

PROGRAM of the

One Hundred Twenty-Eighth Annual Meeting

AMERICAN OTOLOGICAL SOCIETY, INC.

April 29-30, 1995

Marriott's Desert Springs Resort Palm Desert, California

OFFICERS JULY 1, 1994 - JUNE 30, 1995

PRESIDENT

Robert A. Jahrsdoerfer, M.D. 6431 Fannin Street, Suite 6.132 Houston, TX 77030

PRESIDENT-ELECT

Derald E. Brackmann, M.D. 2100 West Third Street Los Angeles, CA 90057

SECRETARY-TREASURER

Gregory J. Matz, M.D. Loyola University Medical Center 2160 S. First Avenue Bldg. 105-Room 1870 Maywood, IL 60153

EDITOR-LIBRARIAN

Joseph C. Farmer, Jr., M.D. Division of Otolaryngology - Box 3805 Duke University Medical Center Durham, NC 27710

COUNCIL

The above Officers and Mansfield F.W. Smith, M.D. Robert I. Kohut, M.D. A. Julianna Gulya, M.D. C. Gary Jackson, M.D.

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

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

SATURDAY, APRIL 29, 1995

REGISTRATION - 7:00 a.m.

BUSINESS MEETING - 7:00 a.m.

ROOM: SALON F (Restricted to Members)

Minutes of the Previous Annual Meeting

Introduction of New Members

Election of Nominating Committee

Report of the Secretary/Treasurer

Report of Editor/Librarian

SCIENTIFIC PROGRAM - 7:30 a.m.

ROOM: SALON F (Open to Non-Members)

Remarks by the President Robert A. Jahrsdoerfer, M.D.

Remarks by the Guest of Honor Richard R. Gacek. M.D.

Presidential Citation Eiji Yanagisawa, M.D.

- 8:00 a.m. Transtympanic Gentamicin Titration Therapy for Meniere's Disease Loren J. Bartels, M.D.
 Jonathan S. Sillman, M.D.* (by invitation)
- 8:10 a.m. Transtympanic Gentamicin Therapy: A New Dosing Regimen and Protocol for Monitoring Its Effects Mitchell K. Schwaber, M.D.*

Mitchell K. Schwaber, M.D.*
Faith C. Wurm, M.S. (by invitation)
James W. Hall III, Ph.D. (by invitation)

3. 8:20 a.m. Dexamethasone Perfusion of the Labyrinth Plus Intravenous Dexamethasone for Meniere's Disease

John J. Shea, M.D.*

Xianxi Ge, M.D. (by invitation)

^{*}speaker

4. 8:30 a.m. Intratympanic Steroid Therapy in the Treatment of Sensorineural Deafness in Meniere's Disease and Autoimmune Inner Ear Disease

Herbert Silverstein, M.D. *

Seth I. Rosenberg, M.D. (by invitation)

8:40 a.m. Discussion

5. 8:50 a.m. "Feeling Well" or "Not" After
Compensation of Unilateral Vestibular
Loss: Is There an Objective Measure?
Athanasios Katsarkas, M.D.*
Henrietta Galiana, Ph.D. (by invitation)
Heather Smith, M.Eng. (by invitation)

9:00 a.m. What is the Minimal Vestibular Function Required for Compensation?
 F. Owen Black, M. D.*
 Lewis M. Nashner, Sc.D. (by invitation)

7. 9:10 a.m. A Comparison of Hearing Results After Endolymphatic Sac Decompression and Posterior Fossa Vestibular Neurectomy Seth I. Rosenberg, M.D.* (by invitation) Herbert Silverstein, M.D. Michael E. Hoffer, M.D. (by invitation)

8. 9:20 a.m. A Human Temporal Bone Study of Changes in the Basilar Membrane of the Apical Turn in Endolymphatic Hydrops Benny Nageris, M.D. (by invitation)
Saumil N. Merchant, M.D.* (by invitation)
Joe C. Adams, Ph.D. (by invitation)

9:30 a.m. Discussion

9:40 a.m. Intermission

PANEL: "Benign Positional Vertigo - New 10:00 a.m. Treatments" Moderator - lack M. Kartush, M.D. Panelists: Philip F. Anthony, M.D. (by invitation) Brian W. Blakley, M.D., Ph.D. (by invitation) John M. Epley, M.D. (by invitation) Richard R. Gacek, M.D. John F. Kveton, M.D. (by invitation) 10:50 a.m. Discussion 9. 11:00 a.m. Revision Stapedectomy With and Without the CO2 Laser: A Comparison of Results Thomas J. Haberkamp, M.D.* (by invitation) Steven A. Harvey, M.D. (by invitation) Yasser Khafagy, M.D. (by invitation) Reporting Operative Hearing Results in 10. 11:10 a.m. Stapes Surgery: Does Choice of Outcome Measure Make a Difference? Karen I. Berliner, Ph.D.* (by invitation) Robert A. Goldenberg, M.D. Karen J. Doyle, Ph.D., M.D. (by invitation) 11. 11:20 a.m. Endoscopic Stapedectomy: A Preliminary Report Muaaz Tarabichi, M.D. * (by invitation) 12. 11:30 a.m. Bioglass Middle Ear Prosthesis: Long Term Results Kevin R. Rust, M.D.* (by invitation) George T. Singleton, M.D. June Wilson, Ph.D. (by invitation) 13. 11:40 a.m. Long Term Hearing Results From Primary

Ossicular Reconstruction with Autologous

Bone

Jay B. Farrior, M.D.*

*speaker

14. 11:50 a.m. 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, B.S.E.* (by invitation)

Douglas A. Miller, B.S.E.E. (by invitation)

Alec N Salt, Ph.D. (by invitation)
John M. Fredrickson, M.D.

12:00 noon Discussion

12:10 p.m. PHOTOGRAPH

(All members remain for group photo.)

6:30 p.m. PRESIDENT'S RECEPTION -

South & West Foyer

7:30 p.m. PRESIDENT'S DINNER -

The Springs Ballroom For Members,

Officially Invited Guests, their Ladies or Escorts

-Black Tie-

^{*}speaker

SUNDAY, APRIL 30, 1995

REGISTRATION - 7:00 a.m.

BUSINESS MEETING - 12:30 p.m.

Room: SALON F (Restricted to Members)

Report of the:

- a. Board of Trustees of Research Fund
- b. American Board of Otolaryngology
- c. Award of Merit Committee
- d. American College of Surgeons
- e. American Academy of Otolaryngology-Head and Neck Surgery

Report of the Audit Committee

Report of the Nominating Committee

Reading of Communications

Unfinished Business

New Business

SCIENTIFIC PROGRAM - 1:00 p.m.

ROOM: SALON F (Open to Non-Members)

15. 1:00 pm. Laser Doppler Vibrometry (LDV) — A New

Clinical Tool for the Otologist Richard Goode. M.D.*

16. 1:10 p.m. Osseointegration and Growth Effects of

Temporal Bone Percutaneous Pedestals

James L. Parkin, M.D.*

Roy D. Bloebaum, Ph.D. (by invitation) Brett D. Parkin, B.S. (by invitation)

17. 1:20 p.m. Magnetic Resonance Imaging in Idiopathic

Sudden Sensorineural Hearing Loss

George A. Gates, M.D. *

Todd Richards, M.D. (by invitation)
Jay Tsuruda, M.D. (by invitation)
Edwin W. Rubel, Ph.D. (by invitation)

^{*}speaker

18. 1:30 p.m.	The Use of Temporoparietal Fascial Flap in Temporal Bone Reconstruction Mack L. Cheney, M.D. (by invitation) Cliff A. Megerian, M.D.* (by invitation) Mark T. Brown, M.D. (by invitation) Michael J. McKenna, M.D. (by invitation) Joseph B. Nadol, Jr. M.D.
19. 1:40 p.m.	Invasion Patterns of Advanced Temporal Bone Malignancies John P. Leonetti, M.D. * (by invitation) Peter G. Smith, M.D., Ph.D. G. Robert Kletzker, M.D. (by invitation) Ricardo Izquierdo, M.D. (by invitation)
1:50 p.m.	Discussion
20. 2:00 p.m.	Neurophysiological Approach to Tinnitus Patients Pawel J. Jastreboff, Ph.D., Sc.D.* (by invitation) William C. Gray, M.D. (by invitation) Susan L. Gold, M.A. (by invitation)
21. 2:10 p.m.	Patient Performance With the Cochlear Corporation 20+2 Implant: Bipolar versus Monopolar Activation Terry A. Zwolan, Ph.D. (by invitation) Paul R. Kileny, Ph.D.* Carissa A. Moeggenberg, M.A. (by invitation)

Steven A. Telian, M.D. (by invitation)

Defining Functional Limitation, Disability, and Societal Limitations in Patients With Facial Paresis: Initial Pilot Questionnaire J. Gail Neely, M.D.*

Peggy S. Neufeld, M.A. (by invitation)

23. 2:30 p.m.	The Variable Relationship Between the Lower Cranial Nerves and Jugular Foramen Tumors: Implications for Neural Preservation Lawrence R. Lustig, M.D.* (by invitation) Robert K. Jackler, M.D.
24. 2:40 p.m.	Hearing Conservation in Surgery for Glomus Jugulare Tumors C. Gary Jackson, M.D.* David S. Haynes, M.D. (by invitation) Michael E. Glasscock, III, M.D. Anne F. Josey-Tallent, M.S. (by invitation)
2:50 p.m.	Discussion
3:00 p.m.	Intermission
25. 3:20 p.m.	The Diagnosis of Intra-axial Posterior Fossa Lesions Arvind Kumar, M.D.* Marlos A.G. Viana, Ph.D. (by invitation) Albert Pieri, B.S. (by invitation)
26. 3:30 p.m.	Histologic Evaluation of Aeration Routes in Temporal Bones with Cholesteatoma Atsushi Haruta, M.D.* (by invitation) Patricia A. Schachern, B.S. (by invitation) Tetsuya Tono, M.D. (by invitation) Michael M. Paparella, M.D. Tamotsu Morimitsu, M.D. (by invitation)
27. 3:40 p.m.	Management of Labyrinthine Fistulae Secondary to Cholesteatoma

Jacques A. Herzog, M.D.* (by invitation)
G. Robert Kletzker, M.D. (by invitation)

Kenneth S. Maxwell, M.D. (by invitation)

Peter G. Smith, M.D., Ph.D.

