

**1995**

TRANSACTIONS  
AMERICAN OTOLOGICAL SOCIETY, INC.  
1995

VOLUME EIGHTY-THREE



ONE HUNDRED TWENTY-EIGHTH ANNUAL MEETING  
MARRIOTT'S DESERT SPRINGS RESORT  
PALM DESERT, CALIFORNIA  
APRIL 29 AND 30, 1995

≈

 **Lippincott - Raven**  
P U B L I S H E R S  
Philadelphia • New York

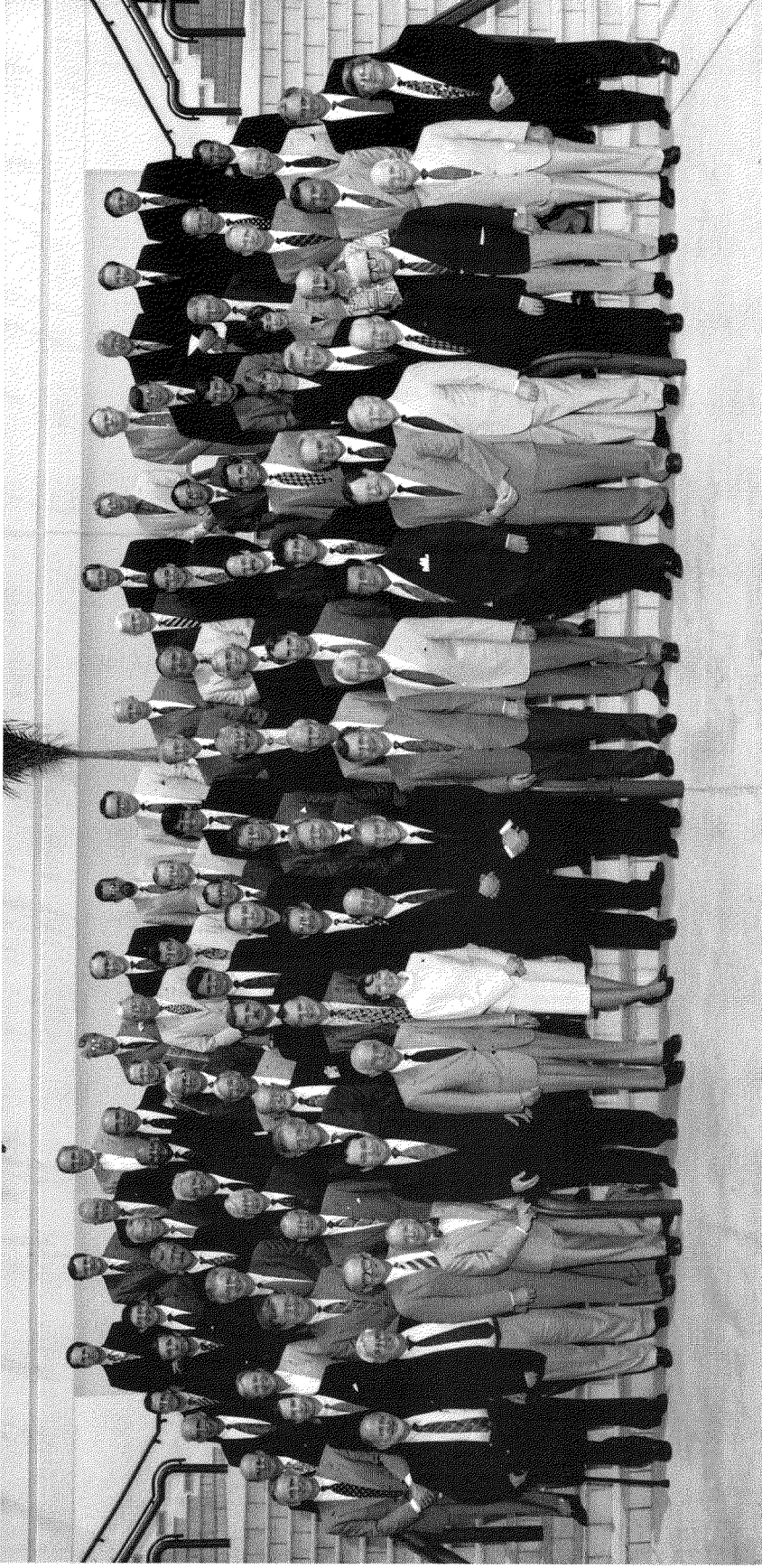
# CONTENTS

ANNUAL PHOTOGRAPH .....	vii
1995 OFFICERS .....	viii
1996 OFFICERS .....	viii
INTRODUCTION OF AWARD OF MERIT RECIPIENT D. THANE R. CODY, M.D., Ph.D. .... Mansfield F.W. Smith, M.D.	ix
RESPONSE OF AWARD OF MERIT RECIPIENT .....	ix
D. Thane R. Cody, M.D., Ph.D.	
AWARD OF MERIT RECIPIENTS 1949–1995 .....	x
GUESTS OF HONOR 1949–1995 .....	x
<b>SCIENTIFIC SESSIONS</b>	
PRESIDENTIAL ADDRESS: THE DARK SIDE OF MANAGED CARE .....	1
Robert A. Jahrsdoerfer, M.D., F.A.C.S.	
PRESENTATION OF GUEST OF HONOR: RICHARD R. GACEK, M.D., F.A.C.S. ....	3
Robert A. Jahrsdoerfer, M.D., F.A.C.S.	
REMARKS OF GUEST OF HONOR: THE PERIODICITY OF THE PROFESSIONAL CAREER .....	4
Richard R. Gacek, M.D., F.A.C.S.	
PRESIDENTIAL CITATION: EIJI YANAGISAWA, M.D. ....	6
Robert A. Jahrsdoerfer, M.D., F.A.C.S.	
1. TRANSTYMPANIC GENTAMICIN TITRATION THERAPY FOR MENIERE’S DISEASE .....	7
Loren J. Bartels, M.D. and Jonathan S. Sillman, M.D.	
2. TRANSTYMPANIC GENTAMICIN THERAPY: A NEW DOSING REGIMEN AND PROTOCOL FOR MONITORING ITS EFFECT .....	8
Mitchell K. Schwaber, M.D., Faith C. Wurm, M.S., and James W. Hall III, Ph.D.	
3. DEXAMETHASONE PERFUSION OF THE LABYRINTH PLUS INTRAVENOUS DEXAMETHASONE FOR MENIERE’S DISEASE .....	10
John J. Shea, Jr., M.D., Xianxi Ge, M.D., and Michael J. Ruckenstein, M.D.	
4. INTRATYMPANIC STEROID THERAPY IN THE TREATMENT OF SENSORINEURAL DEAFNESS IN MENIERE’S DISEASE AND AUTOIMMUNE INNER EAR DISEASE .....	12
Herbert Silverstein, M.D. and Seth I. Rosenberg, M.D.	
<b>DISCUSSION PERIOD ONE</b> .....	13
5. “FEELING WELL” OR “NOT” AFTER COMPENSATION OF UNILATERAL VESTIBULAR LOSS: IS THERE AN OBJECTIVE MEASURE? .....	15
Anthanasios Katsarkas, M.D., Henrietta Galiana, Ph.D., and Heather L. Smith, M.Eng.	

6. WHAT IS THE MINIMAL VESTIBULAR FUNCTION REQUIRED FOR COMPENSATION? . . . . .	17
F. Owen Black, M.D., Steven W. Wade, M.S., and Lewis M. Nashner, Sc.D.	
7. A COMPARISON OF HEARING RESULTS AFTER ENDOLYMPHATIC SAC DECOMPRESSION AND POSTERIOR FOSSA VESTIBULAR NEURECTOMY . . . . .	18
Seth I. Rosenberg, M.D., Herbert Silverstein, M.D., and Michael E. Hoffer, M.D.	
8. A HUMAN TEMPORAL BONE STUDY OF CHANGES IN THE BASILAR MEMBRANE OF THE APICAL TURN IN ENDOLYMPHATIC HYDROPS . . . . .	19
Benny Nageris, M.D., Joe C. Adams, Ph.D., and Saumil N. Merchant, M.D.	
<b>DISCUSSION PERIOD TWO</b> . . . . .	20
PANEL DISCUSSION—BENIGN POSITIONAL VERTIGO: NEW TREATMENTS . . . . .	22
Moderator: Jack M. Kartush, M.D.	
Panelists: Jack M. Kartush, M.D., John M. Epley, M.D., Brian W. Blakley, M.D., Ph.D., John F. Kveton, M.D., Richard R. Gacek, M.D., and Phillip F. Anthony, M.D.	
PANEL OPEN DISCUSSION . . . . .	29
9. REVISION STAPEDECTOMY WITH AND WITHOUT THE CO <sub>2</sub> LASER: AN ANALYSIS OF RESULTS . .	31
Thomas J. Haberkamp, M.D., Steven A. Harvey, M.D., and Yasser Khafagy, M.D.	
10. REPORTING OPERATIVE HEARING RESULTS IN STAPES SURGERY: DOES CHOICE OF OUTCOME MEASURE MAKE A DIFFERENCE? . . . . .	32
Karen I. Berliner, Ph.D., Karen Jo Doyle, Ph.D., M.D., and Robert A. Goldenberg, M.D.	
11. ENDOSCOPIC STAPEDECTOMY: A PRELIMINARY REPORT . . . . .	33
Muaaz Tarabichi, M.D.	
12. BIOGLASS MIDDLE EAR PROSTHESIS: LONG-TERM RESULTS . . . . .	34
Kevin R. Rust, M.D., George T. Singleton, M.D., June Wilson, Ph.D., and Patrick J. Antonelli, M.D.	
13. LONG-TERM RESULTS USING OSSICULAR GRAFTS . . . . .	35
Jay B. Farnior, M.D. and Stacy W. Nichols, M.A., CCC-A	
14. THE USE OF EVOKED POTENTIAL RECORDINGS AND STAPES DISPLACEMENT MEASUREMENTS TO EVALUATE THE IN VIVO FUNCTION OF AN IMPLANTABLE ELECTROMAGNETIC MIDDLE EAR TRANSDUCER . . . . .	36
Thomas C. Robey, B.S.E., Douglas A. Miller, B.S.E.E., Alec N. Salt, Ph.D., and John M. Fredrickson, M.D.	
<b>DISCUSSION PERIOD THREE</b> . . . . .	38
15. LASER DOPPLER VIBROMETRY (LDV): A NEW CLINICAL TOOL FOR THE OTOLOGIST . . . . .	40
Richard L. Goode, M.D.	
16. OSSEOINTEGRATION AND GROWTH EFFECTS OF TEMPORAL BONE PERCUTANEOUS PEDESTALS . . . . .	42
James L. Parkin, M.D., M.S., Roy D. Bloebaum, Ph.D., Brett D. Parkin, B.S., and Matthew J. Parkin, B.S.	
17. MAGNETIC RESONANCE IMAGING IN IDIOPATHIC SUDDEN SENSORINEURAL HEARING LOSS . . . . .	43
George A. Gates, M.D., Todd Richards, M.D., Jay Tsuruda, M.D., and Edwin W. Rubel, Ph.D.	
18. THE USE OF THE TEMPOROPARIETAL FASCIAL FLAP IN TEMPORAL BONE RECONSTRUCTION . .	44
Mack L. Cheney, M.D., Cliff A. Megerian, M.D., Mark T. Brown, M.D., Michael J. McKenna, M.D., and Joseph B. Nadol, Jr., M.D.	

19. INVASION PATTERNS OF ADVANCED TEMPORAL BONE MALIGNANCIES .....	45
John P. Leonetti, M.D., Peter G. Smith, M.D., Ph.D., G. Robert Kletzker, M.D., and Ricardo Izquierdo, M.D.	
<b>DISCUSSION PERIOD FOUR .....</b>	<b>46</b>
20. NEUROPHYSIOLOGICAL APPROACH TO TINNITUS PATIENTS .....	47
Pawel J. Jastreboff, Ph.D., Sc.D., William C. Gray, M.D., and Susan L. Gold, M.A.	
21. PATIENT PERFORMANCE WITH THE COCHLEAR CORPORATION "20+2" IMPLANT: BIPOLAR VERSUS MONOPOLAR ACTIVATION .....	48
Teresa A. Zwolan, Ph.D., Paul R. Kileny, Ph.D., Carissa Ashbaugh, M.A., and Steven A. Telian, M.D.	
22. DEFINING FUNCTIONAL LIMITATION, DISABILITY, AND SOCIETAL LIMITATIONS IN PATIENTS WITH FACIAL PARESIS: INITIAL PILOT QUESTIONNAIRE .....	49
J. Gail Neely, M.D. and Peggy S. Neufeld, M.A.	
23. THE VARIABLE RELATIONSHIP BETWEEN THE LOWER CRANIAL NERVES AND JUGULAR FORAMEN TUMORS: IMPLICATIONS FOR NEURAL PRESERVATION .....	50
Lawrence R. Lustig, M.D. and Robert K. Jackler, M.D.	
24. HEARING CONSERVATION IN SURGERY FOR GLOMUS JUGULARE TUMORS .....	52
C. Gary Jackson, M.D., David S. Haynes, M.D., Paul A. Walker, M.D., Michael E. Glasscock III, M.D., S. Storper, M.D., and Anne Forrest Josey, M.S.	
<b>DISCUSSION PERIOD FIVE .....</b>	<b>53</b>
25. THE DIAGNOSIS OF INTRA-AXIAL POSTERIOR FOSSA LESIONS .....	54
Arvind Kumar, M.D., Marlos A.G. Viana, Ph.D., and Albert Pieri, B.S.	
26. HISTOLOGIC EVALUATION OF AERATION ROUTES IN TEMPORAL BONES WITH CHOLESTEATOMA .....	56
Atsushi Haruta, M.D., Patricia A. Schachern, B.S., Tetsuya Tono, M.D., Michael M. Paparella, M.D., and Tamotsu Morimitsu, M.D.	
27. MANAGEMENT OF LABYRINTHINE FISTULAE SECONDARY TO CHOLESTEATOMA .....	58
Jacques A. Herzog, M.D., Peter G. Smith, M.D., Ph.D., G. Robert Kletzker, M.D., and Kenneth S. Maxwell, M.D.	
28. MECHANICAL VERSUS CO <sub>2</sub> LASER OCCLUSION OF THE POSTERIOR SEMICIRCULAR CANAL IN HUMANS .....	59
Patrick J. Antonelli, M.D., Larry B. Lundy, M.D., Jack M. Kartush, M.D., Don L. Burgio, M.D., and Malcolm D. Graham, M.D.	
29. DIRECT COCHLEAR NERVE ACTION POTENTIALS AS AN AID TO HEARING PRESERVATION IN MIDDLE FOSSA ACOUSTIC NEUROMA RESECTION .....	60
Joseph P. Roberson, Jr., M.D., Allen Senne, MA, Derald E. Brackmann, M.D., William E. Hitselberger, M.D., and James Saunders, M.D.	
<b>DISCUSSION PERIOD SIX .....</b>	<b>61</b>
30. IDENTIFICATION OF PHOTOACOUSTIC TRANSIENTS DURING PULSED LASER ABLATION OF THE HUMAN TEMPORAL BONE .....	63
Brian J.F. Wong, M.D., Mark Dickinson, Ph.D., Joseph Neev, Ph.D., Karen J. Doyle, M.D., Ph.D., and Michael W. Berns, Ph.D.	
31. POLYMERASE CHAIN REACTION AMPLIFICATION OF A MEASLES VIRUS SEQUENCE FROM HUMAN TEMPORAL BONE SECTIONS WITH ACTIVE OTOSCLEROSIS .....	65
Michael J. McKenna, M.D., Arthur Kristiansen, M.S., and Jonathan Haines, Ph.D.	

32. THE ROLE OF THE NEUROTROPHINS IN MATURATION AND MAINTENANCE OF POSTNATAL AUDITORY INNERVATION . . . . .	66
Hinrich Staecker, M.D., Vera Galinovic-Schwartz, M.D., Wei Liu, B.S., Philippe Lefebvre, M.D., Ph.D., Richard Kopke, M.D., Brigitte Malgrange, Ph.D., Gustave Moonen, M.D., Ph.D., and Thomas R. Van De Water, Ph.D.	
33. EXTERNAL AND MIDDLE EAR PATHOLOGY IN TGF- $\alpha$ -DEFICIENT ANIMALS . . . . .	67
Charles G. Wright, Ph.D., Karen S. Robinson, B.S., and William L. Meyerhoff, M.D., Ph.D.	
34. IMMUNOHISTOCHEMICAL FINDINGS IN THE COCHLEA OF AIDS CASES . . . . .	68
Jessica W. Lim, M.D., J. Thomas Roland, Jr., M.D., Jin S. Lim, M.D., James Lee, B.A., Bernard Ong, B.A., and Dean E. Hillman, Ph.D.	
35. EFFECT OF LEUKOTRIENE INHIBITOR ON OTOACOUSTIC EMISSIONS IN SALICYLATE OTOTOXICITY . . . . .	69
Johnny Arruda, M.D., Timothy T.K. Jung, M.D., Ph.D., and David G. McGann, M.S.	
<b>DISCUSSION PERIOD SEVEN</b> . . . . .	70
INTRODUCTION OF NEW PRESIDENT: DERALD E. BRACKMANN, M.D. . . . .	71
Robert A. Jahrsdoerfer, M.D., F.A.C.S.	
REMARKS OF NEW PRESIDENT . . . . .	71
Derald E. Brackmann, M.D.	
<b>EXECUTIVE SESSIONS</b> . . . . .	72
Business Meeting	
Reports	
Secretary-Treasurer . . . . .	72
Editor-Librarian . . . . .	74
Board of Trustees of the Research Fund . . . . .	75
American College of Surgeons . . . . .	75
COSM. . . . .	76
<i>American Journal of Otology</i> . . . . .	76
Audit Committee . . . . .	77
American Board of Otolaryngology. . . . .	77
Award of Merit Committee . . . . .	77
Nominating Committee . . . . .	78
American Academy of Otolaryngology—Head and Neck Surgery. . . . .	78
Reading of Communications. . . . .	79
In Memoriam	
David A. Dolowitz, M.D. . . . .	80
Victor Goodhill, M.D. . . . .	81
Ralph J. McQuiston, M.D. . . . .	83
Ruth E.B. Parks, B.A. . . . .	84
Kinsey M. Simonton, M.D. . . . .	85
Walter P. Work, M.D. . . . .	87
Members	
New Members. . . . .	89
Active . . . . .	94
Senior . . . . .	96
Emeritus . . . . .	97
Associate. . . . .	97
Corresponding. . . . .	98
Honorary. . . . .	98
Deceased . . . . .	98
Index	
Subject . . . . .	99
Author. . . . .	100



Starting from Bottom, Left to Right:

- Row 1: Jerry Goldstein, Jack Clemis, Robert Keim, Robert Wehrs, Jack Pulec, Robert Kohut, Julia Gulya, Derald Brackman, Robert Jahrsdoerfer, Gregory Matz, Mansfield Smith, Gary Jackson, Joseph Farmer, Donald Kamerer, Sam Kinney, James Sheehy, Eugene Derlacki, Arnold Schuring
- Row 2: Charles Luetje, Gordon Hughes, George Gates, Thane R. Cody, Peter Alberti, Aram Glorig, Gail Neely, James Olsson, Patrick Brookhouser, Robert Mathog, Charles Mangham, Mitchell Schwaber, Richard Gacek, Alexander Schleuning, Sean Althaus, Antonio DeLaCruz, Phillip Daspit
- Row 3: Raul Hinojosa, George Lesinski, Robert Ruben, James Snow, Ron Konrad, Robert Sataloff, Hill Britton, Newton Coker, Leonard Rybak, Ruediger Thalmann, Thomas Eby, Ronald Amedee, Carol Jackson, Karen Berliner, John House, Howard House
- Row 4: Herbert Silverstein, Jack Kartush, Jean-Bernard Causse, Dan Bagger-Sjoberg, Malcolm Graham, Jay Farrow, Ralph Nelson, Anthanasios Katsarkas
- Row 5: Dennis Poe, John Shea, Arvind Kumar, Frank Ritter, John Emmet, Clough Shelton, Jim Parkin, Julian Nedzelski, Jack Hough, Roger Boles, John Niparko, Richard Miyamoto, Bruce Gantz, Dudley Weider, Owen Black, Joel Goebel
- Row 6: John Dickins, Paul Lambert, Paul Ward, Harold Pillsbury, Shokri Radpour, Cecil Hart, Edwin Monsell, Mohamed Hamid, Anthony Maniglia, Stephen Harner, Thomas McDonald, William Meyerhoff, Richard Babin, Richard Goode, James Pappas, John Frederickson, Edward Applebaum, Jeffrey Harris

AMERICAN OTOLOGICAL SOCIETY, INC.

1995 OFFICERS

PRESIDENT

ROBERT A. JAHRSDOERFER, M.D.

VICE-PRESIDENT (PRESIDENT-ELECT)

DERALD E. BRACKMANN, M.D.

SECRETARY-TREASURER

GREGORY J. MATZ, M.D.

EDITOR-LIBRARIAN

JOSEPH C. FARMER, JR., M.D.

COUNCIL

The above officers and

MANSFIELD F.W. SMITH, M.D.

ROBERT I. KOHUT, M.D.

A. JULIANNA GULYA, M.D.

(also Editor-Librarian Elect)

C. GARY JACKSON, M.D.

1996 OFFICERS

PRESIDENT

DERALD E. BRACKMANN, M.D.

VICE-PRESIDENT (PRESIDENT-ELECT)

JOSEPH C. FARMER, JR., M.D.

SECRETARY-TREASURER

GREGORY J. MATZ, M.D.

EDITOR-LIBRARIAN

A. JULIANNA GULYA, M.D.

COUNCIL

The above officers and

ROBERT I. KOHUT, M.D.

ROBERT A. JAHRSDOERFER, M.D.

C. GARY JACKSON, M.D.

CHARLES M. LUETJE, M.D.



## INTRODUCTION OF AWARD OF MERIT RECIPIENT D. THANE R. CODY, M.D., Ph.D.

*Mansfield F. W. Smith, M.D.*

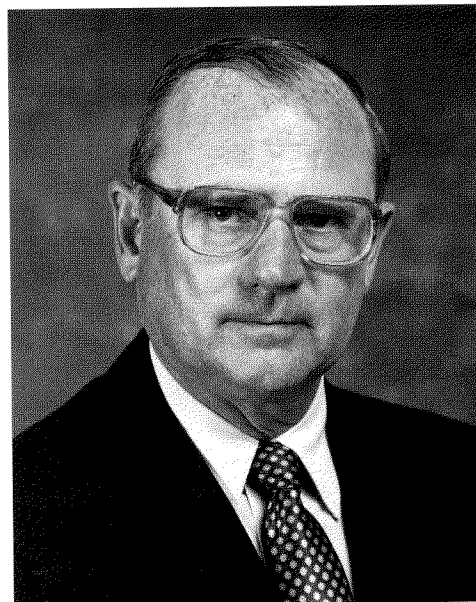
It is a great honor for me to introduce the Award of Merit recipient. By now, you recognize from the photograph, Douglas Thane Romney Cody. Born in Saint John, New Brunswick, Canada, it is our good fortune that he became a United States citizen. After receiving an M.D. and a C.M. at Dalhousie University, and a Ph.D. in Otolaryngology and Physiology from the University of Minnesota, he distinguished himself as Chairman of the Department of Otolaryngology at the Mayo Clinic, which he developed into one of the finest training programs in the world.

His service to this Society, as member and Chairman of the Board of Trustees of the Research Fund, Secretary-Treasurer, and President, was recognized as he was our Guest of Honor in 1993.

His activities in research and teaching are evidenced by nearly 200 scientific publications, including two books.

But these accomplishments pale in comparison to his good fortune to have married Joanne Dae Gerow, with whom he has had two wonderful children, Thane and Romney Joanne.

It is with great pleasure that I present the Award of Merit to Thane Cody, a close friend and a person whom I hold in the highest regard.



Douglas Thane Romney Cody, M.D., Ph.D.

## RESPONSE OF AWARD OF MERIT RECIPIENT

*D. Thane R. Cody, M.D., Ph.D.*

I sincerely thank the Society for this most appreciated, and most unexpected, honor.

## AWARD OF MERIT RECIPIENTS 1949–1995

- |  |                                    |
|--|------------------------------------|
| 1949 George M. Coates, M.D.                                | 1975 Catherine A. Smith, Ph.D.     |
| 1951 Barry J. Anson, Ph.D., and<br>Theodore H. Bast, Ph.D. | 1976 Harry Rosenwasser, M.D.       |
| 1952 Edmund P. Fowler, M.D.                                | 1977 Frank D. Lathrop, M.D.        |
| 1953 Julius Lempert, M.D.                                  | 1978 Juergen Tonndorf, M.D.        |
| 1954 Stacy R. Guild, M.D.                                  | 1979 John E. Bordley, M.D.         |
| 1957 Georg von Békésy, Ph.D.                               | 1980 Ben H. Senturia, M.D.         |
| 1959 E. Glen Wever, Ph.D.                                  | 1981 J. Brown Farrior, M.D.        |
| 1960 Hallowell Davis, M.D.                                 | 1982 William F. House, M.D.        |
| 1961 John R. Lindsay, M.D.                                 | 1983 Victor Goodhill, M.D.         |
| 1962 William J. McNally, M.D.                              | 1984 Harold F. Schuknecht, M.D.    |
| 1965 Anderson C. Hilding, M.D.                             | 1985 Wesley H. Bradley, M.D.       |
| 1966 Gordon D. Hoople, M.D.                                | 1986 John J. Shea Jr., M.D.        |
| 1967 Merle Lawrence, Ph.D.                                 | 1987 Jack V. Hough, M.D.           |
| 1968 Lawrence R. Boies, M.D.                               | 1988 George T. Nager, M.D.         |
| 1969 Sir Terence Cawthorne                                 | 1989 Brian F. McCabe, M.D.         |
| 1970 Senator Joseph Sullivan, M.B.                         | 1990 Eugene L. Derlacki, M.D.      |
| 1971 Samuel Rosen, M.D.                                    | 1991 Richard R. Gacek, M.D.        |
| 1972 Howard P. House, M.D.                                 | 1992 James L. Sheehy, M.D.         |
| 1973 Moses H. Lurie, M.D.                                  | 1993 James A. Donaldson, M.D.      |
| 1974 George E. Shambaugh Jr., M.D.                         | 1994 Fred H. Linthicum, Jr., M.D.  |
|  | 1995 D. Thane R. Cody, M.D., Ph.D. |

## GUESTS OF HONOR 1949–1995

- |                                    |                                     |
|------------------------------------|-------------------------------------|
| 1949 Harris P. Mosher, M.D.        | 1975 John E. Bordley, M.D.          |
| 1950 D. Harold Walker, M.D.        | 1976 Ben H. Senturia, M.D.          |
| 1951 John Mackenzie Brown, M.D.    | 1977 Henry B. Perlman, M.D.         |
| 1952 Edmund P. Fowler, M.D.        | 1978 Howard P. House, M.D.          |
| 1953 H. I. Lillie, M.D.            | 1979 Hallowell Davis, M.D.          |
| 1956 Stacy R. Guild, Ph.D.         | 1980 Victor Goodhill, M.D.          |
| 1958 Ralph A. Fenton, M.D.         | 1981 Harold F. Schuknecht, M.D.     |
| 1961 Julius Lempert, M.D.          | 1982 George E. Shambaugh Jr., M.D.  |
| 1962 Philip Meltzer, M.D.          | 1983 Wesley H. Bradley, M.D.        |
| 1963 William J. McNally, M.D.      | 1984 Brown Farrior, M.D.            |
| 1964 Kenneth M. Day, M.D.          | 1985 Bruce Proctor, M.D.            |
| 1965 Senator Joseph Sullivan, M.B. | 1986 Merle Lawrence, Ph.D.          |
| 1966 Dean M. Lierle, M.D.          | 1987 Robert M. Seyfarth, Ph.D.      |
| 1967 Lawrence R. Boies, M.D.       | 1988 G. Dekle Taylor, M.D.          |
| 1968 Sir Terence Cawthorne         | 1989 Eugene L. Derlacki, M.D.       |
| 1969 Gordon D. Hoople, M.D.        | 1990 William F. House, M.D.         |
| 1970 John R. Lindsay, M.D.         | 1991 Michael E. Glasscock III, M.D. |
| 1971 E. Glen Wever, Ph.D.          | 1992 William E. Hitselberger, M.D.  |
| 1972 Frank D. Lathrop, M.D.        | 1993 D. Thane R. Cody, M.D., Ph.D.  |
| 1973 Moses H. Lurie, M.D.          | 1994 Cesar Fernandez, M.D.          |
| 1974 Harry Rosenwasser, M.D.       | 1995 Richard R. Gacek, M.D.         |

# SCIENTIFIC SESSIONS

## 1995 PRESIDENTIAL ADDRESS

### THE DARK SIDE OF MANAGED CARE

*Robert A. Jahrsdoerfer, M.D., F.A.C.S.*

Earlier this month there appeared in the *Houston Chronicle* an opinion article stating that "HMOs and similar health care networks are proving that managed care systems are providing quality care at affordable rates" (1). The author further stated that "the common bond among this country's 545 HMOs is their determination to deliver quality care at cost-conscious rates, an objective achieved in almost epic proportions." There is no question that managed care is sweeping the country. There is considerable concern, however, that quality of care is lagging behind.

Managed care enterprises, typically health care insurers, function by assimilating networks of physicians and contracting with other providers and blocks of patients to deliver services at a discounted rate. The primary care physician, really a family practitioner, internist, or pediatrician, often functions as a gatekeeper, limiting the patient's access to specialty physicians, restricting the number and degree of diagnostic tests and curtailing the use of hospitals and other paramedical services. To keep the patient within the system, the primary care physician may be offered monetary incentives. These may take the form of withholdings or bonuses which reward the primary care physician at year end for coming in under budget, or fiscally punish him for exceeding a quota. In this milieu, the playing field is fertile for a physician conflict of interest.

A physician has a fundamental obligation, indeed a fiduciary responsibility, to act as the patient's advocate. This covenant of trust dictates that if a patient's interests are being denied, it is incumbent upon the physician to disclose the reasons why (2). Often, this may run counter to the contract between physician and employer in which the physician may be forbidden to inform patients of these referral limitations. Abrogating this gag order may effectively cost the physician his/her job.

From my vantage point, I see that quality of care is being eroded. I recognize that the word "quality" is not easily definable as it pertains to medicine. Outcome studies comparing health care rendered by primary physicians, vis-a-vis the specialist, have been

inconclusive. Similarly, other studies have tried to measure health outcomes of managed care groups versus fee-for-service arrangements, again with mixed results (3). While these studies suggest that the patient may be at increased risk of harm in managed care settings, the evidence has been largely anecdotal. But how can it be otherwise? Although there is a call for better documentation of the benefits of specialty care, the means to measure this outcome are crude. One can hardly expect a managed care entity to commission a study comparing a patient's outcome within the plan to treatment rendered by a specialist outside the plan, particularly when the results may be antithetical to the company's mission.

It is my contention that quality of care has taken a hit under managed care. I believe that perverse financial incentives unduly influence the primary care physician to retain the patient too long. By doing so, the primary care physician assumes the role of a marginal specialist.

Over the past four months, I have been made aware of six cases in which the level of care has been unacceptable. In three cases, the physical condition of the patient had deteriorated to a point where specialty care was sought outside the plan. In the remaining cases, the patients were kept within the managed care plan for an excessive period of time before being referred to a specialist. In each case the outcome was adversely affected by these actions.

#### CASE REPORTS

**Case 1.** 11 year old male with severe headache, neckache, loss of memory, and draining ear.

PCP diagnosis: viral bilateral ear infection.

Specialist diagnosis: papilledema and brain abscess. MRI: 6 cm posterior fossa mass (astrocytoma).

Referral?: yes, after two months

**Case 2.** 9 year old male with a bulging eye and orbital cellulitis. PCP diagnosis: viral upper respiratory infection.

**Specialist diagnosis:** orbital abscess, acute ethmoiditis.

Referral?: no.

**Case 3.** 35 year old female with severe otalgia and facial paralysis.

PCP diagnosis: external otitis.

Specialist diagnosis: Ramsey-Hunt syndrome.

Referral?: no.

**Case 4.** 59 year old female with CSF rhinorrhea for 2½ years.

PCP diagnosis: post-surgical CSF leak

Specialist diagnosis: CSF otorrhea through bony defect in the posterior petrous ridge.

Referral?: yes, to neurotologist after 6 months.

**Case 5.** 60 year old female with a personality change for 8 years.

PCP diagnosis: personality disorder.

Specialist diagnosis: MRI showed a 9 cm frontal lobe meningioma.

Referral?: yes, after MRI showed the tumor.

**Case 6.** 34 year old female with a history of decreased vision and hearing loss. Headaches for one year and recent ataxia and facial palsy.

PCP diagnosis: ?

Specialist diagnosis: MRI showed a 6 cm cerebellopontine angle tumor

Referral?: no.

I perceive that quality of care in managed care plans is on the decline. To document the extent of the problem, I propose an on-line network to solicit information from otolaryngologists to determine the scope of the problem. Part of the solution lies in abolishing fiscal incentives that restrain the primary care physician from the timely referral of patients to specialists. Whether or not this is accomplished through public opinion, or legislation, or lawsuits, is immaterial. It is imperative that we remove our patients from harm's way. Only then can we restore their confidence in our system of medical care.

## REFERENCES

1. Schauerte CM. Must not sabotage the HMOs' successes. *The Houston Chronicle*: Outlook April 9, 1995, sec. C.
2. Pelligrino, ED. Words can hurt you: some reflections on the metaphors of managed care. *JABFP* 194;7:505-10.
3. Council on Ethical and Judicial Affairs, American Medical Association. Ethical issues in managed care. *JAMA* 1995;273:330-5.

## PRESENTATION OF GUEST OF HONOR RICHARD R. GACEK, M.D., F.A.C.S.

*Robert A. Jahrsdoerfer, M.D., F.A.C.S.*

I have known Dick Gacek for most of his medical career. He has achieved a level of success matched by few others. He served as President of this Society, President of the American Neurotology Society, and President of the Triological Society. His contributions to otology are the standards by which all others are measured. He has pioneered singular neurectomy surgery for the control of benign posi-

tional vertigo. He has shown us how to plan diagnostic and surgical interventions with astute laboratory investigation. More than anything else, however, you can believe what Dick Gacek says and I learned that long time ago. I am proud to have him as a friend. He is this year's American Otologic Society Guest of Honor and I can think of no one more deserving of this honor.

