



**PROGRAM and ABSTRACTS**

of the

*One Hundred Thirty-Sixth Annual Meeting*

**AMERICAN OTOLOGICAL  
SOCIETY, INC.**

**May 3-4, 2003**

**Gaylord Opryland Resort &  
Convention Center  
Nashville, Tennessee**

**OFFICERS**  
**JULY 1, 2002- JUNE 30, 2003**

**PRESIDENT**

Horst R. Konrad, M.D.  
Southern Illinois University School of Medicine  
P. O. Box 19662  
Springfield, IL 62794-9662

**PRESIDENT-ELECT**

Jeffrey P. Harris, M.D., Ph.D.  
UCSD Medical Center  
200 W. Arbor Drive 8895  
San Diego, CA 92103-8895

**SECRETARY TREASURER**

Clough Shelton, M.D.  
University of Utah School of Medicine  
50 North Medical Drive, 3C120  
Salt Lake City, UT 84132

**EDITOR-LIBRARIAN**

Sam E. Kinney, M.D.  
60 Pebblebrook Lane  
Moreland Hills, OH 44022

**COUNCIL**

The above officers and  
A. Julianna Gulya, M.D.  
Richard A. Chole, M.D., Ph.D.  
John K. Niparko, M.D.  
Antonio De La Cruz, M.D.

The American Otological Society is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

This Continuing Medical Education offering meets the criteria for eight (8) credit hours in Category One (1) of the Physician's Recognition Award of the American Medical Association.

**SATURDAY, May 3, 2003**

REGISTRATION – 7:00 am

BUSINESS MEETING – 7:00 am

ROOM: Delta Ballroom B

(Restricted to Members)

Minutes of the Annual Meeting 2002

Introduction of New Members

Election of Nominating Committee

Report of the Secretary-Treasurer

Report of the Editor-Librarian

SCIENTIFIC PROGRAM – 7:30 am

ROOM: Delta Ballroom B

(Open to Non-Members)

\*Speaker

7:30 am      Remarks by the President  
**Horst R. Konrad, MD\***

7:45 am      Introduction of the Guest of Honor  
**James F. Battey, Jr., MD, PhD**

7:50 am      Presidential Citation  
**Maureen T. Hannley, PhD**

### **Auditory Basic Science Topics**

1. 7:55 am      **Audiological and Clinical Outcomes  
in Auditory Neuropathy**  
Colm Madden, MB, FRCSI  
Mark Boston, MD\*  
John Greinwald, MD  
Daniel Choo, MD
  
2. 8:05 am      **Spiral Ligament and Stria Vascularis  
Changes in Cochlear Otosclerosis:  
Effects on Hearing Level**  
Joni K. Doherty, MD, PhD\*  
Fred H. Linthicum, Jr., MD
  
3. 8:15 am      **Caspase Inhibition Prevents Apoptosis of  
Trophic Factor Deprived Auditory  
Neurons**  
Francois Lallemand, BS, Syed Ahsan, MD  
Adrian A Eshraghi, MD, Marek Polak, PhD  
Brigitte Malgrange, PhD  
Philippe P Lefebvre, MD, PhD  
Fred F Telischi, MEE, MD \*  
Thomas J Balkany, MD  
Thomas R Van De Water, PhD\*

4. 8:25 am **Sound Evoked Motion of the Ossicles in the Guinea Pig Middle Ear**  
 Manoj Kumar, MS, FRCS\*  
 Wei Dong, PhD  
 Nigel Cooper, PhD
- 8:35 am **DISCUSSION**
- 8:45 am **Panel— Noise-Induced Hearing Loss: Current Status and New Directions**  
 Moderator: Leonard P. Rybak, MD, PhD  
 Panelists:  
 Kathleen Campbell, PhD  
 Robert A. Dobie, MD  
 Richard D. Kopke, MD  
 Brenda L. Lonsbury-Martin, PhD
- 9:30 am **DISCUSSION**
- 9:45 am **INTERMISSION—Break with Exhibitors**

**Autoimmune Inner Ear Disease**

5. 10:15 am **Kinetics of Round Window Permeability**  
 Douglas E. Mattox, MD\*  
 Don R. Christian, MD  
 Jake A. Gilbert, BA  
 Girlandia Goepfert, MD  
 Henry F. Edelhauser, PhD
6. 10:25 am **Intratympanic Gentamicin Therapy for Meniere's Disease: A Meta-Analysis**  
 Stanley H. Chia, MD\*  
 John P. Anderson, PhD  
 Jeffrey P. Harris, MD, PhD
7. 10:35 am **Randomized Trial of Methotrexate for Autoimmune Inner Ear Disease**  
 Jeffrey P. Harris, MD, PhD\*  
 M. Jennifer Derebery, MD  
 Mark A. Espeland, PhD  
 Bruce J. Gantz, MD  
 A. Julianna Gulya, MD  
 Michael Weisman, MD  
 for the AIED Study Group
8. 10:45 am **Hearing Loss as an Early Manifestation and Indicator of Exacerbation in Wegener's Granulomatosis**  
 Mark S. Driver, MD\*  
 Sivasanker Bakthavachalam  
 Jeffrey H. Spiegel, MD  
 Clarke Cox, PhD  
 Kenneth M. Grundfast, MD  
 Peter A. Merkel, MD, MPH

**Surgical Treatment of Conductive Hearing Loss**

9. 11:05 am    **A Comparison of Ossiculoplasty with Stapes to Malleus and Stapes to Eardrum Prostheses**  
Manohar Bance MB, MSc, FRCSC\*  
David P. Morris MBBS, FRCS  
Rene G. van Wijhe BSc, M.Eng  
Rachael Smith MD
10. 11:15 am    **Hearing Outcome of Laser Stapedotomy Minus Prosthesis (STAMP) Versus Conventional Laser Stapedotomy**  
Herbert Silverstein, MD  
Karen K. Hoffmann, MD\*  
Lance E. Jackson, MD  
Jack H. Thompson, Jr., PhD, PA-C  
Joshua P. Sleeper, BA
11. 11:25 am    **Partial Promontory Technique in Stapedotomy Cases with Narrow Niche**  
Michelle M. Inserra, MD\*  
Patricia J. Yoon, MD  
Theodore P. Mason, MD  
Joseph B. Roberson, MD
12. 11:35 am    **A New Approach for Malleus/Incus Fixation: No Prosthesis Necessary**  
Michael D. Seidman, MD\*  
Seilesh Babu, MD
13. 11:45 am    **Trans Facial Recess Ossicular Chain Reconstruction: Surgical Technique and Early Results**  
Nikolas H. Blevins, MD\*

11:55 am    **DISCUSSION**

12:15 pm    **Group Photograph**  
Members of AOS  
Location to be Announced

**Lunch with Exhibitors**

Sunday, May 4, 2003

REGISTRATION – 12:00 Noon

BUSINESS MEETING – 12:30 pm

ROOM: Delta Ballroom D

(Restricted to Members)

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-Head and Neck Surgery

Report of the Audit Committee

Report of the Membership Development Committee

Report of the Nominating Committee

Unfinished Business

New Business

SCIENTIFIC PROGRAM – 1:00 pm

ROOM: Delta Ballroom D

(Open to Non-Members)

\*Speaker

### Superior Canal Dehiscence

- 14. 1:00 pm      **Investigations of the Effect of Superior Semicircular Canal Dehiscence on Hearing Mechanisms**  
John J. Rosowski PhD\*  
Jocelyn E. Songer MS  
Heidi H. Nakajima MD, PhD  
Kelly M. Brinsko BS  
Saumil N. Merchant MD
  
- 15. 1:10 pm      **Superior Semicircular Canal Dehiscence Presenting as "Conductive" Hearing Loss Without Vertigo**  
Anthony A. Mikulec, MD\*  
Mitchell J. Ramsey MD  
Michael J. McKenna MD  
Joseph B. Nadol, Jr., MD  
Steven D. Rauch, MD  
John J. Rosowski PhD  
Hugh D. Curtin, MD  
Saumil N. Merchant MD

16. 1:20 pm **Long Term Results of Mastoid Obliteration with Bone Cements**  
Jennifer L. Maw, MD\*

1:30 pm **DISCUSSION**

1:40 pm **Vestibular Panel**  
Moderator: Phillip A. Wackym, MD  
Topics and Panelists  
**Use of Intratympanic Steroids**  
Lloyd B. Minor, MD

**Use of Intratympanic Gentamicin**  
John P. Carey, MD

**Differentiating Meniere's Disease from Superior Semicircular Canal Dehiscence**  
Phillip A. Wackym, MD

**Vestibular Rehabilitation**  
Neil T. Shepard, PhD

2:30 pm **DISCUSSION**

2:45 pm **INTERMISSION—Break with Exhibitors**

### **Cochlear Implants**

17. 3:15 pm **Central Auditory System Development and Plasticity in Children with Cochlear Implants Who are Implanted Later in Childhood**  
Anu Sharma, PhD\*  
M. F. Dorman, PhD  
N. Wendell Todd, MD  
Jolie Fainberg, MS  
Phillip Gilley, MS  
Kathryn Martin, MS  
Anthony Spahr, MS

18. 3:25 pm **Language and Speech Development in Deaf Children and Infants Following Cochlear Implantation**  
Richard T. Miyamoto, MD\*  
Derek M. Houston, PhD  
Karen Iler Kirk, PhD  
Amy E. Perdew, MS  
Mario A. Svirsky, PhD

19. 3:35 pm **Optimizing Cochlear Implant Efficiency with Modiolus-Based Return Electrode**  
Steven Y. Ho, MD\*  
Richard J. Wiet, MD  
Claus-Peter Richter, MD

20. 3:45 pm **Meningitis in Cochlear Implant Recipients: The North American Experience**  
Noel L. Cohen, MD\*  
J. Thomas Roland, Jr., MD
- 3:55 pm **DISCUSSION**
21. 4:05 pm **Neonatal Middle Ear Effusion and Chronic Otitis Media with Effusion**  
Karen Jo Doyle, MD, PhD\*  
Ying Yee Kong, MA  
Patricia Dallaire, MA  
Karen Strobel, MA  
Mark Ray, MD
22. 4:15 pm **Hearing Preservation Rates During Vestibular Schwannoma Resection: Retrosigmoid Approach and Direct Cochlear Nerve Monitoring**  
Christopher J. Danner, MD\*  
Roberto A. Cueva, MD
23. 4:25 pm **Comparison of Vestibular Nerve Afferent and Eye Movement Responses to Galvanic and Rotational Stimuli**  
Charley C. Della Santina, PhD, MD\*  
Timothy E. Hullar, MD  
John P. Carey, MD  
Americo A. Migliaccio, PhD  
Lloyd B. Minor, MD
24. 4:35 pm **Benign Paroxysmal Positional Nystagmus in Patients Receiving Ototoxic Medications**  
F. O. Black, MD\*  
S. C. Pesznecker, RN  
Valerie Stallings
- 4:45 pm **DISCUSSION**
- 4:55 pm **Introduction of New President Jeffrey P. Harris, MD, PhD**

## **2003 Program Advisory Committee**

**Hilary A. Brodie, MD, PhD  
Karen Jo Doyle, MD  
Rick A. Friedman, MD, PhD  
Joel A. Goebel, MD  
Anil K. Lalwani, MD  
John T. McElveen, Jr., MD  
Saumil N. Merchant, MD  
Allan Rubin, MD  
N. Wendell Todd, Jr., MD  
Thomas R. Van De Water, MD  
Phillip A. Wackym, MD**

**COSM 2004**

**137<sup>th</sup> AOS Annual Meeting**

**May 1-2, 2004**

**J. W. Marriott Desert Ridge Resort & Spa  
Phoenix, Arizona**

**Abstract Deadline: October 15, 2003**

**Abstract form available from**

**Website—[www.americanotologicalsociety.org](http://www.americanotologicalsociety.org)**

**E-Mail—[segossard@aol.com](mailto:segossard@aol.com)**

**Administrative Office**

**American Otological Society, Inc.**

**2720 Tartan Way**

**Springfield, IL 62707**

**Ph/Fax: 217.483.6966 (Voice)**

## **Audiological and Clinical Outcomes in Auditory Neuropathy**

Colm Madden, MB, FRCSI, Mark Boston, MD  
John Greinwald, MD, Daniel Choo, MD

**Objective:** To medically and audiotically characterize a population of children diagnosed with auditory neuropathy (AN).

**Study Design:** A retrospective chart review of patients diagnosed with AN.

**Setting:** Tertiary care pediatric referral center.

**Patients:** Thirty-five patients with AN identified from a pediatric otology/audiology clinic.

**Outcome Measures:** Clinical data, audiometric thresholds.

**Results:** Thirty-five children were diagnosed with AN at our institution. A genetic factor in AN is suggested by our identification of 3 families (each with 2 affected siblings). Clinical features common amongst our population included a history of hyperbilirubinemia (57%), prematurity (54%), ototoxic drug exposure (48%), neonatal ventilator dependence (37%) and a family history of AN (17%). At least one risk factor was present in 83% of our patients. Full clinical and audiological data was available for all of the 35 children. This included Otoacoustic Emissions (OAEs), Auditory brainstem responses (ABR) with Cochlear Microphonics (CM) and age-appropriate audiometry. Significantly, 16 of these 35 patients showed improvement in behavioral thresholds over time (mean follow-up 31 months, range 0-119), indicating that a subset of children with AN may recover useful hearing levels. A significant improvement was seen in those children with a history of ototoxic medication use at birth ( $p=0.02$ ). Cochlear implantation provided an effective habilitation in 6 children.

**Conclusions:** This data shows that management of children with AN requires serial clinical and audiometric evaluations. Prematurity, genetics, hyperbilirubinemia and ototoxic medication appear to be significant factors in the development of AN. A history of exposure to ototoxic medication is associated with spontaneous improvement in hearing thresholds.

## **Spiral Ligament and Stria Vascularis Changes in Cochlear Otosclerosis: Effects on Hearing Level**

Joni K. Doherty, MD, PhD, Fred H. Linthicum, Jr., MD

**Objective:** To investigate the pathogenesis of sensorineural hearing loss (SNHL) in cochlear otosclerosis, we (1) define the relationship between spiral ligament (SL) hyalinization, stria vascularis (SV) atrophy, and SNHL; and (2) describe changes within the lateral wall of the cochlea in terms of ion transport channel expression and fibrocyte survival.

**Study Design:** Retrospective

**Setting:** Tertiary referral center

**Patients:** Seventy-five cochleas from 57 temporal bone donors, ages 49-85 (avg. 71.3), with histologic evidence of CO, including SL hyalinization.

**Intervention:** Audiography.

**Main Outcome Measures:** In basal, middle posterior, middle anterior, and apical turns of celloidin-embedded cochlear sections, measurements of SL width and hyalinization were compared with SV and bone conduction hearing thresholds (BCHT). To exclude other causes of SNHL, cochleas were assessed for hair cell, dendrite, and spiral ganglion counts. Expression of the ion transport molecules Na-K-ATPase, connexin26 (Cxn26) and carbonic anhydrase II (CAII) were assessed by immunohistochemical techniques.