^{*}speaker

Mechanical versus CO2 Laser Occlusion 28. 3:50 p.m. of the Posterior Semicircular Canal in Humans Patrick J. Antonelli, M.D.* (by invitation) lack M. Kartush, M.D. Larry D. Lundy, M.D. (by invitation) Don L. Burgio, M.D. (by invitation) Direct Cochlear Nerve Action Potentials as 29. 4:00 p.m. an Aid to Hearing Preservation in Middle Fossa Acoustic Neuroma Resection Ioseph P. Roberson, Jr., M.D.* (by invitation) Allen Senne, M.A. (by invitation) Derald E. Brackmann, M.D. William E. Hitselberger, M.D. (by invitation) Discussion 4:10 p.m. Identification of Photoacoustic Transients 30. 4:20 p.m. During Pulsed Laser Ablation of the Human Temporal Bone Brian J.F. Wong, M.D.* (by invitation) Mark R. Dickinson, Ph.D. (by invitation) Joseph Neev, Ph.D. (by invitation) Karen J. Doyle, M.D., Ph.D. (by invitation) Michael W. Berns, Ph.D. (by invitation) 31. 4:30 p.m. Polymerase Chain Reaction Amplification

Polymerase Chain Reaction Amplification of a Measles Virus Sequence from Human Temporal Bone Sections with Active Otosclerosis

Michael J. McKenna, M.D.*

(by invitation)

Arthur Kristiansen, M.S. (by invitation) Jonathan Haines, Ph.D. (by invitation)

^{*}speaker

32. 4:40 p.m. Effects of Neurotrophic Factors on the Survival and Regeneration of Auditory Neurons Hinrich Staecker, M.D.* (by invitation) Richard Kopke, M.D. (by invitation) Philippe Lefebvre, M.D., Ph.D. (by invitation) Bridgitte Malgrange, Ph.D. (by invitation) Wei Liu, B.S. (by invitation) Ioseph Feghali, M.D. (by invitation) Gustave Moonen, M.D., Ph.D. (by invitation) Thomas Van De Water, Ph.D. Robert Ruben, M.D. Pathological Changes of the External and 33. 4:50 p.m. Middle Ear in Animals with TGF-Alpha Deficiency Charles G. Wright, Ph.D. (by invitation) Karen S. Robinson, B.S. (by invitation) Sarah A. Comerford, Ph.D. (by invitation) William L. Meyerhoff, M.D., Ph.D.* Immunohistochemical Findings in the 34. 5:00 p.m. Cochlea of AIDS Cases Jessica W. Lim, M.D.* (by invitation) J. Thomas Roland, Jr., M.D. (by invitation) Jin S. Lim, M.D. (by invitation) James Lee, B.A. (by invitation) Bernard Ong, B.A. (by invitation) Dean E. Hillman, Ph.D. (by invitation) Effect of Leukotriene Inhibitor on 35. 5:10 p.m. Otoacoustic Emissions in Salicylate Ototoxicity Timothy T.K. Jung, M.D., Ph.D. Johnny Arruda, M.D.* (by invitation) Discussion 5:20 p.m. INTRODUCTION OF NEW PRESIDENT 5:30 p.m. Derald E. Brackmann, M.D.

ADJOURNMENT

*speaker

NAMES AND ADDRESS OF PRIMARY AUTHORS

Patrick I. Antonelli, M.D.

Department of Otolaryngology University of Florida Box 100264 Gainesville, FL 32610

Loren I. Bartels, M.D.

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

Karen I. Berliner, Ph.D.

House Ear Institute 2100 West Third Street, Fifth Floor Los Angeles, CA 90057

F. Owen Black, M.D., F.A.C.S.

Neurotology Research N010 1040 N.W. 22nd Avenue Portland, OR 97210

Derald E. Brackmann, M.D.

House Ear Clinic 2100 West Third Street Los Angeles, CA 90057

Mack L. Cheney, M.D.

Massachusetts Eye and Ear Infirmary Department of Otolaryngology 243 Charles Street Boston, MA 02114

Jay B. Farrior, M.D.

509 West Bay Street Tampa, Florida 33606

George A. Gates, M.D.

Virginia Merrill Bloedel Hearing Research Center University of Washington XF-01 1325 4th Avenue, Suite 2000 Seattle, WA 98101

Richard L. Goode, M.D. 300 Pasteur Dr., R-135

Stanford University Medical Center Stanford, CA 94305-5328

Thomas J. Haberkamp, M.D. 9200 W. Wisconsin Avenue

Milwaukee, WI 53226

Atsushi Haruta, M.D.

Room 226 Lions Research Building Dept. of Otolaryngology 2001 Sixth Street S.E. Minneapolis, MN 55455

Jacques A. Herzog, M.D.

The Center for Hearing & Balance Disorders 11155 Dunn Road, Suite 209 East St. Louis. MO 63136

C. Gary Jackson, M.D.

The Otology Group 300 20th Avenue North, Suite 502 Nashville, TN 37203

Pawel J. Jastreboff, Ph.D., Sc.D.

University of Maryland at Baltimore M.S.T.F. Building, Room 434F 10 South Pine Street Baltimore, Maryland 21201-1192

Timothy T.K. Jung, M.D., Ph.D.

Loma Linda University 11790 Pecan Way Loma Linda, CA 92354

A. Katsarkas, M.D.

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

Arvind Kumar, M.D.

Room B.42 1855 W. Taylor, M/C 648 Chicago, IL 60612

John P. Leonetti, M.D.

Department of Otolaryngology-HNS Loyola University Medical Center 2160 S. First Avenue, Bldg. 105, Rm. 1870 Maywood, IL 60153

Jessica W. Lim, M.D.

New York University Medical Center 550 First Avenue New York, NY 10016 Lawrence Lustig, M.D. U California-San Francisco 400 Parnassus Avenue - A717 San Francisco, CA 94143-0342

Michael J. McKenna, M.D. Massachusetts Eye & Ear Infirmary 243 Charles Street Boston, MA 02114

Saumil N. Merchant, M.D.
Massachusetts Eye and Ear Infirmary
Department of Otolaryngology
243 Charles Street
Boston. MA 02114

J. Gail Neely, M.D.
Department of Otolaryngology-HNS
Washington University School of
Medicine
517 S. Euclid Ave, Box 8115
St. Louis, Mo 63110

James L. Parkin, M.D. University of Utah School of Medicine 50 North Medical Drive Salt Lake City, Utah 84132

Thomas C. Robey, B.S. 3261 Sulphur Avenue, #1 St. Louis, MO 63139

Seth Rosenberg, M.D. 1901 Floyd Street Sarasota, FL 34239

Kevin R. Rust, M.D. Dept. of Otolaryngology University of Florida Box 100264 Gainesville, Florida 32610 Mitchell K. Schwaber, M.D. Vanderbilt University Medical Center S-2100 Medical Center North Nashville, TN 37232-2559

Herbert Silverstein, M.D. 1901 Floyd Street Sarasota, FL 34239

John J. Shea, Jr., M.D. Shea Clinic 6133 Poplar Pike Memphis, Tennessee 38119

Hinrich Staecker, M.D. Albert Einstein College of Medicine Kennedy Center 302 1410 Pelham Pkwy South Bronx, New York 10461

Muaaz Tarabichi, M.D. 3535 30th Avenue, Suite #204 Kenosha, WI 53144

Brian J.F. Wong, M.D. Beckman Laser Institute 1002 Health Sciences Rd., East Irvine, CA 92715

Charles G. Wright, Ph.D. Dept. of Otolaryngology UT Southwestern Medical Center 5323 Harry Hines Blvd. Dallas, TX 75235

Terry Zwolan, Ph.D.
University of Michigan
Department of Otolaryngology
1500 E. Medical Center Driv
Ann Arbor, MI 48109-0312

AWARD OF MERIT RECIPIENTS

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

GUESTS OF HONOR (1974-1994)

- 1974 Harry Rosenwasser, M.D.
- 1975 John E. Bordley, M.D.
- 1976 Ben H. Senturia, M.D.
- 1977 Henry B. Perlman, M.D.
- 1978 Howard P. House, M.D.
- 1979 Hallowell Davis, M.D.
- 1980 Victor Goodhill, M.D.
- 1981 Harold Schuknecht, M.D.
- 1982 George E. Shambaugh, Jr., M.D.
- 1983 Wesley H. Bradley, M.D.
- 1984 Brown Farrior, M.D.
- 1985 Bruce Proctor, M.D.
- 1986 Merle Lawrence, Ph.D.
- 1987 Robert M. Sevfarth, Ph.D.
- 1988 G. Dekle Taylor, M.D.
- 1989 Eugene L. Derlacki, M.D.
- 1990 William F. House, M.D.
- 1991 Michael E. Glasscock III, M.D.
- 1992 William E. Hitselberger, M.D.1993 D. Thane R. Cody, M.D.
- 1994 Cesar Fernandez, M.D.

21

** ACTIVE	
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	
1970 Alford, Bobby R	6501 Fannin Street Houston, TX 77030
1987 Althaus, Sean R	5201 Norris Canyon Rd. #230 San Ramon, CA 94583-5405
1985 Applebaum, Edward	
1980 Austin, David F	2860 Channing Way, Suite 202 Idaho Falls, ID 83404
1993 Babin, Richard W River Bend	l Head & Neck Assoc. 6570 Stage Road, Suite 245 Bartlett, TN 38134
1991 Balkany, Thomas J L	Iniv. of Miami School of Medicine Dept of Otolaryngology PO Box 016960 Miami, FL 33101
1992 Bartels, Loren J	
1983 Black, F. Owen	2525 N.W. Lovejoy, Suite 406 Portland, OR 97210

1977 Bluestone, Charles D.125 DeSoto Street

Pittsburgh, PA 15213

San Francisco, CA 94143

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	
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	
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
1972 Crabtree, James A	1332 Westhaven Road San Marino, CA 91108
1975 Dayal, Vijay S	
1991 De la Cruz, Antonio	

1991 Dickins, John R.E
1985 Dobie, Robert A Dept of Otolaryngology, UTSA 7703 Floyd Curl Drive San Antonio, TX 78284
1987 Doyle, Patrick J
1988 Duckert, Larry G Department of Otolaryngology P. O. Box 351928 RL-30, University of Washington Seattle, WA 98195
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, III, Jay B 509 Bay Street Tampa, FL 33606
1978 Fredrickson, John M517 South Euclid - Box 8115 St. Louis, MO 63110
1969 Gacek, Richard R
1987 Gantz, Bruce J
1983 Gardner, Jr., L. Gale899 Madison Avenue, Suite 602A Memphis, TN 38103

1987 Gates, George AUniversity of Washington Department of Otolaryngology 1959 NE Pacific St. RL-30 Seattle, WA 98195
1973 Glasscock,III, Michael E 300 20th Avenue, North Suite 502 Nashville, TN 37203
1989 Goldenberg, Robert A111 West First St, Suite 1000 Dayton, OH 45402
1990 Goode, Richard L
1992 Goycoolea, Marcos V San Crescente 70 Las Condes, Santiago, Chile
1979 Graham, Malcolm D Georgia Ear Institute Provident Professional Bldg. 4750 Waters Avenue Savannah, GA 31404
1991 Gulya, Julianna
1987 Harker, Lee A Boystown National Research Hospital 555 North 30th Street Omaha, NE 68131
1987 Harner, Stephen GMayo Clinic 200 First Street SW Rochester, MN 55905
1988 Harris, Jeffery PUniv. of California Medical Ctr 225 Dickinson St H-895 San Diego, CA 92102
1992 Hart, Cecil W. J
1984 Hawke, W. Michael
1992 Hoffman, Ronald A 1430 Second Avenue New York, NY 10021
1984 House, John W

1964 House, William F
1987 Hughes, Gordon B Dept of Otolaryngology Cleveland Clinic 9500 Euclid Avenue Cleveland, OH 44195
1992 Jackler, Robert K
1994 Jackson, Carol A
1990 Jackson, C. Gary
1992 Jahn, Anthony 556 Eagle Rock Avenue Roseland, NJ 07068
1982 Jahrsdoerfer, Robert A 6431 Fannin Street Ste.6.132 Houston, TX 77030
· ·
1987 Jenkins, Herman A Dept of Otolaryngology Baylor College of Medicine Houston, TX 77030
Baylor College of Medicine
Baylor College of Medicine Houston, TX 77030 1990 Jung, Timothy K ENT Division, Loma Linda 11790 Pecan Way
Baylor College of Medicine Houston, TX 77030 1990 Jung, Timothy K
Baylor College of Medicine Houston, TX 77030 1990 Jung, Timothy K
Baylor College of Medicine Houston, TX 77030 1990 Jung, Timothy K

1976 Kohut, Robert I Bowman Gray School of Medicine Dept of Otolaryngology Medical Center Boulevard
Winston-Salem, NC 27157-1034 1991 Konrad, Horst
1993 Kumar, Arvind 1855 W. Taylor St., M/C 648 Chicago, IL 60612
1993 Lesinski, S. George
1987 Lindeman, Roger C 1100 Ninth Avenue - #900 Seattle, WA 98101
1988 Lippy, William H
1969 Litton, Ward B
1991 Luetje, Charles M Otologic Center, Inc Penntower Office Center 3100 Broadway, Suite 509 Kansas City, MO 64111
1970 Maddox, H. Edward
1987 Mangham, Jr., Charles A Seattle Ear Clinic 600 Broadway, Suite 340 Seattle, WA 98122
1989 Maniglia, Anthony J University Hospitals of Cleveland 2074 Abington Road Cleveland, OH 44106
1985 Mathog, Robert H540 East Canfield Avenue Detroit, MI 48201
1992 Mattox, Douglas E