# REMARKS OF GUEST OF HONOR

## THE PERIODICITY OF THE PROFESSIONAL CAREER

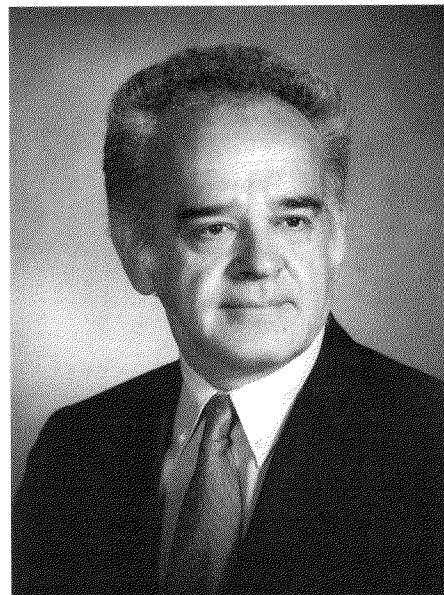
*Richard R. Gacek, M.D., F.A.C.S.*

I am deeply honored and appreciative of this special recognition by the American Otological Society—the world's oldest and most prestigious body devoted to the clinical and scientific advancement of ear disorders. I wish to thank President Jahrsdorfer and the Council, in particular, for nominating me for this honor.

As Guest of Honor, I should like to make a few remarks regarding the role of scientific contribution in the development of the professional career.

The induction this morning of new members into this Society is a reminder that the high standards of the Society are maintained by young otologists who have made significant contributions to otology and show promise of further contributions to the field.

Sir William Osler remarked that a physician's career was composed of two "fixed periods." The first period covers the years from 25 to 40 (or thereabouts) when one has the creative energy and time to make a mark on the field and establish a reputation upon which to further grow in stature. The substantive nature of these early contributions depends heavily on the time and interest invested in preparation for clinical and research investigation. Cultivation of the young physician's career development is also influenced by the environment where the role model of a proven teacher/investigator is vital to the maturation of the young investigator. Important also is protected time and support with resources in a busy clinical program to permit serious creative investigative activity. Given this setting, the young specialist has only to apply himself/herself in the preparation of an investigative career to be successful. The fruits of this research effort must be realized early, before the growth of time commitments to committees, clinical practice, and other administrative responsibilities which will detract from serious research efforts. I believe the extraordinary sacrifice made during these years, clinical and research fellowships, are vital to the initiation, development and durability of these scientific contributions.



Richard R. Gacek, M.D., F.A.C.S.

The second fixed period in one's career, whether it be as a program director, laboratory chief, academician or clinician, traditionally comes to an end at about age 65 years. Frequently, administrative burdens and national society activities create a drain on the energy and time available for continued scientific contributions during this period. Unless a concerted effort is made to control the time spent in accepting responsibilities which are not mandatory, the opportunity to contribute scientifically is eventually lost during this period. Vital to continued meaningful contributions during this harried period is the formation of close collaborative ties with other senior and junior investigators in the basic science disciplines.

These two fixed periods are followed by a third, or "extended period," one which is more flexible and is unlimited in time and scope. It is a period for aspiration rather than retirement, unencumbered by administrative burdens and committee meetings. At this stage one can continue clinical,

surgical and research activities, if curiosity remains and abilities permit, and write creatively, culling together a lifetime of experience. To a large extent the interest and the energy to pursue such activities during this extended period depends upon groundwork laid down early in a career by

acquiring fundamental research and clinical skills. This can and should be the most fruitful period professionally of one's career.

I wish to again thank the Council and the Society for this most unexpected and appreciated opportunity to be your Guest of Honor.

## PRESIDENTIAL CITATION EIJY YANAGISAWA, M.D.

*Robert A. Jahrsdoerfer, M.D., F.A.C.S.*

This year I have chosen to present the Presidential Citation to Dr. Eiji Yanagisawa. Dr. Yanagisawa is not a member of this Society, but he is very well known in our field and I can tell you, from personal knowledge, he is an excellent ear surgeon.

He launched my career in general ear surgery. I will tell you one brief story. When I was chief resident, a patient came in with a unilateral aural atresia that I wanted to do, but the then attending otologist on service refused to allow me to do it because he said "we do not do unilateral atresias." I asked, "Would you have any objections if I got someone else to staff it?" He said, "No," so I then asked Dr. Yanagisawa. Little did I know that I got him into trouble by doing that. However, he persevered and the patient was operated and we found

a cholesteatoma, so there was another reason why she needed the operation. But I remember him well for his willingness to stick his neck out for me and I very much appreciate that. All those who have subsequently gone through the residency training program will remember Eiji as a very fine, innovative educator. So, Eiji, if you would please come up and accept your certificate.

[Reading from certificate] "The American Otolological Society presents this Presidential Citation to Dr. Eiji Yanagisawa in acknowledgment of his primary pioneering contribution as an educator, clinical scientist, and innovator. This distinguished physician's service is recognized as a consummate mentor in Otolaryngology-Head and Neck Surgery."

There are no truer words spoken.

## REPLY

*Eiji Yanagisawa, M.D.*

Dr. Jahrsdoerfer, thank you very much for this most unanticipated Presidential Citation.



# TRANSTYMPANIC GENTAMICIN TITRATION THERAPY FOR MENIERE'S DISEASE

*Loren J. Bartels and Jonathan S. Sillman*

## **ABSTRACT**

While transtympanic gentamicin therapy has been well established in Germany, its utilization in the USA has not achieved widespread acceptance. In recent reports, multiple injections through a tympanostomy catheter stop the vertigo spells in 80% of patients, with significant incidence of further hearing loss and a 10% rate of profound hearing loss. In contrast, Magnuson demonstrated that a single transtympanic gentamicin treatment was quite effective. We elected to treat a series of Meniere's disease patients with transtympanic gentamicin until either subjective evidence of vestibular dysfunction or significant drop in pure tone thresholds occurred. The treatment protocol was stretched over three to seventeen days to allow for delay in appearance of vestibular toxicity. We present results with a low incidence of cochlear toxicity and a high rate of control of episodic vertigo. One patient eventually required a labyrinthectomy. As with surgical approaches, persistent disequilibrium troubles some patients. We postulate that some patients will fail transtympanic gentamicin therapy because of a high jugular bulb, a strong mucosal fold in the round window niche or fibrosis that limits access to the round window membrane. Overall, the Meniere's vertigo control rate equals vestibular neurectomy results and approaches labyrinthectomy results.

---

Department of Surgery, University of South Florida, MDC-16, 12901 Bruce B. Downs Boulevard, Tampa, Florida 33612.

## TRANSTYMPANIC GENTAMICIN THERAPY: A NEW DOSING REGIMEN AND PROTOCOL FOR MONITORING ITS EFFECT

*Mitchell K. Schwaber, Faith C. Wurm, and James W. Hall III*

### **ABSTRACT**

Transtympanic instillation of gentamicin has been increasingly used as a treatment for the incapacitating vertigo associated with unilateral Meniere's disease. The purpose of this study is to report the results of a new dosing regimen, specifically instilling the gentamicin twice weekly. In addition, we report the results of our investigation using rotational testing as a means of monitoring the efficacy of this therapy. Ten patients with a history of recurring, incapacitating vertigo consistent with unilateral Meniere's disease were treated with transtympanic administration of gentamicin. All patients were seen on an outpatient basis. Three patients were treated with 40 mg/cc of gentamicin buffered with sodium bicarbonate to a pH of 7.4 and diluted to 30 mg/cc, three times a day for four consecutive days. At each instillation, 0.5 cc of the gentamicin preparation was injected through an anesthetized tympanic membrane into the middle ear space with a 25 gauge spinal needle. Due to a high incidence of sensorineural hearing loss with this dose regimen, the protocol was changed to the same preparation administered once daily for two consecutive days. This regimen was repeated the next week if no effects were seen with the initial treatment. With this more conservative protocol, patients experienced no change in hearing sensitivity. Although caloric responses are often used to determine the effects of gentamicin therapy, there are several advantages of using rotational testing for monitoring vestibular function. These include a more controlled stimulus which enables the detection of small changes within the VOR, as well as the ability to monitor vestibular compensation. A second major advantage is that the perforated tympanic membrane is not exposed to caloric irrigation. Vestibular response to rotational stimulation was assessed with rotational chair testing and the Vestibular Autorotation Test (VAT) within four hours prior to initial dose, one week following gentamicin treatment and two months following treatment. Phase, gain and asymmetry measures were obtained from rotational chair testing at 0.01, 0.02, 0.04, 0.08, 0.16, 0.32, and 0.64 Hz. The horizontal and vertical VORs were tested with the VAT at 2 to 6 Hz. Response parameters

calculated were phase and gain. Binaural bithermal caloric testing was used as a unilateral measurement of labyrinthine function prior to treatment and two months following gentamicin instillation. The presence of spontaneous and/or positional nystagmus was also noted. The Dizziness Handicap Inventory (DHI) was administered pre-treatment and two months post-treatment to quantify the outcome of the gentamicin treatment. The rotary chair test was most sensitive to the effects of vestibular disturbance following gentamicin treatment in all patients. With the exception of one patient, no significant difference was observed on the VAT during serial testing. Post-treatment, subjects presented with an asymmetry and abnormal phase on the rotary chair, consistent with an acute peripheral lesion. At two months post-treatment, asymmetry decreased, reflecting central compensation. Electronystagmography documented decreased vestibular function with absent or decreased response to caloric stimulation. All subjects reported vertigo spells decreased or abolished, although most complained of persistent disequilibrium following therapy.

---

Vanderbilt University Medical Center, S-2100 Medical Center North, Nashville, Tennessee 37232-2559.

## DEXAMETHASONE PERFUSION OF THE LABYRINTH PLUS INTRAVENOUS DEXAMETHASONE FOR MENIERE'S DISEASE

*John J. Shea, Jr., Xianxi Ge, and Michael J. Ruckenstein*

### ABSTRACT

**Objective:** To improve the hearing, reduce the fullness and low-frequency tinnitus and stop the dizzy spells in patients with Meniere's disease.

**Design:** A protocol was created to confirm the diagnosis of Meniere's disease in 28 patients, some in stage I Meniere's disease, without dizzy spells, but most in stages II and III, with dizzy spells, in which the ears of these patients were perfused through the round window with dexamethasone while receiving intravenous dexamethasone.

**Patients and Setting:** All those in this report were the private patients of the senior author, treated at the SHEA CLINIC in Memphis, Tennessee.

**Interventions:** Dexamethasone perfusion of the labyrinth plus intravenous dexamethasone was done on all patients.

**Main Outcome Measures:** Careful history was taken, plus complete hearing test, ice water caloric test, sinusoidal harmonic acceleration test and trans-tympanic electrocochleography were all performed before and after dexamethasone perfusion plus intravenous dexamethasone, to confirm the diagnosis of Meniere's disease, and observe the response to dexamethasone perfusion plus intravenous dexamethasone after operation.

**Results:** Results were generally very good, with improvement of hearing in 42.9%, reduction in low-frequency tinnitus in 85.7% and fullness in 92.9%, and relief from dizzy spells, when present, in 100%. The hearing in no patient was made worse.

**Conclusions:** It has already been demonstrated by Shea Jr. and Ge that streptomycin perfusion of the labyrinth through the round window plus intravenous streptomycin is of great value in the treatment of the dizzy spells, fullness, low-frequency tinnitus and hearing loss of Meniere's disease. Just how and where dexamethasone works on the inner ear in Meniere's disease is not known, but it must work mostly on the stria vascularis and the endolymphatic sac, to reduce the hydrops, and in so doing, improve the hearing, reduce the fullness, low-frequency tinnitus, and stop the dizzy spells when present. This is an ideal

operation, easy to perform, safe, effective, and does not make the hearing worse. The question is how long will these initial good results last, will they be permanent? Perfusion of the labyrinth, with other drugs than streptomycin and dexamethasone, through the round window plus the same drug intravenously, has great potential for the treatment of other diseases than Meniere's disease, and to determine the size and test the function of the endolymphatic sac as well. This report demonstrates that dexamethasone perfusion of the labyrinth plus intravenous dexamethasone in Meniere's disease improves the hearing, reduces the fullness, low-frequency tinnitus and dizzy spells, when present and does not make the hearing worse.

---

Shea Clinic, 6133 Poplar Pike, Memphis, Tennessee 38119.

# INTRATYMPANIC STEROID THERAPY IN THE TREATMENT OF SENSORINEURAL DEAFNESS IN MENIERE'S DISEASE AND AUTOIMMUNE INNER EAR DISEASE

*Herbert Silverstein and Seth I. Rosenberg*

## **ABSTRACT**

It is well accepted that prednisone (20 mg TID), taken systemically, will produce an improvement in cochlear function in some cases of unilateral or bilateral Meniere's disease, autoimmune inner ear disease and sudden deafness. Two methods of topical application have been devised to directly treat the cochlea with steroids applied to the round window membrane, thus avoiding the systemic effects of steroids. The first method is to inject 0.3 cc methylprednisolone suspension (80 mg/cc) or 0.3 cc methylprednisolone sodium succinate (40 mg/cc) through the posterior inferior portion of the tympanic membrane using a tuberculin syringe (27 gauge needle). The second method is to place a Meroceel wick into the round window niche through a myringotomy incision. The patient then uses Decadron ophthalmic solution (0.1 cc) as ear drops, three times a day for two weeks. Thirteen patients have been treated with these two methods. Dramatic improvements in hearing occurred in four patients, moderate improvement in one patient and no effect was observed in eight patients. Vertigo attacks were not improved. No patient suffered ill effects from the treatment and the acceptance was high. Preliminary animal experiments will be presented showing the effects of topical steroids on inner ear function. Detailed techniques and results will be presented of a larger series of patients.

---

Ear Research Foundation, 1901 Floyd Street, Sarasota, Florida 34239-2909.

## DISCUSSION PERIOD ONE

### Papers 1–4

**Dr. Ruben** (New York, New York): Thank you very much Dr. Jahrsdoerfer. I appreciated these papers, which presented interesting observations; however, there are a number of substantive problems. Firstly, one must recognize the heterogeneity of disease in different biological states. Some of us consider Meniere's disease a syndrome, caused by many different things. Secondly, the total lack of any control, placebo, no treatment, or "other" treatment groups for comparison. I feel that perhaps we have some observations, and medicine does progress by clinical observations. The next stage, which Dr. Silverstein so nicely indicated, is a double-blind, cross-over study, or something like that, which is really essential.

**Dr. Jahrsdoerfer** (Houston, Texas): For the purpose of recording the discussion I ask the speakers to give their names and locations. First microphone, please.

**Dr. Anthanasios Katzarkas** (Montreal, Canada): I would like to make a comment on the first two papers. It has been described in the literature for many years now that the ototoxic effects of phytotoxic agents continue for a long time after administration has been discontinued. Therefore, I believe that neither subjective nor objective symptoms are appropriate indicators for determining the dosage required by a particular individual.

**Dr. Jahrsdoerfer** (Houston, Texas): Thank you—second microphone.

**Dr. William L. Meyerhoff** (Dallas, Texas): I have an anecdotal caveat in support of the papers from Nashville and Florida. Over the past year I have seen four patients with ataxic gait disturbance who have had prophylactic gentamicin ophthalmic drops used following tympanostomy tube insertion. All four patients had no vestibular response to caloric stimulation in the ear(s) subjected to that treatment.

**Dr. Jahrsdoerfer** (Houston, Texas): Thank you Bill—next, please.

**Dr. Robert A. Dobie** (San Antonio, Texas): I wanted to comment on the first two papers that discussed non-vertiginous disequilibrium after treatment and ask the authors whether these people had

clear-cut rotatory vertigo before treatment. My observation has been that many patients who do not give a history of vertigo and deny rotational vertigo, will, after an ENG say "you know, that was exactly like my spells," suggesting to me that patients often have a hard time describing vertigo.

**Dr. Jahrsdoerfer** (Houston, Texas): Thank you. Dr. Coker.

**Dr. Newton J. Coker** (Houston, Texas): Regarding the first paper by Drs. Bartels and Sillman, I seek clarification on two points. The authors report that of 29 patients, 38% sustained hearing loss and in 7% the hearing loss was profound. Could they please clarify the degree of hearing loss in that 38%? Also, in their conclusion they stated that this figure is comparable to the results seen after vestibular neurectomy. I would like to have more information on that because I do not recall the incidence of hearing loss reaching 38% following vestibular neurectomy.

**Dr. Jahrsdoerfer** (Houston, Texas): Dr. Hamid.

**Dr. Mohamed A. Hamid** (Cleveland, Ohio): The second paper presented some shared results after ototoxic medications and showed decrease of the high frequency rotary chair response, as opposed to the low frequency response. Also, it was stated that the caloric response was terminated in the sense that it disappeared. I find this interesting and I would like to hear some comments on that.

**Dr. Jahrsdoerfer** (Houston, Texas): Any comments from the audience? The speakers respond please.

**Dr. Sillman** (Tampa, Florida): To Dr. Coker's questions: by profound hearing loss we meant a pure tone average worse than 90 decibels with speech discrimination worse than 25%. The incidence of hearing loss subsequent to nerve section has been reported to be 28% by Dr. McElveen and 38% by Dr. Brackmann and colleagues. Our series reported an incidence of hearing loss in the range of 20–40%; with these sorts of numbers we would consider that there is no statistical difference between our hearing loss rate and others reported, especially as other gentamicin series have reported even lower hearing loss rates.

I agree with Dr. Katsarkas, that using subjective criteria to determine when to stop treatment probably is not adequate.

In response to Dr. Dobie's question, the patients in our series that reported episodic disequilibrium all had true rotatory vertigo prior to treatment, and the post-treatment episodes were clearly different from the vertigo they had before treatment.

**Dr. Newton J. Coker** (Houston, Texas): I have one quick question. How are we defining the degree of hearing loss? Are we talking about pure tone averages, discrimination, and at what frequencies?

**Dr. Sillman** (Tampa, Florida): We defined hearing loss as a ten decibel or greater loss of pure tone average or 15% or more drop in speech discrimination. We used the frequencies between 500 and 2,000 Hz.

**Dr. Jahrsdoerfer** (Houston, Texas): Dr. Schwaber, would you like to respond?

**Dr. Schwaber** (Nashville, Tennessee): I have just a couple of comments. Firstly, we have treated a group of patients using Dr. Julian Nedzelski's protocol, consisting of two injections a day for four

days in a row; we found a very high incidence of sensorineural loss with that regimen. By cutting back to the once or twice a week regimen, I think there is less hearing loss, perhaps because of the reduced exposure to gentamicin. In answer to one of the other questions, our patients indeed did have rotary vertigo, which was one of the criteria for treatment. Their vertigo subsequently changed to this disequilibrium or whatever that "pulling sensation" that they described represents, after treatment.

In answer to Dr. Hamid, our data showed that it was the low frequency rotary chair response that demonstrated the shift in symmetry. One would think that it would be the high frequency response that would show this change after gentamicin, because gentamicin knocks out the high frequency fibers more selectively. I have been told that the explanation for this apparently paradoxical finding is that the rotary chair is a much more sensitive and accurate test for the low frequencies.

**Dr. Jahrsdoerfer** (Houston, Texas): Any other comments or questions? We will go on to the next paper.



## "FEELING WELL" OR "NOT" AFTER COMPENSATION OF UNILATERAL VESTIBULAR LOSS: IS THERE AN OBJECTIVE MEASURE?

*Anthanasios Katsarkas, Henrietta Galiana, and Heather L. Smith*

### ABSTRACT

Acute loss of unilateral peripheral vestibular function induces vertigo, loss of postural control, nausea and vomiting. These symptoms slowly subside due to central compensation. In the compensated patient, however, two questions are of clinical relevance: 1) Are there any permanent functional deficits? 2) Can how "well" the patient feels be quantified? In a recent series of experiments, normal subjects ( $n = 9$ ) and compensated patients ( $n = 14$ ) were exposed to passive sinusoidal head oscillations. All experiments were conducted in total darkness at 1/6 Hz, with protocols (52 s duration) of increasing velocity. The estimated parameters of the vestibulo-ocular reflex (VOR) were the gain (nystagmus slow phase velocity/head velocity), the phase shift (eye vs head velocity), the offset (bias or mean deviation of nystagmus slow phase velocity from zero) and the significance of non-linearities (using a polynomial [cubic] fit) by calculating an asymmetry index. The gain (mean value) was not statistically different in normal subjects vs patients taken as a group. The bias, never equal to any spontaneous nystagmus, showed a tendency to be larger in patients than in normal subjects, although there was a substantial overlap between the two groups. At low head velocities (up to 900/s), the bias was located on either side (healthy or lesioned), depending on the patient. At the peak head velocity (1800/s), however, the bias was invariably located on the lesioned side (Katsarkas A, Galiana H, Smith H: *Acta Otolaryngol* 1994 [submitted]). Phase shifts in patients were always greater than in normals but there was no difference associated with the side of the lesion. The asymmetry index reflected always the side of the lesion and distinguished normals from some patients. Thus these parameters, taken separately, distinguished frequently normals from patients but such distinction was not possible in all cases. Normals had small indexes of asymmetry, for instance, but there were also patients with similar indexes. Individual parameters could not be correlated with "well being" of patients either. When all three parameters (phase shift, bias, asymmetry index), however, were considered in the three-dimensional domain, patients could always be

identified from normals and the side (right vs left) of the peripheral lesion was consistently detected. When correlated with the clinical profile of "well being," it would appear that patients feeling well were located closer to the group of normals in the three-dimensional domain. It is concluded that: 1) large biases and/or phase shifts were more easily tolerated than VOR non-linearities; 2) low asymmetry index of the VOR was the most sensitive individual measure of functional vestibular compensation and the best predictor of functional recovery; 3) when all three parameters were considered together, it appeared that they could distinguish the patients who felt well vs the patients who did not.

---

Royal Victoria Hospital, E4.48, Montreal, Quebec H3A 1A1, Canada.

## WHAT IS THE MINIMAL VESTIBULAR FUNCTION REQUIRED FOR COMPENSATION?

*F. Owen Black, Steven W. Wade, and Lewis M. Nashner*

### **ABSTRACT**

Living with an uncompensated, abnormal vestibular system requires oppressive modification of life style and often prevents return to work and activities of daily living. Patients with vestibular abnormalities were studied to determine the minimal residual vestibular function required to achieve compensation. Three groups of patients with (a) complete unilateral loss of vestibular function with normal horizontal canal-vestibulo-ocular (HCVOR) function in the opposite ear, (b) complete unilateral loss with abnormal HCVOR function in the opposite ear, and (c) bilateral reduction of vestibular function from aminoglycoside toxicity underwent vestibulo-ocular (VOR), optokinetic (OKN), visual-VOR (VVOR), and computerized dynamic posturography (CDP) tests before and after therapeutic procedures. Results suggest that a minimal VOR response amplitude must be present for compensation of VVOR function to occur. The roles of VOR and OKN phase shifts in vestibular compensation are more complicated and require further study. Compensation of vestibulospinal function does not necessarily accompany VOR or VVOR compensation. Ascending and descending vestibular compensatory mechanisms may involve different spatial sensory inputs. Results of these studies have important implications for the diagnosis and treatment of patients with vestibular disorders, including selection and monitoring of patients for therapeutic regimens such as vestibular nerve section and streptomycin therapy.

---

Neurotology Research, Portland, Oregon, U.S.A.

Presented at COSM.

Reprint requests: Dr. F.O. Black, Neurotology Research NO10, 1040 N.W. 22nd Avenue, Portland, Oregon 97210, U.S.A.

## A COMPARISON OF HEARING RESULTS AFTER ENDOLYMPHATIC SAC DECOMPRESSION AND POSTERIOR FOSSA VESTIBULAR NEURECTOMY

*Seth I. Rosenberg, Herbert Silverstein, and Michael E. Hoffer*

### **ABSTRACT**

Endolymphatic sac decompression and posterior fossa vestibular neurectomy are accepted procedures for controlling refractory vertigo associated with Meniere's disease. Using the 1985 AAO hearing criteria, the hearing results of 109 patients undergoing endolymphatic sac subarachnoid shunt (ESS) were compared to 150 patients undergoing retrolabyrinthine or combined retrolabyrinthine-retrosigmoid vestibular neurectomy (VN). At one month postoperative, no change in hearing occurred in 60% (66/109) in the ESS group and in 68% (102/150) of the VN group. Hearing was improved at one month postoperative in 13% (14/109) of the ESS group and in 10% (15/150) of the VN group. Hearing was worse at one month postoperative in 22% (24/109) of the ESS group and in 19% (28/150) of the VN group. After surgery, profound hearing loss occurred in 5% (5/109) of the ESS group and in 3% (5/150) of the VN group. At one year postoperative, 37% (26/70) of the ESS group had worse hearing as compared to 19% (14/73) of the VN group ( $p < 0.05$ ). From these results, it appears that the short-term hearing results are similar after ESS or VN; however, at one-year postoperative there is more hearing deterioration in the ESS group.

---

Ear Research Foundation, 1901 Floyd Street, Sarasota, Florida 34239-2909.

# A HUMAN TEMPORAL BONE STUDY OF CHANGES IN THE BASILAR MEMBRANE OF THE APICAL TURN IN ENDOLYMPHATIC HYDROPS

*Benny Nageris, Joe C. Adams, and Saumil N. Merchant*

## ABSTRACT

We observed that some temporal bones with endolymphatic hydrops (EH) showed varying degrees of basal-ward displacement (towards the scala tympani) of the basilar membrane (BM) in the apical turn of the cochlea. In some, the BM was adherent to the bony wall of the scala tympani (i.e., the interscalar septum). Such mechanical distortion of the BM could conceivably alter cochlear mechanics and lead to sensorineural hearing loss. The results of a systematic evaluation of 234 temporal bones to characterize, quantify and determine the functional significance of this observation are presented. Four groups of bones were evaluated: normal (n = 78), presbycusis (n = 96), Ménière's disease (n = 23), and EH secondary to labyrinthitis (n = 37). The incidence of extreme displacement of the BM in the apical turn such that it adhered to the interscalar septum was 52% in Ménière's disease, 57% in EH secondary to labyrinthitis, 10% in presbycusis, and 1% in normals. These differences were significant and could not be explained on the basis of age, sex, postmortem time, or artifact of technique or processing. Displacement of the BM was not observed in other turns of the cochlea. Its pathogenesis is not known, but may be related to atrophy of the spiral ligament. It is likely that such BM displacement results in sensorineural hearing loss. However, our data and theoretical analyses both indicate that such a loss will be restricted to frequencies below 100 Hz and that this pathologic change alone is not likely to cause appreciable hearing loss at clinically tested frequencies of 250 Hz and higher. Hence, even though this pathologic finding is common in endolymphatic hydrops, it cannot explain the low-frequency hearing loss observed in Ménière's disease.

---

Department of Otolaryngology, Massachusetts Eye and Ear Infirmary, and the Department of Otolaryngology, Harvard Medical School, Boston, Massachusetts, U.S.A.

Results of this study were presented at the 128th Annual Meeting of the American Otolaryngological Society Inc., Palm Desert, California, April 29–30, 1995.

Reprint requests: Dr. S.N. Merchant, Department of Otolaryngology, Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston, Massachusetts 02114, U.S.A.

## DISCUSSION PERIOD TWO

### Papers 5–8

**Dr. Jahrsdoerfer** (Houston, Texas): The preceding four papers are now open for discussion.

First microphone.

**Dr. Kumar** (Chicago, Illinois): I have a comment for Drs. Black's and Katsarkas' papers. The matter of vestibular compensation is interesting and I think we ought to differentiate between pure labyrinthine loss and the compensation associated with that, and the compensation that we are looking for in patients with vestibular neuritis or following acoustic neuroma surgery. In the latter two instances there is potential involvement of the central nervous system as well. So, if we differentiate between the two, maybe we can get some better answers.

**Dr. Jahrsdoerfer** (Houston, Texas): Second microphone.

**Dr. Jason Henson** (St. Louis, Missouri): I would like to ask Dr. Black what kind of head fixation was used in his rotational chair testing and what kind of visual stimulus was used for the VVOR testing.

**Dr. Jack Pulec** (Los Angeles, California): I too wish to respond to Dr. Black's paper and comment that one must put in pure data in order to make certain analysis. He is talking about labyrinthectomy. Simonton showed many years ago, as have I, that labyrinthectomy does not mean complete ablation of vestibular function. Yet Dr. Black, when he presented his own case, was surprised to find that the patient was not entirely compensated, and by the second case, done by someone else, that showed normal VOR responses, indicating that the patient did not have complete loss of vestibular function. I think that this evidence of residual function would seriously contaminate any results of studies on vestibular compensation.

**Dr. Michael Seidman** (Detroit, Michigan): I have a question for Dr. Merchant, whose presentation was really fascinating. As I am sure you know, there is a lot of work being done in Ann Arbor regarding basilar membrane micromechanics. I wonder if you have any animal models currently looking at basilar membrane micromechanics, because I think they may very well explain your fascinating finding.

**Dr. Jahrsdoerfer** (Houston, Texas): Dr. Hamid.

**Dr. Mohamed A. Hamid** (Moreland Hills, Ohio): I have a question for Dr. Katsarkas about the asymmetry index you used to quantify compensation in terms of the phase relationship as well. If I understood your comments correctly, it can be used to determine the side of lesion. Would you comment on that, please?

Dr. Black, excellent presentation and good scientific work. What is the temporal pattern of VOR versus VSR compensation? Do patients compensate first using their vestibulospinal reflex or do they use the vestibulo-ocular reflex first? Thank you.

**Dr. Jahrsdoerfer** (Houston, Texas): Are there any comments from the audience?

**Dr. John J. Shea, Jr.** (Memphis, Tennessee): I would like to comment on Dr. Black's paper first. I read the literature and I am always impressed by Dr. Black's emphasis on the fact that totally destructive procedures, including streptomycin perfusion, of which we have all been guilty, leave the patient with a deficit, for which they may have trouble compensating. I wonder if there is any benefit in shifting away from destructive operations in the vestibule and going to reparative ones, for example, dexamethasone perfusion, which does not destroy the rehabilitative abilities of the patient. I think Dr. Black's message to all of us has always been to leave the vestibular system as intact as possible.

**Dr. Jahrsdoerfer** (Houston, Texas): I will ask Dr. Black to respond first.

**Dr. F. Owen Black** (Portland, Oregon): First of all, I would like to thank everyone for their comments.

To Dr. Kumar, you are quite right; I did not have time in the presentation to mention that we always rule out cerebellar or other central nervous system abnormalities. There are, as you know, sensitive techniques for ruling out such abnormalities, and we did not include any subjects who had central nervous system findings. With regard to the technique of opticokinetic stimulation, we used a full field opticokinetic stimulus beyond theoretical infinity; it was about a meter and a half from the subjects, and we gave them approximately the same pseudo-random stimulus with the head fixed in the rotatory chair for the opticokinetic. For the VVOR,

the subject was rotated in the chair with the same stimulus as given for the VOR, but with eyes open, viewing the visual surround. In terms of the technique of the signal processing, that is a very involved discussion; I would rather talk to you about that personally, if I could, after the meeting.

Dr. Pulec, perhaps I was not clear enough in my description of these patient groups and in my haste I probably forgot to mention that the subjects that we selected to present were subjects that we were very concerned about compensating. We knew ahead of time that their functions were not normal; we advised the patients that they were not normal and that for that reason we could not predict their outcome. In the first patient, the severe episodes of vertigo were reduced, as a matter of fact eliminated, with the operation, but the patient was still unsteady. When the subjects moved their heads quickly they had the sensation that they continued to move, which they found very disruptive. So, I apologize to you for that omission. With respect to Dr. Hamid's questions, the vestibulospinal system, if the subject starts walking, compensates much more rapidly during the initial phase of recovery. Typically, subjects can do that within minutes to hours once they get moving around. The longer term recovery, which is the second phase of recovery, requires days to weeks. The VOR asymmetry that Dr. Katsarkas talked about takes about ten days to recover, and I think everyone pretty much agrees with those dynamics.

With respect to Dr. Shea's comments, I could not agree more; we think of not harming the hearing, but we do not hesitate to wipe out vestibular function. One of our motivations is to try to find ways to give the clinician tools to help to predict and document when this might occur so we can deal with this problem. Thank you very much.

**Dr. Jahrsdoerfer** (Houston, Texas): Dr. Katsarkas, would you like to respond next?

**Dr. Anthanasios Katsarkas** (Montreal, Canada): To Dr. Pulec's comment I would like to repeat that the definition of compensation is purely clinical. Firstly, there is no such a thing as total and complete compensation simply because the persons who have lost vestibular function on one side will have functional deficits for the rest of their lives. Secondly, I would like to point out to Dr. Kumar that I have the impression that brain stem dysfunction is like pregnancy—it is an absolute, not a matter of degrees. If there is residual brain stem dysfunction after surgery it is going to show. At least for the vestibulo-ocular system, we have the tools today to determine if there are residual symptoms after

surgery. Our cases were all less than 2 cm; I did not do the surgeries, but I could find neither pre-operative nor postoperative brain stem dysfunction in the vestibulo-ocular system. Finally, I would like to comment on Dr. Hamid's question. The asymmetry index shows how much on the side of the pathology the system has become nonlinear. In other words, talking in engineering terms, a linear system is a system which can predict, or rather the person can predict when he moves his head, where the eyes will go. When the function becomes nonlinear, it means that when the person moves his head, the eyes will go somewhere where the system cannot predict, and this results in the person becoming dizzy. As you saw on my slide, what happens is that the system is capable of linearizing function within small velocities. That is why these people will not complain that they have dizziness when they move their heads slowly, but do when they go above a certain range of velocities. It is the asymmetry index which shows the degree of nonlinearity; it does not show if the person feels well or not, but when you put the whole thing together it shows how much the person is able to tolerate his nonlinearity.

**Dr. Jahrsdoerfer** (Houston, Texas): Dr. Merchant.

**Dr. Merchant** (Boston, Massachusetts): Thank you for the comments. We do not have any animal work going on in our labs to test for the defect of change I showed you. There are two additional pieces of evidence that support the fact that this change in the apex would not cause significant hearing loss at the frequencies 250 cycles per second and higher, which I did not show in the interest of time. The first is the fact that the traveling wave has a very steep slope on the apical side; so, if one were to mark displacement at the apex and the 250 cycles per second spot on the basilar membrane was stimulated, the spread of excitation would be much greater toward the base of the cochlea and very little toward the apex. The second piece of evidence comes from examination of the temporal bone in our collection from a patient with Meniere's disease who had an audiogram two weeks before he died. His thresholds were 30 dB for 125, 250, and 500 cycles per second, and his temporal bone shows extreme displacement of the basilar membrane at the apex with no displacement in any of the other turns. Accordingly, the fact that he had a 30 dB shift in thresholds for 125, 250, and 500 cycles, but no greater shift at 125 despite the displacement, makes me feel justified in saying what I did. Thank you.