**Results:** SL hyalinization correlated directly with SV atrophy and hearing loss (BCHT) in the middle posterior and apical turns of the cochlea ( $p < 0.05$ ). These relationships did not achieve significance for the basal or middle anterior turns. Decreased CAII, Cxn26, and Na-K-ATPase immunostaining of type I, II, and IV fibrocytes of the SL was observed in CO sections compared with normal cochlea. However, overall immunostaining results were inconsistent.

**Conclusions:** These data suggest that SL structure and function are essential for SV survival. Additionally, malfunctioning SV may result in altered endocochlear potential and SNHL.

This work was supported by a grant from the NOHR Foundation.

## **Caspase Inhibition Prevents Apoptosis of Trophic Factor Deprived Auditory Neurons**

Francois Lallemand, BS, Syed Ahsan, MD

Adrian A Eshraghi, MD, Marek Polak, PhD

Brigitte Malgrange, PhD, Philippe P Lefebvre, MD, PhD

Fred F Telischi, MEE, MD, Thomas J Balkany, MD

Thomas R Van De Water, PhD

**Hypothesis:** Caspases participate in the apoptosis of auditory neurons that occurs following a loss of trophic factor support (i.e. auditory hair cells).

**Background:** The current trend to implant people with substantial residual hearing requires preservation of enduring sensorineural elements. Because hair cells provide trophic support for auditory neurons, it would be desirable to preserve these sensitive cells in addition to the neurons. Loss of trophic support causes oxidative-stress damage in the auditory neurons and apoptosis. Procaspases are present in healthy hair cells and neurons and when activated are key participants in oxidative-stress initiated apoptosis.

**Methods:** Dissociated spiral ganglion cell cultures were grown either with or without neurotrophin (hrBDNF). Immunolabeling and Western blots identified caspase activation. Neurotrophin-deprived cultures were treated with pan-and specific caspase inhibitors. Cultures were stained for neurofilaments and the percentage of neuron survival was determined.

**Results:** These in vitro studies show that several members of the caspase family actively participate in the apoptotic cell death of neurotrophin-deprived auditory neurons. Because more than one caspase participated in the apoptosis of trophic factor deprived auditory neurons the most effective treatment was a pancaspase inhibitor.

**Conclusion:** Treatment of neurotrophin-deprived auditory neurons with a pancaspase inhibitor prevented the loss of these neurons due to oxidative-stress induced apoptosis. Because caspases have been shown to participate in the apoptotic cell death of both hair cells and neurons a pancaspase therapeutic strategy may have application to cochlear implantation by reducing the loss of hair cells and any subsequent loss of neurons.

## Sound Evoked Motion of the Ossicles in the Guinea Pig Middle Ear

Manoj Kumar, MS, FRCS, Wei Dong, PhD  
Nigel Cooper, PhD

**Objective:** The aim of the experiment was to find out the relative movement of the malleus and incus in response to varying frequencies of sound stimuli.

**Method:** Experiments were performed in 5 anaesthetized guinea pigs. Surgically, the bulla was exposed. Small holes were made in the bulla to visualize and measure the ossicular movements. A heterodyne laser interferometer was used to measure the sound evoked ossicular motion.

**Background:** Three orthogonal components are necessary in describing the motion of a point in 3-D space. Direct measurement of these displacement components of the ossicles were not possible, since the interferometer system we used provided information only in one direction of the object under study at a time. The measurements were carried out from a single point on the malleus and incus from a wide range of viewing angles, by rotating the goniometer system attached to the guinea pig head, around the vertical and horizontal axes. This allowed 3D reconstruction of the motion of the ossicles.

**Results and Conclusion:** 3D reconstruction of the umbo and the end of long process of the incus showed linear motion for low frequency stimuli and more elliptical or circular motion for higher frequency stimuli. The mean lever ratio was calculated from the above recordings increased when the frequency of the sound stimuli exceeded 3.2kHz. The change in the lever ratio appears to reflect a change in the ossicle's mode of vibration between low and high frequencies.

## Kinetics of Round Window Permeability

Douglas E. Mattox, MD, Don R. Christian, MD  
Jake A. Gilbert, BA, Girlandia Goepfert, MD  
Henry F. Edelhauser, PhD

**Hypothesis:** This study used a novel in vitro perfusion chamber to establish quantitative data on the permeability of the round window membrane (RWM) to 3H-dexamethasone, 3H-water and diclofenac (NSAID).

**Background:** Intratympanic drug delivery is becoming an increasingly important tool in the management of inner ear diseases including autoimmune hearing loss, sudden hearing loss and Meniere's disease. Although the transport of numerous molecules and particles across the RWM has been described, an actual permeability constant ( $K_{trans}$ ) for various drugs and molecules has not been defined. We have established an in vitro model to test the permeability of the RWM to various molecules.

**Methods:** The RWM of adult guinea pigs was mounted on a perfusion device, which clamped the RWM between two chambers. The upper chamber (middle ear side) allowed for a depot of drug. The lower chamber (inner ear side) was continuously perfused with balanced salt solution. A fraction collector collected the outflow every hour for 24 hours. The amount compound that had diffused through the RWM was measured in a liquid scintillation counter. The average permeability constant,  $K_{trans}$ , (cm/sec) over the 24 hours period was calculated.

**Results:** The  $K_{trans}$  for 3H-dexamethasone was  $1.5 \pm 0.4 \times 10^{-7}$  and  $3.1 \pm 0.8 \times 10^{-5}$  for 3H-water ( $p < 0.001$ ). The RWM was impermeable to diclofenac for 12 hours.

**Conclusions:** This study shows the feasibility of an in vitro method to measure RWM permeability. Both 3H-dexamethasone and 3H-water were permeable to the RWM, with the later having a significantly higher permeability constant. The RWM was impermeable to diclofenac.

Supported by the American Otologic Society Research Fund and Alcon Laboratories, Ft. Worth, Texas.

## **Intratympanic Gentamicin Therapy for Meniere's Disease: A Meta-Analysis**

Stanley H. Chia, MD, John P. Anderson, PhD  
Jeffrey P. Harris, MD, PhD

**Objective:** This study compares the effectiveness of different techniques of intratympanic gentamicin administration for Meniere's Disease.

**Data Sources:** Medline search for English language literature, 1978-2002, was performed using key words: intratympanic, gentamicin, therapy, Meniere's, disease.

**Study Selection:** Inclusion criteria to select articles for meta-analysis were: clear description of gentamicin delivery technique; clearly reported vertigo control results; report of hearing loss post-treatment. Eight studies (n=247) describing the multiple daily dosing technique (TID delivery for  $\geq 4$  days); seven studies (n=262) describing the weekly/biweekly dosing technique (weekly/biweekly injections for fixed number of doses or termination with vertigo relief); four studies (n=103) of the low dose technique (1-2 injections with retreatment for recurrent vertigo); four studies (n=156) of continuous microcatheter delivery; and five studies (n=241) of the titration technique (daily or weekly doses until onset of vestibular symptoms) were entered into the model.

**Data Extraction:** Vertigo control results were stratified into complete, substantial, or poor control. Hearing results were separated by profound, partial, or no hearing loss. Patients were further divided into those receiving  $< 120$  mg of gentamicin (low dose group, n=278), and  $\geq 120$  mg (high dose, n=323).

**Data Synthesis:** Rates of vertigo control and hearing loss between delivery techniques and dosage groups were analyzed by chi-square.

**Conclusions:** Maintaining gentamicin dosage under 120 mg can decrease hearing loss ( $p < 0.05$ ) without sacrificing vertigo control. Titration method of delivery provides the greatest complete vertigo control ( $P < 0.01$ ). Low dose therapy has the least associated hearing loss ( $p < 0.05$ ), but also less vertigo control ( $p < 0.05$ ) when compared to titration and multiple daily dosing methods.

## Randomized Trial of Methotrexate for Autoimmune Inner Ear Disease

Jeffrey P. Harris, MD, PhD, M. Jennifer Derebery, MD  
Mark A. Espeland, PhD, Bruce J. Gantz, MD  
A. Julianna Gulya, MD, Michael Weisman, MD  
for the AIED Study Group

**Context:** A number of therapies have been proposed for the long-term management of steroid-responsive rapidly progressive bilateral sensorineural hearing loss (RPBSHL), i.e. autoimmune inner ear disease (AIED). However, none of these therapies had been rigorously evaluated.

**Objective:** To assess the efficacy of methotrexate (MTX) in maintaining hearing gains achieved with corticosteroid (prednisone) therapy in AIED.

**Design, Setting, Participants, Intervention:** A double-blind, randomized, placebo-controlled trial conducted from January 1998 to September 2002 at 10 otolaryngology out patient clinics in the United States. Of 116 participants with RPBSHL, 67 “responded” to prednisone and accepted randomization to either oral MTX (up to 15 – 20 mg weekly; 33 patients) or placebo (34 patients), in combination with prednisone taper. Follow-up examinations, including audiometric evaluation, were carried out at 4, 8, 12, 24, 36, 48, and 52 weeks, or until hearing loss was documented, whichever came first.

**Main Outcome Measure:** Time after the date of randomization to loss of hearing gain achieved with prednisone.

**Results:** In the intention-to-treat analysis, MTX was no more effective than placebo (fitted relative hazard  $1.31 \pm 0.34$ ,  $p = 0.29$ ) in preventing the loss of hearing gained with prednisone.

**Conclusions:** MTX does not appear to be effective at the dosage used in this study in maintaining the hearing gain achieved with prednisone therapy in AIED.

**Acknowledgement:** NIH/NIDCD and the American Academy of Otolaryngology/Head and Neck Surgery.

## **Hearing Loss as an Early Manifestation and Indicator of Exacerbation in Wegener's Granulomatosis**

Mark S. Driver, MD, Sivasanker Bakthavachalam  
Jeffrey H. Spiegel, MD, Clarke Cox, PhD  
Kenneth M. Grundfast, MD, Peter A. Merkel, MD, MPH

**Objective:** Describe the frequency, type, and clinical course of hearing loss in patients with Wegener's Granulomatosis (WG).

**Study Design, Setting, Patients:** Retrospective cohort study of all patients with WG seen in one year at an academic medical center.

**Main Outcome Measures:** Hearing loss documented by pure-tone audiogram.

**Results:** 35 patients included: 19 men, 16 women; mean age 55 years (range 22-87); 32 were ANCA-positive, mean disease duration of 47 months. (2-196). 15 patients (42.8%) had documented hearing loss: Accounting for mixed losses, 12 (34.2%) had sensorineural (SNHL) and 10 had conductive loss (CHL). 3 of 9 cases of CHL improved with treatment of WG (cyclophosphamide) 1 worsened and 1 remained stable. Of 12 patients with SNHL; 1 improved, 1 worsened, and 3 remained stable (on cyclophosphamide). 7 patients had hearing loss requiring amplification. 5 of 35 (14%) patients had established hearing loss months-years prior to diagnosis of WG. Hearing loss occurred both upon initial presentation and with disease relapse.

**Conclusions:** Both SNHL and CHL are common in WG, may result in significant morbidity, and may precede the diagnosis of WG by years. CHL is more likely to improve with treatment of WG than SNHL. The significance of both types of hearing loss in patients with WG may be used as a manifestation helpful in diagnosis and as an indicator of severity before other manifestations are manifest. These data suggest that it may be appropriate to perform screening audiograms in all patients with newly diagnosed or relapsed WG.

## **A Comparison of Ossiculoplasty with Stapes to Malleus and Stapes to Eardrum Prostheses**

Manohar Bance, MB, MSc, FRCSC

David P. Morris, MBBS, FRCS

Renie G. van Wijhe, BSc, M.Eng, Rachael Smith, MD

**Hypothesis:** In the ear with a missing incus, reconstruction from the stapes head to the malleus will result in different stapes responses compared to reconstruction to the eardrum.

**Background:** In the ear with a missing incus, two commonly used reconstruction methods either a prosthesis from the stapes head to the malleus (Malleus Stapes Assembly (MSA)), or a prosthesis from the stapes head to the eardrum (Partial Ossicular Replacement Prosthesis - PORP). The differences in function are not clear. Our objective was to compare these reconstructions in a cadaveric human middle ear model.

**Methods:** Eight fresh human cadaveric temporal bones were harvested within 48 hours after death. The stapes footplate vibrations were measured using a Laser Doppler Vibrometer, and compared for the two types of reconstruction. The tympanic membrane was stimulated with a sound input of 80 – 95 dB SPL over a frequency range of 0.2 to 8kHz. Measurements were made for several lengths of each prosthesis to allow for the confounding effects of tension.

**Results:** Our results show substantial differences in the performance of the MSA and PORP prostheses, in some narrow frequency ranges. On average, the results were within 10dB of each other. The differences are complex and frequency dependant. In general, MSA resulted in slightly better high frequency responses.

**Conclusions:** The stapes responses for MSA and PORP prostheses show differing resonances at narrow frequency ranges within each bone. Overall, the results are not separated by more than 10dB.

## **Hearing Outcome of Laser Stapedotomy Minus Prosthesis (STAMP) Versus Conventional Laser Stapedotomy**

Herbert Silverstein, MD, Karen K. Hoffmann, MD  
Lance E. Jackson, MD, Jack H. Thompson, Jr., PhD, PA-C  
Joshua P. Sleeper, BA

**Objective:** To compare short and long-term hearing outcomes for patients undergoing primary laser stapedotomy minus prosthesis (STAMP) versus conventional laser stapedotomy.

**Study design:** Retrospective case review of 156 patients over a 9-year period between 1993-2002.

**Setting:** Otolaryngology/neurotology tertiary referral center.

**Patients:** Those with clinical otosclerosis without previous otologic surgery.

**Interventions:** Patients with otosclerosis confined to the fissula ante fenestram underwent STAMP. Patients with more extensive otosclerosis or anatomical contraindications to STAMP underwent standard laser stapedotomy.

**Main Outcome Measures:** Pure-tone audiometry was performed before surgery, post-operatively, and on follow-up examination.

**Results:** Of the 174 ears in 156 patients, 110 (63.2%) underwent laser stapedotomy, and 46 (36.8%) underwent STAMP. Of the 40 patients in the STAMP group with an average of 701 days (SD 439) follow-up, the air-bone gap closed from a mean of 21dB (SD 10 dB) to 5 dB (SD 6 dB). In comparison, 83 stapedotomy patients with an average of 717 days (SD 772) follow-up, the air-bone gap closed from a mean of 27 dB (SD 11 dB) to 7 dB (SD 8 dB). There was a statistically significant improvement in high-frequency hearing in the 2000-8000 Hz range in the STAMP patients, with an average difference of 22 dB compared to the stapedotomy group. Five patients required revision surgery after STAMP, but were successfully repaired with conventional stapedotomy.

**Conclusion:** Laser STAMP, when used for isolated anterior footplate otosclerosis, provides improved high frequency hearing compared to conventional laser stapedotomy, and has a low incidence of refixation necessitating revision surgery.

## **Partial Promontory Technique in Stapedotomy Cases with Narrow Niche**

Michelle M. Inserra, MD, Patricia J. Yoon, MD  
Theodore P. Mason, MD, Joseph B. Roberson, MD

**Objective:** Examine clinical and audiometric outcomes of laser partial promontory approach to stapedotomy cases with a narrow oval window niche.

**Study design:** Retrospective chart review.

**Setting:** Tertiary referral center.

**Patients:** Fifty-nine patients who underwent a partial promontory technique with stapedotomy between 1994 and 2000. Seventy-two patients who underwent primary stapedotomy without promontory technique served as a control group.

