loss, and unrelated controls is in progress in preparation for gene expression studies. Identification of genes that are significantly up- or down-regulated in otosclerosis as compared to controls will allow delineation of pathways important in development and maintenance of normal stapedial function and will suggest candidate genes for the other otosclerosis loci as well.

Research Grant 2008-2009—Progress Report
Title: Validation of a Mouse Model of Endolymphatic Hydrops
PI: Cliff A. Megerian, M.D. , Case Western Reserve University

In the guinea pig model of Ménière's disease (MD), surgery is required to induce endolymphatic hydrops (ELH), and the resulting inner ear sequelae. Based on preliminary data we hypothesized that the *PhexHyp-Duk* mouse will be a valuable model for study of the ELH-related ear condition. To test our hypothesis, we proposed:
Aim 1: To determine (part a) the degree and chronology of hearing deterioration in *PhexHyp-Duk*/Y (*PhexH-D*/Y) mice and (part b) the correlation between the development and degree of hearing loss with the onset and severity of ELH in this mutant.
Aim 2: To determine (part a) the pattern of histological progression of ELH and cochlear deterioration in and (part b) the correlation between the degree of deterioration and severity of ELH in *PhexH-D*/Y mutants.
Aim 3: To confirm whether the *PhexH-D*/Y mouse demonstrates immunohistochemical and molecular changes recently noted in the hydropic guinea pig model (1). We have made significant progress with respect to aim 1, part a and aim 2, part a. Briefly, ABR recordings at various times points (P21-P90) and histological analysis of representative temporal bones reveal that *PhexH-D*/Y mice typically develop adult onset, asymmetric, progressive hearing loss closely followed by the onset of ELH. ABR and histological data show that functional degeneration precedes structural degeneration. The major degenerative correlate of hearing loss and ELH in the mutants is the primary loss of spiral ganglion cells. Further, *PhexH-D*/Y mice develop ELH without evidence of endolymphatic duct (ED) obstruction, supporting the idea that ELH can be induced by a mechanism other than the blockade of longitudinal flow of endolymphatic fluid. We also recorded Vestibular Evoked Myogenic Potentials (VEMPs) to evaluate vestibular function in *PhexH-D*/Y mice. This is the first report of VEMP recordings in mice. Biphasic potentials were recorded from all normal animals. The mean threshold of the VEMP response in normal adult mice was 60 dB nHL with a mean peak latency of 6.25 ± 0.46 ms and 7.95 ± 0.42 ms for p1 and n1 peaks, respectively. At the maximum sound intensity used (100 dB nHL), no VEMPs were recorded from 4/5 mutants and 1/5 mutants showed an elevated threshold, but normal response, with regard to peak latency and amplitude. The histological findings in all of these *PhexH-D*/Y mice were consistent with distended membranous labyrinth, displaced Reissner's membrane, ganglion cell loss, and ELH. These results were published (2, 3).We are currently working on part b of aim 1 and 2. Similarly, we have started working on aim 3. These experiments are well underway and we will complete all initially proposed work by 6/30/09.

References (*PI served as the corresponding author in these publications):
1. Anne *et al**. Molecular changes associated with the endolymphatic hydrops model. Otol. Neurotol. **2007**; 28(6):834-41.
Publications directly relevant to the specific aims:
2. Megerian *et al.* Hear Res. **2008**; 237(1-2):90-105.
3. Sheykholeslami *et al**. Otology & Neurotology (accepted **Jan 2009**)
Other relevant publication:
4. Bixenstine PJ, *et al**. Spiral ganglion degeneration patterns in endolymphatic hydrops. Laryngoscope. **2008**; 118(7):1217-23.

Fellowship - American Otological Society Research Fund
Pharmacological Promotion of Neurite Outgrowth from Adult Inner Ear Neurons

PI: Samit Shah, B.S.

Interim Progress Report 02/01/2009

Loss of hearing is the most common sensory deficit in developed countries and ranks second only to arthritis among the common physiological dysfunctions affecting older adults. The prevalence of this debilitating perceptual defect is far greater than that of Down's syndrome, spina bifida, or phenylketonuria, and the effects are negative in regards to both the standard of living and life expectancy. Listeners with severe to profound SNHL cannot rely on hearing aids to perceive sound, so most listeners receive cochlear implants. However, the success of these devices is limited by the reduced availability of surviving SGNs that can be targeted for electrical stimulation. The significant distance between the implanted electrode array and the SGN cell bodies and the formation of scar tissue at the interface create a need for a large current spread to stimulate the target neurons. This increases the negative interference between neighboring electrodes, resulting in poor spectral resolution, frequency smearing, and ultimately reduced performance.

