

**THE
AMERICAN ACADEMY
OF
NEUROLOGICAL SURGERY**



61st Annual Meeting



THE RITZ-CARLTON®
AMELIA ISLAND

November 10-13, 1999



Jointly Sponsored by the American
Association of Neurological Surgeons



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Richard Morawetz

GENERAL INFORMATION

REGISTRATION

Meeting Registration will be located in the foyer area of Salon III in the Ritz-Carlton.

REGISTRATION HOURS ARE:

Wednesday, November 10 3:00 PM – 9:00 PM

Thursday, November 11 7:00 AM – 5:00 PM

Friday, November 12 7:00 AM – 5:00 PM

Saturday, November 13 7:00 AM – 1:00 PM

SLIDE PREVIEW ROOM

The Slide Preview Room is located in the Boardroom and will be open during official registration hours.

MESSAGE CENTER

A telephone Message Center will be available in the Registration Area from Wednesday, November 10th through Saturday, November 13th during official registration hours. The message center has been assigned the following number: 904/277-1039

PROGRAM SUMMARY

Tuesday, November 9

12:00 Noon ABNS Primary Exam Committee—
Kings Bay

7:00 PM – 9:00 PM ABNS Dinner—*Amelia Room*

Wednesday, November 10

7:00 AM – 8:00 AM ABNS Breakfast—*Kings Bay*

8:00 AM – 4:00 PM ABNS Primary Exam Committee—
Kings Bay

12:00 PM – 1:00 PM ABNS Luncheon—*Kings Bay*

2:00 PM – 3:00 PM American Academy Executive
Committee Meeting—*The Ambassador*

3:00 PM – 5:00 PM American Academy Membership
Committee—*The Ambassador*

3:00 PM – 9:00 PM Registration—*Salon III Foyer*

4:00 PM – 9:00 PM Speaker Ready Room—*The Boardroom*

6:30 PM – 10:00 PM Welcome Reception—*Oceanfront Lawn*

7:30 PM – 10:00 PM American Academy Executive
Dinner—*The Amelia Room*

Thursday, November 11

7:00 AM – 5:00 PM Registration—*Salon III Foyer*

7:00 AM – 5:00 PM Speaker Ready Room—*The Boardroom*

7:00 AM – 9:00 AM Buffet Breakfast
(Spouses and Guests)—
The Talbot Room

7:00 AM – 8:00 AM Buffet Breakfast and Meeting
(Members)—*Salon II*

8:00 AM – 1:00 PM Scientific Session I—*Salon III*

9:54 AM – 10:15 AM Coffee Break—*Salon III Foyer*

1:00 PM – 2:30 PM	ABNS Advisory Council Luncheon— <i>The Ambassador</i>
1:00 PM	Golf— <i>The Golf Club of Amelia Island</i>
3:00 PM	Tennis— <i>Ritz-Carlton Tennis Courts</i>
3:00 PM – 5:00 PM	Journal of Neurosurgery Meeting— <i>Director's Room</i>
6:30 PM – 7:00 PM	Reception— <i>Walkers Landing</i>
7:00 PM – 8:30 PM	Dinner— <i>Walkers Landing</i>

Friday, November 12

7:00 AM – 5:00 PM	Registration— <i>Salon III Foyer</i>
7:00 AM – 5:00 PM	Speaker Ready Room— <i>The Boardroom</i>
7:00 AM – 9:00 AM	Buffet Breakfast (Spouses and Guests)— <i>The Talbot Room</i>
7:00 AM – 8:00 AM	Buffet Breakfast and Meeting (Members)— <i>Salon II</i>
8:00 AM – 1:00 PM	Scientific Session II— <i>Salon III</i>
10:02 AM – 10:22 AM	Coffee Break— <i>Salon III Foyer</i>
11:55 PM – 12:30 PM	<i>Presidential Address: J. Charles Rich</i> Introduction by: Donald O. Quest — <i>Salon III</i>
1:00 PM	Golf— <i>The Golf Club of Amelia Island</i>
3:00 PM	Tennis— <i>Ritz-Carlton Tennis Courts</i>
6:30 PM – 7:30 PM	Cocktails— <i>Ballroom Foyer</i>
7:30 PM – 10:30 PM	Black-tie Reception— <i>Salon II</i>