**Methods:** Preoperative and postoperative audiometric results were obtained for 59 patients undergoing laser stapedotomy with a narrow oval window niche. The partial promontory takedown was performed with a KTP laser. Results were compared with 72 primary laser stapedotomy cases without promontory technique within the same time period and analyzed using a paired student's t-test.

**Results:** Ninety percent of the partial promontory cases were successful ( $ABG \leq 10$  dB). The mean postoperative ABG was 5.1 dB which was comparable to the non-promontory cases ( $p=0.7$ ). The mean change in postoperative bone conduction was also comparable ( $p=0.98$ ). There were no cases of sensorineural hearing loss. An overhanging facial nerve was present in 32% of the narrow niche cases and a dehiscent facial nerve encountered in 17% of these cases.

**Conclusions:** Partial laser takedown of the promontory as an adjunct to laser stapedotomy cases with a narrow oval window niche is a safe, effective technique with comparable results to primary laser stapedotomy.

## **A New Approach for Malleus/Incus Fixation: No Prosthesis Necessary**

Michael D. Seidman, MD, Seilesh Babu, MD

**Objective:** To describe a novel approach to manage malleus/incus fixation.

**Study Design:** Retrospective review of 363 patients with conductive hearing loss operated on since 1992.

**Setting:** Academic tertiary referral center

**Patients:** 363 patients with conductive hearing loss, an intact tympanic membrane and without history for chronic infection underwent middle ear exploration. 341 had otosclerosis and underwent laser stapedotomy; the remaining 22 patients had laser release of their malleus/incus fixation.

**Intervention:** 22 patients were diagnosed with malleus fixation prior to surgery. Conductive hearing loss was identified using audiometry and tuning forks. The diagnosis was confirmed using micropneumotoscopy and noting immobility of the malleus. A transcanal approach was used and the malleus/incus fixation was released using a laser. A 1.5 to 2.0 mm space was created where the ossicular fusion existed, thereby reducing the likelihood of re-fusion.

**Main Outcome Measure:** Audiometric studies pre and post intervention were compared. 1-10 years of follow up are provided.

**Results:** Pre-operative air-bone gaps ranged from 25 dB to 60dB and averaged 45 dB. Post-operative air-bone gaps ranged from 0-25 and averaged 10 dB. No patients have experienced re-fusion. There were two complications: One perforation requiring a tympanoplasty and one patient sustained a 20 dB high frequency sensorineural loss

**Conclusion:** The idea that malleus/incus fixation should be repaired by removing the incus and using a POP is outdated. The approach presented provides an opportunity to leave the anatomy relatively undisturbed. The results are excellent and this approach should be considered in most cases with malleus/incus fixation. A video will be presented.

## **Trans Facial Recess Ossicular Chain Reconstruction: Surgical Technique and Early Results**

Nikolas H. Blevins, MD

**Objectives:** To present the technique of trans facial recess ossicular chain reconstruction (TFROCR) for use in selected patients with cholesteatoma.

**Study Design:** Retrospective review of all candidates for TFROCR between 8/98 and 10/02.

**Setting:** Tertiary referral center

**Patients:** At their first procedure, 19 patients (8 children, 11 adults) with cholesteatoma and ossicular discontinuity were identified as candidates for staged TFROCR. Seven patients had undergone previous tympanomastoid surgery.

**Intervention:** The first stage included canal wall up mastoidectomy with resection of disease, wide opening of the facial recess, cartilage graft tympanoplasty, and placement of silastic in the middle ear. Approximately 6 months later, patients underwent a second stage post-auricular procedure. Endoscopes were used to inspect the middle ear through the facial recess. When possible, TFROCR was then performed, without elevating a tympanomeatal flap.

**Main Outcome Measures:** Variations in anatomy, disease characteristics, and hearing results were studied.

**Results:** Of the 19 candidates, 15 successfully underwent TFROCR, and 4 required traditional second stage procedures with canal incisions. There were no surgical complications. Early hearing results are promising, with an average air-bone gap of less than 20 dB. There have been no early failures from recurrent disease or prosthesis displacement.

**Conclusions:** In carefully selected patients, TFROCR may be safe and effective for disease control and hearing restoration. It may provide for optimal prosthesis placement and almost immediate hearing improvement, avoiding the need for canal incisions, middle ear packing, and dry ear precautions. One must consider the potential risk of missing residual disease secondary to limited exposure.

## Investigations of the Effect of Superior Semicircular Canal Dehiscence on Hearing Mechanisms

John J. Rosowski, PhD, Jocelyn E. Songer, MS  
 Heidi H. Nakajima, MD, PhD, Kelly M. Brinsko, BS  
 Saumil N. Merchant, MD

**Hypothesis:** Superior Semicircular Canal Dehiscence (SSCD) affects hearing function by introducing a third window into the inner ear which: (a) lowers cochlear input impedance, (b) shunts sound away from the cochlea, and (c) improves bone conduction thresholds by increasing the difference in impedance between the vestibule and round-window membrane.

**Background:** Besides affecting the vestibular system, SSCD has also been linked to a “conductive” hearing loss characterized by a decrease in the sensitivity to air-conducted sound and hyper-sensitivity to bone-conducted sound.

**Methods:** Laser-Doppler vibrometer measurements of sound-induced umbo velocity were performed in patients with CT confirmed SSCD. The effect of SSCD on bone and air-conducted sounds were studied in chinchillas. Anatomically-based theoretical analyses of sound flow through the cochlea and semi-circular canals were performed.

**Results:** (1) The umbo-velocity in five SSCD patients with no other complications ranged from normal through hyper-mobile. Such hypermobility is consistent with a decrease in cochlear impedance produced by a shunt path that allows sound to flow away from the cochlea. (2) Measurements in eight chinchilla demonstrated that creating an SSCD can lead to increases in the cochlear potentials produced by bone-conduction stimuli. This increase mimics the hyper-sensitivity to bone-conducted sound observed in patients with SSCD. (3) An anatomically based model predicts changes in auditory sensitivity that have features in common with the clinical and experimental measurements of hearing function in SSCD.

**Conclusions:** The results are consistent with the hypothesis that SSCD introduces a third window into the inner ear.

IRB Approval number for Human studies: 00-09-041

IRB Approval number for Animal studies: 91-11-027

Work supported by NIDCD R01 DC 04798 and R01 DC 00194

## Superior Semicircular Canal Dehiscence Presenting as "Conductive" Hearing Loss Without Vertigo

Anthony A. Mikulec, MD, Mitchell J. Ramsey, MD  
 Michael J. McKenna, MD, Joseph B. Nadol, Jr., MD  
 Steven D. Rauch, MD, John J. Rosowski, PhD  
 Hugh D. Curtin, MD, Saumil N. Merchant, MD

**Objective:** To describe superior semicircular canal dehiscence (SSCD) presenting as otherwise unexplained "conductive" hearing loss without vestibular symptoms.

**Study Design:** Retrospective.

**Setting:** Tertiary referral center.

**Patients:** Seven patients (9 ears); ages 29-61 yr;  
 M:F = 3:4.

**Diagnostic Tests and Results:** All 9 ears had SSCD on high resolution temporal bone CT scan. There were no middle ear findings to explain the air-bone gap in these 9 ears (including negative middle ear exploration in 6 ears; of these 6, stapedectomy had been performed in 3 ears but the air-bone gap was unchanged post-operatively). Air-bone gaps were largest at 250, 500 and 1000 Hz: the averaged gap for these 3 frequencies for the 9 ears ranged from 32-52 dB. Bone conduction thresholds below 2000 Hz were negative (-5 to -15 dB) at one or more frequencies in all 9 ears. Laser vibrometry showed umbo motion to be above mean normal in all 8 ears tested. Vestibular evoked myogenic potentials (VEMP) were present in all 3 tested ears. The audiometric and laser vibrometry data are consistent with SSCD producing an apparent conductive loss by shunting air-conducted sound away from the cochlea and improving thresholds for bone-conducted sounds by increasing the difference in impedance between the oval- and round- windows.

**Conclusions:** SSCD can present with a "conductive" hearing loss that mimics otosclerosis, and may explain some cases of persistent conductive hearing loss after uneventful stapedectomy. Audiometric testing with attention to absolute bone conduction thresholds, laser vibrometry of the umbo, VEMP testing and CT scanning can help to identify patients with SSCD presenting with apparent conductive hearing loss without vertigo.

## Long Term Results of Mastoid Obliteration with Bone Cements

Jennifer L. Maw, MD

**Objective:** To report the long-term outcome of mastoid obliteration using bone cements.

**Study design:** Case series

**Setting:** Private otologic practice

**Patients:** Thirteen patients aged 17 to 51 with a history of a mastoid cavity and chronic mastoid problems who wanted to undergo a mastoid obliteration procedure

**Intervention:** Revision tympanomastoidectomy and mastoid obliteration or canal wall reconstruction with a commercially available bone cement.

**Main Outcome Measures:** Complete healing of ear, dry ear and time to required revision surgery.

**Results:** Eight patients underwent mastoid obliteration with dahllite bone cement (Norian CRS cement (Synthes)) and three patients with hydroxyapatite cement (Bone Source (Leibinger)). Palva flaps were performed in 2 of each procedures. Two patients underwent canal wall reconstruction, one with each cement. Initial results looked promising with near or complete healing and dry ears at 6 months in 9 patients. Eleven of the 13 procedures eventually failed over 2 years with occurrence of granulation tissue formation and/or cement exposure in the ear canal and otorrhea. One patient fistulized through the post-auricular incision. Revision surgery was required in 10 patients (mean time from obliteration surgery was 20 months) and all underwent successful removal of the bone cement and mastoid obliteration with bone pate. There were no cases of sensorineural hearing loss. One patient had extensive middle ear fibrosis and obliteration of the round window with a maximum conductive hearing loss. Granulation tissue showing chronic inflammation and foreign body response was present between the temporal bone and cement except for the bony labyrinth.

**Conclusions:** While early results look promising, mastoid obliteration with the currently available dahllite and hydroxyapatite bone cements have an unacceptable long-term complication rate. Canal wall reconstruction with use of a Palva flap requires further consideration and study.

**Central Auditory System Development and Plasticity  
in Children with Cochlear Implants  
Who are Implanted Later in Childhood**

Anu Sharma, PhD, M. F. Dorman, PhD,  
N. Wendell Todd, MD, Jolie Fainberg, MS, Phillip Gilley, MS  
Kathryn Martin, MS, Anthony Spahr, MS

**Hypothesis:** We examined the effect of pre-implantation hearing thresholds on central auditory development in late-implanted children. We compared P1 cortical response latencies of: 1) congenitally profoundly deaf children who had very poor aided performance prior to implantation and, 2) children who had better aided hearing thresholds and speech perception scores prior to implantation.

**Background:** We have recorded the P1 response in a large number of congenitally-deaf children fit with cochlear implants. Children with 7 or more years of auditory deprivation before implantation had abnormal cortical response latencies. Children with 3.5 years or less of deprivation evidenced age-appropriate latencies. In children implanted after age 7, abnormal P1 latencies were observed even after years of implant use, suggesting that too long a period of deprivation results in reduced plasticity in auditory thalamo-cortical pathways.

**Results:** Preliminary results show that children with significant aided benefit prior to implantation evidenced age-appropriate cortical response latencies after implantation.

**Conclusion:** A small amount of auditory stimulation preserves auditory plasticity in profoundly deaf children. At issue is how little stimulation is sufficient to keep pathways plastic. The answer has a direct bearing on the issue of when is it too late to implant a child and expect a good result.

## **Language and Speech Development in Deaf Children and Infants Following Cochlear Implantation**

Richard T. Miyamoto, MD, Derek M. Houston, PhD  
Karen Iler Kirk, PhD, Amy E. Perdew, MS  
Mario A. Svirsky, PhD

**Objective:** The purpose of this study was to evaluate the speech and language benefits of cochlear implantation in infancy and compare these observations to those obtained in slightly older children. A re-evaluation of the lower age limits appropriate for cochlear implantation has been mandated by the increased number of newly identified deaf infants found through newborn hearing screening programs.

**Study design:** Longitudinal, prospective, repeated measures.

**Setting:** Tertiary referral center

**Patients:** Performance of our youngest patient who received a cochlear implant at age 6 months is compared to a group of implanted children who received implants at 4-5 years, 3-4 years and less than 3 years.

**Interventions:** Implanted infants and children were assessed using standard language measurements including the Reynell Developmental Language Scales and The GAEL-P. Assessing infants requires the development of new measures. We have modified the Visual Habituation Procedure to document early skills. This procedure has been used extensively to assess normal hearing infants ability to discriminate speech contrasts but has not previously been applied to implanted infants.

**Results:** By age 2 years the youngest implanted infant achieved age equivalent scores on the Reynell Development Language Scales and scores on the GAEL-P which were nearly equivalent to scores achieved at age 5.5 years by children implanted at later ages. Speech pattern discrimination was demonstrated by the Visual Habituation Procedure.

**Conclusion:** Enhanced speech and language growth in an infant vs. children implanted at older ages is demonstrated. A clear rationale for earlier implantation is in evidence.

Supported by: NIH-NIDCD RO1 DC00064, RO1 DC00423, and K23 DC00126.

## **Optimizing Cochlear Implant Efficiency with Modiolus-Based Return Electrode**

Steven Y. Ho, MD, Richard J. Wiet, MD  
Claus-Peter Richter, MD

**Hypothesis:** By placing the return electrode in the modiolus, the current generated by a cochlear implant will be directed into that area and, therefore, improve its operating efficiency.

**Background:** Ideal cochlear implant should maximize current flow into the modiolus in order to stimulate the cochlear nerve. The latest cochlear implants attempt to accomplish this by several techniques, such as the pre-curved electrodes designed to “hug” the modiolus and silastic positioners designed to appose the electrodes against the modiolus. This study is designed to explore the effects of return electrode placement on current and potential field distributions.

**Methods:** The effects of return electrode position on potential field distributions are studied in two different models designed to simulate human cochlea. Actual measurements are then taken in the modiolus of human temporal bone implanted with Clarion HiFocus implant. Return electrode is placed either within the modiolus, or remotely, outside of the temporal bone, simulating current cochlear implant configuration.

**Results:** Cochlear models’ results clearly show that voltage and current distribution are greatly influenced by the location of return electrode. Temporal bone data reflect similar findings. Voltages recorded in the modiolus are 3-5 times higher with return electrode in the modiolus than outside of the temporal bone.

**Conclusion:** Modiolus-based return electrodes significantly reduce the power requirements by a factor of three to five in a cochlear implant. The power reduction should lead to improved efficiency, safer long-term use, and longer device life.

## **Meningitis in Cochlear Implant Recipients: The North American Experience**

Noel L. Cohen, MD  
J. Thomas Roland, Jr., MD

**Introduction:** Until recently, post-implant meningitis was infrequently reported and felt to be uncommon. However, in the spring of 2002 there appeared to be a sudden increase in occurrence of post-implantation meningitis in both Europe and North America.

**Objective:** Since complications of surgery often tend to be under-reported, we decided to survey all cochlear implant centers in North America to determine the true incidence of post-implant meningitis and to learn more about the demographics and risk factors.