1979 Matz, Gregory J Dept of Otolaryngology-HNS 2160 South First Avenue Maywood, IL 60153
1965 McCabe, Brian F University of Iowa Dept of Otolaryngology Iowa City, Iowa 52242
1987 McDonald, Thomas J.P
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
1975 Montgomery, William
1988 Nadol, Jr., Joseph B243 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. GailWashington University School of Med. 517 South Euclid Avenue, Box 8115 St. Louis, MO 63110
1993 Olsson, James E Texas Neurosciences Institute 4410 Medical Drive, Suite 550 San Antonio, TX 78229
1968 Paparella, Michael M701 25th Avenue South - Ste.200 Minneapolis, MN 55454
1985 Pappas, Dennis
1983 Pappas, James J
1982 Parisier, Simon C

Salt Lak	chool of Medicine se City, UT 84132
231 Bethe Cincinnati,	illege of Medicine esda Ave, ML 528 OH 45267-0528
	mack Bldg, 229H of North Carolina el Hill, NC 27514
	more, MD 21209
	ngeles, CA 90017
	oush, VA Med Ctr West 10th Street napolis, IN 46202
1992 Roland, Peter S 5323 Dalla:	Harry Hines Blvd. s, TX 75235-9035
1972 Ronis, Max L 3400 N Philad	orth Broad Street elphia, PA 19140
	e Medical Center 0th Street VCA-4 , NY 10467-2490
•	ingfield, IL 62794
333 Ceda New I	of Otolaryngology our Street, Box 333 Mayen, CT 06510
1990 Sataloff, Robert TPhilad	1721 Pine Street elphia, PA 19103
	mbus, OH 43210
1983 Schindler, Robert A A717, 400 P San Fran	arnassus Avenue acisco, CA 94143
1990 Schuring, Arnold G 3893 E	ast Market Street arren, OH 44484

1993 Schwaber, Mitchell
1967 Shea, Jr., John J Box 17987 6133 Poplar Pike Memphis, TN 38119
1973 Silverstein, Herbert
1973 Simmons, F. Blair 300 Pasteur Drive, Room R-135 Palo Alto, CA 94025
1972 Singleton, George T University of Florida JHMHC, Box J-264 Gainesville, FL 32610
1993 Sismanis, Aristides
1973 Smith, Mansfield F.W2120 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
1975 Wehrs, Roger E
1990 Weider, Dudley J
1987 Wiet, Richard J
1992 Wilson, David F

1993-94 MEMBERSHIP LIST OF THE AMERICAN OTOLOGICAL SOCIETY

**	SE	Ν	O	R
----	----	---	---	---

1988 (1960) Armstrong, Beverly W	Charlotte, NC 28203
1994 (1969) Bailey, Jr., H.A.Ted	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	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, III, Claude C	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	
1989 (1968) Compere, Wesley E	3755 Avocado Blvd #503 LeMesa, CA 91941
1981 (1961) Daly, John F	1500 Palisase Avenue #27C Fort Lee, NJ 07024-5318
1989 (1958) Derlacki, Eugene L	
1979 (1959) Dolowitz, David A	
1994 (1974) Donaldson, James A	

1971 (1939) Druss, Joseph G 145 East 92nd Street New York, NY 10028
1993 (1971) Duvall, III, Arndt J Dept of Otolaryngology University of Minnesota, Box 478 Minneapolis, MN 55455
1988 (1957) Farrior, J. Brown 509 West Bay Street Tampa, FL 33606
1973 (1953) Glorig, Aram
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, Jerome409-1700 Lexington Avenue St. Paul, MN 55118
1990 (1970) Hohmann, Albert
1990 (1960) Hough, Jack V
1975 (1947) House, Howard P.2100 West Third Street Los Angeles, CA 90057
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
1981 (1954) Kos, Clair M.6801 W Poly Webb Road, #124 Arlington, TX 76016
1991 (1967) Linthicum, Jr., Fred H 2122 West Third Street Los Angeles, CA 90057
1987 (1975) Marcus, Richard E.6
1980 (1952) McQuiston, Ralph J20 North Meridian Street Indianapolis, IN 46204

1993-94 MEMBERSHIP LIST OF THE AMERICAN OTOLOGICAL SOCIETY

1990 (1974) Michelson, Robin P A717, 400 Parnassus Avenue San Francisco, CA 94143
1989 (1965) Moon, Jr., Cary N 1135 Inglecress Drive Charlottesville, VA 22901
1987 (1952) Moore, James A 525 East 68th Street New York, NY 10021
1978 (1957) Myers, David
1994 (1974) Myers, Eugene
1994 (1988) Nager, George T
1993 (1968) Naunton, Ralph FDCSD-NIDCD EPS-400B 6120 Executive Boulevard Rockville, MD 20892
1993 (1973) Pennington, Claude LPO Box 1916 Macon, GA 31202
1992 (1975) Powers, W. Hugh728 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
1993 (1967) Ruggles, Richard L11201 Shaker Boulevard Cleveland, OH 44104
1991 (1969) Robinson, Mendell
1992 (1967) Rubin, Wallace
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, Jr., George 40 South Clay St Hinsdale, IL 60521
1994 (1965) Sheehy, James L 2100 West Third Street Los Angeles, CA 90057
1980 (1958) Smith, J. Brydon
1993 (1973) Snow, Jr., James B National Institute on Deafness and Communicative Disorders 9000 Rockville Pike, 313C02 Bethesda, MD 20892
1990 (1967) Stroud, Malcolm H 517 South Euclid Avenue St. Louis, MO 63110
1971 (1947) Stuart, Edwin A.
1990 (1961) Tabb, Harold G1430 Tulane Avenue New Orleans, LA 70112
1985 (1965) Taylor, G. Dekle 13500 Mandarin Road Jacksonville, FL 32223
1984 (1974) Torok, Nicholas
1972 (1946) Truex, Edward H
1981 (1962) Waltner, Jules G
1994 (1972) Ward, Paul H
1989 (1972) Wilson, William H 1133 Oneida Street Denver, CO 80220
1986 (1964) Withers, Ben T
1994 (1971) Wolfson, Robert J
1987 (1964) Wright, William K 3671 Delmonte Houston, TX 77019

1993-94 MEMBERSHIP LIST OF THE AMERICAN OTOLOGICAL SOCIETY

** EMERITUS

1992 (1977) Bergstrom, Lavonne	
	Manhattan Beach, CA 90266
1979 (1963) Boyd, Harold M.E	313 Via Anita Redondo Beach, CA 90277-6621
1994 (1987) 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

1993-94 MEMBERSHIP LIST OF THE AMERICAN OTOLOGICAL SOCIETY

** ASSOCIATE

1992 Altschuler, Richard A. PhDKresge Hearing Research Inst. University of Michigan 1301 N. Ann Street Ann Arbor, MI 48109-0506
1979 Bohne, Barbara A. PhD 517 South Euclid Avenue St. Louis, MO 63110
1978 Butler, Robert A. PhD
1973 Fernandez, Cesar MD
1959 Graybiel, Ashton MDPO Box 4063 Warrington, FL 32507
1977 Gussen, Ruth MD.
1992 Hamid, Mohamed A. PhD
1992 Hannley, Maureen T. PhD
1972 Hawkins, Jr, Joseph E. PhD Kresge Hearing Research Inst. Ann Arbor, MI 48109
1989 Hinojosa, Raul MD 5316 Hyde Park Boulevard Chicago, IL 60615
1972 Honrubia, Vincente MD850 North Beverly Glen Blvd Los Angeles, CA 90024
1973 Igarashi, Makota MD Moto Azabu 3-12-18 Minato-Ku Tokyo, Japan 106
1994 Iurato, Salvatore J. MD Cattedra Di Bioacustica dell-Universita di Bar Policlinico, 70124 Bari, Italy
1960 Johnson, Walter H. PhD.
1979 Johnsson, Lars-Goran MD Simmarstigen 10A2 Helsinki 33, Finland

1993-94 MEMBERSHIP LIST OF THE AMERICAN OTOLOGICAL SOCIETY

1980 Juhn, S.K. MD
1969 Kiang, Nelson Y.S. PhD 18 Cedar Lane Way Boston, MA 02108
1994 Kileny, Paul R., PhD
1978 Kimura, Robert S. PhD243 Charles Street Boston, MA 02114
1959 Lawrence, Merle PhD 1535 Shorelands Dr. East Vero Beach, FL 32963
1973 Lim, David J. MD9000 Rockville Pike Bldg. 31, Room 3C06, NIH Bethesda, MD 20892
1986 Merzenich, Michael PhD
1979 Miller, Josef M. PhD
1985 Morizono, Tetsuo MD Dept. of Otolaryngology Fukuoka University Medical School Nanakuma 7-45-1 Jonak-Kufukuoka, JAPAN 814-01
1978 Neff, William D. PhD Center for Neural Sciences Indiana University Bloomington, IN 47401
1970 Rosenblith, Walter A. PhD
1986 Rubel, Edwin W. PhD Dept of Otolaryngology RL-30 University of Washington Seattle, WA 98195
1989 Ryu, Jai H. PhD Dept of Otolaryngology Bowman Gray School of Medicine Winston-Salem, NC 27157

1975 Sando, Isamu MD
1992 Schacht, Jochen PhD Kresge Hearing Research Inst University of Michigan 1301 East Ann Street Ann Arbor, MI 48109-0506
1950 Silverman, S. Richard PhD 2510 NW 38th Street Gainesville, FL 32601
1962 Smith, Catherine A. PhD
1992 Snyder, Jack McLean PhD Dept of Otolaryngology RL-30 University of Washington Seattle, WA 98195
1971 Thalmann, Ruediger MD 517 South Euclid Avenue St. Louis, MO 63110
1970 Valvassori, Galdino MD
1987 Van De Water, Thomas MDAlbert Einstein College of Med Kennedy Center 302 1410 Pelham Pky. S. Bronx, NY 10461-1101
1974 Vernon, Jack A. PhD3515 S.W. Sam Jackson Park Rd. Portland, OR 97201
1971 Ward, W. Dixon PhD246 Maple Hill Road Hopkins, MN 55343
1984 Zwislocki, Jozef J. ScD Institute of Sensory Research Syracuse University Syracuse, NY 13210

1993-94 MEMBERSHIP LIST OF THE AMERICAN OTOLOGICAL SOCIETY INC.