Saturday, November 13

7:00 AM – 5:00 PM	Registration— <i>Salon III Foyer</i>
7:00 AM – 5:00 PM	Speaker Ready Room— <i>The Board-</i> <i>room</i>
7:00 AM – 9:00 AM	Buffet Breakfast (Spouses)— <i>The Talbot Room</i>
7:00 AM – 8:00 AM	Buffet Breakfast and Meeting (Members and Guests)— <i>Salon II</i>
8:00 AM – 1:00 PM	Scientific Session III— <i>Salon III</i>
10:02 AM – 10:22 AM	Coffee Break— <i>Salon III Foyer</i>
1:00 PM	Meeting Adjourns

SCHEDULE OF ACTIVITIES FOR SPOUSES

The spouses of the American Academy members and guests are welcome to all events.

Wednesday, November 10

6:30 PM – 10:00 PM Welcome Reception—*Oceanfront Lawn*

Thursday, November 11

7:00 AM – 9:00 AM Buffet Breakfast—*The Talbot Room*

7:00 AM – 3:00 PM Hospitality Suite—*The Talbot Room*

1:00 PM Golf & Tennis

6:30 PM – 7:00 PM Reception—*Walkers Landing*

7:00 PM – 8:30 PM Dinner—*Walkers Landing*

Friday, November 12

7:00 AM – 9:00 AM Buffet Breakfast —*The Talbot Room*

7:00 AM – 3:00 PM Hospitality Suite—*The Talbot Room*

11:55 PM – 12:30 PM Presidential Address: J. Charles Rich
Introduction by: Donald O. Quest—
Salon III

1:00 PM Golf, Tennis, Shopping, Touring, etc.

6:30 PM – 7:30 PM Cocktails—*Ballroom Foyer*

7:30 PM – 10:30 PM Black-tie Reception—*Salon II*

Saturday, November 13

7:00 AM – 9:00 AM Buffet Breakfast (Spouses)—
The Talbot Room

7:00 AM – 3:00 PM Hospitality Suite—*The Talbot Room*

1:00 PM Meeting Adjourns

**SCIENTIFIC PROGRAM
AMERICAN ACADEMY
OF
NEUROLOGICAL SURGERY
1999 LEARNING OBJECTIVES**

Jointly Sponsored by The American Academy of Neurological Surgery November 10-13, 1999.

**Following the Scientific Sessions, the participants will be able to:
Critique the value of the recommended surgical and non-surgical options presented in the scientific papers.**

Evaluate the relevance of the research methodologies, the findings, and the potential usefulness in practice of the topics presented for cerebrovascular, neoplastic, spinal and developmental and functional nervous system diseases.



The American Association of Neurological Surgery is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education of physicians.

The American Association of Neurological Surgery designated this continuing medical education activity for 14.25 credit hours in Category 1 of the American Medical Association.

SCIENTIFIC PROGRAM AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

Thursday, November 11 Moderator—William Chandler, MD

- 8:00 AM-9:00 AM Point-Counter-Point - Treatment of an unruptured 8 mm Aneurysm.
Robert Spetzler, L. Nick Hopkins,
David G. Piepgras
- 9:00 AM-9:18 AM Posterior circulation aneurysms: results using combined surgical and endovascular techniques
Christopher S. Ogilvy, Christopher Putman, Ronald Budzik, Alex Norbash, In Sup Choi
- 9:18 AM-9:36 AM Surgical repair of endovascularly untreatable traditional cavernous carotid aneurysms Fredric B. Meyer, Jonathan A. Friedman, Douglas A. Nichols
- 9:36 AM-9:54 AM Retrospective analysis of carotid revascularization at the Univ. of Buffalo, Department of Neurosurgery
Lee R. Guterman, James L. Budny, L. Nick Hopkins
- 9:54 AM-10:15 AM Coffee
- 10:15 AM-10:33 AM Unilateral acoustic tumors in younger individuals with out neurofibromatosis
Jon H. Robertson, Gale Gardner, John J. Shea
- 10:33 AM-10:51 AM Dendritic cell immunotherapy for patients with glioblastoma multiforme and anaplastic astrocytoma
Keith L.Black, Christopher Wheeler, Paul Zeltzer, Divina Nacis, Paul Lee, John S. Yu