**Study Design:** This prospective study asked surgeons the number of implants performed and whether they had any meningitis following implantation. If the answer was affirmative, they were asked to respond to a 20-point questionnaire. This instrument was sent with the help of the manufacturers to all 401 cochlear implant centers in North America.

**Setting:** Tertiary care referral centers

**Patients:** All patients having received cochlear implants in North America.

**Interventions:** None

**Main Outcome Measures:** Number of cases of post-implant meningitis, age of patients, device used, cochlear and temporal bone abnormalities, treatment and outcomes

**Results:** Meningitis is more common than previously thought. Risk factors include: young age, cochlear and temporal bone abnormalities, and the use of a two-part electrode system. This survey led to the involvement by the FDA and CDC in a much more intensive analysis of a group of cases.

**Conclusions:** Post-implant meningitis is related to patient, surgical and device factors. By improving surgical technique, vaccinating high-risk cases, and eliminating potentially traumatic devices the incidence of meningitis should be much diminished.

## **Neonatal Middle Ear Effusion and Chronic Otitis Media with Effusion**

**Karen Jo Doyle, MD, PhD, Ying Yee Kong, MA  
Patricia Dallaire, MA, Karen Strobel, MA  
Mark Ray, MD**

Many "failures" on newborn hearing screening tests are subsequently attributed to middle ear fluid (effusion) in the newborn period. It is not known whether effusion in the newborn period is a risk factor for the development of chronic otitis media with effusion (COME). The goal of this study is to determine whether newborn middle ear effusion present at age 30 to 48 hours is related to later diagnosis of COME.

Fourteen experimental infants with effusion in at least one ear and 14 control infants with no effusions were recruited and followed with examinations at 3, 6, 9, and 12 months. When otoscopic examination revealed effusion, the infant was re-examined the following month. At each visit, the infant underwent pneumatic otoscopy, multifrequency tympanometry, transient evoked otoacoustic emissions (TEOAE), and visual reinforcement audiometry (starting at age six months).

Our data from 28 infants indicate that infants with middle ear effusions in this newborn period are more likely to develop COME in the first year of life. Seven of 14 experimental (50%) and 3 of 14 control (21%) infants developed COME during the first year of life, as defined by three consecutive months of middle ear effusion. The results of audiologic testing will be discussed. The data obtained from this study may be of great value to clinicians who must make referral and treatment decisions based on results of hearing screening tests

## **Hearing Preservation Rates During Vestibular Schwannoma Resection: Retrosigmoid Approach and Direct Cochlear Nerve Monitoring**

Christopher J. Danner, MD, Roberto A. Cueva, MD

**Objectives:** To discuss the effectiveness and ease of direct eighth nerve monitoring and the advantages it offers over ABR when attempting to preserve hearing during vestibular schwannoma resection.

**Study Design:** Prospective study

**Setting:** Tertiary referral center

**Methods:** Six year prospective study of the use of direct eighth nerve monitoring during vestibular schwannoma removal. Tumors were removed via a retrosigmoid craniotomy.

**Results:** Hearing preservation was attempted in over 80 patients with vestibular schwannomas. Tumor sizes ranged from 0.5cm to 2cm. Hearing was preserved in over 80% of patients with tumors less than 1cm and in 50% of patients with tumors between 1 - 2cm when the direct eighth nerve monitoring was used. When ABR was used hearing results were over 40% for both groups. Facial nerve preservation rates were over 90% (HB 1 - 2) for tumors less than 1.5cm.

**Conclusions:** A remarkable increase in the hearing preservation rate can be achieved with the use of direct eighth nerve monitoring over ABR. The retrosigmoid approach is a viable option when considering hearing preservation during vestibular schwannoma resection.

## Comparison of Vestibular Nerve Afferent and Eye Movement Responses to Galvanic and Rotational Stimuli

Charley C. Della Santina, PhD, MD, Timothy E. Hullar, MD  
John P. Carey, MD, Americo A. Migliaccio, PhD  
Lloyd B. Minor, MD

**Hypothesis:** Electrical stimulation of semicircular canal cristae elicits vestibular nerve activity and eye movements with characteristics spanning the range of normal responses to head rotation.

**Methods:** We measured chinchilla vestibular nerve afferent activity (using glass micropipettes) and eye movements (using magnetic scleral search coils) during galvanic stimulation of semicircular canal cristae and during controlled head rotation. Galvanic stimuli were delivered using metal electrodes implanted through a superior canal fenestration, with the return electrode in the round window niche. Galvanic stimuli were 0-90  $\mu$ A pk-pk about a baseline of -20 to 40  $\mu$ A, and included sinusoids from 0.5-100 Hz and band-limited white noise. Rotational stimuli were acceleration steps or 0.5-18 Hz sinusoids, 20-150 deg/sec in the plane of the canal innervated by the afferent fiber.

**Results:** DC current stimuli modulated vestibular nerve responses from 0-130 action potentials (spikes)/sec, with higher currents evoking responses with increased mean rate and decreased variability. Sinusoidal stimuli elicited a single spike at the instant the fenestration electrode became cathodic above a threshold, plus additional spikes at a nearly fixed interspike interval during the cathodic phase. Current stimuli also elicited eye movements in the plane of the stimulated canal. The range of mean afferent spike rates and eye movements evoked by electrical stimulation encompassed the range observed for head rotation stimuli.

**Conclusions:** Electrical stimulation of semicircular canal cristae can evoke vestibular nerve activity and eye movements similar to those evoked by natural head rotation, although the stochastic properties of electrically-evoked nerve activity and natural responses may differ.

Supported by a Clinician Scientist Career Development Award from the American Otological Society and by NIH DC002390-08.

## **Benign Paroxysmal Positional Nystagmus in Patients Receiving Ototoxic Medications**

F. O. Black, MD

S. C. Pesznecker, RN, Valerie Stallings

**Objective:** To investigate the occurrence of benign paroxysmal positional nystagmus (BPPN) in subjects undergoing treatment with ototoxic medications.

**Study design:** Retrospective record review.

**Setting:** Tertiary referral neurotology clinic; clinical research and technology center

**Subjects:** One hundred eighteen subjects undergoing in-hospital treatment of infectious disease or carcinoma with ototoxic medications.

**Intervention(s):** (1) records review, (2) tests of vestibular function.

**Main outcome measure(s):** (1) results of Cawthorne-Hallpike positional tests for BPPN (electrooculography or videonystagmography).

**Results:** Fifty-six of 118 subjects were female, 62 were male. Age range was 10-81 years, with mean age of 48 years. All subjects underwent Cawthorne-Hallpike testing; 56 of 118 subjects (47%) had an unequivocally positive Cawthorne-Hallpike test for BPPN in one or both ears. The occurrence of BPPN in the Hallpike-positive population was distributed equally across the age decades.

**Conclusion:** The high occurrence rate of BPPN in subjects receiving potentially ototoxic medications is compatible with the observation that BPPN occurs in combination with other pathological conditions such as Meniere's syndrome and vestibular neuritis. Occurrence of BPPN was independent of increasing age. The presence of BPPN may complicate the clinical identification of ototoxicity or obfuscate management.

Supported in part by NIH grants RO1 NS 19221, RO1 DC00205 and NASA grant NAG5-6329.

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

### CONFLICT OF INTEREST DISCLOSURE FORM

I, as senior author, am confirming that I/we have no real or apparent conflict of interest related to my/our participation in the American Otological Society's Annual Spring Meeting to be held May 3-4, 2003. In this regard, please be advised that I am disclosing below any publication, public positions, or memberships, as well as any personal financial interests (including equity positions, consulting agreements or employment arrangements) related to the proposed conference topic.

\_\_\_ I have no financial interests or advocacy positions related to the issues under discussion.

\_\_\_ My relevant financial interests are:

\_\_\_ My relevant publications, public positions, or memberships are:

### 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. 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): \_\_\_\_\_

# **AMERICAN OTOLOGICAL SOCIETY, INC.**

## **MISSION STATEMENT**

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

- to advance and promote medical and surgical otology including the rehabilitation of the hearing and balance impaired.
- to encourage, promote, and sponsor research in otology 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 dialog 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 and neurotology.

After attending this meeting, the participant will have a

- better understanding of sound transmission in the middle ear and operations to improve conductive hearing loss.
- better understanding of the physiology and biochemical prevention of noise-induced hearing loss.
- better understanding of transtympanic treatment of Meniere's Disease and autoimmune inner ear disease.
- better understanding of cochlear implants and prevention of meningitis.

## NAMES AND ADDRESS OF PRIMARY AUTHORS

---

Manohar Bance, MB, MSc, FRCSC  
Rm 3184, Dickson Bldg,  
VGH Site, QEII HSC  
1278 Tower Rd.  
Halifax, NS, CANADA  
B3H 2Y9

F. O. Black, MD  
Legacy Holladay Park  
Clinical Res & Tech Ctr  
Dept of Neurotology Research  
1225 NE 2nd Ave, Ste 303  
PO Box 3950  
Portland, OR 97208-3950

Nikolas H. Blevins, MD  
750 Washington St.  
NEMC #850  
Boston, MA 02111

Stanley H. Chia, MD  
3412 Herman Ave, Unit A  
San Diego, CA 92104

Noel L. Cohen, MD  
Dept. of Oto-NYU Med Ctr  
530 First Avenue  
New York, NY 10016

Christopher J. Danner, MD  
200 Arbor Drive - 8895  
San Diego, CA 92103

Charley C. Della Santina, PhD, MD  
Johns Hopkins Outpatient Center  
601 North Caroline St, Rm 6260B  
Baltimore, MD 21287

Joni K. Doherty, MD, PhD  
632 N. Topanga Canyon Blvd  
Topanga, CA 90290

Karen Jo Doyle, MD, PhD  
6392 Harmon Drive  
Sacramento, CA 95831

Mark S. Driver, MD  
Dept. of Oto-HNS  
Boston Uni School of Med  
D616, 88 E. Newton St.  
Boston, MA 02118

Jeffrey P. Harris, MD, PhD  
Univ of CA-San Diego  
Div of Oto-HNS  
200 W. Arbor Dr. #8895  
San Diego, CA 92103-8895

Steven Y. Ho, MD  
1000 Central St., Ste 610  
Evanston, IL 60201

Michelle M. Inserra, MD  
Stanford Univ Med Ctr  
Div of Oto-HNS  
Rm 135, Edwards Bldg  
Stanford, CA 94305-5328

Manoj Kumar, MS, FRCS  
223 Martin Luther King Dr, Apt. 4  
Cincinnati, OH 45219

Colm Madden, MB, FRCSI  
Ctr of Hearing & Deafness Research  
Dept of Pediatric Otolaryngology  
Childrens Hospital Med Ctr.  
3333 Burnet Ave  
Cincinnati, OH 45229

Douglas E. Mattox, MD  
Dept of Oto-HNS  
Emory Clinic, Rm A 2328  
1365 Clifton Rd, NE  
Atlanta, GA 30322

Jennifer L. Maw, MD  
2030 Forest Ave Ste 210  
San Jose, CA 95128

Saumil N. Merchant, MD  
Dept. of Otolaryngology  
Massachusetts Eye and Ear Infirmary  
243 Charles St.  
Boston, MA 02114-3096

Richard T. Miyamoto, MD  
Riley Hospital, Ste 0860  
702 Barnhill Dr.  
Indianapolis, IN 46202

John J. Rosowski, PhD  
Eaton-Peabody Lab  
Massachusetts Eye and Ear Infirmary  
243 Charles St.  
Boston, MA 02114

Michael D. Seidman, MD  
Henry Ford Health System  
Dept. of Otolaryngology-HNS  
6777 W. Maple Rd  
W. Bloomfield, MI 48323

Anu Sharma, PhD  
University of Texas at Dallas  
Callier Center for Communication  
Disorders  
1966 Inwood Road  
Dallas, TX 75235

Herbert Silverstein, MD  
1961 Floyd St., Ste, A  
Sarasota, FL 34239

Thomas R. Van De Water, PhD  
Univ of Miami Ear Institute  
RMSB 3160  
1600 NW 10th Ave  
Miami, FL 33136

# **AOS Research Grantee Progress Report**

## **Electrical Stimulation to Restore Vestibular Function AOS Clinician-Scientist Career Development Award**

Charley C. Della Santina, PhD, MD  
Division of Otolaryngology, Neurotology and Skull Base Surgery  
Department of Otolaryngology – Head & Neck Surgery  
Johns Hopkins School of Medicine

Progress July-December 2002

### Synopsis of Project

The goal of this project is to lay a biologic foundation for development of a prosthesis to restore vestibulo-ocular reflex (VOR) function in patients disabled by bilateral vestibular deficiency due to Ménière's disease, ototoxic drugs, or other insults to the labyrinth.

Specific aims include:

- 1) Establish a chinchilla model of peripheral vestibulopathy using intratympanic gentamicin, and determine whether viable vestibular nerve afferent fibers remain in treated cristae after hair cell destruction.
- 2) Characterize the effect of semicircular canal electrode placement and stimulation on the cochlea and other endorgans.
- 3) Measure the eye movements elicited by electrical stimuli applied via electrodes near canal cristae, and assess their dynamics, range and conjugacy. Evaluate stimulation strategies for expanding the range of eye movements driven by purely unilateral canal stimulation.
- 4) Evaluate the extent to which electrical crista stimulation encoding actual head rotation can stabilize gaze in bilaterally vestibular-deficient chinchillas.

### Summary of Progress During This Period

The main focus of work in the initial 6 months of this 3-year project has been on refining experimental techniques and establishing normative data against which results for treated animals will be compared. Significant progress has been made in this regard for each of the specific aims.

#### **Aim 1)**

After an initial 4-month period of training and adapting techniques on 8 normal control chinchillas, extracellular single-unit recording from vestibular afferents during rotational and galvanic stimulation is now working reliably. We are typically able to record from 20-30 single vestibular afferents in each nerve, holding some for >120 minutes and most for >5 minutes (adequate for spontaneous rate measurement and identification of target endorgan). Spontaneous firing rates have been similar to those noted in prior studies, as has been the distribution of fiber types based on discharge regularity and endorgan. We

## (PI: Della Santina—Continued)

are now ready to begin side-to-side comparisons in a cohort of 8 chinchillas treated unilaterally with intratympanic gentamicin. Those animals will also provide material for side-to-side histological comparison.

We also measured chinchilla vestibular nerve single-unit afferent activity during galvanic stimulation of the superior semicircular canal crista and during controlled head rotation. (We chose the superior canal for ease of surgical access in the chinchilla.) Galvanic stimuli (0-90  $\mu$ A pk-pk about a baseline of -20 to 40  $\mu$ A) were delivered using metal wire electrodes implanted through a superior canal fenestration, with the return electrode in the round window niche. Rotational stimuli were acceleration steps or 0.5-18 Hz sinusoids, 20-150 deg/sec in the plane of the canal innervated by the afferent fiber. DC currents modulated afferent responses from 0 to 130 action potentials/sec, with higher currents evoking responses with increased mean rate and decreased variability. Sinusoidal stimuli reliably elicited a single spike at the instant the fenestration electrode became cathodic above a threshold, plus additional spikes at a nearly fixed inter-spike interval during the cathodic phase. The range of mean afferent spike rates induced by electrical stimulation encompassed the range observed for head rotation stimuli. Electrical stimuli also elicited eye movements that appeared to align in the plane of the stimulated superior canal (see below).