**	Н	O	N	O	R	A	R'	Υ
----	---	---	---	---	---	---	----	---

7.0.10.10.11
1993 Albernaz, Pedro
1993 Babal, Aziz Egypt
1993 Chiossone, Edgar
1985 Fisch, Ugo Forchstrasse 26 Erlenbach, Switzerland
1992 Goldstein, Jerome C
1968 Jongkees, L.B.W
1985 Morrison, Andrew
1992 Nomura, Yasuya Dept of Otolaryngology Showa University 1-5-8 Hatanodai, Shinagawa-ku Tokyo 142, Japan
1983 Portmann, Michel

DECEASED SINCE 1994 MEETING

Dr. John Bordley Active Membership 1955 Senior Membership 1985 Died June 11, 1993	Dr. Kinsey M. Simonton Active Membership 1952 Senior Membership 1976 Died December 14, 1994
	Died December 11, 1994
Senior Membership 1985	Active Membership 1952 Senior Membership 1976

Dr. Victor Goodhill
Active Membership 1950
Senior Membership 1985
Died December 26, 1994

Dr. Walter P. Work
Active Membership 1953
Senior Membership 1975
Died November 4, 1994

Dr. J. William Wright, Jr. Active Membership 1978 Senior Membership 1991 Died February 14, 1994

PAST PRESIDENTS

	E 14 EM	1010	BLACKE MB
1868-69	E. Williams, M.D.	1948	B.J. McMahon, M.D.
1870-73	H.D. Noyes, M.D.	1949 1950	Marvin F. Jones, M.D. Philip E. Meltzer, M.D.
1874-76	D.B.St.John Roosa, M.D.	1950	
1877-78	C.J. Blake, M.D.		Kenneth M. Day, M.D.
1879-80	A.H. Buck, M.D.	1952	Gordon D. Hoople, M.D.
1881-83	J.O. Green, M.D.	1953	A.C. Furstenberg, M.D.
1884-85	C.H. Burnett, M.D.	1954	Frederick T. Hill, M.D.
1886-89	J.S. Prout, M.D.	1955	D.E.S. Wishart, M.D.
1890	O.D. Pomeroy, M.D.	1956	William J. McNally, M.D.
1891-94	Gorham Bacon, M.D.	1957	John R. Lindsay, M.D.
1895-99	Arthur Mathewson, M.D.	1958	Dean M. Lierle, M.D.
1900-02	H.G. Miller, M.D.	1959	Moses H. Lurie, M.D.
1903-05	B. Alex Randali, M.D.	1960	Robert C. Martin, M.D.
1906-07	Emil Gruening, M.D.	1961	Henry L. Williams, M.D.
1908	C.J. Kipp, M.D.	1962	Lawrence R. Boies, M.D.
190 9- 10	Frederick L. Jack, M.D.	1963	Joseph A. Sullivan, M.D.
1911-12	Edward B. Dench, M.D.	1964	Theodore E. Walsh, M.D.
1913-14	J.F.McKernon, M.D.	1965	Harry Rosenwasser, M.D.
1915-16	C.W. Richardson, M.D.	1966	Howard P. House, M.D.
1917	C.R. Holmes, M.D.	1967	James A. Moore, M.D.
1918	Norval H. Pierce, M.D.	1968	G.E. Shambaugh,Jr., M.D.
1919	Ewing W. Day, M.D.	1969	Frank D. Lathrop, M.D.
1920	Robert Lewis, M.D.	1970	Francis L. Lederer, M.D.
1921	W.P. Eagleton, M.D.	1971	John E. Bordey, M.D.
1922	H.S. Birkett, M.D.	1972	Walter P. Work, M.D.
1923	G. Shambaugh, Sr., M.D.	1973	Ben H. Senturia, M.D.
1924	John B. Rae, M.D.	1974	Wesley H. Bradley, M.D.
1925	E.A. Crockett, M.D.	1975	Lester A. Brown, M.D.
1926	Thomas J. Harris, M.D.	1976	Victor Goodhill, M.D.
1927	Arthur B. Duel, M.D.	1977	Harold Schuknecht, M.D.
1928	M.A. Goldstein, M.D.	1978	Clair M. Kos, M.D.
1929	J.G. Wilson, M.D.	1979	G. Dekle Taylor, M.D.
1930	S. Mac C. Smith, M.D.	1980	Eugene Derlacki, M.D.
1931	D.H. Walker, M.D.	1981	Richard J. Belluci, M.D.
1932	L.W. Dean, M.D.	1982	J. Brown Farrior, M.D.
1933	G.I. Tobey, Jr., M.D.	1983	Jack V.D. Hough, M.D.
1934	John R. Page, M.D.	1984	Cary N. Moon, Jr., M.D.
1935	Samuel J. Crowe, M.D.	1985	Francis A. Sooy, M.D.
1936	F.R. Packard, M.D.	1986	Brian F. McCabe, M.D.
1937	E.P. Fowler, M.D.	1987	Harold G. Tabb, M.D.
1938	Harris P. Mosher, M.D.	1988	Richard R. Gacek, M.D.
1939	Isidore Friesner, M.D.	1989	D. Thane Cody, M.D.
1940	Horace Newhart, M.D.	1990	H.A. Ted Bailey, Jr., M.D.
1941	George M. Coates, M.D.	1991	William F. House, M.D.
1942	L. M. Seydell, M.D.	1992	Michael Glasscock, III, M.D.
1943-44	W.C. Bowers, M.D.	1993	Mansfield F.W. Smith, M.D.
1945-46	Gordon Berry, M.D.	1994	Robert I. Kohut, M.D.
1947	William E. Grove, M.D.		
174/	TTIMON E. CIOTE, ITED.		

PAST SECRETARY-TREASURER OF AOS

1868-1870	C.E. Ryder, M.D.
1870-1879	J.O Green, M.D.
1879-1898	J.J.B. Vermyne, M.D.
1898-1907	Frederick L. Jack, M.D.
1907-1912	James F. McKernon, M.D.
1912-191 <i>7</i>	John B. Rae, M.D.
191 <i>7</i> -1919	George E. Shambaugh, M.D.
1919-1925	Thomas J. Harris, M.D.
1925-1927	D. Harold Walker, M.D.
1927-1940	Thomas J. Harris, M.D.
1940-1945	Isidore S. Friesner, M.D.
1945-1950	Gordon D. Hoople, M.D.
1950-1955	John R. Lindsay, M.D.
1955-1960	Lawrence R. Boies, M.D.
1960-1965	James A. Moore, M.D.
1965-1972	Wesley H. Bradley, M.D.
1972-1977	G. Dekle Taylor, M.D.
1977-1982	Cary N. Moon, Jr., M.D.
1982-1987	D. Thane Cody, M.D.
1987-1992	Robert I. Kohut, M.D.

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

CONFLICT OF INTEREST DISCLOSURE FORM

Otological Society's ar In this regard, please b publication, public pos financial interests (incli	ted to my/our participation in the American nual Spring meeting to be held April 29-30, 1995. e advised that I am disclosing below any sitions, or memberships, as well as any personal uding equity positions, consulting agreements or nents) related to the proposed conference topic.
	I have no financial interests or advocacy positions related to the issues under discussion
	My relevant financial interests are:
	My relevant publications, public positions, or memberships are:

Las senior author, am confirming that I/we have no real or apparent

PUBLICATION STATEMENT

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

44

1995 PROGRAM ADVISORY COMMITTEE

Newton J. Coker, M.D. John R.E. Dickins, M.D. Robert A. Dobie, M.D. Maureen Hannley, Ph.D. Stephen G. Harner, M.D. Robert K. Jackler, M.D. Jack M. Kartush, M.D. Charles Luetje, M.D. Douglas E. Mattox, M.D.

John T. McElveen, Ir., M.D.





ABSTRACTS

of the

ONE HUNDRED TWENTY-EIGHTH ANNUAL MEETING

AMERICAN OTOLOGICAL SOCIETY, INC.

April 29-30 1995

Marriot Dessert Springs Resort Palm Desert, California

OFFICERS JULY 1, 1994 - JUNE 30, 1995

PRESIDENT
Robert A. Jahrsdoerfer, M.D.
6431 Fannin Street, Suite 6.132
Houston, TX 77030

PRESIDENT-ELECT
Derald E. Brackmann, M.D.
2100 West Third Street
Los Angeles, CA 90057

SECRETARY-TREASURER
Gregory J. Matz, M.D.
Loyola University Medical Center
Bldg 105, No 1870
2160 S. First Avenue
Maywood, IL 60153

EDITOR-LIBRARIAN
Joseph C. Farmer, Jr., M.D.
Division of Otolaryngology, Box 3805
Duke University Medical Center
Durham, NC 27710

COUNCIL
The above Officers
and
Mansfield F.W. Smith, M.D.
Robert I Kohut, M.D.
A. Julianna Gulya, M.D.
C. Gary Jackson, M.D.

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

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

TRANSTYMPANIC GENTAMICIN TITRATION THERAPY FOR MENIERE'S DISEASE

Loren J. Bartels, M.D., Jonathan S. Sillman, M.D.

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.

- To verify efficacy of transtympanic gentamic therapy in comparison to published surgical results.
- 2. To present a simplified method of transtympanic membrane delivery of gentamicin.
- To utilize titration method based on known delay in appearance of vestibular toxicity of topically applied gentamicin.

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

Mitchell K. Schwaber, M.D., Faith C. Wurm, M.S., James W. Hall, III, Ph.D.

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, .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 gentamic in 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 month 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 c

omplained of persistent disequilibrium following therapy.

- To report our experience with transtympanic gentamicin therapy for incapacitating Meniere's disease, including an improved dosing regimen.
- To demonstrate the usefulness of rotational testing as a means to monitor the efficacy of gentamicin therapy.
- To report the incidence of sensorineural hearing loss and persistent disequilibrium with transtympanic gentamicin therapy.

DEXAMETHASONE PERFUSION OF THE LABYRINTH PLUS INTRA-VENOUS DEXAMETHASONE FOR MENIERE'S DISEASE

John J. Shea, Jr., M.D., Xianxi Ge, M.D.

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 & 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:

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.

- To explain the pathology of Meniere's disease and how it is affected by dexamethasone perfusion of the labyrinth through the round window plus intravenous dexamethasone.
- To explain how patients are selected for dexamethasone perfusion plus intravenous dexamethasone and how the operation is performed.
- 3. To present the results with dexamethasone perfusion plus intravenous dexamethasone and explain the implications of these results for the pathology and treatment of Meniere's disease.

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

Herbert Silverstein, M.D., Seth I. Rosenberg, M.D.

It is well accepted the 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 Merocel 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.

- 1. To describe two methods to treat the inner ear directly with topical steroids.
- To propose a method to study the topical application of various drugs in the treatment of sensorineural deafness.
- To present the results of using intratympanic steroids in the treatment of Meniere's disease and autoimmune inner ear disease.

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

Athanasios Katsarkas, M.D., Henrietta Galiana, Ph.D, and Heather L. Smith, M.Eng.

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 1/6Hz, with protocols (52s 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.

- 1. To discuss the theorectical aspects of a clinical problem.
- 2. To show an example of basic science applied in the clinic.
- 3. To relate objective data to a psychologic situation.

WHAT IS THE MINIMAL VESTIBULAR FUNCTION REQUIRED FOR COMPENSATION?

F. Owen Black, M.D., Lewis M. Nashner, Sc.D.

Living without a vestibular system requires oppressive modification of life style and often prevents return to previous occupation and activities of daily living. Patients with unilateral and bilateral vestibular loss were studied to determine the minimal residual vestibular function required to achieve compensation. Three groups of patients with: 1) complete unilateral loss of vestibular function with normal horizonal canal-vestibulocular (HCVOR) function in the opposite ear, 2) complete unilateral loss with abnormal HCVOR function in the opposite ear, and 3) varying degrees of bilateral, symmetrical and asymmetrical vestibular functional loss, underwent vestibulo-ocular (VOR), visual-VOR (VVOR), & computerized dynamic posturography (CDP) tests before and after surgical procedures or ototoxic drug administration. Results suggest quantitative limits of residual vestibular function, either in the unoperated ear of patients with complete unilateral loss or a combination of the two ears in patients with bilateral loss, which must be present for the compensation of vestibular function loss. Data will be presented which supports findings from animal studies suggesting that intact otolith function in one ear is necessary for recovery of normal postural control after loss of vestibular function. Results of these studies have important implications for management of patients with vestibular disorders, including selection of patients for vestibular nerve section and streptomycin therapy.

OBJECTIVES:

- To review functional consequences and documentation of unilateral and bilateral loss of vestibular function.
- 2. To review published criteria for vestibular compensation.
- To compare residual vestibular function test results in patient groups who did and who did not achieve compensation after loss of vestibular function.

This study supported in part by NIH (NIDCD) grant R01 DC00205, NASA grant NAGW-3799 and a Legacy/Portland Hospitals Research Advisory Committee grant to Dr. Black.

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

Seth I. Rosenberg, M.D., Herbert Silverstein, M.D., Michael E. Hoffer, M.D.

