2000

TRANSACTIONS
AMERICAN OTOLOGICAL SOCIETY, INC.
2000

VOLUME 88
ONE HUNDRED THIRTY-THIRD ANNUAL MEETING
ORLANDO WORLD CENTER MARRIOTT
ORLANDO, FLORIDA
MAY 13–14, 2000

LIPPINCOTT WILLIAMS & WILKINS

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INTRODUCTION OF AWARD OF MERIT WINNER

Charles Luetje, M.D.

It is a great pleasure as past President of the American Otological Society to present to you our Award of Merit winner. Our first slide shows our Award of Merit winner with all seven brothers and sisters. The awardee was born in New York and had four brothers and two sisters.

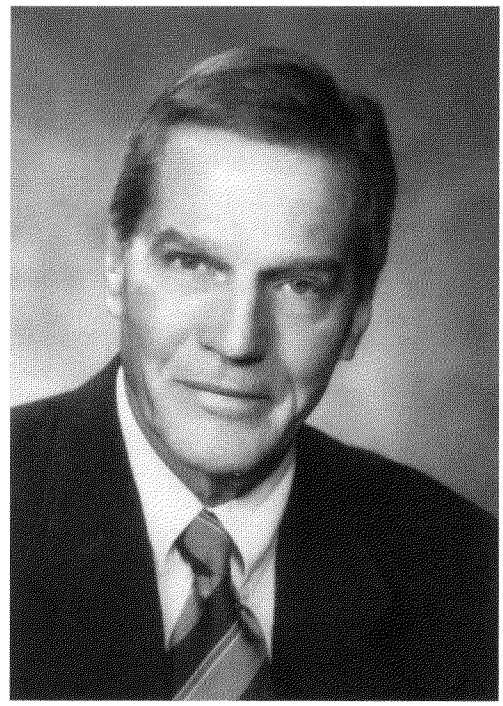
He developed a flare for fast cars at an early age, and also learned to enjoy water sports. His father felt it was important to keep the family together, and every other Sunday he would drive the 1932 Buick from Glendale, Queens, New York, to Lake Ron Kon Kama. Our Award of Merit winner loved all kinds of sports, particularly football. He was a champion on his basketball team.

To earn money during the early days, our awardee collected live Japanese beetles and sold them for five cents a pound, and fetched golf balls. He also worked at an ice cream parlor. He always seemed to work late or come home late. The real reason was that he and his brother shared a room. Each had a window. He wanted it warm, window closed, and his brother wanted it cold, window open. This didn't work, and it was agreed that the last one in would control the window.

Our Award of Merit winner has always been interested in being physically fit. He had other hobbies. A direct quote from his brother is, "He was known as a ladies' man." In high school he was so struck by the appearance of the beauty queen, Ms. Steel Pier, across the river in New Jersey, he was able to get a date with her. This fame continued into his first year at college. He was the first to go to college–in Rochester, New York. He was selected by Kodak to appear for a poster that hung in Grand Central Station. Ah, yes, college life was good.

College life was expensive. It was not exactly proceeding the way his father had in mind. The funding was cut off, and our Award of Merit winner joined the Navy for three years.

Basketball continued, in addition to his duties, and aerial photography. He is seen in this picture home on leave in 1957. After the Navy he enrolled in George Washington University, and so did 90% of his brothers' clothes, I was told. He received his undergraduate degree from George Washington. He then received his medical degree from the University of Virginia School of Medicine in 1961. After completing his residency at Yale–New Haven Hospital in 1966, he joined the faculty at the University of Virginia. In 1982 he moved to Houston to accept the chairmanship of the Otolaryngology Department at the University of Texas; he returned to the University of Virginia in 1995. As can be seen, our Award of Merit honoree is Robert H. Jahrsdoerfer, M.D.



Robert H. Jahrsdoerfer, M.D.

Our Award of Merit winner's many accomplishments include receiving the 1978 Triological Society's Mosher Award for work on congenital atresia. He is also past vice president of the Triological Society–Southern Section, past president of the American Neurotology Society, and past president of our own Otological Society. His own personal experience in surgery for congenital aural atresia exceeds 1,200 cases, and his expertise is known the world over. It is a pleasure to introduce to the members of the American Otological Society Dr. Robert H. Jahrsdoerfer as our Award of Merit winner on the occasion of the 133rd Annual Meeting of the Society.

AWARD OF MERIT RECIPIENTS 1949–2000

1949 George M. Coates, M.D.
1951 Barry J. Anson, Ph.D., and Theodore H. Bast, Ph.D.
1952 Edmund P. Fowler, M.D.
1953 Julius Lempert, M.D.
1954 Stacy R. Guild, M.D.
1957 Georg von Békésy, Ph.D.
1959 E. 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. Boies, M.D.
1969 Sir Terence Cawthorne
1970 Senator Joseph 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 D. Lathrop, M.D.
1978 Juergen Tonndorf, M.D.
1979 John E. 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 Jr., M.D.
1987 Jack V. Hough, M.D.
1988 George T. 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.
1995 D. Thane R. Cody, M.D., Ph.D.
1996 F. Blair Simmons, M.D.
1997 Michael E. Glasscock III, M.D.
1998 Michael M. Paparella, M.D.
1999 Mansfield F. W. Smith, M.D., M.S.
2000 Robert A. Jahrsdoerfer, M.D.

GUESTS OF HONOR 1949–2000

1949 Harris P. Mosher, M.D.
1950 D. Harold Walker, M.D.
1951 John Mackenzie Brown, M.D.
1952 Edmund P. Fowler, M.D.
1953 H. I. Lillie, M.D.
1956 Stacy R. Guild, Ph.D.
1958 Ralph A. Fenton, M.D.
1961 Julius Lempert, M.D.
1962 Philip Meltzer, M.D.
1963 William J. McNally, M.D.
1964 Kenneth M. Day, M.D.
1965 Senator Joseph Sullivan, M.B.
1966 Dean M. Lierle, M.D.
1967 Lawrence R. Boies, M.D.
1968 Sir Terence Cawthorne
1969 Gordon D. Hoople, M.D.
1970 John R. Lindsay, M.D.
1971 E. Glen Wever, Ph.D.
1972 Frank D. Lathrop, M.D.
1973 Moses H. Lurie, M.D.
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 F. 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. Seyfarth. 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., Ph.D.
1994 Cesar Fernandez, M.D.
1995 Richard R. Gacek, M.D.
1996 James L. Sheehy, M.D.
1997 Mansfield F. W. Smith, M.D., M.S.
1998 Robert A. Jahrsdoerfer, M.D.
1999 Barbara A. Bohne, Ph.D.
2000 Derald E. Brackmann, M.D.

SCIENTIFIC SESSIONS
2000 PRESIDENTIAL ADDRESS

C. Gary Jackson, M.D.

Good afternoon, and welcome to the 133rd Annual Meeting of the Otological Society. The first part of the program offers your President an opportunity to wax a bit philosophical, and that is what I will do. I will begin with some remarks that will, I hope, put things together for us as to where we are and what I think we can do to make some progress. My grandfather used to tell me that knowing something is wrong creates a problem. The problem is saying it out loud. The problem, out loud, is that medicine has collided head on with its moment in history. It is a profession in crisis.

Physicians have lost the meaning to the system. DeBakey has reminded us that in the last 10–15 years, the system has been subjected to the imperious intrusion of self-appointed overseers of health care reform, the theorists. The theorists have limited or no clinical experience, have not participated extensively in patient care, have evolved only a concept of managed care, and have used medical theory to make medical decision policy at a set physician compensation–all based on a hypothesis. As a result, medicine has undergone the most significant reprioritization of principles in its history. Control of medicine and health care has taken a back seat to business; the money changers are clearly ruling the temple.

As a medical community, we are left surveying the wreckage of what was once the best medical system in the world. In addition, the public has figured out that managed care is discounted fee for service, rationing, or service denial, and that something is dreadfully wrong. Our situation is this: Costs are increasing after a hiatus due to managed care, a health care crisis for employers is looming, and health care spending is about to take off in a tight labor market as the government mandates increased benefits and HMOs face increased accountability. Business will struggle to manage costs to retain workers by quality insurance options.

This crisis will likely come to a head with the next economic slowdown, as small companies opt out of providing insurance to employees and large companies shift costs to employees. The ranks of the uninsured will swell. Put simply, the country has ceased to exalt professional achievement–unless, of course, it is exhibited by a professional athlete, a movie star, or a corporate executive. Managed care advocates continue to argue that costs must be cut by decreasing physicians' salaries; they have enunciated a preferred place in health care for the generalist, and simultaneously they have worked out a redistribution of income to attract generalists. Not surprisingly, patients are in open revolt, critically assessing every aspect of care.

Quite simply, we are dealing with a new kind of patient. Because of the greater availability of information and the abuses consequent on managed care, the attitude of the new patient is different. There is a trend toward patient empowerment, as the system regularly acts to erode patient trust in itself and in doctors. There is an advocacy gap whose horizons are unknown. The economic, legislative, and strategic upheavals of the past decade have marginalized the fundamental work of physicians–to deliver health care–and have positioned

virtually every health care professional as a possible victim of an attitude of hopelessness that could pervade the entire system he or she influences.

The public has evolved to accept the generalist as a caregiver for purposes of prevention and minor medical monitoring. Once sick, however, the public demands the attention of a well-trained specialist. Patients simply want us to treat them, and they have rejected the hick for the generalist model. Access to care is a toothy issue. Marketplace competition and the dynamics of managed care have stifled the specialist community and the procedure-oriented subspecialties.

Organized practitioners of audiology have in several ways indicated their desire to expand their scope of practice to achieve unregulated access to patients, along the lines of the optometry model. The theorists currently mangling health care would grant audiologists unregulated access to patients, direct reimbursement for services, and membership on physician panels. Physician coalitions, on the other hand, are proactively proposing comprehensive positions on hearing health care that ensure the primacy of the physician. A reminder to us all that medical diagnosis should not be confused with mechanical tasks or with the legislative process. The lines in the sand have been clearly drawn, as we as specialists reject the cooperative organization that elaborates a common message and that redefines "us" and "them."

Here is my perspective: As this situation begins to unfold, health care is broken, and employers, employees, patients, and the government are brought to a stalemate relative to the increasingly organized medical presence. If the stalemate is not resolved, then, as Uvi Reinholt has cautioned, we may find ourselves the medical analogue of a rate-regulated utility. As one of my favorite philosophers has declared, predicting the future just isn't what it used to be. I don't know how this is going to end. But on this occasion I would like to suggest a beginning.

The circumstances in which we find ourselves in medicine are cosmically uninspiring. In pursuit of professional and emotional prosperity, we can be driven to counterintuitive places and solutions we might not otherwise resort to. Our responses cannot be antisocial. We must create something that enriches us all. Instead of inhabiting those counterintuitive places, we must spend more time in creative places, effecting change. Change heals progress; progress is changed with a purpose. Change for the better, however, can only be achieved through individual responsibility–that is, through a willingness to express and act upon what we each know about right and wrong.

All great battles are a series of scrimmages, and scrimmages are won or lost by the actions of individuals. As a culture, we have become far too enamored of government of a social, and a professional group sophistication centralized to cure all of our problems for us. In point of fact, however, a profession, like society, can flourish only when the nuclear unit–the physician–is secure. Change clearly is required to achieve that security to inspire. Change is intellectual. Change is slow, but it cannot be too slow, or the individual inspiration becomes bogged down, peptic and conservative–witness the Chicago Cubs in the 90th year of their rebuilding effort. George Will has suggested a concept of change that I like. He talks about changing one by one, from the inside out. Change that is achieved by living the littorals, with big consequences. Furthermore, unwritten rules are more important than those written. The latter we violate, with or without trifling consequences. The unwritten rules, when broken, disrespect the profession that gave rise to them and disgrace their perpetrator. As an example, a prime concern to me that represents one of those many points at which to begin the change and that constitutes as well one of those counterintuitive places to which some have been driven is a sporadically appearing trend in neurotology to abandon the cooperation between otologists and neurosurgeons in acoustic neuroma surgery.

From time to time I am made freshly aware of that. Neurotologists have operated on acoustic tumors with the input, expertise, and support of our partners for better than 40 years. This cooperation, I suggest, forms one of those unwritten micro-rules of professional behavior, the violation of which can have huge consequences. This battle was met, endured, and won by Bill House, and cast in stone by the excellence exhibited by his succeeding teams. Cooperation between neurotology and neurosurgery exists because it is teleologically right. It underwrites excellence. Once we disavow it, we are left to consider mediocrity–and in so doing generate mediocrity as an option.

For years, otologists criticized neurosurgeons who operated independently as irresponsible. It appears illogical and recidivistic to abandon the team concept. I can hear the neurosurgeons now as they toss back their accusations. But the one accusation that we must shoulder, the one accusation based in fact, is that we simply cannot care for all of our potential complications. Perhaps some practitioners become too comfortable in the danger zone of the

posterior cranial fossa by virtue of repeated success. Perhaps they have been driven to consider a counterintuitive position for convenience because of hospital dynamics or politics, or because cooperation has been made impossible by managed care shibboleths. The fact is, some form of team approach to acoustic tumor surgery constitutes the standard in most communities.

The new patient is discriminating and informed. Neurosurgeons now understand it. Not a week goes by in which a neurosurgeon doesn't call me and say, "I could do this procedure but I don't have a neurotologist." My heart leaps when they tell me that. It provides care of excellence. The tradition of cooperation has been forged in the blood, sweat, and tears of our predecessors, on the shoulders of their patients. It has come about through the science of health care, not because of some demand from the theorists. It should not be modified by nor consumed within the records we know now as managed care. This elegantly derived concept cannot be allowed to be aberrated by the theorists who cannot and will not understand the caring cooperation that is cost-effective. Excellence enriches us all. Excellence is inspiring, and inspiration can dissipate some of the hopelessness that afflicts the entire medical system. Ladies and gentleman, the ghosts of greatness that inhabit this society, this profession, and every one of us wouldn't have it any other way. So please, effect the change, inspire by excellence, and remember the unwritten small rules with big consequences: do the work. It is an individual prerogative. It begins with each one of us. And good luck. It isn't going to be easy. Thank you.

INTRODUCTION OF GUEST OF HONOR: DERALD E. BRACKMANN, M.D.

C. Gary Jackson, M.D.

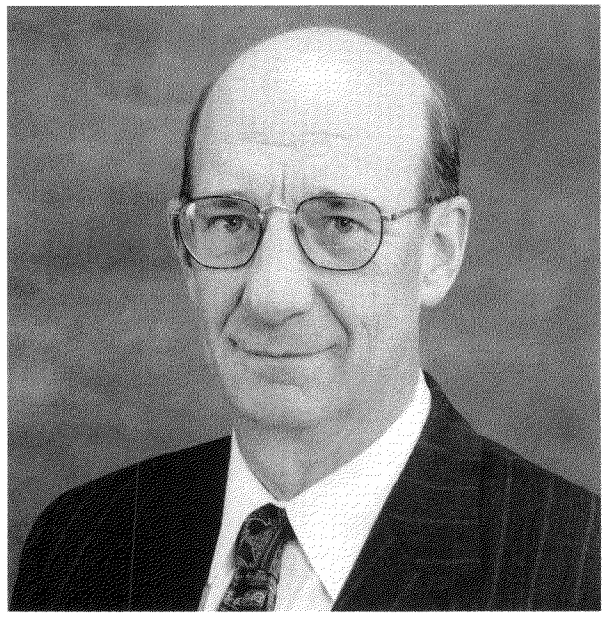
It is with great pleasure that I introduce Dr. Derald E. Brackmann, the 2000 American Otological Society's Guest of Honor. In 1970, upon completion of his training at the University of Southern California, Dr. Brackmann joined the House Ear Clinic, of which he is now president. He has led the specialty from his post as president of the world's leading societies, including the American Neurotology Society, the American Academy of Otolaryngology–Head and Neck Surgery, and the North American Skull Base Society. Most notably he is past president of the American Otological Society. Dr. Brackmann has contributed over 260 scientific papers and book chapters to the literature and has edited or co-edited numerous books. He is a sought-after guest speaker the world over, and his list of honors and awards is awe-inspiring. He is among the finest otologists in the world. Like fine artists, we microsurgeons are commonly accused of consigning our souls to our profession, at great cost to our personal lives. In contradistinction to this stereotype, Derald has maintained a balanced and capacious life. With Char, his lovely wife of 41 years, he has four sons, two grandchildren, and a wonderful family. He is an avid sportsman. If he's not on his boat fishing with his sons, he might be found hunting in the field. Although I can't vouch for the company he keeps, it is appropriate and just that the senior society acknowledge his continuing service to the AOS, and more broadly to the academic and clinical domains of otology and neurotology. I proudly introduce Derald E. Brackmann as the Guest of Honor for the 133rd Annual Meeting of the Otological Society.

REMARKS OF GUEST OF HONOR

Derald E. Brackmann, M.D.

Gary and I had not rehearsed at all. I didn't know what he was going to talk about. I had prepared just a few remarks, and I will keep them brief. As Gary has just described, many outside forces are impact-

ing on our enjoyment of our practice. But I present to you that nevertheless, medicine–and I will be even more specific and say otology–is the greatest profession in the world. Where else can you make a decent living (and we're all going to make a decent living!)–where else can you do that, help people, and at the same time get the great personal satisfaction that we all feel, the hugs and all the things that you all experience as I do? I've been very fortunate that when I go to bed Sunday night I can't wait to get up Monday morning and go to work. How lucky it is to be able to go through life and do that! Gary mentioned that Jens Thompson concluded a talk as the guest of honor at the ANS by showing a slide saying that being guest of honor is the beginning of the end. Of course, I can always point to Howard, who was guest of honor 40 years ago, and the end is nowhere in sight for him, so I won't take it as all bad. When you do get to this stage, you have a little bit of license to be philosophical. [*Dr. Brackmann shows a slide reading, "The worse day fishing is better than the best day working."*] As Gary said, while you are going through all of this, and no matter how much you love your work, never forget that slide. So go fishing, and take your kids with you. It's the greatest thing you can do for them.



Thank you.

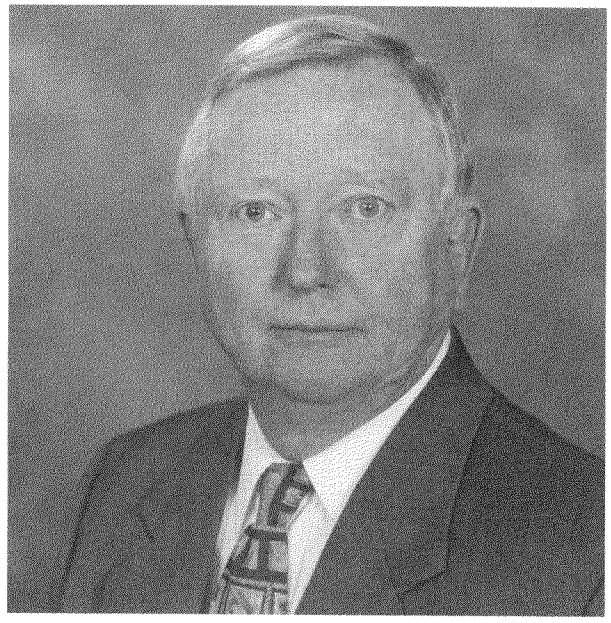
Dr. Jackson: On this occasion, Derald, allow me to present you with this certificate to commemorate it, and thank you.

Dr. Brackmann: Thank you so much, Gary.

PRESENTATION OF PRESIDENTIAL CITATION: WILLIAM B. WILLIAMS, ESQ.

C. Gary Jackson, M.D.

I next have the pleasure of awarding a presidential citation. William B. Williams joined his father-in-law, Harry Treece, at Richard's Medical nearly 30 years ago. Since then he has represented multiple companies that impact otology. It is, however, not these companies but the man I wish to cite. For 30 years, Bill has represented a capacity to serve this specialty in a manner that, but for the likes of Jack Urban, is virtually unparalleled. His simple, honest, and straightforward dialogue with doctors for 30 years has made him a trusted colleague at the ready to serve production, innovation, academic interchange, and an old school collegiality that has enriched otology scientific congresses for decades. It is therefore my privilege and great pleasure to present this presidential citation to acknowledge the ongoing career of service to otology to Mr. William B. Williams, my dear friend. Allow me, Bill, to present you with this certificate in commemoration of this occasion.



RESPONSE OF PRESIDENTIAL CITATION RECIPIENT

William B. Williams, Esq.

Thank you, sir. I'd like to thank Dr. Jackson. I'd like to thank Mr. Harry Treece for starting me off in this business, and I'd like to thank the two gentleman, Dr. Bev Armstrong and Dr. Ed Stevenson, who came down here because they pushed me off on this thing 28 years ago. Dr. Jackson, Dr. Brackmann, and Dr. Owens keep me going. Thanks to all of the otologists who have made it a wonderful career, and to my wife for letting me fly all over the world and making it go. Thank you.

FELINE IMMUNODEFICIENCY VIRUS–MEDIATED GENE THERAPY OF MIDDLE EAR MUCOSA CELLS

Hamid R. Djalilian, M.D., Yasuhiroz Tsuboi, M.D., Wesley Obritsch, and Jizhen Lin, M.D.

ABSTRACT

Hypothesis: To investigate the feasibility of gene therapy of the middle ear mucosa using a novel vector.

Background: Insofar as present medications are inadeqate to address chronic otitis media, cholesteatoma, or tympanic membrane perforation, newer methods of treatment for these diseases, such as gene therapy, need be explored. Genes could be used to alter cytokines in the middle ear, slow or stop cholesteatoma growth, or improve tympanic membrane perforation healing. Feline immunodeficiency virus (FIV), a new lentiviral vector, has been found to have greater than 90% efficacy in transfecting epithelial cells. Therefore, in vivo gene therapy of middle ear mucosal cells was attempted.

Methods: Twenty microliters of 107 vectors/mL FIV carrying the gene for green fluorescence protein (GFP) was introduced into the middle ears of Sprague-Dawley rats via a bulla approach.

Results: Expression of the GFP gene was observed in the middle ear mucosal cells, indicating transfection.

Conclusion: Gene therapy of the middle ear is feasible and has a potential human application in treating patients with chronic otitis media, cholesteatoma, or tympanic membrane perforation.

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ANALYSIS OF THE DYSFUNCTIONAL EUSTACHIAN TUBE BY VIDEO ENDOSCOPY

Dennis S. Poe, M.D., Ashraf A. Halawa, M.B.B.Ch., M.S., and Osama A. Razek, M.B.B.Ch., M.S.

ABSTRACT

Objective: Human eustachian tubes (ETs) with known ear pathology were inspected endoscopically and video recordings were made for slow-motion analysis of pathophysiology.

Setting: Ambulatory office in a tertiary referral center.

Subjects: Forty-four adults with 64 pathological ears.

Interventions: Transnasal endoscopic examination of the nasopharyngeal opening of the ET during rest, swallowing, and yawning to study ET dilatory movements.