**Dr. Jahrsdoerfer** (Houston, Texas): We will now have an intermission. Please return at 10:00 o'clock. Thank you.

## PANEL DISCUSSION

### BENIGN POSITIONAL VERTIGO: NEW TREATMENTS

**Moderator:** Jack M. Kartush, M.D.  
**Panelists:** Jack M. Kartush, M.D., John M. Epley, M.D.,  
Brian W. Blakley, M.D., John F. Kveton, M.D.,  
Richard R. Gacek, M.D., Phillip F. Anthony, M.D.

**Dr. Jack Kartush** (Farmington Hills, Michigan): Good morning, Dr. Jahrsdoerfer, Dr. Matz, members and guests. Our topic today is benign paroxysmal positional vertigo. We have an outstanding array of guest speakers who will update us not only on new treatments for this disease, but also analyze and give us historical perspective. To keep things interesting, I have gathered a collection of speakers with differing opinions, to make for an intellectually stimulating as well as entertaining event. Doctors Epley and Blakley will discuss their contradictory views on particle repositioning. Dr. Kveton will discuss whether or not particles within the semicircular canal are indeed pathologic. Finally, Drs. Gacek, Anthony, and I will discuss surgical treatment. In order to minimize duplication and allow our speakers to focus on their particular topic, I will begin with a brief overview.

Positional vertigo is extremely common. Because particles have been identified within the posterior semicircular canal, it has been felt by many to be a post-traumatic disorder but patients often have no antecedent history of trauma; consequently, viral, vascular and degenerative causes may be more common. The Hallpike test is usually diagnostic and demonstrates a transient rotary nystagmus with the affected ear down. It is important to recall that, because the nystagmus is rotary, electronystagmography is often unable to detect it. Therefore, it is important that the otologist perform the Hallpike test and evaluate the eyes.

Since Barany's 1921 classic description of benign positional vertigo, there have been many contributors to this topic some of whom are with us today. Drs. Schuknecht and Parnes were invited but unfortunately were not able to attend. When I spoke with Dr. Schuknecht recently, he wanted me to emphasize a point to this audience. Some have re-

ported that Schuknecht's prior work only demonstrated particles attached to the cupula whereas Lorne Parnes' posterior semicircular canal occlusion surgery was the first to demonstrate free floating particles within the canal. But this is not the case. In fact, in Dr. Schuknecht's 1969 article he not only postulated cupulolithiasis but also stated that presumably these particles might also exist free in the endolymph. From this 1969 article, you can see a normal cupula, versus one weighted down with otoliths, i.e., cupulolithiasis. But in addition, Figure 6 demonstrates free particles in the canal, i.e., canalolithiasis.

The treatment for benign positional vertigo has often been simple observation. Many believe that tincture of time is the only treatment that is needed. However, some patients will have prolonged and occasionally severe symptoms. Many seem to benefit from the canalith repositioning procedure which Dr. Epley will discuss later. A few patients will have disabling symptoms despite all conservative treatments. Dr. Gacek was the first to describe singular neurectomy and this was helpful not only in developing a cure for the disease but also in confirming the site of pathology. Dr. Anthony will describe indirect laser partitioning and I will discuss our work using direct laser-assisted occlusion.

With this as our foundation I think our panelists will be able to get right to their particular discussion sections. First, I would like to invite Dr. John Epley to discuss canalith repositioning.

#### PARTICLE REPOSITIONING IS EFFECTIVE

**Dr. John Epley:** Good morning. Particle repositioning presupposes that the canalith hypothesis is



true. You have to have faith that BPPV is caused by hydrodynamic forces produced by free particles within the semicircular canal. The original concept as Dr. Kartush mentioned was that there were particles attached to or pinching upon the cupula but this would obviously cause a persistent nystagmus which is different from the typical transient nystagmus we see with BPPV. So, obviously it makes more sense that this condition is caused by free particles passing down the posterior canal after the person has been moved from upright to the supine position. Now, if they are further moved into the Hallpike position, notice that the cupula actually is in a vertical position and would not be affected by gravity.

As these particles move down the canal we envision that there is a hydrodynamic drag which pulls the cupula downward and of course when you reverse it the same thing happens in reverse. These particles then act like a leaky piston. As these particles pass downward the fluid has to pass on each side of them in the opposite direction and this fluid in a way acts like almost a lubrication that prevents the particles from dragging along the sides. Because of the differential in cross section between the canal and the ampulla (their areol ratio is approximately 25), we get a hydraulic mechanical advantage like a typical hydraulic jack. This gives the free particles extra capacity to move the cupula and overcome the cupular resistance. This also presupposes that the particles are together in one area of the endolymph loop if you think of this as a complete loop. If the particles are on one side of the loop, they will produce a unidirectional flow in the fluid and a force in this direction. But if they are dispersed, and, as we know, many of these have been found in patients with no particular symptoms, then you get a cancelling force in both directions as they move earthward through the canal.

Canalith repositioning is a theoretically based treatment of positional vertigo due to canalithiasis designed to induce out-migration of free densities from the pathological semicircular canal to the utricle. Once they are in the utricle, they do not cause this effect just described.

The procedure is well outlined in the literature. Basically the patient is moved from a sitting to a supine position, and then their head is rotated through 180° so that the canaliths move through the canal. This should be timed because the canaliths are going to move slowly, so that by the time you reach the end of the procedure the particles will have moved into the utricle.

These are the protocols and results from the six published studies regarding canalith repositioning or modifications thereof. These are the principal au-

thors, the various protocols, and the results arranged in descending order of effectiveness. Epley is at the top of the list. In my protocol, I call for 180° effective movement of the head after the person is supine, use of a vibrator, and repetition of the procedure at the same session until the nystagmus is gone. Also, on follow-up we repeat the procedure.

Now, most of the other results are stated in terms of subjective response. However, I have reported mine in terms of the patient actually remaining resolved for at least a month with no symptoms and they must have a negative Hallpike test with no nystagmus. This is a much more rigid test criteria. Obviously, the results are very good for most of the studies. The one outlier is Blakley and the reason for this appears, from my reading of his article, and he can comment on this, that he only used a 90° effective rotation of the head in the supine position. This, then, possibly makes Blakley's study a "control." He also waited until one month out, and then he did not test these people with a Hallpike test, according to his article, but he only went on the basis of subjective responses.

In summary, the canalith repositioning procedure and the various modifications for particle repositioning have been shown to be very effective. I feel that this should be the treatment of choice, and certainly it should be tried, and a good attempt made, before we go into the various surgical procedures. Thank you.

#### PARTICLE REPOSITIONING IS INEFFECTIVE

**Dr. Brian Blakley:** I would like to thank Jack for asking me to present my views on the canalith repositioning maneuver. He did this probably because I published a study last year about a trial testing the maneuver. I would like to say at the outset that even though my opinions are different than those of Dr. Epley's they should not be interpreted as a personal thing. I have a lot of respect for Dr. Epley in attacking this rather difficult problem that bothers many patients. The canalith theory gives me a lot of problems when I think about it. For this reason I thought it would be most appropriate to do a randomized trial. The thought I would like to start and finish with is the idea that when you consider diseases such as benign positional vertigo and dizziness which tend to get better anyway, it is very important to consider the natural course of the disease. The statement that 80% got better or that any number got better, by itself, is not meaningful unless we also consider how many people got better without treatment.

This is stolen from one of the articles which outlines the basis behind the canalith theory. The idea is that in contrast with the cupulolithiasis theory of Schuknecht, basically these solid bodies, whether they be the otoliths, or calcium carbonate, or something else are found in the posterior semicircular canal of some people. Under the influence of gravity, if the head is positioned in a provocative position, they may move and cause negative pressure in this region deflecting the cupula.

The problem that I have with that is that a solid body falling through a liquid does not affect the pressure of the liquid above it unless the diameter of that object is similar to the diameter of the vessel. In other words, it acts like a piston in a car. If that is the case that means there is some resistance, some slowness of reactivity if you will, and therefore repositioning these canaliths should take a long time—I think much longer than the 13 seconds that Dr. Epley has indicated. There are many other problems I have as well with the model as you will see in a minute. What I wanted to do with the study was to try to duplicate or enhance Epley's technique as much as I could. Actually it is not possible as far as I could see, to duplicate exactly all of what has been described. The idea of randomization I think has value and accounts for the natural course of the disease. There are many other reasons in particular diseases that are more important to randomize; however, I must agree that not all clinical treatments need to have a randomized trial. In fact, most of the things that we do in clinical medicine are not, let's say, verified by a randomized trial. There are many randomized trials that are baloney.

This slide is the orientation in the coordinate scheme that most people use and I have chosen this which I think corresponds with Dr. Epley's. This side shows what are supposed to be human beings, believe it or not, and the head is flexed backwards and if we view it from the top, these are the ears, and nose. This is what I will refer to as 0 degrees, this is 45 degrees towards the lesion, 45 degree away, 90 degree away, and so forth. Dr. Epley has indicated that the degrees that the canaliths can negotiate between each maneuver is 90 degrees but we do not have good information on what the effect of the diameter of the tube is and the viscosity of the endolymph and so forth. One of the analogies I like to think of is that if you take mud and put it into water and swirl it around and set it down, it takes longer than 30 seconds usually depending on the size of the material and so forth, to settle out. If you shake that material as with an oscillator, it will take longer. Those are the optimal circumstances—when the vessel is large and

the walls are some distance from the particles, in the canalith situation we try to negotiate a very small, curved canal which presents more problems.

This is a repeat of the slide Dr. Epley just showed, outlining his technique. The patient starts in an upright position and then is leaned back with the head flexed and turned 45 degrees. However, if we look at this diagram, if the canaliths are here it might be better to even rotate more to 90 degrees perhaps and to do more stops. This enhancement is what I chose to do. The oscillator also gives me some problems. Dr. Epley has indicated that one particular brand given down here should be used. The print did not come out very well at all but there is another brand which is identified which should be used. In another article a still different brand of oscillator was used and we were cautioned against using "unproven oscillators." The oscillator question gives me problems so I decided not to use that. The parts of the Epley technique which have given me problems are these, and the 6 to 13 seconds seems very short to me. So, I thought it might be wise to prolong those, prolong the positions, in other words, position the head for a longer period of time. The vibrator is a big question, and I have decided not to use one. How often the maneuver is repeated is not exactly clear. Some of my readings from literature have indicated to stop when the symptoms go away, some indicated when the nystagmus goes away. It is hard for me to differentiate that from the fatigue which we know happens in these patients.

The follow up time is not clear, I think, from reading the literature. "How long until you reassess patients?" So, to do this study I wanted to give the same stimulus to everybody and follow them up in the same length of time which, in my view, should be a reasonably long length of time, so I did this. I took five positions, turned the subjects 90 degrees, then 0 degrees, and then 90 degrees back and then 135 degrees as Dr. Epley does and sit the patient forward. Each of these positions is held for 30 to 60 seconds so that the whole maneuver takes at least 2 minutes. I did not use a vibrator, but followed up with patients in a month.

The trial consisted of 16 treated subjects and 22 controls, chosen at random; I found no difference in response after a month. There was a great spontaneous improvement rate. All participants felt better after three months. This graph which shows the numbers and chi-square analysis does not suggest any statistical difference. Thank you.

**Dr. Kartush:** Thank you, Brian. Our next speaker is Dr. John Kveton who will discuss whether or not particles are indeed pathological in the posterior canal.

## PARTICLES ARE NOT PATHOLOGICAL

**Dr. John Kveton:** Thank you Dr. Kartush, Dr. Jahrsdoerfer, members, and guests. I would like to briefly discuss what we know about the substances in the vestibular labyrinth that had been implicated historically in benign positional vertigo. For the sake of clarity I will divide this discussion into an identification of these substances, their origin and hopefully how they function within the labyrinth. These substances have been identified by several names. Dr. Schuknecht described them as statoconia. They have been described as particles, otoliths, canaliths, and Dr. Gantz had described them in an affectionate manner as Parnesicles. That is my favorite one by the way. The fact that we have yet to settle on one term to describe these substances indicates our uncertainty regarding their origin. There is no doubt that these substances exist though. Vyzlonsil first histopathologically identified basophilic staining material within the lumen of the non-ampullary portion of the posterior canal in patients who had suffered ototoxic injury after streptomycin therapy. Dr. Kartush has already alluded to Dr. Schuknecht's identification of the same basophilic deposits within the non-ampullary portion of the posterior canal in one of his patients in whom he had based his theory of cupulolithiasis. In 1991 Lorne Parnes demonstrated these substances in live human subjects in his landmark intraoperative videotape, which most of us have seen, of one of his canal occlusion procedures. Dr. Epley has felt that canaliths are more appropriate descriptors of these substances. In 1994 we demonstrated the presence of these substances within the non-ampullary portion of the posterior canal using transmission EM. What was interesting was that only one patient in ten studied complained of any positional vertigo. This obviously raised the question of whether the mere presence of these particles was pathologic or not.

There still remains a question about the origin of these substances. Previous reports reconfirmed by more recent studies indicate that otoconia can be displaced from the maculae by linear acceleration. Once studied carefully, though, this linear acceleration exceeds the physiologic ranges of normal vestibular function so it cannot possibly explain all the cases of positional vertigo. As importantly, while senile degeneration has been implicated as a reason for otoconial loss through analysis of histologic specimens, Johnson and Hawkins in 1972 were careful to note the likelihood of preparation artifact as an explanation for the apparent degener-

ation of neuroepithelial structures in the appearance of such basophilic deposits. In addition, another possible origin was raised by our identification of vacuole structures within the luminal epithelium of the non-ampullary portion of the posterior canal. These vacuole structures raise a possible secretory capacity which to our knowledge has not been identified in any other region of the inner ear. These vacuoles contain moderately electron dense material consistent with lipid or protein, and a particulate matter is also composed of varying electron densities characteristic of partially mineralized protein. At present, the function of these particles remains theoretical.

It is my personal opinion that it is the degree of particulate matter within the lumen of the non-ampullary portion of the canal exerting a frictional effect that influences endolymph flow within the system. It is endolymph flow, not cupular deflection, that stimulates the sensory cells of the crista. This is supported by variable scientific data, one demonstrating that there is imperceptible cupular displacement at physiologic stimulation parameters. Another piece of evidence is that initial cupular displacement does not influence hair cell response. This theory is further supported by the identification of a subcupular space that measures about 60 to 100 microns that permits endolymph flow over the ampullary hair cells. Also, that calorically sustained endolymph flow will maintain ampullary excitation. And also, that the ampullary hair cells that generate this excitation are indeed deflected by fluid displacements. In conclusion this slide best explains my attitude towards the subject, mainly one of a study in bewilderment. Thank you.

**Dr. Kartush:** Thank you, John. The next speaker is also our Society's Guest of Honor, Dr. Richard Gacek, who will discuss singular neurectomy.

## SINGULAR NEURECTOMY

**Dr. Richard Gacek:** I am not going to get into the debate about what to do with these particles. I think a lot of this is very theoretical. The fact is that most patients with this disorder, perhaps 90%, undergo spontaneous resolution or live with the disorder. If I see any patients early in the course of their disease I do not treat them with anything but time. The small number of patients that do have chronic benign paroxysmal positional vertigo, and by chronic I mean at least one year in duration and in many cases longer, are significantly disabled. These are patients that are candidates for surgical relief. The

approach that I have taken for the last 23 years is like the first patient I had in 1972 when I performed a selective ablation of the posterior canal sense organ. There are a number of reasons why I believe that the pathology conceptually is located in the posterior canal. It is the most dependent sense organ. The debris, or what you call dislodged otoconia, probably is a degenerated form of the basophilic material that we see in histological sections. The logical source of this material is the utricle and I think the utricle plays a role in this disorder. It is not just the posterior canal that is activated by the positional stimulus but the lack of normal utricular control over all the three semicircular canal responses, in this case with the posterior canal, that contributes to the clinical condition that we see.

The anatomical landmarks and relationships that are important to performing selective ablation through the middle ear approach is the round membrane that we see outlined here in this left ear which will need to be completely exposed surgically by removal of the bony overhang of the niche. The intermediate portion of the singular canal travels just inferior to the posterior-superior portion of that round window membrane. The surgical procedure is performed under local anesthesia just as you would perform a stapedectomy and other middle ear exploration procedures. It requires one to one and a half hours of operating time at most. After exposure of the middle ear with an elevated tympanomeatal flap, the first step is to remove the bony overhang of the niche so the entire round window membrane can be visualized. This portion anterior-inferiorly is easily seen after removal. The posterior-superior portion being parallel to the surgeon's view is not easily seen and needs to be confirmed by displacement of the ossicular chain to confirm the bulging of that portion of the membrane. The approach to the singular canal is carried out on the floor of the niche and usually at the depth of 2 mm or more one encounters the singular nerve. At that point the patient responds with an experience of vertigo, or pain, or both symptoms. The nerve will vary in position. Usually it is partially exposed but it may be completely exposed which is a favorable position. In a small number of cases it is hidden, that is, tucked under the attachment of the round window membrane. The advantage of local anesthesia is very critical in these cases. The nerve is transected with hooks followed by a diamond burr to impact bone dust into the proximal end of the canal. This promotes osteoneogenesis and prevents regeneration of nerve fibers. Only the proximal end of the canal is probed since the ampulla of the pos-

terior canal is located just posterior to the round window niche.

Over the past 23 years I have performed this procedure on 144 patients. There is the usual female to male predominance that is characteristic for this disorder with quite a wide age spectrum. It is very unusual to see a symptomatic patient in the younger age group. The peak age group is in the 6th decade. Incidence of bilaterality with one side being more symptomatic than the other is about 18% and that has been confirmed by a number of other authors. The characteristic etiologies that were reviewed by Dr. Kartush are seen in this series. Idiopathic is the most common cause where there is no antecedent trauma but probably a viral insult occurs. This is typified by a prolonged vestibular episode that requires takes several days or weeks to resolve and is followed by the positional component. Head trauma and a number of miscellaneous etiologies complete the list of categories.

The most important data are the results. Out of the 153 singular neurectomies performed, 9 patients had bilateral singular neurectomies sequentially separated by periods of six months to one year. A number of those bilateral cases, after having their worse ear relieved by surgery, did not elect to have the contralateral ear operated. But 145 of the 153 experienced complete relief (95%). Complete relief means complete absence of a vestibular-ocular response and subjective symptoms on the provocative maneuver. Three patients experienced incomplete relief but still had some residual symptoms and findings. There were 5 failures; these were all cases where the nerve was tucked under the round window membrane attachment, and a blind approach was utilized. Partial sensorineural hearing loss occurred in 4 out of the 153 procedures for an incidence of less than 3%. The advantages of selective ablation are shortness of the procedure, short hospitalization of 1 to 2 days, local anesthesia and an excellent success rate for the complete relief of positional vertigo with a minimal risk of sensorineural hearing loss. Thank you.

**Dr. Kartush:** Thank you, Dick. Next is Dr. Phil Anthony who will discuss laser partitioning.

#### LASER PARTITIONING FOR CANAL PARTICLES

**Dr. Phillip Anthony:** I am Phil Anthony and I practice otology in Fort Worth, Texas. Jack has done me the courtesy of inviting me to comment on partitioning which is a laser technique for causing heat

constriction of the membranous posterior semicircular canal. Jack has also given me the option of having a change of heart and I have not shared this with him yet but I have decided that within that vein I am going to spend two minutes summarizing partitioning which is the laser closing of the posterior semicircular canal and the next six minutes describing a new technique that I believe will be a marked improvement and goes more to the heart of the matter in this regard that I call macular ablation.

Partitioning is a technique using the heat of the laser to coagulate the posterior semicircular canal membrane protein and causes the circumferential constriction and longitudinal shortening of the membranous canal. The technique itself requires a general anesthetic, simple mastoidectomy, and blue lining as you can see here on the posterior semicircular canal (and then 1 or several 12 watt laser burns that create a single or several chars usually with a central perilymphatic fistula created which is subsequently covered with soft tissue). The results of partitioning are that it allows control of positional vertigo in 96% of the patients; the resolution of the positional vertigo takes from 3 to 21 days, so it is a reasonably rapid resolution. The dominant side effect of this procedure is that it has marked postoperative motion sensitivity and although this motion sensitivity resolves reasonably rapidly in the first few days it still takes fully four to eight weeks for the motion sensitivity symptoms to resolve. Hearing complications are limited to a previously reported insulin-dependent diabetic who had a postoperative hearing loss; and unreported at this point is the incidence of about 10% of the patients over 65 (even when given steroids post partitioning) will have about a 10 decibel three frequency average hearing loss. There continues to be, as Dr. Kartush has pointed out and I failed to recognize early on, some non-positional symptoms remaining in patients who had non-positional symptoms preoperatively. In concluding, partitioning (in greater than 60 cases) is a technically straightforward technique for controlling positional vertigo with a low hearing loss and high success rate. It is best used in patients who are less than 65 years of age and non-diabetic.

My thoughts about positional vertigo mimicked the thought that the utricular macula is the central item in this disease and everything else is mechanical effects on the posterior canal. It occurred to me that all our surgeries on the posterior canal whether they be denervation procedures or mechanical or laser occlusion procedures are all fishing behind the net. We

ought to be operating on the utricular macula. So I call this new procedure macular ablation. The animal work has been nicely done by Drs. Namuro and Okuna. They demonstrated that following argon radiation, after stapedectomy in monkey and guinea pig, of the utricular macula at ten weeks you see the loss of utricular macula sensory epithelium, the loss of supporting epithelium, the loss of nerve fibers and the replacement of that whole organ with cuboidal epithelium without the loss of continuity of the membranous labyrinth. I have performed this operation on 14 patients, one of whom has been lost to follow up. I call this operation macular ablation. You can see conceptually what is going on. The operation is done through the exterior canal with a stapedectomy-like flap as an outpatient, under local anesthesia. By creating a small laser rosette in the posterior superior stapes and then using a second angled fiberoptic passed from the promontory through the cura and with the tip of it placed just into the perilymph, a three and a half watt burst of energy can be delivered posterior superior medially for half a second. Following that, the rosette is covered with a small piece of tissue and the patient is taken in a wheelchair back to the outpatient unit.

100% of these patients have been improved. The resolution has been complete in 87% of the patients. The marked advantage of this procedure is that there is minimal (relative to posterior canal occlusion) postoperative vertigo. I have had hearing loss in only one patient, a 70 year old lady with a fifteen year history of inactive Meniere's disease. In conclusion, I believe that macular ablation is clearly a new procedure. It allows resolution of positional vertigo and it has the marked advantages of local anesthetic and markedly less postoperative vertigo. Most importantly in this whole conversation, we are beginning to collect techniques that allow us to shape the membranous labyrinth with heat (either argon or CO<sub>2</sub>); with macular ablation we can destroy selectively special sensory epithelium and blood vessels on the medial surface of the endolymphatic and perilymphatic spaces with a visible light laser. What an exciting time. Thank you so very much.

#### CO<sub>2</sub> LASER POSTERIOR CANAL OCCLUSION

**Dr. Jack M. Kartush:** I will now discuss CO<sub>2</sub> laser assisted occlusion of the posterior canal and would like to acknowledge our alumni, Eric Sargent, M.D., Patrick Antonelli, M.D., and Gerard Gianoli, M.D., who have contributed to the clinical and histologic work.

Most patients with BPV will improve spontaneously or with the Epley maneuver. But there are some who have persistent and disabling disease. Singular neurectomy can be very effective but few have been able to do it as safely as Dr. Gacek who is the master of his own procedure. Consequently, many clinicians have sought alternate surgical techniques.

Although labyrinthotomy surgery seems new, otologists have, in fact, been doing so for years, starting with the fenestration operation. Lorne Parnes was instrumental in doing the first human posterior canal occlusion operations: initially in patients with profound sensorineural hearing loss and subsequently in those with normal hearing. Similar work has been done in the past during animal studies but often for different reasons. In 1962, for example, Money and Scott selectively occluded the semicircular canals in an attempt to discern the specific functions of each canal and the associated nystagmus. In 1981, Wipilsesky studied the effect of lasering the outer surface of non-fenestrated canals in guinea pigs with CO<sub>2</sub> and argon. More recently, McElveen, Hirsch, Hakuba and others have demonstrated the feasibility of occluding various canals during neurotologic procedures to increase exposure while preserving hearing.

Dr. Anthony was the first to apply Wipilsesky's laser technique to human ears which he referred to as canal partitioning. Although Phil's histologic studies demonstrated that laser heat applied to the outer surface of the posterior canal does generate an internal fibrosis, I was concerned that the fibrosis might be too variable to assure complete ablation. Consequently, we wanted to develop an open technique that guaranteed complete ablation while minimizing the risk of hearing loss.

I was not aware that Phil Anthony was going to be talking about a new procedure today to replace his partitioning operation but not only are we entitled to change our minds, we are obliged to do so if results demands it.

As some of you know, Ted McGee, George Lesinski, and I have had a friendly, ongoing debate regarding the relative efficacy and safety of visible versus non-visible lasers for stapes surgery. We believe KTP, argon and CO<sub>2</sub> lasers can all be used safely in stapes surgeries if you use the correct parameters. However, when it comes to the micro-environment of the posterior canal, the CO<sub>2</sub> laser appears best suited as will be shown.

Our goal is to occlude the canal while minimizing mechanical trauma and preventing loss of endolymph. We felt that the non-vibrational effects of the laser would be helpful but were unsure which

laser would be best. Thus, we investigated the differential laser effects which are demonstrated on this video. Whereas the CO<sub>2</sub> laser occludes the membrane by shrink wrapping it, the KTP and argon lasers are ineffective because they go through the nearly clear membrane. In fact, this probably explains why KTP and argon lasers are safe in stapes surgery: even though, as Dr. Lesinski has shown, these visible lasers will pass through fluid and tissue, they need a chromophore to initiate vaporization of which there is virtually none in the nearly clear inner ear membranes.

The video shows the posterior canal blue lined and then exposure of the endolymphatic membrane within the canal. You do have to wick away the perilymph in order to be able to expose the membrane. If you use the CO<sub>2</sub> laser without doing so, it does not work well because its energy is absorbed by the perilymph. Here we see an example on soft tissue of the CO<sub>2</sub> shrink wrapping tissue of any color. Now we will see the effect of the CO<sub>2</sub> on the membranous labyrinth. We have wicked away some of the perilymph and are seeing progressive shrinking of the membrane. In doing so, we have not opened the membrane and so there is no endolymph leakage. In contrast, the KTP passes through the nearly invisible membrane but causes virtually no coagulation effect even with repeated applications. Now, similar to Lorne Parnes, we go ahead with the bone pate occlusion, we put a little blood on it and then finish up with fascia. Here are the pre- and postoperative audiograms of our first four patients we reported on with disabling, persistent vertigo. Tomorrow, Dr. Patrick Antonelli will report in detail on twelve of our patients, half of whom had the CO<sub>2</sub> laser and the other half did not. All patients had complete resolution of vertigo with no change of hearing.

As exciting as these results are, I am most interested in extrapolating the concept of selective vestibular ablation to other causes of dizziness. Although total ablative procedures such as labyrinthectomy and vestibular neurectomy are effective in resolving vertigo, I am troubled by the incidence of postoperative disequilibrium and lightheadedness. These symptoms are often worse after surgery and I believe are under-reported in the literature.

Perhaps selective vestibular ablation techniques can help us restrict the area of ablation in order to spare uninvolved tissue. Therefore, in the future, rather than performing a total labyrinthectomy, we may be able to selectively occlude all three canals (triple canal occlusion; TCO) to ablate the accelerometers while preserving the statoliths. Conversely, if we identify the statoliths as the cause of a

particular patient's dizziness, the utricle and saccule could be selectively treated via the stapes footplate while preserving the canals. Phil Anthony's comments today show that he has apparently been thinking along the same line.

The benefits of selective vestibular ablation remain to be seen but the persistence or exacerbation of disequilibrium and lightheadedness following labyrinthectomy and vestibular neurectomy clearly show that total ablation procedures are not the final answer.

In conclusion, the safety and efficacy of evolving procedures for BPV reveal that we are indeed approaching an era of intralabyrinthine surgery.

## PANEL QUESTIONS AND OPEN DISCUSSION

**Dr. Kartush:** Gentlemen, thank you very much for your outstanding presentations. I will start out directing this to Dr. Epley: There has been some controversy; is the vibrator really necessary for the Epley maneuver?

**Dr. Epley:** It certainly is not necessary, and there are some patients where it may be contraindicated. However, in our study with and without, we got about a 20% increase in our results with the vibrator.

**Dr. Kartush:** So, have you done that in a controlled fashion, John? Did you alternate treatment?

**Dr. Epley:** Yes, this was many years ago.

**Dr. Kartush:** So, 20% improvement in efficacy using the vibrator. Any other thoughts from the panelists regarding using the vibrator? How many of you here are doing particle repositioning for your patients? About half do and half do not.

If particles are not part of the histopathology, how are these patients actually recovering spontaneously? Why is there such a high rate of improvement? John Kveton, do you want to address that?

**Dr. Kveton:** I think one of the biggest problems with this whole condition is that we are still not sure what we are trying to treat. I think the "Art of Medicine" plays a huge role in this condition. My own perspective is that these particles do exist and, through the simple metabolic course of inner ear function, some of these particles become reabsorbed or shift on their own. So, I think that as Dr. Gacek points out a huge majority of these patients will get better on their own.

**Dr. Kartush:** And that is happening by what mechanism John? By becoming dispersed?

**Dr. Kveton:** Yes, that is my own perception, yes.

**Dr. Kartush:** Rather than by resolution or dissolution of the particles?

**Dr. Kveton:** It could be a combination of those two. Again, this is completely hypothetical but I feel that there probably are some metabolic factors that are involved here as well.

**Dr. Anthony:** I have some observations. First of all, I agree with Dr. Gacek's numbers in that about the third of the patients I have operated upon have been traumatic and about two thirds have been spontaneous. I have wondered semi-privately whether the traumatics were not where big chunks of the otoconia were knocked off which made them longer running and tending to be more continuous rather than intermittent and in the two-thirds spontaneous onset are degenerative, viral, or whatever if they are not more affected by flakes. The flakes dissolve more rapidly, hence the episodes come and go and come and go. John Epley and I have talked about this and John you said to me that you thought there were a finite number of otoconia. I got to look at some of Harada's work which is scanning EM stuff and he pointed out that in the material underneath the otoconia there was a particularly high concentration of calcium. So, my theory had been that these otoconia either get knocked off or they come off and then they dissolve. They are probably replaced.

**Dr. Blakley:** I think that is right. There are studies in the basic science literature that indicate that there is otoconia turnover and that might be the mechanism of recovery. However, we are also well aware that the vestibular system has a great capacity for compensation in other situations so I do not think it should surprise us that people do tend to recover from this particular disorder by whatever mechanism.

**Dr. Kartush:** Dick, I look at some of the newer procedures as really being just another way to obtain some very selective ablation. What is your assessment of some of these new procedures in contrast to the one which you know has worked for many years.

**Dr. Gacek:** I think the big difference is that these procedures are all invasive procedures. They involve invading the labyrinth and so incur some degree of labyrinthitis. Serous labyrinthitis hopefully is reversible. As compared to a non-invasive, as far as the labyrinth is concerned, the procedure of selective neurectomy. That is the big difference in my mind between all of these procedures and the selective extra-labyrinthine ablation which avoids any trauma to the labyrinth. And the trauma to the labyrinth is expressed in both auditory and vestibular symptoms and findings like sensorineural hearing loss which may be reversible or may not be. As I have talked around the country and around the world with other surgeons performing canal occlu-

sion the results are not as good as some of the results that we have heard here today. I do not know if anybody in the audience had some experience but I have heard some very discouraging reports about sensorineural hearing loss, persistence of vertigo, tinnitus, and all of these sequela indicate that there is a traumatic labyrinthitis that has been incurred.

**Dr. Kartush:** We have just two minutes so are there some questions from the audience?

**Dr. Stephen G. Harner** (Rochester, Minnesota): The question I would like to ask is—how much objective data do we have in terms of follow up? I think I have heard Dr. Gacek refer to follow up ENG or some objective data. I guess I would like to ask all the folks up there how much objective data do you have in terms of the vestibular studies before and after treatment, and what kind of changes have been seen?

**Dr. Gacek:** I think it is a good question. The auditory studies speak for themselves, and we obtained hearing tests both at the early periods of one month, as well as one year and longer. I do not do vestibular testing (that is, ENG) in all my patients as many of my patients come from a distance, making that very difficult. The patients that we do have near Syracuse we have tested both pre- and post-operatively with caloric stimulation. The results are interesting, because we find that after ablation of the posterior canal, with stimulation of the labyrinth in the position that is optimally supposed to stimulate the lateral semicircular canal, there is a decrease in the overall response post-neurectomy, which varies anywhere from 10% to 20%.

**Dr. Kartush:** We have time for one or two very brief questions. Dr. Katsarkas.

**Dr. Anthanasios Katsarkas** (Montreal, Canada): I was very surprised to hear Dr. Blakley saying that the diagnosis of BPPV is subjective. Sometimes during the attack the eye movements are so intense you have the impression the eyes will jump on the floor

and go. That is number one. Number two, the type of nystagmus, which is always rotational when the gaze is toward the floor and always linear when it is toward the ceiling. It cannot be from the otolith organs. So Dr. Anthony has to change that and I hate to give him a heartache but the otolithic organs cannot induce this kind of nystagmus. Finally, I would like to point out that there is a possibility that endolymph, like any other fluid in the human body, may make deposits.

**Dr. Kartush:** Dr. Hamid, you have 22 seconds to comment.

**Dr. Mohamed Hamid** (Cleveland, Ohio): First, I have a comment on the Semont maneuver. The number of patients that you actually see with the classical benign paroxysmal positional vertigo with nystagmus (we have a data base of about ten thousand), and we probably have about two hundred patients with the classical definition of this syndrome. Some of the data in the literature using 150, 170, 190, 200, 300 patients. I would like the panel to address that point.

**Dr. Blakley:** With regard to Dr. Katsarkas' question about the diagnosis being subjective. I did not mention the basis for diagnosis. We only had five minutes but benign paroxysmal positional vertigo is partly on subjective but also on objective findings of geotropic rotatory nystagmus with the latency and symptoms. In other words if you have nystagmus without symptoms it does not count. My suggestion about subjectivity was based on the Epley maneuver and when do you stop. Is it based on symptoms? I mentioned that I do not understand when are you supposed to stop. Is it based on symptoms, nystagmus, or both and how is that different from fatigue?