To date, there has been little investigation into the roles of the *Frizzled* receptors and their secreted ligands, the *Wnts*, in the inner ear, despite extensive work on their roles elsewhere in the nervous systems of both embryonic and adult animals. *Wnt* signaling has been implicated in axon guidance, synapse formation, and dendrite morphogenesis during development and our recent studies show that the expression of *Wnt* genes persists in the intact and noise-damaged adult cochlea. The general goal of the current research is to determine whether the activation or inhibition of *Wnt*-Frizzled signaling in the inner ear can promote the guidance and regeneration of dendritic projections from adult spiral ganglion neurons toward electrodes on cochlear implants to form private channels of

communication between the electrodes and their target neuronal populations. These biomolecular neural prosthetics could enhance device performance in noisy environments and improve acoustical fidelity of complex sounds. The specific focus of this research project is an investigation into the modulation of neurite outgrowth from auditory neurons using pharmacological activators of Wnt and neurotrophin signaling. During the first half of the fellowship period several key objectives of this project have been addressed.

First, a complete mRNA expression profile of *Frizzled* receptors in the spiral ganglion neurons was completed and a manuscript detailing our thorough analysis of *Wnt/Frizzled* expression in the adult cochlea is in the final phase of preparation. Furthermore, the *in vitro* cell culture technique previously developed by our group has been optimized for the current application. At this time, a systematic comparison of neurite growth effects induced by the application of *Wnt* modulatory drugs is underway and the results will provide an overview of pharmacologically-mediated *Wnt* function in adult spiral ganglion neurons. If a successful candidate is discovered in the future course of these experiments, the research plan will be refined to focus on the mechanistic action of a particular drug/target, commencing in *in vivo* trials.

AOS Clinician-Scientist Award
Molecular Mechanisms of Noise-Induced Delayed Primary Auditory Neuropathy
PI: Konstantina M. Stankovic, MD, PhD

Progress Report: January 31, 2009

Noise-induced hearing loss (NIHL) is a problem of profound clinical significance, growing magnitude, and major societal impact, because opportunities for overexposure abound, and exposures that damage hearing are not necessarily painful. Permanent threshold shift (PTS) in NIHL is caused by loss of hair cells or damage to their stereocilia bundles, which appears within hours or days post exposure. Loss of spiral ganglion neurons (SGNs), in contrast, is not seen until weeks or months post-exposure and is most dramatic in regions of inner hair cell loss, long suggesting that noise-induced SGN loss is "secondary" to inner hair cell loss, presumably via loss of the neurotrophin (NT) support formerly provided by the hair cells. However, recent experiments show that "primary" SGN degeneration, i.e. in the absence of hair cell loss and even in the absence of lingering threshold shifts, is a common sequela of NIHL. Within 24 hrs post exposure, confocal immunohistochemistry reveals loss of up to 50% of inner hair cell synapses and retraction of SGN terminals within the organ of Corti; comparable SGN loss is seen but only 1-2 yrs later. Identifying molecular mechanisms of this delayed neuropathy is our focus. To understand molecular mechanisms of this neuropathy, we have embarked on a genome-wide quantitative survey using a new technology of unprecedented sensitivity, Solexa deep sequencing. As a proof of principle, we have initially selected noise that causes permanent rather than temporary threshold shift to demonstrate that we can detect noise-induced changes in genes that span a spectrum of expression levels. We have generated two libraries of the organ of Corti from 6 week old mice: the unexposed library and the library 24 hrs after noise overexposure that causes permanent threshold shift. For each library, tissue was pooled from 20 ears (10 animals) to generate enough RNA. Each library of tags produced over five million sequences, which mapped to over 31,000 transcripts. In the unexposed library, we detected genes known to be uniquely or preferentially expressed in the adult inner ear, spanning a broad range of abundance, from prestin (3 tags/ millinon) to otospiralin (12313 tags per million). Over 1000 genes were differentially expressed between the unexposed and exposed organ of Corti ($p<0.001$ after accounting for multiple hypothesis testing). The complexity of these data was substantially simplified by performing Ingenuity Pathway Analysis, which identified claudins and genes involved in tight junctional signaling as potentially key molecular players in the pathogenesis of PTS. Having demonstrated feasibility of the Solexa sequencing approach in the cochlea, and the utility of analyzing pathways rather than individual genes, we now plan to apply this technology to uncover pathways that control noise-induced delayed primary neuropathy.