Main Outcome Measures: Slow-motion video analysis of ET opening movements.

Results: Sixty-four pathological ears and ETs were studied. Tubal function was graded on:

1. Extent of lateral excursion and progression of dilatory wave as estimates of tensor veli palatini and dilator tubae muscle function. Reduced function was observed in 43 tubes.
2. Degree of mucosal disease, which was significant in 48 tubes.
3. Polypoid or other obstructive mucosal changes, present in 15 tubes.
4. Ease and frequency of tubal opening with maneuvers: 26 tubes opened moderately, 21 opened minimally, and 11 were unable to open.
5. Patulous tubes. All 6 clinically patulous tubes showed concavities in the superior third of the tube, which is convex in normals.

All tubes with active ear pathology (otitis media with effusion, tympanic membrane retraction, draining ear, cholesteatoma) had significant abnormalities. Correlation could not be made between the severity of middle ear disease and the severity of observed ET dysfunction.

Conclusions: Slow-motion endoscopic video analysis was a useful technique in classifying types of ET pathology. Additional studies of dysfunctional tubes are needed to predict outcomes in operative ear cases and to design intratubal therapy for chronically dysfunctional tubes.

Reprint requests: Dennis S. Poe, M.D., Zero Emerson Place, Suite 2C, Boston, MA 02114; ph. 617-725-3300; fax 617-725-2797. E-mail: dpoe@massmed.org

LASER STAPEDOTOMY WITH CONSERVATION OF THE STAPEDIAL TENDON

Rodney C. Perkins, M.D.

ABSTRACT

Objective: The objective of this study was to develop a procedure that allows the stapedial tendon to be conserved in the surgical correction of otosclerosis, and to assess the results. Conservation of the tendon theoretically should provide protection against noise trauma in this group of patients.

Study Design: Patients in whom the procedure was done were studied prospectively.

Setting: Surgery was performed in an ambulatory surgical center, with pre- and postoperative studies done in an outpatient clinic.

Patients: Patients who had clinical otosclerosis and who were candidates for surgery were selected for the study.

Interventions: Patients in the study group underwent laser stapedotomy with conservation of the stapedial tendon. The procedures were done under local analgesia on an outpatient basis.

Main Outcome Measures: Audiometric improvement in hearing and maintenance of stapedial reflex on impedance audiometry were assessed. Air conduction, bone conduction, and speech discrimination testing and impedance audiometry were performed pre- and postoperatively.

Results: Audiometric results were comparable with results in control patients who had undergone conventional laser stapedotomy with vaporization of the stapedial tendon. The stapedial reflex could be demonstrated postoperatively in the study group. There was no evidence of adverse effect, increased cost, or significantly increased surgical time, and there was no increase in morbidity.

Conclusion: The technique provides a method for conserving the stapedial tendon in patients undergoing laser stapedotomy for otosclerosis. In these patients it is expected that the protective function of the stapedial reflex will be maintained.

Reprint requests: Rodney C. Perkins, M.D., CA Ear Institute, 801 Welch Rd., Palo Alto, CA 94304-1611; ph. 650-494-1000; fax 650-323-2365.

CURRENT USE OF IMPLANTS IN MIDDLE EAR SURGERY

Robert A. Goldenberg, M.D., and John R. Emmett, M.D., F.A.C.S.

ABSTRACT

Hypothesis (Objective): Members of the American Otological Society (AOS) and American Neurotology Society (ANS) were surveyed regarding their use of currently available prostheses for ossiculoplasty and stapedectomy. The results were compared with findings of a similar study conducted by one of the authors in 1989.

Methods: Questionnaires were sent to the entire membership of the AOS and ANS with questions regarding biomaterial and prosthesis usage for stapes and chronic ear surgery, as well as satisfaction with each type of prosthesis used. Of the 575 questionnaires mailed, 274 were returned (47%). Only 248 of the respondents performed middle ear surgery (43%), and their responses constitute the database for this study.

Results: For those respondents who performed stapes surgery in both 1989 and 1999, the mean number of cases per year increased from 32 to 37 (P = 0.004). The mean number of chronic ear cases also increased, from 95 in 1989 to 110 in 1999 (P = 0.001). As a biomaterial, hydroxylapatite prostheses are used by most surgeons (82%), followed by autograft and homograft bone (72%), autograft and homograft cartilage (62%), and Plastipore® (59%). (Although 62% of respondents use cartilage, only 4.4% ranked it first in preference.) In 1989, bone was used most often (93%), followed by cartilage (78%) and Plastipore (81%). Hydroxylapatite, which had just been introduced as a biomaterial, was used by only 9% of respondents. For stapes prostheses in 1999, the majority of respondents used stainless steel/platinum (71%), bucket-handle (69%), or partial fluoroplastic (56%) prostheses. Overall satisfaction with most of these prostheses was high (>85%), with several exceptions. The lowest satisfaction rate was 71% for Plastipore PORP and TORP. Usage and satisfaction rates are presented for specific types of implants and compared with the earlier survey findings.

Conclusion: The current use of implants in middle ear surgery demonstrates a specific pattern with a high degree of user satisfaction. Respondents' preference for implants has remained stable over the past ten years; there has been a decrease in the percentage of use of bone, cartilage, and Plastipore and a corresponding increase in the use of hydroxylapatite.

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DISCUSSION PERIOD I: MIDDLE EAR/MASTOID
Papers 1–4

Dr. C. Gary Jackson (Nashville, TN): These papers are now open for discussion.

Dr. John Shea, Jr. (Memphis, TN): I congratulate Dr. Poe for this excellent presentation. It is important work that teaches us what is going on in the eustachian tube, something I am fascinated with. I certainly want to come see Dr. Poe's work and begin to do it myself. It is really Dr. Perkins's paper that I want to comment on. I applaud the fact that he has used vein interposition in a clever way. Rod has always been a very clever person, and I congratulate him. At the American Otological Society meeting last year Dr. Causse presented a series of papers on preserving the stapedius tendon. As you know, we are physicians, practitioners of physics, and the physics of the ear are that the stapedius tendon contracts the stapes in the oval window and is jammed in there, and unless you preserve the stapes footplate in the oval window, there is no reason to preserve the stapes tendon. It is interesting that Rod ends by saying only that you preserve the circulation, and that is good, but to say that you protect the physics of the inner ear is just not so. It is interesting that he made no such claim. So, Rod, I enjoyed your paper, but I am glad you never claimed that the physics of the ear is better when you preserve the stapedius tendon, because unless you have the oval window of the footplate joint in place, the stapes tendon doesn't do anything.

Dr. Jackson: Rod, do you have a comment?

Dr. Richard Bellucci (New York): I'd like to say a few words about Dr. Poe's paper. I have been interested in eustachian tube function with regard to middle ear infection for many years. He's shown some very interesting views of how the tube opens, but not anything about the etiology, and he admits that he has to do some more work on it. Basically, I believe that outside of this function between the two muscles that he shows, the fifth nerve activates the middle ear muscles as well as the eustachian tube muscles. I think there is a sequence that follows in swallowing between the eustachian tube muscles and the middle ear muscles; however, I feel that the basic problem in eustachian tube function is the anatomy of the nasal pharynx. As we see in cleft palate, the muscles are attached poorly, and therefore the function of the two muscles varies. There is a gradient–I have published on this topic before. I think that what has to be shown is this type of dysfunction with relation to the anatomy of the nasal pharynx and cleft palate in particular. That will be revealing in trying to determine the etiology of eustachian tube malfunction.

Dr. Jackson: Thank you. George?

Dr. George Lesinski (Cincinnati, OH): I wish to echo Dr. Shea's comments regarding Dr. Perkins's very elegant and meticulous surgery. The physics would not allow a sound protection mechanism from the stapedius tendon, at least as I understand it. I would like to comment further. We have an ongoing study that now comprises in excess of 300 stapedectomy revisions undertaken because of hearing failure; in 78% of those patients the procedure failed because the prosthesis migrated out of the fenestration and into the vestibule and became fixed against the solid otic capsule bone or against the solid fixed stapes bone. In careful analysis and video documentation, one of the most common reasons the stapes prosthesis migrates apparently is because the collagen that is used as a seal in the healing process contracts and over time tends to lift the prosthesis out of the stapedotomy opening, giving it an opportunity then to migrate. And it will migrate, on the basis of adhesions that contract, or it may migrate because of the angle at which the prosthesis enters mechanically if it is not directly perpendicular. Moreover, in consequence of that, in the vast majority of these cases–perhaps 75% of the fixed prostheses–we are seeing at least partial erosion of the incus. The partial erosion invariably occurs on the undersurface of the incus. As the incus vibrates against this fixed prosthesis–the biological living bone vibrating against a fixed inner material, whatever the material–we begin to see erosion of the bone, just as we would anywhere else in the body. So, for these reasons, I have been attempting to create a very precise, 0.6-mm opening when I do the stapedotomy and not use a collagen tissue seal but rather clotted blood, and that I believe is safely possible to do only if you can create a round, symmetrical, perfectly sized 0.6-mm opening.

Dr. Jack Pulec (Los Angeles, CA): I wish to compliment Dr. Poe on the quality of his photographs and video recording. This is a very technically difficult thing to accomplish. I was very pleased to see Dr. Poe's work; it is one of the first times in a long time that we have seen great interest and progress in this area. Very few people can even make the diagnosis of an abnormally patent eustachian tube. It is a severely missed or undiagnosed problem. I'm certain Dr. Poe's major accomplishment here is the technique, the details of what causes serous or chronic otitis media, a totally different part of this study. With a thin, atrophic mucous membrane, the fat does tend to show through; in many cases the fat is missing, so that is part of the problem as well. I congratulate you, Dr. Poe, and remind the audience what a great series of pictures those were.

Dr. Rodney C. Perkins (Palo Alto, CA): I would like to comment on Dr. Shea's and Dr. Lesinski's comments. As Dr. Shea pointed out, we have no evidence that this is protective over the long run. I didn't make any claim for that, and I don't think any claim should be made. The best recent work on this topic has been done at MIT, by Pang, Peak, and Gillian. They have shown that the effect of the stapes reflex is mediated through a stiffening of the annular ligament. This allows the low-frequency masking to be decreased, and hearing is better in the high frequencies. They also found that the stapes was the only thing that moved in the stapedial reflex. When they tried to measure incus movement and malleus movement, they didn't get that. I believe that is true, and probably because there is a stapes there. One might speculate that if there were no stapes footplate attached to that, would part of that be damaging to the incus. I submit that probably some of it would, but that is not the way it is mediated in the normal situation.

One of the things I'd like to do before I forget a second time is to thank my co-authors, who were not mentioned on the slide: Dr. Catrina Stidern, who is a fellow with us and is going into practice in California, and Dr. Yoon, who helped prepare the paper.

With regard to Dr. Lesinski's comments, I agree with Dr. Shea that the vein seal in the fenestra is important, and I know Dr. Causse has also championed this. I also agree, though, with Dr. Lesinski that the method by which the prosthesis ejects is probably the tightening of a vein under the piston. It's the same mechanism by which a tympanic membrane will lateralize if it is cup-shaped when it is put in. It contracts, and there is no force on it pulling out.

In the first few hundred laser stapedotomies we used a blood seal and it worked quite well, so I believe others would find it works fine as well. I expect we will continue the study in a little more detail with audiometric studies of sound and noise, and I hope we will have a further report later.

VIBRATORY SAMPLE MAGNETOMETRY OF STAPES PROSTHESES TO ASSESS MR SAFETY AND COMPATIBILITY

Mark J. Syms, M.D., and Derrick W. Peterman, Ph.D.

ABSTRACT

Objective: To assess the ferromagnetivity of stapes prostheses using a vibratory sample magnetometer (VSM).

Data Sources: Previously, stapes prostheses from different manufacturers were placed in a 1.5-tesla MRI field to determine their ferromagnetic properties. Two series of Xomed prostheses were found to be ferromagnetic. VSM was performed on 16 samples, including ferromagnetic 420F stainless steel. VSM testing was performed using an LDJ model 9600 VSM, in accordance with American Society for Testing and Materials standard A894.

Results: A VSM measures the magnetic dipole moment of a sample in a magnetic field. The magnetic field is swept over a range of magnetic fields, and the magnetic dipole moment is plotted as a function of field. In a ferromagnetic material, the dipole moment plot demonstrates hysteresis. The samples made with 316L stainless steel, which is used in otological implants, are fairly nonmagnetic relative to the 420F stainless steel. The torque and linear force on the prosthesis in a given magnetic field can be calculated from the results of VSM.

Conclusion: On VSM, prostheses made with 316L stainless steel were relatively nonferromagnetic when compared with 420F stainless steel. The forces acting on a prosthesis in a given magnetic field can be calculated using VSM. The safety of performing MRI in patients with these implants needs to be reassessed.

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MIDDLE EAR PROSTHESIS DISPLACEMENT IN HIGH STRENGTH MAGNETIC FIELDS

Michelle D. Williams, M.S.4, Patrick J. Antonelli, M.D., F.A.C.S., and Lorna Williams, M.D.

ABSTRACT

Hypothesis: Middle ear prostheses made from nonmagnetic, magnetic resonance (MR)-compatible metals reportedly displace ex vivo in the presence of high magnetic fields used in MR imaging. We postulated that the prosthesis displacement seen with nonmagnetic, MR-compatible prostheses ex vivo may not be clinically significant in vivo.

Methods: Middle ear prostheses made from ferromagnetic (420F stainless steel) and nonmagnetic MR-compatible metals (316L stainless steel and platinum) were examined for magnetic field interactions at 4.7 tesla (T). Ex vivo testing consisted of measurements of the translational and rotational motion of the prosthesis induced by the static magnetic field. In vivo testing entailed implanting prostheses in cadaveric temporal bones and performing clinical MR sequences. Prosthesis displacement was measured semiquantitatively.

Results: Angular deflection was observed in all samples made from nonmagnetic stainless steel. The negative control (platinum) demonstrated no deflection, and the positive controls (ferromagnetic stainless steel) deflected more than 90 degrees. Torque analysis showed movement in five of five nonmagnetic stainless steel prostheses. Prostheses made from nonmagnetic stainless steel remained in place without appreciable loosening in vivo following MR imaging. Prostheses made with known ferromagnetic properties were displaced at 4.7 T but not at 1.5 T.

Conclusion: Middle ear prostheses made from low-magnetic stainless steel do move in the presence of high magnetic fields ex vivo; however, this does not appear to be clinically or statistically significant in vivo at 4.7 T. MR imaging should be undertaken with caution in individuals with prostheses made from stainless steel with strong ferromagnetic properties.

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PROGNOSTIC FACTORS IN OSSICULOPLASTY: A STATISTICAL STAGING SYSTEM

John L. Dornhoffer, M.D., and Edward K. Gardner, M.D.

ABSTRACT

Objective: To determine factors that predict hearing results using a standard prosthesis system.

Study Design: Retrospective chart review.

Setting: Tertiary referral center.

Patients: All patients undergoing ossiculoplasty with the Dornhoffer HAPEX Partial and Total Ossicular Replacement Prostheses (PORP and TORP) from February 1995 to May 1999 who had documented postoperative follow-up and no congenital atresia or stapes fixation. A total of 185 patients (200 ears), 105 men and 80 women, were evaluated.

Intervention(s): Ossiculoplasty with the Dornhoffer prostheses.

Main Outcome Measure(s): Hearing results using a four-frequency pure-tone average air-bone gap (PTA-ABG) were measured. Multivariate statistical analysis determined the effect of mucosal status, ossicular chain status, and type of reconstruction techniques on hearing.

Results: PTA-ABGs were 13.4 ± 8.1 dB and 14.0 ± 8.4 for the PORPs (n = 114) and TORPs (n = 86), respectively, which was not statistically different. When the malleus handle was present (n = 126), the PTA-ABG was 11.6 ± 6.2 dB, compared to 16.9 ± 10.1 dB when it was absent (n = 74), which was statistically significant ($P < 0.05$). Mucosal fibrosis, drainage, revision ear surgery, and type of surgical procedure had a significant detrimental impact on hearing. The type of pathology (perforation versus cholesteatoma) had no significant impact on hearing results.

Conclusions: The revised staging system, the Ossiculoplasty Outcome Parameter Staging (OOPS) Index, more adequately predicts hearing outcome in our series of 200 cases.

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DISCUSSION PERIOD II: MIDDLE EAR/MASTOID
Papers 5–7

Dr. C. Gary Jackson (Nashville, TN): This series of papers is now open for discussion.

Oats (United Kingdom): Our radiologists are worried about the possibility of heating the prosthesis in an MR field. Can you address that issue?

Dr. Mark Syms (Honolulu, HI): They're right. Heating is a big concern, and as the fields get stronger, it will become an even bigger concern. It is not just gross displacement of the prosthesis that will be a concern but the very small, quick, back-and-forth movements. One of the problems with a stapes prosthesis is that the FDA considers it a static implant, similar to an aneurysm clip, in terms of evaluation. But it is actually meant to move, so it is a unique implant, different from other kinds of metallic implants.

Michelle Williams (Gainsville, TX): Previous studies done on small prostheses have indicated no heating when the prostheses were subjected to long trials within an MR machine, nor was there any magnetism induced in the small prostheses. There have been no reports of vertigo in patients with middle ear prostheses, as might occur if there was heating within the middle ear.

Dr. Charles Luetje (Kansas City, MO): I have some practical concerns with regard to the migration of these metallic prosthetic devices. In the office, not infrequently we are called from the radiology department. The radiologists say, we need to have data about the implant you did on this patient (it's usually a stapedectomy)–do you have the number or the catalog number, etc., because otherwise we will not do the MRI. I have told the radiologists to go ahead and do the study, it won't hurt anything. Maybe I shouldn't say it, but I do. Sometimes they want documentation and sometimes they don't. I know that there are no catalog numbers left for certain of the tantalum wire prostheses. I know some of the Robinson prostheses were molybdenum, nickel, and so on. The in vivo studies look pretty good. Can I continue to tell the radiologists not to worry about it?

Dr. Jackson: Some of our radiologists have told me they would do an MRI study in a patient with a one-sided stapedectomy but not in patients with bilateral stapedectomies, so I would like some guidance as well.

Dr. Rick Chole (St. Louis, MO): Perhaps this is an unfair question to ask in the current medicolegal environment, but has anyone here ever had a stapedectomy patient with a serious complication because of an MR machine?

Dr. Jackson: Can we have a show of hands from any who have? (*No hands were up.*)

Dr. Chole: I think that pretty well answers the question. I hate to be unscientific about it, but. . . .

Dr. Syms: Actually, there are two issues. One is that most people are currently using 1.5-T MR fields, and the scanners are getting stronger. The boutique scanners use 3-T magnetic fields, and in Great Britain 8-T machines are being tested. The problem is not whether they are safe now, but whether they will be safe 20 years from now when patients are undergoing this type of evaluation.

The FDA has a working paper asking that the safety of the prosthesis be specified within the magnetic field in which it was tested. In other words, the documentation has to say, This prosthesis or implant can be safely scanned in a 1.5-T field. It's a working paper–the regulation hasn't been adopted yet–but that is the direction the FDA is moving in with regard to certification of MR safety and compatibility.

Dr. Doug Backous (Seattle, WA): I think we are looking at it backwards. I was called by a radiologist because a patient who underwent MRI of the shoulder had a stapes prosthesis and claimed that her head heated up. As it turned out, she had two or three psychiatric diagnoses, but when it came time for the attorneys, the question was not whether this prosthesis heated up or moved. It was proved to us that it didn't, but the problem is, that can lead to a settlement, which leads to a precedent, which is a problem, so we exercise lots of caution. I think it's very ambiguous.

CHOLESTEATOMA: CANAL WALL UP, CANAL WALL DOWN?

PANEL DISCUSSION I

Moderator: Bruce Gantz, Iowa City, IA

Participants: Paul Lambert, Charleston, SC; Joe Nadol, Boston, MA; and Simon Parisier, New York, NY

Dr. Bruce Gantz: May I introduce Paul Lambert, from Charleston, South Carolina

Dr. Paul Lambert: Thank you very much. I am delighted to be part of this panel as we explore a subject that continues to elicit as much controversy as it did when I began my training, nearly 25 years ago. As I present some thoughts on cholesteatoma surgery, my goals will be twofold: first, to discuss some concepts regarding intact canal wall and canal-wall-down mastoidectomy, and second, to discuss my experience with these procedures in treating pediatric cholesteatomas.

The advantages of an intact canal wall procedure are well recognized and include preservation of normal anatomy, faster healing, and fewer long-term care issues. An intact canal wall procedure also facilitates use of a hearing aid, which is often needed in this patient group. There is a price to pay, however, in the increased incidence of both residual and recurrent disease. Inadvertently leaving a small focus of squamous epithelium behind is a distinct possibility, given the greater technical problems with this procedure as well as the diminished exposure. Maintaining the canal wall intact provides spaces in which retractions can occur, and thus also recurrent disease. This recidivism is not a trivial problem, particularly in children.

In contrast, removing the canal wall essentially eliminates the problem of recurrent disease from retraction pockets, and the improved exposure greatly lessens the incidence of residual disease. The accumulation of squamous debris and the possibility of infection, however, must always be borne in mind, and hearing aid use can be more problematic.

The problems of residual and recurrent disease are particularly important in children, in whom rates tend to be significantly higher than in adults. Why is this? Is the biology of a pediatric cholesteatoma somehow different?

There are several factors to be considered. First is the physical parameters of the disease. Children often have well- pneumatized temporal bones with deep cell tracks, and this can complicate complete disease removal. Adults may have a sclerotic mastoid secondary to childhood infections. A second factor–and this may be the most important–is the poor function of the eustachian tube in children, which predisposes to otitis media and secondary infection of the cholesteatoma, thus promoting a more aggressive disease process. It also predisposes to retraction pockets, and thus recurrent disease. Also to be considered is that the potential for tissue growth in children is greater than in adults, owing to the normal elaboration of various growth factors.

Several years ago we published a series of pediatric cholesteatomas. The average age was 10 years. Follow-up lasted from 1 to 12.5 years (average, 3 years). I prefer to perform an intact wall procedure for all cholesteatomas, and in this particular series of children, it was the initial procedure in 70% of those needing a mastoidectomy. Some patients were treated with middle ear exploration only. It is also my practice to stage these ears, returning in 8–10 months to perform the ossiculoplasty and to check for any residual disease. Even though the intent was to maintain the canal wall intact, almost one in six patients did need conversion to a canal-wall-down procedure, and 15% of the patients required a third procedure because of persistent disease. The overall incidence of residual and recurrent disease was about 40%, a figure consistent with what has been published in the literature. In 19% disease was left intentionally–for example, around an intact stapes that was to be removed in a second stage. In most of the patients with recurrent disease the procedure was converted to a canal-wall-down procedure. In patients initially treated with a canal-wall-down procedure, staging was common, and only one patient required a third procedure. In the canal-wall-down group, recurrent cholesteatoma from a retraction pocket did not occur, and the rate of residual cholesteatoma was only 12%.