**Dr. Kartush:** We are out of time; I would love to continue, but in the interest of the next speaker we must conclude this panel. I thank all the panelists for their time.



# REVISION STAPEDECTOMY WITH AND WITHOUT THE CO<sub>2</sub> LASER: AN ANALYSIS OF RESULTS

*Thomas J. Haberkamp, Steven A. Harvey, and \*Yasser Khafagy*

## ABSTRACT

A retrospective review is presented of revision stapedectomies performed by the senior authors between 1986 and 1994. A total of 106 stapedectomies was performed during that period, of which 30 were revisions. These revisions were within the first 100 stapedectomies performed by the senior authors. Most failures occurred early in the series. The overall success rate for closure to within 10 dB was 52%, and the average closure was 12.72 dB. Five cases were performed without the laser with no successes and an average closure of 27 dB. After the use of the laser, the success rate was 64%, and the average closure was 9.75 dB. There was a statistically significant difference between the hearing results with and without the laser ( $p < 0.01$ ). The prognosis was better when surgery was performed primarily for hearing loss and with the laser, with success in 13 (72%) of 18 cases. In two cases, there was a sensorineural decline >10 dB. The only dead ear occurred preoperatively in a patient with a granuloma. We have found the use of the CO<sub>2</sub> laser in revision stapedectomy to be a safe technique that produces reliable results.

---

Department of Otolaryngology and Human Communication, the Medical College of Wisconsin, Milwaukee, Wisconsin, U.S.A.; and \*Department of Otolaryngology, Faculty of Medicine, Mansoura University, Mansoura, Egypt.

Presented at The Annual Meeting of the American Otologic Society, Palm Desert, California, April 29–30, 1995.

Reprint requests: Dr. T.J. Haberkamp, Department of Otolaryngology and Human Communication of the Medical College of Wisconsin, 9200 W. Wisconsin Ave., Milwaukee, Wisconsin 53226, U.S.A.

## REPORTING OPERATIVE HEARING RESULTS IN STAPES SURGERY: DOES CHOICE OF OUTCOME MEASURE MAKE A DIFFERENCE?

*Karen I. Berliner, \*Karen Jo Doyle, and †Robert A. Goldenberg*

### ABSTRACT

In a prior study, findings indicated that when reporting results of chronic ear surgery, neither choice of pre- versus postoperative bone-conduction scores nor choice of frequencies to include in averaging makes a substantial difference in reported outcome. In this study, audiologic data from 240 stapes surgery patients at three different institutions were used to generate a variety of outcome measures. Use of preoperative rather than postoperative bone conduction values in computing postoperative air-bone gap resulted in an ~5-dB smaller mean gap and a 2% higher success rate. Frequencies included in averaging made little difference in mean computed air-bone gap, although success rate (gap <10 dB) was lower by 6% when 4 kHz was used in a four-frequency average rather than 3 kHz. Results for air conduction were similar to those for air-bone gap regarding choice of frequencies to include in averaging. When using air conduction pure-tone average (PTA) as the outcome measure, those with normal preoperative sensorineural hearing had a >20% higher success rate than the general population of stapes surgery patients. The greatest differences in success rate were based on definition of and criteria for success. Success rate was higher when based on air-bone gap than when based on air conduction PTA. As in the prior chronic ear study, differences in outcome were more drastically affected by criteria for "success" than by frequencies included. Unlike similar data from chronic ear surgery, however, success rate differed depending on choice of air-bone gap or air conduction PTA as the definition for success. Further, air and bone scores from the same test interval must be used to accurately reflect air-bone gap in stapes surgery.

---

House Ear Institute, Los Angeles, \*Department of Otolaryngology Head and Neck Surgery, University of California at Irvine, Orange, California, and †Department of Otolaryngology, Wright State University School of Medicine, Dayton, Ohio, U.S.A. Presented at the American Otological Society 128th Annual Meeting, Palm Desert, California, April 29, 1995.

Reprint requests: Dr. K I. Berliner, House Ear Institute, 2100 W. Third Street, Los Angeles, California 90057, U.S.A.

## ENDOSCOPIC STAPEDECTOMY: A PRELIMINARY REPORT

*Muaaz Tarabichi*

### **ABSTRACT**

All of the surgical tasks involved in stapedectomy were performed using the endoscope, a video camera and monitor instead of the microscope in six patients with otosclerosis and secondary fixation of the footplate. Small fenestra technique was utilized in all patients. All patients had closure of the air-bone gap to within 10 dB (pure tone average of 0.5, 1 and 2 kHz) at two months post-operation. Four patients had one year follow-up with three patients maintaining hearing and the fourth one redeveloping conductive hearing loss at three months post-operatively. This particular patient was a revision of a previous stapedectomy (performed by a different surgeon) with almost the exact same post-operative course. None of the patients developed sensorineural hearing loss. The average operative time was 48 minutes and was comparable to the surgeon's previous operative time with the microscope. The main advantage is a better visualization and control of footplate drilling. There are no compelling reasons to perform stapedectomy with the endoscope except for the surgeon's choice and preference. As more clinicians develop the necessary skills, the endoscope will be the instrument of choice in stapedectomy for many surgeons. A video clip of the surgery will be shown.

---

Private practice, 3535 30th Avenue, Suite 204, Kenosha, Wisconsin 53144.

## BIOGLASS MIDDLE EAR PROSTHESIS: LONG-TERM RESULTS

*Kevin R. Rust, George T. Singleton, \*June Wilson, and Patrick J. Antonelli*

### **ABSTRACT**

The purpose of this study was to review the University of Florida's long-term results with Bioglass middle ear prostheses. Between April 1984 and November 1987, 37 patients were implanted with Bioglass prostheses (25 total and 12 partial ossicular replacements). Twenty-one patients had postoperative data of at least 24 months (range, 24 to 126 months; mean, 86 months; median, 100 months), and five patients had >10 years' follow-up. In three cases, portions of fractured prostheses extruded, leaving an intact tympanic membrane. One patient with a total ossicular prosthesis was reexplored at 38 months for conductive hearing loss and found to have a prosthesis fracture ( $n = 1$ ). There were no extrusions of intact prostheses, even in patients in whom the prosthesis was placed directly under the tympanic membrane or graft ( $n = 12$ ). After 24 months, the mean pure-tone average air-bone gap was 24 dB (24% had ABG  $\leq 10$  dB; 53% had ABG  $\leq 20$  dB). Air-bone gap closures were stable over time. Our results demonstrated that Bioglass middle ear prostheses have excellent long-term tissue compatibility. The four failures are attributed to fractures in early experimental prototypes.

---

Department of Otolaryngology and the \*Bioglass Research Center, University of Florida, Gainesville, Florida, U.S.A.

Presented at the American Otologic Society Annual Meeting, April 29–30, 1995, in Palm Desert, California.

Reprint requests: Dr. P.J. Antonelli, Department of Otolaryngology, University of Florida, Box 100264, Gainesville, Florida 32610, U.S.A.

# LONG-TERM RESULTS USING OSSICULAR GRAFTS

*Jay B. Farrior and Stacy W. Nichols*

## ABSTRACT

Sculpted autologous ossicle and cortical bone grafts were the first materials successfully used to reconstruct the ossicular chain in chronic ear surgery. Over the last 20 years, the use of biocompatible implants has been popularized; as a result, bone grafts have fallen into disfavor with most otologists. To determine if autologous bone grafts remain stable with time, 115 cases in which autologous bone grafts were used between 1971 and 1984 were reviewed. Eighty patients underwent Type III tympanoplasty, stapes arch present. Thirty-five underwent Type IV tympanoplasty, stapes arch absent. Minimum follow-up was 2 years; 30 patients were followed for  $\geq 10$  years. In Type III tympanoplasty, overall the initial air/bone gap was 19.7 dB at 6 months, with 59% of those with improved hearing at 15 dB air/bone gap or better. Hearing remained stable for 10 years with overall hearing of 19.2 dB air/bone gap and 50% with an air/bone gap of  $\leq 15$  dB. In Type IV tympanoplasty, the average air/bone gap was 26 dB at 6 months, with 70% of those having improved hearing with  $\leq 20$  dB air/bone gap. At 10 years, the overall air/bone gap was 29.3 dB, with only 28% maintaining an air/bone gap of  $\leq 20$  dB. Poor eustachian tube function and collapse of the middle ear air space were found to be the primary causes for long-term failure. The initial hearing results using autologous bone are comparable with those achieved with synthetic prosthesis. Hearing results using autologous bone remained stable through 5 years. Beyond 5 years, Type III tympanoplasty remained stable, while there was deterioration in Type IV tympanoplasty due to poor eustachian tube function.

---

University of South Florida, Tampa, Florida.

Presented at the 128th Annual Meeting of the American Otological Society, April 29–30, 1995, Palm Desert, California.

Reprint requests: Dr. J.B. Farrior, 509 West Bay Street, Tampa, Florida 33606, U.S.A.

# THE USE OF EVOKED POTENTIAL RECORDINGS AND STAPES DISPLACEMENT MEASUREMENTS TO EVALUATE THE IN VIVO FUNCTION OF AN IMPLANTABLE ELECTROMAGNETIC MIDDLE EAR TRANSDUCER

*Thomas C. Robey, Douglas A. Miller, Alec N. Salt, and John M. Fredrickson*

## **ABSTRACT**

An electromagnetic middle ear transducer, implanted in a rhesus monkey model, was mounted in the temporal bone just postero-superior to the external ear canal and was coupled to the body of the incus. Each animal was followed chronically with auditory brainstem responses and otoacoustic emissions for at least 6 months prior to final assessment. As an additional means to assess function of the implanted middle ear device, the output of the middle ear transducer was determined by measuring the displacement of the stapes with a fiber optic lever and by recording evoked potentials at the round window. These results were then compared with those recorded in response to acoustically generated input in the same animal. To obtain these measurements, the animal was anesthetized and access to the middle ear cleft was gained via an inferior mastoid air cell approach. With the round window and the stapes exposed, hemostasis was achieved and the fiber optic lever positioned just medial to the incudostapedial joint. A continuous swept tone of 500 to 10,000 Hz served as both the acoustic and mechanical input. The acoustic input was delivered by an insert receiver coupled to a hollow metal ear bar placed in the bony external auditory canal. When mechanically driven by the middle ear transducer, stapes displacement amplitude was measured to be maximal at 4 kHz and was within 15 dB of this output over the entire input frequency range. This suggests that the middle ear transducer has good sound fidelity from 500 to 10,000 Hz. To obtain evoked potentials, a silver ball electrode was placed on the round window with a differential electrode at the vertex and a reference electrode at the neck.

The acoustic and mechanical input consisted of a 7 millisecond pure tone burst delivered in quarter octave steps over a frequency range of 500 to 8000 Hz. Action potentials (APs) were then recorded for both acoustically generated and mechanically coupled input. At 2 kHz, the direct mechanical stimulation of the ossicles by the transducer with a 1 volt input produced AP responses equivalent to those generated by an acoustic input of 136 dB SPL in the external ear canal.

---

Washington University Medical Center, 517 South Euclid Avenue, St. Louis, Missouri  
63110-1007.

## DISCUSSION PERIOD THREE

### Papers 9–14

**Dr. Robert Jahrsdoerfer** (Houston, Texas): We have time for some discussion.

**Dr. Stephen Harner** (Rochester, Minnesota): First of all I would like to congratulate Dr. Berliner on an excellent presentation. I also urge that program organizers and journal editors make an effort to put results into a common format so we can compare apples to apples. One further comment—I noticed in Dr. Farrior's presentation that in the type III tympanoplasty, success was defined as a 15 decibel or better air-bone gap, and for type IV tympanoplasty, it was set at 20 decibels, which means that we are not using the same criteria for success in the two different procedures, at least as I have interpreted it.

**Dr. Jean Bernard Causse** (Beziers, France): I would like to make a short comment on two excellent presentations. The first is on the use of the laser in revision stapedectomy. I really think that this is the place for the laser. The laser is most useful when there is fibrous tissue in the oval window, and the only failure occurs when fibrous tissue has penetrated into the anterior labyrinth. Then the laser will not help. If not, then this is really the moment to use the laser. My second comment is on using the endoscope for otosclerosis surgery. I would like to congratulate the presenter for an excellent presentation. He just proved that one should not use the endoscope. A few months ago, Dr. Silverstein and I tried this just to prove that actually lot of things can be done using the endoscope, except stapes surgery. Using both hands at the same time as using a speculum holder is extremely important, as well as having a good binocular view of the footplate. This paper is very useful because it helps prove this point.

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Thank you. Gordon Hughes.

**Dr. Gordon B. Hughes** (Cleveland, Ohio): I congratulate the authors of these papers and I do not direct my comments to anyone in particular, except to point out that previous publications have documented clearly that autologous bone is the best thing to use in ossicular reconstruction in chronic ear surgery, and also the technique used to compare pre and postop hearing in stapes replacement surgery

will determine the final measurement of the result. Instead of producing postop air bone gaps I suggest that we, in the future, try to concentrate on the definitions of success. Most stapes surgeons, for example, propose that 90% of patients should achieve a 10 decibel air-bone gap closure, or better, with less than 1% sensorineural hearing loss. In chronic ear surgery, it is well known that the technique is not as important as the skill of the surgeon and the disease of the ear; however, a 20% decibel result 50% of the time for five years or longer is acceptable in most severe disease. When I wrote a paper a few years ago on the learning curve in stapes surgery, and got a lot of advice, I generally learned from the masters that one should compare both the postop air with preop bone, to avoid missing a sensorineural hearing loss when there was complete closure postop, and compare the postop air with the postop bone, which is the more realistic measure to avoid the ambiguity of overclosure. So, to repeat, I think we should begin to focus on the definitions of good results rather than bantering about the postop air-bone gaps.

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Jay, do you want to respond to those comments?

**Dr. Jay Farrior** (Tampa, Florida): Regarding postoperative results in chronic ear surgery, the definition of success varies widely among surgeons. Dr. Wehrs has one of the tightest criteria, at 20 decibels. Dr. Brackmann and Dr. Sheehy came out with a paper in 1983 or 1984 that defined 15 decibels as satisfactory for type III tympanoplasty, which most people agree has a higher degree of success than type IV, and their criterion for success was 25 decibels for type IV tympanoplasty. Dr. Glasscock and Dr. Jackson published criteria using 30 decibels. So, the problem is the criteria for what is successful varies widely from author to author over the past twenty years or so. What I tried to do is to pick some fairly stringent criteria and also what I consider to be acceptable when I am talking to patients. Thank you.

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Would you like to respond, Dr. Haberkamp?

**Dr. Thomas Haberkamp** (Milwaukee, Wisconsin): I wanted to thank Dr. Causse for his comments.



I left it out because I did not think we would have time. We thought that the reason for such a dismal prognosis in the excessively long prosthesis was probably because they had some fibrosis within the vestibule that we could not address with or without lasers. Thank you very much, Dr. Causse.

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Dr. Rodney Perkins.

**Dr. Rodney Perkins** (Palo Alto, California): I would like to comment on Dr. Tarabichi's paper. I would say that I am very impressed with the potential of the endoscope, more than what we see here. As opposed to Dr. Causse in saying this is not potentially

the way to do things (I think he is right in stapedectomies and middle ear areas), if you look at the areas that went on earlier, such as labyrinthine surgery, you are getting down to finite areas for stereoscopic surgeries and entry may not be possible. I think that some of these procedures in the future will probably be done endoscopically with very small endoscopes going into the middle ear and that will be the method of observation. So, although all these look a little "clugy" at the present time, and there are some limitations, for example, the two handed approach, etc, I think this is a pioneering work and I would like to congratulate the author. Thank you.

## LASER DOPPLER VIBROMETRY (LDV) A NEW CLINICAL TOOL FOR THE OTOLOGIST

*Richard L. Goode*

### **ABSTRACT**

The laser Doppler vibrometer (LDV) has been used for many years in the research laboratory to measure ossicular velocity and displacement in human temporal bones and live subjects. It has only been recently that the technology has developed to the point where clinical applications can be considered, including the diagnosis of certain types of sensorineural hearing loss. The LDV is a very sensitive, non-contacting optical measurement system capable of making displacement measurements down to 0.001 micron at frequencies up to 1.5 mHz. It uses a helium-neon laser aimed at any vibrating site through an operating microscope. The reflected beam from the target site is analyzed in the detector portion of the system using the Doppler principle, producing an output voltage proportional to the velocity of the target. The target can be quite small, less than 1.0 millimeter in diameter. A sound generating system is required to produce and maintain a constant sound pressure level at the tympanic membrane (TM) at 200–6000 Hz. The measurement takes less than one minute. The LDV has the potential to provide important information about the acoustic-mechanical function of the ear that cannot be obtained in any other way and that could begin a new era in otologic diagnosis and treatment. Experience with the LDV in patients and temporal bones has shown significant individual variation in umbo displacement at key hearing frequencies in response to a constant sound pressure input. These differences appear due to differences in TM acoustic function; some TMs are much better than others. As would be expected, umbo displacement is decreased in ears with obviously damaged TMs, such as perforation or extensive tympanosclerosis; these TM abnormalities usually produce a conductive hearing loss. What is not well known is that a large percentage of TMs with commonly seen minor abnormalities (scars, monomeric membranes, retraction pockets, etc), previously thought to be acoustically innocuous, also have abnormally low umbo displacements but do not have a conductive hearing loss on air-bone testing. These patients may have mild to moderate hearing losses that appear entirely or mostly sensorineural. It appears that there is a mechanical component contributing to the loss that can be up to 25 dB and can be identified with LDV. By knowing of the existence

and the probable cause of this type of loss, surgical correction can be considered. Inefficiency in the transmission of vibration from malleus to stapes is also present in many ears at about 1.0 kHz and contributes to abnormal hearing thresholds at higher frequencies. It appears that this is due to excessive translational (in and out) movement of the rotation axis of the malleus and incus; LDV assessment of short process displacement can analyze the extent of this inefficiency, which is also potentially correctable by surgery. The LDV system can also be useful in the operating room to determine ossicular fixation as well as prosthesis function. Details of the technique and clinical experience to date will be provided in the paper.

---

Stanford University Hospital, 300 Pasteur Drive, R-135, Palo Alto, California 94025.

## OSSEOINTEGRATION AND GROWTH EFFECTS OF TEMPORAL BONE PERCUTANEOUS PEDESTALS

*James L. Parkin, \*Roy Bloebaum, Brett D. Parkin, and Matthew J. Parkin*

### ABSTRACT

The percutaneous temporal bone pedestal has shown significant utility for the attachment of bone-anchored hearing aids, attachment of cosmetic auricular prostheses, and as connector between external sound processors and implanted cochlear implants. The biological acceptance of these implants by temporal bone hosts is affected by many factors including the maturity of the bone, the design of the pedestal fixation system, and the pedestal construction material. The first phase of this study evaluated the effect of the pyrolyzed graphite pedestal fixation on maturing temporal bones. Pedestals were implanted in young swine temporal bones using single screw and multiple screw fixation systems. The effect on temporal bone growth is demonstrated with photomicrographs and gross photography showing acceptable host-tissue response to the presence of multiple and single screw attachment techniques. Osseointegration of the attaching screws occurred. The second phase of the study evaluated osseointegration of smooth, beaded, and textured titanium pedestals in feline temporal bones. High-resolution temporal bone/pedestal sectioning has been accomplished with high-performance microtomes, showing the osseointegration of the pedestal by the temporal bone. This is demonstrated with tetracycline labeling and histologic assessment. Percutaneous pedestals are of increasing importance in otologic practice. This study assists in the understanding of biologic acceptance of pedestals as influenced by the pedestal composition and fixation design. This basic understanding is essential for design improvements in percutaneous temporal bone pedestals.

---

Division of Otolaryngology–Head and Neck Surgery, University of Utah Health Sciences Center, and \*Bone and Joint Research Laboratory, Department of Veterans Affairs Medical Center, Salt Lake City, Utah, U.S.A.

Reprint requests: Dr. J.L. Parkin, Division of Otolaryngology–Head and Neck Surgery, University of Utah Health Sciences Center, 50 North Medical Drive, Salt Lake City, Utah 84132, U.S.A.

# MAGNETIC RESONANCE IMAGING IN IDIOPATHIC SUDDEN SENSORINEURAL HEARING LOSS

*George A. Gates, Todd Richards, Jay Tsuruda, and Edwin W. Rubel*

## **ABSTRACT**

Idiopathic sudden sensorineural hearing loss (ISSHL) remains an etiologic enigma. Two likely theories of etiology involve a) hypoperfusion of the cochlea and b) viral cochleoneuritis. Study of patients with ISSHL has been hampered by the lack of anatomic corroboration of the site of lesion. Modern magnetic resonance imaging (MRI) using phased array coil technology provides unparalleled quality in imaging the cochlea, vestibule, and contents of the internal auditory canal. We present the MR findings in 6 cases of ISSHL for whom complete audiometric assessment including ABR and otoacoustic emission testing has been done as part of a prospective study. The preliminary findings suggest a cochlear site of lesion in the majority of cases.

---

University of Washington, 1959 Northeast Pacific Street, RL-30, Seattle, Washington 98195.

## THE USE OF THE TEMPOROPARIETAL FASCIAL FLAP IN TEMPORAL BONE RECONSTRUCTION

*Mack L. Cheney, Cliff A. Megerian, Mark T. Brown, Michael J. McKenna, and Joseph B. Nadol, Jr.*

### ABSTRACT

After routine canal wall down mastoidectomy, local muscle flaps with and without bone paté, cartilage and fascia are the standard techniques available to otologists wishing to obliterate the mastoid and reconstruct the external auditory canal. Reconstructive options for temporal bone defects after extirpative surgery for cancer, osteoradionecrosis, and revision surgery for chronic granulomatous otitis media, however, are few. Although the neighboring temporoparietal fascia flap (TPFF), based on the superficial temporal vessels, has been frequently employed for auricular reconstruction, its versatility in temporal bone reconstruction has not been widely explored. The TPFF has recently been employed at our institution in 11 patients who presented with a variety of reconstructive problems, including defects after temporal bone resection, surgery for malignant otitis externa, and revision mastoid surgery. Follow-up in these patients ranged from 1 to 43 months (average 18.4 months) and surgical objectives of achieving a dry mastoid bowl, fully epithelialized canal, and/or reduction of mastoid cavity volume was attained in 100% of cases. The TPFF offers many advantages to the otologic surgeon when faced with reconstruction dilemmas that center around a poorly vascularized mastoid cavity and temporal bone. The TPFF is a reliable source of local well-vascularized tissue that is extremely pliable and facilitates both hearing and nonhearing preservation temporal bone reconstruction.

---

Department of Otolaryngology, Massachusetts Eye and Ear Infirmary and the Department of Otology and Laryngology, Harvard Medical School, Boston, Massachusetts, U.S.A.

Results of this study were presented at the Annual Meeting of the American Otologic Society, May 1995, Palm Desert, California.

Reprint requests: Dr. M.L. Cheney, Department of Otolaryngology, Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston, Massachusetts 02114, U.S.A.

## INVASION PATTERNS OF ADVANCED TEMPORAL BONE MALIGNANCIES

*John P. Leonetti, †Peter G. Smith, †G. Robert Kletzker, and \*Ricardo Izquierdo*

### ABSTRACT

Primary malignancies of the temporal bone may originate in the external auditory canal, the middle ear, the endolymphatic sac, or the eustachian tube. The surgical treatment of advanced tumors in these regions is strictly dependent upon the radiographic delineation of disease extent and the tumor relationship to adjacent neurovascular structures. Twenty-six cases of stage III or IV squamous cell carcinoma of the temporal bone were retrospectively reviewed to correlate preoperative clinicoradiographic analysis with intraoperative findings. The following patterns of tumor invasion were identified: (a) superior erosion through the tegmen tympani into the middle cranial fossa; (b) anterior extension into the glenoid fossa and infratemporal space; (c) inferior growth through the hypotympanum and jugular foramen; (d) posterior involvement of the mastoid air cells; and (e) medial involvement of the middle ear and carotid canal. While otic capsule erosion was uncommon, several of these patients did present with lower cranial nerve palsies. Complex surgical procedures exist for the en bloc resection of advanced temporal bone cancers. Appropriate operative planning must be based upon a knowledge of potential patterns of tumor extension and meticulous radiographic assessment.

---

Departments of Otolaryngology–Head and Neck Surgery and \*Plastic Surgery, Loyola Center for Cranial Base Surgery, and †Midwest Otologic Group, Maywood, Illinois, U.S.A.

Reprint requests: Dr. J.P. Leonetti, Department of Otolaryngology-HNS, Loyola University Medical Center, 2160 S. First Ave., Bldg. 105, Rm. 1870, Maywood, Illinois 60153, U.S.A.

## DISCUSSION PERIOD FOUR

### Papers 15–19

**Dr. Robert Jahrsdoerfer** (Houston, Texas): The previous five papers are now open for discussion.

**Dr. Brian Blakley** (Detroit, Michigan): The question I have is for Dr. Goode. His paper was excellent, and I think there should be more study of the acoustics of the middle ear. I wonder where did he get his equipment from—the laser Doppler/vibrometer; did the engineers at his institution develop these, or modify them from some other source? Are they commercially available? The enhancements that he has shown us, are they commercially available?

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Dr. Goode, would you like to respond?

**Dr. Richard Goode** (Palo Alto, California): The laser Doppler/vibrometer we used is commercially available and is made by Polytech, although there are other manufacturers. It only makes the vibrometer; you also need the sound producing system that ties in. Then it is also nice to have something to decrease the noise, because swallowing movement of the head can be a problem. All of this equipment is commercially available.

The material we have here is immense, when you think about phase and multiple sites of the eardrum, so I did not go through and analyze all that. We are going through those data now.

We will be able to do the same analysis of live ears, both before and after surgery, with the multi-scanning. That equipment, though, I borrowed, because it is twice as expensive as the single spot one.

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Dr. Ruckenstein.

**Dr. Michael Ruckenstein** (San Diego, California): I have a question for Dr. Leonetti. Presumably recognition of undercalling of the extent of the carcinoma on radiologic evidence at the time of operation meant that you operated on those areas and

tried to get a total resection. Were you implying by your talk that the underestimation on radiologic evidence predisposed in and of itself to recurrence, or was it just that this showed there is very aggressive disease?

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Thank you. We take the other question from the floor now.

**Dr. Moises Arriaga** (Lackland AFB, Texas): I have another question for Dr. Leonetti, following along the same lines. What was the extent of surgery in those patients in whom the underestimation occurred? Namely, were these people who could have had a larger resection—were they among the group with the total temporal bone resections, or were they the ones with the lesser extent where perhaps a larger resection could conceivably have prevented a local recurrence?

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Other questions or comments from the floor. John, would you care to respond, please.

**Dr. John Leonetti** (Maywood, Illinois): Maybe I can answer both of those questions with one sentence. The main goal of looking at the patterns of invasion was to determine whether we should have planned a different surgical resection based upon the preoperative radiographic assessment. For example, instead of doing a type two resection, had we actually known that there was disease in the mastoid mucosa, perhaps we would have proceeded with a total temporal bone resection, knowing that there are other factors involved with the patient's overall prognosis, overall survival and local cure. But specifically, we are trying to determine if there is anything radiographically that would have changed what we did before we entered the operating room.

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Thank you. We will continue with the scientific program.



## NEUROPHYSIOLOGICAL APPROACH TO TINNITUS PATIENTS

*Pawel J. Jastreboff, William C. Gray, and Susan L. Gold*

### **ABSTRACT**

The principal postulate of the neurophysiological model of tinnitus is that all levels of the auditory pathways and several nonauditory systems play essential roles in each case of tinnitus, stressing the dominance of nonauditory systems in determining the level of tinnitus annoyance. Thus it has been proposed to treat tinnitus by inducing and facilitating habituation to the tinnitus signal. The goal is to reach the stage at which, although patients may perceive tinnitus as unchanged when they focus on it, they are otherwise not aware of tinnitus. Furthermore, even when perceived, tinnitus does not evoke annoyance. Habituation is achieved by directive counseling combined with low-level, broad-band noise generated by wearable generators, and environmental sounds, according to a specific protocol. For habituation to occur, it is imperative to avoid masking tinnitus by these sounds. Since 1991, >500 tinnitus patients have been seen in our center. About 40% exhibited hyperacusis to varying degrees. A survey of >100 patients revealed >80% of significant improvement in groups of patients treated with the full protocol involving counseling and the use of noise generators. Notably, in patients who received counseling only, the success rate was <20%. The improvement in hyperacusis was observed in ~90% of treated patients.

---

Tinnitus and Hyperacusis Center, Department of Surgery, University of Maryland School of Medicine, Baltimore, Maryland, U.S.A.

Presented at the 128th Annual Meeting of the American Otological Society, Palm Desert, California, April 29–30, 1995.

Reprint requests: Dr. P.J. Jastreboff, Department of Surgery, University of Maryland School of Medicine, 10 S. Pine Street, MSTF Bldg, Rm 434F, Baltimore, Maryland 21201, U.S.A.

## PATIENT PERFORMANCE WITH THE COCHLEAR CORPORATION "20 + 2" IMPLANT: BIPOLAR VERSUS MONOPOLAR ACTIVATION

*Teresa A. Zwolan, Paul R. Kileny, Carissa Ashbaugh, and Steven A. Telian*

### **ABSTRACT**

A within-subjects comparison of monopolar versus bipolar stimulation was performed using a modified version of the Nucleus mini-22 cochlear implant, the Nucleus "20 + 2" implant. Six subjects underwent implantation with this device, which is identical to the Nucleus 22 cochlear implant with the addition of two extracochlear indifferent electrodes. These electrodes provide two monopolar modes of stimulation in addition to the standard bipolar modes used with the Nucleus 22 device. One of the indifferent electrodes is a ball placed under the temporalis muscle (MP1), whereas the second electrode is mounted on the lateral aspect of the receiver-stimulator (MP2). After a pre-experimental phase, subjects used each of three stimulation modes (BP + 1, MP1, and MP2) for a total of 4 weeks each. Variables tested with each mode included electric thresholds, comfort levels, dynamic ranges, and speech recognition. Both the MP1 and MP2 modes of stimulation required significantly less current than the bipolar mode (BP + 1) to reach threshold and comfort level. Analysis of dynamic range data also indicated a significant stimulation mode effect. However, stimulation in the various modes did not significantly affect speech recognition scores, although two subjects demonstrated significantly improved speech recognition scores when programmed in a monopolar mode.

---

Department of Otolaryngology, University of Michigan, Ann Arbor, Michigan.  
Presented at the Annual Meeting of the American Otological Society, Palm Desert, California, April 30, 1995.

Address correspondence and reprint requests to Dr. Teresa A. Zwolan, Cochlear Implant Program, University of Michigan, 475 Market Place, Bldg. 1, Suite A, Ann Arbor, Michigan 48108, U.S.A.

## DEFINING FUNCTIONAL LIMITATION, DISABILITY, AND SOCIETAL LIMITATIONS IN PATIENTS WITH FACIAL PARESIS: INITIAL PILOT QUESTIONNAIRE

*\*J. Gail Neely and †Peggy S. Neufeld*

### **ABSTRACT**

Experiences with patients with facial paralysis over the last 25 years and recent efforts to develop objective measures of paresis and synkinesis led us to three hypotheses: (a) dysfunction in certain regions of the face is more disturbing than that in others, (b) there are major psychosocial impacts of facial paralysis, and (c) the impact of facial paralysis is underestimated. An initial questionnaire of 10 open-ended items was submitted to 11 subjects stabilized after acoustic tumor resection. Responses were tabulated qualitatively, and frequency counts were made of responses. These results show that the region of the face that is most disturbing is the mouth; however, early in the time course of paralysis, the eye is most disturbing. Synkinesis about the eye is ultimately more disturbing than paresis in that region, and it may worsen. Major psychosocial impacts of paralysis appear common and underestimated. These pilot data qualitatively support the hypotheses.

---

\*Department of Otolaryngology–Head and Neck Surgery and †Occupational Therapy Program, Washington University School of Medicine, St. Louis, Missouri, U.S.A.  
Presented at the American Otological Society Annual Meeting, April 29–30, 1995, Palm Desert, California.

Reprint requests: Dr. J.G. Neely, Washington University School of Medicine, 517 S. Euclid Ave Box 8115, St. Louis, Missouri 63110, U.S.A.

## THE VARIABLE RELATIONSHIP BETWEEN THE LOWER CRANIAL NERVES AND JUGULAR FORAMEN TUMORS: IMPLICATIONS FOR NEURAL PRESERVATION

*\*Lawrence R. Lustig and \*†Robert K. Jackler*

### ABSTRACT

Tumors involving the jugular foramen (JF) have a variable relationship to the neurovascular structures (jugular vein, cranial nerves IX–XI) that traverse this conduit through the skull base. The surgeon familiar with the site of origin, growth pattern, and geometry of each of the common lesions affecting this region with respect to surrounding nerves and vessels is at a considerable advantage when undertaking a function-sparing procedure. Anatomically, the JF has two vascular compartments that may be affected by tumor: the jugular bulb laterally and a passage for the inferior petrosal sinus medially. Tumors may also penetrate the JF along the fibro-osseous diaphragm, which divides these two vascular channels. The lower cranial nerves lie on either side of this partition, which is connected to the posterior cranial fossa via a curved, funnel-shaped cone of dura. Tumors that arise within or penetrate the JF lateral to this neural plane displace the nerves medially, a position favorable for their preservation during tumor extirpation. By contrast, medially positioned tumors displace the cranial nerves onto the lateral tumor surface, where they interpose between surgeon and tumor—an unfavorable location. Glomus tumors consistently arise in the lateral aspect of the JF, displacing the lower cranial nerves medially. This positioning accounts for the high rate of neural preservation in small and medium-size glomus tumors that have not invaded the foramen's central partition. Meningiomas that arise lateral to the JF (e.g., the posterior petrous surface, sigmoid sinus) favorably displace the lower cranial nerves medially. By contrast, tumors that originate medial to the JF (e.g., clivus, foramen magnum) are unfavorable, laterally displacing the multiple small rootlets that coalesce into cranial nerves IX–XI into a vulnerable location. Schwannomas arise within the neural plane and have a variable geometry that depends, in part, upon the nerve of origin. Theoretically, tumors that arise from the ninth nerve, which is located on the lateral surface of the neural plane, should be more favorable than those originating from the tenth or eleventh nerves, which lie on its deep surface. The propensity of these three tumor types toward thrombosis of the

jugulosigmoid complex also carries important surgical implications. Because glomus tumors arise from the jugular bulb, the jugulosigmoid complex is nearly always occluded. In both meningiomas and schwannomas, however, the jugular system may occasionally remain patent. This is important to recognize through angiography and/or magnetic resonance venography, since sacrifice of a patent, dominant system risks intracerebral venous infarction.