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.

- 1 To compare hearing results after endolymphatic sac subarachnoid shunt (ESS) and posterior fossa vestibular neurectomy (VN) at one month and one year.
- 2. To show at one month no statistical difference in hearing between ESS and VN groups.
- To show statistical differences in hearing at one year with ESS group having worse hearing.

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

Benny Nageris, M.D., Saumil N. Merchant, M.D., Joe C. Adams, Ph.D.

The authors have observed that some temporal bones with endolymphatic hydrops (EH) show varying degrees of basalward displacement (towards the scala tympani) of the basilar membrane (BM) in the apical turn of the cochlea. Such mechanical distortion of the BM could conceivably alter cochlear mechanics and lead to sensorineural hearing loss.

This study is a systematic evaluation of 220 temporal bones in the collection at the Massachusetts Eye and Ear Infirmary designed to characterize and quantify this observation, and to determine its functional significance, if any. Medical histories were scrutinized and clinical otologic diagnoses assigned for all cases while being blinded to the histopathologic findings (in order to avoid observer bias). Similarly, histopathologic data were gathered without knowledge of the medical histories. Histologic evaluation included displacement of the BM (measured as the angle between the osseous spiral Iamina and BM), degree of hydrops, and assessments of the organ of Corti and stria vascularis. There were four groups of temporal bones: (1) normal, (2) presbycusis, (3) Meniere's disease with EH, (4) EH secondary to bacterial or viral labyrinthitis.

In the apical turn of the cochlea, there was severe basalward displacement of the basilar membrane such that it abutted the bony wall of the scala tympani in 50% of bones with hydrops (both Meniere's and secondary hydrops), 5% of bones with presbycusis, and in none of the normal bones. This marked difference between hydropic and normal/presbycusis bones was statistically significant. Two bones showing the basilar membrane plastered to the bony wall of the scala tympani had been obtained from two patients who had audiometric testing performed five hours and two weeks prior to death. Hearing thresholds at the lowest clinical frequencies (250 and 500 Hz) in the two cases were 20 dB and 30 dB respectively.

The high incidence of basilar membrane deformation in hydropic ears at the cochlear apex indicates a mechanical vulnerability of the basilar membrane where it is the widest. However, this pathologic change alone is not likely to cause hearing loss at clinical audiometric frequencies.

- To describe and characterize deformation of the basilar membrane in the apical turn in temporal bones with endolymphatic hydrops.
- To compare the occurrence of this change in hydropic bones to bones from normal and presbycusis ears.
- To determine the functional significance of this change by comparison to clinical audiometric findings.

REVISION STAPEDECTOMY WITH AND WITHOUT THE CO2 LASER: AN ANALYSIS OF RESULTS

Thomas J. Haberkamp, M.D., Steven A. Harvey, M.D., Yasser Khafagy, M.D.

A rapidly decreasing number of primary stapes cases and increasing number of trained Otologic surgeons have combined to make the goal of closure to within 10 dB air bone gap in 90% of cases difficult to maintain. This has made revision surgery an increasingly important aspect of stapes surgery and has led to the search for reliable technique that can be used in both primary and revision stapedectomy.

Laser stapedotomy has been proposed as a technique to increase the success rate of revision stapes surgery. All cases of revision stapes surgery performed by the senior authors performed between 1986 and 1994 were analyzed. There were 23 revision cases which represented 29% of the total of 78 stapes surgeries performed during that period. Five cases were performed without the laser all before 1988. In an attempt to increase the success rate for revision stapedectomy we began to use the laser in 1988 so it was the primary instrument used since that time. The success rate for closure of the air bone gap to within 10 dB was 56% for all cases with an average residual gap of 17 dB. Five cases were done without the laser with closure to within 10 dB air bone gap in 1 case (20%) and an average post operative air bone gap of 27 dB. The laser was used in 18 cases with 11 closing to within 10 dB (61%) and an average post operative conductive hearing loss of 14 dB. There was a statistically significant difference between the hearing results with and without the laser (p=.04).

The prognosis for a good hearing result appeared worse when the primary indication for surgery was vertigo. The average hearing result was 19.4 dB air bone gap and only 2/6 cases closed to within 10 db. This was because the most common finding in these cases was a long prosthesis which was successfully revised to within 10 dB in only 1 of 5 cases. When surgery was primarily performed for hearing the success rate for closure to within 10 dB with the use of the laser was 75% (9/12), and the average air bone gap was 12 dB. These results approached statistical significance (p=.06).

The most common reasons for failure of the previous surgery were bony refixation (11 cases), displaced prosthesis (8 cases), and an excessively long prosthesis (5 cases). In 12 cases there were multiple reasons for failure and in one no cause for failure was found.

There were no dead ears in the series and in two cases there was a sensorineural decline of 11 dB each. Revision stapes surgery represented a constant 30 percent of cases throughout this series. We have found CO2 laser stapedectomy to be a safe technique which produces reliable results in revision stapedectomy.

- 1. To discuss the results of revision stapes surgery with and without the use of the C02 laser.
- 2. To analyze the reasons for failure of the original surgery in this series.
- To present the technique of CO2 laser stapedectomy used in this series and discuss the prognostic factors for success.

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

Karen I. Berliner, Ph.D., Robert A. Goldenberg, M.D., and Karen Jo Doyle, Ph.D., M.D.

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 make a substantial difference in reported outcome. In otosclerosis surgery, however, the potential for surgical trauma to hearing is greater since the inner ear is 'opened'. Further, occlusion of the oval window and fixation of the stapes have both been shown to affect bone conduction responses, often resulting in improved postoperative bone scores. It was the purpose of this study to evaluate the effect of different choices regarding three factors commonly used in defining outcome: 1) choice of pre- or postoperative bone conduction scores for calculating postoperative air-bone gap, 2) choice of frequencies to include in averaging, and 3) choice of "success/failure" criterion.

Audiological data from 211 stapes surgery patients at three different institutions was used to generate a variety of outcome measures, including pure-tone thresholds for frequencies from .5 kHz to 8 kHz and different frequency combination PTAs and air-bone gaps. Subjects included 124 females and 87 males with a mean age of 49.4 years (range 12.4 to 80.2 years).

Paired comparison statistical analyses showed a significant improvement in postoperative bone conduction thresholds for all of the frequencies from .5 to 3 kHz as well as for the average of 1, 2, and 4 kHz. The largest mean improvement was 7.6 dB at 2 kHz.

Mean postoperative air-hone gap differed little across six different frequency combination PTAs. There was no difference between the means for the traditional 3-frequency (.5, 1, 2 kHz) and 4-frequency (.5, 1, 2, 3 kHz) average air-bone gaps. "Success rate" was slightly lower when 4 kHz was included in the frequency averaging.

For evaluating results in terms of absolute air conduction threshold, only data from the 70 patients with preoperative normal sensorineural hearing (bone conduction PTA for .5, 1,2, kHz or .5, 1,2,3 kHz of < 20 dB) were used. Mean postoperative air conduction threshold varied no more than 2.5 dB when any combination of the thresholds from .5 to 4 kHz was used in computing the PTA, and the percentage of subjects with "successful" result differed by only 4% for the traditional PTAs of .5, 1, 2 kHz vs .5, 1, 2,3, kHz.

As in the prior chronic ear study, differences in outcome were more drastically affected by definition of "success" than by frequencies included. Unlike similar data from chronic ear surgery, however, use of air and bone scores from the same test interval are crucial in stapes surgery for accurately reflecting air-bone gap. Use of preoperative bone score and postoperative air scores to compute the postoperative air-bone gap would artificially inflate results by making the gap appear smaller than it might actually be.

- 1. To increase awareness of how hearing results in stapes surgery can be presented.
- 2. To make the audience aware of implications of various methods of reporting results.
- To provide data-based support for the AAO-HNS's desire to standardize reporting procedures.

ENDOSCOPIC STAPEDECTOMY: A PRELIMINARY REPORT.

Muaaz Tarabichi, M.D.

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 .5, 1 and 2 kHz) at two months post-op. 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.

- 1. To describe the technique of endoscopic stapedectomy.
- 2. To discuss the advantages and disadvantages of endoscopic stapedectomy.
- 3. To discuss the results of endoscopic stapedectomy.

BIOGLASS* MIDDLE EAR PROSTHESIS: LONG-TERM RESULTS

Kevin R. Rust, M.D., George T. Singleton, M.D., June Wilson, Ph.D

The purpose of this study was to review the University of Florida's long-term results with Bioglass* middle ear prosthesis. In a clinical trial between April 1984 and November 1987, 37 patients were implanted with Bioglass* prostheses (19 TORPs, 12 PORPs and 6 stapes). Twenty-four patients had sufficient follow up for inclusion in this study, including five patients who are more than ten years out from surgery (range 24-126 months). TORPs have been removed because of early tympanic membrane graft failure (N=3) and late development of conductive hearing loss secondary to prosthesis fracture (N=3). In two cases, portions of fractured prosthesis extruded, leaving an intact tympanic membrane. There were no extrusions of intact prosthesis, even in patients where the prosthesis was placed directly under the tympanic membrane or graft (N=16). Mean postsurgical three frequency pure tone air bone gap (ABG) was 24.5dB (23% had ABG < 10dB, 45% had ABG < 20dB). Air-bone closure remained stable over time.

Our results demonstrate that Bioglass* middle ear prosthesis have excellent long-term tissue compatibility. Extrusions were only seen after prosthesis fracture. The three failures are attributed to stress fractures in early experimental prototypes.

OBJECTIVES:

- 1. To summarize the University of Florida's experience with Bioglass* middle ear prosthesis.
- 2. To demonstrate excellent long term results with Bioglass* in terms of tissue compatibility.
- 3. To demonstrate low extrusion rates.

This study supported in part by U.S. Biomaterials Corp.

^{*} Registered trademark.

LONG TERM HEARING RESULTS FROM PRIMARY OSSICULAR RECONSTRUCTION WITH AUTOLOGOUS BONE

Jay B. Farrior, M.D.

Restoration of hearing in chronic ear surgery remains a challenge for the otologic surgeon.

Various types of synthetic implants made have been advocated for middle ear reconstruction. For optimal success with synthetic prostheses, a second surgical procedure is often required. The long term hearing results in 124 patients who underwent primary ossicular reconstruction using bone grafts were reviewed. Type III tympanoplasties were performed in 84 patients and Type IV tympanoplasties in 40.

Post operative audiograms 2 through 15 years were reviewed. Long term hearing results were found to be stable using bone grafts after reconstruction at the primary surgery. The surgical techniques, long term hearing results and causes of failure are discussed. Tympanoplasty with reconstruction of the ossicular chain using bone grafts is an effective technique for restoring hearing in a single operation.

- To present long term hearing results using sculptured ossicle, homograft and cortical bone graft.
- To demonstrate that hearing results using bone graft remains stable for 2-10 years follow-up.
- 3. To discuss causes of failure.

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, B.S.E., Douglas A. Miller, B S.E.E., Alec N. Salt. Ph.D., John M. Fredrickson, M.D.

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.

- To present results of stapes displacement measurements when driven by an electromagnetic middle ear transducer using a fiber optic lever.
- To present results of action potential measurements for an electromagnetic middle ear transducer.
- 3. To show efficacy of an electromagnetic middle ear transducer with this data.

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

Richard L. Goode, M.D.

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 .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 TM's are much better than others. As would be expected, umbo displacement is decreased in ears with obviously damaged TM's, 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 TM's 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.

- To describe the laser Doppler vibrometer system and associated sound generating equipment.
- To detail how the LDV is used to make measurements of ossicle and prosthesis displacement.
- To discuss how these measurements can be used clinically to diagnose certain types of conductive hearing loss that mimic a sensorineural loss.

OSSEOINTEGRATION AND GROWTH EFFECTS OF TEMPORAL BONE PERCUTANEOUS PEDESTALS

James L. Parkin, M.D., M.S., Roy D. Bloebaum, Ph.D., Brett D. Parkin, B.S.