What about hearing? Some believe that hearing results are better when the canal wall is left intact; however, that is disputed in the literature. It was

not our finding in this particular series. Instead, hearing depended more on whether there was an intact stapes or whether only a footplate was available for reconstruction. In a review of 17 published studies (comprising almost 1,500 patients) on pediatric cholesteatomas, the cumulative rate of recurrent and residual disease was 42% with an intact canal wall, and about half that when the canal wall was removed.

Even though I prefer to perform an intact canal wall procedure, there are certain situations in which a canal-wall-down procedure may be preferred, namely, a single hearing ear when follow-up is problematic, or if the patient is a poor anesthetic risk and only one surgery is desired. Intraoperatively, the findings of a small mastoid or horizontal canal fistula or significant erosion of the canal wall may also lead to a canal-wall-down approach. Significant erosion of the posterior and superior canal wall can be repaired with cartilage or bone. Repair with tragal cartilage is a very satisfactory way to deal with this problem.

In conclusion, in this series of pediatric patients, the majority were managed with an intact canal wall procedure, and 84% of patients so treated achieved a disease-free state during the follow-up period while maintaining the canal wall intact. We recognize that an intact wall approach will necessitate more operations to completely eradicate the cholesteatoma, but it is my belief that the extra morbidity and cost, when averaged over many decades of life, is justifiable.

Dr. Gantz: Next is Dr. Joe Nadol, from Massachusetts Eye and Ear Infirmary, who has a completely different way of managing cholesteatoma.

Dr. Joe Nadol: Thank you, Bruce. I'm pleased to be invited. Bruce asked me to give a short summary of how to decide between a canal-up and a canal-down procedure. I think he was assuming that most of the cases we do are canal wall down, and in fact that's true. Most of the procedures I do are revision surgeries, and that probably biases me. The reasons for choosing an open cavity or canal-wall-down technique are, obviously, to revise a previous canal-wall-down procedure, to treat recurrence with loss of integrity of the posterior canal wall (which Paul just mentioned), and for exposure purposes, mainly in the case of a large cholesteatoma on a small mastoid. If the dimension of the mastoid from the lateral venous sinus to the posterior wall is about the same as the dimension from the posterior wall to the anterior wall, I would consider that a small mastoid. Finally, if the patient seems to have chronic eustachian tube dysfunction in both ears, I tend to use the canal-wall-down technique.

The technique is very straightforward: a postauricular incision is made, followed by the development of an inferiorly based musculoperiosteal flap (I do this canal up or canal down, either to reconstitute the lateral cortex or to use it to cover an obliterative material). The flap is elevated and the mastoidectomy is done. When obliteration is done for the canal-down technique, I almost always use bone paté, which is collected from the lateral mastoid cortex at the beginning of the procedure using a Sheehy collector.

Over the years I have learned much from revision surgery, and especially why the first operation tends to fail. This is one of our least successful procedures–certainly much less successful than, for example, stapes surgery. It is not always the cholesteatoma that is the problem. It may be mechanical factors, something simple, such as a high facial ridge in canal-wall-down surgery, or a very poor meatoplasty preventing cleaning of the mastoid bowl, followed by residual or recurrent granulation tissue in predictable areas–tegmental cells, sinal dural cells, tip, the facial recess, and the hypotympanum. In a certain sense, it is harder to do a good canal-wall-down procedure because it is more important to eliminate as many cells as possible to prevent recurrent disease. The areas that tend to cause trouble in the canal-wall-down technique are the residual tegmental cells and sinodural cells; another area that is particularly problematic is the so-called hypotympanic or infralabyrinthine cell tract.

I was trained to respect the middle ear mucosa, and it was generally prohibited to do much in the way of drilling in the middle ear. The fact remains, however, that in a number of patients I have seen with recurrent disease, the recurrence is exclusively (or almost exclusively) in the hypotympanic cells. This can usually be determined with CT. Isolated hypotympanic cell or infralabyrinthine disease occasionally can even erode into the membranous labyrinthine. How well do we do with these cases? We conducted a study about 3 years ago in which we looked at 272 patients, most of them with cholesteatoma, some without, and followed them for a minimum of 12 months (mean of 30 months) to see how we did and what factors determined success. Most of the procedures were revision procedures, which is a characteristic of our practice, and the majority were canal wall down because of that. We used a grading scale for evaluations: 0 for complete cure and 3 for total failure (continued daily otorrhea); grades 1 and 2 represented episodes of otorrhea of increasing duration that could be managed medically and did not require revision surgery. Of the 272 patients, about 55% had a dry ear that

stayed dry through the period, and another group had recurrences but did not require revision surgery. Interestingly, in those without cholesteatoma it was more difficult to achieve a dry ear. This was statistically significant in the cholesteatoma patients, who did a little bit better than those without. The outcome was not influenced by primary ear revision surgery with the canal up or down, by the extent of the cholesteatoma, or even by the extent of the granulation tissue. This remains one of our least successful procedures and leads to many revisions.

Dr. Gantz: Thank you, Joe. Finally, Simon Parisier, of Lenox Hill, New York City, will address the group.

Dr. Simon Parisier: Bruce asked me to talk about suitable cases for canal-wall-up or canal-wall-down procedures. I individualize the decision, and although some of the decision making is done preoperatively, basically it is an intraoperative decision. In trying to decide whether I will leave the canal wall up, one of the factors I am concerned with is recurrent cholesteatoma, or cholesteatomas that result from the formation of retraction pockets, either because of poor eustachian tube function or because of other unknown factors. It is very difficult for us to assess eustachian tubule function. We know how to work in the nasal pharynx, we know how to work in the ear, but we really don't have anything that tests or addresses the eustachian tube. Clinically, I look at these ears and I look at the pars tensa, to determine whether it is normal, and then I look at two other factors–the middle ear mucosa and the size of the mastoid. I will illustrate with the case of a person who has a pars flaccid, or retraction with an attic antral block. (The illustration is from Brackmann's Atlas, so I'd like to thank him.) The middle ear is clear. But there is aeration of the middle ear and disease in the epitympanum, and that fact would lead me to want to preserve the canal wall, reconstructing the damaged part. If I enter an ear and it has a mucosa that resembles what I encounter doing a middle ear exploration for otosclerosis, that is a very favorable sign that the eustachian tube ventilation is normal. If there is middle ear effusion and the eustachian tube is not working functionally, in some of these ears I will put a ventilating tube.

I look at the size of the mastoid. This is a small epitympanic cholesteatoma in an otherwise pneumatized mastoid. The cholesteatoma is abutting the lateral semicircular canals, so it is doing some damage, but this ear would lend itself well to a canal wall procedure.

The indications for performing a canal-wall-down procedure include ears that are poorly ventilated and inadequate operative exposure. I prefer to do procedures as one operation, with reconstruction performed simultaneously with ablation of the disease, and if regrowth occurs, I consider the procedure to have failed. I don't go back for a second-look procedure. If the eardrum is retracted so that I have an epitympanic defect but a retracted pars tensa, or if there is squamous epithelium lining the middle ear space, I will take the canal wall down. I don't think that these ears will be ventilated properly, and I am concerned that there will be recurrent disease in ears with poor eustachian tube function.

Hyperplastic polypoid middle ear mucosa is a very poor prognostic indicator. In ears with a very thickened lamina propria obliterating the middle ear space, if I have to peel off the middle ear mucosa, the likelihood of having normal mucosa growing back is problematic. If the mastoid is really sclerotic, such that the ear really has an ossified antrum, I would perform a canal-wall-down procedure.

We looked at the treatment of congenital cholesteatoma in children; the average was 4 years (range, 1–12 years). A canal-wall-down procedure was performed in 17%. Facial recess approach closed procedure in 7%, the remainder of these ears could be handled with a tympanotomy. In acquired pediatric cholesteatoma (216 ears), the cholesteatomas were either acquired primarily or following a previous surgery. A closed canal-wall-up procedure was performed in 52% and an open canal-wall-down procedure was performed in 48%.

I believe that the cholesteatoma should be removed completely at the initial operation, even if it involves the stapes footplate or facial nerve. With regard to recidivism, insofar as the follow-up of these young patients isn't perfect, we have adopted the Kaplan-Meyer statistical analysis for survival that is popular with head-and-neck cancer surgeons. In a study of cholesteatoma follow-up in adults, recidivism following all procedures plateaued at about 10 years, because of the recurrent cholesteatomas. Residuals only occurred out to 3 years. In pediatric cholesteatoma there is a similar curve that plateaus at 10 years, but it is a little bit higher. When adult cholesteatoma data are analyzed by canal-wall-up as opposed to canal-wall-down procedures, regrowth occurred earlier, and surprisingly, there wasn't that much difference. The pediatric cholesteatoma data show that the canal-wall-down approach results in a 15% regrowth rate that plateaus at about 4 years, but the recurrent cholesteatomas continue up to about 40%, and they can occur as late as 10 years postoperatively.

Dr. Gantz: I have had difficulty for 17 years in

trying to decide between canal wall up and canal wall down. Like Simon, I showed a lot of my canal-wall-up decisions, the canal walls came down, and I was frustrated. Even after putting large pieces of cartilage in the posterior-superior quadrant, I would find that the eustachian tube didn't function, and eventually the retraction would go underneath the cartilage and would start to accumulate debris, and we would have to take the canal wall down. We had to do that in more than 50% of children we treated at Iowa. At the 1976 Cholesteatoma Conference I heard a presentation by a Swedish group that described 122 cases with a 5-year follow-up and no recurrent cholesteatomas. Residual disease at 5 years was zero, there were only three perforations, there were no retraction pockets, and ears were dry in 100%. The 10-year results from this group were similar. I tried to do the procedure. The highlights of it are a complete mastoidectomy, with a lot of the steps that others did before the Swedish group, such as use of an extended facial recess and collecting the paté with the Sheehy bone paté collector. The skin of the posterior canal wall is elevated forward. No incisions are made in the posterior canal wall. Remove the posterior canal wall (as the Wulsteins used to do with the microsaw), get all of the cholesteatoma out, and you have a canal wall down at that time. Then you put a Silastic spacer in and come back in a second stage for reconstruction, during which you replace the posterior canal wall. You block the attic with bone chips that you take with a chisel, and then you fill the mastoid with bone paté. The objective is to get rid of all of the mucosa in the mastoid, which is very problematic because it resorbs nitrogen. What I demonstrate here is after you've done a complete mastoidectomy, using a nasal chisel as you would a plane to take some very thin bone chips. The canal skin is taken and lifted forward before you cut out the canal wall. Then, with a reciprocating saw–this is a Storz microsaw, but you can use a handpiece on a Fisch drill and do the same thing–you cut out the canal wall so that it is at an angle, so that when you place it back it will not fall back in the mastoid. Now you have a canal-wall-down situation, as you have taken out the piece of bone, and you can do a very thorough job of removing the cholesteatoma. You take a large piece of fascia. You put a Silastic spacer in the middle ear. If you don't have a stapes, you put another piece in there in the oval window, and you put the Silastic spacer on top of that and then use a big piece of fascia to go all the way underneath the tympanic membrane and up the canal wall where you've made your cuts. This fascia goes outside of where you've made the canal wall cuts. You line with bone chips up to the attic so that the bone paté does not go into the middle ear space. Then you fill the mastoid with bone paté, you put the posterior canal wall back, and you use a cottel speculum to see all the way down to the tympanic membrane. Nu gauze and bacitracin are placed in the ear canal and left for 1 week. I have drained the mastoid with a Penrose drain for 48 hours and we have run the bone paté through aqueous bacitracin to try and reduce the chance of postoperative infection. This is an x-ray of one of my patients who complained of some ear pain, but you can see that this bone fills in, the posterior canal wall is in good shape, and in this situation you have the bone all the way up to the cortex.

These results were tabulated by Paul Canti, our fellow, and one of our residents, Marlin Hansen. We have treated 60 ears this way since January 1997. Our follow-up is only 26 months, so our results are very early and preliminary. One of the problems was that 13% had postoperative infection for which they had to be rehospitalized and given IV antibiotics. All of the infections cleared with antibiotics. We had no loss of posterior canal wall. In one patient a partial resorption occurred, but the posterior canal wall is intact. Of the 60 ears, we have looked at 47 so far. Two had a pearl in the oval window that we removed. The posterior canal wall retractions and the posterior-superior quadrant are zero. We had two that had perforations after the first procedure. We repaired them, and they are now healed. All of these ears are dry, meaning they don't have to be maintained and the patients don't have to make clinic follow-up visits. We will continue to follow these patients.

How did we do with hearing? We are not doing as well with hearing. We looked at our patients preoperatively, 0–10 Db, air-bone gap, 11–20 Db, 21–30 Db, and greater than 30 Db. Postoperatively we are reducing these figures, but we are still having significant air-bone gaps. The air-bone gaps in these 37 patients are evident from these audiograms, which are the most recent audiograms obtained, some only 3 months postoperatively and others a year postoperatively. These results are not as good as have been reported. We are not putting tubes in these ears, we are not trying to re-aerate them. This is just the disease process of the eustachian tube dysfunction again, and it's probably the reason. The advantages here for me are improved access and removal of all the disease. I think we try to remove all of the mucosa and get rid of it so that we don't have the negative resorption of nitrogen. Long-term debridement is not necessary. It's a one-type procedure for all comers, and I don't have to

make a decision. This is a big problem for me right now, because it is still a two-stage procedure. So that is another alternative. I will continue to follow these patients. We do not yet have long-term results, but when I was doing canal wall ups and following the patients, I was taking down a lot more at this time period.

Now, some questions for our panelists. Simon discussed his preoperative parameters for determining which procedure to use. Joe, what procedure do you perform if you have a virgin ear that has not been operated on before?

Dr. Nadol: I would predict a canal-wall-up procedure–and almost always do it–when I encounter an entity that I call chronic inactive otitis media with frequent reactivation. That is an ear without cholesteatoma. It is an ear with a perforation that drains intermittently; in most cases there is an attic block. For me, that is the perfect case for a canal-wall-up procedure, which can almost always can be done. In general, on the first time through, I approach these cases as a canal wall up. The circumstances that will make me go to canal wall down we have already talked about.

As to the mastoid cavity, I do not believe there is any intrinsic value to preserving it, so I have nothing against obliterating it completely. In fact, mastoid cavity obliteration is part of (almost) every canal-wall-down procedure that I do. I do not do an obliteration if the cholesteatoma is adherent to the dura in the posterior fossa. I simply can't get it off that dura reliably, and therefore I don't try.

Dr. Gantz: Paul, what parameters do you use when you are making this decision? Can you make it preoperative?

Dr. Lambert: Again, my preference is to have an intact canal wall, so I look for reasons to do a canal-wall-down procedure. If there is a large degree of erosion of the posterior canal wall, that might sway me, but not always. If I see a very retracted tympanic membrane, not just in the posterior-superior quadrant but the entire pars tensa, that is sometimes a red flag. If the patient has any vertigo or sensorineural hearing loss, and particularly a sensorineural hearing loss associated with a possible fistula into the horizontal canal or even into the cochlea itself, that would certainly sway me toward a canal wall down preoperatively. With regard to the intraoperative situation, I agree what has been said here, but I reiterate that I approach just about every case with the intention of maintaining the canal wall.

Dr. Gantz: Now, for the people who are doing their canal wall ups, Simon and Paul: When you do this canal wall up, do you place a large piece of cartilage in every case in the posterior-superior quadrant of the whole tympanum? Paul?

Dr. Lambert: I don't. At the first stage, things I do to try to prevent a retraction–and again, most of my cases are staged–include placing cartilage in the defect of the canal if some erosion of the medial aspect of the canal has occurred, stabilizing that cartilage by removing the perichondrium on one side, and then draping that onto the canal wall. Staging is helpful in terms of placing a large Silastic sheet into the middle ear. That extends back into the epitympanum, back into the mastoid, and will frequently abut the medial aspect of the superior and posterior-superior canal wall and help prevent retractions there. If at the second stage I see changes in the middle ear that are a little disconcerting, if the mucosa is very thick, and certainly if there is fluid in the middle ear, if any initial retraction is evident, then at that point I would reconstruct a large part of the tympanic membrane with cartilage.

Dr. Gantz: Simon, do you use cartilage primarily to try to prevent that re-retraction?

Dr. Pariser: Yes.

Dr. Gantz: Does it work?

Dr. Pariser: Not always, no. Sometimes it retracts around the cartilage, and that's a problem. Inserting middle ear ventilating tubes has not been universally successful either.

Dr. Gantz: Paul, are you in the same situation? That is, do you put tubes in these kids when you start to see fluid re-accumulating?

Dr. Lambert: Yes. I don't do that at the first stage, but at the second stage if I see fluid, certainly at that point, and then subsequently during follow-up.

Dr. Gantz: In other words, follow-up of these young patients has to continue for a long time? Both you and Simon see your canal-wall-up patients on a yearly basis? Joe, with your mastoid cavity obliteration technique, do you do skin grafting? If so, do you have to see patients yearly to clean the grafts and keep them free of disease?

Dr. Nadol: Every mastoid I do gets skin grafted. I take skin at the beginning of the procedure before the ear is even draped out. But most of that skin is applied to the anterior canal wall, and the principal reason for skin grafting is to maintain the anterior angle between the anterior canal wall and the tympanic membrane. Skin grafting over fascia, at least in my hands, doesn't work very well, at least initially. I do not attempt to do a skin graft in the bowl area or over the musculoperiosteal flap covering bone paté. A minority of patients will need a delayed split-thickness skin graft in the office. I will do that graft about 6 weeks postoperatively if they are not epithelizing adequately. The procedure is

done under local anesthesia and takes about 20 minutes.

As far as follow-up is concerned, canal up or canal down gets followed, although it's not a question of the cleaning requirements: they all have to be followed. Follow-up averages about twice a year, but some patients need to be seen three or four times a year. I wish I knew why that is the case, but that is the case. And some patients return after 5 years with no follow-up in the interim, and there's not very much in them, even though it was a canal-wall-down technique. I also let patients swim if they have never had any skin breakdown and have a dry, clean bowl. I don't prevent them from swimming or using hearing aids. Generally that is not an issue, as long as they have a stable, dry, epithelialized bowl.

Dr. Gantz: Simon, do you follow patients and clean the bowls on a yearly basis, or do you do something to make them self-cleaning?

Dr. Pariser: No, I don't skin graft them. I preserve the anterior canal wall skin universally on these ears and widen the canal. I think the meatoplasty plays a critical role in how often they have to be cleaned. If you have a small opening, that is going to be a problem. But it's unpredictable. I've never been able to figure out why some ears are self-cleaning after a canal-wall-down procedure and others aren't. I agree with Joe.

Dr. Gantz: Joe said that he wasn't concerned about closing off or obliterating the mastoid, which he does in almost all of his cases. Paul, does that concern you? Is the technique I have described something you think will be a problem 10–15 years from now?

Dr. Lambert: You have to consider where the recurrent disease or, better said, the residual disease actually occurs in these ears. It is usually in the middle ear space or epitympanum, and typically not back in the mastoid. I routinely place bone paté or some fascial graft over the mastoid area to smooth the contour, so I am not too concerned as long as I feel comfortable that I've removed the cholesteatoma. Having staged these ears, I get a chance to look again about a year later, and if there is no disease there, I feel very comfortable.

Dr. Gantz: Simon, what is your feeling about obliterating the mastoid cavity?

Dr. Pariser: In a large pneumatized mastoid, I amputate the tip completely and sew down the periosteal flap to the digastric muscle, which effectively makes the large mastoid recess smaller (it doesn't eliminate it completely, but it makes it manageable). I routinely graft the mastoid cavity with a piece of connective tissue, the lateral surface of the temporalis fascia. So, in most ears, most of the bone is grafted primarily. I do not depend on secondary granulation tissue.

Dr. Gantz: I'd like to open this discussion to the floor. You have heard from the experts. All of us have some problems, and we haven't found all of the answers. Are there questions for these experts?

Dr. Kevin McKennan (Sacramento): I have a question for Dr. Nadol. In my experience with revision surgery on patients using the canal-wall-down technique, in the vast majority of cases, if I took off the mastoid tip and enlarged the meatus, I found I did not have to do early skin grafting. I had sparingly done skin grafting in mastoid cavities, but usually in patients who had undergone multiple operations or who were elderly. In the patients whom I have seen with poor epithelialization of the mastoid at 2 or 3 months who underwent regular cleaning of that ear over a period of 6 or 12 months, the epithelium would sometimes keratinize and look great at a year when it might not have looked very good at 3 and 6 months. So, my question is, what is your rationale for doing early skin grafting in a mastoid cavity approached by a canal-wall-down technique?

Dr. Nadol: I perform skin grafting on all of my patients during the procedure. I elevate the anterior canal skin in all cases so that it is basically the mirror image of a Kerner flap–that is, the skin is elevated based on the cartilaginous auricle. I put that back in, but it never quite goes all the way back down. So, I might not do it in a case where a good part of the tympanic membrane, which has epithelialized, is preserved, but I would almost always do it in total drum replacement cases. So, the early skin grafting is not so much to achieve epithelialization but to make more predictable the achievement of a sulcus anteriorly between the tympanic membrane graft and the anterior canal wall. That is something I strongly feel influences the hearing outcome. Delayed grafting is not always performed after granulation has occurred. Most of them, as you say, will epithelialize on their own, and during that period the bowl is actually getting smaller. So there is a certain advantage in not rushing it. I tell patients that this is a bit like gum surgery. It's going to take a long while before this ear is healed, measured in weeks or sometimes months, and they should not be disappointed with the fact that postoperative care continues for several weeks.

ANTERIOR SUBANNULAR T-TUBE FOR PROLONGED MIDDLE EAR VENTILATION DURING TYMPANOPLASTY: LONG-TERM FOLLOW-UP

Ravindhra G. Elluru, M.D., Ph.D., Reena Dhanda, M.D., Joel A. Goebel, M.D., F.A.C.S., and J. Gail Neely, M.D., F.A.C.S.

ABSTRACT

Objective: We have previously described the use of anterior subannular T-tubes (n = 20) for long-term middle ear ventilation. In the present study we examine a larger patient population (n = 38) and a longer follow-up interval (average > 2 years) to evaluate the efficacy and safety of anterior subannular tympanostomy.

Study Design: Retrospective nonrandomized case review.

Setting: Tertiary referral hospital.

Patients: Our series consisted of 38 consecutive patients with a diagnosis of eustachian tube (ET) dysfunction, adhesive otitis, media and/or chronic otitis media with a perforation who underwent a tympanoplasty.

Intervention: A subannular T-tube was placed anteriorly at the time of tympanoplasty to provide long-term middle ear ventilation.

Main Outcome Measures: The main outcomes of this study were tube position, tube patency, and middle ear ventilation. In addition, hearing was evaluated both pre- and postoperatively, and any complications were noted.