---

Departments of \*Otolaryngology–Head and Neck Surgery and †Neurological Surgery,  
University of California, San Francisco, California, U.S.A.

Presented in part at the Meeting of the American Otological Society, Palm Desert, California, April 29–30, 1995.

Reprint requests: Dr. Robert K. Jackler, 350 Parnassus Avenue, no. 210, San Francisco, California 94117, U.S.A.

## HEARING CONSERVATION IN SURGERY FOR GLOMUS JUGULARE TUMORS

*C. Gary Jackson, David S. Haynes, Paul A. Walker, Michael E. Glasscock III,  
S. Storper, and Anne Forrest Josey*

### ABSTRACT

The most common ground on which surgery for glomus jugulare (GJ) tumors is criticized is the perceived risk of functional incapacity that attends possible cranial nerve (CN) loss. It is aggregate lower CN loss that is most often highlighted as particularly disabling to the quality of post-surgical survival. The documented success of both conservation surgery and operative rehabilitation of phonopharyngeal surgical deficits has, however, neutralized much of this criticism. The issue of hearing conservation (HC) in neurotologic skull base surgery, on the other hand, has not been well documented toward this end. The presence of a GJ neoplasm need not reflexly nor technically forfeit preexisting hearing. HC is, admittedly, a subordinate priority to total tumor removal, successful distal control of the internal carotid artery and even facial nerve integrity. Yet, in appropriately selected patients, existing operative technology permits hearing preservation, a noteworthy addition to the high-grade functional outcome we have come to reasonably expect of conservation surgery. Hearing salvage further serves to define the concept of neurotologic skull base surgery. Hearing preservation in 122 GJ tumor patients is reviewed. Intuitively, as for acoustic tumor, HC appears tumor size related. Selection criteria for conservation surgery and its operative technique are detailed. Outcome is appropriately scored. The radiation therapy literature on this subject will be assiduously scrutinized for comparison.

---

Otology Group, Nashville, Tennessee, U.S.A.

This work was presented at the 128th Annual Meeting of the American Otological Society, Palm Desert, California, U.S.A., April 29–30, 1995.

Reprint requests: Dr. C.G. Jackson, the Otology Group, 300 20th Ave. N., Suite 502, Nashville, Tennessee 37203, U.S.A.

## DISCUSSION PERIOD FIVE

### Papers 20–24

**Dr. Robert Jahrsdoerfer** (Houston, Texas): The floor is open to discussion of the preceding five papers.

**Dr. John Niparko** (Baltimore, Maryland): I have two questions for Dr. Terry Zwolan. Terry, were there effects of monopolar versus bipolar stimulation on electrode differentiation in pitch ranking? Secondly, did the patients with better performance with monopolar stimulation have a longer duration of deafness?

**Dr. Terry Zwolan** (Ann Arbor, Michigan): We have just started to look at place-pitch discrimination, and we do see a trend for better place-pitch discrimination with bipolar stimulation. We have done some other studies in our laboratory that suggest that better place-pitch discrimination is not positively correlated with better speech recognition, as many people believe. So, that is sort of an interesting finding in itself. Regarding your second question, I do not know, to be honest with you, about the duration of deafness. I would have to look at that, but from eyeballing it, the first patient who had the biggest effect did have a very long duration of deafness. So, it might be that is why she demonstrated a better monopolar performance.

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Any other questions or comments?

**Dr. John Niparko** (Baltimore, Maryland): I have a question for Dr. J. Gail Neely. Did the age or

level of education affect the perception of facial paresis?

**Dr. J. Gail Neely** (St. Louis, Missouri): We did not systematically study that in this pilot questionnaire. When you start digging into this, the permutations and combinations are just overwhelming. There are many disassociations to intuition, meaning that your intuition might lead you to conclude that age would have an effect and a cursory look at the data says no.

**Dr. James S. Brown** (Calgary, Canada): I would like to compliment Dr. Jackson on his presentation on glomus jugulare tumors. Looking at his results for preservation of hearing, it is still important to make an early diagnosis, because the smaller tumors give you the opportunity to preserve hearing. These are innocent tumors, but they are important because of their location; a complete excision is still a treatment of choice. Certainly in Canada radiotherapy seems to be a lot more, and has been over the years a lot more, the choice procedure. If you look at the 15 and 20 year results of cases in your series that have had radiotherapy, certainly the long term results are very important and very discouraging, because the radiotherapy, over a long period of time, particularly to the base of the skull area, causes a lot of severe damage. Thank you.

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Thank you. Any other comments or questions?

## THE DIAGNOSIS OF INTRA-AXIAL POSTERIOR FOSSA LESIONS

*Arvind Kumar, Marlos A.G. Viana, and Albert Pieri*

### ABSTRACT

Dizziness, disequilibrium, vertigo, and hearing loss can be caused by lesions of the posterior fossa. Today with modern imaging techniques, it is possible to non-invasively detect such lesions as well as establish their tissue characteristics. However, in many instances the cause of the symptoms is not in the posterior fossa and imaging studies are negative. The work-up in such cases is not cost effective. Consequently the need for screening tests which reliably localize the lesion to the posterior fossa is a continuing need. Since extra-axial lesions of the posterior fossa have a potential for surgical cure, the screening tests for lesions of this region are now at a high level of sophistication and imaging is considered early in the work-up. Intra-axial lesions on the other hand are more difficult to diagnose and subtle abnormalities of the posterior neuro-axis (PNA) are either missed or disregarded because of a lack of good clinico-radiological correlation. In an effort to address this issue, and to overcome the inherent limitations of the caloric test, a variety of newer, microprocessor based tests have been developed. In principle, they test the vestibulo-ocular reflex, the saccadic system, the smooth pursuit system and the balance system as a whole. These newer tests include computerized rotational tests, auto-rotational tests, tests of oculomotor function and posturography. The purpose of this study was to do meta-analysis of the literature which has reported the diagnostic yield of these newer tests as well as those reported for the caloric test. The objective was to determine if the stated conclusions were valid. The results of this review show that the simple caloric test still provides the most reliable and cost-effective topodiagnostic information. The second objective of this study was to test the validity of this conclusion. To this end, we retrospectively reviewed the Torok monothermal caloric test results of all patients with magnetic resonance imaging (MRI) confirmed lesions of the PNA. The PNA lesions confirmed in these 70 patients included Type I Chiari malformation (30), brain stem/cerebellar infarct (10), vertebro-basilar insufficiency (2), arterio-venous malformation (2), multiple sclerosis (9), IVth ventricle cyst (1), arachnoid cyst (1), interhemispheric epidermoid (1), active cysticercosis (1), pontine venous angioma (1), Dandy-Walker cyst (1), basilar impression (2), cerebellar metastases (1), cere-



bellar atrophy (1), autoimmune encephalopathy (1). The data from these 70 patients was analyzed and vestibular decruitment (VDEC) was noted in 59 patients. In previous reports, we have shown VDEC is a reliable sign of posterior fossa lesions and from this review, we find that the sensitivity of the test is 84%. To establish the specificity of the test, we examined 18 normal healthy subjects. The caloric results were normal in all these subjects and VDEC was found in none. On the basis of this study, we conclude that the Torok Monothermal caloric test is a valuable screening test for defining PNA pathology and VDEC is a valid clinical sign, even though diverse pathologic lesions involving different anatomic sites of PNA provide the same result.

---

University of Illinois, Eye and Ear Infirmary, 1855 West Taylor Street, Chicago, Illinois  
60612-7242.

## HISTOLOGIC EVALUATION OF AERATION ROUTES IN TEMPORAL BONES WITH CHOLESTEATOMA

*Atsushi Haruta, Patricia A. Schachern, Tetsuya Tono,  
Michael M. Paparella, and Tamotsu Morimitsu*

### ABSTRACT

Aeration disorder of the middle ear and mastoid is one of the most important causes of acquired cholesteatoma. Proctor (1964) described two aeration routes between the middle ear and mastoid, the so-called anterior and posterior tympanic isthmus routes. In most cases of cholesteatoma surgery, the aeration route between the eustachian tube and mastoid is observed to be obstructed by cholesteatoma, granulation tissue and/or effusion. However, in some patients an aerated region in the middle ear and mastoid air cells is revealed, indicating the existence of an aeration route from the Eustachian tube to that region. The purpose of this study was to determine the area of aeration in the middle ear and mastoid, and to evaluate the three possible routes of aeration from the eustachian tube to mastoid; via the anterior tympanic isthmus, the posterior tympanic isthmus, and directly to the attic. Ten temporal bones from patients with no or conservative treatment for cholesteatoma were collected at autopsy, processed routinely in celloidin, and examined by light microscopy. To determine the area of aeration, the middle ear was divided into five areas:

1. The anterior area of the mesotympanum and hypotympanum—AA
2. The posterior area of the mesotympanum and hypotympanum—PA
3. The supratubal recess—SR
4. The epitympanum—ET
5. The mastoid antrum and air cells—MA

Cases with an aerated mastoid were further evaluated to determine the route of aeration between the eustachian tube and mastoid. The results of this study were as follows: All cases (100%) were aerated in the AA; eight cases (80%) revealed aeration in the SR; three cases (30%) showed aeration in the PA; four cases (40%) showed aeration in the ET; five cases (50%) revealed aeration in the MA. Of the five aerated mastoid cases which were further investigated to determine the aeration route between the eustachian tube and mastoid, two cases were aerated through a patent anterior tympanic isthmus, one case was

aerated directly from the supratubal recess to the epitympanum, and one case was aerated directly from the mesotympanum to the epitympanum through a perforation in the anterior-inferior quadrant of the tympanic membrane. The high percentage of area aerated in the SR indicates that this compartment may be more resistant to cholesteatoma invasion. Moreover, in addition to the anterior and posterior tympanic isthmus, the aeration route directly from the SR to epitympanum was demonstrated histopathologically to contribute to mastoid aeration. It would seem important, therefore, in surgical cases using the intact canal wall technique, to keep this route patent for mastoid aeration to prevent recurrence of cholesteatoma.

---

Minnesota Ear, Head, and Neck Clinic, 701 25th Avenue South, Suite 200, Minneapolis,  
Minnesota 55454-1443.

## MANAGEMENT OF LABYRINTHINE FISTULAE SECONDARY TO CHOLESTEATOMA

*Jacques A. Herzog, \*Peter G. Smith, \*G. Robert Kletzker, and \*Kenneth S. Maxwell*

### ABSTRACT

Improvements in diagnosis and management of chronic ear disease in general and cholesteatoma in particular have led to a decreased incidence of serious labyrinthine complications. Unfortunately, significant disease still does occur and, if unrecognized, may result in significant morbidity. Labyrinthine fistulae secondary to cholesteatoma cause potentially irreversible symptoms such as hearing loss and vertigo. This study reviews 17 patients who developed labyrinthine fistula secondary to cholesteatoma. Sixteen involved the horizontal semicircular canal and one involved the oval window. The cholesteatoma matrix was removed in all cases and the underlying fistula repaired primarily. Cochlear function was preserved in all patients. Sixteen of 17 patients have had no further difficulty with vertigo beyond the immediate postoperative period. The evaluation and contemporary management of this difficult problem are discussed.

---

Center for Hearing and Balance Disorders and \*Midwest Otologic Group, St. Louis, Missouri, U.S.A.

This work was presented at the 128th Annual Meeting of the American Otological Society, Palm Springs, California, U.S.A, April 30, 1995.

Reprint requests: Dr. J.A. Herzog, 11155 Dunn Rd., Suite 209 E., St. Louis, Missouri 63136, U.S.A.

## MECHANICAL VERSUS CO<sub>2</sub> LASER OCCLUSION OF THE POSTERIOR SEMICIRCULAR CANAL IN HUMANS

*Patrick J. Antonelli, \*\*†Larry B. Lundy, \*\*†Jack M. Kartush, †Don L. Burgio, and §Malcolm D. Graham*

### ABSTRACT

The purpose of this study was to compare the effectiveness of mechanical and laser-assisted posterior semicircular canal occlusion (PCO) for the treatment of retractable benign paroxysmal positional vertigo (BPPV). Twelve consecutive patients with intractable BPPV underwent PCO by three surgeons, six with mechanical PCO and six with CO<sub>2</sub> laser-assisted PCO. PCO eliminated positional vertigo in all patients treated with the laser and five of six patients treated without the laser. Dysequilibrium was present in all patients immediately postoperatively. This resolved in all patients treated with the CO<sub>2</sub> laser but in only two of six patients treated without the laser ( $p=10.03$ ). Patients were hospitalized for dysequilibrium for an average of 5.2 and 2.8 days for the mechanical and laser-assisted groups, respectively. Preoperative and postoperative hearing was not significantly different between the groups. No clinically significant postoperative hearing loss was encountered in either group. These results suggest that PCO is an effective treatment for intractable BPPV. The incidence of dysequilibrium that persists following PCO may be reduced by using the CO<sub>2</sub> laser to seal the membranous canal prior to occluding the bony canal.

---

Department of Otolaryngology, University of Florida, Gainesville, Florida; \*Michigan Ear Institute, Farmington Hills, †Providence Hospital, Southfield, and ‡Department of Otolaryngology, Wayne State University, Detroit, Michigan; and §Georgia Ear Institute, Savannah, Georgia, U.S.A.

This work was presented at the American Otological Society Meeting, April 29–30, 1995, Palm Desert, California, U.S.A.

Reprint requests: Dr. P.J. Antonelli, Department of Otolaryngology, University of Florida, Box 100264, Gainesville, Florida 32610, U.S.A.

## DIRECT COCHLEAR NERVE ACTION POTENTIALS AS AN AID TO HEARING PRESERVATION IN MIDDLE FOSSA ACOUSTIC NEUROMA RESECTION

*Joseph P. Roberson, Jr., \*Allen Senne, \*\*Derald Brackmann, †William E. Hitselberger,  
and ‡James Saunders*

### ABSTRACT

A new application of auditory evoked potentials using direct cochlear nerve action potentials (CNAPs) for monitoring middle fossa acoustic neuroma resection with attempted hearing preservation is described. Twenty patients have been studied to date. With this technique, a monitoring electrode is secured between the floor of the internal auditory canal and the dura adjacent to the cochlear nerve in an extradural location. Standard auditory evoked potential techniques with click stimuli and microelectrical recording allow observation of nearfield waveforms in seconds versus several minutes required for farfield potentials recorded from the scalp. Advantages of this technique over auditory brain stem response monitoring may include nearly real time measurement of potentials, improved surgeon learning curve and possibly higher rates of hearing preservation, and applicability to all patients undergoing hearing-preservation surgery independent of presence or absence of ABR tracing. Immediate changes in amplitude and latency of waveforms appear to compare with reversible and irreversible intraoperative auditory system damage, thereby guiding surgical maneuvers.

---

California Ear Institute at Stanford, Palo Alto; \*House Ear Clinic and †House Ear Institute and ‡private practice neurological surgery, Los Angeles, California; and §private practice otolaryngology, Oklahoma City, Oklahoma, U.S.A.

Reprint requests: Dr. D.E. Brackmann, House Ear Clinic, 2100 West Third Street, Los Angeles, California 90057, U.S.A.

## DISCUSSION PERIOD SIX

### Papers 25–29

**Dr. Robert Jahrsdoerfer** (Houston, Texas): We will now open the floor to discussion of the previous five papers. Questions or comments? Dr. Sheehy.

**Dr. James L. Sheehy** (Los Angeles, California): Dr. Herzog, it is fine to do a canal-wall-down procedure, but why would you remove the matrix and the fistula if you took the canal wall down? This would be a situation in which all my associates and I would leave the matrix there. If we were doing canal-wall-down, and we did not remove it, why would we go back and remove it at a second stage? Maybe there is something here I do not understand, but this does not fit in with what we would do. It is like in the old days, you see, in a modified radical mastoidectomy, you just left the matrix over the fistula, because if you take the matrix off, the skin is just going to grow back. So, why not just leave it? Then you do not have the risk involved in its removal. I am questioning here if he knows why it is that whoever did most of those surgeries decided that with a canal-wall-down procedure, an open cavity, the matrix should be removed?

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Second microphone.

**Dr. Julian Nedzelski** (Toronto, Canada): A question, perhaps a comment, for Dr. Roberson. I enjoyed your presentation Dr. Roberson. We have been using this technique for the better part of ten years. My comment would be that if you are going to put an electrode toward the lateral end of the internal auditory canal, you would be just as well advised to put it on the promontory, unless you are prepared to put the electrode on the cochlear nerve, medial to the tumor in the cerebellopontine angle. Secondly, I think the parameters you should be looking for to tell you what is happening are not latency or amplitude, but rather shift of threshold.

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Other questions or comments from the floor?

**Dr. F. Owen Black** (Portland, Oregon): I have a question for Dr. Antonelli. Postural discontrol in uncompensated BBPN subjects is fairly high, so it seems to me that one of the controls you would want to use is a stratified control. You can obviously

cannot do a cross-over, but stratification could control for selection bias. I wonder if you want to comment on that?

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Dr. Herzog, would you like to respond please?

**Dr. Jacques Herzog** (St. Louis, Missouri): Dr. Sheehy, I will answer you in two parts. To the second part of your question first (that was about leaving the larger ones behind and coming back at a second stage), with those patients we were discussing the idea of leaving an intact canal wall with the larger fistulas, and coming back at a later date. In terms of dealing with matrixes over a fistula less than 2 millimeters in size, my training always was to remove that for the concern of guarding against potential enzymatic destruction of the otic capsule and labyrinthine complications down the road. I do respect your work and you have great data; this is just the way we have done it, and we have shown here that we can remove all the disease and not cause any labyrinthine trauma.

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Dr. Roberson, would you like to respond?

**Dr. Joseph Roberson** (Los Angeles, California): Dr. Nedzelski, thank you for your comments. I heard yesterday during the panel your comments about the threshold, and I look forward to being able to try that. One of the advantages, I hope, of this technique is that the electrode is situated proximal to the tumor; that is the way I understand it, and maybe that is wrong. I think it would be better if we could get the electrode close to the brain stem in order to follow wave V, because that appears to be the area we want to know the electrical stimulation gets to. Practically, we have not been able to develop a way to hold the electrode in the same place to generate that, therefore, we have come up with this.

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Dr. Antonelli, would you care to respond?

**Dr. Patrick Antonelli** (Gainesville, Florida): If I understand the question correctly, is there selection bias in our patients, and I think that the answer is maybe. We were doing the mechanical occlusion technique first; we were doing it on people with the worst cases, and it was only when we had more fa-

miliarity with it were we doing people with lesser degrees of disease. So, that certainly is a possibility. However, some of the surgeons that were doing this technique were doing both, and it really is not clear to me that that is the answer. It is possible that that is responsible. There definitely was a tendency to have more of a disease symptom duration before, and it was not statistically significant, but there was certainly a longer period of time. The other thing I

would like to point out is that we had about three or four patients out of the six in the carbon dioxide laser assisted group that had abnormal ENGs suggesting more diffuse pathology than just the posterior canal. There were fewer, I think only one, who had an abnormal ENG in the mechanically occluded group which tended to speak against that.

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Any further discussion?



# IDENTIFICATION OF PHOTOACOUSTIC TRANSIENTS DURING PULSED LASER ABLATION OF THE HUMAN TEMPORAL BONE

*Brian J.F. Wong, Mark Dickinson, Joseph Neev, Karen J. Doyle, and Michael W. Berns*

## ABSTRACT

Laser ablation of hard tissues during neurotologic operations has been accomplished with continuous-wave (CW) lasers in the visible and mid-infrared spectrum. The mechanism of ablation at these wavelengths is secondary to photothermal induced tissue destruction. As a result, significant heating of neuroepithelia and inner ear fluids can occur. Pulsed ultraviolet (UV) lasers have been suggested as an alternative to the argon, KTP-532, and CO<sub>2</sub> lasers currently used in clinical practice. The pulse length of Excimer UV lasers are considerably shorter than the thermal relaxation time of bone tissue and hence very little thermal diffusion occurs. This makes pulsed lasers an attractive tool for non-thermal tissue ablation, in essence a "cold knife". However, the short pulse width of Excimer lasers (typically 10–150 ns) can create large thermoelastic waves resulting in photoacoustic transients in the ablation specimen. This study identifies the presence of these photoacoustic waves during the Excimer laser treatment of the human temporal bone. We investigated the basic characteristics of these waves in cadaveric human temporal bones. Mastoidectomies were performed in five cadaveric human temporal bones. Care was taken to preserve the bone covering the facial ridge. The temporal bones were mounted on glass plates and then secured on an x-y calibrated microstage that allowed the precise movement of the tissue specimen relative to the laser beam in a highly reproducible and consistent manner. A XeCl 308-nm Excimer laser (Lumonics HyperEX-400) was used to ablate hard tissue surrounding the oval window and facial ridge influences varying from 15 to 75 mJ per pulse. Spot size was estimated to be 0.5 mm<sup>2</sup>. A silicon photodiode detecting scattered laser light was used to provide the trigger signal, coincident with the onset of ablation. High frequency transducers were fabricated from polyvinylidene difluoride (PVDF) piezoelectric film (10 GHz bandwidth) and attached to the promontory, round window niche, and facial ridges (various locations). The PVDF films were secured with cyanoacrylate adhesives. Electrodes were attached to these transducers. The signals were amplified using a low noise pre-amplifier (SRS 650) and

recorded on a digitizing oscilloscope (Textronix DSA 601). Signals were transferred to a Macintosh lab system via a GPIB interface. Signals were recorded at the promontory, round window niche, and facial ridges. Photoacoustic waves were clearly identified along with the presence of pyroelectric transducer signals. Photoacoustic waves were measured exceeding  $0.30 \text{ N/M}^2$  in magnitude and at frequencies exceeding 1 MHz. Notably, large acoustic waves were measured on the promontory and on both sides of the facial ridge. This is the first report of a photoacoustic wave in laser surgery of the ear. The implications and clinical relevance of these findings is discussed, and compared to findings obtained from a model system.

---

Beckman Laser Institute, 1002 Health Sciences Road East, Irvine, California 92715.  
This work was supported by Grants ONR N0014-91-C-0134, DOE DE-FG03-91ER61227, and NIH 5P41RR01192. Dr. Wong was supported by the Research Fund of the American Otological Society.

## POLYMERASE CHAIN REACTION AMPLIFICATION OF A MEASLES VIRUS SEQUENCE FROM HUMAN TEMPORAL BONE SECTIONS WITH ACTIVE OTOSCLEROSIS

*\*Michael J. McKenna, †Arthur Kristiansen, and †Jonathan Haines*

### **ABSTRACT**

Investigation of a possible viral etiology for otosclerosis was initiated because of the clinical and histopathologic similarities between otosclerosis and Paget's disease of bone and the mounting evidence of a viral etiology in Paget's disease. Thus far, ultrastructural and immunohistochemical studies have revealed measles-like structures and antigens in active otosclerotic lesions. A method for isolation and identification of both DNA and RNA sequences in archival human temporal bone specimens using the polymerase base chain reaction technique has been developed. Using this technique, a 115 base pair sequence of the measles nucleocapsid gene has been identified in 8 of 11 different temporal bone specimens with histologic evidence of otosclerosis. Zero of 9 control specimens without histologic evidence of otosclerosis were positive. The association between the presence of the measles nucleocapsid gene sequence and histologic otosclerosis is significant ( $P < 0.01$ ). This study provides further evidence for a possible measles virus etiology in otosclerosis.

---

\*Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston, Massachusetts 02114. †Massachusetts General Hospital, Boston, Massachusetts 02114.

## THE ROLE OF THE NEUROTROPHINS IN MATURATION AND MAINTENANCE OF POSTNATAL AUDITORY INNERVATION

*\*Hinrich Staecker, \*Vera Galinovic-Schwartz, \*Wei Liu, \*Philippe Lefebvre,  
\*‡Richard Kopke, Brigitte Malgrange, \*Gustave Moonen, and \*†Thomas R. Van De Water*

### ABSTRACT

Auditory hair cells produce trophic factors that directly affect maturation and survival of auditory neurons. These factors include two members of the neurotrophin family, brain-derived neurotrophic factor (BDNF) and neurotrophin-3 (NT-3). Loss of hair cells, as a result of either noise trauma or ototoxic damage, results in the degeneration of auditory neurons. An in vitro model of early postnatal rat organ of Corti/spiral ganglion explants was used to study the effects of deprivation and supplementation of nerve growth factor (NGF), BDNF, and NT-3 on neuronal survival. Immunolocalization of receptors for these neurotrophins correlated with their effectiveness as promoters of neuronal survival. BDNF affected early neuronal survival, whereas NT-3 was the most important survival factor for maturing auditory neurons. NGF was shown to maintain axonal morphology. Our results support the hypothesis that changes in the expression of these neurotrophins and their specific receptors in the maturing cochlea may control the postnatal processes of neuronal apoptosis and maturation of the innervation of both inner and outer hair cells. The results suggest that these growth factors have potential for preventing neuronal degeneration as well as enhancing the repair of damaged neuronal processes in the traumatized auditory system.

---

Departments of \*Otolaryngology and †Neuroscience, Albert Einstein College of Medicine, Bronx, New York, and ‡U.S. Army Medical Department Center and School, Fort Sam Houston, Texas, U.S.A.; and Department of Human Physiology and Pathophysiology, University of Liege, Liege, Belgium.

This article was presented at the 128th American Otologic Society Meeting in Palm Desert, California, U.S.A., April 29–30, 1995.

Reprint requests: Dr. T.R. Van De Water, Department of Otolaryngology, Albert Einstein College of Medicine, 1410 Pelham Parkway South, Kennedy Rm. 302, Bronx, New York 10461, U.S.A.

## EXTERNAL AND MIDDLE EAR PATHOLOGY IN TGF- $\alpha$ -DEFICIENT ANIMALS

*Charles G. Wright, Karen S. Robinson, and William L. Meyerhoff*

### ABSTRACT

Transforming growth factor- $\alpha$  (TGF- $\alpha$ ) is a growth-regulatory peptide found in a wide range of embryonic and adult tissues TGF- $\alpha$  is produced by keratinocytes and has been reported to be overexpressed in several epidermal diseases, including middle ear cholesteatoma. This report describes ear pathology in the waved-1 mutant mouse, which is severely deficient in TGF- $\alpha$ . Morphologic changes of the external and middle ear were studied histologically in waved-1 mutants 2 weeks to 6.5 months of age. Abnormalities found in the mutants included epidermal hyperplasia of the external ear canal (EAC) and tympanic membrane (TM) and enlargement of specialized sebaceous glands adjacent to the cartilaginous EAC. Sebum and desquamated keratin progressively accumulated within the EAC, displacing the TM into the middle ear. These changes appear similar to those occurring in Mongolian gerbils, which are known to develop cholesteatoma. The alterations found in waved-1 mutants are discussed in relation to the possible involvement of TGF- $\alpha$  in cholesteatoma pathogenesis.

---

Department of Otolaryngology, University of Texas Southwestern Medical Center, Dallas, Texas, U.S.A.

Presented at the American Otological Society Meeting, Palm Desert, California, April 29-30, 1995.

Reprint requests: Dr. C.G. Wright, Department of Otolaryngology, University of Texas Southwestern Medical Center, 5323 Harry Hines Blvd, Dallas, Texas 75235-9035, U.S.A.

## IMMUNOHISTOCHEMICAL FINDINGS IN THE COCHLEA OF AIDS CASES

*Jessica W. Lim, J. Thomas Roland, Jr., Jin S. Lim, James Lee, Bernard Ong, and Dean E. Hillman*

### ABSTRACT

Neurotologic manifestations of human immunodeficiency virus (HIV) infection are documented but poorly understood. Recent studies described degenerative ultrastructural changes in cochlear and vestibular neuroepithelia from humans infected with HIV. Additionally, HIV-like particles in various stages of viral maturation were observed in these tissues. In this study, we analyzed cochlear neuroepithelia of post-mortem HIV cases using immunohistochemistry. Each cochlea was perilymphatically perfused in situ with a mixed solution of glutaraldehyde and paraformaldehyde within 8 hours of death. Temporal bones were removed at autopsy. The cochleas were microdissected, and samples of cochlear neuroepithelia were removed. Mouse monoclonal antibodies to the p 17 and p 24 HIV antigens were localized using nano-sized colloidal gold with silver enhancement. After immunostaining, the tissue samples were embedded in Durcupan and sectioned at 1 micron for light microscopy. We observed intracytoplasmic staining of cells within the stria vascularis, particularly in the vascular epithelium, and adjacent connective tissue, as well as in the basilar membrane. This localization is consistent with our ultrastructural findings previously published and support the hypothesis of a direct invasion of the cochlea by HIV, which produces cochleotoxic effects. Further immuno-histochemical studies are being performed on vestibular tissues, with planned immuno-electron microscopy of cochlear and vestibular neuroepithelia.

---

New York University Medical Center, 550 First Avenue, New York, New York 10016.

# EFFECT OF LEUKOTRIENE INHIBITOR ON OTOACOUSTIC EMISSIONS IN SALICYLATE OTOTOXICITY

*\*Johnny Arruda, \*Timothy T. K. Jung, and \*†David G. McGann*

## ABSTRACT

Our previous work showed that salicylate ototoxicity is associated with decreased levels of prostaglandins (PGs) and increased levels of leukotrienes (LTs) in the perilymph. Pretreatment with LT inhibitor was found to prevent salicylate ototoxicity. Other studies demonstrated that salicylate ototoxicity is associated with decreased cochlear blood flow, reversible changes in cochlear outer hair cells, and decreased otoacoustic emissions. The purpose of our study was to determine the effect of LT blocker (Sch 37224) on transient-evoked otoacoustic emissions (TEOAEs) in salicylate or LT ototoxicity. Chinchillas were divided into five groups. Transient-evoked otoacoustic emissions were measured after salicylate application on the round window membrane (RWM), with (Group 1) and without (Group 2) LT blockade; after LTC<sub>4</sub> (a type of leukotriene) application on the RWM, with (Group 3) and without (Group 4) LT blockade; and in the control group after saline application on the RWM. The overall response differences from the baseline measurements over time in each case were compared with each other. Both salicylate and LTC<sub>4</sub> application on the RWM were followed by significant decreases in TEOAEs, and the decrease was prevented by pretreatment with LT blocker. There was no significant change in TEOAEs in the control group. Salicylate ototoxicity appears to be mediated by the elevated levels of leukotrienes as a consequence of cyclooxygenase inhibition. This study also provides further evidence that the site of action in salicylate ototoxicity is the outer hair cell.

---

\*Division of Otolaryngology–Head and Neck Surgery and the Jerry L. Pettis Memorial VA Hospital, and †Audiology Services; Loma Linda University Medical Center, Loma Linda, California, U.S.A.

Presented at the Annual Meeting of the American Otological Society, Palm Desert, California, April 29–30, 1995.

Reprint requests: Dr. T. Jung, 11790 Pecan Way, Loma Linda, California 92354, U.S.A.

## DISCUSSION PERIOD SEVEN

### Papers 30–35

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Thank you. The previous papers are now open for discussion.

**Dr. Richard Chole** (Davis, California): I enjoyed Bill's paper on the TGF-alpha-deficient mouse. I have a couple of observations and questions. We did a number of studies ligating the external canals of a number of animals including mice, rats, hamsters, guinea pigs, cats, and so forth, and never got any cholesteatoma/retraction, except in the gerbil, which of course develops cholesteatoma. This animal seems to respond very much like the gerbil with a couple of exceptions. There is a lot more inflammation and the middle ear changes appear to be a lot greater than we see in the gerbil, at least the uninfected gerbil. So, do you have any idea about why there may be those differences? The other question I have is that TGF alpha is thought to be active, not on the resorption side of bone, but on the deposition side of bone. There is not a lot of evidence for that, but did you see any differences in the bone in these animals, and do these animals develop and grow normally?

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Any other questions? Vincente.

**Dr. Vincente Honrubia** (Los Angeles, California): Yes. Regarding the mechanism of the effect of the leukotrienes in preventing damage to otoacoustic emissions—what is the cellular mechanism of this process? And that implies that the effect of acetylsalicylic acid is also a hair cell mechanism?

**Dr. Robert Jahrsdoerfer** (Houston Texas): Dr. Meyerhoff, would you like to respond please?

**Dr. William Meyerhoff** (Dallas, Texas): Rick, I really do not know the answer to your question. First of all, the animals have developed normally; they have no abnormalities of bone structure or anything of that nature. They have meibomian gland atrophy just like the zymbals gland which causes some ophthalmic problems but otherwise they appear to be quite pleasant mice. I do not think that it is the occlusion of the external auditory canal that is causing this cholesteatoma-like formation, but 100% of these animals are getting it. I think we have to study this a little bit further.

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Dr. Arruda, would you like to respond please?

**Dr. John Arruda** (Loma Linda, California): Regarding the mechanism of the leukotriene, it is not completely understood, but there are two possible explanations. One has to do with a decrease in cochlear blood flow, which has a vasoconstrictive effect. At the same time we also have a decrease in prostaglandin levels, which tend to be vasodilating. The other mechanism probably has to do with membrane conductance of the outer hair cell (similar to what you have with salicylates, having to do with potassium diffusion and so forth). That is about the best I can explain that.

**Dr. Robert Jahrsdoerfer** (Houston, Texas): Any final comments or questions?



## INTRODUCTION OF NEW PRESIDENT: DERALD E. BRACKMANN, M.D.

*Robert A. Jahrsdoerfer, M.D., F.A.C.S.*

As President, I just want to say three things. Firstly, we tend to take it for granted when these meetings run smoothly, but I can tell you that it takes a lot of preparation, dedication, and hard work. I want to give credit to our Secretary-Treasurer, Dr. Gregory Matz, who has performed admirably over the past year. I can tell you that he had made my year as President an easy one. Greg, thank you.

Secondly, I want to thank the audio-visual people for two days of flawless presentation. I thought they did a fantastic job.

Lastly, I have the honor of introducing the new President, someone who really needs no introduction, Dr. Derald Brackmann. In his hands, I am sure that this Society will stay the course.