- 10:51 AM-11:09 AM Tumor control and reversal of cranial nerve deficits after focused radiation therapy for intracavernous meningiomas
John M. Tew, Abhay Sanan, Rashid M. Janjua, Harry R. Van Loveren, John C. Breneman
- 11:09 AM-11:27 AM The contemporary surgical management of Chiari I malformation in children
Mark D. Krieger, Michael L. Levy, J. Gordon McComb
- 11:27 AM-11:45 AM Emergency decompression for cervical spinal cord injury: improved outcome and reduced cost
Stephen M. Papadopoulos, Nathan R. Selden, Nayna Patel, Brenda Gillespie, Douglas J. Quint, Susan Grube
- 11:45 AM- 12:03 PM Effect of timing of decompression on neurological outcome following spinal cord injury Christopher B. Shields, John R. Dimar, George H. Raque, Y. Ping Zhang, Steven D. Glassman
- 12:03 PM-12:21 PM Controlled cortical impact injury to reat brain up-regulates peripheral-type benzodiazapine receptor expression in the thalamus Robert J. Dempsey, V. L. Raghavendra Rao, A. Dogan, K. K. Bowen
- 12:21 PM-12:41 PM *Academy Award Paper.***
Reduction of post-traumatic spinal cord injury by inhibition of the caspase cascade Robert Friedlander, M. Li, V. O. Chen, M. Kaul, L. Tenneti, Phillip Stieg, S.A. Lipton
- 12:41 PM-1:00 PM *Academy Award Paper- Runner up.***
Fas upregulation in high-grade gliomas results in increased apoptosis and survival
Bruce Frankel, Timothy Ryken, Sharon Longo, Michele Kyle

Friday, November 12 Moderator—Howard Eisenberg, MD

8:00 AM-8:50 AM Point-Counter-Point - To plate or not to plate in a single level anterior discectomy Volker Sonntag, Stephen Papadopoulos

8:50 AM-9:08 AM Surgical experience with an artificial cervical joint James T. Robertson, S. Gill, R. Nelson, N. Metcalf

9:08 AM-9:26 AM Outcome of 51 cases of unilateral locked cervical facets: interspinous braided cable for lateral mass plate fusion compared with interspinous wire and facet wiring with iliac crest J. Kevin Kaufman, Scott A. Shapiro, Paul B.Nelson

9:26 AM-9:44 AM Image guidance in spinal surgery: an update Gerald Rodts, Kevin Foley

9:44 AM- 10:02 AM Developing new technology: An accurate laser targeting system for fluoroscopically assisted procedures Michael K. Landi

10:02 AM-10:22 PM COFFEE

10:22 AM-10:40 AM Outcomes studies in carotid endarterectomy Robert E. Harbaugh

10:40 AM-10:58 AM Improved management of childhood medulloblastoma using proton beam radiotherapy Paul H. Chapman, Nancy J. Tarbell, William E. Butler, Jay S. Loeffler

10:58 AM-11:16 AM Radical resection of gliomas in functioning brain regions utilizing awake cortical and subcortical mapping and frameless stereotaxis Fredric B. Meyer, Lisa M. Bates, Stephen J. Goerss, Wanda L. Windschitl, Jonathan A. Friedman