The percutaneous temporal bone pedestal has shown significant utility for the attachment of bone-anchored hearing aids, attachment of cosmetic auricular prostheses and as connectors 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 evaluates the effect of the pyrolized 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 less growth effect by a central single screw attachment system. Osseointegration of the attaching screws was experienced.

The second phase of the study evaluates 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 biological 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.

- 1. To demonstrate that osseointegration of titanium temporal bone pedestals will occur.
- 2. To show the effects of pedestal fixation to rapidly growing swine temporal bones.
- To emphasize the importance of pedestal fixation design and pedestal material design in the biologic acceptance by the temporal bone for such implants.

MAGNETIC RESONANCE IMAGING IN IDIOPATHIC SUDDEN SENSORINEURAL HEARING LOSS

George A. Gates, M.D., Todd Richards, M.D., Jay Tsuruda, M.D., Edward W. Rubel, Ph.D.

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 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.

- 1. To demonstrate the site of lesion in patients with ISSHL
- 2. To display the diagnostic value of phased array MRI in otology.
- 3. To correlate physiologic and anatomic findings in ISSHL.

THE USE OF THE TEMPOROPARIETAL FASCIAL FLAP IN TEMPORAL BONE RECONSTRUCTION

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

Reconstruction options for temporal bone defects following extirpative surgery for cancer, osteoradionecrosis, or chronic otitis media are few. Local muscle flaps with and without bone pate, cartilage and fascia are the main techniques available to otologists wishing to obliterate the mastoid and rebuild the external auditory canal (EAC). Although the neighboring temporoparietal fascial flap (TPFF) supplied by the superficial temporal vessels has been widely employed in auricular reconstruction, its versatility in temporal bone reconstruction has not been widely explored. The TPFF with its axial blood supply is a reliable tool for reconstruction of mastoid and EAC defects as this flap is thin, durable, conforming, and well suited to accept split thickness skin grafts (STSG). With superior extension of a standard post-auricular incision and minimal scalp undermining, flaps as large as 170 cm2 can be raised and safely rotated into the mastoid or EAC.

This technique was employed in 9 patients who presented with reconstructive dilemmas. Seven patients had undergone temporal bone resection for neoplasms. Two patients with chronic otitis media had defects secondary to revision mastoid surgery. Reconstruction with standard muscle flaps was not possible due to scarring, radiation damage, and disruption of tissue integrity during previous surgery. In 4 patients, the TPFF provided a healthy bed for STSG and mastoid obliteration with preservation of hearing. In 4 cases in which the EAC or concha had been removed, the TPFF provided the vascular foundation for EAC reconstruction with STSG. In a final case of temporal bone osteoradionecrosis in which standard fat tympanomastoid obliteration was obviated due to concerns for tissue viability in devascularized bone, the TPFF provided well vascularized tissue bulk. Follow up in these patients ranged from 8 to 43 months (average 22.5). There were no flap failures during this time; however, one patient experienced post-op EAC stenosis.

The TPFF flap has many advantages to the otologic surgeon who by virtue of previous surgery, cancer, or radiation is faced with reconstruction dilemmas centering around a poorly vascularized mastoid and temporal bone. The flap is extremely hardy and has been shown to have minimal risks of necrosis or flap failure. The TPFF is a reliable source of local, well vascularized tissue which is extremely pliable and facilitates both hearing and non-hearing preservation temporal bone reconstruction. We conclude that the TPFF flap is an important technique for otologic surgeons who routinely perform revision mastoidectomy for chronic otitis media and surgery for osteoradionecrosis and cancer of the temporal bone.

- To discuss the rationale for usage of the temporoparietal fascial flap in temporal bone reconstruction.
- To present the results of surgery in nine patients in which the temporoparietal fascial flap was employed for external auditory canal reconstruction as well as mastoid and tympanomastoid obliteration surgery.
- 3. To discuss the suitability of the temporoparietal fascial flap for other otologic surgery uses.

INVASION PATTERNS OF ADVANCED TEMPORAL BONE MALIGNANCIES

John P. Leonetti, M.D., Peter G. Smith, M.D., Ph.D., G. Robert Kletzker, M.D., Ricardo Izquierdo, M.D.

Primary malignancies of the temporal bone may originate in the external auditory canal, the middle ear cleft, 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 patients with stage III or IV malignancies of the temporal bone were retrospectively reviewed in order to correlate preoperative clinicoradiographic analysis with intra-operative findings. The following patterns of tumor invasion were identified: 1) Superior erosion through the tegmen tympani into the middle cranial fossa 2) Anterior extension into the glenoid fossa and infratemporal space, 3) Inferior growth through the hypotympanum and jugular foramen, and 4) Posterior involvement of the mastoid air cells. While otic capsule erosion was uncommon, a majority of these patients did present with lower cranial nerve palsies.

Complex surgical procedures exist for the en-block resection of advanced temporal bone cancers, and the functional as well as cosmetic reconstitution of the resultant defect. Appropriate operative planning must be based upon a knowledge of potential patterns of tumor extension and meticulous radiographic assessment.

- To present the preoperative clinical and radiographic findings in a series of 26 patients with advanced temporal bone malignancies.
- 2. To compare these preoperative findings with intraoperative findings.
- 3. To utilize this information to describe patterns of invasion of temporal bone malignancies.

NEUROPHYSIOLOGICAL APPROACH TO TINNITUS PATIENTS.

Pawel J. Jastreboff, Ph.D., Sc.D., William C. Gray, M.D., and Susan L. Gold, M.A.

A neurophysiological model of tinnitus has been successfully implemented for treating tinnitus patients. The main point of the theory of tinnitus based on neurophysiological principles is the postulate that all levels of the auditory pathways and several nonauditory systems. particularly the limbic system, are essential parts of each case of tinnitus, contribute in varying degrees in the emergence of tinnitus perception, and determine the level of its annoyance (Jastreboff, Neuroscience Research, 8:221-254, 1990). Furthermore, the model stresses the dominance of nonauditory systems in determining the level of tinnitus annoyance. From this perspective, the initial events on the periphery which trigger the cascade of processes resulting in the emergence of tinnitus are secondary; the primary stress is on the detection and processing of the tinnitus-related signal by the auditory and non-auditory nervous systems. Realizing our limitation in repairing the initial peripheral source of tinnitus, but cognizant of the extensive plastic property of the nervous system, it has been proposed to treat tinnitus patients by inducing and further facilitating habituation of the tinnitus signal. The goal is to reach a stage that, although patients can perceive tinnitus as unchanged when they focus attention on it, otherwise subjects are not aware of the presence of tinnitus. Furthermore, even when perceived. tinnitus does not evoke annoyance.

Habituation is induced utilizing low level broad band noise generated by wearable noise generators, and by appropriately used, if necessary amplified, environmental sounds according to a specific protocol. For habituation to occur it is imperative to avoid masking of tinnitus by these sounds. Since 1991 over 450 tinnitus patients have been seen in our Center. About 40% exhibited hyperacusis to varying degrees. A survey of over 100 patients revealed that the neurophysiological approach aimed at the habituation of tinnitus yielded over 80% of significant improvement in groups of patients treated with the full protocol involving counseling and the use of noise generators or properly fitted and used hearing aids. Notably, in patients who received counseling only, the success rate was below 20%. Furthermore, for some patients tinnitus disappeared totally. The improvement in hyperacusis was observed in about 90% of treated patients.

OBJECTIVES:

- To outline the theoretical basis of a new neurophysiological approach to tinnitus based on inducing and facilitating habituation of tinnitus.
- 2. To delineate clinical implications of the neurophysiological model, including rationale for interrelation of tinnitus and hyperacusis.
- To outline practical aspects of the habituation approach and to present a summary of clinical results.

Supported by NIH/NIDCD R01 DC00299 grant.

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

Terry A. Zwolan, Ph.D., Paul R. Kileny, Ph.D., Carissa A. Moeggenberg, M.A., Steven A. Telian, M.D.

To date, the experience in the United States with the Cochlear Corporation 22 electrode device has been confined to bipolar stimulation. Bipolar intracochlear stimulation is generally considered to result in current delivery to a restricted area and, thus, better place pitch discrimination when compared to monopolar stimulation. Monopolar intrascalar stimulation involves an active intracochlear electrode and a remote indifferent electrode. This mode of stimulation has been associated with a wider spread of current. Six patients have been implanted at the University of Michigan Medical Center with the new 20+2 version of the Cochlear Corporation device under an investigational device exemption. This device consists of the standard Cochlear Corporation intrascalar array carrying 20 electrodes and two remote indifferent electrodes. The first indifferent electrode consists of a ball electrode in the temporalis muscle, the second is a plate mounted on the lateral surface of the receiver stimulator. This configuration allowed the comparison of three modes of stimulation: 1) standard bipolar; 2) monopolar with the ball electrode; and 3) monopolar with the plate electrode. Performance with these three configurations is being investigated employing a within-subject balanced crossover design. Of the six subjects implanted to date, four have completed testing in all three modes; the two remaining subjects will complete testing by January, 1995. Results indicate substantially lower threshold and comfort levels in the monopolar when compared to the bipolar mode. In three of the four subjects who have completed testing to date, speech recognition results are significantly improved in the monopolar mode when compared to the standard bipolar mode of activation. The lower current requirement associated with the monopolar mode of stimulation is a distinct advantage, especially when considering life-long electrical stimulation. Additionally, this mode is likely to offer a larger dynamic range in patients with poor neural survival. Since this new device offers both monopolar and bipolar stimulation it should be considered to replace the current bipolar only device.

- To provide information about the configuration of the new Cochlear Corporation 20+2 implant.
- To provide information about the surgical differences between the standard and the 20+2 implant.
- 3. To provide information about the differences in patient performance when programmed in the standard bipolar versus the monopolar mode, both available with the 20+2 implant.

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

J. Gail Neely, M.D., Peggy S. Neufeld, M.A.

Experiences with patients with facial paralysis over the last twenty-five years and recent efforts to develop a computer assisted method to measure paresis and synkinesis lead us to three hypotheses: (1) Specific areas of the face are more disturbing, when dysfunctional, than others; (2) There are multiple occupational, social, and personal impacts of even minor degrees of facial paralysis; and (3) The impact of facial paralysis is markedly underestimated by those who do not have it.

An initial questionnaire of ten open ended items was submitted to eleven recruited volunteer subjects with varying degrees of facial paralysis following acoustic tumor resection. The qualitative and tabulated results form the basis of this report.

The results indicate that paresis of the eye initially is most disturbing; later the most disturbing paresis is of the mouth. Synkinesis is often more disturbing than paresis. Major occupational, social, and personal impacts of paralysis were common. There was strong evidence that the impacts of paralysis are underestimated by those unaffected.

On the basis of this study, we have undertaken a major project to develop and validate a quantitative questionnaire, beginning with the use of focus groups. It would appear that serious attention to these outcome issues is required.

- 1. To identify the facial region most distressing to the patient with facial paralysis.
- 2. To identify occupational, social, and personal impacts of facial paralysis.
- 3. To determine how patients with paralysis feel others appreciate the handicap.

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

Lawrence R. Lustig, M.D., Robert K. Jackler, M.D.