## REMARKS OF NEW PRESIDENT

*Derald E. Brackmann, M.D.*

Well, Bob, let's see; if you had three things to say, I can say three things, too, I guess. The first is that it is a great honor to serve this Society, and I thank you all for that privilege. Secondly, I invite all of your submissions of abstracts for papers. You know, there is a Program Committee now, and the abstracts are reviewed without authors' names. We hope to make this a very fair selection of papers, and we invite all of your submissions. My third thing, Bob, is to thank you. It has been a great privilege to be on the Council with you, and the Society does have a small token of our appreciation [reading from plaque]: "Presented to Dr. Robert Jahrsdoerfer, President, in appreciation and recognition of his service to this Society." Bob, thank you very much.

**Dr. Jahrsdoerfer:** Thank you. We are adjourned.

# EXECUTIVE SESSIONS

## BUSINESS MEETING

MINUTES—APRIL 29–30, 1995

Dr. Jahrsdoerfer called the meeting to order at 7:00 AM, April 29, 1995. The minutes of the previous American Otological Society Annual Meeting, held in Palm Beach, Florida, May 7–8, 1994, were approved.

The following new members were presented to the Society, along with their respective proposers:

### *Active Members*

Ronald G. Amedee, M.D. Proposed by Harold G. Tabb, M.D.; seconded by Newton J. Coker, M.D.  
Charles W. Beatty, M.D. Proposed by Stephen G. Harner, M.D.; seconded by John W. House, M.D.  
C. Phillip Daspit, M.D. Proposed by James L. Sheehy, M.D.; seconded by John W. House, M.D.  
Thomas L. Eby, M.D. Proposed by Harold C. Pillsbury, M.D.; seconded by Dennis Pappas, M.D.  
Joel A. Goebel, M.D. Proposed by J. Gail Neely, M.D.; seconded by John M. Fredrickson, M.D.  
Paul R. Lambert, M.D. Proposed by Cary N. Moon, M.D.; seconded by Malcolm D. Graham, M.D.  
John P. Leonetti, M.D. Proposed by Peter G. Smith, M.D.; seconded by Robert A. Goldenberg, M.D.  
Edwin M. Monsell, M.D., Ph.D. Proposed by James L. Parkin, M.D.; seconded by John W. House, M.D.  
Ralph A. Nelson, M.D. Proposed by Aram Glorig, M.D.; seconded by Howard P. House, M.D.  
John K. Niparko, M.D. Proposed by Malcolm D. Graham, M.D.; seconded by Noel L. Cohen, M.D.  
Dennis S. Poe, M.D. Proposed by Michael E. Glasscock III, M.D.; seconded by Robert K. Jackler, M.D.  
Alexander J. Schleuning, M.D. Proposed by Harold C. Pillsbury, M.D.; seconded by Paul H. Ward, M.D.  
Clough Shelton, M.D. Proposed by James L. Parkin, M.D.; seconded by James L. Sheehy, M.D.

### *Associate Members*

Karen I. Berliner, Ph.D. Proposed by Robert A. Goldenberg, M.D.; seconded by John W. House, M.D.

### *Corresponding Members*

J. Barton Booth, M.B. Proposed by Howard P. House, M.D.; seconded by Aram Glorig, M.D.  
Dan Bagger-Sjöbäck, M.D., Ph.D. Proposed by Gordon B. Hughes, M.D.; seconded by Jack Pulec, M.D. and Michael M. Paparella, M.D.  
Jean-Bernard Causse, M.D. Proposed by Gordon B. Hughes, M.D.; seconded by Herbert Silverstein, M.D.

A Nominating Committee was elected to prepare the slate of officers for the 1995–1996 year. The Committee consisted of: Dr. John House, Chairman; Dr. Roger Boles; Dr. Jack Kartush; Dr. Gail Neely; Dr. Harold Tabb.

## REPORT OF THE SECRETARY-TREASURER

### REPORT OF THE SECRETARY

Report of present membership

Active Members	133
Senior Members	71
Emeritus Members	4
Honorary Members	9
Associate Members	40
Corresponding Members	3
<b>Total Members</b>	<b>256</b>

Deaths since the last annual meeting:  
John Bordley, M.D., joined the Society in 1955, elected Senior Member in 1985, died July 12, 1993; David A.

Dolowitz, M.D., joined the Society 1959, elected Senior Member 1979, died December 7, 1994; Victor Goodhill, M.D., joined the Society in 1950, elected Senior Member in 1985, died December 23, 1994; Ralph J. McQuiston, M.D., joined the Society 1952, elected Senior Member in 1980, died March 22, 1995; Kinsey M. Simonton, M.D., joined the Society in 1952, elected Senior Member in 1976, died December 14, 1994; Walter P. Work, M.D., joined the Society in 1953, elected Senior Member in 1975, died November 4, 1994; J. William Wright, Jr., M.D., joined the Society in 1978, elected Senior Member in 1990, died February 14, 1994.

TRANSACTIONS 1995 / AMERICAN OTOLOGICAL SOCIETY

Candidates for Senior Membership of the Society were announced. Bylaws require membership of 20 years or attainment of the age of 70 years for Senior Membership. A voice vote for Senior Membership on each of the following Candidates was taken and approved: James A. Crabtree, M.D., joined the Society in 1972; William F. House, M.D., joined the Society in 1964; Ward B. Litton, M.D., joined the Society in 1969; Joseph Sataloff, M.D., joined the Society in 1960; and F. Blair Simmons, M.D., joined the Society in 1973.

New Members were introduced to the Society on Saturday, April 29, 1995. These included 13 Active Members, 1 Associate Member, and 3 Corresponding Members.

**REPORT OF THE TREASURER**

July 1, 1994–March 31, 1995	
Beginning Balance	.....\$69,140.52
Income:	
COSM Payments	.....\$6186.00
Membership Dues	.....\$51000.00
Initiation Fees	.....\$600.00
Reimbursements	
Member	.....\$76.00
I.R.S	.....\$1609.24
Research Fund Transfer	.....\$221.00
(Insurance & Bond)	
Research Fund Transfer	.....\$14432.75
(Taxes, Audit Fees)	
<i>Transactions and History</i>	.....\$605.00
Interest	.....\$1941.40
Total Income	.....\$76,671.39
Balance	.....\$145,811.91

Expenses:	
ACCME	.....\$500.00
Accounting Fees	.....\$6565.00
Dekker Publishers	.....\$24129.40
1993 <i>Transactions</i>	\$16004.40
1995 <i>AJO</i> Subscriptions	\$ 8125.00
Donations	.....\$1200.00
DRF	\$1000.00
Ruth Parks Memorial	\$ 200.00
Fall Council Meeting	.....\$201.51
Insurance	.....\$3847.00
Travel	\$267.00
Dishonesty Bond	\$221.00
Officer Liability	\$3359.00
IRS Penalty	.....\$1831.00
(most reimbursed)	
Mid-winter Council Meeting	.....\$12016.47
Miscellaneous	.....\$3037.29
Deposit Box	\$10.00
Bank Charges	\$28.79
New Lapel Pins (100)	\$2185.00
Travel, President	\$770.00
Travel, Sec'y-Treas	\$43.50
Postage, Printing, Supplies	.....\$2343.13
Society Operational Expenses	.....\$6238.22
Secretary Stipends (2)	\$4600.00
Editor-Librarian	\$1638.22
Reimbursements Due from	
1993 Audit	.....\$1303.50
LUMC	\$1003.50
Univ. Michigan	\$300.00
COSM Meeting 1995	.....\$555.00
Taxes 1993 & 1994	.....\$500.00
Total Expenses	.....\$64267.52
Balance	.....\$81,544.39

## EXECUTIVE SESSIONS

Dr. Joseph Farmer presented the report of the Editor-Librarian, indicating that Volume 82, the 1994 edition of the *Transactions*, was being prepared for the publisher. The cost of printing, postage, and handling will be \$67, and any Senior, Associate, Corresponding, or Emeritus Member who wishes to purchase the *Transactions* should send payment to the office of the Editor-Librarian.

A committee has been appointed to study whether republication in the *Transactions* of papers already published in the *American Journal of Otology* (AJO) represents unnecessary repetition. Eliminating the repetition could result in a savings of thousands of dollars.

It has been tentatively agreed that Lippincott-Raven Publishers, the same company that will be publishing the AJO beginning in 1996 with Volume 17, will publish the *Transactions* at a lower unit cost.

The office of the Editor-Librarian continues to work closely with Mr. Phillip Seitz, the Historian at the American Academy of Otolaryngology-Head & Neck Surgery in Alexandria, Virginia, to insure that a complete set and back up copies of the *Transactions* are available. Microfilm duplication of decaying volumes has been arranged. Under consideration by the AOS Council is a request to give \$2000 to defray a portion of the shelving and equipment costs involved in storing and displaying this collection.

Dr. Robert A. Jahrsdoerfer called the Society members' attention to the proposed Bylaws change:

9.1.0.d. "Any Candidate vetoed by eight percent of the eligible voting members shall be eliminated from the list of applicants for membership." The Society voted to approve the modification of the Bylaws.

Dr. Robert Keim, President of the AJO Board of Directors, presented an update on the AJO and expressed the *Journal's* great appreciation for the years of expertise and excellence under the Editorship of Dr. C. Gary Jackson. He introduced the new Editor of the AJO, Dr. Robert K. Jackler.

Dr. Robert Jahrsdoerfer thanked the members of the Program Advisory Committee: Drs. Newton J. Coker, John R.E. Dickins, Robert A. Dobie, Maureen T. Hannley, Stephen G. Harner, Robert K. Jackler, Charles M. Luetje, Douglas E. Mattox, and John T. McElveen.

The Business Meeting was adjourned and the first Scientific Session started at 7:30 AM with remarks by the President, Robert A. Jahrsdoerfer, M.D. Remarks by the

Guest of Honor were presented by Richard R. Gacek, M.D. President Jahrsdoerfer presented the Presidential Citation to Eiji Yanagisawa, M.D. The Scientific Session was adjourned at 12:10 PM, with all members staying for the group photograph.

The President's Reception and dinner/dance began at 6:30 PM and was attended by 175 persons. President Robert A. Jahrsdoerfer, M.D. introduced the new members and their spouses. Dr. Mansfield F.W. Smith presented the Award of Merit to D. Thane R. Cody, M.D., Ph.D.

The Business Meeting was reconvened at 12:30 PM, on Sunday, April 30, 1995. Reports were received as follows:

The report of the Board of Trustees of the Research Fund was delivered by Richard T. Miyamoto, M.D., the Secretary-Treasurer. Dr. Robert I. Kohut presented the report of the American Board of Otolaryngology for Dr. Warren Y. Adkins, who was unable to attend because of illness in the family.

Dr. Mansfield Smith reported that the Award of Merit Committee had chosen Dr. Thane Cody as the Award of Merit Recipient for 1995.

Dr. Gregory Matz reported on the American College of Surgeons. Dr. Harold Pillsbury presented the report of the American Academy of Otolaryngology-Head & Neck Surgery.

Dr. Michael Maves updated the membership on COSM.

Dr. Robert Jackler, Chairman of the Audit Committee, presented the report of the Audit Committee.

Dr. John House, Chairman of the Nominating Committee, presented the slate of officers for the 1995-1996 year. They are: Dr. Derald Brackmann, President; Dr. Joseph C. Farmer, Jr., President-Elect; Dr. Gregory J. Matz, Secretary-Treasurer; Dr. A. Julianna Gulya, Editor-Librarian; and Charles M. Luetje, new Council member. Drs. C. Gary Jackson, Robert I. Kohut, and Robert A. Jahrsdoerfer will continue their terms on the Council.

The Business Meeting was adjourned and the second session of the Scientific Program began at 1:00 PM. At the close of the Scientific Session, Dr. Jahrsdoerfer presented the gavel to Dr. Derald Brackmann, the incoming President. Dr. Jahrsdoerfer in turn was presented a commendation certificate and was thanked by the membership for his service as President. The meeting was adjourned at 5:30 PM.

Respectfully submitted,  
Gregory J. Matz, M.D.

## REPORT OF THE EDITOR-LIBRARIAN

All the materials for Volume 82, the 1994 edition of the *Transactions*, are expected to be in the hands of the publisher by June 1, 1995, and available for distribution early this fall. The Bylaws of the Society require that Senior, Emeritus, Corresponding and Associate members pay the cost of the *Transactions*, which we expect will remain at \$67 per issue, including postage and handling. Any Senior, Emeritus, Associate, or Corresponding member who wishes to purchase the *Transactions* should send payment to our office. It should be noted that these issues contain

edited transcriptions of the panel discussions presented at the meeting each year.

The President of the Society, Dr. Robert Jahrsdoerfer, at the direction of the Council, has formed a committee to study whether the publishing of papers in the *Transactions* that have already been published in the *American Journal of Otology* represents unnecessary repetition. In 1993 we used only the abstracts and did not publish the full texts of those papers which were accepted for publication in the *American Journal of Otology*, and did not publish any of the

panel session or discussion periods, we would have saved the cost of approximately 160 pages, which comprise about 64% of the total pages of this volume. This may result in substantial savings of thousands of dollars. The committee appointed by President Jahrsdoerfer will study the pros and cons of this issue and present a cost/benefit analysis. If members have comments they wish to make to the committee, it is recommended that they send their comments to my office, or to any member of the Council, and we will see that the comments are forwarded to the committee chairperson.

The Council has tentatively approved a bid from Lippincott-Raven Publishing Company to publish the 1995 *Transactions* at a lower unit cost. It should be noted that Lippincott-Raven will be publishing the *American Journal of Otology* beginning with Volume 17 in 1996. This volume will contain most of the papers presented at this meeting. The *American Journal of Otology, Inc.*, Directors and the AOS Council look forward to this relationship with Lippincott-Raven.

The Editor-Librarian's office continues to work closely with Mr. Phillip Seitz, Historian at the American Academy of Otolaryngology-Head and Neck Surgery Museum in Alexandria, Virginia, to ensure that a complete set and backup copies of the *Transactions* are available. We continue to receive, on occasion, successful responses to our requests for locating missing volumes. We still are seeking

any copies of Volume 2, covering the years 1875-1879, Volume 15, covering 1919, and Volume 16, for 1924.

With Mr. Seitz's assistance, we have arranged for microfilm duplication of decaying volumes by the History of Medicine Division of the National Library of Medicine. We are working with this institution to have the material beyond 1940 microfilmed. If any member is interested in this project, we invite your participation and any assistance that you may wish to give. This is a worthwhile and necessary effort to preserve these valuable materials. The Council is considering a request to display and store this collection of the *Transactions*, which documents the history of the second oldest subspecialty (after the AAOO) in North America.

Finally, I wish to urge the members to proceed promptly to Mrs. Judy Matz's desk at the close of this meeting and obtain a number to use for the annual photograph. Please take a number and make sure that your name and number are recorded before you leave Judy's desk. As before, we will take the photograph with each member holding the card so that it can be seen by the camera, and so that it is not obscured by the individual standing in front. We will then collect the cards and take another photograph, hopefully with everybody remaining in the same location.

Respectfully submitted,  
Joseph C. Farmer, Jr., M.D.

## REPORT OF THE BOARD OF TRUSTEES OF THE RESEARCH FUND

The Trustees of the American Otolological Society Research Fund, chaired by Dr. Robert Dobie, met in St. Louis, Missouri on March 18th, 1995. At that time, according to the accounting from Mr. Art Schweithelm, account advisor, our balance on March 8, 1995, was \$5.6 million. Despite the very difficult investment climate the Fund earned over 4% during this past year, which is quite good compared to what most mutual funds have done. The allocation of the assets was 63% to common stock and 37% to fixed-income securities.

The past year was marked by a very successful effort to track more high quality grant applications. The Trustees reviewed 27 grant applications, and funded 7 research

grants—five traditional research grants and two fellowships. The Trustees also voted to contribute \$10,000 to the Friends of the NIDCD to support their efforts to increase the NIDCD budget; through consultation with Drs. Robert Ruben and George Gates it was determined that the Friends possess an appropriate tax mechanism to receive such funds from the AOS.

At the meeting Dr. George Gates was installed as the new Chairman for the coming year and Dr. Jeffrey Harris was elected as the new Trustee.

Respectfully submitted,  
Richard T. Miyamoto, M.D.

## REPORT OF THE REPRESENTATIVE TO THE BOARD OF GOVERNORS OF THE AMERICAN COLLEGE OF SURGEONS

The American College of Surgeons met in the second week of October, 1995, in Chicago. Dr. Murray, the Executive Director, outlined five major issues before Congress in health care reform:

1. Protecting the patient's right to choose his/her physician or surgeon;
2. Increasing patient access to specialists and safeguarding quality of care;
3. Protecting the surgeon's or physician's autonomy in medical decision-making;

4. Maintaining a reimbursement system which pays surgeons for services provided to patients; and,
5. Controlling health-care costs and developing a simple and more workable administrative system.

No dues increase was recommended, and it was noted that none had been presented for the past three to four years.

There has been a net increase in membership in Otolaryngology within the College. With 3769 members, our specialty constitutes nearly 8% of the membership. There

## EXECUTIVE SESSIONS

were 167 new members inducted this year. Otolaryngology was the second largest group after general surgery, which has been consistent over the past few years. The College is especially concerned about the National Data Bank for malpractice suits, and a special level of concern is related to the surgical residents whose names appear in a data bank in cases where they have no involvement in the lawsuit. Another data bank is needed to develop insurance data, especially for capitated fees. The College continues to urge HCFA to refute its methodology for determining adjustment to medicare RBRVS.

The College has prepared an updated statement on "The Surgeon and HIV and Hepatitis B Infection," a copy of which can be obtained from the Chicago office.

Dr. Gerald B. Healy was appointed Chairman of the Advisory Council on Otolaryngology, succeeding Dr. Charles W. Cummings. Drs. Nicholas J. Cassisi, Frank E. Lucente, and Ernest A. Weymuller, Jr., were nominated for the Residency Review Committee.

Respectfully submitted,  
*Gregory J. Matz, M.D.*

## REPORT ON COSM

The American Academy of Otolaryngology-Head and Neck Surgery has been active on a variety of fronts during the past year. We have been engaged in the five-year review of the medicare RBRVS and we are very active both on the federal and state levels with legislative advocacy issues. One issue which demands immediate attention is before the Congress, actually the Senate, right now, and involves significant medical malpractice reform. There is a move to eliminate the \$250,000 cap on pain and suffering which was included in the House version of this legislation (HR10) that is now being considered in the Senate. The American Trial Lawyer Association has pledged \$20 million to this effort and the AMA has contributed about one-half million dollars to try to have this cap remain. It is a critical time and I ask each one of you to call both your Senators this Monday. Two phone calls will be necessary, but it will not take you very much time. It is critically important. Mr. John Williams, who is our Federal Affairs Manager, has a complete list of the Senate phone numbers for your convenience. Call and make your opinion known. We have been battling for tort reform for many, many years, and finally we have some gleam of hope for significant legislation being passed on a national level. It is imperative for us to act.

I also wanted to report on some other activities at the Academy. We are undertaking a survey of continuing educational needs for all of our members. By the end of the year it should be on-line, either through an on-line service or through the Internet, and we have just recently secured the services of a marketing firm in Northwest Washing-

ton, D.C., to embark upon the "marketing otolaryngology" initiative. We have been very active in practice management issues and will have a number of publications on capitation and referral guidelines ready for the Academy meeting this fall.

We continue to participate actively in research. We are submitting a bid for a clinical trial initiative.

We have been active in trying to obtain more support from our corporate partners, as indicated by the very nice turnout of corporate people here at this meeting; the exhibitors in large part support the success of COSM, so I ask each one of you to make a point of going to the exhibit hall.

Regarding future meetings, this fall, September 17-20, we will be in New Orleans, Louisiana. COSM will go to the Hyatt Grand Cypress, in Orlando, Florida, next year (May 4-9) and then next fall (September 29-October 3) is the Centennial Meeting of the American Academy of Otolaryngology-Head and Neck Surgery, and Foundation, which will be in Washington, D.C. We are engaged in devising a number of special events to mark the occasion of our 100th anniversary. In 1997, COSM will be in Scottsdale, Arizona, and the Academy's Foundation Meeting will be in San Francisco, California.

Respectfully submitted,  
*Michael D. Maves, M.D.*  
Executive Vice-President,  
American Academy of Otolaryngology-  
Head & Neck Surgery

## REPORT OF THE AMERICAN JOURNAL OF OTOTOLOGY

It is my pleasure, as President of the *American Journal of Otology*, to speak on the behalf of the Board of Directors in announcing the new Editor-in-Chief. It was not a small task to select the new editor; it took us nearly a year, for we had many fine candidates to evaluate. After our deliberations, we have selected Dr. Robert K. Jackler. I ask

you at this time to join me in supporting his work. I think it is also appropriate at this time to recognize the fine, dedicated work of Dr. C. Gary Jackson, who has worked for twelve years to make the *Journal* the one which we now enjoy as our representative voice in this specialty. Please join me in applauding his work.

## REPORT OF THE AUDIT COMMITTEE

The Audit Committee, consisting of Dr. Richard Wiet, Dr. Eugene Derlacki and myself, has reviewed the financial reports of the Society for the period from July 1, 1994 to March 31, 1995. Our findings include:

1. Financial totals appear correct according to the data provided:  
Beginning bank balance = \$69,140  
Income during the audit period = \$76,671  
Expenses during the audit period = \$81,544

2. Expenses: The cash disbursement register shows consecutive checks except where voided. We identified no unusual expenditures.
3. All monies dispersed to Council members were appropriately justified as expenses incurred through travel to official Society functions.
4. The Society maintains an admirable level of financial reserves.

Respectfully submitted,  
*Robert K. Jackler, M.D.*

## REPORT OF THE AMERICAN BOARD OF OTOLARYNGOLOGY

## EXAM STATISTICS

Four successful examination cycles have been completed using the new format. The candidates must first pass a written, qualifying exam and then pass an oral exam in order to become certified; scores are not combined for the final score. The written examination was taken by 353 candidates in September 1994. Of those candidates, 16% failed and nearly 84% became candidates for the oral examination, which was conducted by 82 Guest and Associate examiners and the 25 ABO Directors for 336 candidates on April 8-9, 1995, at the Palmer House.

## ASSOCIATE EXAMINERS

The position of Associate Examiner was initiated last year. To be elected an Associate Examiner, an individual must have served as an ABO examiner at least twice, and must be prominent in the specialty, especially in the areas of patient care and medical education. The individual must demonstrate an interest and ability in the creation of educational and test materials. The ABO is committed to electing and training new examiners while maintaining consistency in administering the examination. The Associate Examiners are a corps of experienced examiners to fill this need along with the Directors. Associate Examiners are elected for a three-year term and are eligible for re-election for one additional term. The Associate Examiners are listed as follows:

**Elected in 1993:** Hugh H. Biller, M.D.; Nicholas J. Cassisi, M.D.; A. Julianna Gulya, M.D.; Lauren D. Holinger, M.D.; Jonas T. Johnson, M.D.; Frank E. Lucente, M.D.; Dale Rice, M.D.; James Y. Suen, M.D.

**Elected in 1994:** Robert A. Dobie, M.D.; Paul J. Donald,

M.D.; Ellen M. Friedman, M.D.; Jack L. Gluckman, M.D.; G. Richard Holt, M.D.; Herman A. Jenkins, M.D.; Douglas E. Mattox, M.D.; Michael D. Maves, M.D.; Richard T. Miyamoto, M.D.; William J. Richtsmeier, M.D.; Clarence T. Sasaki, M.D.; Nancy L. Snyderman, M.D.; Ernest A. Weymuller, M.D.

**Elected in 1995:** Kenneth M. Grundfast, M.D.; J. David Osguthorpe, M.D. Stanley M. Shapshay, M.D.; James N. Thompson, M.D.; W. Frederick McGuirt, M.D.; Gary L. Schecter, M.D. Bruce J. Gantz, M.D.; Jeffrey P. Harris, M.D.; Paul R. Lambert, M.D.; James L. Parkin, M.D.; Karen H. Calhoun, M.D.; Richard L. Goode, M.D.; Wayne F. Larrabee, M.D.; Ira D. Papel, M.D.; J. Regan Thomas, M.D.

## ELECTIONS

The American Board of Medical Specialties is the umbrella organization of the 24 recognized certifying organizations in the United States. Otolaryngology is represented by Drs. Robert W. Cantrell, Gerald B. Healy and M. Eugene Tardy, Jr. Alternate representatives are Drs. Charles W. Cummings, Michael E. Johns, and Frank N. Ritter. Dr. Byron J. Bailey is Treasurer of the ABMS, and Dr. Gerald B. Healy serves on the Committee on Subcertification and Recertification. Dr. Jerome C. Goldstein represents the Council of Medical Specialty Societies to the ABMS.

**1995-1996 Examination Dates:** The 1995 written examination will be September 24, 1995, and the subsequent oral examination will be March 10-11, 1996. Both examinations will be given at the Palmer House.

Respectfully submitted,  
*Robert I. Kohut, M.D.*  
for Warren Y. Adkins, Jr., M.D.

## REPORT OF THE AWARD OF MERIT COMMITTEE

The Award of Merit Committee, consisting of our distinguished President, Dr. Robert A. Jahrsdoerfer, Dr. Robert I. Kohut, Dr. Derald E. Brackmann, Dr. A. Julianna Gulya, and myself as Chairman, met February 11, 1995 at 3:00 p.m. Dr. D. Thane R. Cody, a distinguished and mar-

velous otologist who has contributed greatly to this organization, was selected by a unanimous vote.

Respectfully submitted,  
*Mansfield F.W. Smith, M.D.*  
Chairman, Award of Merit Committee

**REPORT OF THE NOMINATING COMMITTEE**

The Nominating Committee, consisting of myself, Dr. Roger Boles, Dr. Jack Kartush, Dr. Gail Neely, and Dr. Harold Tabb met and proposes the following slate for your consideration: President, Dr. Derald E. Brackmann; President-Elect, Dr. Joseph C. Farmer, Jr.; Editor-Librar-

ian-Elect, Dr. A. Julianna Gulya; Council, Dr. Charles M. Luetje II, Dr. C. Gary Jackson, Dr. Robert A. Jahrsdoerfer, and Dr. Robert I. Kohut.

Respectfully submitted,  
*John W. House, M.D.*

**REPORT OF THE BOARD OF GOVERNORS REPRESENTATIVE TO THE AMERICAN ACADEMY OF OTOLARYNGOLOGY-HEAD & NECK SURGERY**

The efforts of the Academy continue in a traditional manner in terms of the Annual Meeting and the Continuing Education curriculum. One new program is an audio-cassette series, and the Home Study Course has been re-structured.

On a separate and more intense plane, the Academy has endeavored to proceed with a public relations campaign. The Widmeyer Group, headquartered in Arlington, Virginia, has been secured to spearhead this effort. I am on the Planning Committee and have worked with Academy staff, as well as other members of the Board of Governors, to help direct this public relations campaign. An extensive questionnaire was used to develop an idea of what otolaryngologists in the Academy would most like the campaign to address. It was clear that patients are the target group. The vehicle for getting the message across will be packets that are sent out through state representatives and members of the Board of Governors, as well as interested private practitioners, to use in various programs around the country; the packets will be designed to inform the constituents not only what an otolaryngologist is, but also how the ear, nose, and throat physician has expanded his or her practice to incorporate new technology. In addition, media packets will be developed. All of this bodes well, in my opinion, for the American Otological Society, which will be one of the benefi-

ciaries of this campaign. It seems clear that members of the AOS should be seen as the primary diagnosticians for ear disorders that go beyond the realm of the primary otolaryngologist. Our efforts in terms of the diagnosis and long-term management, both operative and non-operative, of complex ear problems, and our expertise in these areas, will be one of the items addressed in the Public Relations Campaign. It is my personal interest to have this area pursued, and I feel that we can do so without inhibiting the thrust of the message in terms of the primary otolaryngologist. As soon as we get our campaign packet together, I will send a copy to the Council so that it will remain informed about this initiative.

The Academy is involved in developing the position of Research Coordinator, and I serve on the Search Committee. Several individuals have been suggested, and Dr. Edwin Monsell is regarded as an excellent candidate. Should he be selected, I believe that this will be an excellent opportunity for otologists in the Academy to have an increased impact in terms of outcome studies and issues of that nature, which will undoubtedly be addressed by the Research Coordinator. The Academy is also seeking a replacement for Dr. Frank Lucente, Coordinator for Instruction; I am on the relevant Search Committee.

Respectfully submitted,  
*Harold C. Pillsbury, M.D.*



## READING OF COMMUNICATIONS

### **Dr. Joseph C. Farmer, Jr.**

The Council asked me to say a few words today about Dr. C. Gary Jackson. Indeed, the Directors of the *American Journal of Otology*, as well as the entire field of otology/neurotology, take great pride in giving sincere thanks to Gary for years of outstanding leadership as Editor-in-Chief of the *American Journal of Otology*. All physicians concerned with the care of patients with neurotologic disease have benefited from the contributions of notable and well-known leaders in the field over the years. Dr. Jackson certainly belongs to this select group. The Directors of the *Journal*, as well as the members of the Council, express their sincere admiration and respect for

his accomplishments, especially his major contributions toward enhancing the education of, and communication among, otologists around the world. Gary, we shall all be in your debt, and we extend you our sincere gratitude.

### **Dr. Robert A. Jahrsdoerfer**

I would like to commend the excellent work that Mrs. Judy Matz is doing on behalf of the Society; I think that she deserves public recognition for her consistently outstanding efforts. Frankly, I cannot see how the Society would function without her. Please join me in giving her a round of applause.

**Editor's Note:** The following obituary and photograph appeared in *The Deseret News* on Thursday, December 8, 1994, and are reprinted with permission of the author and editor, Mr. Fred Keller. The photograph was kindly provided by his son, David S. Dolowitz, Esq. Dr. Dolowitz was elected to the American Otological Society in 1959 and to Senior Membership in 1979.

A. Julianna Gulya, M.D., Editor

David Augustus Dolowitz, born November 3, 1913 in the Bronx, New York City, New York, son of Florence L. and Alexander Dolowitz, died December 7, 1994.

Married Frances Fleisher Dolowitz in 1938; she died in 1968. Children, David S. (Anne), Julia D. Reagan (William), Wilma F. Dolowitz, Dr. Susan D. Morgan, and Fridolyn D. Hicks (Larry).

Married Emma Halvorsen, 1969. Stepchildren, Barbara K. Bank (Barry), and Carole K. Tucker.

Survived by 16 grandchildren.

Education: A.B. from Johns Hopkins University 1933; M.D. Yale University 1937; M.A. in anatomy, University of Utah 1951; Honorary Ph.D. of Science Southern Utah State University 1978. Member Sigma Xi 1948. Sigma Alpha Eta 1958. University of Utah Emeritus Alumni Association-Merit Honour Award 1983. NIH Fellowship University of Lund Sweden 1960-1961 Lund Sweden. Captain United States Army Medical Corp with service in the Pacific Theater 1943-1945. Taught at Johns Hopkins University 1939-1943; University of Utah 1943-1979. Chairman of Division of Otolaryngology at University of Utah 1949-1968, then returned to private practice. Remained as adjunct clinical professor until 1979 U of U Medical School. Dixie College-Instructor of human biology 1987-1991. Memberships: American Otological Society Centurian Club, American Laryngological Rhinological, and Otological Association, vice president, Western Section. American Bronchoesophagological Association-Senior Member. American Academy Otophological and Otolaryngology Association. Multiple of Medical awards and community service awards.

Served on the board of directors of the Pioneer Craft House from 1957 until the time of his death. Past president of Yale Club of Utah, served on Chamber of Commerce of Salt Lake City 1948-1978. Served on the board of the Friends of the University Libraries 1973 until the time of his death. Member of the Utah Endowment for the Humanities Committee 1988 until the time of his death. Wrote for the



David A. Dolowitz  
1913-1994

Bulletin, and was a member of the editorial board of the Utah Section of the American Medical Society, Received Certificate of Award Retired Senior Volunteer Programs 1992. Served as local judge for Sterling Scholarships.

Wrote numerous articles for medical journals and medical texts, one of which was used by medical schools as the basic text for otolaryngology.

Past mayor of Toquerville, Utah, as well as City Councilman and Treasurer.

Served in many other social and political and medical positions.

Donations can be made to the Dolowitz Fund at the University of Utah in either the School of Nursing or the Department of Communications.

Funeral will be Friday, December 9, 1994, 10 a.m. at Evans and Early Mortuary, 570 East 100 South, Salt Lake City.

**Editor's Note:** The following obituary and photograph appeared in the February, 1995 *American Academy of Otolaryngology–Head and Neck Surgery Bulletin*, and is reprinted with the permission of the author, Loring W. Pratt, M.D., and the Editor of the *American Academy of Otolaryngology–Head and Neck Surgery Bulletin*, Jerome C. Goldstein, M.D. Dr. Goodhill was elected to the American Otological Society in 1950, to the Presidency in 1976, and to Senior Membership in 1985.

A. Julianna Gulya, M.D., Editor

Victor Goodhill, M.D., one of our premier otologists and Past President of the American Academy of Otolaryngology–Head and Neck Surgery (1979) died on 23 December 1994 at the age of 83. He was a distinguished surgeon, teacher, and musician.

Born in Boston, he graduated from the USC Medical School in 1936, where he later taught as Clinical Professor. He was a founding partner of the Los Angeles Otosurgical Group, and maintained his private practice with that organization. In 1960 Dr. Goodhill joined the faculty at UCLA where he remained for the duration of his career.

He was an accomplished and dedicated teacher, "the sort of teacher who changes a student's life." Many of his students have succeeded as department heads at universities throughout the world. As an example of his commitment to teaching, and his innate showmanship, one can remember with pleasure the presentation he made on Beethoven's deafness and its effect on the musical compositions of the Master. He first played Beethoven's music and then replayed it through electronic filters with tinnitus and hearing loss simulated, so that the listener could hear how the music sounded to Beethoven as his hearing loss progressed. He clearly made the point that the composition of music, for Beethoven, was a cerebral and not an auditory function. He postulated Paget's disease as the cause of Beethoven's deafness, rather than the more commonly accepted syphilis.

He wrote the first textbook on stapes surgery in 1960 and in 1980 wrote *Ear Diseases, Deafness and Dizziness*, now in revision for its second edition. He was the author of over 150 articles in medical journals, and 25 chapters in textbooks. In 1992 he was a consultant for an AMA video clinic on hearing loss.

His scholarly accomplishments were recognized by Phi Beta Kappa and Alpha Omega Alpha. He was awarded the degree of Doctor of Humane Letters (honoris causa) by the University of Judaism and the Jewish Theological Seminary. He received the Maimonides Award of Wisconsin in 1974.

Dr. Goodhill's surgical career began in the pre-antibiotic era, and he was skilled in the treatment of



Victor Goodhill  
1911–1994

both infectious and rehabilitative surgery of the ear and temporal bone. He progressed through all of the varieties of surgery for otosclerosis.