11:16 AM-11:34 AM FasL expression in tumor vessels of patients with glioblastoma multiforme John S. Yu, Yun-hui Liu, Chunren Liu, Ken Samoto, Moneeb Ehtasham, Keith L. Black

11:34 AM- 11:52 AM Stereotactic radiosurgery: the treatment of choice for jugular foramen region tumors? Bruce E. Pollock, Deborah A. Gorman

11:55 AM- 12:30 PM ***Presidential Address:***
Athletic Performance Enhancement: The Difference between Sports Science and Sports Medicine J. Charles Rich

Saturday, November 13 Moderator: **Robert Spetzler, MD**

8:00 AM-8:50 AM Point-Counter-Point - Aggressive resection versus biopsy of a low grade glioma Mitchell L. Berger, L. Dade Lunsford

8:50 AM-9:08 AM Results of a phase I study of the treatment of malignant gliomas with the genetically-engineered herpes simplex virus G207 James A. Markert, Michael D. Medlock, Samuel Rabkin, Yancey Gillespie, Frank Feigenbaum, William D. Hunter, Tomoki Todo, Carlo Tornatore, Robert L. Martuza

9:08 AM-9:26 AM Clinical and economic consequences of early discharge after stereotactic brain biopsy Gene H. Barnett, Wayel Kaakaji, Diane Bernhard, Kren Valaitis, Sarah Stamp, Narongsak Boonswag

9:26 AM- 9:44 AM An ideal syngeneic mouse glioma for testing immunotherapy strategies Warnick RE, Weiner NE, Pyles RB, Chalk CL, Balko GO, Miller MA, Dyer CA, Parysek LM

- 9:44 AM-10:02 AM Intraoperative human sensorimotor and language mapping using optical intrinsic signal imaging: comparison with electrophysiologic techniques and fMRI in 40 patients Neil Martin, Andrew Cannestra, Nader Pouratian, Donald Becker, Susan Bookheimer, Nancy Sicotte, Arthur Toga
- 10:02 AM-10:22 AM COFFEE
- 10:22 AM-10:40 AM New neurosurgical perspectives on spontaneous intracerebral hemorrhage Issam A. Awad
- 10:40 AM-10:58 AM 3-D computer modeling of the cerebral vasculature Paul S. Larson, Chuck Sites, Ashraf Mohamed, Ayman M. Eldeib, Todd Vitaz, Aly A. Faraq, Thomnas M. Moriarty, Christopher B. Shields
- 10:58 AM-11:16 AM Instrumentated fusion in the management of post-laminectomy lumbar stenosis Charles L. Branch, David Jones
- 11:16 AM-11:34 AM Early moderate hyperventilation does not reduce cerebral metabolism following severe traumatic brain injury Robert L. Grubb, Thomas Videen, Allyson R. Zazulia, Ellen Deibert, Venkadesh Aiyagari, Ralph G. Dacey, Michael N. Diringer, William J. Powers
- 11:34 AM-11:52 AM Intracranial hypertension and cerebral perfusion pressure: their influences on neurological deterioration and outcome in severe head injury N. Juul, G.F. Morris, S. B. Marshall, The Executive Committee of the International Selfotel Trial, L. F. Marshall

Saturday, November 13

- 11:52 AM-12:10 PM Fluorescence-guided surgery of malignant gliomas utilizing 5-ALA-induced porphyrins. Experience with 66 consecutive patients. H.J. Reulen, R. Baumgartner, W. Stummer
- 12:10 PM-12:28 PM Genetically engineered cytotoxic T lymphocytes targeted against angiogenesis: A novel anti-glioma strategy Zoher Ghogawala, Robert Carter, Thomas Niederman, Richard C. Mulligan
- 12:28 PM-12:46 PM Elevation of the internal auditory canal pressure by vestibular schwannomas Behnam Badie, Mark Pyle, Peter Nguyen

THURSDAY PROGRAM

THURSDAY, NOVEMBER 11

9:00-9:18 AM

Posterior Circulation Aneurysms: Results Using Combined Surgical and Endovascular Techniques