Results: Thirty-nine ears in 38 patients (24 females, 14 males) received an anterior subannular T-tube at the time of tympanoplasty. Median patient age was 36 years (range, 10–75). All 38 patients had ET dysfunction. In addition, 22 had adhesive otitis media, 23 had chronic otitis media, 13 had a cholesteatoma, 11 had tympanic membrane perforations, and 3 had a cleft palate. All patients underwent tympanoplasty; one patient received a subannular tube in the contralateral ear without tympanoplasty. Eighteen patients underwent a concomitant ossiculoplasty and 7 underwent mastoidectomy.

Follow-up ranged from 1 to 48 months (average, 26 months). Three tubes had extruded within 2 years, in one case resulting in a persistent perforation. Postoperative complications included one case of a partially extruded prosthesis, two cases of tipped prosthesis and persistent tympanic membrane retraction, and case of a plugged tube. All other tubes were patent and showed no evidence of migration. Furthermore, there were no cases of anterior canal blunting or ingrowth of epithelium around the tube.

Conclusions: Anterior subannular tympanostomy is a safe and effective

method for long-term middle ear ventilation in patients with chronic ET dysfunction.

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DELAYED FACIAL PALSY AFTER STAPEDECTOMY

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

ABSTRACT

Objective: To study the incidence, pathogenesis, and prevention of delayed facial palsy after stapedectomy.

Study Design: Retrospective case review.

Setting: Otology/neurotology referral center.

Patients: A series of 2,152 stapedectomy procedures performed in the past 12 years.

Intervention: Delayed facial palsy after stapedectomy was studied.

Main Outcome Measure: House-Brackmann facial nerve grading system and serum antibody titer tests for herpes simplex virus types I and II and varicella zoster virus.

Results: Delayed facial palsy occurred in 11 of 2,152 procedures. Delayed facial palsy occurred from 5 to 16 days (mean, 8 days) after stapedectomy. Predisposing factors were bony facial canal dehiscence, with bare facial nerve herniation in 5 patients, chorda tympani nerve stretched, manipulated, or cut in 2 patients, granulomatous reaction to Gelfoam in 1 patient, fever blisters on the upper lip in 1 patient, and viral sinusitis in 2 patients. Elevated antivaricella antibody titers were found in all 6 patients studied. Anti-HSV type I and II antibody titers were elevated in 5 of 6 patients. Acyclovir was effective in preventing delayed facial palsy in one revision stapedectomy patient, who had delayed facial palsy after prior stapedectomy in the same ear with an elevated anti-HSV antibody titer.

Conclusion: Delayed facial palsy occurred in 0.51% after stapedectomy. Serologic investigation suggests activation of latent herpesvirus. Mechanical irritation of the facial or chorda nerve during operation may trigger the activation. The anti-herpes virus agent acyclovir may prevent delayed facial palsy after stapedectomy in patients suspected of this complication.

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INCIDENCE OF FACIAL NERVE DEHISCENCE AT SURGERY FOR CHOLESTEATOMA

Samuel H. Selesnick, M.D., F.A.C.S., and Alastair G. Lynn-Macrae, M.S.

ABSTRACT

Objective: Facial paralysis can occur after surgery for cholesteatoma. The risk of facial nerve injury is great when the nerve is not covered by its normal bony fallopian canal. The objective of this study was to identify the incidence of facial nerve dehiscence in patients undergoing surgery for cholesteatoma.

Study Design: Retrospective chart review.

Setting: Tertiary referral hospital.

Patient Population: An assessment of all cases performed by the senior author from 1991 to 1999 identified 59 patients in whom adequate data were available for analysis. These patients ranged in age from 3 to 92 years. Sixty-seven operations were performed in total.

Intervention: Surgery for cholesteatoma, including tympanoplasty and mastoidectomy.

Outcome Measures: The presence of facial nerve bony dehiscence after exenteration of disease, and postoperative facial nerve function.

Results: Facial nerve bony dehiscence occurred in 33% of the total operations analyzed, including 30% of the initial surgeries and 35% of the revision surgeries. The dehiscence was present in the tympanic portion of the facial nerve in the vast majority of patients. Of the 97% of patients with normal preoperative facial nerve function, all retained normal function postoperatively.

Conclusions: Facial nerve dehiscence in our series was far greater than that reported in the literature, underscoring the fact that this is an underappreciated finding. These findings merit increased vigilance when surgeons dissect near the facial nerve.

Intraoperative facial nerve monitoring has proved to be of value in facial nerve preservation during acoustic neuroma resections, and may have a role during surgery for cholesteatoma.

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DISCUSSION PERIOD III: Papers 8–10

Dr. C. Gary Jackson (Nashville, TN): This series of papers is now open for discussion.

Dr. Gerry Gianoli (New Orleans, LA): I have a couple of questions for Dr. Ge. How long after surgery did you obtain the titers in your patients? You mentioned they were elevated. Were you referring to IGM or IGG titers? Finally, did you measure titers in patients who did not develop facial palsy?

Dr. Richard Ruggles (Cleveland, OH): A quick comment regarding the persistent problem following mastoid surgery, which I didn't get to last time. Granulation tissue is usually the cause of this problem. If one takes the time to put the patient on zinc sulfate by mouth and to clean the ear canal and mastoid bowl and paint it with gentian violet once a week, I have not seen a failure with this routine. I think it works very well.

Dr. Xianxi Ge (Memphis, TN): The antibody titer tested was IGG.

Dr. Douglas Green (Jacksonville, FL): I have a question for Dr. Selesnick or his assistant: Did you use intraoperative facial monitoring with the high incidence of dehiscence of the facial nerve and the good postoperative nerve results? I am curious as to whether it was used, and helpful.

Dr. Samuel Selesnick (New York, NY): Yes, we routinely used nerve monitoring in that situation.

A COMPARISON OF HEARING RESULTS IN INTRATYMPANIC GENTAMICIN THERAPY

Michelle L. Facer, D.O., Colin L. W. Driscoll, M.D., Stephen G. Harner, M.D., George W. Facer, M.D., Charles W. Beatty, M.D., and Thomas J. McDonald, M.D.

ABSTRACT

Objective: Intratympanic gentamicin is a well-established and effective treatment for intractable vertigo of a peripheral vestibular etiology. Sensorineural hearing loss is a potential complication of the treatment and is directly related to the dose delivered. The risk of hearing loss with various treatment regimens has not been clearly delineated. The objective of this study was to establish the risk of hearing loss and to clarify the relationship between hearing loss and drug dose.

Data Sources: A MEDLINE search of the English literature up to June 1999 was conducted using the search terms *intratympanic, gentamicin, vertigo,* and *Ménière's disease.* The bibliographies of each article were reviewed to identify other relevant publications.

Study Selection: All studies reporting pre- and posttreatment hearing results and treatment dose were included. The analysis also included the prospectively collected data from approximately 100 patients treated at the authors' institution.

Data Synthesis: Pre- and posttreatment hearing results were compared, and the relationship with treatment dose was explored.

Conclusions: Intratympanic gentamicin can be delivered with limited risk to hearing in patients with vertigo of peripheral labyrinthine origin. In patients with useful hearing, the goal of treatment should be to deliver the lowest dose of gentamicin that relieves the symptoms. Some current protocols may use a dose higher than necessary for vertigo control and increase the risk of hearing loss.

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IMPACT OF MÉNIÈRE'S DISEASE ON QUALITY OF LIFE

John P. Anderson, Ph.D., and Jeffrey P. Harris, M.D., Ph.D.

ABSTRACT

Objective: To describe the health-related quality-of-life situation of patients with Ménière's disease in whom conventional therapy has failed and who are candidates for further medical intervention.

Study Design: Pretreatment interviews to establish baseline characteristics in terms of quality-of-life measures before further medical intervention.

Setting: Tertiary referral center.

Patients: Nineteen adult patients with Ménière's disease (12 women and seven men) in whom conventional therapy had failed. Ages ranged from 32 to 83 years.

Interventions: None as yet. Pretreatment baseline interviews have been conducted.

Main Outcome Measures: Quality of Well-being (QWB) Scale, SF-12 Physical and SF-12 Mental scores, Center for Epidemiologic Studies C Depression Scale.

Results: The QWB score for patients with Ménière's disease of .561 indicated that they were losing 43.9% of wellbeing compared with patients with no symptoms and full functional status. On days patients had symptoms identified as characteristic of acute Ménière's disease episodes, QWB scores were lower than on days patients did not report such symptoms ($P = 0.000$). Patients' scores of 38.9 on the SF-12 Physical scale were below the general mean of 50 by more than 1 SD, and scores of 44.2 on the SF-12 Mental scale were below the general mean of 50 by 0.5 SD. The CES-D score was 23.3 (a score of 16 or above indicates clinically significant depression).

Conclusions: These findings indicate that (1) the pretreatment condition of patients with Ménière's disease can be measured by these instruments; (2) the instruments appear to be in substantial agreement about a serious impairment of patients' quality of life; and (3) days with acute episodes of Ménière's disease are significantly worse than days without such episodes. Treating physicians indicated surprise at the breadth and degree of debilitation experienced by patients with Ménière's disease (IRB #980609).

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THE WANING ROLE OF VESTIBULAR NERVE SECTION AND LABYRINTHECTOMY FOR INTRACTABLE MÉNIÈRE'S DISEASE

Anis A. Ahmadi, B.S., Patrick J. Antonelli, M.D., and George T. Singleton, M.D.

ABSTRACT

Objective: To assess the impact of intratympanic gentamicin (ITG) therapy on the need for invasive surgery (labyrinthectomy and vestibular nerve section) for intractable Ménière's disease.

Study Design: Retrospective case review.

Setting: Tertiary referral center.

Patients: All patients receiving surgical treatment for Ménière's disease not controlled by medical therapy, for the 5 years preceding our adoption of ITG (1987–1991) and the most recent 5-year period (1994–1998).

Main Outcome Measures: Type, efficacy, and complications of surgical therapy.

Results: From 1987 through 1998, 61 patients with intractable Ménière's disease underwent labyrinthectomy (18 patients), vestibular nerve section (2), ITG (29), or endolymphatic sac surgery (12). Although the volume of surgical cases doubled from the first 5 years (16) to the last 5 years (30), the need for labyrinthectomy dropped from 87.5% of cases before ITG to 10% after ITG. After the introduction of ITG, the use of ITG rose to 67% of cases. Of the three patients treated with labyrinthectomy in the past 5 years, two did not have adequate support to perform ITG at home or to return for outpatient therapy, and one patient was not offered ITG. Vestibular nerve section has not been needed in the past 5 years. Only one patient with bilateral disease reported no improvement with ITG. Complete or substantial control of vertigo was similar with ITG and invasive ablative techniques (90% vs. 95%).

Conclusion: ITG therapy markedly reduced the need for more invasive ablative surgery for intractable Ménière's disease.

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DISCUSSION PERIOD IV: MÉNIÈRE'S DISEASE
Papers 11–13

Dr. C. Gary Jackson (Nashville, TN): These papers are now open for discussion. Dr. Leutje?

Dr. Charles Luetje (Kansas City, MO): In light of the excellent paper by Dr. Harris and the plight of patients with Ménière's disease, I'd like to ask Dr. Antonelli and Dr. Facer whether they would use intratympanic gentamicin in the only hearing ear of a person with Ménière's disease.

Dr. Patrick Antonelli (Gainesville, FL): Nothing like starting with an easy question! If a patient's life was basically on hold because of severe, intractable vertigo, I would certainly consider the use of gentamicin over some other modalities. Obviously labyrinthectomy is not a major consideration, but with the success of cochlear implantation, we can be a bit more aggressive than we were 10–15 years ago. As for performing neurectomy in an elderly patient, that's a judgment call. I don't think there is any answer.

Dr. Michelle Facer (Rochester, MN): At our institution we have not used gentamicin in that situation; however, because of the low risk to hearing (and here I concur with Dr. Antonelli), it would be a reasonable alternative to consider instead of surgical intervention.

Dr. Michael Seidman (Detroit, MI): A few quick questions for Dr. Facer. You mentioned that 2–3 dB was statistically significant in your patients. We usually use at least 5 dB and call it intertest variability. I'm curious as to how you get statistical significance out of those numbers, or perhaps I misunderstood what you were doing.

Do you have any data on streptomycin? Dr. Shea uses streptomycin and frequently reports that its use is associated with a lower risk of hearing loss, and–no offense–the statement of virtually no hearing loss was rather bold, and may be dangerous to make.

A quick question for Dr. Antonelli: Vincente Honrubia has indicated that pericells regenerate even in the malleus, and so symptoms might recur in 2–3 years. Could you comment?

Dr. Facer: Those are good questions. With regard to the threshold, no statistical change in the mean hearing threshold was seen after either initial or subsequent injections or at the varying concentrations. The only significant changes were seen at the high frequencies of 6 and 8 kHz with 40 mg/mL and at 8 kHz with 20 mg/mL.

Dr. Antonelli: A number of patients come back after a year or so and say they are starting to get woozy spells and feel as though the condition is beginning to come back very slightly. We do a tune-up in the office and give a little injection, and they do very well.

Dr. Richard Friedman (Los Angeles, CA): This question is for Dr. Antonelli. I didn't get the duration of follow-up in your study. In my experience the vast majority of these patients, symptoms recur within months to a year. Again, I haven't been practicing as long as some in the audience, but virtually all of the have gone to the nerve section, so I personally have not seen the positive results that you are reporting.

Dr. Richard Ruggles (Cleveland, OH): I used to do sac surgery too, and some of the other procedures. For the past 10 years I have been using allergy management with skin end-point testing and provocative food testing. We have seen no failures with this method. The disease is controlled very well, and patients are much more comfortable, as invasive procedures are not being done.

Dr. Newton Coker (Houston TX): I have a question for Dr. Harris. A number of years ago, when we studied patients with Ménière's disease, we administered a battery of psychological tests, because–as most here would concur–these patients have a different personality from most patients we see in the office. The psychological testing indicated a high profile of depression in this group of patients. These patients had active Ménière's disease, and by that I mean they were either suffering from chronic disequilibrium or had recurrent attacks of vertigo that were poorly managed by medical therapy. But what was not clear from our study was the actual problem: was depression aggravating the vestibular problems or were the vestibular problems leading to an altered lifestyle that in turn was causing the depression? It was the old chicken-or-the-egg problem. I wondered if you could give us some insight from your experience.

Dr. Jeffrey Harris (San Diego, CA): No, I can't–but that is a great question. I expect most clinicians today would say that patients who are severely limited develop neuroses as a consequence of the impairment, because they begin to wonder what might trigger the medical condition. So they start to avoid things, and start to change their life.

I'm not sure how you could answer the question. You would need to obtain a psychological profile before the problem began to manifest, in order to segment disability-related problems from underlying personality problems, but thank you for the question.

Dr. John Shea, Jr. (Memphis, TN): I would like to compliment Dr. Facer's presentation. I thought it was absolutely brilliant. I find it interesting that they had 83 patients and one total hearing loss. There are a lot of reports in the literature on people with a familial sensitivity to aminoglycoside antibiotics; the rate is about 1%. I have read about half a dozen papers in the literature, and the sensitivity is to streptomycin and gentamicin. They had one total hearing loss, and the minute I heard that, I thought, well, that's the 1% familial sensitivity to any aminoglycoside.

It's wonderful that we now have nearly complete acceptance of aminoglycoside treatment for Ménière's disease. I still prefer streptomycin, but that's why they make chocolate and vanilla, you know–it's a difference of opinion. We have come a long way, in this Society, to be talking about the advantages of aminoglycosides and the disadvantages of labyrinthectomy and vestibular nerve sections.

Dr. Kevin McKennan (Sacramento, CA): I have used both gentamicin and vestibular neurectomy for treatment, and my conclusion is exactly the opposite of some of the authors'. Of the two treatments, I prefer the vestibular neurectomy, because it is definitive and patients are basically cured of vertigo forever. Gentamicin injections are very safe and easy to do, but patients come back after 2 or 3 years with wooziness, disequilibrium, and attacks of vertigo, and I have to reinject them. And second, I never used just one injection. Dr. Facer mentioned that 80% of patients were treated with one injection. I would be curious to know the follow-up in those patients, because I was never able to accomplish much of anything with one injection.

Dr. Facer: Our mean follow-up interval is 35.6 months. Those patients have been followed for a significant length of time, and they have not had recurring symptoms or significant hearing loss.

IMPLANTATION OF THE SEVERELY MALFORMED COCHLEA

Andrew J. Fishman, M.D., J. Thomas Roland, M.D., George Alexiades, M.D., and Noel L. Cohen, M.D.

ABSTRACT

Objective: To evaluate the feasibility, safety, and efficacy of cochlear implantation in a series of patients with severe cochlear malformations, including common cavity deformities and small hypoplastic cochlear buds of only a few millimeters. An initial case report of fluoroscopically assisted implantation of a common cavity deformity is provided.

Study Design: Retrospective case review.

Setting: Tertiary referral center.

Patients: Patients with severe cochlear malformations, including common cavity deformities and small hypoplastic cochleas of less than one complete turn, were included in the review.

Interventions: High-resolution CT, MRI, plain radiography, and age-appropriate pre and postoperative audiologic and speech perception assessments were performed in all patients, with the exception of one recently implanted individual. Fluoroscopically assisted implantation was performed in one patient.

Main Outcome Measures: Nature of cochlear malformation, active intracochlear electrodes currently in use, and complications and their management were documented, in addition to audiologic testing.

Results: All but one of the patients derived tangible benefit from cochlear implantation. One patient who recently received an implant has yet to be tested postoperatively, although intraoperative electrophysiologic testing revealed that neural response telemetry and stapedial reflexes were present.

Conclusions: Cochlear implantation can be safely and successfully performed in patients with severe cochlear malformations at experienced centers.

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THE MANAGEMENT OF FAR-ADVANCED OTOSCLEROSIS IN THE ERA OF COCHLEAR IMPLANTATION

Michael J. Ruckenstein, M.D., M.S., F.A.C.S., Kristine O. Rafter, M.A., and Douglas C. Bigelow, M.D.

ABSTRACT

Objective: To evaluate issues pertaining to cochlear implantation in patients with far-advanced cochlear otosclerosis.

Study Design: Prospective cohort.

Setting: Tertiary care referral center.

Patients: Eight adult patients (18 years of age or older) referred for the management of profound hearing loss, the etiology of which was determined to be otosclerosis.

Intervention: Cochlear implantation with a multichannel cochlear implant device.

Main Outcome Measures: Benefit from cochlear implant as measured by CID sentence scores, the incidence and management of facial nerve stimulation, and technical issues pertaining to cochlear implantation in this patient population.

Results: All patients showed significant improvement in auditory function as measured by CID sentence scores and ability to engage in telephone conversations. Facial nerve stimulation occurred in two of eight patients and was managed by deactivating the stimulating electrodes. Ossification in the basal turn of the cochlea, detected on preoperative CT, necessitated placement of the electrode into scala vestibuli in two patients and the utilization of a thinner electrode (Nucleus 24) in a third patient.

Conclusion: Patients with profound hearing loss secondary to otosclerosis derive excellent benefits from cochlear implantation. Surgical implantation can be complicated by ossification of the cochlea, which can be detected on preoperative CT. Electrode activation may be complicated by facial nerve stimulation, which can be addressed with programming strategies.

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IS COCHLEAR IMPLANTATION POSSIBLE AFTER ACOUSTIC TUMOR REMOVAL?

Aziz Belal, M.D.

ABSTRACT

Methods: Eight temporal bones in seven patients who underwent acoustic tumor removal during life were histologically examined. Special emphasis was placed on examining the patency of the cochlear turns, survival of the spiral ganglion cells, and the cochlear nerve.

Results: After middle fossa removal of an acoustic tumor with an unsuccessful attempt at hearing preservation, the cochlea was ossified, the spiral ganglion cells had degenerated, and the cochlear nerve was fibrosed. Following translabyrinthine acoustic tumor removal, the cochlear turns were filled with blood, which gradually organized into fibrous tissue and bone. Total cochlear ossification was complete within months after the surgery. The spiral ganglion cells and the cochlear nerve had almost completely degenerated.

Conclusions: The possibility of cochlear implantation after acoustic tumor surgery depends on two factors: patency of the cochlear turns, and survival of the spiral ganglion cells and cochlear nerve. There is progressive osteneogenesis of the cochlear turns following acoustic tumor removal. The process seems to take months to be completed and is directly related to preservation of the blood supply to the cochlea. If cochlear implantation is indicated, the earlier it is performed the better. Following retrosigmoid or middle fossa approaches, cochlear implantation may be done after 1 month of the initial surgery. Following translabyrinthine acoustic tumor removal, the internal coil may be inserted at the time of initial surgery.

Survival of the neural structures in the cochlea and of the cochlear nerve is also directly related to preservation of cochlear blood supply. Determination of nerve survival by the promontory test may be a crucial prerequisite in cases with unsuccessful hearing preservation.

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ADULT COCHLEAR IMPLANT PATIENT PERFORMANCE WITH NEW ELECTRODE TECHNOLOGY

Terry Zwolan, Ph.D., Paul R. Kileny, Ph.D., Sharon Smith, M.S., Dawna Mills, M.S., and Mary Joe Ogberger, Ph.D.

ABSTRACT

Objective: In 1998, clinical trials were initiated to evaluate the Clarion precurved electrode plus Electrode Positioning System (EPS) in adults with severe to profound hearing impairment. In 1999, clinical trials were initiated to assess the Clarion HiFocus Electrode plus EPS in a similar group of adults. This retrospective study evaluated the benefit of these new electrode designs and compared the postoperative speech perception abilities of 60 patients implanted with the precurved electrode + EPS and 43 patients implanted with the HiFocus electrode + EPS.

Study Design: All subjects participated in preoperative testing with hearing aids and postoperative testing (1 month and 3 months) with either the precurved electrode + EPS or the HiFocus electrode + EPS. Demographic characteristics and pre- and postoperative speech perception results were compared within and between the two groups.

Setting: The clinical trial data presented here were collected at 26 cochlear implant programs affiliated with tertiary medical centers located in the United States and Canada.

Patients: Postlinguistically deafened adults who received a Clarion cochlear implant.

Results: Speech perception results demonstrate the improved communication benefit provided by these two electrode designs when compared with results obtained preoperatively when using conventional amplification. A comparison of demographic data showed that the HiFocus group had a significantly longer duration of deafness than the precurved electrode group. Statistical comparison of speech perception abilities showed no significant difference between patients using the precurved electrode + EPS and those using the HiFocus electrode + EPS, although the mean and median scores for word and sentence recognition were higher for the HiFocus + EPS group.

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DISCUSSION PERIOD V: IMPLANTABLE DEVICES
Papers 14–17

Dr. C. Gary Jackson (Nashville, TN): This set of papers is now open for discussion.

Dr. Mansfield Smith (San Jose, CA): I'd like to see about 30 seconds of the video that Dr. Fishman was showing; we were just getting into it and he had to stop.