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1909-10	Frederick L. Jack, MD
1911-12	Edward B. Dench, MD
1913-14	J.F.McKernon, MD
1915-16	C.W. Richardson, MD
1917	C.R. Holes, MD
1918	Norval H. Pierce, MD
1919	Ewing W. Day, MD
1920	Robert Lewis, MD
1921	W.P. Eagleton, MD
1922	H.S. Birket, MD
1923	G. Shambaugh, Sr., MD
1924	John B. Rae, MD
1925	E.A. Crockett, MD
1926	Thomas J. Harris, MD
1927	Arthur B. Duel, MD
1928	M.A. Goldstein, MD
1929	J.G. Wilson, MD
1930	S. Mac C. Smith, MD
1931	D.H. Waler, MD
1932	L.W. Dean, MD
1933	G.I. Tobey, Jr., MD
1934	John R. Page, MD
1935	Samuel J. Crowe, MD
1936	F.R. Packard, MD
1937	E.P. Fowler, MD
1938	Harris P. Mosher, MD
1939	Isidore Friesner, MD
1940	Horace Newhart, MD
1941	George M. Coates, MD
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	Frederick T. Hill, MD
1955	D.E.S. Wishart, MD
1956	William.J McNally, MD
1957	John R. Lindsay, MD
1958	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
1965	Harry Rosenwasser, MD
1966	Howard P. House, MD
1967	James A. Moore, MD
1968	G. Shambaugh, Jr., MD
1969	Frank D. Lathrop, MD
1970	Francis L. Lederer, MD
1971	John E. Bordley, MD
1972	Walter P. Work, MD
1973	Ben H. Senturia, MD
1974	Wesley H. Bradley, MD
1975	Lester A. Brown, MD
1976	Victor Goodhill, MD
1977	Harold Schuknecht, MD
1978	Clair M. Kos, MD
1979	G. Dekle Taylor, MD
1980	Eugene Derlacki, MD
1981	Richard J. Bellucci, MD
1982	J. Brown Farrior, MD
1983	Jack V. Hough, MD
1984	Cary N. Moon, Jr., MD
1985	Francis A. Sooy, MD
1986	Brian F. McCabe, MD
1987	Harold G. Tabb, MD
1988	Rihard R. Gacek, MD
1989	D. hane Cody, MD
1990	H.A. Ted Bailey, Jr., MD
1991	William F. House, MD
1992	Mihael Glasscock, III, MD
1993	Mansfield F.W. Smith, MD
1994	Robert I. Kohut, MD
1995	Robert A. Jahrsdoerfer, MD
1996	Derald E. Brackmann, MD
1997	Joseph C. Farmer, Jr., MD
1998	Charles M. Luetje, MD
1999	Gregory J. Matz, MD
2000	C. Gary Jackson, MD
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2006	John K. Niparko, MD
2007	Antonio De La Cruz, MD
2008	Clough Shelton, MD
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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
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Houston, TX

P. Ashley Wackym, MD (Active 1997)
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Francis I. Catlin, MD (1996 (1975))
Houston, TX

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

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L.B.W. Jongkees, (Honorary 1968)
S2 1071, THE NETHERLANDS

Yasuya Nomura, (Honorary 1992)
Tokyo 142, JAPAN

Michel Portmann, (Honorary 1983)
Bordeaux 33000, FRANCE

Naoaki Yanagihara, MD (Honorary 2008)
Matsyama, JAPAN

Members Deceased Since Last Spring Meeting

Patrick J. Doyle, MD (Senior 1996 (1987))
Vancouver, BC
Active Member: 1987
Senior Member : 1996
Date of Death: 5/21/2008

Richard E. Marcus, MD
Winnetka, IL
Active Member: 1975
Senior Member: 1987
Date of Death: 1/30/2009

Joseph Sataloff, MD (Senior 1994 (1960))
Philadelphia, PA
Active Member: 1960
Senior Member: 1994
Date of Death: 9/26/2008

Summary of Professional Practice Gaps, educational needs and educational planning constructs for American Otological Society CME activities.

Professional practice gap	Educational need	Knowledge or competence (educational obj)
Lack of knowledge about importance of molecular genetics and biology	Deeper understanding of the role of molecular genetics and biology in scientific advances in otology and neurotology	Competence Describe the potential limitations in drug delivery to the inner ear
Need for new information/ strategies for identification/mgt of vestibular disorders	Current practices and standards for the appropriate diagnosis and management of vestibular disorders	Knowledge Name two or three contemporary and emerging technologies in the diagnosis and treatment of vestibular disorders
Lack of knowledge about the clinical history/ presentation of acoustic neuromas	Current practices and standards for the diagnosis and medical management of acoustic neuromas	Knowledge Identify emerging technologies in the medical management of acoustic neuroma
Lack of knowledge about non-surgical treatments for skull base tumors	Current practices and standards for non-surgical treatment of skull base tumors	Knowledge Discuss current and prospective medical management protocols for otologic disease
Need for knowledge and clinical application of implantable devices as a treatment alternative	Availability and role of implantable devices such as cochlear implants, etc	Competence Recognize the applicability and limitations in use of implantable devices for hearing rehabilitation
Need for discussion of current approaches in surgical and medical management of patients with otologic conditions	Discuss current and prospective medical management protocols for otologic disease	Knowledge Discuss current and prospective medical management protocols for otologic disease

Relationship to desirable attribute (ACGME)	**Program ID #**
1, 2, 4	Papers 1, 2, 3, 8 Basic Science Lecture 1, 2
1, 2, 3, 4	Papers 8, 21, 23, 24; Basic Science Lecture 1, Panels 1, 2
1, 2, 3	Basic Science Lecture 1; Papers 8, Panel 1
1, 2, 3	Paper 8, Basic Science Lecture 1, Panel 1
1, 2, 3	Papers 6, 7, 10, 12, 13, 14, 15, 17, 18, 19, 20, 24 Basic Science Lecture 2
1, 2, 3, 6	Papers 1, 3, 4, 5, 6, 7, 9, 10, 11, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24 Basic Science 1, 2 Panels 1, 2