He helped establish the Tracy Clinic, working with Mrs. Spencer Tracy. He was affiliated with the Cedars-Sinai Medical Center, Los Angeles Children's Hospital, UCLA Hospital, and he was both a Fellow and former Governor of the American College of Surgeons.

He enjoyed much recognition for his work.

He received the Fifth Joseph Toynbee Memorial Lecturership at the Royal College of Surgeons in England in 1976, the Sir William Wilde Discourse, Royal Society of Surgeons of Ireland in 1983 and the

## IN MEMORIAM

Distinguished Service Award from the California Speech, Language, Hearing Association in 1985.

As a boy, he studied at the Boston Conservatory of Music. This interest never left him, and he was an accomplished violinist. He was involved with the Music Guild, and brought world class chamber music to Los Angeles.

He was committed to and active in the Jewish community. He was chairman of the Board of Overseers of the University of Judaism, Member of the Board of Governors at Sinai Temple, Member of the

Board of Overseers of the Jewish Theological Seminary in New York and a Vice President of the Brandeis-Dardan Institute in Brandeis, California.

He is survived by his wife Ruth, son Dean, daughter Barbara, and grandchildren Gina, Spencer, Marissa, and Lily.

Funeral Services were held on Wednesday, 28 December 1994 in the TaNaCh Chapel of Mount Sinai Cemetery in the San Fernando Valley.

*Loring W. Pratt, M.D.*

**Editor's Note:** The following obituary and photograph appeared in the Friday, March 24, 1995 *Indianapolis Star*, and are reprinted with permission of the author. The photograph was kindly provided by his son, Robert D. McQuiston, M.D. Dr. Ralph McQuiston was elected to the American Otological Society in 1952 and to Senior Membership in 1980.

A. Julianna Gulya, M.D., Editor

Services for Dr. Ralph J. McQuiston, 89, Indianapolis, a prominent otorhinolaryngologist, will be at 1 p.m. Saturday in Flanner & Buchanan Broad Ripple Mortuary, with calling from 11 a.m.

Burial will be in Crown Hill Cemetery.

He died Wednesday.

An ear, nose and throat specialist, Dr. McQuiston practiced medicine 50 years, retiring in 1984.

He was professor emeritus of the Department of Otolaryngology at Indiana University Medical Center.

He had served as chairman of the Credential Committee of the American College of Surgeons.

Honored by three different governors, Dr. McQuiston received a Citation for Meritorious Service in 1956, was named Sagamore of the Wabash in 1967 and was recognized as a Distinguished Hoosier in 1971.

He was an Army veteran of World War II, serving as a flight surgeon.

Dr. McQuiston was a member of Geist Christian Church. He was a member of the Indiana Basketball Hall of Fame.

He was a graduate of Franklin College, where he played basketball as a guard, and the Indiana University School of Medicine. He later served on the board of trustees at Franklin College and was an associate professor at IU for 20 years.

Memorial contributions may be made to the church or Franklin College.

Survivors: wife Jean Billings McQuiston, daughter Peggy Jay Kitterman, son Dr. Robert D. McQuiston, stepdaughter Tonya Caruso, sister Mary Redmeier, 10 grandchildren, and a great-grandchild.



Ralph J. McQuiston  
1906-1995

## IN MEMORIAM

**Editor's Note:** The following obituary and photograph are provided by Robert I. Kohut, M.D. Ms. Parks admirably served the Society in an administrative capacity from 1982 to 1995.

A. Julianna Gulya, M.D., Editor

Ruth Ellen Boles Parks died March 10, 1995. This memoriam is written to acknowledge our gratitude for her untiring contribution to The American Otolological Society from 1982 to 1995. Her diligence enhanced solid growth of the Society.

Ruth was born in New Jersey, December 7, 1942 and grew up in North Carolina in the town of Elkin in the foothills of the Brushy Mountains. From Elkin, one can observe the first snow falls on the peaks. She often recalled the beauty and peaceful sense of this sight. Her organizational abilities, it seems, were partially endowed by her genes. Her parents delegated family responsibilities to Ruth, her sister and her brothers. Ruth sometimes commented that the responsibilities were "lopsided" in that she and her sister "had to do more than the boys."

Her time in college added to these organizational abilities. Just before the age of 20, she joined the staff at the Bowman Gray School of Medicine of Wake Forest University as Secretary to Dr. Henry Valk. Dr. Valk, who is a legend in his own right, had many responsibilities with which "Ruthie," as he called her, was instantly involved. In other words, she was the person "who made it go." She worked with Dr. Valk for 16 years until his retirement, an example of her loyalty.

She joined the Department of Otolaryngology in 1979 as Administrative Secretary and soon became Administrative Manager of the Department. Budget matters always confronted the department. As she developed budget proposals, it became obvious that the salary increases she proposed for herself were always the smallest, an example of her giving nature. She was the confidant of not only the Department Chairman but all of the support staff and many of the faculty, an example of her personal warmth and respect for others.

She joined in the administration matters of the AOS making cogent observations, giving sound advice, and listening to the members, all of whom she recognized and to many she became a close friend. Her abilities were astounding. One AOS member stated, "I know why our societies secretary appears to be doing a good job. Ruth was able to straighten out a major problem in my office—and she did it over the phone!" Being pragmatic and expedient about cash flow problems, she agreed to help solve some of the Societies financial problems by having the Secretary and the Editor pay the bills with reim-



Ruth E.B. Parks  
1942–1995

bursement coming only after identifying adequate cash reserves. Only Ruth could reassure two individuals such as these about the logic of this solution. Her organization of the Society's records and archival materials contributed towards clarification of the Society's table of organization regarding the Otolological Society and its Foundation for research in otology.

She had to handle the administration matters of the Society's IRS audit. The auditor was impressed with her candid nature and stated that his review was pleasant, contrary to some of his experiences elsewhere. He gave the Society "a clean bill of health" and thanked Ruth for her help. Things generally worked out that way when Ruth was involved.

Ruth Parks was very active in the Association of Academic Administrators of Otolaryngology. She was recruited to become President of the Association. A commemorative lecture fund has been established in her memory.

**Editor's Note:** The following obituary is provided by Bruce W. Pearson, M.D., and the photograph is furnished by Thomas J. McDonald, M.D. Dr. Simonton was elected to the American Otological Society in 1952 and to Senior Membership in 1976.

A. Julianna Gulya, M.D., Editor

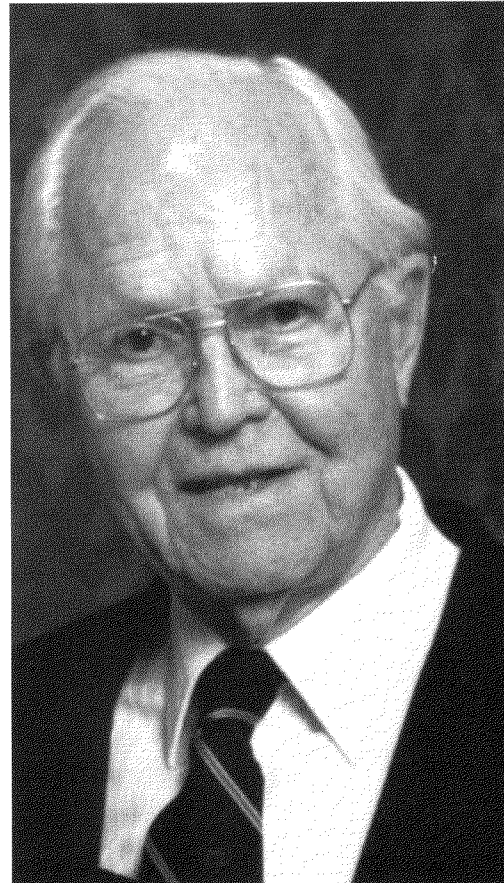
Kinsey Simonton was born in Wamego, Kansas, February 21, 1908, the son of Harriet Viola Deweese and Edgar Locke Simonton. He attended the public schools in Wendell, Idaho, and in 1925 enrolled in the University of Oregon. In 1927, he transferred to George Washington University in Washington, D.C., where he received the degrees of Bachelor of Science in 1930 and Doctor of Medicine in 1933. He served his internship at Gallinger Hospital in Washington.

In 1934 he entered the Mayo Graduate School of Medicine in Rochester Minnesota as a resident in otolaryngology and rhinology. He was appointed to the staff of the Mayo Clinic in 1937 and received the degree of Master of Science in Otolaryngology and Rhinology from the University of Minnesota in 1938. He was certified as a specialist in otolaryngology and rhinology in 1939, and became an instructor in the Mayo Graduate School of Medicine that same year. He was advanced to assistant professor in 1943, to associate professor in 1947, and to professor in 1961.

Dr. Simonton was an officer in the Medical Reserve, U.S. Army, when he was ordered to active duty in 1943 with the grade of Major. Assigned to the 237th Station Hospital, an affiliated unit formed at the Mayo Clinic, he served in New Guinea and the Philippines and with the 71st General Hospital and 188th General Hospital in the Philippine Islands. He was released to civilian life in 1946 with the rank of Lieutenant Colonel.

In 1958, Dr. Simonton became chairman of the section of Otolaryngology and Rhinology of the Mayo Clinic in Rochester, and became a senior consultant in 1968. Throughout his career he maintained a particular interest in diseases of the ear, and especially the mechanisms of hearing in airline pilots and others subjected to excessive background noise. Pediatric otology was also a focus of his attention, and he collaborated in the development a screening test of hearing for preschool children.

Dr. Simonton contributed extensively to the literature of his specialty, and served as a member and leader in several professional societies. A partial listing includes the Minnesota Academy of Ophthalmology and Otolaryngology, president 1957; the Minnesota State Medical Association, chairman of the Committee on Conservation of Hearing, and



Kinsey M. Simonton  
1908-1994

the Advisory Committee to the Selective Service System; The Deafness Research Foundation, Minnesota Chairman; the American Academy of Ophthalmology and Otolaryngology, Committee on Conservation of Hearing, Award of Merit; the American Laryngological, Rhinological, and Otological Society, Inc., Vice President; the American Otologic Society; the American Laryngological Association; the American College of Surgeons; the Communicative Disorder Research Training Committee of the National Institutes of Health; the Society of Sigma Xi and the Sigma Chi social fraternity.

In 1970, Dr. Simonton moved to Atlanta Georgia, where he worked in private practice until 1976. At

## IN MEMORIAM

the invitation of the Persian government, he then went to Shiraz, Iran, where for two years he taught on the medical faculty of Palavi University. He retired to Boca Raton, Florida in 1979, and in 1988, he moved to Ponte Vedra, Florida, near Jacksonville. As the Section of Otolaryngology at Mayo Clinic Jacksonville developed, he attended rounds, lectured eloquently on Otology and on Mayo history, and created the original temporal bone dissection set at that site.

Dr. Simonton was married to Miss Anita Brunet Dunlap of Washington, D.C., on March 30, 1937.

They have two children: Kay (Mrs. Charles Urquart Foster of Duxbury, MS) and Bruce Dunlap Simonton of Des Moines, IA. Simie, as he was known to many of his friends and colleagues, died at St. Lukes Hospital on Dec 14. He was a skillful surgeon, a consummate gentleman, and a lifelong contributor to our art and science. Si, it was our great privilege to know you as a colleague and a friend. Thanks for enriching this world with your many talents and gifts. You will be deeply missed and long remembered.



**Editor's Note:** The following obituary and photograph appeared in the *American Journal of Otolaryngology*, July, 1995, Volume 16, Number 4, and are reprinted with permission of the author, Roger Boles, M.D., and the Editor of *The American Journal of Otolaryngology*, Robert K. Jackler, M.D. Dr. Work was elected to the American Otological Society in 1953, to the Presidency in 1972, and to Senior Membership in 1975.

A. Julianna Gulya, M.D., Editor

Walter P. Work, M.D., Professor and Chairman Emeritus of the Department of Otorhinolaryngology at the University of Michigan, passed away on November 4, 1994, at the age of 85, in his retirement home at Green Valley, Arizona. Dr. Work was a leader of national stature in our field as well as an inspirational teacher of hundreds of medical students and residents. His career spanned the end of the pre-antibiotic era and the emergence of our expanded otolaryngology interests following World War II, including head and neck cancer surgery and other otologic surgery. He was a fundamentally sound clinician and teacher who always stressed the importance of the patient in the medical scheme of things and emphasized basic fundamentals of diagnosis and treatment and particularly of surgery.

Dr. Work was born in Cannonsburg, Pennsylvania, as one of a large family. While he was still young the family moved to a farm in Oxford, Ohio, where he completed his early schooling and attended Miami University for a brief period before transferring to the University of Michigan. He received his B.A. degree from the University of Michigan in 1931 and went on to medical school there, receiving his M.D. degree in 1935. He took his surgical otolaryngological residency training at the University of Michigan, under the direction of Drs. Albert C. Furstenberg and James E. Maxwell. He was certified by the American Board of Otolaryngology in 1941 and practiced in San Francisco for a short period before entering the Army Medical Corps, in which he served for 4 years throughout World War II. During his time in the Army he did pioneering work in audiology and helped to set some of the early standards in this new field. Following World War II, he returned to private practice in San Francisco and joined the clinical faculty of the Stanford Medical School. He also established an independent residency program at the San Francisco Veteran's Administration Hospital that he directed for almost 15 years. Dr. Robin Michelson, who pioneered the cochlear implant, was Dr. Work's first resident in his VA residency program. In 1961, Dr. Work was appointed Chairman of the Department of Otolaryngology at the University of Michigan, and he remained there until his retirement in the



Walter P. Work  
1909–1994

late 1970s. Following retirement Dr. and Mrs. Work moved to Green Valley, Arizona, near Tucson, where Dr. Work joined the clinical faculty at the University of Arizona School of Medicine and taught medical students and residents for almost a decade.

There was hardly an area or society in otolaryngology in which Dr. Work did not participate and make major leadership and clinical contributions. He served for many years on the American Board of Otolaryngology and became both President and Executive Secretary. He was a founding member of the Society of University Otolaryngologists and was its President in 1967–68. He was President of the Amer-

## IN MEMORIAM

ican Otolological Society in 1971–72 and President of the Triological Society in 1973. He was also Second Vice President of the American College of Surgeons. He was a member of the American Laryngological Association and was honored with its Newcomb Award in 1968.

In his 1983 Triological Guest of Honor address, Dr. Work summarized his career philosophy of “having invested in people” rather than to have built buildings or to have built or acquired other material things. This philosophy was not lost on all of the residents he trained who, before his retirement from Michigan, established The Walter P. Work Society, which has grown and has met every

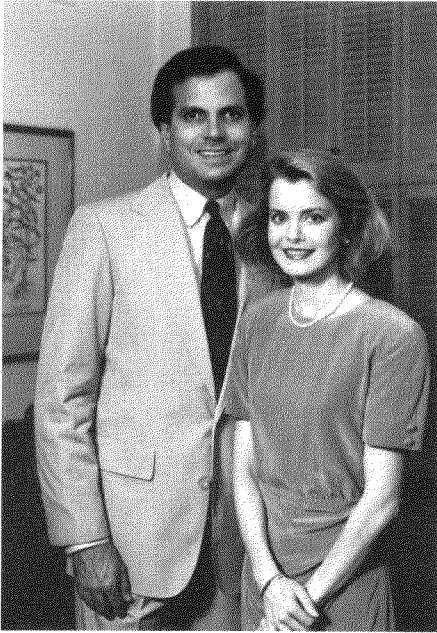
year since and has been a most popular and pleasant scientific-social gathering for all of us who are so grateful to him for his leadership and inspiration. He was in attendance at the meeting of the society in the fall of 1994, just a few weeks before his death.

Dr. Work was married to Alice Tyler in 1939 and they had four children who have borne them eight grandchildren.

*Roger Boles, M.D.*  
Professor of Otolaryngology—  
Head and Neck Surgery  
The University of California, San Francisco  
San Francisco, California

## NEW MEMBERS 1995

### *Active*



**Ronald Amedee, M.D.**

Department of Otolaryngology–Head and Neck Surgery  
SL-59, 1430 Tulane Avenue  
New Orleans, LA 70112-2699  
(with wife Elisabeth)



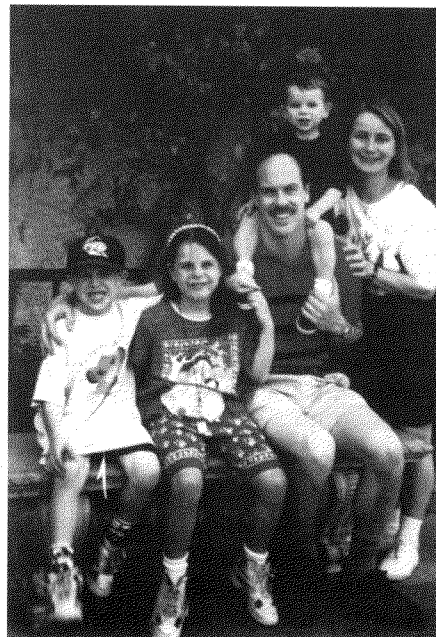
**Charles W. Beatty, M.D.**

Mayo Clinic  
Department of Otolaryngology  
200 First Street, SW, Suite 100  
Rochester, MN 55905-0001  
(with wife Ann)



**C. Phillip Daspit, M.D.**

222 West Thomas Road, Suite 114  
Phoenix, AZ 85013-4420  
(with wife Diane)



**Thomas L. Eby, M.D.**

University of Alabama-Birmingham  
Department of Otolaryngology  
1501 5th Avenue South  
Birmingham, AL 35233-1614  
(on his right are Brendon and Margaret,  
and on his left are Conor and wife Mary)



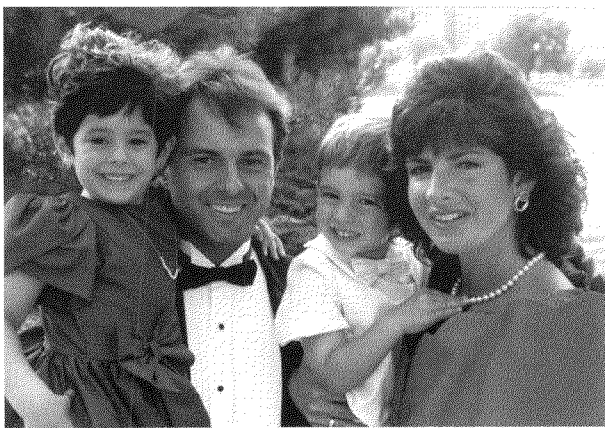
**Joel A. Goebel, M.D.**

Department of Otolaryngology  
 Washington University School of Medicine  
 517 South Euclid Avenue  
 Box 8115  
 St Louis, MO 63110-1007  
 (with wife Vicky, Shannon (left), and Stacey (right))



**Paul R. Lambert, M.D.**

Department of Otolaryngology–Head and Neck Surgery  
 University of Virginia Health Sciences Center  
 Box 430  
 Charlottesville, VA 22908-0430  
 (with wife Debbie in the foreground; in the back row,  
 from left to right, are Paul, Leslie and Lara)



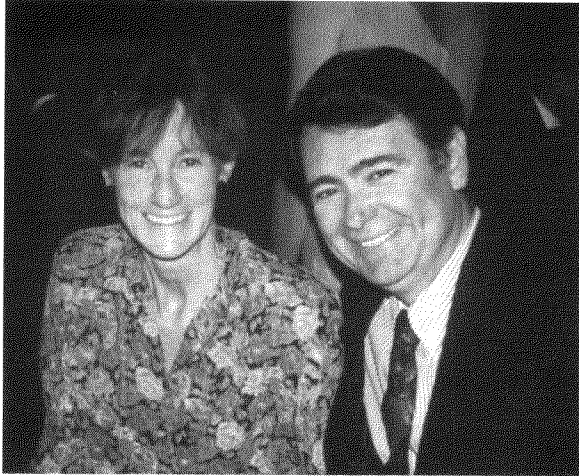
**John P. Leonetti, M.D.**

Department of Otolaryngology  
 Loyola University Medical Center  
 2160 South First Avenue  
 Building 105, Room 1870  
 Maywood, IL 60153-3304  
 (holding Gianna Marie, wife Mary Grace holding  
 Michael Patrick)

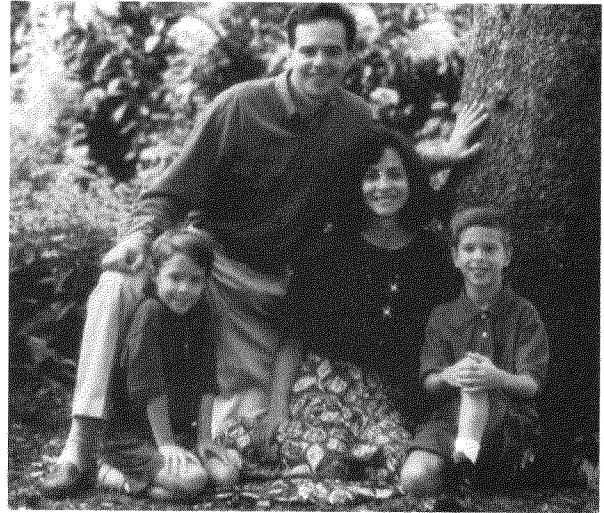


**Edwin M. Monsell, M.D., Ph.D.**

Department of Otolaryngology–Head and Neck Surgery  
 K8, Henry Ford Hospital  
 2799 West Grand Boulevard  
 Detroit, MI 48202-2608  
 (with wife Wendy, Sarah (left) and Susan (right))



**Ralph A. Nelson, M.D.**  
House Ear Institute, Inc.  
2100 West Third Street  
Los Angeles, CA 90057-1922  
(with wife Anne)



**John K. Niparko, M.D.**  
601 North Caroline Street  
JHOC-6  
Johns Hopkins Hospital  
Baltimore, MD 21205-1809  
(with wife Angela, Nathan (left) and Kevin (right))



**Dennis S. Poe, M.D.**  
Zero Emerson Place  
Suite 2C  
Boston, MA 02114  
(with wife Milja and Lara)



**Alexander J. Schleuning, M.D.**  
Department of Otolaryngology  
University of Oregon Health Sciences Center  
3181 S.W. Sam Jackson Park Road  
Portland, OR 97201-3011  
(with granddaughter Kristina Bode)



**Clough Shelton, M.D.**

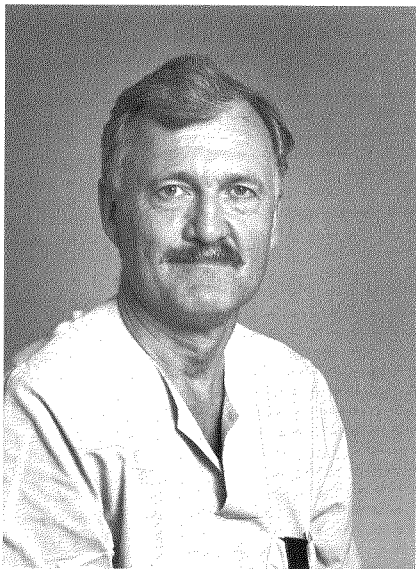
Division of Otolaryngology–Head and Neck Surgery  
University of Utah Medical Center  
3C120  
50 North Medical Drive  
Salt Lake City, UT 84132  
(with wife Kay)

*Associate*



**Karen I. Berliner, Ph.D.**  
2252 Linnington Avenue  
Los Angeles, CA 90064

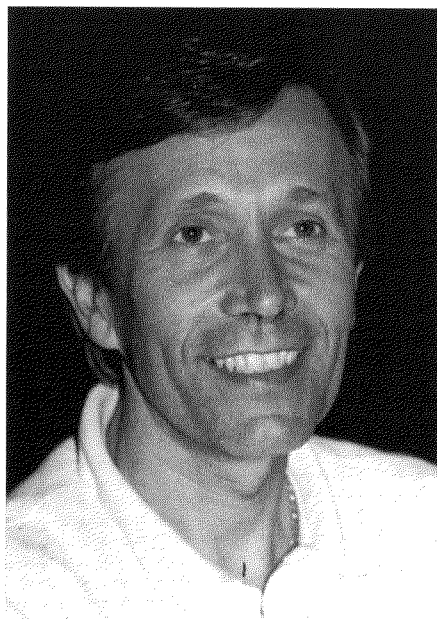
*Corresponding*



**Dan Bagger-Sjöbäck, M.D., Ph.D.**  
Karolinska Hospital  
Department of Otolaryngology  
Stockholm 17176  
Sweden



**J. Barton Booth, M.B.**  
18 Upper Wimpole Street  
London W1M 7TB  
England



**Jean-Bernard Causse, M.D.**  
J. Causse Clinic  
Traverse de Beziers  
Colombiers 34440  
France

# MEMBERS OF THE AMERICAN OTOLOGICAL SOCIETY, INC. 1995–1996

## *Active Members*

- 1987 Adkins, Warren Y., Department of Otolaryngology, Medical Univ. of South Carolina, 171 Ashley Avenue, Charleston, SC 29425
- 1988 Adour, Kedar, Sir Charles Bell Society, 1000 Green Street #1203, San Francisco, Ca 94133
- 1982 Alberti, Peter W., 259 Glencairn Avenue, Toronto, Ontario, Canada M5N 1T8
- 1970 Alford, Bobby R., 6501 Fannin Street, Houston, TX 77030
- 1987 Althaus, Sean R., 5201 Norris Canyon Rd. #230, San Ramon, CA 94583-5405
- 1995 Amedee, Ronald, Dept. of Otolaryngology-HNS, SL-59, 1430 Tulane Avenue, New Orleans, LA 70112-2699
- 1985 Applebaum, Edward, 1855 West Taylor Street, Chicago, IL 60612
- 1993 Babin, Richard W., River Bend Head & Neck Assoc., 6570 Stage Road, Suite 245, Bartlett, TN 38134
- 1991 Balkany, Thomas J., Univ. of Miami School of Medicine, Dept of Otolaryngology, PO Box 016960, Miami, FL 33101
- 1992 Bartels, Loren J., Harbourside Medical Tower-Ste 610, 4 Columbia Drive, Tampa, FL 33606
- 1995 Beatty, Charles W., Mayo Clinic, Dept. of Otolaryngology, 200 First Avenue, SW, Rochester, MN 55905
- 1983 Black, F. Owen, 2222 N.W. Lovejoy, Suite 411, Portland, OR 97210
- 1977 Bluestone, Charles D., 125 DeSoto Street, Pittsburgh, PA 15213
- 1982 Boles, Roger, 400 Parnassus Avenue, San Francisco, CA 94143
- 1979 Brackmann, Derald E., 2100 West Third Street-1st Floor, Los Angeles, CA 90057
- 1978 Britton, B. Hill, Univ. of Oklahoma-HSC, Dept. of Otorhinolaryngology, 3SP226 P.O. Box 26901, Oklahoma City, OK 73190-3048
- 1988 Brookhouser, Patrick E., Boystown National Institute of Communication Disorders in Children, 555 N. 30th Street, Omaha, NE 68131
- 1991 Canalis, Rinaldo F., Div. Head & Neck Surgery, Harbor-UCLA, 1000 W. Carson Street, Torrance, CA 90509
- 1979 Cantrell, Robert W., University of Virginia, Department of Otolaryngology, Box 430 Charlottesville, Va 22908
- 1975 Catlin, Francis I., 13307 Queensbury Lane, Houston, TX 77079
- 1984 Chole, Richard, Otology Research Lab, 1159 Surge III, Davis, CA 95616
- 1976 Clemis, Jack D., 151 North Michigan Avenue, Chicago, IL 60601
- 1985 Cohen, Noel L., Dept of Otolaryngology, NYU Medical Center, 550 First Avenue, New York, NY 10016
- 1991 Coker, Newton J., Dept. of Otolaryngology, Baylor College of Medicine, One Baylor Plaza, Houston, TX 77030
- 1995 Daspit, C. Phillip, 222 W. Thomas Rd., Suite 114, Phoenix AZ 85013
- 1975 Dayal, Vijay S., Department of Otolaryngology, University of Chicago Medical Ctr, Chicago, IL 60637
- 1991 De la Cruz, Antonio, 2100 W. Third Street, Los Angeles, CA 90057
- 1991 Dickins, John R.E., 1200 Medical Towers Building, 9601 Lile Drive, Little Rock, AR 72205
- 1985 Dobie, Robert A., Dept of Otolaryngology, UTSA, 7703 Floyd Curl Drive, San Antonio, TX 78284
- 1987 Doyle, Patrick J., #150-809 West 41st Avenue, Vancouver, BC, Canada V5Z 2N6
- 1988 Duckert, Larry G., Department of Otolaryngology, P.O. Box 351928, RL-30, University of Washington, Seattle, WA 98195
- 1995 Eby, Thomas L., University of Alabama-Birmingham, Dept. of Otolaryngology, 1501 5th Avenue South, Birmingham, AL 35233
- 1988 Eden, Avrim R., Dept. of Otolaryngology, Mount Sinai Medical Ctr, Box 1189, Fifth Ave & 100 St, New York, NY 10029-6574
- 1990 Emmett, John R., 6133 Poplar Pike at Ridgeway, Memphis, TN 38119
- 1981 Eviatar, Abraham, 1575 Blondell Avenue, Suite 150 Bronx, NY 10461
- 1994 Facer, George W., Mayo Clinic, 200 First Street, S.W., Rochester, MN 55905
- 1984 Farmer, Joseph C., Division of Otolaryngology, Duke Univ Medical Ctr, Box 3805, Durham, NC 27710
- 1990 Farrior, Jay B. III, 509 Bay Street, Tampa, FL 33606
- 1978 Fredrickson, John M., 517 South Euclid, Box 8115, St. Louis, MO 63110
- 1969 Gacek, Richard R., 750 East Adams Street, Syracuse, NY 13210
- 1987 Gantz, Bruce J., Dept of Otolaryngology, University of Iowa, 200 Hawkins Drive, Iowa City, IA 52242
- 1983 Gardner, L. Gale Jr., 899 Madison Avenue, Suite 602A, Memphis, TN 38103
- 1987 Gates, George A., University of Washington, Department of Otolaryngology, 1959 NE Pacific St. RL-30, Seattle, WA 98195



- 1973 Glasscock, Michael E. III, 300 20th Avenue, North, Suite 502, Nashville, TN 37203
- 1995 Goebel, Joel A., 517 South Euclid, Box 8115, St. Louis, MO 63110
- 1989 Goldenberg, Robert A., 111 West First St, Suite 1000, Dayton, OH 45402
- 1990 Goode, Richard L., 440 Old Oak Ct., Los Altos, CA 94022
- 1992 Goycoolea, Marcos V., San Crescente 70, Las Condes, Santiago, Chile
- 1979 Graham, Malcolm D., Georgia Ear Institute, 4700 Waters Avenue, Box 23665, Savannah, GA 31404-3665
- 1991 Gulya, A. Julianna, Georgetown Univ. Medical Ctr., 3800 Reservoir Road, N.W., Washington, DC 20007
- 1987 Harker, Lee A., Boystown National Research Hospital, 555 North 30th Street, Omaha, NE 68131
- 1987 Harner, Stephen G., Mayo Clinic, 200 First Street SW, Rochester, MN 55905
- 1988 Harris, Jeffrey P., 9350 Campus Point Drive, BOX 0970, LaJolla, CA 92037-0970
- 1992 Hart, Cecil W.J., 707 North Fairbanks Ct, Ste 1000, Chicago, IL 60611
- 1984 Hawke, W. Michael, 1849 Yonge Street, Ste. 10, Toronto, Ontario M4S 1Y2 Canada
- 1992 Hoffman, Ronald A., 1430 Second Avenue, Suite 110, New York, NY 10021
- 1984 House, John W., 2100 West Third Street, Los Angeles, CA 90057
- 1987 Hughes, Gordon B., Dept of Otolaryngology, Cleveland Clinic, 9500 Euclid Avenue Cleveland, OH 44195
- 1992 Jackler, Robert K., Univ of California-San Francisco, 350 Parnassus Ave, Suite 210, San Francisco, CA 94117
- 1994 Jackson, Carol A., 361 Hospital Road, Suite 325 Newport Beach, CA 92663
- 1990 Jackson, C. Gary, The Otology Group, 300 20th Avenue, North, Nashville, TN 37203
- 1992 Jahn, Anthony, 556 Eagle Rock Avenue, Roseland, NJ 07068
- 1982 Jahrsdoerfer, Robert A., Dept. of Otolaryngology, University of Virginia Med.Ctr., Box 430, Charlottesville, VA 22908
- 1987 Jenkins, Herman A., Dept of Otolaryngology, Baylor College of Medicine, Houston, TX 77030
- 1990 Jung, Timothy K., ENT Division, Loma Linda, 11790 Pecan Way, Loma Linda, CA 92354
- 1988 Kamerer, Donald B., Eye and Ear Hospital, 203 Lothrop Street, Suite 500, Pittsburgh, PA 15213
- 1991 Kartush, Jack, Michigan Ear Institute, 27555 Midlebelt Road, Farmington Hills, MI 48334
- 1992 Katsarkas, Anthanasios, Royal Victoria Hospital, 687 Pine Avenue, W Montreal, PQ H3A, Canada
- 1987 Keim, Robert J., 13504 Green Cedar Lane, Oklahoma City, OK 73131
- 1981 Kinney, Sam E., 9500 Euclid Avenue, Cleveland, OH 44106
- 1976 Kohut, Robert I., Bowman Gray School of Medicine, Dept of Otolaryngology, Medical Center Boulevard, Winston-Salem, NC 27157-1034
- 1991 Konrad, Horst, Southern Illinois University, School of Medicine, Div of Otolaryngology, PO Box 19230, Springfield, IL 62794
- 1993 Kumar, Arvind, 1855 W. Taylor St., M/C 648, Chicago, IL 60612
- 1995 Lambert, Paul R., Dept. of Otolaryngology-HNS, University of Virginia Med.Ctr., Health Sciences Center, Charlottesville, VA 22908
- 1995 Leonetti, John P., Loyola University Medical Center, 2160 S. First Avenue, Bldg. 105, Room 1870, Maywood, IL 60153
- 1993 Lesinski, S. George, 629 Oak Street, Suite 201, Cincinnati, OH 45206
- 1987 Lindeman, Roger C., 1100 Ninth Avenue, #900, Seattle, WA 98101
- 1988 Lippy, William H., 3893 East Market Street, Warren, OH 44484
- 1991 Luetje, Charles M., Otologic Center, Inc., Penntower Office Center, 3100 Broadway, Suite 509, Kansas City, MO 64111
- 1970 Maddox, H. Edward, 7777 Southwest Freeway, Houston, TX 77074
- 1987 Mangham, Charles A. Jr., Seattle Ear Clinic, 600 Broadway, Suite 340, Seattle, WA 98122
- 1989 Maniglia, Anthony J., Dept. of Otolaryngology, Case Western Reserve Hosp., 11100 Euclid Avenue, Cleveland, OH 44106-1736
- 1985 Mathog, Robert H., 540 East Canfield Avenue, Detroit, MI 48201
- 1992 Mattox, Douglas E., 1314 Locust Avenue, Ruxton, MD 21204
- 1979 Matz, Gregory J., Loyola University Medical Center, Dept of Otolaryngology-HNS, 2160 South First Avenue, Bldg. 105, Room 1870, Maywood, IL 60153
- 1965 McCabe, Brian F., University of Iowa, Dept of Otolaryngology, Iowa City, Iowa 52242
- 1987 McDonald, Thomas J.P., Mayo Clinic, 200 First Street, SW, Rochester, MN 55905
- 1981 Meyerhoff, William L., Univ of Texas Health Science Ctr., 5323 Harry Hines Blvd. GL-208, Dallas, TX 75235
- 1987 Miyamoto, Richard T., 702 Barnhill Drive, Ste. 860, Indianapolis, IN 46202
- 1995 Monsell, Edwin M., Dept. of Otolaryngology-HNS, Henry Ford Hospital, 2799 W. Grand Blvd., Detroit, MI 48202
- 1975 Montgomery, William, 243 Charles Street, Boston, MA 02114
- 1988 Nadol, Joseph B. Jr., 243 Charles Street, Boston, MA 02114
- 1987 Nedzelski, Julian M., Dept of Otolaryngology, Sunnybrook Medical Center, 1075 Bayview Avenue, Toronto, Ontario M4N3M5, Canada
- 1985 Neely, J. Gail, Washington University School of Med., 517 South Euclid Avenue, Box 8115, St. Louis, MO 63110
- 1995 Nelson, Ralph A., House Ear Institute, Inc., 2100 West Third Street, Los Angeles, CA 90057
- 1995 Niparko, John P., Dept. of Otolaryngology-HNS, Johns Hopkins Hospital, P.O. Box 41402, Baltimore, MD 21203-6402