Dr. Andrew Fishman (New York, NY): We precurved the tip. Here's the common cavity in the internal auditory canal. The important aspect of using this fluoroscopic technique is to avoid inserting it right into the internal auditory canal, which we did on the previous common cavity. Here the device makes a complete turn. It probably snakes around to the back of the hypoplastic semicircular canal. Here's the nice curve of the device, right here. And here are the common cavity, the internal auditory canal, and the final configuration.

Dr. Bradley Welling (Columbus, OH): There seems to be an unusually high failure rate for these devices in the malformed common cavity. Could you comment on that?

Dr. Fishman: There are two device failures. Neither of them have to do with electrode problems. They have to do with the receiver stimulator type of device failure. It's coincidental that those two happened to be in malformed cochleas, but they were not specific to electrode malfunction. There does seem to be a higher number of short-circuit electrodes, which you would expect from the fact that these are inserted into a kinked or a looped or a spiral configuration, but those are programmed out, and they are usually left with an average of 10 or 12 working electrodes to use in the program map.

Dr. Richard Wiet (Chicago, IL): This question is for Dr. Ruckenstein. If you were confident of your diagnosis of far-advanced otosclerosis–and I realize that's a very rare problem–why did you not consider primary stapes surgery in six of eight patients? In other words, how did you arrive at that management decision?

Dr. Michael Ruckenstein (Philadelphia, PA): Thank you, Dr. Wiet. I want to mention that your paper was one of the papers we carefully reviewed before deciding on management strategy. The decision was made in conjunction with the patient and our evaluation. The six patients all had undergone primary stapedectomy previously. We found no evidence of any bone audition on examination. They had longstanding disease, and we had no good evidence to suggest that there would be a positive response to revision stapedectomy. So, after discussing the situation with each patient, and based on our somewhat poor results with revision procedures in patients with far-advanced otosclerosis, we decided to proceed with a cochlear implantation.

Dr. John McElveen (Raleigh Durham, NC): Dr. Fishman, what particular approach did you use for the common cavity malformations as well as the other malformations?

Dr. Fishman: A surgical approach is not dissimilar to a standard cochlear implant procedure performed through a facial recess with the canal wall intact and utilizing preoperative CT to determine the position for the cochleostomy. So, in comparing the surface features, if you see something like a common window depression, or perhaps a round window or an oval window, you can use those features to determine the location of the cochleostomy.

Dr. McElveen: Did you do facial recess on all of the common cavities? I ask because you might avoid problems with advertently coursing facial nerves if you go directly into the area of the lateral semicircular canal using the transmastoid labyrinthotomy approach. I don't know whether you have considered that.

Dr. Fishman: I have seen it. Most of the aberrant facial nerves were still identifiable in a case in which the facial nerve was entirely inferior to the common cavity. It was not identified, but the chorda was identified, and there is ample exposure (or view) of the mesotympanum, especially if you take down the incus bar. We do tend to put a little stimulator over the surface of the promontory just before drilling, just to make sure that nerve fibers are not splayed over the surface of the promontory.

Dr. Noel Cohen (New York, NY): A comment for Dr. Belal: We first reported the use of a cochlear implant following acoustic neuroma surgery in 1991, and the patient is still using his cochlear implant. He is a lawyer and on the telephone all day.

Dr. Anthony De La Cruz (Los Angeles, CA): Dr. Ruckenstein, what made you decide not to do a stapedectomy in the other ear?

Dr. Ruckenstein: Again, it was the absence of any hint of bone audition in the other ear. A second consideration is when the electrode goes into the internal auditory canal. Usually those elec trodes need to be turned off from the map because of facial nerve stimulation, so it effectively allows us to have fewer electrodes active in the common cavity. So it's just a suboptimal placement.

HEARING REHABILITATION USING THE BAHA BONE-ANCHORED HEARING AID: RESULTS IN 40 PATIENTS

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ABSTRACT

Objective: To evaluate the experience of the first 40 patients who have undergone audiologic rehabilitation with the BAHA (Bone-Anchored Hearing Aid) in the United States.

Study Design: Multicenter, nonblinded, retrospective case series.

Setting: Twelve tertiary referral medical centers in the United States.

Patients: Patients eligible for BAHA device implantation were those with hearing loss and inability to tolerate a conventional hearing aid, with bone conduction pure-tone average levels of 60 db or less at 0.5, 1, 2, and 4 kHz.

Intervention: Patients who met audiologic and clinical criteria were implanted with the BAHA Bone-Anchored Hearing Aid (BAHA, Entific Corp).

Main outcome measures: (1) Preoperative air and bone conduction thresholds and air-bone gap; (2) postoperative BAHA-aided thresholds; (3) hearing improvement as a result of implantation; (4) implantation complications; and (5) patient satisfaction.

Results: The most common indications for implantation included chronic otitis media and/or draining ears (18 patients) and external auditory canal stenosis or aural atresia (7). Overall, each patient had an average improvement of 32 dB _ 19 dB with the use of the BAHA device. Closure of the air-bone gap to within 10 dB of the preoperative bone conduction thresholds (postoperative BAHA-aided threshold vs. preoperative bone conduction threshold) occurred in 32 patients (80%), while closure to within 5 dB occurred in 24 patients (60%). In 12 patients (30%) there was "overclosure" of the preoperative bone conduction threshold of the better hearing ear. Complications were limited to local infection and inflammation at the implant site in three patients, and failure to osseointegrate in one patient. Patient response to the implant was uniformly satisfactory. Only one patient reported dissatisfaction with the device.

Conclusions: The BAHA device provides a reliable and predictable adjunct for auditory rehabilitation in appropriately selected patients, offering a means of dramatically improving hearing thresholds in patients with conductive or mixed hearing loss who are otherwise unable to benefit from traditional hearing aids.

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UPDATE ON CONSERVATIVE MANAGEMENT OF PATIENTS WITH ACOUSTIC NEUROMAS

Dick L. Hoistad, M.D., George A. Melnik, M.D., Bulent Mamikoglu, M.D., Cathleen A. O'Connor, M.S., and Richard J. Wiet, M.D., F.A.C.S.

ABSTRACT

Objective: To update our 1995 experience with conservative management of acoustic neuromas (ANs).

Study Design: Retrospective chart review.

Setting: Private practice and tertiary care referral setting.

Intervention: Of 600 patients with an AN, 102 were treated with a "wait and scan" treatment option. At least two magnetic resonance imaging (MRI) studies were required in all patients.

Main Outcome Measures: Change in tumor size over time, and clinical symptoms (hearing status, tinnitus, balance disturbance, aural fullness, vertigo, headache, and facial pain, numbness, or weakness).

Results: The average follow-up time in the 102 patients was 28.5 months. In 45 (44%) of the 102 patients, there was a change in tumor size, which grew on average 2.17 mm per year. In the remaining 54 patients (53%), no growth was demonstrated on a mean follow-up of 28.5 months. In three patients the tumor shrank. Of the 102 patients managed conservatively, 85 (84%) reported hearing loss, 67 (66%) tinnitus, 37 (36%) balance disturbance, 29 (28%) aural fullness, 28 (27%) vertigo, 7 (7%) headache, 4 (4%) facial numbness, 2 (2%) facial weakness, and 0 (0%) facial pain.

Conclusion: Conservative management–'wait and scan'–in selected patients with AN a reasonable management alternative to radiation therapy or microsurgery. There are situations in which the individual morbidities associated with surgery or radiation therapy are not in the patient's best interests. A third option should be available for patients who cannot or do not wish to undergo these other treatments.

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COMPARISON OF THE KI-67 AND C-*FOS* STAINING PATTERN IN GLOMUS JUGULARE AND GLOMUS TYMPANICUM TUMORS

Mohammed Mujtaba, M.D., J. Thomas Roland, M.D., Dennis G. Pappas, M.D., and Dean E. Hilman, Ph.D.

ABSTRACT

Hypothesis: The size of the jugulotympanic paraganglioma (JTP) is directly related to the density of Ki-67 antibody- and c-*fos* antibody-labeled cells, and is indicative of tumor aggressiveness.

Background: Jugulotympanic paragangliomas are usually slow-growing benign tumors of the temporal bone; however, some tumors may show aggressive growth rates and become malignant. In this study, we utilized c-*fos* and Ki-67 antibodies for labeling cells in the active phase of replication. The density of c-*fos*- or Ki-67-labeled cells was compared to tumor size for determining a possible relationship to the rate of growth.

Method: Nine surgical tumor specimens that included both the glomus tympanicum (GT) and glomus jugulare (GJ) were investigated using immunohistochemical and ultrastructural analysis. Tumor sections labeled with Ki-67, c-*fos*, γ-tubulin, and S-100 antibodies were analyzed using a light microscope interfaced with a computer-based mapping system. Ultrastructural analysis of the tumor sections was performed to compare morphological features.

Result: Large-sized and recurrent glomus tumors (most aggressive types) had a higher density of Ki-67- and c-*fos*-labeled cells with a low density of the γ-tubulin-labeled cells than the small-sized nonaggressive tumors. In addition, malignant and recurrent glomus tumors had an increased number of mitochondria as compared to the small-sized tumors.

Conclusion: There is a positive correlation between the aggressiveness of glomus tumors and the density of Ki-67- and c-*fos*-labeled cells, but a negative correlation for density of γ-tubulin-labeled cells. An increased number of cellular organelles such as mitochondria might reflect the rapid tumor growth rate. We conclude that Ki-67 and c-*fos* antibodies are indicative of a faster growth rate and susceptibility for recurrence in glomus tumors.

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DISCUSSION PERIOD VI: HEARING LOSS/INNER EAR
Papers 18–21

Dr. C. Gary Jackson (Nashville, TN): These papers are now open for discussion.

Dr. Michael Seidman (Detroit, MI): This question is for Dr. Hoistad. I enjoyed your talk but might question the age cutoff of 60 years for a "wait and scan" approach. The average life span in the United States today is 76.6 years, which gives this tumor 16 years to grow. Now, if you scan every 6 or 12 months, you will probably catch something, but if you go from a 5-mm tumor–which I think is reasonable to watch–and find on the next study that it is 1.5 cm, the risks increase significantly, and the ethics of this situation become questionable. I certainly offer 55- to 60-year-old patients a "wait and scan" choice if the tumors are small, but I wonder whether their average life span should be a consideration.

Dr. Richard Hoistad (Evanston, IL): If I understand your question correctly, my response is that even younger people might present with a tumor and end up in the same predicament. Do you want to comment one more time so that I can try to explain?

Dr. Seidman: My only comment is that 60 years seems awfully young when the average life span is now 76.6 years.

Unidentified Speaker: Can I help you out, Dick? –This issue of wait and scan can be applied to young people. There doesn't need to be an age cutoff of 60 years. The point is, if you are diagnosing people with 2-mm tumors that are intracanalicular, there is time. I think that's an arbitrary rule that is established but perhaps not appropriate. We would want to clarify that.

Dr. Bruce Gantz (Iowa City, IA): Dr. Mujtaba, I enjoyed your paper. We do encounter these aggressive tumors and we don't know what to do with them. What do you do at NYU when you find an aggressive tumor? Do you alter your postoperative management strategy, and what is that management strategy?

Dr. J. Thomas Rowland (New York, NY): We are not sure, because our data are preliminary, but in general, if we have a tumor that is very aggressive, very invasive, and has a lot of carotid artery involvement, we do not sacrifice the carotid arteries. Either we observe these patients or, depending on the institution in which they are being treated, they often receive postoperative radiation therapy. We hope that some of the data we are acquiring will help us decide which of those patients should be offered radiation therapy earlier rather than later.

Dr. Donald Kamerer (Pittsburgh, PA): Dr. Lustig, I enjoyed your paper very much. We certainly agree that the bone-anchored hearing aid has a place. You reported only one failure at osseointegration, and I was a little surprised by your waiting time, only 6 weeks before hookup. In our small series we have waited 3-4 months. Could you comment on that?

Dr. Lawrence Lustig (Baltimore, MD): We used 6 weeks because that's what the Swedish groups have used, and they are the ones who developed osseointegration, so we followed their lead. The one patient in whom osseointegration did not occur was the youngest patient in our series. The Swedish groups are now recommending 12 weeks for adolescents and younger patients for osseointegration. In adults, 6 weeks is adequate, but in adolescents or younger pediatric patients, at least 12 weeks should be allowed before osseointegration.

Dr. Julian Nedzelski (Toronto, ON): With respect to Dr. Hoistad's paper, I would just like to make a plea that irrespective of what we as a fraternity decide to use as measuring guidelines with respect to tumor growth, let's adhere to those guidelines. I'm chagrined that we would decide that the tumor is growing or not growing on the basis of a single measurement, which seems to be the longest dimension of the tumor. There is ample precedent in the literature for deciding how we will measure tumors. Otherwise, what we report as growing or nongrowing tumors will be even more variable, and we need some data that are at least uniform.

Dr. Richard Wiet (Chicago, IL): I'd like some clarification on the paper on immunohistochemical evaluation of glomus tumors. Will your paper shed any light on the emerging reports of gamma knife treatment for glomus tumors versus standard radiation? Will you have information in that area? There are recent reports that in individuals with

large glomus jugulare tumors, the gamma knife may effectively retard tumor growth. It doesn't remove the tumor, of course, but it is now being used for slowing tumor growth. Does your paper give us information to help us with management strategies? Can you comment on that?

Dr. Rowland: I'm going to help out on this one. This is just preliminary information; these are postoperative evaluations. Perhaps you are referring to the possibility that one might want to biopsy the tumor to get information, and then decide on treatment preoperatively. In general, we have not been using the gamma knife as a treatment option in patients with glomus tumors.

Dr. Lawrence Duckert (Seattle, WA): Two questions for Dr. Lustig regarding the BAHA. First, is the BAHA currently FDA-approved for use in the pediatric population? Second, do you have any idea what your audiologist charges for the device itself?

Dr. Lawrence Lustig: The answer to both questions is no. The BAHA is not approved for use in the pediatric population under 5 years of age; that issue is being worked on right now. John Neparko, who is also one of the authors of the study, is working with Antifik to try to get that approval. And no, I don't know what my audiologists charge.

Dr. Brad Pickett (NM): Which of your patients had bilateral mixed or conductive hearing loss, and how did unilaterality affect your indications for surgery and your results?

Dr. Lustig: All the patients with otosclerosis or conductive hearing loss had bilateral hearing loss. Many of these patients had bilateral mastoid bowls and bilateral chronically draining ears that could not be fixed, and as a result they could not wear a hearing aid on either ear. That was probably the most common indication for implantation. To answer the second question, the most common unilateral indication was a patient who underwent external auditory canal closure following a skull base procedure. Hearing in the contralateral ear was fine, but the patient didn't have any hearing by air conduction on the bad side, so we felt that was an excellent indication to proceed. All of those patients had subjectively improved results and were very happy with their device.

Dr. Dudley Weider (Hanover, NH): What was your maximum overclosure?

Dr. Lustig: The maximum overclosure was about 5 dB.

Dr. Steve Telian (Ann Arbor, MI): Can you comment on tinnitus suppression with the BAHA?

Dr. Lustig: I have no data at all indicating that the BAHA does suppress tinnitus, and we had no reports from any other patients that their tinnitus was improved by the device. So I don't think I would recommend it at this point for tinnitus, but it might be something to look into in the future. For the present, I have no data to suggest that it helps tinnitus.

ETANERCEPT THERAPY FOR IMMUNE-MEDIATED COCHLEOVESTIBULAR DISORDERS: PRELIMINARY RESULTS IN A PILOT STUDY

Hyon K. Choi, M.D., M.P.H., Dennis S. Poe, M.D., and Mahboob U. Rahman, M.D., Ph.D.

ABSTRACT

Objective: Immune-mediated cochleovestibular disorders (IMCVDs) continue to present a management challenge to the otolaryngologist. Antirheumatic agents, commonly employed for IMCVDs, are associated with variable efficacy and sometimes with serious side effects. In this report, we describe preliminary results in patients with IMCVDs treated with etanercept, a TNF-γ receptor blocker recently approved by the FDA for the treatment of rheumatoid arthritis.

Study Design: Retrospective case series.

Setting: Tertiary care hospital.

Patients: Twelve patients suspected of having IMCVDs unresponsive to conventional therapies or who developed side effects to conventional therapies.

Intervention: Etanercept, 25 mg, given by subcutaneous injection twice a week.

Main Outcome Measures: Assessment of hearing change by air conduction pure-tone audiograms and/or word discrimination. When present, vertigo, tinnitus, and aural fullness were assessed as well.

Results: Follow-up in excess of 5 months was available for all patients (range, 5–12 months). Eleven (92%) of 12 patients had improvement or stabilization of hearing and tinnitus; 7 (88%) of 8 patients who had vertigo and 8 (89%) of 9 patients who had aural fullness experienced resolution or significant improvement in their symptoms. The benefit persisted until the last visit (5–12 months after starting etanercept). In one patient the initial dramatic improvement deteriorated after 5 months. The patient's hearing was rescued and stabilized with the addition of leflunomide to etanercept. Three other patients needed a second antirheumatic agent to stabilize their hearing. There were no significant side effects from the etanercept therapy.

Conclusions: Our limited data suggest that etanercept therapy is safe and may be efficacious in carefully selected patients with IMCVDs, at least on a short-term basis. These preliminary efficacy and safety results are encouraging enough to warrant further follow-up and studies for better determination of the potential clinical utility of etanercept for IMCVDs.

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RISK FACTORS FOR HEARING LOSS IN NEONATES

Stilianos E. Kountakis, M.D., Ph.D., John Skoulas, M.D., Diane Phillips, M.S., and C. Y. Joseph Chang

ABSTRACT

Objective: To identify potential risk factors for neonatal hearing loss that are not included in the current variables recognized by the Joint Committee on Infant Hearing (JCIH).

Methods: A series of consecutively born neonates with risk factors for hearing loss based on the 1994 JCIH registry were screened prospectively. There were 110 subjects with hearing loss and 636 subjects without hearing loss. Data collected as potential risk factors for infant hearing loss included not only those on the JCIH list but also others that we believed could be significant. The infant hearing screening was performed on each subject using auditory brain stem testing. Statistical analysis of data was performed using the chi-squared test.

Results: In addition to the variables listed by the JCIH, we identified 11 other risk factors that were associated with hearing loss in our neonatal population: length of stay in the intensive care unit, respiratory distress syndrome, retrolental fibroplasia, asphyxia, meconium aspiration, neurodegenerative disorders, chromosomal abnormalities, drug and alcohol abuse by the mother, maternal diabetes, multiple births, and lack of prenatal care.

Conclusion: This study identified 11 risk factors in addition to those currently on the high-risk registry published by the JCIH for neonatal hearing loss. The inclusion of these additional risk factors in neonatal screening programs may improve the detection rate of neonates with hearing loss. Further study will be needed to determine whether inclusion of these additional risk factors in a hearing screening program can provide an efficacious alternative to the use of universal infant screening.

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LIDOCAINE PERFUSION OF THE INNER EAR PLUS IV LIDOCAINE FOR TINNITUS

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

ABSTRACT

Objective: To determine the results of lidocaine perfusion of the inner ear plus intravenous (IV) lidocaine for intractable tinnitus.

Study design: Retrospective case review.

Setting: Otology/neurotology referral center.

Patients: Lidocaine perfusion of the inner ear plus IV lidocaine was performed on 71 ears of 63 patients with intractable tinnitus. Patients were followed up for 1 month to 1 year.

Intervention: Approximately 0.5 mL of hyaluronan (Amvisc) containing 20 mg of lidocaine per milliliter was injected into the round window niche. The patient remained with the operate ear up while receiving 500 mg of lidocaine IV over 2 hours. The procedure was performed on each of 3 consecutive days. Hearing and spontaneous nystagmus were tested on the second and third days.

Main Outcome Measure: Subjective evaluation of tinnitus by the patient. Complete relief was indicated by no more tinnitus, partial relief by occasional troublesome tinnitus, and no relief by tinnitus remaining the same.

Results: Complete or partial relief of tinnitus was achieved in 35 (70%) of 50 ears within 1 month, in 20 (76.9%) of 26 ears within 3 months, and in 10 (83.3%) of 12 ears within 1 year. Hearing remained the same in all patients. Temporary paralytic spontaneous nystagmus occurred in 22 ears, irritative in 21 ears, and no nystagmus in 25 ears.

Conclusion: Lidocaine perfusion of the inner ear plus IV administration of lidocaine is a safe and effective treatment for intractable tinnitus.

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ROLE OF IMAGING IN THE CLINICAL DIAGNOSIS OF INNER EAR DISORDERS

Arvind Kumar, M.D., Mahmood Mahfee, M.D., Scott W. DiVenere, M.D., and Han Soo Bae, B.S.

ABSTRACT

Objective: In the clinical setting of unilateral hearing loss, unilateral tinnitus, dizziness, and facial paralysis, modern imaging has effectively served to "rule out acoustic tumor." However, in the majority of patients, no tumor is found, and the cause of the symptoms remains unclear. This study sought to demonstrate the diagnostic potential of advanced imaging studies for disorders of the inner ear and adjacent nerves.

Study Design: Retrospective case review.

Setting: Tertiary referral center.

Patients: Individuals presenting with unilateral hearing loss, unilateral tinnitus, dizziness, and/or facial paralysis.

Interventions: Diagnostic review of patients' clinical, audiologic, vestibular, and imaging studies.

Main Outcome Measure: Comprehensive clinical data in patients with unilateral inner ear symptoms were correlated with results of advanced imaging.

Methods: Comprehensive clinical data are correlated with the results of advanced imaging studies, and specific inner ear diagnoses were established. Examples of such diagnoses include hemorrhage into the inner ear, cochlear dendritic demyelination, cochlear otosclerosis, inflammatory lesions of the meatal and intralabyrinthine facial nerve and inner ear, intralabyrinthine schwannoma, and endolymphatic sac tumor.

Conclusions: When advanced imaging of the inner ear is correlated with comprehensive clinical data, specific pathologic entities of the inner ear can be confidently diagnosed. Should all patients with unilateral inner ear symptoms undergo this costly imaging procedure? More data are needed to answer the question. A multicenter study of patients with unilateral inner ear symptoms would provide data that could be used in developing appropriate guidelines.

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DISCUSSION PERIOD VII: HEARING LOSS/INNER EAR
Papers 22–25

Dr. C. Gary Jackson (Nashville, TN): These papers are now open for discussion.

Dr. Mark Gustafson (Cincinnati, OH): I was very interested in Dr. Shea's paper. Some of the previous papers on lidocaine perfusion in the middle ear space mentioned a lot of postprocedure vertigo necessitating hospitalization. I was wondering if you saw this type of impact. Also, because of the heart monitoring you talked about, were you keeping the patients in the hospital, or doing the procedure in the office?

Dr. John Shea (Memphis, TN): Yes, they all experienced significant vertigo after the treatment for the first couple of hours. It's interesting because the patients we have treated have not had any facial weakness, and it is also strange that we always provoke a very strong spontaneous nystagmus. Sometimes the nystagmus is away from the treated ear, but in about half the cases it is toward the treated ear, so something different is going on. The dizziness lasts only a short while, about 2 hours. But it is almost always quite severe.