### Aim 2)

We have used two approaches to discern the effect of semicircular canal electrode implantation on function of the normal cochlea and labyrinth. First, we have recorded from vestibular afferents before and after implantation and galvanic stimulation of the superior semicircular canal in otherwise normal chinchillas. In 4 of 5 acutely chinchillas implanted with perilymphatic superior canal electrodes thus far, we have recorded single unit afferents with responses (rate and preferred stimulus/orientation) typical of normal fibers innervating the horizontal, anterior and superior canals and utricle. Thus, it is possible to place and galvanically drive electrodes without ablating function of other endorgans in the labyrinth. One animal developed signs of unilateral vestibular hypofunction 36 hours after attempted implantation of electrodes very close to the superior semicircular canal crista. The electrode may have perforated the membranous labyrinth, whereas others likely remained in the perilymphatic space.

Evoked auditory brainstem response measurement with sound field stimulation (using a system designed by Bradford May, Johns Hopkins Otolaryngology - Head & Neck Surgery) is now working well. ABR's have been recorded in 6 control chinchilla ears, yielding thresholds of 29 +/- 13 dB SPL. Occluding an ear with a foam plug raises ipsilateral thresholds by 45 +/- 16 dB SPL. When the test ear is plugged but the non-test ear is not (simulating testing of an ear deafened by

unilateral ototoxic treatment), crosstalk attenuation is sufficient to prevent recording from the contralateral non-test ear. Thus, although statistically rigorous estimates must await further normative data, these initial studies suggest we should be able to detect a unilateral ABR threshold change of 26 dB SPL even with a normal contralateral ear. We are now ready to perform pre- and post-operative ABR testing of animals implanted with semicircular canal electrodes.

**Aims 3 and 4)**

In order to make maximal use of the opportunity to collaborate closely with Americo Migliaccio, PhD (a visiting post-doctoral bioengineer with significant experience in 3-D scleral coil recording) we have shifted significant effort towards eye movement recording techniques to address Aims 3 and 4. Initial eye experiments using glued-on scleral coils and IM anesthesia in control animals were disappointing. Glued-on coils tended to shift position and restrict eye rotation due to impingement on retracted eyelids. Intramuscular anesthetic effects lingered for many hours, with vestibular-ocular reflex gains rising slowly with level of consciousness. We have now converted to surgically implanted scleral coils and inhalant anesthetics, with excellent results. In 2 animals tested so far using these techniques, 1-dimensional vestibulo-ocular reflex gains have been close to the expected value (1.0 in light for 0.5 Hz horizontal rotations).

Importantly, we are successfully recording eye movements in 3 dimensions(which requires two coils implanted on each eye). This will enable us to directly test our hypotheses regarding the direction and conjugacy of electrically driven eye movements.

Presentations and Publications

Submissions acknowledging American Otological Society support during this initial 6 months include:

- 1) J.T. Rubinstein and C.C. Della Santina, "Development of a biophysical model for vestibular prosthesis research," submitted to Journal of Vestibular Research Dec 2002
- 2) C.C. Della Santina, T.E. Hullar, J.P. Carey, A.A. Migliaccio, L.B. Minor, "Comparison of Vestibular Nerve Afferent and Eye Movement Responses to Galvanic and Rotational Stimuli," accepted for presentation at the Annual COSM/AOS Meeting May 2003.

# AOS Research Grantee Progress Report

## Growth Factor Signaling of Otic Capsule Chondrogenesis: Implications for Otosclerosis

Dorothy A. Frenz, PhD  
Albert Einstein College of Medicine

Progress Report – October 2002 – January 2003

**Summary of research study:** Bone morphogenetic proteins (BMP) and transforming growth factor beta (TGF $\beta$ ) regulate chondrification of the otic capsule during embryonic development. These signaling molecules (i.e. BMP, TGF $\beta$ ) are also present at sites of new bone formation in specimens of the stapedial footplate from patients with active otosclerosis. This observation suggests that BMP and TGF $\beta$  play a role not only in normal otic capsule formation, but also in the abnormal chondrification and bone remodeling of otosclerosis. Smad6 and Smad7 are antagonists of BMP/TGF $\beta$  signaling. During the current funding period, we have investigated the functional role of Smad6 and Smad7 in the developing otic capsule, and have tested the ability of manipulation of levels of expression of Smad6 or Smad7 to modify otic capsule chondrogenesis as a putative means to control chondrogenesis and prevent overgrowth of the otic capsule. A summary of our progress is provided below.

**Smad6 is expressed in the developing mouse inner ear.** We examined the pattern of expression of Smad6 in the developing mouse inner ear between ages 10.5 and 14 days of embryonic development (E10.5, E14). Although Smad6 was present in the mesenchyme of the E10.5 and E12 inner ear, expression was most intense at E14 days, when Smad6 protein was localized to the chondrifying otic capsule and to the mesenchyme of the forming perilymphatic spaces. The presence of Smad6 in the cartilaginous otic capsule and perilymphatic space mesenchyme at this stage of embryonic development is consistent with a function for Smad6 in the turning off of chondrogenesis to prevent overgrowth of the otic capsule and promote the capsular remodeling associated with normal inner ear development.

**Expression of Smad7 in the developing mouse inner ear.** An antibody directed against Smad7 was used to define the pattern of expression of Smad7 in the developing mouse inner ear. At E10.5 days, Smad7 localized to the loosely organized periotic mesenchyme surrounding the lateral aspect of the developing otocyst. In the E12 inner ear, Smad7 was present in the now condensing periotic mesenchyme that is dorsal and lateral to the developing horizontal semicircular duct. Expression of Smad7 at E14 days is currently under investigation.

**Stimulation of chondrogenesis by blocking of Smad6 or Smad7.** An antisense oligonucleotide approach was used to define a function for Smad6 and Smad7 in otic capsule chondrogenesis. High density cultures of E10.5 mouse periotic mesenchyme containing otic epithelium (periotic mesenchyme + otic epithelium) were treated with Smad6 or Smad7 specific antisense oligonucleotide (60 or 90  $\mu\text{g/ml}$ ) for a period of 7 days *in vitro*. On day 7, the cultures were fixed then stained with Alcian blue 8GX at pH 1.0, a stain which at this pH binds specifically to sulfated glycosaminoglycans in the matrix of chondrifying cells. In cultures treated with Smad6 or Smad7 specific antisense oligonucleotide (60 or 90  $\mu\text{g/ml}$ ), a significant enhancement of chondrogenesis, as indicated by a significant increase in mean values for binding of Alcian blue stain (pH 1.0), occurred in comparison to untreated or sense oligonucleotide-treated (60 or 90  $\mu\text{g/ml}$ ) control cultures. This finding supports a role for Smad6 and Smad7 as negative regulators of otic capsule chondrogenesis in the developing mouse inner ear. The significance of this study is that it demonstrates the ability of antisense oligonucleotides to modulate the chondrogenic response of cultured periotic mesenchyme by blocking an endogenous signaling molecule.

**Redundant function of Smad2 and Smad3 in the inner ear.**

We previously reported that the phenotype of the inner ear in normal (CBA C57/BL6; wild-type) mice and Smad3 null mutant mice is similar, and suggested that the absence of an effect of targeted mutation of the Smad3 gene on the inner ear may be due to functional redundancy between Smad3 and related Smad2. Since Smad2 null homozygote mice are embryonic lethal and the phenotype of the inner ear cannot be examined in the mutant embryos, we began to test for redundancy using an antisense oligonucleotide approach in high density cultures. Smad2 or Smad3 specific antisense oligonucleotide was added, either alone or in combination, to cultured E10.5 periotic mesenchyme containing otic epithelium. In cultures treated either with Smad2 or Smad3 antisense oligonucleotide, the extent of chondrogenesis, as measured by binding of Alcian blue stain, pH 1.0, was comparable to their respective sense oligonucleotide treated or untreated control cultures. However, when cultures were treated with a combination of Smad2 and Smad3 antisense oligonucleotides, a significant decrease in chondrogenesis occurred in comparison to their single-treated antisense oligonucleotide counterparts. Addition of a combination of Smad2 and Smad3 sense oligonucleotides had no effect on the chondrogenic differentiation of the cultured periotic mesenchyme. These findings support functional redundancy between Smad2 and Smad3 in the developing capsule of the inner ear. A manuscript reporting these results is in preparation.

**(PI: Frenz—Continued)**

**Specimens of human otosclerotic bone.** We have been accumulating and processing surgical specimens consisting of parts of the stapedial footplate from patients with otosclerosis. Over the course of the next few weeks, these specimens will be immunostained for Smad2,-3, -5, and -6.

# AOS Research Grantee Progress Report

## Role of Noggin in Ear Development

Margaret I. Lomax, PhD

University of Michigan

Department of Otolaryngology-Head & Neck Surgery

Progress Report

Several autosomal dominant human syndromes are known to cause conductive hearing loss due to stapes ankylosis, the fusion of the middle-ear ossicle to the oval window of the cochlea. These syndromes include proximal symphalangism (SYM1) and multiple synostosis (SYNS1) syndromes, whose principal features are multiple joint fusions and conductive hearing loss caused by stapes ankylosis (Gong et al, 1999). Dominant missense mutations in the human *NOGGIN* gene (*NOG*) have been identified in several unrelated families with either SYM1 or SYNS1. Noggin is one of the most powerful antagonists of bone morphogenetic proteins (BMPs) and decreased noggin levels would be expected to affect development of joints, the skeleton, and the bony structures of the ear. Recently, Dr. Lesperance and her colleagues identified a family (Family 16) with conductive hearing loss that was originally attributed to otosclerosis (Brown et al., 2002). The clinical features of affected family members indicated that the conductive hearing loss was only one feature of a syndrome that included congenital stapes ankylosis, broad thumbs and toes, hyperopia and skeletal anomalies (SABTH), but did not involve symphalangism. They identified a chain termination (nonsense) mutation in the *NOG* gene in Family 16 and an insertion mutation that leads to premature chain termination in another family (Family G) with this same syndrome. Thus human *NOG* mutations can cause at least three different syndromes involving skeletal abnormalities, underscoring the role of noggin in development of joints, the skeleton, and the bony structures of the ear. The homozygous null *Nog*<sup>-/-</sup> mouse mutant also has skeletal defects and dies at birth because of problems with development of the brain, whereas the *Nog*<sup>+/-</sup> heterozygote is apparently normal with no obvious neurological, skeletal or otologic phenotype (Brunet et al., 1998). To understand how expression of *NOG* mutants affects BMP signaling and subsequent development of bony structures, including the stapes and otic capsule, we are beginning to examine expression of the mouse *Nog* gene during normal ear development, along with other genes known to be expressed in the otic capsule during development, such as *Brn4*. These studies have not yet been initiated. We also want to understand the structure-function relationships between the mutations in the human *NOG* gene and the effects of these chain terminating mutations on Noggin protein. These experiments involve the expression of the mutant *NOG* sequence in cultured cells. The mutant *NOG* genes identified

**(PI: Lomax—Continued)**

by Dr. Lesperance have been subcloned into the eukaryotic expression vector pcDNA3.1-V5-His. The resulting plasmids have been introduced into COS7 cells by transfection. These expression constructs should produce truncated Noggin proteins that are epitope-tagged, i.e., that have short sequences at the C-terminus that can be detected by antibodies to either V5 or His on Western blots. These Western blot experiments to analyze the expression of mutant NOG are currently in progress. These experiments on the structure-function relationship of *NOG* mutations should provide insight into the role of Noggin in the correct development of the bony structures of the ear, the ossicles and the otic capsules. We anticipate that these results will eventually provide information that will enable otologists to distinguish between otosclerosis and these syndromes when dealing with patients with conductive hearing loss.

# AOS Research Grantee Progress Report

## Kinetics of Round Window Permeability

2002-2003 American Otologic Society Research Grant

Progress Report: July 1, 2002 – January 2003

Principal Investigator: Douglas E. Mattox, MD

**Abstract of proposed research:** The round window membrane is permeable to a wide variety of substances of varying molecular size and charge. Based on this permeability, the application of various agents to the round window membrane is an increasingly popular method of treating a number of inner ear disorders and Meniere's Disease in particular. Intratympanic aminoglycosides, particularly gentamicin, is currently the most commonly used non-surgical treatment for Meniere's Disease after diuretics and salt restriction. The local application of corticosteroids and antimetabolites has also been reported for Meniere's. The various clinical trials, outlined in more detail in the Background Section, are confusing because a diverse number of techniques and dosing schedules have been used. The authors propose that one of the underlying problems in interpreting and applying these potential treatments is a lack of understanding of the permeability characteristics and kinetics of the round window membrane. The authors have developed an *in vitro* system to test and calculate permeability constant for the round window membrane for virtually any substance.

To test round window permeability, a guinea pig round window is isolated with its bony annulus and mounted in a double sided Lucite perfusion chamber. The upper chamber, representing the middle ear, allows for a 50 $\mu$ L depot of drug solution. Samples aliquots are taken from the middle ear chamber before and after each experiment to verify the extent of drug depletion. The lower chamber, representing the inner ear, is continuously perfused with balanced salt solution. The outflow is collected every hour for 24 hours in a fraction collector. The permeability constant,  $K_{trans}$  (cm/sec), over 24 hours is calculated as follows:

$$K_{trans} = \frac{R_{total}}{(t)(A)} \times \frac{1}{[D]}$$

where  $R_{total}$  equals the total moles through the RWM in time  $t$ ,  $A$  = the surface area of the exposed RWM (cm<sup>2</sup>),  $t$  = time interval (sec), and  $D$  = concentration of compound in the middle ear chamber (mol/mL).

**Specific Aim 1a.** Determine the stability of the round window membrane in the perfusion chamber over time. Initial experiment lasted 24 hours and the permeability of the round window was stable overtime after an initial equilibrium phase. Subsequent experiments with  $^3\text{H}$ -Dexamethasone carried over 48 hours demonstrated integrity of the round window membrane for the entire experiment.

**Specific Aim 1b.** Determine the permeability constant for the guinea pig round window membrane *in vitro* for gentamicin. The  $K_{\text{trans}}$  for  $^3\text{H}$ -dexamethasone was  $1.5 \pm 0.4 \times 10^{-7}$  cm/s. Radiolabeled prednisone and triamcinolone have just been obtained and will be tested in the second six months.

**Specific Aim 2:** Determine if there is a differential directional permeability of the round window to water.

Witte (2000) evaluated round window membrane transport in a static two-chambered preparation and found that there was net water flow from the middle ear to the inner ear side of the round window membrane. We evaluated the differential diffusion of  $\text{H}^3\text{-H}_2\text{O}$  from the middle ear to the inner ear and vice versa by mounting the round window membrane in the normal configuration and inverted (with the inner ear surface toward the drug reservoir. The  $K_{\text{trans}}$  for the normal orientation was  $2.8 \times 10^{-5}$  verses  $4.1 \times 10^{-5}$  for the inverted membrane. This result is directly contrary to the previous finding in that the membrane was more permeable in the direction from the inner ear to the middle ear. More experiments and histology are pending to determine if this result is the result of an unidentified artifact.