A thorough understanding of the microanatomical relationships of the most common tumors presenting in the jugular foramen (JF) is critical for the surgical preservation of associated neurovascular structures. Anatomically, the JF has 3 loci in which these tumors may reside: a lateral compartment containing the jugular vein and bulb, a medial hiatus transmitting the inferior petrosal sinus, and an intervening fibro-osseous plate upon which cranial nerves IX-XI lie. A preoperative awareness of the most probable relationship of a JF tumor to the nerves and vessels within this complex structure has several important surgical implications. Tumors which arise lateral to the neural plane are favorable as they are more readily microdissected from the nerve trunks displaced on their deep surface. By contrast, medially positioned tumors are unfavorable because the nerves lie splayed on the superficial aspect of the tumor, making surgical removal more difficult. Glomus tumors, which have the most stereotypical growth pattern of all JF tumors, consistently arise in the lateral compartment and thus displace the lower cranial nerves into a favorable medial position. Schwannomas arise along the central axis of the foramen and, depending upon the nerve of origin, may displace the uninvolved nerves laterally, as seen with tumors arising from the Xth nerve (unfavorable), or medially, as in the case of a IXth nerve tumor (favorable). Most jugular foramen meningiomas originate from dural surfaces of the posterior fossa and secondarily invade the foramen. Those originating from the posterior surface of the petrous pyramid or sigmoid sinus region typically displace the lower cranial nerves on their medial surface, a position which favors neural preservation. In contrast, meningiomas arising from the lower clivus are unfavorable, splaying the multiple small rootlets which coalesce into CN IX-XI between the tumor and the surgeon.

The propensity of these 3 tumors to cause progressive occlusion of the jugulosigmoid complex also has important surgical implications. Because glomus tumors arise within the jugular bulb, the venous system is nearly always occluded at an early stage. In both meningiomas and schwannomas, however, the jugular system may occasionally remain patent, even with relatively large lesions. A patent venous system positioned lateral to the tumor may constitute a significant impediment to surgical extirpation, and may need to be sacrificed when complete removal is the desired goal. It is important to evaluate venous sinus patency and dominance preoperatively through angiography and/or MR venography, since abrupt interruption of a dominant system risks intracerebral venous infarction. Illustrative cases will be presented to demonstrate the predominant neurovascular relationships of each of major type of tumor which involves the JF.

- To discuss the microanatomy of the three most common tumors of the jugular foramen (JF).
- To describe how the presentation of each tumor impacts on the surgeon's ability to preserve individual cranial nerves and vascular structures in the jugular foramen.
- To use case illustrations to support the assertion that jugular foramen tumors present a predictable manner with regard to JF neurovascular structures.

HEARING CONSERVATION IN SURGERY FOR GLOMUS JUGULARE TUMORS

C. Gary Jackson, M.D., David S. Haynes, M.D., Michael E. Glasscock, III, M.D., Anne Forrest Josey-Tallent, M.S.

The most common grounds on which surgery for glomus jugulare tumors is criticized is the perceived risk of functional incapacity which attends possible cranial nerve loss. It is aggregate lower cranial nerve loss which 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 phono-pharyngeal surgical deficits has, however, neutralized much of this criticism.

The issue of hearing conservation in neurotologic skull base surgery, on the other hand, has not been well documented toward this end. The presence of a glomus jugulare neoplasm need not reflexly nor technically forfeit pre-existing hearing. Hearing conservation is, admittedly, a priority subordinate 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've come to reasonably expect of conservation surgery. Hearing salvage further serves to define the concept of neurotologic skull base surgery.

Hearing preservation in 122 glomus jugulare tumor patients is reviewed. Intuitively, as for acoustic tumors, hearing conservation 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.

- To clearly define selection criteria for hearing conservation in glomus jugulare tumor surgery.
- To elaborate the technical feasibility of hearing conservation in glomus jugulare tumor surgery by evaluating the (operative) technique which maximizes conservation potential without sacrificing oncogenically sound resection.
- To compare the success of hearing conservation in glomus tumor surgery to that presented in the radiation therapy literature.

THE DIAGNOSIS OF INTRA-AXIAL POSTERIOR FOSSA LESIONS

Arvind Kumar, M.D., Marlos A.G. Viana, Ph.D., Albert Pieri, B.S.

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 is 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, micro-processor 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 imaging (MR) confirmed lesions of the PNA. The PNA lesions confirmed in these 70 patients included Type I Chiari malformation (30), brainstem/cerebellar infarct 10, vertebro-basilar insufficiency 2, arterio-venous malformation 2, multiple sclerosis 9, IVth ventricle cyst 1, arachanoid cyst 1, interhemispheric epidermoid 1, active cysticercosis 1, pontine venous angioma 1, Dandy Walker cyst 1, basilar impression 2, cerebellar metastases 1, cerebellar 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 the VDEC is a reliable sign of posterior fossa lesion 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.

- 1. To report the results of a meta-analysis of the literature on diagnostic tests for PNA lesions.
- To show that vestibular decruitment has a sensitivity of 84% for PNA lesions and a specificity of 100%.
- 3.To report that the Torok monothermal caloric test is a cost effective and reliable diagnostic test.

HISTOLOGIC EVALUATION OF AERATION ROUTES IN TEMPORAL BONES WITH CHOLESTEATOMA

Atsushi Haruta, M.D., Patricia A. Schachern, B.S., Tetsuya Tono, M.D., Michael M. Paparella, M.D., Tamotsu Morimitsu, M.D.

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 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 resistent 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 intact canal wall technique, to keep this route patent for mastoid aeration to prevent recurrence of cholesteatoma.

- 1. To clarify the histologic aeration remaining in temporal bones with cholesteatoma.
- To evaluate the aeration routes histologically in temporal bones with cholesteatoma with aeration routes.
- To present the significance of the aeration route from eustachian tube directly to attic and mastoid.

MANAGEMENT OF LABYRINTHINE FISTULAE SECONDARY TO CHOLESTEATOMA

Jacques A. Herzog, M.D., Peter G. Smith, M.D., Ph.D., G. Robert Kletzker, M.D., Kenneth S. Maxwell, M.D.

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 sixteen patients who developed labyrinthine fistula secondary to cholesteatoma. Fifteen involved the horizontal semi-circular canal and one resulted in a fistulization of the oval window. The cholesteatoma matrix was removed in all cases and the underlying fistula repaired primarily. Cochlear function was preserved in all patients. Fifteen of sixteen patients have had no further difficulty with vertigo beyond the immediate postoperative period. The evaluation and contemporary management of this difficult problem is discussed.

- To properly evaluate patients who may have a labyrinthine fistula secondary to cholesteatoma.
- To understand the appropriate technique for cholesteatoma matrix removal from a labyrinthine fistula.
- 3.To discuss the outcome of total cholesteatoma removal in the patient with labyrinthine fistula utilizing this technique.

MECHANICAL VERSUS CO₂ LASER OCCLUSION OF THE POSTERIOR SEMICIRCULAR CANAL IN HUMANS

Patrick J. Antonelli, M.D., Jack M. Kartush, M.D., Larry B. Lundy, M.D., and Don L. Burgio, M.D.

Carbon dioxide and argon lasers have been promoted as helpful adjuncts in posterior semicircular canal occlusion (PCO) for intractable benign paroxysmal positional vertigo (BPPV), but no studies have compared these laser-assisted techniques to the original, mechanical occlusion technique. The purpose of this study was to compare the effectiveness of mechanical against laser-assisted PCO.

From January 1992 to April 1994, 12 consecutive patients with intractable BPPV underwent PCO by 3 surgeons. Six were treated with mechanical PCO, and 6 were treated with CO2 laser-assisted occlusion of the membranous labyrinth, followed by mechanical occlusion with bone pate and fascia. PCO eliminated positional vertigo in 5 of 6 patients from each group. Dysequilibrium was present in all patients immediately postoperatively. This resolved in 5 of 6 patients treated with the CO2 laser but in only 1 of 6 patients treated without the laser (p=0.04). Patients were hospitalized for dysequilibrium for an average of 5.4 and 3.0 days for the mechanical and laser-assisted groups, respectively. Preoperative and postoperative hearing was not significantly different between the groups. No 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 CO2 laser to seal the membranous canal prior to occluding the bony canal.

- To assess the effectiveness of posterior semicircular canal occlusion with mechanical and laser-assisted techniques in the treatment of benign paroxysmal positional vertigo.
- To compare vestibular sequelae in patients treated with posterior canal occlusion, using mechanical and laser assisted techniques.
- To compare audiometric outcomes in patients treated with posterior canal occlusion, using mechanical and laser assisted techniques.



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

Joseph B. Roberson, Jr., M.D., Allen Senne, M.A., Derald E. Brackmann, M.D., William E. Hitselberger, M.D.

A new application using direct cochlear nerve action potentials (CNAP's) for monitoring middle fossa acoustic neuroma resection with attempted hearing preservation is described. Advantages of this technique over auditory brainstem response monitoring include real time or very near real time measurement of evoked auditory potentials, improved surgeon learning curve, applicability to all patients undergoing hearing preservation surgery independent of the quality of the clinically averaged ABR waveform morphology or in the absence of a reliable ABR tracing, and, possibly, interpretation of changes in amplitude and latency of waveforms as predictors of hearing preservation.

Four patients have been monitored with this procedure to date. Based on work with these cases, an extradural site was selected for electrode placement, the dural flap arrangement was developed, and early results were obtained suggesting that changes in latency and waveform morphology do occur as a result of surgical manipulations.

In the current technique, the electrode is placed between the bony floor of the IAC and the dura in a position adjacent to the eighth nerve. The dura is opened along with the posterior margin of the superior vestibular nerve and reflected anteriorly, covering the electrode and a portion of the wire. Waveforms may be recorded using electrode placement over the entire area from the cochlea to the carotid to the superior semicircular canal. Small variations in waveform size and morphology can occur with changes in placement. Thus, stability of placement is important. Brisk responses can be obtained with a single or very few stimulus presentations. Data from additional subjects, including prediction of hearing preservation, will be available shortly.

- To describe a new procedure for cochlear nerve monitoring during acoustic tumor surgery with the goal of hearing preservation.
- To demonstrate the effectiveness of the procedure in providing rapid feedback to the surgeon.
- To elucidate the advantages of this cochlear nerve action potential procedure over ABR monitoring.



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

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.

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 CO2 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 a 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 in fluences varying from 15 to 75 mJ per pulse. Spot size was estimated to be a 0.5mm2. 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 polyvinyldifluoride (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 N/M2 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.

OBJECTIVES:

- To report the finding of photoacoustic/photomechanical transients during pulsed laser ablation of the temporal bone.
- To characterize photoacoustic events during laser ablation of the temporal bone and discuss the clinical significance.
- To discuss pulsed laser phenomenology and its sequela in terms of laser mediated trauma during middle ear surgery.

This work was supported by the following grants ONR N0014-91-C-0134, DOE DE-FG03-91ER61227 and NIH 5P41RR01192. Dr. Wong 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, M.D., Arthur Kristiansen, M.S., Jonathan Haines, Ph.D.

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.

- To discuss the scientific rationale for the investigation of a possible measles virus etiology in otosclerosis.
- 2. To review the evidence to date supporting a possible measles virus etiology in otosclerosis.
- To present molecular biologic evidence of measles virus gene sequences in archival human otosclerotic temporal bone specimens with otosclerosis.



EFFECTS OF NEUROTROPHIC FACTORS ON THE SURVIVAL AND REGENERATION OF AUDITORY NEURONS

Hinrich Staecker, M.D., Richard Kopke, M.D., Philippe Lefebvre, M.D., Ph.D., Bridgitte Malgrange, Ph.D., Wei Liu, B.S., Joseph Feghali, M.D., Gustave Moonen, M.D., Ph.D., Thomas Van De Water, Ph.D., Robert Ruben, M.D.