Dr. Mohamed Hamid (Cleveland, OH): I have a question for Dr. Poe and his group. I realize that the study is a pilot study. My question is, have you had patients treated with methotrexate, and can you comment on the results of both? Second, in my experience speech discrimination is also very responsive to this particular treatment. In fact, we have already increased speech discrimination from 20% to 70%–80% with prednisone and methotrexate. Is that the case with Entanercept?

Dr. Hyon Choi (Boston, MA): We have many patients who are on methotrexate, and also some patients on another methotrexate-like medication called Araba. Interestingly, the results we saw in the mediated cochlear vestibular disorder parallels our experience with the treatment of rheumatoid arthritis. In rheumatoid arthritis, the experience with methotrexate is very long, about 30 years. The patients get better, but after some time the efficacy of methotrexate diminishes, and it is never 100%. Patients show about 60%–80% improvement, and it takes 3–4 months to achieve full effect, similarly with Araba. But with Entanercept or Ambrel, the efficacy is extremely high compared to methotrexate. One of our patients had a dramatic response within 2 weeks. She was using hearing aids, and her hearing improved so much that after 2–3 weeks she did not need a hearing aid anymore. Your other question was speech discrimination. Yes, we see that. In fact, some of our patients that did not show significant improvement in pure tone had dramatic improvement in speech discrimination. One of our patients had only 34% speech discrimination before the treatment was started, and within 3–4 weeks it went up to 94%, although the patient continued to need a hearing aid, because his pure-tone levels were low, in the 40- to 50-dB range.

Dr. Larry Duckert (Seattle, WA): I have a couple of comments regarding Doctor Shea's paper. I'm afraid I do not share his enthusiasm for the use of IV lidocaine–at least I don't have any experience with profusion. Some time ago, my audiological colleagues and I conducted a double-blind study using IV lidocaine, and we found that by comparison with the control group, there was no significant difference. In some cases the patients who received the IV lidocaine said their tinnitus got worse. That was the first half of the study; the results were described here, before this Society. The next year we recalled our patients and told those who had received the placebo that they would be getting the drug in the new study. But instead, we gave them the placebo again, and on that particular occasion, many of them got better, and impressively so. So we concluded that to a great degree, the effect–if there was an effect–was a placebo effect.

Dr. Shea (Memphis, TN): I am surprised, but not totally surprised, at your results. I think it's possible to prove anything if you start with the right set of patients and the right mind-set. My paper referred to about 10 articles in the literature that do report beneficial effects of IV lidocaine, including a series of papers by Melding and his group in Auckland. I'm fascinated by what you have to say. Our experience is exactly the opposite. The basic premise that you have to use in dealing with these people is that they have a disorder that is mostly located in the ear, and then in the brain, and we are beginning

to understand the brain function of tinnitus a lot better. I think there will be drugs coming that we can use to treat the depression. The one we are most interested in, in addition to IV lidocaine, is called Effexor, which increases the body's uptake of both serotonin and dopamine; it has a dramatic effect. I had one man with tinnitus that was not helped by anything we did. IV lidocaine and all these things would help him for a while, and then he'd come back. We put him on Effexor, and he is a dramatically improved person. So this is shotgun therapy; it isn't just IV lidocaine. But the IV lidocaine is a dramatic treatment. I couldn't disagree with your results more, but, is as I said yesterday, that is why they make chocolate and vanilla–it's a matter of opinion.

Dr. John Lisek (Columbus, OH): This question is for Dr. Choi. What creiteria did you use to determine improvement in patients on Entanercept, and were those responses sustained? Were the steroids continued during the treatment?

Dr. Hyon Choi: We used the American Academy criteria for the improvement or stabilization of hearing, which include more than a 10-dB improvement in pure tone in two consecutive wavelengths or more than a 15-dB improvement in one wavelength or more than 15% improvement in the word discrimination score. When we started the patients on tinnitus therapy, ten of the patients were still on very high-dose prednisone. Of those ten, eight were already off prednisone at the time of the report, and the dosages for the other two had been reduced from 80 mg to 10 and 5 mg. We hope we will be able to taper them off completely.

Dr. Manohar Bance (Toronto, ON): My question is on the same topic. Do you have any patients in your group in whom steroid treatment failed, and do you have any experience with salvaging steroid failure with Entanercept? Is there a possibility that patients in whom steroid treatment fails could be effectively treated with Etanercept?

Dr. Choi: All of our 12 patients had a good response to prednisone; however, in our experience with Dr. Dennis Poe, we had a few patients who either had a questionable response to Prednisone or did not have a good response to prednisone but had other indications suggesting an autoimmune process.

For example, one of the patients did not respond to steroids but did have the entire HSP 70 antibody positive, so we tried methotrexate and got similar results, even though the patient did not respond to prednisone. However, we have also treated some patients who were unresponsive to prednisone and subsequently unresponsive to methotrexate. We do not have any experience with patients who were unresponsive to prednisone then being treated with Entanercept. It is a very expensive medication, and we are using it off study, as it is FDA-approved only for rheumatoid arthritis, so we have to make a good case before we use Entanercept. As I mentioned, 58% of our patients had tried methotrexate, Cytoxan or Araba, other forms of Plaquenil, other forms of rheumatic disease therapies. At that point we used Entanercept.

ACOUSTIC NEUROMA

PANEL DISCUSSION II

Dr. Bradley Welling (Columbus, OH): We asked Dr. Thompson to join the panel also.

Dr. John Flickinger is a radiation oncologist and professor in the Department of Radiation Oncology and in Neurosurgery. He has published extensively on the use of the gamma knife on intracranial and other tumors, including more than 250 articles in peer-reviewed journals and chapters. We appreciate him being with us here today.

Dr. Jens Thompsen from Copenhagen, the William House guest of honor for the American Neurotology Society, has also extensively published and has a wealth of experience in the treatment of acoustic tumors.

Dr. Derald Brackmann needs no introduction. He is past president of the American Otological Society and has more than 260 publications and chapters to his credit.

Finally, Dr. Kevin McKennan is a neurotologist who has been in practice in Sacramento for 14 years and has recently taken the gamma knife course and gone through the rigors of becoming trained to perform gamma knife stereotactic radiation therapy as well as microsurgical removal.

I will start by asking our panelists to spend 5 minutes each introducing their area of expertise to us. I would like to start with Dr. Brackmann, followed by Dr. Flickinger, and then Dr. Thompsen and Dr. McKennan.

Dr. Derald Brackmann: Thank you very much, Bradley, and congratulations, Julia and Gary, for running an on-time meeting.

The most recent article on the gamma knife for acoustic tumors was an excellent review from the University of Virginia. It just came out in this month's *Neurosurgery*. The conclusion was that microsurgery remains the primary modality for the treatment of most acoustic tumors, but that surgeons who can't achieve the high level of excellent reported results with microsurgery should consider the gamma knife as their primary modality for the treatment of tumors. I would like to reverse that opinion. I am going to present the neurosurgical, neuro-otological, and microsurgical results and state that it is the challenge of gamma knife users to meet those results, because I think that the results with microsurgery are as good as or superior to the results of radiotherapy. You don't have a tumor when you get done with microsurgery. [With the gamma knife] you still have a tumor, and I'm sure that will come up in the discussion.

The goal of acoustic tumor microsurgery is to completely remove the tumor. Preservation of hearing is a somewhat elusive goal but nevertheless a realistic one in many cases. Of course, the goal of radiotherapy is not total tumor removal, and that also will be discussed. This is nothing new to any of you here. I will briefly review the data because we have presented these statistics before.

We use three approaches–middle fossa, retrosigmoid, and translabyrinthine–for acoustic neuromas. We use the translabyrinthine approach in about half of our cases. Any tumor that results in nonserviceable hearing or any tumor that is more than 2½ cm we treat with the translabyrinthine approach, feeling that hearing preservation would be extremely unlikely.

The results for the middle fossa approach were presented here last year. We have some measurable hearing preserved in about 80% of cases. The hearing is serviceable, very audible in about 60% of cases, and the complication rate for the middle fossa approach is extremely low. The mortality has been zero.

The results in NF2 are the same as in our unilateral cases. We are writing up these results right now. We have 40 patients who have been treated by a middle fossa approach for small tumors and NF2, and the results are actually equivalent to what they are in unilateral tumors–in fact, they are slightly better.

We have preserved class A or B hearing in about 60% of those patients and some hearing in about 65%. Those results, by the way, are superior to the results achieved with the gamma knife or fractionated stereotactic radiotherapy. So, particularly in NF2, there is a real question as to whether you should ever irradiate a patient with NF2.

I prepared a talk for the Lexcell Society. They were kind enough to invite me, and I had a lot of time going there. On the right side [of the slide] is a quotation from the *Archives of Otolaryngology* in the 1930s where they say that any treatment other than x-ray therapy for tonsils–well, anyway, you should

never surgically remove tonsils. They should only be treated with radiotherapy. That's one benign disease that was treated that way, and I think we are going to learn this lesson again.

Dr. John Flickinger: I'd like to thank Dr. Welling and the Society for inviting me to speak today. All of the patients I see with acoustic neuromas at the University of Pittsburgh come in requesting stereotactic radiosurgery, and since I'm on a straight salary and I get to sleep in if there are no radiosurgery cases, my approach is to try to talk the patients out of it, or to scare them out of it. So I give them a list of the top ten reasons why they should not have stereotactic radiosurgery for acoustic neuromas. I tell the neurosurgeons that I work with, Dave Lundsford and Doug Consioca, that this is obtaining informed consent.

Number ten on the list, there's no guarantee that radiosurgery will stop the tumor from growing. In our *New England Journal of Medicine* article, with 5–10 years of follow-up, 2% of patients progress to resection. You might ask what results we are getting now, because we have lowered the doses. I'm just revising our data for the *Journal of Neurosurgery*, and we found that with the lower doses, with most patients receiving 13 Gy, so far 1% have progressed to resection.

Dr. Brackmann talked about salvage surgery after radiosurgery, particularly in patients who have previously undergone a resection that failed. These patients have already received radiosurgery because their tumor grew back after surgical resection. In our combined article with the Mayo Clinic, which Bruce Polek wrote up, we had five out of six cases labeled as difficult, three out of seven without prior surgery. In one patient both radiosurgery and microsurgery failed to control the tumor. The tumor progression rate was about 6%. In the latest review, which covered the past 5 years, I also included the patients treated initially to make it more comparable to a surgical experience.

This didn't include any failures, whereas I think in that paper the failure rate was about 6%–that is, 6% of patients went on to further resection. If you plot that out actuarially, it's probably about 10%, so I worry a bit more about patients with recurrent tumors; the tumors may be more aggressive.

The other thing in the salvage surgery case is we found one patient in that series who did not have an acoustic neuroma but who underwent resection after radiosurgery. The tumor was found to be a malignant triton tumor that had been treated and then progressed. That is one of the risks.

That brings us to reason number nine, which is to tell the patients about chemotherapy. I tell the patients that they may feel safer if we put their tumor into chemotherapy. We cut the tumor out, we throw it into a bucket, and when it stops twitching we take a good look at it under the microscope and be sure what it was. You still have to worry. There is a chance, perhaps 1 in 500 or 1 in 1,000, that it was something other that should have been treated with radiation therapy, but you still have to remember all the risks of surgery.

Number eight, trigeminal neuropathy, is something that bothers me a lot about this procedure. When we started doing the procedure, 27% of patients developed some facial numbness. This figure dropped to 3% in our latest review. In patients who received 13 Gy or less, it's still about 3.1%. One patient has developed typical trigeminal neuropathy at age 79, and in that case the radiosurgery may have been a contributing factor. The etiology of trigeminal neuralgia is complex, but it may be adding to the wear and tear on the nerve.

Facial neuropathy is number seven. There's no guarantee that a patient won't get some facial weakness from the procedure. In our original experience it was 21%, and was so stated in the *New England Journal of Medicine* article. Recently, in the 1992–1997 period, that figure dropped to 1.2%. When plotted, it was 0 out of 103 patients who received 13 Gy or less. Actuarially, it is 0 out of 167 patients who received 14 Gy or less. Because of these results, even though we think there is zero risk of facial neuropathy, because it is a slightly higher dose, we still have cases. I tell patients there may be a 1 in 200 chance that they will end up with some facial weakness after radiosurgery.

Number six is hearing loss from radiosurgery. You may still lose hearing if you undergo radiosurgery. In our original experience, half of the patients–that is all patients, not just the ones in whom we tried to preserve the hearing–half of the patients experienced some drop in their speech discrimination. In the recent series that figure was about 27%, and at the 13 Gy dose level it was 25%–not that much different, because there wasn't that much spread in the data in terms of doses. So, they dropped more than one hearing level, with 11% losing all of their hearing and the other 14% sustaining a drop in hearing level but still having testable hearing.

Number five, which goes along with this, you may hold on to your hearing better if we observe you. I won't go into that too long. A nice series was reported from Shiroto, Japan, in which 27 patients who were observed were compared with 50 who underwent stereotactic radiotherapy. There was no difference in hearing preservation but a dramatic difference in tumor growth. The hearing preserva-

tion suggests–I don't know how well you can see these small numbers–that perhaps at least for the first 3 years you are going to have patients holding on to hearing with observation versus radiosurgery, but after that, there will most likely be a benefit in terms of hearing preservation.

Number four, we just need more time to assess the results of radiosurgery. Twenty-five years of experience in Sweden and 10 years of experience in the United States does not tell you everything you want to know. We would like to see 25-year results of using the low doses published from all of the U.S. centers, but maybe by then we will all be retired and the HMOs will only let chiropractors and LPNs manage these tumors.

Number three, radiation can cause new tumors in the developing 10–30 years or malignant degeneration. Some of the best data on radiotherapy for an entity like this come from studies of radiotherapy for pituitary adenoma. Thousands of patients have been treated in large series with follow-up to 20–30 years. Because of the 5-cm fields used for treating pituitary adenomas, the risk of a new tumor developing is a bit larger, about 1%–2% in large series. The new tumors are about evenly divided between benign and malignant.

We think the risk with single-fraction radiosurgery to smaller fields may be higher by a factor of 10. We are working with the University of California, San Francisco, and other centers to try to get more reliable figures, butt it will still take some time to get hard data.

Number two is a good one for scaring them away: Didn't gamma rays turn mild-mannered Bruce Banner into the Incredible Hulk in that awful TV show? We have [a picture of] Hulk saying, "Hulk should have had translab," with the gamma knife zapping him. If this doesn't scare them away, then I get to the last reason you can use. This is one for the surgeons to use before the patient gets to the radiation oncologist: "If you don't get radiation, you don't have to talk to any creepy radiation oncologists." And why do they hide radiation therapy departments in hospital basements next to morgues if they are not creepy?

These are my top ten reasons, but unfortunately, I haven't been able to convince any of the patients to let me sleep in.

All of us want our patients to have the best treatment. We'd like to offer them a treatment that achieves 100% tumor control with no complications, and we don't have such a treatment. We do have stereotactic radiosurgery, and it is coming closer and closer to reaching that goal. If you discuss the complications with your patients honestly, talk about all the patients who are operated on, all the risks that they go through, and compare all those things with radiosurgery, most patients will decide that they would be better off with radiosurgery. Thank you.

Dr. Jens Thompsen (Copenhagen, Denmark): I am going to elaborate on some of the issues I discussed yesterday. First, it was our opinion some years ago–and this fits very well with what Derald Brackmann was just saying–that you should operate on these patients to achieve these goals. However, we have changed a bit because of our experience with the "wait and scan" group of patients. Again, our study was a prospective study that involved 123 patients. Six had cystic [disease] and nine were NF2 patients. If we omit the NF2 and cystic patients, the yearly growth rate was 2.4 mm, and it did not differ between the 30–60-year age group and the 60–80-year age group.

You cannot use patient age as a factor in deciding how and when to treat the patient. Clearly, cystic tumors grow very fast compared to other tumors, and if you see a picture like this on MRI, it's advisable that you operate on that patient as quickly as possible, because these tumors have a great propensity to grow. Here I show four pictures of a 78-year-old man taken at 2-year intervals, and this is where we made our mistake. We should have operated on the patient here, where the tumor has doubled in size. In this picture he is 80, this one was taken at age 82, and this was taken at age 84, when he was brought in and had to be operated on acutely. So, age will not tell you anything.

What might tell you something is symptom duration. Those who have a very short duration of symptoms tend to have much faster growing tumors. This also applies to elderly patients who come in with relatively acute symptoms. In fact, this is also supported by the experiments we have done on nude mice. The tumors we implanted in the nude mice that came from patients with a short history had a much higher growth rate than tumors from patients with a longer duration of symptoms. As I mentioned yesterday, we have designated five types of growth. One is type A, steady growth; B is no growth over time; C is a silent period with no growth; D is shrinkage (which is something we have to accept, and in large tumors this happens); and the E-type tumor is growing in a different way. In our series, 74% of tumors exhibited growth over time and 26% exhibited no growth or shrinkage. Growth was unrelated to age, sex, or initial tumor size, but it was related to tumor radiological architecture, and these are the cystic tumors.

If we introduce duration of symptoms, it is possible to derive a mathematical formula that will tell you whether or not a patient has a chance of having a growing tumor, but we have not yet started figuring such probabilities. With regard to hearing preservation, "wait and scan" is not a good tactic over time. If we use a 50/50 cutoff, 62% lose their hearing in the observation period, and at a 30/70 cutoff, about 70% lose hearing in the observation period–and this occurs even in patients with no tumor growth. Hearing can decline independently of whether the tumor grows or not.

In this study we followed patients until 1999; 85% of the tumors exhibited growth, 89% of patients lost eligibility for hearing preservation, and 6% died of the tumor. From these figures you could conclude that we should have operated on these tumors a long time ago in order to improve these results. However, you could also do a 180-degree turn and look at things differently. For example, 19% of the patients who died, died with a tumor but not because of the tumor. Moreover, 42% are alive and not being treated. Some of the tumors are growing, but the patients are still well, and they are quite happy with not having to undergo operation.

Some tumors, less than 30%, we treated along the way. They, of course, were the tumors that grew faster than the others.

Now we come to the change in attitude. Three or four years ago, the majority of studies in the literature said that most tumors did grow, but some studies said that less than 50% did so. We were reluctant to accept that figure because, as we have just shown, it was close to 90% if we waited long enough. We just said, from now on we are not operating on patients until we have shown that the tumors grow. This made us collect data on 225 patients from 1993 to 1998 with an evaluation time of at least 1 year. We excluded the cystic and NF2 patients, and then 51 only had one MRI study. When I looked at it a couple of months later, I was sure that they could be included. So, basically looking at an additional 162 patients, we got some different results with regard to the growth rate. Eleven patients had to be operated on, six died of unrelated causes, and three patients had radiation therapy. When we put all of this together, we can see that basically, these tumors grew less than 1 mm per year–with great variation, of course. If we look at types A and D, those that are not growing at all (and these are the tumors that are shrinking in the new series), 61% of the tumors did not grow in the observation time. So we had to change our attitude. This attitudinal change was also influenced by the Danish Acoustic Neuroma Association, which tried to force us not to operate on patents purposely. Of course, we were not doing operations against the patient's will, but that is the way they think.

They want to postpone everything, and the problem is, they are not explaining to patients what might happen. Later on, in talking to patients, we found that if you have a disease that allows you to wait almost forever, you don't take it seriously. Then, once you operate, if anything happens it's the doctor's fault because he should have recommended that you be treated earlier. Patients forget that they decide for themselves not to have the surgery. This is a fact of life in our country, and we have to accept it. Today we are not operating on any patient unless the tumor is more than 2 cm in diameter and we have two scans saying that this tumor is growing. Then, of course, we have to look at our facial nerve results in 900 patients operated on. Until the tumor reaches about 2 cm, there is no great increase in facial nerve problems. If the patient with a 10-mm tumor outside the meatus comes in and asks do I really need this surgery now? If it's 2 mm bigger or 3 mm bigger, will the facial nerve results be otherwise? We have to admit that this is not the case. Maybe we're just cutting the branch we are sitting on but we are being forced to operate on larger and larger tumors. This is my opinion of the "wait and see" tactic. It hasn't solved any problems for us, but it has forced us to change our attitude in respect to degree of aggressiveness in treating.

Dr. Kevin McKennan: Dr. Welling asked me to address the audience on an issue that is more philosophy than science: What is the neuro-otologist's role in gamma knife treatments? Every few years our subspecialty arrives at a fork in the road where we must choose either to take on a new technology or to maintain the status quo. Sometimes the status quo is superior. The field of neurotology has encountered many such decision points in the past as result of pioneering work by people like Dr. William House. We perform cochlear implants, vestibular neurectomies, and acoustic neuroma resections. Each of these procedures has required us to learn new surgical techniques and take on new technologies. Before we decide what role the gamma knife will play in neurotology, it is helpful to review its history and progress today.

The gamma knife was first developed by a Swedish neurosurgeon, Dr. Lars Lexcell, in the 1960s. Twenty years later, in 1987, the first gamma knife unit was installed at the University of Pittsburgh, where Dr. Flickinger practices. During the first few years of use, the gamma knife had no significant

impact on the treatment of acoustic neuromas in the United States because Pittsburgh's unit was the only one available.

I thought, and I recall colleagues commenting, that the unit in Pittsburgh was somewhat of an oddity, that it really didn't affect our practices. I cannot recall a single patient in my practice asking about gamma knife treatment prior to 1998. Now, with the expansion of the Internet, I would say that probably 50% of my patients inquire about the gamma knife, whether I bring it up or not. There are now 112 gamma knife units throughout the world. More than 60,000 patients have been treated, and more than 8,000 acoustic neuromas.

During the past 10 years, 42 gamma knife units were installed in the United States. This proliferation of gamma knife treatment centers occurred because of the effectiveness and low morbidity of the technique, not necessarily in application to acoustic neuromas, for it has many other application's.

With regard to acoustic neuromas, a hundred articles have been published reporting the results of gamma knife treatment in acoustic neuromas. For example, in 1998 Dr. George Noren noted that in a series of 669 patients, tumor control occurred in 95% of patients who were followed up for a minimum of 5 years. Hearing was preserved in 65%, and facial nerve weakness occurred in only 2% of patients. These results must be weighed against the results of the natural course of the disease and the results of surgery, but there are numerous other articles, many from the University of Pittsburgh, reporting similar short-term results with the use of the gamma knife.

The continued treatment of acoustic neuromas with the gamma knife is evidence that the gamma knife has a role to play in treating acoustic neuromas. We need to define that role. Elderly patients with small to medium-size tumors that are growing are probably ideal candidates for this treatment. They may not necessarily need the long-term solution afforded by surgical excision. Patients with recurrent or residual tumors, those with tumors in an only hearing ear, and patients who have medical contraindications to surgery are certainly candidates for gamma knife treatment.

On the other hand, the most ardent supporters of the gamma knife advise against its use in large tumors with brain stem or cerebellar compression. The long-term role of gamma knife treatment is uncertain in patients with small to medium-size tumors, especially those with good hearing. Either neurotologists will help determine the role or others will determine it without our input or expertise.