**AWARD OF MERIT RECIPIENTS (1949-2002)**

---

1949	George M. Coates, MD
1951	Barry J. Anson, PhD Theodore H. Bast, PhD
1952	Edmund P. Fowler, Sr., MD
1953	Julius Lempert, MD
1954	Stacy Guild, PhD
1957	Georg von Bekesy, PhD
1959	Ernest Glen Wever, PhD
1960	Hallowell Davis, MD
1961	John R. Lindsay, MD
1962	William J. McNally, MD
1965	Anderson C. Hilding, MD
1966	Gordon D. Hoople, MD
1967	Merle Lawrence, PhD
1968	Lawrence R. Boles, MD
1969	Sir Terence Cawthorne
1970	Senator Joseph A. Sullivan, MB
1971	Samuel Rosen, MD
1972	Howard P. House, MD
1973	Moses H. Lurie, MD
1974	George E. Shambaugh, Jr., MD
1975	Catherine A. Smith, PhD
1976	Harry Rosenwasser, MD
1977	Frank Lathrop, MD
1978	Juergen Tonndorf, MD
1979	John Bordley, MD
1980	Ben H. Senturia, MD
1981	J. Brown Farrior, MD
1982	William F. House, MD
1983	Victor Goodhill, MD
1984	Harold F. Schuknecht, MD
1985	Wesley H. Bradley, MD
1986	John J. Shea, Jr., MD
1987	Jack V. Hough, MD
1988	George D. Nager, MD
1989	Brian F. McCabe, MD
1990	Eugene L. Derlacki, MD
1991	Richard R. Gacek, MD
1992	James L. Sheehy, MD
1993	James A. Donaldson, MD
1994	Fred H. Linthicum, Jr., MD
1995	D. Thane Cody, MD
1996	F. Blair Simmons, MD
1997	Michael E. Glasscock, III, MD
1998	Michael M. Paparella, MD
1999	Mansfield F. W. Smith, MD
2000	Robert A. Jahrsdoerfer, MD
2001	Derald E. Brackmann, MD
2002	Gregory J. Matz, MD

## GUESTS OF HONOR (1974-2002)

---

1974	Harry Rosenwasser, MD
1975	John E. Bordley, MD
1976	Ben H. Senturia, MD
1977	Henry B. Perlman, MD
1978	Howard P. House, MD
1979	Hallowell Davis, MD
1980	Victor Goodhill, MD
1981	Harold Schuknecht, MD
1982	George E. Shambaugh, Jr., MD
1983	Wesley H. Bradley, MD
1984	Brown Farrior, MD
1985	Bruce Proctor, MD
1986	Merle Lawrence, PhD
1987	Robert M. Seyfarth, PhD
1988	G. Dekle Taylor, MD
1989	Eugene L. Derlacki, MD
1990	William F. House, MD
1991	Michael E. Glasscock III, MD
1992	William E. Hitselberger, MD
1992	D. Thane R. Cody, MD
1994	Cesar Fernandez, MD
1995	Richard R. Gacek, MD
1996	James L. Sheehy, MD
1997	Mansfield F.W. Smith, MD
1998	Robert A. Jahrsdoerfer, MD
1999	Barbara A. Bohne, Ph.D.
2000	Derald E. Brackmann, MD
2001	James B. Snow, Jr., MD
2002	David J. Lim, MD

## PAST PRESIDENTS OF AMERICAN OTOLOGICAL SOCIETY

1868-69	E. Williams, MD	1965	Harry Rosenwasser, MD
1870-73	H.D. Noyes, MD	1966	Howard P. House, MD
1874-76	D.B.St.John Roosa, MD	1967	James A. Moore, MD
1877-78	C.J. Blake, MD	1968	G. Shambaugh, Jr., MD
1879-80	A.H. Buck, MD	1969	Frank D. Lathrop, MD
1881-83	J.O. Green, MD	1970	Francis L. Lederer, MD
1884-85	C.H. Burnett, MD	1971	John E. Bordley, MD
1886-89	J.S. Prout, MD	1972	Walter P. Work, MD
1890	O.D. Pomeroy, MD	1973	Ben H. Senturia, MD
1891-94	Gorham Bacon, MD	1974	Wesley H. Bradley, MD
1895-99	Arthur Mathewson, MD	1975	Lester A. Brown, MD
1900-02	H.G. Miller, MD	1976	Victor Goodhill, MD
1903-05	B. Alex Randall, MD	1977	Harold Schuknecht, MD
1906-07	Emil Gruening, MD	1978	Clair M. Kos, MD
1908	C.J. Kipp, MD	1979	G. Dekle Taylor, MD
1909-10	Frederick L. Jack, MD	1980	Eugene Derlacki, MD
1911-12	Edward B. Dench, MD	1981	Richard J. Bellucci, MD
1913-14	J.F.McKernon, MD	1982	J. Brown Farrior, MD
1915-16	C.W. Richardson, MD	1983	Jack V. Hough, MD
1917	C.R. Holmes, MD	1984	Cary N. Moon, Jr., MD
1918	Norval H. Pierce, MD	1985	Francis A. Sooy, MD
1919	Ewing W. Day, MD	1986	Brian F. McCabe, MD
1920	Robert Lewis, MD	1987	Harold G. Tabb, MD
1921	W.P. Eagleton, MD	1988	Richard R. Gacek, MD
1922	H.S. Birkett, MD	1989	D. Thane Cody, MD
1923	G. Shambaugh, Sr., MD	1990	H.A. Ted Bailey, Jr., MD
1924	John B. Rae, MD	1991	William F. House, MD
1925	E.A. Crockett, MD	1992	Michael Glasscock, III, MD
1926	Thomas J. Harris, MD	1993	Mansfield F.W. Smith, MD
1927	Arthur B. Duel, MD	1994	Robert I. Kohut, MD
1928	M.A. Goldstein, MD	1995	Robert A. Jahrsdoerfer, MD
1929	J.G. Wilson, MD	1996	Derald E. Brackmann, MD
1930	S. Mac C. Smith, MD	1997	Joseph C. Farmer, Jr., MD
1931	D.H. Walker, MD	1998	Charles M. Luetje, MD
1932	L.W. Dean, MD	1999	Gregory J. Matz, MD
1933	G.I. Tobey, Jr., MD	2000	C. Gary Jackson, MD
1934	John R. Page, MD	2001	A. Julianna Gulya, MD
1935	Samuel J. Crowe, MD	2002	Richard A. Chole, MD, PhD
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. McMahan, 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		

**PAST SECRETARY-TREASURERS OF AMERICAN OTOLOGICAL SOCIETY**

---

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

**AMERICAN OTOLOGICAL SOCIETY 2002-2003 Membership Roster**  
(please send corrections to AOS Administrative Office)

**ACTIVE MEMBERS**

Professor P. W. Alberti (1982)  
107 Clemanti Road Kent Vale Block F, #13-03, Singapore 129790

Sean R. Althaus, MD (1987)  
5201 Norris Canyon Rd. #230 San Ramon, CA 94583-5405

Ronald G. Amedee, MD (1995)  
Dept. of Otolaryngology Tulane University Medical Center SL-59  
1430 Tulane Avenue  
New Orleans, LA 70112-2699

Patrick J. Antonelli, MD (2001)  
Dept. of Otolaryngology University of Florida PO Box 100264  
Gainesville, FL 31620-0264

Edward Applebaum, MD (1985)  
Dept. of Otolaryngology-HNS Northwestern University Medical School  
303 E. Chicago Ave., Searle 12-573 Chicago, IL 60611

Mosies A. Arriaga, MD (2002)  
Pittsburgh Ear Associates 420 East North Avenue, Suite 402  
Pittsburgh, PA 15212

H. Alexander Arts, MD (2001)  
2670 Apple Way Ann Arbor, MI 48104

Richard W. Babin, MD (1993)  
1830 Hwy 51 South P. O. Box 99 Covington, TN 38019

Thomas J. Balkany, MD (1991)  
University of Miami Ear Institute Dept. of Otolaryngology  
PO Box 016960-D48  
Miami, FL 33101

David M. Barrs, MD (1997)  
Carolina Ear & Hearing Clinic 3100 Duraleigh Road, Ste. 300  
Raleigh, NC 27612

Loren J. Bartels, MD (1992)  
4 Columbia Drive Suite 610 Tampa, FL 33606

Charles W. Beatty, MD (1995)  
Mayo Clinic Dept. of Otolaryngology 200 First St. SW  
Rochester, MN 55905

F. Owen Black, MD (1983)  
1225 NE 2nd Ave (97232) PO Box 3950  
Portland, OR 97208-3950

Brian Blakley, MD (1996)  
Dept. of Otolaryngology Room GB 421- 820 Sherbrook St.  
Winnipeg, Manitoba, Canada R3A 1R9

Charles D. Bluestone, MD (1977)  
Children's Hospital of Pittsburgh Dept. of Pediatric Otolaryngology  
3705 Fifth Avenue  
Pittsburgh, PA 15213-1583

Derald E. Brackmann, MD (1979)  
2100 West Third St. 1st Floor Los Angeles, CA 90057

Hilary A. Brodie, MD, PhD (2001)  
Otolaryngology Research Labs University of California Davis  
1515 Newton Court #209  
Davis, CA 95616

Patrick Brookhouser, MD (1988)  
Boystown National Institute 555 N. 30th St. Omaha, NE 68131

Rinaldo F. Canalis, MD (1991)  
457-15th St. Santa Monica, CA 90402

Stephen P. Cass, MD (2000)  
4200 East 9th Ave., B-205 Denver, CO 80262

Margaretha L. Casselbrant, MD (2001)  
3705 Fifth Avenue Children's Hospital of Pittsburgh  
Pittsburgh, PA 15213

Richard A. Chole, MD (1984)  
Dept. of Otolaryngology Washington University Medical School  
660 S. Euclid, Box 8115  
St. Louis, MO 63110

Jack D. Clemis, MD (1976)  
734 LaVergne Avenue Wilmette, IL 60091

Noel L. Cohen, MD (1985)  
Dept. of Otolaryngology NYU Medical Center 530 First Avenue  
New York, NY 10016

Newton J. Coker, MD (1991)  
Baylor College of Medicine Department of Otorhinolaryngology  
6550 Fannin, Suite 1727  
Houston, TX 77030

C. Phillip Daspit, MD (1995)  
222 W. Thomas Road Suite 114 Phoenix, AZ 85013-4480

Antonio De La Cruz, MD (1991)  
2100 W. Third St. 1st Fl Los Angeles, CA 90057

M. Jennifer Derebery, MD (2002)  
House Ear Clinic, Inc. 2100 West Third Street  
Los Angeles, CA 90057-1922

John R.E. Dickins, MD (1991)  
10201 Kanis Road Little Rock, AR 72205

Robert A. Dobie, MD (1985)  
Division of Extramural Research, NIH/NIDCD EPS, MSC  
7180 6210 Executive Blvd, Ste 400C Bethesda, MD 20892-7180

Karen Jo Doyle, MD, PhD (2002)  
2521 Stockton Blvd. #7200 Sacramento, CA 95817

Larry G. Duckert, MD (1988)  
Dept. of Otolaryngology MS Box 357923  
University of Washington  
Seattle, WA 98195-7923

Thomas L. Eby, MD (1995)  
University of AL-Birmingham Dept. of Otolaryngology  
1501 5th Ave. South  
Birmingham, AL 35233

John R. Emmett, MD (1990)  
6133 Poplar Pike at Ridgeway Shea Clinic Memphis, TN 38119

John M. Epley, MD (2001)  
545 NE 47th Ave. Ste. 212 Portland, OR 97213

George W. Facer, MD (1994)  
3643 Hidden Cove N.E. Rochester, MN 55906

Joseph C. Farmer, Jr., MD (1984)  
Division of Otolaryngology Duke University Medical Ctr. Box 3805  
Durham, NC 27710

Jay B. Farrow, III, MD (1990)  
509 Bay Street Tampa, FL 33606

Joseph G. Feghali, MD, FACS (2002)  
3340 Bainbridge Ave. Bronx, NY 10467

Rick Friedman, MD; PhD (2001)  
House Ear Clinic 2100 West 3rd. St. Los Angeles, CA 90057

Bruce J. Gantz, MD (1987)  
Dept. of Otolaryngology-HNS University of Iowa  
200 Hawkins Drive Iowa City, IA 52242

L. Gale Gardner, Jr., MD (1983)  
1750 Madison Avenue, Suite 280 Memphis, TN 38104-6492

George A. Gates, MD (1987)  
Dept. of Otolaryngology PO Box 357923 University of Washington  
Seattle, WA 98195-6515

Joel A. Goebel, MD (1995)  
Washington Univ SOM 517 S. Euclid Avenue Box 8115  
St. Louis, MO 63110

Robert A. Goldenberg, MD (1989)  
255 North Main St. Centerville, OH 45459

Richard L. Goode, MD (1990)  
300 Pasteur Dr. R135 Stanford, CA 94305

Marcos V. Goycoolea, MD (1992)  
Pedro Lira Urquieta 11154 Lo Barnechea Santiago, CHILE

A. Julianna Gulya, MD (1991)  
1558 North Colonial Terrace Arlington, VA 22209

Thomas J. Haberkamp, MD (1997)  
6726 N. Wildwood Avenue Chicago, IL 60646

Paul E. Hammerschlag, MD (2001)  
650 First Avenue New York, NY 10016

Lee A. Harker, MD (1987)  
Deputy Director Boys Town National Research Hospital  
555 North 30th St. Omaha, NE 68131

Stephen G. Harner, MD (1987)  
Mayo Clinic 200 First St., S.W. Rochester, MN 55905

Jeffrey P. Harris, MD, PhD (1988)  
UCSD Head & Neck Surgery 200 W. Arbor Drive 8895  
San Diego, CA 92103-8895

Barry E. Hirsch, MD (1996)  
Eye & Ear Institute Bldg. 200 Lothrop St., Suite 500  
Pittsburgh, PA 15213

Ronald A. Hoffman, MD (1992)  
10 Union Square E Frnt 2 New York, NY 10003-3314

Karl L. Horn, MD (2001)  
201 Cedar S.E., Suite #808 Albuquerque, NM 87106

John W. House, MD (1984)  
House Ear Clinic/Institute 2100 W. 3rd Street Los Angeles, CA 90057

Gordon B. Hughes, MD (1987)  
A-71 Cleveland Clinic 9500 Euclid Avenue Cleveland, OH 44195

Robert K. Jackler, MD (1992)  
Univ of CA-San Francisco, 400 Parnassus Ave. A-730  
San Francisco, CA 94143-0342

Carol A. Jackson, MD (1994)  
361 Hospital Road Suite 325 Newport Beach, CA 92663

C. Gary Jackson, MD (1990)  
The Otology Group 300 20th Avenue North Suite 502  
Nashville, TN 37203

Anthony Jahn, MD (1992)  
556 Eagle Rock Avenue Roseland, NJ 07068

Herman A. Jenkins, MD (1987)  
University of Colorado - Dept. of Otolaryngology 4200 E. 9th Ave. B205  
Denver, CO 80262

Glenn D. Johnson, MD (2001)  
Dartmouth-Hitchcock Medical Society One Medical Center Drive  
Lebanon, NH 03756-0001

Timothy K. Jung, MD (1990)  
3975 Jackson St., Suite 202 Riverside, CA 92503

Donald B. Kameron, MD (1988)  
Eye & Ear Hospital 200 Lothrop St., Suite 500 Pittsburgh, PA 15213