Auditory hair cells have been shown to produce a variety of trophic factors that directly affect growth and survival of auditory neurons. These include the neurotrophins NGF, BDNF and NT-3, which are all members of a family of small peptides. When hair cell death occurs after noise trauma or ototoxic damage, these factors are no longer produced and neuronal degeneration results. We have developed a number of in vitro models for testing the role of these growth factors in supporting neuronal survival after removal of hair cell trophic support. Auditory neurons grown in dissociated cultures or in mass organotypic cultures degenerate after 24 hrs. in vitro. This degeneration could be prevented through addition of NT-3 or BDNF to the culture medium. Survival of adult auditory neurons grown without their peripheral (hair cell) and central (brain stem) targets was significantly increased when compared to controls. Addition of NGF did not increase survival, but brought about a regeneration of injured neuritic processes. Using whole modiolus cultures of the adult auditory (spiral) ganglion from which the organ of Corti had been stripped has helped us to identify a second type of growth factor, i.e., ciliary neurotrophic factor (CNTF), that can affect the survival of injured auditory neurons. Presently the most effective factor tested to stimulate the repair and regrowth of injured or severed neuritic processes of adult auditory neurons has been NGF, a factor that when used alone does not affect neuronal survival. Therefore, NGF must be used in combination with a survival promoting factor, i.e. BDNF or NT-3 and possibly CNTF to maintain the neuronal population of the spiral ganglion after hair cell injury. There is sufficient documentation of the in vitro effects of trophic factors on cultures of developing auditory neurons and on cultures of adult injured auditory neurons in vitro to begin to formulate in vivo strategies for the use of growth factors as drugs to treat the effects of injury or aging on adults auditory neurons. Possible therapeutic strategies and methods of delivery will be discussed.

OBJECTIVES:

- 1. To discuss the role of neurotrophins in the survival of auditory neurons with emphasis on prevention of neuronal degeneration.
- To review experimental data regarding the role of trophic factors in neuritogenesis and regeneration.
- 3. To discuss possible delivery methods for neurotrophin therapy.

This study supported by grants from NIDCD (DC00088) to TRV, Deafness Research Foundation to HS, and National Fund for Scientific Research of Belgium to PPL and GM.



PATHOLOGICAL CHANGES OF THE EXTERNAL AND MIDDLE EAR IN ANIMALS WITH TGF-ALPHA DEFICIENCY

Charles G. Wright, Ph.D., Karen S. Robinson, B.S., Sarah A. Comerford, Ph.D., William L. Meyerhoff, M.D., Ph.D.

Transforming growth factor alpha (TGFa) is a growth regulatory peptide present in a variety of epithelial tissues. It is produced by keratinocytes and has been reported to be over expressed in several epidermal diseases, including middle ear cholesteatoma. Although TGFa and various other growth factors are believed by some investigators to influence cholesteatoma formation, no animal models are currently available in which this disease occurs in association with a specific growth factor defect. A mutant mouse (designated Waved-1 due to pelage abnormalities) which may provide such a model is now under investigation in the authors' laboratory.

Waved-1 mutants have a genetically determined, life-long deficiency of TGFa. In addition to previously documented eye defects and hair follicle abnormalities, our studies have revealed that these animals develop striking alterations of the external ear canal and middle ear which may be relevant to the pathogensis of aural cholesteatoma. By 8-10 weeks of age, most mutants demonstrate cellular and keratinaceous debris filling the middle ear cavity. These lesions originate in the medial portion of the external ear canal with hyperplasia of the epidermis and its accessory structures, including hair follicles and sebaceous glands. At this stage, while the tympanic membrane is still intact, the middle ear shows serous effusion, mucosal metaplasia, and osteoneogenesis. Morphological studies on the sequence of histopathologic changes occurring in the ears of Waved-1 mutants of different ages are currently underway and findings of that work will be reported. As the sequential development of aural abnormalities seen in this model becomes better understood we expect to gain new insights into the role of transforming growth factor alpha in external and middle ear disease.

- 1. To describe a new animal model for the study of external and middle ear disease.
- 2. To document histopathologic changes occurring in this model.
- To discuss the possible role of transforming growth factor alpha in diseases of the external and middle ear.

IMMUNOHISTOCHEMICAL FINDINGS IN THE COCHLEA OF AIDS CASES

Jessica W. Lim, M.D., J. Thomas Roland, Jr., M.D., Jin S. Lim, M.D., James Lee, B.A., Bernard Ong, B.A., Dean E. Hillman, Ph.D.

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, HIVlike 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 p17 and p24 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.

- To show positive identification of the human immunodeficiency virus in human cochlear tissue.
- 2. To lend further evidence to the hypothesis of a direct viral cochlectoxic effect.
- To correlate immunohistochemical findings to previously published ultrastructural changes seen in HIV-infected cochlear tissue.

EFFECT OF LEUKOTRIENE INHIBITOR ON OTOACOUSTIC EMISSIONS IN SALICYLATE OTOTOXICITY

Timothy T.K. Jung, M.D., Ph.D., Johnny Arruda, M.D.

Our previous work has shown that salicylate ototoxicity is associated with decreased levels of prostaglandins and increased levels of leukotrienes (LTs) in perilymph. Pretreatment with LT inhibitor was found to prevent salicylate ototoxicity. Other studies have 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 the current study was to determine the effect of LT blocker (Sch. 37224) on otoacoustic emissions in salicylate ototoxicity.

In the first part of the study, the effect of systemic salicylate ototoxicity on transient-evoked otoacoustic emissions (TEOAEs) with and without LT blockade was investigated in chinchillas. In the next two parts, effects of the round window membrane (RWM) application of salicylate and LTC4 respectively, on TEOAEs both with and without LT blockade were determined. Saline application on the RWM served as control.

With systemic salicylate ototoxicity, the TEOAEs decreased significantly over 8 hours and this was prevented by pretreatment with LT blocker (Sch. 37224). Both salicylate and LTC4 application on the RWM were followed by significant decreases in TEOAEs and the decrease in responses was prevented by pretreatment with LT blocker. There was no significant change in TEOAEs in the control group.

This study provides further evidence that the site of action in salicylate ototoxicity is the outer hair cell. Moreover, salicylate ototoxicity appears to be mediated by the elevated levels of leukotrienes as a consequence of cyclooxygenase inhibition.

- 1. To review what is known about salicylate ototoxicity.
- To report the effect of leukotriene inhibitor on octracoustic emissions in salicylate ototoxocity.
- 3. To propose a mechanism of salicylate ototoxicity.

NAMES AND ADDRESSES OF PRIMARY AUTHORS

Patrick J. Antonelli, M.D. Department of Otolaryngology University of Florida Box 100264 Gainesville, FL 32610

Loren I. Bartels, M.D.
University of South Florida
Department of Surgery MDC-16
12901 Bruce B. Downs Blvd.
Tampa, FL 33612

Karen I. Berliner, Ph.D.
House Ear Institute
2100 West Third Street, Fifth Floor
Los Angeles, CA 90057

F. Owen Black, M.D. Neurotology Research N010 1040 N.W. 22nd Avenue Portland, OR 97210

Derald E. Brackmann, M.D. House Ear Clinic 2100 West Third Street Los Angeles, CA 90057

Mack L. Cheney, M.D.

Massachusetts Eye and Ear Infirmary
Department of Otolaryngology
243 Charles Street
Boston, MA 02114

Jay B. Farrior, M.D. 509 West Bay Street Tampa, Florida 33606

George A. Gates, M.D.
Virginia Merrill Bloedel Hearing
Research Center
University of Washington XF-01
1325 4th Avenue, Suite 2000
Seattle, WA 98101

Richard L. Goode, M.D. 300 Pasteur Dr., R-135 Stanford University Medical Center Stanford, CA 94305-5328

Thomas J. Haberkamp, M.D. 9200 W. Wisconsin Avenue Milwaukee, WI 53226 Atsushi Haruta, M.D.
Room 226 Lions Research Building
Dept. of Otolaryngology
2001 Sixth Street S.E.
Minneapolis, MN 55455

Jacques A. Herzog, M.D.
The Center for Hearing & Balance Disorders
11155 Dunn Road, Suite 209 East
St. Louis, MO 63136

C. Gary Jackson, M.D.
The Otology Group
300 20th Avenue North, Suite 502
Nashville, TN 37203

Pawel J. Jastreboff, Ph.D., Sc.D. University of Maryland at Baltimore M.S.T.F. Building, Room 434F 10 South Pine Street Baltimore, Maryland 21201-1192

Timothy T.K. Jung, M.D., Ph.D. Loma Linda University 11790 Pecan Way Loma Linda, CA 92354

A. Katsarkas, M.D. Royal Victoria Hospital #E4.48, Montreal, Quebec H3A 1A1 Canada

Arvind Kumar, M.D. Room B.42, 1855 W. Taylor, M/C 648 Chicago, IL 60612

John P. Leonetti, M.D.
Department of Otolaryngology-HNS
Loyola University Medical Center
2160 S. First Avenue
Bldg. 105, Rm. 1870
Maywood, IL 60153

Jessica W. Lim, M.D. New York University Medical Center 550 First Avenue New York, NY 10016

Lawrence R. Lustig, M.D. UC San Francisco 400 Parnassus Avenue - A 717 San Francisco, CA 94143-0342

NAMES AND ADDRESSES OF PRIMARY AUTHORS

Michael J. McKenna, M.D. Massachusetts Eye & Ear Infirmary 243 Charles Street Boston, MA 02114

Saumil N. Merchant, M.D. Massachusetts Eye and Ear Infirmary Department of Otolaryngology 243 Charles Street Boston, MA 02114

J. Gail Neely, M.D.
Department of Otolaryngology-HNS
Washington University School of Medicine
517 S. Euclid Ave, Box 8115
St. Louis, MO 63110

James L. Parkin, M.D. University of Utah School of Medicine 50 North Medical Drive Salt Lake City, Utah

Thomas C. Robey, B.S.E. 3261 Suphur Avenue, #1 St. Louis, MO 63139

Seth Rosenberg, M.D. 1901 Floyd Street Sarasota, FL 34239

Kevin R. Rust, M.D. Dept. of Otolaryngology Box 100264 Gainesville, FL 32610

Mitchell K. Schwaber, M.D. Vanderbilt University Medical Center S-2100 Medical Center North Nashville, TN 37232-2559

Herbert Silverstein, M.D. 1901 Floyd Street Sarasota, FL 34239

John J. Shea, Jr., M.D. Shea Clinic 6133 Poplar Pike Memphis, TN 38119 Hinrich Staecker, M.D. Albert Einstein College of Medicine Kennedy Center 302 1410 Pelham Pkwy South Bronx, NY 10461

Muaaz Tarabichi, M.D. 3535 30th Avenue, Suite #204 Kenosha, WI 53144

Charles G. Wright, Ph.D.
Dept. of Otolaryngology
UT Southwestern Medical Center
5323 Harry Hines Blvd.
Dallas, TX 75235

Brian J.F. Wong, M.D. Beckman Laser Institute 1002 Health Sciences Rd., East Irvine, CA 92715

Terry Zwolan, Ph.D. University of Michigan Department of Otolaryngology 1500 E. Medical Center Drive Ann Arbor, MI 48109-0312

NOTES:

NOTES:

NOTES:

Author's signature on the following statements were required on all papers submitted to the American Otological Society. Each author was advised that the submitted paper is property of THE AMERICAN JOURNAL OF OTOLOGY and cannot be reprinted without permission of the Journal.

CONFLICT OF INTEREST DISCLOSURE FORM

I, as senior author, am confirming that I/we have no real or apparent conflict of interest related to my/our participation in the American Otological Society's annual Spring meeting to be held April 29-30, 1995. In this regard, please by advised that I am disclosing below any publication, public positions, or memberships, as well as any personal financial interests (including equity positions, consulting agreements or employment arrangements), related to the proposed conference topic.
Thomas Constitution of the

 I have no financial interests or advocacy positions related to the issues under discussion.
 My relevant financial interests are:
 My relevant publications, public positions or, memberships are:

PUBLICATION STATEMENT

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

1995 PROGRAM ADVISORY COMMITTEE

Newton J. Coker, M.D.
John R.E. Dickins, M.D.
Robert A. Dobie, M.D.
Maureen Hannley, Ph.D.
Stephen G. Harner, M.D.
Robert K. Jackler, M.D.
Jack M. Kartush, M.D.
Charles Luetje, M.D.
Douglas E. Mattox, M.D.
John T. McElveen, Jr., M.D.