As I see it, we have three choices. First, we can stick our heads in the sand like ostriches and simply refuse to accept the repeated published results of successful gamma knife treatment. Second, we can refer all of these potential acoustic neuroma patients to a neurosurgeon who performs gamma knife radiosurgery. I think this is unnecessary and not in the best interest of patients. We as ENT surgeons diagnose the vast majority of acoustic neuromas. We have the best understanding of the auditory, vestibular, and facial nerves. We are the best trained to treat the complications of cranial nerve lesions. We have the diagnostic and research capability to study the effects of gamma knife therapy on acoustic neuromas. When all of these factors are considered, the second choice of referring patients to a neurosurgeon who performs gamma knife surgery seems as absurd to me as simply ignoring the literature. The third and most logical option for neurotologists is for us to perform this treatment ourselves when the treatment is indicated.

By having both surgical and gamma knife privileges, we would be able to offer either treatment to potentially every patient with an acoustic neuroma. We would be able to discuss with firsthand knowledge the pros and cons of each treatment. These two treatments are not necessarily mutually exclusive. Many neurosurgeons, including those I work with, practice both surgery and gamma knife radiosurgery. Neuro-otologists can do the same. Last year I came to the conclusion that I would like to use the gamma knife in my practice in selected cases of acoustic neuromas and other skull-base tumors, especially those of the jugular foramen.

A number of otologists here today have expressed a similar interest. In May 1999 I took a weeklong gamma knife course in London. I submitted my proposal to our hospital's gamma knife unit. I had the support of several local neurosurgeons, all of my ENT colleagues, and many other physicians on the hospital staff.

Unfortunately the neurosurgical director of the unit vigorously opposed my proposal. I know of similar opposition in virtually every other center in the United States. At this time, all 42 gamma knife centers in the United States are co-directed by neurosurgeons. If our specialty does not break this monopoly, we will lose the opportunity to define the role of the gamma knife, not only in the treatment of acoustic neuromas but in the treatment of other tumors of the skull base as well. In the end, our patients may suffer from this turf battle. If the gamma knife proves to be a successful long-term management tool, we, not the neurosurgeons, should be using it. If the gamma knife does not provide successful long-term management, that

also needs to be exposed. As for the proverbial fork in the road I pointed to earlier, I think we have only one choice, and that is to get involved with the gamma knife and define its role.

Dr. Welling: We have some diverse opinions on the panel; that's good. Could we have the next tray of slides, please? Let's go through a couple of cases and get the feelings of the panel.

The first case is of a 56-year-old retired nurse who came in with a 6-month history of right-sided tinnitus and mild hearing loss. Her past medical history was most significant for insulin-dependent diabetes and a coronary artery bypass graft 4 years previously. She was asymptomatic. She had searched the net. She came in with a note from her cardiologist saying that it was okay if she had surgery, although she was at increased risk. The physical examination was otherwise unremarkable. An audiogram showed very mild high-frequency loss but essentially normal discrimination, almost symmetric hearing, and a fairly small intracanalicular tumor. Dr. Brackmann, how much role do you allow your patient in the choice of treatment, and what would you recommend to this patient?

Dr. Brackmann: The treatment choice is always the patient's. You apprise them of the possibilities, including radiotherapy, and then they make the decision. Because of her special medical problems, I would recommend repeating her scan in 6 months and doing nothing at this time to see if this is a growing tumor. I do that in almost all tumors except the small tumors in patients with good hearing, where there is a significant risk to patients' hearing if they wait. If she did not have medical contraindications or relative contraindications, I would recommend surgery right away but since she does have contraindications, I would follow her for the present.

Dr. Flickinger: I think the standard approach for somebody who is at medical risk, and also the standard approach for a small tumor of this size, is observation. For years, that is what we always presented. Even patients who really wanted radiosurgery we for the most part talked out of it for something this small–particularly when we were starting out, when we thought the risk of hearing loss and facial weakness would be the same for all of these tumors. With the newer results, and seeing how many patients have lost their hearing over time, I now tend to offer longer observation or radiosurgery as equal choices. I still lean toward observation. I feel better psychologically if I can follow patients closely and pick up hearing loss when it is still very slight, or at the earliest sign of progression, and treat them at that point. I think it's a little bit better, but with the newer results, we have found concern about trigeminal neuropathy. If you treat these tumors while they are still intracanalicular, then there should be zero risk of developing any facial numbness, and also the risk of hearing loss should be less. We have had no patient lose hearing with moderate doses in intracanalicular tumors. One patient dropped in hearing from grade 1 to grade 2, so I think that such patients have about an equal choice between observation and radiosurgery. With the medical contraindications, microsurgery could be considered, but you would want to think twice about that.

Dr. Welling: Dr. Flickinger, there was a report out of Seattle recently that showed that intracanalicular tumors seem to be associated with a higher rate of facial nerve injury at the time of radiation, although I think the dose used was around 18 Gy. Is it more difficult to irradiate a small intracanalicular tumor than one that has more mass to it, in the CP angle?

Dr. Flickinger: I don't think so. That may have been a function of the high doses used in early radiosurgery. In the early days of surgery planning, and particularly for the small tumors, they were probably treating volumes that were much larger, so even though they quoted 18 Gy, that dose is the minimum dose. The dose to the facial nerve may actually have been higher than it is with modern planning. Now we use lower doses to begin with, so that may turn out to be a different problem. But certainly with the low dosages and modern techniques, we have found zero risk of facial neuropathy.

Dr. Welling: Does the panel have any treatment recommendation for this patient other than wait and watch?

Dr. Thompsen: I would obviously wait and watch in this patient, but I would also warn against doing MRI too often. If the average growth rate of these tumors is somewhere between 1 and 2 mm [per year] and you obtain an MRI study after 6 months, you may not be able to discern any difference between the two images. We would wait at least 1 year between imaging studies in order to control the tumor in this patient.

Dr. Welling: Do you fear missing the patient who has a tumor growing at 2 cm per year if you don't get that first 6-month scan?

Dr. Thompsen: Not in this situation. If it was a cystic tumor, then the situation would be different, but in the solid tumors, we are not seeing a 2-cm expansion.

Let's go to our next case.

Dr. Welling: This is a 39-year-old man who un-

derwent a suboccipital excision of a right acoustic neuroma at age 32 with profound sensorineural hearing loss and seventh nerve loss of continuity. A primary seventh nerve anastomosis was performed. When he underwent follow-up MRI at 2 and 4 years, there was no evidence of recurrence. Now, however, at the 7-year follow-up, he does show some evidence of recurrence. His facial nerve is House grade 3 with moderate synkinesis. He does achieve full eye closure with effort. The audiogram confirms a profound sensorineural hearing loss and suboccipital exposure, profound hearing loss, and then a small recurrent tumor after suboccipital excision. Dr. Linthicum presented some interesting data yesterday about the influence of the ganglia on possible recurrence. Dr. Brackmann, what is the difference in recurrence in your practice in terms of tumors that have been removed suboccipitally versus through a translabyrinthine approach?

Dr. Brackmann: Brad, as you know, we don't have a great suboccipital series. The ones that we have seen have primarily been treated elsewhere. About 32 patients have now come to us after having undergone suboccipital surgery elsewhere with recurrent disease that we have removed by a translab procedure. Usually the recurrence is here in the suboccipital approach. In our translab series we have had seven recurrences in over 4,000 cases, so it is extremely uncommon, and all of the lesions have been very large tumors. Specifically in this case, Brad, how was the facial nerve reconstructed?

Dr. Welling: It was by a primary anastomosis.

Dr. Brackmann: From where to where?

Dr. Welling: It was in the cerebellar pontine angle toward the meatus, where it was reconstructed, I believe.

Dr. Welling: What would your treatment options be? This fellow actually said that he would rather die than go through facial rehabilitation again.

Dr. McKennan: In a 39-year-old patient who is already deaf in the ear, I would do a translab excision. With modern monitoring techniques, I would anticipate being able to resect the tumor completely and cure him of the problem. That would not be my recommendation if this fellow were 69 years old. That is a different story. In a 39-year-old I would also have concerns about the long-term management over his expected lifetime, which might be 40 years more.

Dr. Brackmann: That would be my preference too. I would not expect the facial nerve to be greatly involved within the internal auditory canal, and I think you could preserve it. He may have already made the decision, although if he said he would rather die than risk facial nerve weakness, that would probably be the decision that he would make not to undergo operation.

Dr. Flickinger: It seems that a lot of patients and physicians think that if you have two different approaches to control a tumor, then, if the first one you try fails, you can try the other, so in that respect, radiosurgery after failed surgery seems logical. But the tumor control rate for these recurrent tumors may be lower with radiosurgery, perhaps down around 90%. This man is a young patient, so the tumor is certainly accessible by a translab approach. It's a small tumor, and we would expect good results with microsurgery, but I would present that as the first treatment option, and I would certainly be willing to do radiosurgery if the patient refused that.

Dr. Thompsen: I would recommend translab removal but would not promise anything about the facial nerve. If the patient is adamantly against any deterioration, then you must wait and see what happens, and then the next time, if the tumor is bigger, he may say, Okay, let's take it out.

Dr. Welling: The next case is of a 21-year-old asymptomatic woman whose father has NF2. Her audiogram is normal, but she has a small intracanalicular tumor on the left and a 2.5-cm brachial plexus tumor. Here is her audiogram. Here is the tumor. Who on the panel would take this out surgically? Who would wait and watch this tumor? Is radiation the primary treatment modality?

Dr. Brackmann: You know that NF2 tumors will grow–why wait and watch? It's a given that they are going to grow, so that's the standard of care in this country: to diagnose NF2 or find asymptomatic patients, and to screen siblings or children and then watch them. You watch them until the lesions are too big to save their hearing. You know she has a disease that will progress, so why wait? Your best chance to save her hearing for her lifetime is to take that tumor out.

Dr. Welling: We went ahead and took this tumor out. The patient did lose her hearing when we took it out,. She was very interested in a prophylactic vestibular nerve resection on the opposite side. Would you consider that?

Dr. Brackmann: We've considered it but we've never done it.

Dr. Mckenna: Dr. Brackmann, I would wait before operating on this patient. She is totally asymptomatic, and I have been impressed with the incredible variation from patient to patient with NF2–even within the same patient. A patient can have a 4-cm tumor on one side and on the opposite side a tiny tumor that may not grow for many, many years. So I don't think you are gaining

anything by operating on a patient who is totally asymptomatic with a small tumor on one side. I think you can safely wait in this case.

Dr. Welling: Thank you. We could stay here the rest of the morning, and probably the rest of the day, discussing interesting cases and management. I thank our panelists. One final short comment.

Dr. Thompsen: I have a comment about the middle fossa, which is of course what Derald had in mind when taking this tumor out. It sounds very reasonable, but I am not sure that everybody can do that with the same success that you have had. At least we have not been that successful operating on our patients to preserve hearing. We have obtained EEGs in all of our middle fossa cases before and after and several years after middle fossa surgery. Over 90% of these patients have EEG changes that are permanent, and on paper the figure looks serious. They don't have any symptoms whatsoever, but in a pilot study, it might create a problem. So we are not that happy about middle fossa surgery. It could be that we are not good enough.

Dr. Welling: There's room for further study on this matter, and I hope we will have the continued support of the membership for prospective blinded controlled studies.

AN INTERACTIVE THREE-DIMENSIONAL COMPUTER MODEL OF THE TEMPORAL BONE

Masayuki Inouye, M.D., Joseph Roberson, M.D., Kevin Montgomery, Ph.D., and Michael Stephanides, M.D.

ABSTRACT

Hypothesis/Goal: Development of a three-dimensional interactive computer model of the temporal bone.

Background: Learning temporal bone anatomy is an integral part of every otolaryngology residency program. The standard curriculum requires temporal bone dissections, operative experience, and examination of histology. Essential to a working understanding of this complex anatomy is the ability to conceptualize the temporal bone in three dimensions. Computer-generated models are the newest addition to the teaching armamentarium. Recent advances in bioimaging and computer technology have enabled the creation of an anatomically accurate three-dimensional model of the temporal bone.

Methods: Fifty serial histologic sections of the temporal bone were scanned into a silicon graphics indigo Elan computer. The images were then processed using RAVE (reconstruction and visualization environment) software, which was developed at this institution. Contours were drawn around various structures, including ossicles, nerves, vessels, and the cochleovestibular system. These contours were then registered and a three-dimensional surface mesh was created. RAVE visualization software was then used to produce a three-dimensional model of each structure. The features of this program include the ability to add or remove any object; to control proximity, rotation, color, and transparency; to produce a cutting plane; to visualize stereoscopically; and to manipulate the model in virtual reality with real otologic instruments which are tracked in space.

Results: An interactive three-dimensional computer model of the temporal bone.

Conclusions: Although otologic training will continue to be based on temporal bone dissections and operative experience, advances in computer technology have allowed the creation of an innovative adjunct to the teaching armamentarium.

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HISTOPATHOLOGY OF RESIDUAL AND RECURRENT CONDUCTIVE HEARING LOSS FOLLOWING STAPEDECTOMY

Joseph B. Nadol, Jr., M.D.

ABSTRACT

Hypothesis: Histopathologic analysis of temporal bones from patients who in life had undergone stapedectomy may provide new information concerning the causes of both residual and recurrent conductive hearing loss (CHL).

Background: Although closure of the air-bone gap to within 10 dB occurs in approximately 90% of primary stapedectomies, a residual CHL occurs in approximately 10% and recurrent CHL may occur in up to 35% of cases. Revision surgery has provided clinical information concerning putative causes of failure of the primary surgery, including erosion of the incus, bony regrowth at the oval window, and displacement of the prosthesis. Most reports on the histopathology of temporal bones from such patients have focused on complications of surgery, with little attempt to correlate the postoperative air-bone gap with the observed histopathology.

Methods: A retrospective review of our collection of temporal bones uncovered 22 cases with a postoperative CHL of 10 dB or greater (air-bone gap averaged at 500, 1000, 2000, 3000, and 4000 Hz, using postoperative air and bone conduction levels) following stapedectomy. These temporal bones were prepared by standard methods for light microscopy.

Results: Of the 22 cases with postoperative CHL equal to or greater than 10 dB, there were 19 with residual CHL, 2 with recurrent CHL, and 1 with both residual and recurrent CHL. The most common histopathologic correlates of residual and recurrent hearing loss included resorptive osteitis of the incus (64%), obliteration of the round window by otosclerosis (23%), the prosthesis lying on a residual footplate fragment (23%), the prosthesis abutting the bony margin of the oval window (18%), adhesions in the middle ear (14%), and new bone formation in the oval window (14%). The mean postoperative CHL in those temporal bones with round window obliteration (n = 5) or resorption of the incus (n = 14) was 38 dB and 27 dB, respectively. Those cases with three findings had a greater postoperative conductive hearing loss than those with one finding.

Conclusions: Histopathologic examination of temporal bones from patients who in life had undergone stapedectomy provides useful information concerning the causes of both residual and recurrent CHL. These data provide a basis for improving both surgical technique and prosthesis design.

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HISTOLOGIC STUDIES OF THE POSTERIOR STAPEDIOVESTIBULAR JOINT IN OTOSCLEROSIS

Saumil N. Merchant, M.D., Armagan Incesulu, M.D., Robert J. Glynn, Sc.D., and Joseph B. Nadol, Jr., M.D.

ABSTRACT

Objective: To determine the prevalence of ankylosis or otosclerosis at the posterior stapediovestibular joint (SVJ) in temporal bones with otosclerosis, with special reference to stapes surgery.

Background: The long-term success of the laser STAMP procedure, anterior crurotomy, and similar partial stapedectomy procedures depends on lack of ankylosis and lack of otosclerosis involving the posterior SVJ. Previous work has shown that the air-bone gap in otosclerosis correlates with narrowing and loss of the SVJ space. However, the prevalence of and histologic features of otosclerotic involvement of the posterior SVJ space have not been well characterized.

Methods: Histologic assessment of serial sections through the oval window niche in 140 temporal bones with otosclerosis that had been sectioned in the axial plane (age range, 20–95 years, mean = 68). Bones with stapes mobilization or stapedectomy were excluded.

Results and Conclusions: Two of 140 bones had otosclerosis exclusively at the posterior SVJ. Of the remaining 138 bones, all of which had otosclerosis at the anterior SVJ, 82 bones also had otosclerosis at the posterior joint. Of the 56 bones without otosclerosis of the posterior joint, three exhibited bony ankylosis of the posterior joint. Thus, 53 bones (38%) had neither ankylosis nor otosclerosis involving the posterior joint, and would be potentially suitable for a laser STAMP or a similar procedure.

There was no correlation between otosclerosis at the posterior SVJ and age, sex, or duration of conductive hearing loss. Otosclerosis at the posterior joint in one ear was significantly associated with its presence at the posterior joint in the opposite ear ($P = 0.01$).

The audiogram could not be used to reliably predict otosclerotic involvement of the posterior SVJ or the degree of footplate pathology such as ankylosis.

Supported by funding from the Silverstein Young Investigator Award, the Lynch Foundation Research Endowment, and NIH grant No. R29 DC003657.

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A COMPARISON OF ENG RESULTS WITH POSTUROGRAPHY FINDINGS FROM THE BALANCETRAK 500

Manali Amin, M.D., Marian Girardi, M.A., Horst R. Konrad, M.D., and Larry F. Hughes, Ph.D.

ABSTRACT

Objective: To determine a correlation between conventional electronystagmography (ENG) findings and results obtained from BalanceTrak 500 posturography assessment.

Study Design: Individuals with a variety of dizziness and balance disorder symptoms were tested with both ENG (ocular motor studies, positional/positioning testing, and caloric testing) and with computer posturography using the BalanceTrak 500J.

Setting: Tertiary referral center.

Patients: Urban/rural Midwesterners referred for dizziness and balance dysfunction symptoms.

Intervention: Results of both testing modalities were sent to referring physicians.

Outcome Measures: ENG and posturography results.

Results: When ENG results were compared with BalanceTrak findings, a majority of those patients whose ENG findings indicated central and mixed etiologies, or peripheral lesions other than BPPV, had abnormal findings on posturography. Specifically, tests similar to the Balance Master Sensory Organization Tests (SOT) 4 and 5 and a new test, Limits of Stability (LOS), presented the most difficulty for these individuals. Patients with normal ENG findings and those with BPPV had mixed results on posturography.

Results for specific individual ENG tests were compared with posturography findings. No correlation was noted among any of the ENG results and posturography findings. Furthermore, there was no correlation between posturography and dizziness etiology.

Conclusion: For many patients with dizziness and/or balance dysfunctions, posturography can provide additional information to that obtained with ENG testing. This is especially apparent for individuals who present with these symptoms but have normal or borderline normal ENG findings.

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A VESTIBULAR PHENOTYPE FOR WAARDENBURG'S SYNDROME?

F. O. Black, M.D., F.A.C.S., S. C. Pesznecker, R.N., K. Allen, M.S., and Claire Gianna, Ph.D.

ABSTRACT

Objective: To investigate vestibular abnormalities in subjects with Waardenburg's syndrome.

Study Design: Retrospective record review.

Setting: Tertiary referral neurotology clinic.

Subjects: Twenty-two adult white subjects with the clinical diagnosis of Waardenburg's syndrome (10 with type I and 12 with type II).

Interventions: (1) Evaluation for Waardenburg's phenotype, (2) review of history for vestibular and auditory symptoms, and (3) tests of vestibular and auditory function.

Main Outcome Measures: (1) Results of phenotyping, (2) results of vestibular and auditory symptom review (history), and (3) results of vestibular and auditory function testing.

Results: There were 17 women and 5 men in the study. The age range was 21–58 years (mean age, 38 years). Sixteen of the 22 subjects presented with a chief complaint of vertigo, dizziness, or imbalance. For subjects with vestibular complaints, vestibulo-ocular tests (calorics, vestibular autorotation, and/or pseudorandom rotation) were abnormal in 77% and vestibulospinal function tests (computed dynamic posturography, EquiTest) were abnormal in 57%, but there were no specific patterns of abnormality. Six had objective sensorineural hearing loss. Thirteen had an elevated summating/action potential (>0.40) on electrocochleography. All subjects except those with severe hearing loss (n = 3) had normal auditory brain stem responses.

Conclusion: Subjects with Waardenburg's syndrome may present with a primary complaint of vestibular symptoms without hearing loss. Electrocochleography and vestibular function tests appear to be the most sensitive measures of otologic abnormalities in persons with Waardenburg's syndrome who present with vestibular complaints.

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DISCUSSION PERIOD VIII: HISTOPATHOLOGY/VESTIBULAR DISORDERS/ANATOMY Papers 26–30

Dr. C. Gary Jackson (Nashville, TN): These papers are now open for discussion.

Dr. Herbert Silverstein (Sarasota, FL): I really enjoyed Dr. Merchant's beautiful paper. When I first started doing this laser STAMP procedure 5 years ago I called Dr. Schuknecht and asked him if this would work, and he said if you pick the right cases with minimal otosclerosis, it probably will work. That was the impetus to ask Joe Nadol to look at the temporal bone to see what would happen. Over the past 5 years we haven't had to re-fix any patient. Right now we have 50 patients. In about 30% of them, after a laser stamp procedure you have a beautiful blue footplate, minimal otosclerosis, and you think you're going to have a great result. Then you touch the stapes, and it is still fixed. This fits the histologic picture that Dr. Merchant detailed, in that about 30% of them have otosclerosis, and you can't actually see the posterior part of the footplate when you're in surgery until you try to mobilize the stapes. So I'm very pleased, and I think this explains our results. If we pick the right cases, we can still give the patient good hearing. If you can't do a stamp procedure you just convert it to a piston procedure at the same time.

Dr. Allan Rubin (Toledo, OH): When we were in Omaha, we would see patients with Waardenburg's syndrome. The presentation was similar to Ménière's disease, so we used to treat them with diuretics. Do you maintain the same type of management, or is there something different with the Waardenburg's syndrome patients?

Dr. F. O. Black (Portland, OR): Thank you for the comment. Yes that's true, we treated them exactly as we do hydrops patients–conservatively. The good news is that they usually respond better than patients with Ménière's disease to conservative management.

Dr. Edward Monsell (Detroit, MI): I have a question for Dr. Nadol. A paper from the Warren group about 2 years ago suggested an association between chronic suppurative otitis media and incus erosion following stapes surgery. I wondered if you saw any evidence of that.

Dr. Joseph B. Nadol (Boston, MA): I'm not sure I understand the question, but certainly the necrosis of the incus can occur in chronic otitis media.

Dr. Monsell: Specifically, following stapes surgery, incus erosion seemed to be more prevalent in patients who had a history of chronic suppurative otitis.

Dr. Nadol: Well, in 19 cases, there were none like that. I think in all of these cases there was no evidence of any otologic disease other than otosclerosis and the surgery that they underwent.

INTRODUCTION OF NEW PRESIDENT: A. JULIANA GULYA, M.D.