Jack M. Kartush, MD (1991)  
Michigan Ear Institute 30055 Northwestern Hwy. #101  
Farmington Hills, MI 48334

Athanasios Katsarkas, MD (1991)  
Royal Victoria Hospital #E 4.48 687 Pine Avenue W  
Montreal, Qc, CANADA H3A 1A1

Barry P. Kimberley, MD (2001)  
701 25th Avenue South Suite 200 Minneapolis, MN 55454

Sam E. Kinney, MD (1981)  
60 Pebblebrook Lane Moreland Hills, OH 44022

Horst R. Konrad, MD (1991)  
SIU School of Medicine Dept. of Otolaryngology PO Box 19662  
Springfield, IL 62794-9662

Arvind Kumar, MD (1993)  
1855 W. Taylor St. Chicago, IL 60612

Anil K. Lalwani, MD (1999)  
400 Parnassus Avenue, A730 San Francisco, CA 94143-0342

Paul R. Lambert, MD (1995)  
Otolaryngology-HNS P. O. Box 250550 135 Rutledge Avenue  
Charleston, SC 29425

K. J. Lee, MD (1997)  
98 York Street New Haven, CT 06511

John P. Leonetti, MD (1995)  
Loyola University Medical Ctr. 2160 S. First Avenue  
Bldg 105-Room 1870  
Maywood, IL 60153

S. George Lesinski, MD (1993)  
10550 Montgomery Road #34 Cincinnati, OH 45242

Samuel C. Levine, MD (1999)  
Box 396 420 Delaware St. Minneapolis, MN 55455

Charles M. Luetje, MD (1991)  
Otolologic Center, Inc. 3100 Broadway, Suite 509  
Kansas City, MO 64111

Charles A. Mangham, Jr., MD (1987)  
Seattle Ear Clinic 600 Broadway  
Seattle, WA 98122-5371

Robert H. Mathog, MD (1985)  
4201 St. Antoine 5E Detroit, MI 48201

Douglas E. Mattox, MD (1992)  
1365 Clifton Road, NE Room 2325 Emory Clinic "A"  
Atlanta, GA 30322

Thomas J. McDonald, MD (1987)  
Mayo Clinic 200 First St., S.W. Rochester, MN 55905

John T. McElveen, Jr., MD (1997)  
Carolina Ear & Hearing Clinic 3100 Duraleigh Road, Ste. 300  
Raleigh, NC 27612

Michael McGee, MD (2002)  
3400 N. W. 56th St. Oklahoma City, OK 73112

Michael J. McKenna, MD (1999)  
Massachusetts Eye & Ear Infirmary 243 Charles Street  
Boston, MA 02114-3096

Saumil N. Merchant, MD (2000)  
Dept. of Otolaryngology Massachusetts Eye & Ear Infirmary  
243 Charles Street  
Boston, MA 02114

Lloyd B. Minor, MD (2001)  
Dept. of Otolaryngology-HNS Johns Hopkins Outpatient Ctr  
601 N. Caroline Street Rm 6253  
Baltimore, MD 21287-0910

Richard T. Miyamoto, MD (1987)  
702 Barnhill Dr., Suite 0860 Riley Hospital  
Indianapolis, IN 46202

William H. Moretz, Jr., MD (1999)  
818 St. Sebastian Way Suite 204 Augusta, GA 30901

Edwin M. Monsell, MD (1995)  
Dept. of Otolaryngology-HNS Wayne State University  
4201 St. Antoine 5E-UHC  
Detroit, MI 48201

Terrence P. Murphy, MD (2002)  
5555 Peachtree Dunwoody Rd, Ste. G-51 Atlanta, GA 30342

Joseph B. Nadol, Jr., MD (1988)  
243 Charles St. Boston, MA 02114

Julian M. Nedzelski, MD (1987)  
Sunnybrook Medical Ctr. 2075 Bayview Avenue  
Toronto, Ontario M4N3M5, CANADA

J. Gail Neely, MD (1985)  
Washington Univ SOM 517 S. Euclid Avenue Box 8115  
St. Louis, MO 63110

Ralph A. Nelson, MD (1995)  
House Ear Institute, Inc. 2100 West Third St., Ste. 111  
Los Angeles, CA 90057

John K. Niparko, MD (1995)  
Dept. of Otolaryngology-HNS Johns Hopkins University  
601 N. Caroline Street, 6th Fl  
Baltimore, MD 21287-0910

James E. Olsson, MD (1993)  
Texas Neurosciences Institute 4410 Medical Drive #550  
San Antonio, TX 78229

Dennis Pappas, MD (1985)  
2937 7th Avenue South Birmingham, AL 35233

Simon C. Parisier, MD (1982)  
210 East 64th St. New York, NY 10021-7471

Lorne S. Parnes, MD (2000)  
University of Western Ontario-Dept. of Otolaryngology  
London Health Sciences Centre  
339 Windermere Road London, Ontario, CANADA N6A 5A5

Steven M. Parnes, MD (2002)  
Albany Medical College 47 New Scotland Avenue, MC-41  
Albany, NY 12208

Myles L. Pensak, MD (1992)  
University of Cincinnati PO Box 670528  
Cincinnati, OH 45267-0528

Harold C. Pillsbury, MD (1988)  
University of North Carolina 610 Burnett-Womack Bldg CB7070  
Chapel Hill, NC 27599-7070

Dennis S. Poe, MD (1995)  
Zero Emerson Place Suite 2-C Boston, MA 02114

Jack Pulec, MD (1969)  
1245 Wilshire Blvd. Ste. 503 Los Angeles, CA 90017

Franklin M. Rizer, MD (1999)  
3893 E. Market Street Warren, OH 44484

Peter S. Roland, MD (1992)  
Dept. of Otolaryngology 5323 Harry Hines Blvd  
Dallas, TX 75235-9035

Seth Rosenberg, MD (2001)  
1961 Floyd St. Sarasota, FL 34239

Allan Rubin, MD (1997)  
Medical College of Ohio Hospital 3065 Arlington Avenue  
Toledo, OH 43614-2807

Jay T. Rubinstein, MD (2002)  
University of Iowa Dept. of Otolaryngology-HNS 200 Hawkins Dr.  
Iowa City, IA 52242

Leonard P. Rybak, MD (1989)  
SIU School of Medicine PO Box 19638  
Springfield, IL 62794-9638

Clarence T. Sasaki, MD (1992)  
Yale Univ. SOM-Sec of Otolaryngology PO Box 208041  
New Haven, CT 06520-8041

Robert T. Sataloff, MD (1990)  
1721 Pine Street Philadelphia, PA 19103

Robert A. Schindler, MD (1983)  
400 Parnassus Avenue Room A-730 San Francisco, CA 94117-3608

Alexander J. Schleuning, MD (1995)  
3181 S.W. Sam Jackson Park Road Portland, OR 97201

Arnold G. Schuring, MD (1990)  
3893 East Market St. Warren, OH 44484

Mitchell K. Schwaber, MD (1993)  
2400 Patterson St. Ste. 418 Nashville, TN 37203

Michael D. Seidman, MD (2001)  
Henry Ford Health System-Dept. of Otolaryngology  
6777 West Maple Road  
West Bloomfield, MI 48322

Samuel H. Selesnick, MD (1999)  
Dept. of Otorhinolaryngology Starr Bldg., Suite 541 520 E. 70th St.  
New York, NY 10021

Clough Shelton, MD (1995)  
50 North Medical Drive, 3C120 Salt Lake City, UT 84132

Herbert Silverstein, MD (1973)  
1961 Floyd St. - Suite A Sarasota, FL 33579

George T. Singleton, MD (1972)  
University of Florida JHMHC, Box 100264 Gainesville, FL 32610

Aristides Sismanis, MD (1993)  
1917 Windingridge Drive Richmond, VA 23233

Peter G. Smith, MD (1988)  
Midwest Otologic Group 621 South New Ballas Road Suite 597-C  
St. Louis, MO 63141-8232

Gershon Jerry Spector, MD (1979)  
517 South Euclid Box 8115 St. Louis, MO 63110

Steven A. Telian, MD (1997)  
Dept. of Otolaryngology-HNS Univ. of Michigan Medical Center  
1500 E. Medical Center Dr. TC1904L Ann Arbor, MI 48109-0312

Fred F. Telischi, MD (2002)  
Dept. of Otolaryngology (D-48) PO Box 016960 Miami, FL 33101

Norman Wendell Todd, Jr. MD (1996)  
1052 Castle Falls Drive Atlanta, GA 30329-4135

Debara L. Tucci, MD (2000)  
Duke University Medical Center Division of Otolaryngology-HNS  
Box 3805  
Durham, NC 27710

Phillip A. Wackym, MD (1997)  
Dept. of Otolaryngology & Communications Sciences  
Medical College of Wisconsin  
9200 W. Wisconsin Avenue Milwaukee, WI 53226

Jack J. Wazen, MD (1993)  
111 East 77th St. New York, NY 10021

Peter C. Weber, MD (2002)  
The Cleveland Clinic Foundation 9500 Euclid Avenue, Ste. A71  
Cleveland, OH 44195

Dudley J. Weider, MD (1990)  
38 Rip Road Hanover, NH 03755

D. Bradley Welling, MD (1998)  
456 West 10th Ave. Columbus, OH 43210-1240

Stephen J. Wetmore, MD (2001)  
P. O. Box 9200 2222 Health Sciences Center  
Morgantown, WV 26506-9200

Richard J. Wiet, MD (1987)  
Chicago Otology Group 950 York Road, Ste 102  
Hinsdale, IL 60521

David F. Wilson, MD (1992)  
911 N.W. 18th Avenue Portland, OR 97209

Eiji Yanagisawa, MD (1996)  
98 York Street New Haven, CT 06511

## SENIOR MEMBERS

Kedar Adour, MD (1999 (1988))  
Sir Charles Bell Society 1000 Green Street #1203  
San Francisco, CA 94133

Bobby R. Alford, MD (1997 (1970))  
One Baylor Plaza, NA-102 Houston, TX 77030

Beverly Armstrong, MD (1988 (1960))  
3034 Hampton Avenue Charlotte, NC 28207

H.A. Ted Bailey, Jr., MD (1994 (1969))  
c/o Bailey Corp. 1400 West Markham Little Rock, AR 72201

Richard J. Bellucci, MD (1990 (1958))  
162 E. 71st St. New York, NY 10021

Roger Boles, MD (1999 (1982))  
Box 620203 Woodside, CA 94062

Wesley H. Bradley, MD (1988 (1961))  
13 Saybrook East Glenmont, NY 12077-9666

Seymour J. Brockman, MD (1988 (1964))  
222 S. McCarty Drive Beverly Hills, CA 90212

Richard A. Buckingham, MD (1994 (1969))  
1420 Sheridan Rd., Apt. 8D Wilmette, IL 60091-1860

Robert W. Cantrell, MD (2000 (1979))  
University of Virginia MSC, P. O. Box 800713  
Charlottesville, VA 22908-0713

Francis I. Catlin, MD (1996 (1975))  
13307 Queensbury Houston, TX 77079-6013

J. Ryan Chandler, MD (1994 (1973))  
3170 Munroe Drive Miami, FL 33133

D. Thane Cody, MD (1992 (1969))  
518 East MacEwen Drive Osprey, FL 34229

James M. Cole, MD (1990 (1966))  
1301 Red Danville, PA 17821-1333

Wesley E. Compere, MD (1989 (1968))  
4519 Mayapan Dr. LeMesa, CA 91941-7145

James A. Crabtree, MD (1995 (1972))  
1332 Westhaven Road San Marino, CA 91108

Vijay S. Dayal, MD (2001 (1975))  
Univ. of Chicago Medical Ctr. MC1035, 5841 South Maryland Ave  
Chicago, IL 60637

Eugene L. Derlacki, MD (1989 (1958))  
700 W. Fabyan Parkway #22A Batavia, IL 60510

James A. Donaldson, MD (1994 (1974))  
5746 65th Ave. NE Seattle, WA 98105-2044

Patrick J. Doyle, MD (1996 (1987))  
301 - 5704 Balsam Street Vancouver, BC V6M4

Joseph G. Druss, MD (1971 (1939))  
145 East 92nd St. New York, NY 10028

Arndt J. Duvall III, MD (1993 (1971))  
420 Delaware St. S.E. Box 396 Minneapolis, MN 55455

Abraham Eviatar, MD (1999 (1981))  
25 Morris Scarsdale, NY 10583

John M. Fredrickson, MD (2002 (1978))  
205 Dartmouth Drive, S.E. Albuquerque, NM 87106

Richard R. Gacek, MD (1998 (1969))  
2880 Dauphin Street Mobile, AL 36606

Michael Glasscock III, MD (1997 (1973))  
100 Pemberton Way Austin, TX 78737

Malcolm D. Graham, MD (2001 (1979))  
Department of Otolaryngology 1904 Taubman Center  
1500 E. Medical Center Drive  
Ann Arbor, MI 48109-0312

Irwin Harris, MD (1993 (1970))  
2160 Century Woods Way Los Angeles, CA 90067-6307

Wiley H. Harrison, MD (1993 (1973))  
Address Unknown - Mailed Returned 6/00

Cecil W.J. Hart, MD (2001 (1992))  
1053 East El Alameda Palm Springs, CA 92262-5815

David A. Hilding, MD (1990 (1972))  
3156 South Plateau Drive Salt Lake City, UT 84109-2358

Jerome Hilger, MD (1975 (1951))  
1700 Lexington Avenue Suite 409 St. Paul, MN 55118

Albert Hohmann, MD (1990 (1970))  
3154 Shoreline New Brighton, MN 55112-3764

Jack V.D. Hough, MD (1990 (1960))  
3400 NW 56th Street Oklahoma City, OK 73112-4466

Howard P. House, MD (1975 (1947))  
2100 West Third St. Los Angeles, CA 90057

William F. House, MD (1995 (1964))  
AllHear, Inc. P. O. Box 330 Aurora, OR 97002

Robert A. Jahrsdoerfer, MD (2001 (1982))  
Dept of Otolaryngology University of Virginia Med. Ctr.  
P. O. Box 800713  
Charlottesville, VA 22908