- 1993 Olsson, James E., Texas Neurosciences Institute, 4410 Medical Drive, Suite 550, San Antonio, TX 78229
- 1968 Paparella, Michael M., 701 25th Avenue South, Ste. 200, Minneapolis, MN 55454
- 1985 Pappas, Dennis, 2937 7th Avenue South, Birmingham, AL 35233
- 1983 Pappas, James J., 1200 Medical Towers Building, 9601 Lile Drive, Little Rock, AR 72205
- 1982 Parisier, Simon C., 210 East 64th Street, New York, NY 10021
- 1986 Parkin, James L., Division of Otolaryngology, Univ of Utah School of Medicine, Salt Lake City, UT 84132
- 1992 Pensak, Myles L., Univ of Cincinnati, College of Medicine, 231 Bethesda Ave, ML 528, Cincinnati, OH 45267-0528
- 1988 Pillsbury, Harold C., 610 Burnett-Womack Bldg, 229H, University of North Carolina, Chapel Hill, NC 27599-7070
- 1995 Poe, Dennis S., Dept. of Otolaryngology, New England Medical Center, Box 850, 750 Washington Street, Boston, MA 02111
- 1989 Proctor, Leonard R., 2007 Fitzwarren, Apt. 102, Baltimore, MD 21209
- 1969 Pulec, Jack, 1245 Wilshire Blvd, Room 503, Los Angeles, CA 90017
- 1989 Radpour, Shokri, Richard L. Roudebush, VA Med Ctr, 1481 West 10th Street, Indianapolis, IN 46202
- 1992 Roland, Peter S., 5323 Harry Hines Blvd., Dallas, TX 75235-9035
- 1972 Ronis, Max L., 3400 North Broad Street, Philadelphia, PA 19140
- 1974 Ruben, Robert, Montefiore Medical Center, 111 East 210th Street VCA-4, Bronx, NY 10467-2490
- 1989 Rybak, Leonard P., 2528 Argonne, Springfield, IL 62794
- 1992 Sasaki, Clarence T., Yale Univ. School of Medicine, Section of Otolaryngology, 333 Cedar Street, Box 333, New Haven, CT 06510
- 1990 Sataloff, Robert T., 1721 Pine Street, Philadelphia, PA 19103
- 1972 Saunders, William H., 456 Clinic Drive, Columbus, OH 43210
- 1983 Schindler, Robert A., A717, 400 Parnassus Avenue, San Francisco, CA 94143
- 1995 Schleuning, Alexander J., 3181 S.W. Sam Jackson Park Road, Portland, OR 97201
- 1990 Schuring, Arnold G., 3893 East Market Street, Warren, OH 44484
- 1993 Schwaber, Mitchell, Vanderbilt University, RM S-2100 Medical Center North, Nashville, TN 37232
- 1967 Shea John J. Jr., Box 17987, 6133 Poplar Pike, Memphis, TN 38119
- 1995 Shelton, Clough, 3C120, 50 North Medical Drive, Salt Lake City, UT 84132
- 1973 Silverstein, Herbert, 1916 Floyd Street, Suite A, Sarasota, FL 33579
- 1972 Singleton, George T., University of Florida, JHMHC, Box J-264, Gainesville, FL 32610
- 1993 Sismanis, Aristides, 1917 Windingridge Drive, Richmond, VA 23233
- 1973 Smith, Mansfield F.W., 2120 Forest Avenue, San Jose, CA 95128
- 1988 Smith, Peter G., Midwest Otologic Group, 621 South New Ballas Rd., St. Louis, MO 63110
- 1979 Spector, Gershon Jerry, 517 South Euclid Avenue, St. Louis, MO 63110
- 1993 Wazen, Jack J., Columbia University, 630 W. 168th Street, New York, NY 10032
- 1975 Wehrs, Roger E., 6465 South Yale, Tulsa, OK 74136
- 1990 Weider, Dudley J., 38 Rip Road, Hanover, NH 03755
- 1987 Wiet, Richard J., 950 York Road, Hinsdale, IL 60521
- 1992 Wilson, David F., 911 N.W. 18th Avenue, Portland, OR 97209
- Senior Members*
- 1988 (1960) Armstrong, Beverly W., 1409 East Blvd 217, Charlotte, NC 28203
- 1994 (1969) Bailey, H.A. Ted Jr., 1200 Medical Towers Bldg., 9601 Lile Drive, Little Rock, AR 72205
- 1990 (1958) Bellucci, Richard J., 162 East 71st Street, New York, NY 10021
- 1988 (1961) Bradley, Wesley H., 13 Saybrook East, Glenmont, NY 12077
- 1988 (1964) Brockman, Seymour J., 222 S. McCarty Dr., Beverly Hills, CA 90212
- 1994 (1969) Buckingham, Richard A., 145 Northwest Highway, Park Ridge, IL 60068
- 1992 (1972) Caparosa, Ralph J., 420 E. North Avenue #402, Pittsburgh, PA 15212-4746
- 1994 (1973) Chandler, J. Ryan, 1700 NW 10th Avenue, Miami, FL 33136
- 1990 (1958) Cody, Claude C. III, 529 E. Friar Tuck Lane, Houston, TX 77024
- 1992 (1969) Cody, D. Thane, 541 LeMaster Dr., Ponte Vedra Beach, FL 32082
- 1990 (1966) Cole, James M., 1301 Red Ln., Danville, PA 17821-1333
- 1989 (1968) Compere, Wesley E., 3755 Avocado Blvd #503, LeMesa, CA 91941
- 1995 (1972) Crabtree, James A., 1332 Westhaven Rd., San Marino, CA 91108
- 1981 (1961) Daly, John F., 1500 Palisase Avenue #27C, Fort Lee, NJ 07024-5318
- 1989 (1958) Derlacki, Eugene L., Northwestern Medical, Faculty Foundation, 707 N. Fairbanks Ct, Ste 1010, Chicago, IL 60611
- 1994 (1974) Donaldson, James A., Seattle Ear Clinic, 600 Broadway, #340, Seattle, WA 98122-5371
- 1971 (1939) Druss, Joseph G., 145 East 92nd Street, New York, NY 10028
- 1993 (1971) Duvall, Arndt J. III, Dept of Otolaryngology, University of Minnesota, Box 478, Minneapolis, MN 55455
- 1973 (1953) Glorig, Aram, 2100 West Third Street, Los Angeles, CA 90057
- 1993 (1970) Harris, Irwin, 10419 Lindbrook, Los Angeles, CA 90024
- 1993 (1973) Harrison, Wiley H., Northwestern Medical Faculty Fnd., 707 N. Fairbanks Ct., Suite 1010, Chicago, IL 60611
- 1992 (1972) Hilding, David A., #1 Hospital Drive, Price, UT 84501

- 1975 (1951) Hilger, Jerome, 409-1700 Lexington Avenue, St. Paul, MN 55118
- 1990 (1970) Hohmann, Albert, 3154 Shoreline Lane, St. Paul, MN 55112-3764
- 1990 (1960) Hough, Jack V., 3400 NW 56th Street, Oklahoma City, OK 73112
- 1975 (1947) House, Howard P., 2100 West Third Street, Los Angeles, CA 90057
- 1995 (1964) House, William F., Newport Lido Medical Center, 361 Hospital Road, Suite 327, Newport Beach, CA 92663
- 1975 (1953) Jordan, Raymond E., 520 Bay Villas Lane, Naples, FL 33963
- 1972 (1952) Juers, Arthur L., 5701 Coach Gate Wynde, Apt 50, Louisville, KY 40207
- 1991 (1967) Linthicum, Fred H. Jr., 2100 West Third Street, Los Angeles, CA 90057
- 1995 (1969) Litton, Ward B., 17 Eagle Pointe Pass, P.O. Box 266, Rapid City, IL 61278
- 1987 (1975) Marcus, Richard E., 691 Sheridan Road, Winnetka, IL 60093
- 1990 (1974) Michelson, Robin P., A717, 400 Parnassus Avenue, San Francisco, CA 94143
- 1989 (1965) Moon, Cary N. Jr., 1135 Inglecress Drive, Charlottesville, VA 22901
- 1987 (1952) Moore, James A., 525 East 68th Street, New York, NY 10021
- 1978 (1957) Myers, David, 2401 Pennsylvania Avenue, Philadelphia, PA 19130
- 1994 (1974) Myers, Eugene, Eye and Ear Institute, 203 Lathrop Street, Suite 500, Pittsburgh, PA 15213
- 1994 (1988) Nager, George T., 550 N. Broadway, Baltimore, MD 21205
- 1993 (1968) Naunton, Ralph F., DCSD-NIDCD EPS-400B, 6120 Executive Boulevard, Rockville, MD 20892
- 1993 (1973) Pennington, Claude L., PO Box 1916, Macon, GA 31202
- 1992 (1975) Powers, W. Hugh, 728 Wind Willow Way, Simi Valley, CA 93065
- 1983 (1959) Proud, Gunner O., 3721 West 87th Street, Shawnee Mission, KS 66206
- 1983 (1958) Rambo, J.H. Thomas, 150 East 77th Street, New York, NY 10021
- 1993 (1972) Ritter, Frank N., 2675 Englave Drive, Ann Arbor, MI 48103
- 1991 (1969) Robinson, Mendell, 130 Waterman Street, Providence, RI 02906
- 1992 (1967) Rubin, Wallace, 3434 Houma Boulevard, Metairie, LA 70006
- 1993 (1967) Ruggles, Richard L., 11201 Shaker Boulevard, Cleveland, OH 44104
- 1994 (1960) Sataloff, Joseph, 1721 Pine Street, Philadelphia, PA 19103
- 1987 (1966) Schlosser, Woodrow D., 1557A Pheasant Walk, Fort Pierce, FL 34950
- 1990 (1957) Schuknecht, Harold F., 243 Charles Street, Boston, MA 02114
- 1975 (1950) Shambaugh, George Jr., 40 South Clay St, Hinsdale, IL 60521
- 1994 (1965) Sheehy, James L., 2100 West Third Street, Los Angeles, CA 90057
- 1995 (1973) Simmons, F. Blair, 300 Pasteur Drive, Room R-135, Palo Alto, CA 94025
- 1980 (1958) Smith, J. Brydon, 21 Farrington Drive, Willowdale, Ontario, M2L 2B4, Canada
- 1993 (1973) Snow, James B. Jr., National Institute on Deafness and Communicative Disorders, 9000 Rockville Pike, 313C02, Bethesda, MD 20892
- 1990 (1967) Stroud, Malcolm H. (address unknown)
- 1971 (1947) Stuart, Edwin A. (address unknown)
- 1990 (1961) Tabb, Harold G., 1430 Tulane Avenue, New Orleans, LA 70112
- 1985 (1965) Taylor, G. Dekle, 13500 Mandarin Road, Jacksonville, FL 32223
- 1984 (1974) Torok, Nicholas, 42 Portwine Road, Claredon Hills, IL 60514
- 1972 (1946) Truex, Edward H., 37 Farmington Road, Wethersfield, CT 06109
- 1981 (1962) Waltner, Jules G., 161 Fort Washington Ave, New York, NY 10032
- 1994 (1972) Ward, Paul H., UCLA School of Medicine, Division of Head and Neck Surgery, 10833 LeConte Ave., 62-132 Center for Health Sciences, Los Angeles, CA 90024
- 1989 (1972) Wilson, William H., 1133 Oneida Street, Denver, CO 80220
- 1986 (1964) Withers, Ben T., 4703 Ivanhoe, Houston, TX 77027
- 1994 (1971) Wolfson, Robert J., 1920 Chestnut Street, Portland, OR 97201
- 1987 (1964) Wright, William K., 3671 Delmonte, Houston, TX 77019
- Emeritus Members*
- 1992 (1977) Bergstrom, Lavonne, 304 20th Street, Manhattan Beach, CA 90266
- 1979 (1963) Boyd, Harold M.E., 313 Via Anita, Redondo Beach, CA 90277-6621
- 1987 (1994) Goin, Donald W., 799 East Hampden Ave., Suite 510, Englewood, CO 80110-2769
- 1973 (1957) Tolan, John F., 3419 47th Avenue NE, Seattle, WA 98105
- Associate Members*
- 1992 Altschuler, Richard A., Ph.D., Kresge Hearing Research Inst., University of Michigan, 1301 N. Ann Street, Ann Arbor, MI 48109-0506
- 1995 Berliner, Karen I., Ph.D. 2252 Linnington Avenue, Los Angeles, CA 90064
- 1979 Bohne, Barbara A., Ph.D., 517 South Euclid Avenue, St. Louis, MO 63110
- 1978 Butler, Robert A., Ph.D., Department of Surgery, University of Chicago, 950 E. 59th Street, Chicago, IL 60637
- 1973 Fernandez, Cesar, M.D., 950 E. 59th Street, Chicago, IL 60637
- 1959 Graybiel, Ashton, M.D., PO Box 4063, Warrington, FL 32507
- 1977 Gussen, Ruth, M.D. (address unknown)
- 1992 Hamid, Mohamed A., Ph.D., 50 Greentree, Moreland Hills, OH 44022
- 1992 Hannley, Maureen T., Ph.D., 2801 Park Center Dr., Alexandria, VA 22302

- 1972 Hawkins, Joseph E. Jr., Ph.D., Kresge Hearing Research Inst., Ann Arbor, MI 48109
- 1989 Hinojosa, Raul, M.D., 5316 Hyde Park Boulevard, Chicago, IL 60615
- 1972 Honrubia, Vicente, M.D., 850 North Beverly Glen Blvd, Los Angeles, CA 90024
- 1973 Igarashi, Makota, M.D., Moto Azabu 3-12-18, Minato-Ku Tokyo, Japan 106
- 1994 Iurato, Salvatore J., M.D., Cattedra Di Bioacustica, dell-Universita di Bari, Policlinico, 70124 Bari, Italy
- 1960 Johnson, Walter H., Ph.D., St. Michael's Hospital, 30 Bond Street, Toronto, Ontario, Canada M5B 1W8
- 1979 Johnsson, Lars-Goran, M.D., Simmarstigen 10A2, Helsinki 33, Finland
- 1980 Juhn, S.K., M.D., Univ of Minn. Medical School, 2001 6th St. SE, Minneapolis, MN 55455
- 1969 Kiang, Nelson Y.S., Ph.D., 18 Cedar Lane Way, Boston, MA 02108
- 1994 Kileny, Paul R., Ph.D., Department of Otolaryngology, 1500 E. Medical Cntr. Dr., Ann Arbor, MI 48109-0312
- 1978 Kimura, Robert S., Ph.D., 243 Charles Street, Boston, MA 02114
- 1959 Lawrence, Merle, Ph.D., 1535 Shorelands Dr. East, Vero Beach, FL 32963
- 1973 Lim, David J., M.D., 9000 Rockville Pike, Bldg. 31, Room 3C06, NIH, Bethesda, MD 20892
- 1986 Merzenich, Michael, Ph.D., University of California, Coleman Laboratory HSE 871, San Francisco, CA 94143
- 1979 Miller, Josef M., Ph.D., University of Michigan, Kresge Hearing Research Inst, 1301 East Ann Street, Ann Arbor, MI 48109
- 1985 Morizono, Tetsuo, M.D., Dept. of Otolaryngology, Fukuoka University Medical School, Nanakuma 7-45-1, Jonak-Kufukuoka, Japan 814-01
- 1978 Neff, William D., Ph.D., Center for Neural Sciences, Indiana University, Bloomington, IN 47401
- 1970 Rosenblith, Walter A., Ph.D., M.I.T., Rm 3-240, Cambridge, MA 02139
- 1986 Rubel, Edwin W., Ph.D., Dept of Otolaryngology, RL-30 University of Washington, Seattle, WA 98195
- 1989 Ryu, Jai H., Ph.D., Dept of Otolaryngology, Bowman Gray School of Medicine, Winston-Salem, NC 27157
- 1975 Sando, Isamu, M.D., 203 Lothrop Street, Pittsburgh, PA 15213
- 1992 Schacht, Jochen, Ph.D., Kresge Hearing Research Inst, University of Michigan, 1301 East Ann Street, Ann Arbor, MI 48109-0506
- 1950 Silverman, S. Richard, Ph.D., 2510 NW 38th Street, Gainesville, FL 32601
- 1962 Smith, Catherine A., Ph.D. (address unknown)
- 1992 Snyder, Jack McLean, Ph.D., Dept of Otolaryngology, RL-30 University of Washington, Seattle, WA 98195
- 1971 Thalmann, Ruediger, M.D., 517 South Euclid Avenue, St. Louis, MO 63110
- 1970 Valvassori, Galdino, M.D., 697 Sheridan Rd., Winnetka, IL 60093
- 1987 Van De Water, Thomas, M.D., Albert Einstein College of Med, Kennedy Center 302, 1410 Pelham Pky. S., Bronx, NY 10461-1101
- 1974 Vernon, Jack A., Ph.D., 3515 S.W. Sam Jackson Park Rd., Portland, OR 97201
- 1971 Ward, W. Dixon, Ph.D., 1666 Coffman St. Apt 315, St. Paul, MN 55108-1340
- 1984 Zwislocki, Jozef J., Sc.D., Institute of Sensory Research, Syracuse University, Syracuse, NY 13210
- Corresponding Members*
- 1995 Bagger-Sjoberg, Dan, M.D., Trastvagen 11, S-182 75 Stockholm, Sweden
- 1995 Booth, Mr. J. Barton, 18 Upper Wimpole Street, London W1M 7TB, England
- 1995 Causse, Jean-Bernard, M.D., Traverse de Beziers, 34440 Colombiers, France
- Honorary Members*
- 1993 Albernaz, Pedro, 4405 N.W. 73rd Ave., Suite 20-40003, Miami, FL 33166
- 1993 Babal, Aziz, Egypt (address unknown)
- 1993 Chiossone, Edgar, 25897 E 30, Apartado 62-277, Caracas, Venezuela 1060
- 1985 Fisch, Ugo, Forchstrasse 26, Erlenbach, Switzerland
- 1992 Goldstein, Jerome C., 1200 N. Nash St. Apt. 1138, Arlington, VA 22209
- 1968 Jongkees, L.B.W., Reijnier Vinkeleskade 71, 1071 S2 Amsterdam, ENT Dept. Wilhelmina Gasthuis, The Netherlands
- 1985 Morrison, Andrew, 38 Devonshire Street. London W1, England
- 1992 Nomura, Yasuya, Dept of Otolaryngology, Showa University 1-5-8, Hatanodai, Shinagawa-ku, Tokyo 142, Japan
- 1983 Portmann, Michel, 114 Ave de'Ares, 33000 Bordeaux, France 33074
- Deceased Members (1994-1995)*
- 1985 (1955) Bordley, John, 830 W. 40th Street, Baltimore, MD. 21205 (deceased 1993)
- 1952 Ruedi, Luzius, Zollikerstrasse 185, CH 8008, Zurich, Switzerland
- 1988 (1967) Schiff, Maurice, UCSD Campus Medical Group, 513 Wm Osler Lane (MO41), La Jolla, CA 92093
- 1975 (1953) Work, Walter P., 172 Paseo De Golf, Green Valley, AZ 85614
- 1991 (1978) Wright, J. William Jr., 7474 Holiday Drive E, Indianapolis, IN 46260-3612
- 1976 (1952) Simonton, Kinsey M., 1000 Vicars Landing Way F302, Ponte Vedra, FL 32082
- 1985 (1950) Goodhill, Victor, 715 North Walden Drive, Beverly Hills, CA 90210
- 1979 (1959) Dolowitz, David A., Box 524, Toquerville, UT 84774
- 1988 (1957) Farrior, J. Brown, 509 West Third Street, Tampa, FL 33606
- 1980 (1952) McQuiston, Ralph J., 20 North Meridian St., Indianapolis, IN 46204

# INDEX

## SUBJECT INDEX

- AIDS, cochlea immunohistochemical findings, 68
- Air-bone gap, stapes surgery, 32, 38
- Air conduction, stapes surgery, 32, 38
- Air conduction pure-tone average, stapes surgery, 32, 38
- American Otological Society (AOS)
- Award of Merit, ix, x
  - executive sessions, 72
  - guests of honor, x, 3-5
  - membership, 94-98
  - new members, 72, 89-93
  - new president, 71
  - obituaries, 72, 80-88
  - officers, viii
  - Presidential Citation, 6
  - professional career periodicity, 4-5
- Ataxic gait disturbance, gentamicin, 13
- Auditory innervation, neurotrophin, 66
- Autoimmune inner ear disease
- prednisone, 12
  - topical steroid, 12
- Autologous bone graft, 38
- long-term results, 35, 38
- Basilar membrane of apical turn
- endolymphatic hydrops, 19, 21
  - micromechanics, 20
- Benign positional vertigo, 22-30
- canalith hypothesis, 22-24
  - CO<sub>2</sub> laser posterior canal occlusion, 27-29
  - cupulolithiasis, 22
  - diagnosis, 22, 23-24, 30
  - etiology, 29
  - follow-up, 30
  - Hallpike test, 22
  - labyrinthitis, 29-30
  - laser partitioning, 26-27
  - nystagmus, 30
  - otoconia, 29
  - overview, 22
  - particle repositioning, 22-25
  - singular neurectomy, 25-26
  - spontaneous recovery, 29
  - treatment, 22-30
  - vestibular labyrinth, 25
- Bioglass middle ear prosthesis, long-term results, 34
- Bone conduction value, stapes surgery, 32, 38
- Brackmann, Derald E., 71
- Brain-derived neurotrophic factor, 66
- Brainstem dysfunction, 21
- Cholesteatoma, aeration route histologic evaluation, 56-57
- CO<sub>2</sub> laser
  - posterior semicircular canal occlusion, 59, 61
  - benign positional vertigo, 27-29
  - revision stapedectomy, 31, 38
- Cochlea
- AIDS immunohistochemical findings, 68
  - Cochlear Corporation "20 + 2" implant, 48
- Cochlear nerve action potential, middle fossa acoustic neuroma resection, 60, 61
- Cody, D. Thane R., ix, x
- Dexamethasone, Meniere's disease, 10-11
- Electromagnetic middle ear transducer
- evoked potential, 36-37
  - stapes displacement measurement, 36-37
- Endolymphatic hydrops, basilar membrane of apical turn, 19, 21
- Endolymphatic sac decompression, hearing loss, 18
- Endoscopic stapedectomy, 33, 38, 39
- Evoked potential, electromagnetic middle ear transducer, 36-37
- External ear pathology, transforming growth factor-alpha, 67, 70
- Facial paralysis, psychosocial impact, 49, 53
- Footplate secondary fixation, 33, 38
- Frequency, stapes surgery, 32, 38
- Gacek, Richard R., 3
- Gentamicin, 7, 8
- ataxic gait disturbance, 13
- Glomus jugulare tumor
- hearing conservation in surgery, 52, 53
  - radiotherapy, 53
- Hallpike test, benign positional vertigo, 22
- Hearing loss, 13-14
- endolymphatic sac decompression, 18
  - measurement, 13-14
  - posterior fossa vestibular neurectomy, 18
- Human immunodeficiency virus, neurotoxic manifestations, 68
- Idiopathic sudden sensorineural hearing loss, magnetic resonance imaging, 43
- Jugular foramen tumor, variable relationship to neurovascular structures, 50-51
- Labyrinthectomy, residual function, 20
- Labyrinthine fistula
- management, 58, 61
  - secondary to cholesteatoma, 58, 61
- Labyrinthitis, benign positional vertigo, 29-30
- Laser Doppler vibrometry, 40-41, 46
- Laser partitioning, benign positional vertigo, 26-27
- Leukotriene inhibitor
- salicylate ototoxicity, 69, 70
  - transient evoked otoacoustic emissions, 69, 70
- Magnetic resonance imaging, idiopathic sudden sensorineural hearing loss, 43
- Managed care, quality of care, 1-2
- Measles, polymerase chain reaction amplification, 65
- Meniere's disease, 13
- dexamethasone, 10-11
  - prednisone, 12
  - streptomycin, 11
  - topical steroid, 12
  - transtympanic gentamicin therapy, 7
  - dosing regimen, 8-9
  - monitoring protocol, 8-9
- Middle ear pathology, transforming growth factor-alpha, 67, 70
- Middle fossa acoustic neuroma resection, cochlear nerve action potential, 60, 61
- Neurotrophin, auditory innervation, 66
- Neurotrophin-3, 66
- Non-vertiginous disequilibrium, 13-14
- Nystagmus, benign positional vertigo, 30
- Opticokinetic stimulation, 20-21
- Ossicular graft, long-term results, 35, 38
- Otoconia, benign positional vertigo, 29
- Otosclerosis, 33, 38
- measles virus etiology, 65
  - polymerase chain reaction amplification, 65
- Outcome measure, stapes surgery, 32, 38
- Particle repositioning, benign positional vertigo, 22-25
- Photoacoustic transient, 63-64
- Place-pitch discrimination, monopolar vs. bipolar stimulation, 53
- Polymerase chain reaction amplification
- measles, 65
  - otosclerosis, 65
- Posterior fossa lesion, diagnosis, 54-55
- Posterior fossa vestibular neurectomy, hearing loss, 18
- Posterior semicircular canal, mechanical vs. CO<sub>2</sub> laser occlusion, 59, 61-62
- Prednisone
- autoimmune inner ear disease, 12
  - Meniere's disease, 12
  - sensorineural deafness, 12
- Professional career periodicity, 4-5
- Pulsed ultraviolet laser, 63-64
- Quality of care, managed care, 1-2
- Revision stapedectomy, CO<sub>2</sub> laser, 31, 38
- Rotatory vertigo, 13-14
- Salicylate ototoxicity, leukotriene inhibitor, 69, 70
- Sensorineural deafness
- prednisone, 12
  - topical steroid, 12
- Singular neurectomy, benign positional vertigo, 25-26
- Squamous cell carcinoma, temporal bone, 45

Stapedectomy. *See* Endoscopic stapedectomy; Revision stapedectomy  
 Stapes displacement measurement, electromagnetic middle ear transducer, 36-37  
 Stapes surgery  
     air-bone gap, 32, 38  
     air conduction, 32, 38  
     air conduction pure-tone average, 32, 38  
     bone conduction value, 32, 38  
     frequency, 32, 38  
     outcome measure, 32, 38  
 Streptomycin, Meniere's disease, 11  
 Temporal bone  
     basilar membrane of apical turn, 19, 21  
     malignancy invasion patterns, 45, 46  
     pulsed laser ablation, identification of photoacoustic transient, 63-64

temporoparietal fascial flap, 44  
 Temporal bone percutaneous pedestal growth effects, 42  
     osseointegration, 42  
 Temporoparietal fascial flap, temporal bone reconstruction, 44  
 Tinnitus, neurophysiological approach, 47  
 Topical steroids  
     autoimmune inner ear disease, 12  
     Meniere's disease, 12  
     sensorineural deafness, 12  
 Transforming growth factor-alpha  
     external ear pathology, 67, 70  
     middle ear pathology, 67, 70  
 Transient evoked otoacoustic emissions, leukotriene inhibitor, 69, 70  
 Transtympanic gentamicin therapy, Meniere's disease, 7  
     dosing regimen, 8-9  
     monitoring protocol, 8-9

Unilateral peripheral vestibular function, measurement after compensation, 15-16  
 Vertigo, 13-14  
 Vestibular compensation, 20, 21  
     measurement, 17, 20  
     minimal residual vestibular function, 17  
 Vestibular function, destructive procedures, 20, 21  
 Vestibular labyrinth, benign positional vertigo, 25  
 Vestibulo-ocular reflex  
     asymmetry index, 16, 20, 21  
     measurement, 15-16  
 Vestibulo-ocular system, postoperative brain stem dysfunction, 21  
 Yanagisawa, Eiji, 6

### AUTHOR INDEX

Adams, Joe C., 19  
 Antonelli, Patrick J., 34, 59  
 Arruda, Johnny, 69  
 Ashbaugh, Carissa, 48  
  
 Bartels, Loren J., 7  
 Berliner, Karen L., 32  
 Berns, Michael W., 63  
 Black, F. Owen, 17  
 Bloebaum, Roy, 42  
 Brackmann, Derald E., 60, 71  
 Brown, Mark T., 44  
 Burgio, Don L., 59  
  
 Cheney, Mack L., 44  
 Cody, D. Thane R., ix  
  
 Dickinson, Mark, 63  
 Doyle, Karen J., 32, 63  
  
 Farrior, Jay B., 38  
 Fredrickson, John M., 36  
  
 Gacek, Richard R., 4  
 Galiana, Henrietta, 15  
 Galinovic-Schwartz, Vera, 66  
 Gates, George A., 43  
 Ge, Xianxi, 10  
 Glasscock, Michael E., III, 52  
 Gold, Susan L., 47  
 Goldenberg, Robert A., 32  
 Goode, Richard L., 40  
 Graham, Malcolm D., 59  
 Gray, William C., 47  
  
 Haberkamp, Thomas, 31  
 Haines, Jonathan, 65  
 Hall, James W., III, 8  
 Haruta, Atsushi, 56  
 Harvey, Steven A., 31  
 Haynes, David S., 52  
 Herzog, Jacques A., 58  
 Hillman, Dean E., 68  
 Hitselberger, William E., 60  
 Hoffer, Michael E., 18  
  
 Izquierdo, Ricardo, 45

Jackler, Robert K., 50  
 Jackson, C. Gary, 52  
 Jahrsdoerfer, Robert A., 1, 3, 6, 71  
 Jastreboff, Pawel J., 47  
 Josey, Anne Forrest, 52  
 Jung, Timothy T. K., 69  
  
 Kartush, Jack M., 59  
 Katsarkas, Anthanasios, 15  
 Khafagy, Yasser, 31  
 Kileny, Paul R., 48  
 Kletzker, G. Robert, 45, 58  
 Kopke, Richard, 66  
 Kristiansen, Arthur, 65  
 Kumar, Arvind, 54  
  
 Lee, James, 68  
 Lefebvre, Philippe, 66  
 Leonetti, John P., 45  
 Lim, Jessica W., 68  
 Lim, Jin S., 68  
 Liu, Wei, 66  
 Lundy, Larry B., 59  
 Lustig, Lawrence R., 50  
  
 Malgrange, Brigitte, 66  
 Maxwell, Kenneth S., 58  
 McGann, David G., 69  
 McKenna, Michael J., 44, 65  
 Megerian, Cliff A., 44  
 Merchant, Saumil N., 19  
 Meyerhoff, William L., 67  
 Miller, Douglas A., 36  
 Moonen, Gustave, 66  
 Morimitsu, Tamotsu, 56  
  
 Nadol, Joseph B., 44  
 Nageris, Benny, 19  
 Nashner, Lewis M., 17  
 Neely, J. Gail, 48  
 Neev, Joseph, 63  
 Neufeld, Peggy S., 49  
 Nichols, Stacy W., 38  
  
 Ong, Bernard, 68  
  
 Paparella, Michael M., 56  
 Parkin, Brett D., 42

Parkin, James L., 42  
 Parkin, Matthew J., 42  
 Pieri, Albert, 54  
  
 Richards, Todd, 43  
 Roberson, Joseph, 60  
 Robey, Thomas C., 36  
 Robinson, Karen S., 67  
 Roland, J. Thomas, Jr., 68  
 Rosenberg, Seth I., 12, 18  
 Rubel, Edwin W., 43  
 Ruckenstein, Michael J., 10  
 Rust, Kevin R., 34  
  
 Salt, Alec N., 36  
 Saunders, James, 60  
 Schachern, Patricia A., 56  
 Schwaber, Mitchell K., 8  
 Senne, Allen, 60  
 Shea, John J., Jr., 10  
 Sillman, Jonathan S., 7  
 Silverstein, Herbert, 12, 18  
 Singleton, George T., 34  
 Smith, Heather L., 15  
 Smith, Mansfield F. W., ix  
 Smith, Peter G., 45, 58  
 Staecker, Hinrich, 66  
 Storper, S., 52  
  
 Tarabichi, Muaaz, 33  
 Telian, Steven A., 48  
 Tono, Tetsuya, 56  
 Tsuruda, Jay, 43  
  
 Van De Water, Thomas R., 66  
 Viana, Marlos A. G., 54  
  
 Wade, Steven W., 17  
 Walker, Paul A., 52  
 Wilson, June, 34  
 Wong, Brian J. F., 63  
 Wright, Charles G., 67  
 Wurm, Faith C., 8  
  
 Yanagisawa, Eiji, 6  
  
 Zwolan, Theresa A., 48