Christopher S. Ogilvy, Christopher Putman, Ronald Budzik, Alex Norbash, In Sup Choi

Massachusetts General Hospital, Harvard Medical School, Boston, MA

Posterior circulation aneurysms can be difficult to treat depending upon the exact anatomic relation to the bony architecture, size, patient's neurologic condition, age, and presence or absence of subarachnoid hemorrhage. In an effort to improve outcome, we have utilized both surgical and endovascular (GD coil therapy) techniques over the past 9 years. As each patient was encountered, surgery was considered the primary mode of therapy. If the surgical risk was estimated to be high, an endovascular strategy was utilized.

We reviewed 199 patients with posterior circulation aneurysms treated over a 9 year interval (1990-1998) at the Massachusetts General Hospital. Of these, 139 patients were treated with surgery and 60 with endovascular techniques. Patients were graded using a previously described system (MGH grade) which incorporates age, clinical condition (Hunt & Hess grade), size of lesion, and density of SAH. Patients in poor neurologic condition were managed aggressively if brainstem function was present. Using this approach, distribution of treatment was as follows: PICA: 21 surgical, 8 GDC; AICA: 3 surgical, 4 GDC; VB junction: 3 surgical, 14 GDC; basilar trunk: 5 surgical, 2 GDC; SCA: 27 surgical, 4 GDC; PCA: 7 surgical, 6 GDC; basilar tip: 73 surgical, 22 GDC.

Overall clinical outcome was evaluated by a nurse practitioner. Followup ranged from 6 months to 8.5 years. Results were excellent/good in 154 patients (77% of total), fair in 16 patients (8%), poor in 8 patients (4%), and fatal in 21 patients (11%). Of these fatalities, 12 were in the endovascular group (7 from hemorrhage or rehemorrhage) and 9 were in the surgical group (1 from rehemorrhage). As endovascular techniques continue to evolve, a careful case-by-case analysis should be utilized to determine the mode of therapy (surgical vs. endovascular) to best improve overall outcome.

Surgical Repair of Endovascularly Untreatable Transitional Cavernous Carotid Aneurysms

Fredric B. Meyer, M.D., Jonathan A. Friedman, M.D.,
Douglas A. Nichols, M.D.

Aneurysms of the carotid artery that originate in the cavernous sinus or extracavernous, extradural segment but extend through the distal dural ring into the subarachnoid space are termed transitional carotid aneurysms. The surgical treatment of these lesions is often difficult because of their complex anatomy. In many circumstances endovascular techniques are the treatment of choice. Analyzed here are the techniques and results in 42 transitional aneurysms not suitable for interventional techniques: wide neck 5 mm, incorporation of the ophthalmic, Acho, PoCom into the aneurysm neck, failure of trial balloon occlusion. There were 39 females and 3 males with an average age of 55. Fifteen patients had bilateral cavernous aneurysms, 5 of whom underwent bilateral craniotomies. Twenty-seven aneurysms were 10-15 mm, nine were 15-25 mm, and six were >25 mm in size. The most common presentation was mass effect including visual loss. Five presented with subarachnoid hemorrhage. Thirty-nine had direct clipping, two had trapping with bypass, and one had trapping alone. The complication rate was 12% consisting of 1 death in a H+H grade 4 patient, 1 major stroke, 2 minor strokes, and 1 treated brain abscess. Based on this experience the following recommendations are made to facilitate management of transitional carotid aneurysms not treatable with contemporary endovascular techniques: 1) CT scan to identify neck calcification, 2) detailed angiogram including aneurysmogram, 3) trial balloon occlusion with CBF studies, 4) radiolucent headholder and exposure of cervical carotid artery for proximal control and planned intraoperative angiogram, 5) extradural removal of sphenoid wing and anterior clinoid, 6) availability of intraoperative doppler flow or CBF measurements.