Arthur L. Juers, MD (1972 (1952)) - Address Unknown

- Robert I. Kohut, MD (1998 (1976))  
Bowman Gray School of Med. Dept. of Otolaryngology  
Medical Center Blvd.  
Winston-Salem, NC 27157-1034
- Fred H. Linthicum, Jr., MD (1991 (1967))  
2100 West Third St. Los Angeles, CA 90057
- William H. Lippy, MD (1999 (1988))  
3893 East Market St. Warren, OH 44484
- Ward B. Litton, MD (1995 (1969))  
17 Eagle Pointe Pass PO Box 266 Rapids City, IL 61278-0266
- H. Edward Maddox III, MD (1996 (1970))  
6249 Terwilliger Houston, TX 77057
- Richard E. Marcus, MD (1987 (1975))  
691 Sheridan Road Winnetka, IL 60093
- Gregory J. Matz, MD (2002 (1979))  
910 North Lake Shore Drive Unit #1119 Chicago, IL 60611
- Brian F. McCabe, MD (1997 (1965))  
University of Iowa Dept. of Otolaryngology  
200 Hawkins Drive-E230 GH  
Iowa City, IA 52242-1078
- William W. Montgomery, MD (1997 (1975))  
243 Charles St. Boston, MA 02114
- William L. Meyerhoff, MD (2002 (1981))  
U.T. Southwestern Medical Center 5323 Harry Hines Blvd  
Dallas, TX 75235-9035
- Eugene N. Myers, MD (1994 (1974))  
Eye and Ear Institute 200 Lothrop St., Suite 500 Pittsburgh, PA 15213
- George T. Nager, MD (1994 (1968))  
Johns Hopkins Hospital Dept. of ORL-HNS 550 N. Broadway  
Baltimore, MD 21205-2020
- Ralph F. Naunton, MD (1993 (1968))  
3303 Pauline Drive Chevy Chase, MD 20815-3919
- Michael M. Paparella, MD (2000 (1968))  
701 25th Avenue South Suite 200 Minneapolis, MN 55454
- James J. Pappas, MD (2002 (1983))  
8 Riding Road Little Rock, AR 72227
- Claude L. Pennington, MD (1993 (1973))  
PO Box 1916 Macon, GA 31202
- Shokri Radpour, MD (1998 (1989))  
Otolaryngology-HNS (112A) VA Medical Center 1481 W. 10th St.  
Indianapolis, IN 46202
- J. H. Thomas Rambo, MD (1983 (1958))  
150 East 77th St. Apt. 8A New York, NY 10021
- Frank N. Ritter, MD (1993 (1972))  
2675 Englave Drive Ann Arbor, MI 48197
- Mendell Robinson, MD (1991 (1969))  
130 Waterman St. Providence, RI 02906

Max L. Ronis, MD (1997 (1972))  
1601 Walnut St. #1405 Philadelphia, PA 19102

Robert Ruben, MD (1996 (1974))  
Montefiore Medical Center 3400 Bainbridge Ave Greene Pavilion  
Bronx, NY 10467-2490

Wallace Rubin, MD (1992 (1967))  
3434 Houma Blvd Suite 201 Metairie, LA 70006

Richard L. Ruggles, MD (1993 (1967))  
11201 Shaker Blvd. Cleveland, OH 44104

Joseph Sataloff, MD (1994 (1960))  
1721 Pine Street Philadelphia, PA 19103

William H. Saunders, MD (1996 (1972))  
4710 Old Ravine Court Columbus, OH 43220

John J. Shea, Jr., MD (1998 (1967))  
6133 Poplar Pike Memphis, TN 38119

James L. Sheehy, MD (1994 (1965))  
2100 West Third St. Los Angeles, CA 90057

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

Mansfield F.W. Smith, MD (2000 (1973))  
417 Mace Blvd. J-330 Davis, CA 95616-6053

James B. Snow, Jr., MD (1993 (1973))  
33506 Tuckahoe River Road Easton, MD 21601

Malcom H. Stroud, MD (1990 (1967))  
4412 Stanhope Ave. Dallas, TX 75205

Harold G. Tabb, MD (1990 (1961))  
1430 Tulane Avenue New Orleans, LA 70112

G. Dekle Taylor, MD (1985 (1965))  
4600 Middleton Park Cir E Apt A325 Jacksonville, FL 32224-3205

Paul H. Ward, MD (1994 (1972))  
UCLA School of Medicine Division of Head & Neck Surgery  
10833 LeConte Ave.  
Los Angeles, CA 90095-3075

Roger E. Wehrs, MD (1996 (1975))  
6909 S. Evanston Tulsa, OK 74136

William H. Wilson, MD (1989 (1972))  
160 South Monaco Parkway, 702 Denver, CO 80224

Robert J. Wolfson, MD (1994 (1971))  
Dept. of Otolaryngology-HNS 219 N. Broad St., 10th Fl  
Philadelphia, PA 19107

## **ASSOCIATE MEMBERS**

Joe C. Adams, PhD (2001)  
Massachusetts Eye & Ear Infirmary ENT Dept. 243 Charles Street  
Boston, MA 02114

Richard A. Altschuler, PhD (1992)  
Kresge Hearing Research Institute University of Michigan  
1301 N. Ann Street  
Ann Arbor, MI 48109-0506

- James F. Battey, Jr., MD, PhD (2001)  
NIDCD Building 31, Room 3C02 31 Center Drive MSC 2320  
Bethesda, MD 20892-2320
- Karen I. Berliner, PhD (1995)  
119 Voyage Mall Marina del Rey, CA 90292
- Barbara A. Bohne, PhD (1979)  
Washington Univ SOM Box 8115 St. Louis, MO 63110
- Robert A. Butler, PhD (1978)  
Dept. of Surgery University of Chicago 950 E. 59th St.  
Chicago, IL 60637
- Ruth Gussen, MD (1977)  
3124 Rehabilitation Center UCLA School of Medicine  
Los Angeles, CA 90024
- Mohamed A. Hamid, MD, PhD (1992)  
Cleveland Hearing & Balance Ctr. 24755 Chagrin Blvd, #310  
Cleveland, OH 44122
- Maureen T. Hannley, PhD (1992)  
American Academy of Otolaryngology-HNS One Prince Street  
Alexandria, VA 22314
- Joseph E. Hawkins, Jr., PhD (1972)  
Kresge Hearing Research Institute University of Michigan  
1301 East Ann Street  
Ann Arbor, MI 48109-0506
- Raul Hinojosa, MD (1989)  
5316 Hyde Park Blvd Chicago, IL 60615
- Vincente Honrubia, MD (1972)  
10833 LeConte Avenue Los Angeles, CA 90024
- Makoto Igarashi, MD (1973)  
Univ. Research Ctr. Nihon University 8-24 Kudan-minami  
4chome Chiyoda-ku  
Tokyo 102, JAPAN 102-0074
- Salvatore J. Iurato, MD (1994)  
via L. Ricchioni 10-N I-70124 Bari, ITALY
- Pawel J. Jastreboff, PhD (1997)  
10127 Frost Way Ellicott, MD 21042
- Walter H. Johnson, PhD (1960)  
201-1833 Bayview Ave. Toronto ONT M4G 3E2, CANADA
- Lars-Goran Johnsson, MD (1979)  
Saynavakuja 4B6 Sipoo 12170, FINLAND
- Steven K. Juhn, MD (1980)  
University of Minnesota Medical Sch 2001 6th St. SE  
Minneapolis, MN 55455
- Nelson Y.S. Kiang, PhD (1969)  
18 Cedar Way Boston, MA 02108
- Paul R. Kileny, PhD (1994)  
University of Michigan Health System Dept. of Otolaryngology  
1500 E. Medical Center Dr TC 1904 Ann Arbor, MI 48109-0312
- Robert S. Kimura, PhD (1978)  
21 Woodchester Drive Weston, MA 02493

Merle Lawrence, PhD (1959)  
3359 Burbank Dr. Ann Arbor, MI 48105

David J. Lim, MD (1973)  
House Ear Institute, Inc. 2100 West Third St., 5th Fl  
Los Angeles, CA 90057

Brenda Lonsbury-Martin, PhD (1997)  
Department of Otolaryngology (B205)  
University of Colorado Health Sciences Center 4200 E. Ninth Ave.  
Denver, CO 80262-0001

Michael Merzenich, PhD (1986)  
University of California Coleman Laboratory HSE 871  
San Francisco, CA 94143

Josef M. Miller, PhD (1979)  
University of Michigan Kresge Hearing Research Inst.  
1301 East Ann Street  
Ann Arbor, MI 48109

Tetsuo Morizono, MD (1985)  
Fukuoka University Medical School 814-01 Rm Jonak-Kufukuoka  
Nanakuma 7-45-1, JAPAN

William D. Neff, PhD (1978)  
3080 Hideway Court Morris, IL 60450

Daniel J. Orchik, PhD (1996)  
6133 Poplar Pike Memphis, TN 38119

Edwin W Rubel, PhD (1986)  
Dept. of Otolaryngology RL-30 University of Washington  
Seattle, WA 98195

Jai H. Ryu, PhD (1989)  
Dept. of Otolaryngology Bowman Gray School of Medicine  
Winston-Salem, NC 27157

Isamu Sando, MD (1975)  
203 Lothrop St. Pittsburgh, PA 15213

Jochen Schact, PhD (1992)  
Kresge Hearing Research Institute University of Michigan  
1301 East Ann Street  
Ann Arbor, MI 48109-0506

S. Richard Silverman, PhD (1950)  
Mailed returned 2/26/02

Catherine A. Smith, PhD (1962)  
17320 Holy Names Drive. Apt. C-302 Lake Oswego, OR 97034

Jack McLean Snyder, PhD (1992)  
1661 Pine Street #932 San Francisco, CA 94109

Ruediger Thalmann, MD (1971)  
Washington Univ SOM Dept. of Otolaryngology  
517 S. Euclid Ave  
St. Louis, MO 63110

Galdino Valvassori, MD (1970)  
697 Sheridan Road Winnetka, IL 60093

Thomas R. Van De Water, PhD (1987)  
Director, Cochlear Implant Research Program  
University of Miami Ear Institute  
1600 N. W. 10th Avenue, RMSB 3160  
Miami, FL 33136

Jack A. Vernon, PhD (1974)  
3515 S.W. Sam Jackson Park Rd. Portland, OR 97201

Charles G. Wright, PhD (1999)  
Dept. of Otolaryngology UT Southwestern Medical Ctr  
5323 Harry Hines Blvd.  
Dallas, TX 75235-9035

Sabina Regina Wullstein, MD (1999)  
Oberer Neubergweg 10 D- 97074, Wurzburg, GERMANY

Joseph J. Zwislocki, ScD (1984)  
Institute of Sensory Research Syracuse University  
Syracuse, NY 13244-5290

## **EMERITUS MEMBERS**

Warren Y. Adkins, MD (2001 (1987))  
1187 Farm Quarter Road Mt. Pleasant, SC 29464

B. Hill Britton, MD (2000 (1978))  
University of OK-HSC Dept. of Otolaryngology PO Box 26901  
Oklahoma City, OK 73190

Donald W. Goin, MD (1994 (1987))  
Attn: Lynn Larson 799 E. Hampton Ave. Ste. 510  
Englewood, CO 80110

Robert J. Keim, MD (1997 (1987))  
13504 Green Cedar Lane Oklahoma City, OK 73131

Roger C. Lindeman, MD (2001 (1987))  
6115 79th Ave. S.E. Mercer Island, WA 98040

Anthony J. Maniglia, MD (1999 (1989))  
Dept. of Otolaryngology University Hospitals of Cleveland  
11100 Euclid Avenue  
Cleveland, OH 44106-5045

James L. Parkin, MD (1997 (1986))  
10 Beechwood Drive Cobham Surrey, England, KT11-2DX UK

Leonard R. Proctor, MD (1997 (1989))  
8102 Halton Road Towson, MD 21204-1817

## **CORRESPONDING MEMBERS**

Soontorn Antarasena, MD (1997)  
Dept. of Otolaryngology Rahvithi Hospital Rajvithi Road  
Phyathai Bangkok 10400, THAILAND

Daniel J. Bagger-Sjoberg, MD (1995)  
Dept. of Otolaryngology Karolinska Hospital 17176  
Stockholm S104 1, SWEDEN

Dr. J. Booth (1995)  
24 The Crofts Castletown Isle of Man IM9 1LZ, UK

Vicente G. Diamante, MD (2000)  
Pasteur 740 1028 Buenos Aires, ARGENTINA

Paul A. Fagan, MD, FRACS (1997)  
352 Victoria Street DARLINGHURST NSW 2010, AUSTRALIA

Bernard Gil Fraysse, MD (1999)  
Dept ORL - Hospital Purpan CHU de Toulouse 31059 Toulouse  
Cedex, FRANCE

Johannes J. Grote, MD, PhD (2002)  
ENT Department Leiden University Medical Center  
PO Box 9600 2300 R.C. Leiden  
THE NETHERLANDS

Chong-Sun Kim, MD (1998)  
Dept. of Otolaryngology Seoul National University Hospital  
28 Yongon-Dong Chongno Gu  
Seoul 110-744, KOREA

Takeshi Kubo, MD (2000)  
Dept. of Otolaryngology Osaka Univ. Graduate Sch of Medicine  
(E8) 2-2 Yamadaoka, Suita  
Osaka, JAPAN 565-0871

Thomas E. Linder, MD (2001)  
Chairman, Dept. of Otorhinolaryngology Kantonsspital  
Luzern CH- 6000  
Luzern, SWITZERLAND

Wolf J. Mann, MD (1996)  
University ENT Department Mainz Medical School 55131  
Langenbeckstr 1 D65101  
Mainz, GERMANY

Mr. David A. Moffat, MA, FRCS (1996)  
Dept of Otoneurological & Skull Base Clinic  
10 Addenbrooke's Hospital Hills Road  
Cambridge CB2 2QQ, ENGLAND

Lars Odkvist, M.D, PhD (1999)  
Rundelsgatan 4 b SE-58721 Linkoping, SWEDEN

Professor Ilmari Pyykko (1997)  
ENT Department Karolinska Hospital S-171 76  
Stockholm, SWEDEN

Helge Rask-Andersen, MD, PhD (1996)  
Luthagsespl - 15B, 75225 Uppsala, SWEDEN

Jens Thomsen, MD (1996)  
ENT Department Gentofte University Hospital  
Hellerup, 2900, DENMARK

Thomas P.U. Wustrow, MD (2000)  
HND-Gemeinschaftspraxis Wittelsbacherplatz I/II D-80333  
Munchen, GERMANY

#### **HONORARY MEMBERS**

Pedro Albermaz (1993)  
4405 NW 73rd Avenue Suite 20-40003 Miami, FL 33166

Aziz Belal, MD (1993)  
Neurotology Section Alexandria Ear & Eye Hospital 1 Sidi Gaber St.  
Alexandria, EGYPT

Edgar L. Chiossone, MD (1993)  
Apartrado 62.277 Caracas 1060-A, VENEZUELA

Graeme M. Clark, PhD (2002)  
Bionic Ear Institute 384 Albert St. East Melbourne  
Victoria, AUSTRALIA 3002

Ugo Fisch, MD (1985)  
Forchstrasse 26 CH-8703 Erlenbach, SWITZERLAND

Jerome C. Goldstein, MD (1992)  
4119 Manchester Lake Drive Lake Worth, FL 33467

William E. Hitselberger, MD (1997)  
2222 Oceanview Suite 199 Los Angeles, CA 90057

L.B.W. Jongkees (1968)  
Reijnier Vinkeleskade 71 1071 S2 Amsterdam ENT Dept.  
Wilhelmina Gasthuis, THE NETHERLANDS

Andrew Morrison (1985)  
"Dyers" Marden Ash Chipping Ongar Essex CM5 9BT, UK

Yasuya Nomura (1992)  
Dept. of Otolaryngology Showa University  
1-5-8 Hatanodai, Shinagawa-Ku Tokyo 142, JAPAN

Michel Portmann (1983)  
114 Ave de' Ares Bordeaux 33000, FRANCE

#### **Deceased Since 2002 Meeting**

**Ralph J. Caparosa, MD**  
Active 1972  
Senior 1992  
Died: 5/2001

**James A. Moore, MD**  
Active 1952  
Senior 1987  
Died: Date unknown

**Walter A. Rosenblith, MD**  
Associate 1970  
Died: 5/1/2002