C. Gary Jackson, M.D.

The next order of business is the introduction of our new President. I'm sure you all recognize by now that the election of Dr. Gulya to the presidency of the American Otological Society is a historical event. This notwithstanding, her career and unique skills portend a presidency of great vitality and purpose. Without further ado, I would like to introduce A. Juliana Gulya as the next President of the American Otological Society.

REMARKS OF NEW PRESIDENT

A Juliana Gulya, M.D.

Thank you very much. I am really looking forward to it myself, but I would be remiss if I did not recognize my eminent predecessor, Gary Jackson. Throughout your career you have consistently distinguished yourself by excellent performance, and I believe the American Otological Society has indeed benefited from your leadership–most recently and most relevantly from your service as President. To commemorate your year as president, on behalf of the American Otological Society I would like to present you with this gold lapel pin and a certificate which reads, "American Otological Society Inc., presented to C. Gary Jackson, M.D., President, 2000, in appreciation and recognition of his service to this society."

Next year we will be in fabulous Palm Desert, California. I invite all of you to attend and, more important, to participate in this meeting. Without further ado, I will call this 133rd Meeting of the American Otological Society to a close. Thank you.

EXECUTIVE SESSIONS

BUSINESS MEETING

MINUTES–May 13–14, 2000 President C. Gary Jackson called the Business meeting to order at 12:30 p.m. The minutes of the April 24–25, 1999, Annual Meeting of the American Otological Society, Inc., held at Marriott's Desert Springs Resort, Palm Desert, California, were approved.

The following new members were introduced to the Society by their respective proposers:

Active Members

Stephen P. Cass, M.D., proposed by Eugene Myers, M.D., and seconded by Malcolm D. Graham, M.D.; Saumil N. Merchant, M.D., proposed by Joseph B. Nadol, Jr., M.D., and seconded by Michael J. McKenna, M.D.; Lorne S. Parnes, M.D., proposed by Brian F. McCabe, M.D., and seconded by Bruce J. Gantz, M.D.; Debara L. Tucci, M.D., proposed by Robert A. Jahrsdoerfer, M.D., and seconded by: Paul R. Lambert, M.D.

Corresponding Members

Vicente G. Diamante, M.D., proposed by Robert A. Jahrsdoerfer, M.D., and seconded by Mansfield F. W. Smith, M.D. (Dr. Diamante was unable to attend this meeting); Takeshi Kubo, M.D., proposed by Richard T. Miyamoto, M.D., and seconded by J. Gail Neely, M.D.;

Thomas P.U. Wustrow, M.D., proposed by Robert A. Jahrsdoerfer, M.D., and seconded by Douglas E. Mattox, M.D.

A Nominating Committee composed of Drs. Joseph Farmer (Chairman), John McElveen, Clough Shelton, Richard Wiet, Derald Brackmann, and alternate Dr. Richard Miyamoto was elected to prepare the slate of nominees for AOS officers for 2000–2001.

REPORT OF THE SECRETARY-TREASURER

Dr. Horst R. Konrad presented the following items of information:

The present Membership totals 274 and includes the induction of new members on May 13, 2000, as follows:

126 Active	10 Honorary
72 Senior	7 Emeritus
43 Associate	16 Corresponding

The AOS Membership Development Committee continues to seek out qualified candidates who would be worthy of proposal for membership in the Society. Dr. Konrad encouraged the membership to continue to propose qualified candidates for membership in the Society. The society is particularly interested in proposing candidates for ACTIVE membership.

Members deceased since the last Annual Meeting are W. Hugh Powers, M.D. (Senior), and Cesar Fernandez, M.D. (Associate).

Members requesting transfer to Senior status are Robert W. Cantrell, M.D., Michael M. Paparella, M.D., and Mansfield F. W. Smith, M.D.

Requesting transfer to Emeritus status is B. Hill Britton, M.D.

INCOME AND EXPENSE STATEMENTS

The following Income and Expense statements were presented to the membership.

AMERICAN OTOLOGICAL SOCIETY, INC.
INCOME STATEMENT
July 1, 1999, to April 30, 2000

Beginning Balance (July 1, 1999)	$ 89,321.32
Membership Dues	52,200.00
Transactions Income	2,405.00

Research Fund 18,532.00
Interest & Dividends.....4,084.90
Certificate of Deposits 75,000.00
COSM 17,125.00
AJO.....7,500.00
Total Income $176,846.90

AMERICAN OTOLOGICAL SOCIETY, INC.
EXPENSE STATEMENT
July 1, 1999, to April 30, 2000

Certificates of Deposit (4 @$25,000).....$100,000.00
Accounting Fees 7,600.00
Annual Meeting 1,695.50
Midwinter Council Meeting 12,058.22
Office Expenses.....3,092.16
AOS Secretarial Stipend 5,250.00
ACCME (Dues & Reaccreditations).....5,050.00
Internal Revenue Service 9,300.00
NY State Dept. of Law 250.00
Insurance (Dir. & Off., AOS/Res Fund) 5,281.00
Other Expenditures–Subscriptions, Transactions, Office Rent, Acoustical Society Membership, Misc.). .36,415.97
Total Disbursements.....$185,992.85

AMERICAN OTOLOGICAL SOCIETY, INC.
FINANCIAL STATEMENT
July 1, 1999, to April 30, 2000

Balance on Hand (July 1, 1999) 89,321.32
Deposits: Income.....176,846.90
Total $266,168.22
Disbursements.....185,992.85
Balance in Checking (4/30/00).....80,175.37
Certificate of Deposit 25,000.00
Balance on Hand (April 30, 2000) 105,175.37

EDITOR-LIBRARIAN REPORT Dr. Julianna Gulya reported that Lippincott–Williams & Wilkins would be publishing the 1999 Transactions. The price remains stable. The archives are missing the volumes for the years 1882, 1919–1924, and 1928. We are still in search of these missing volumes to complete the collection housed at the Adams Center. Dr. Gulya thanked the membership for allowing her to serve as Editor-Librarian of the American Otological Society.

Members were reminded to pick up their numbers for the annual photograph, which was taken immediately following the afternoon program.

PROGRAM ADVISORY COMMITTEE

Dr. Jackson thanked the following individuals for serving on the 2000 Program Advisory Committee: Drs. F. Owen Black, Richard Chole, Bruce J. Gantz, L. Gale Gardner, Herman A. Jenkins, Paul R. Kileny, John P. Leonetti, Brenda Lonsbury-Martin, John K. Niparko, Dennis S. Poe, and Clough Shelton.

PRESIDENT'S REMARKS, INTRODUCTION OF GUEST OF HONOR, PRESIDENTIAL CITATION, May 13, 2000

The Business Meeting was adjourned and the first Scientific Session started at 1:00 p.m. with very brief remarks from President C. Gary Jackson. The President introduced the Guest of Honor, Derald E. Brackmann, M.D. The Presidential Citation was presented to Mr. William B. Williams III.

MINUTES–May 14, 2000

President C. Gary Jackson called the Business meeting to order at 7:00 a.m.

REPORTS OF COMMITTEES

Board of Trustees of AOS Research Fund: Dr. Horst R. Konrad presented to the membership the Research Fund Report in Dr. Douglas Mattox's absence. The value of the Research Fund as of May 15, 1980, was $1,900,000. During the 20-year period to March 24, 2000, the value of the Research Fund increased to $10,535,007.00. The total expenses to the fund for 1999–2000 were $492,608. This amount reflects the administrative fees in the amount of $115,525 and grants totaling $377,148. The budget for 2000–2001 is 258,711.00. The amount reflects administrative fees in the amount of $148,000 and grants totaling $110,711. The increase in administrative fees represents an increase in the investment advisor's fees. New initiatives for the AOS Research Fund include an AOS Scholars Grant to support young investigators with a minimum of 50% time commitment. It provides salary support in the amount of $70,000.00 and research supplies in the amount of $10,000. The first applications will be accepted January 31, 2001. The Research Fund reviewed and recommended for funding by AAO-HNS CORE grant committee a Resident Research Award in the amount of $15,000 per year. At the Trustees meeting on April 1, 2000, eight grants were received and reviewed. The total amount of the requests was $291,925. Two grants were funded (one new and one renewal). The total cost of the funded grants was $110,711.

American Board of Otolaryngology: Dr. Julia Gulya reported on the activities of the Board. The Board continues to administer a two-part examination. Three hundred thirty-four (334) candidates took the written examination in October 1999. Of those individuals, 307 became candidates for the oral examination. One hundred twenty individuals, including ABOto Directors, Senior Examiners, and Guest Examiners, on April 9–10, 2000, conducted the oral examination. Three hundred forty-six (346) candidates for oral boards were examined. Three hundred twenty-one passed the examination and became certified. Dr. Michael E. Johns was elected President of the ABOto, to serve a two-year term. The 2000 written examination will be conducted on Monday, September 18, 2000, in five cities. The oral examination will be conducted in Chicago on April 20–21, 2001.

Dr. Michael E. Johns was elected President of the ABOto, to serve a two-year term. Dr. David E. Schuller was elected President-Elect, also for a two-year term. Dr. Gerald B. Healy was re-elected to a second term as Executive Vice-President. Dr. H. Bryan Neel III continues in his term of service as Treasurer. Drs. Jerome C. Goldstein, Alexander J. Schleuning, and Neil O. Ward were elevated to Senior Counselors. Drs. Richard A. Chole, Jack L. Gluckman, and Jesus E. Medina were elected to the Board of Directors.

American Academy of Otolaryngology: Dr. G. Richard Holt, Executive Vice-President of the American

Academy of Otolaryngology–Head and Neck Surgery, reported on the activities of the Academy. Government and Legislative Affairs: The Infant Hearing and Screening Act was passed. It was universally adopted across the country. The patients' rights bill, which has been strongly supported by the Academy, is now in conference committee, where the disjunction between House and Senate will be worked out. This legislation would allow physicians, not managed care personnel, to make medical decisions; hold health plans accountable when they make bad decisions not in the patient's best interest; initiate a timely and independent appeals process for patients; and mandate adequate access to physicians, including specialists. Prompt Payments Acts of 2000: A number of states have successfully passed this legislation. The Board of Governors and the grassroots legislative network continue to work hard for national passage. Practice Affairs: The RUC and PEAC committees continue to work hard to update CPT codes and to identify those that are undervalued. The Academy plans to conduct a reimbursement and coding meeting for representatives from all societies to plan how otolaryngology can work toward timely and complete response to HCFA and other agencies on coding and reimbursement issues.

The Correct Code system, produced by McKesson HBOC and used by a number of payers, has been shown by the Academy to be faulty, especially with respect to bundling surgical procedures together that make no sense and based on inaccurate principles. Reimbursement for codes that require the use of the operating microscope has been very problematic, especially with combined procedures with neurosurgeons. The Academy is currently working on clarifying those otologic codes that require the use of the microscope through the CPT/RUC committee.

The Academy is very close to launching a new Internet portal for otolaryngology–ENTLink. ENTLink will provide full-service capability for the practitioner, both community and academic, and will link seamlessly to the Academy and Foundation. ENTLink partnering with another company for practitioners will be an expense. Different amounts have been suggested, but it will probably cost less than $1,000 a year to set up a web site with access to patient codes. A demonstration web site will be up at COSM this year. Very soon the popular Antimicrobial Pocket Guide, by Dr. David Fairbanks, will be available for downloading to a Palm Pilot.

REPORT OF THE AMERICAN COLLEGE OF SURGEONS

Dr. Richard Wiet, American College of Surgeons Governor representing the American Otological Society, updated the membership on the activities of the American College of Surgeons. The College has 62,000 members and assets totaling $245 million. The College was formed in 1919; currently 3,972 members are otolaryngologists.

The College has received a report that the Council of Medical Specialty Societies (CMSS), with the American Board of Medical Specialties (ABMS), has met to consider an umbrella organization responsible for overseeing the development of physician assessment.

The Board of Governors has recommended revision of the Guidelines for Optimal Ambulatory Surgery and Office-Based Surgery. The Board also recommended that a statement on Physicians as Expert Witnesses be distributed to American College of Surgeons Fellows for use in trial settings.

The College has initiated ten ACS research fellowships for the year 2000–2001. The fellowships are provided to assist young surgeons in establishing research programs. The award is $35,000 per year for two years.

A report of the Council of Advisory Chairs disclosed Dr. William House's name has been submitted in consideration for the Jacobson's Award for Dr. House's contribution to acoustic neuroma surgery and cochlear implant surgery.

The next course in Otolaryngology will be at the Clinical Congress, to be held in Chicago on October 22–27, 2000, with a focus on cervical paragangliomas and vagal tumors.

REPORT OF THE AWARD OF MERIT COMMITTEE

Dr. Charles M. Luetje, Chairman, reported that he had conferred with his committee members, Drs. C. Gary Jackson, Gregory J. Matz, F. Owen Black, and Paul Lambert, for the selection of the 2000 Award of Merit recipient. Dr. Robert A. Jahrsdoerfer was the recipient of the award at the banquet held on Sunday evening, May 14, 2000.

REPORT OF THE AUDIT COMMITTEE

Dr. Donald B. Kamerer, Chairman, reported on behalf of himself and his committee members, Drs. Gordon B. Hughes and John R. E. Dickins. The committee reviewed the transactions of the society and found all of the transactions to be appropriate, and the consolidated balance sheet of the American Otological Society to be in order. The committee recommended that the Council and the membership accept this report as indication that the financial status of the American Otological Society, Inc., is excellent and being maintained appropriately.

REPORT OF THE NOMINATING COMMITTEE

Dr. Joseph C. Farmer, Chairman, presented the following nominations for the slate of officers for 2000–2001 year: President–Dr. A. Julianna Gulya; President-Elect–Dr. Richard A. Chole; Secretary-Treasurer–Dr. Horst R. Konrad; Editor-Librarian–Dr. Sam E. Kinney; and Council Members–Drs. Gregory J. Matz, C. Gary Jackson, Jef-

frey P. Harris, and John K. Niparko. There were no nominations from the floor. The nominated slate was elected by the membership.

The Award of Merit Committee for the year 2001 will be: Drs. Gregory J. Matz (Chairman), A. Julianna Gulya, C. Gary Jackson, Richard T. Miyamoto, and George W. Facer.

ADJOURNMENT

The Business Meeting was adjourned at 7:30 a.m. The Scientific Program continued until 12:00 noon.

Respectfully submitted,
Horst R. Konrad, M.D.
Secretary-Treasurer

Cesar Fernandez, May 20, 1910–December 15, 1999
Associate 1973

A Full Life

César Fernández was born in Lautaro, a small town near Temuco, Chile. As he was growing up, he was interested in the arts and wanted to be a sculptor, but he acceded to his father's wishes and became a medical doctor. He attended the Instituto de San Jose in Temuco for premedical studies from the age of 14 and received his medical degree from the Universidad de Chile in Santiago. He then interned in the Hospital del Salvador and spent 12 years as a physician there. In 1941, César co-founded *Revista Otorrhinolaringologis,* which has been published without interruption up to the present day (volume 60) as the journal of the Chilean Society of Otolaryngology. He wrote the first article in the journal. In 1948, he received a Kellogg Foundation Fellowship Grant for advanced training. He chose to take this training in the physiology of hearing at the Central Institute for the Deaf (CID) in St. Louis after visits to both Yale Medical School and CID. In coming to CID, he was following in the footsteps of his compatriot, J. Santiago Riesco-MacClure, a colleague from Hospital del Salvador.

While in St. Louis, César divided his time between Hallowell Davis's physiology laboratory at CID and Walter Covell's histology laboratory in the Department of Otolaryngology at Washington University. His most important studies during this time were his collaborations with Ichiji Tasaki and Hallowell Davis, and his study of the innervation of guinea pig cochlea. With this experience, he developed strong interests in both structure and function that were to remain with him throughout his life. Hallowell Davis became a lifelong inspiration to César. In the small room in which César did his microscopy, he hung two pictures: one of Santiago Ramón y Cajal and the other of Hallowell Davis.

The terms of César's fellowship required him to return to Latin America upon its completion even though he was sure that the best opportunities for the research he desired were in the United States. From 1952 to 1954, he conducted otolaryngology research at the Instituto de Cardiolojia, in Mexico City. In 1954, John Lindsay, head of otolaryngology in the Department of Surgery, invited César to join

César Fernández

the research staff at the University of Chicago, where he remained for the rest of his career. Around this time, César met his future wife, Elizabeth Schroeder. They married and had a daughter, Eva.

César collaborated with several of his colleagues, including Henry Perlman and John Lindsay. He and Robert Kimura worked on the effects of venous obstruction of the stria vascularis. He introduced several of his residents to research, including George Allen, Paul Ward, John Fredrickson, and Robert Kohut, who went on to become leaders in academic otolaryngology. In his work in the cochlea, César was joined by Bob Butler, Toruzo Konishi, and two talented postdoctoral trainees, Brian Johnstone and Vicente Honrubia. He received a series of promotions with appointments in both the

Division of Otolaryngology and Department of Physiology, where he rose to professor, eventually became professor emeritus in 1975, and remained active in research for another quarter century.

His longtime collaboration with Jay Goldberg began about 1968. César had published over 50 papers before he met Jay, and published over 50 more during the course of their collaboration. Many of the latter studies are considered landmarks in vestibular physiology. For his contributions, César was awarded the Gold Medal of the Bárány Society in 1978. He also loved teaching and excelled at it. He trained over 30 students, residents, and postdoctoral fellows who are now heads of otolaryngology departments and significant researchers around the country. César was a font of knowledge, which he gladly shared. His love of research and knowledge was an inspiration for all who came under his influence.

César's interest in the arts never waned. He had a Chicago Symphony Orchestra subscription with his daughter Eva and loved to visit the Art Institute in downtown Chicago. He was also an avid reader and especially enjoyed the poetry of Chile's Nobel Prize-winning poet, Pablo Noruda.

César will be greatly missed.

This memorial is reprinted with permission of the Department of Otolaryngology, the University of Chicago.

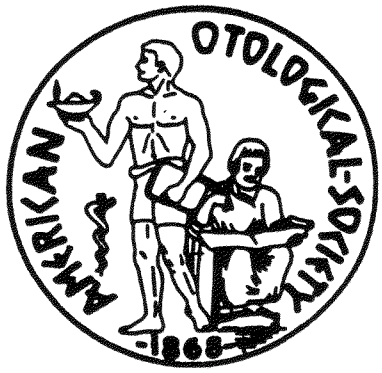
IN MEMORIAM
W. Hugh Powers, M.D.

Hugh Powers, M.D. was an otologist interested in the allergic aspects of otologic disease. He practiced most of his career as part of the Otologic Medical Group in Los Angeles, and subsequently as a member of the Los Angeles Ear Medical Group.

Dr. Powers was born in Dyersville, Iowa, and attended Boone High School and Boone Junior College in Boone, Iowa. He graduated from Creighton University in Omaha, Nebraska, and Creighton University Medical School.

Dr. Powers served his internship at Mercy Hospital in Chicago, Illinois, and his residency at the University of Illinois at Chicago. He then obtained a Fellowship in Otology/Neurotology with the Otologic Medical Group in Los Angeles in 1964–1965. He remained as a member of the Otologic Medical Group with a primary interest in allergies affecting the ear until 1975, at which time he joined the newly developed Los Angeles Ear Medical Group, where he practiced until his retirement.

The information in this obituary was obtained through the kindness of Dr. Lucy Shih of Arcadia, California, who was in practice in California with Dr. Powers at the time of his retirement.



Dr. Powers published numerous papers, most of which were related to allergy and the affects on the inner ear. He was a member of numerous national medical societies, including the Triological Society and the American Academy of Head and Neck Surgery. He was very active in the American Academy of Otolaryngologic Allergy, serving as its president from 1976 to 1977.

Dr. Powers was predeceased by his wife, Jacqueline. He had two children, Mark and Janice.

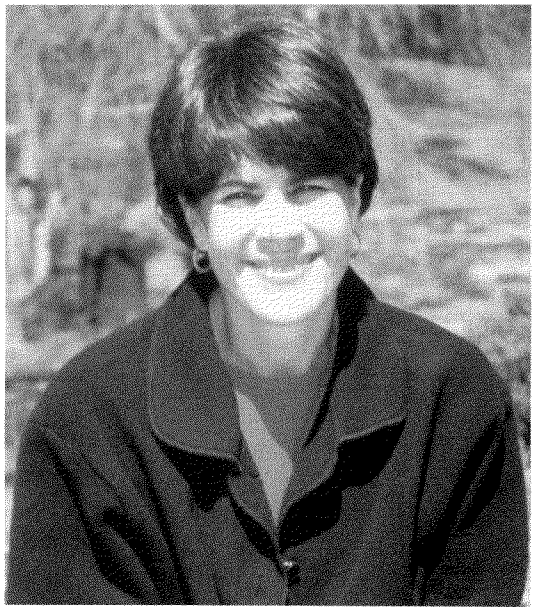
NEW MEMBERS 2000

Active Members

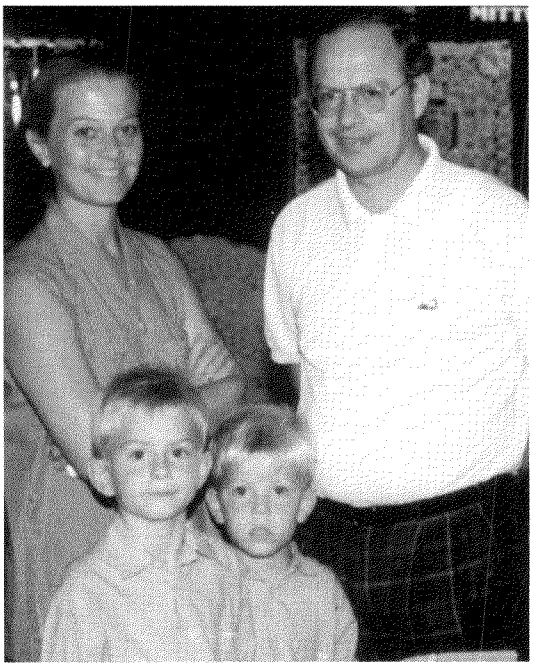
Stephen Cass

Loren Parnes

(photo not available)
Saumil N. Merchant



Debara Tucci



Vincente Diamante

(photo not available)
Thomas P.A. Wustrow

Takeshi Kubo

2000–2001 MEMBERSHIP LIST
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1987 Althaus, Sean R., 5201 Norris Canyon Rd. #230, San Ramon, CA 94583-5405
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1993 Babin, Richard W., 1830 Hwy, 51 So., Covington, TN 38019
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1992 Wilson, David F., 911 N.W. 18th Ave., Portland, OR 97209
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1996 (1975) Catlin, Francis I., 13307 Queensbury Lane, Houston, TX 77079
1994 (1973) Chandler, J. Ryan, 3170 Munroe Dr., Miami, FL 33133
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1989 (1968) Compere, Wesley E., 4519 Mayapan Dr., LeMesa, CA 91941
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