**Retrospective Analysis of Carotid Revascularization at the University of Buffalo, Department of Neurosurgery
May 1, 1995 — July 1, 1998**

Lee R. Guterman PhD MD, James L. Budny MD, and L. N. Hopkins MD

Purpose:

To compare carotid endarterectomy (CEA) to carotid angioplasty and stent as a method for carotid revascularization in 228 consecutive patients who presented to the neurosurgery service study.

- Retrospective chart
- Angiographic film review

Primary Endpoints

- Major stroke 30 d
- Death 30 d
- MI 30 d

Secondary Endpoints

- Minor Stroke 30d
- TIA 30d

Goals

Compare medical comorbidity in each population to quantify the overall state of health for this population. Prior to this review it was assumed patients in the stent group had a higher morbidity index than the CEA group.

Abstract

There were 228 patients treated at Millard Fillmore Hospital between May 1, 1995 and July 1, 1999 who presented with cervical carotid bifurcation disease. There were 94 patients revascularized using carotid angioplasty followed by stent placement and 134 revascularized using endarterectomy. The group was predominantly male (61.4%) with an average age of 69 years. In the stent group 71.3% were male. The majority of patients were Caucasian (88% stent, 97% CEA).

CEA was performed under general anesthesia with EEG monitoring. Pharmacologic burst suppression was used in the majority of these cases.

Angioplasty and stent was performed on awake patients using intravenous sedation.

Comorbid medical conditions were documented and stratified within each group. Diabetes, pulmonary disease, asthma, CCS class, NYSHA class, angina, renal disease, neurologic disease, ejection fraction, arrhythmias, malignancy, surgical hx, smoking hx, coagulation hx, and medications were among 145 variables monitored for each procedure. Angiograms were analyzed in 88 stent patients and 103 endarterectomy patients.

Unstable angina requiring IV nitrates for control was encountered in 16% of stent patients and 0.7 % of CEA patients. CCS class was III/IV in 23.4% of stent patients and 4.4% of CEA patients. Seven patients (7.4%) in the stent group and 1 patient (0.7%) presented after CEA at another institution resulted in stroke. Restenosis was seen in 22 patients (23.4%) in the stent group and 4 patients (3.0%) in the CEA group. Ejection fraction was 40% or less in 12.8 of stent patients and 3.0% of CEA patients. NYSA class III patients 21.3% stent 5.2% CEA while class IV patients 9.6 stent and 0% CEA.

Four patients (4.3%) in the stent group had malignancies while 18 patients (13.4%) in the CEA group were afflicted. These were skin and soft tissue lesions.

Smoking history was essentially equivalent in the two groups. ICU admission within six months prior to surgery was seen in 13.8% of stent patients and 6.7% of the CEA patients.

Contralateral carotid occlusions were present in 23% of the stent population and 9.3% of the CEA population. Carotid string sign was present in 5.7% of stent patients and no CEA patients.

Procedural Complications

Transient ischemic attacks resulted in 1 patient (1.1%) in the stent group and 2 patients (1.5%) in the CEA group. There were 7 procedural related transient events in the stent group (7.4%) and one in the CEA group. There were 3 minor strokes in the stent group (3.2%) and 2 (1.5%) in the CEA group. Neither group had a major stroke. There was one MI in the stent group (1.1%) and 2 in the CEA group (1.5-%). In the stent group there were 4 pseudoaneurysms of the femoral artery and three of these required surgical exploration and repair. There was 1 wound infection in each group.

Unilateral Acoustic Tumors in Younger Individuals Without Neurofibromatosis

Jon H. Robertson, M.D., Gale Gardner, M.D., John J. Shea, III, M.D.

Objective: The purpose of this presentation will be to identify the unique characteristics of acoustic tumors presented in a population group of young individuals (less than 35 years of age) and the results of surgical management of these patients.

Methods: A retrospective chart review of 420 patients who underwent surgery for removal of an acoustic tumor by the senior author during an eighteen year period (1981-1999) was completed. 12% (N=52) of these patients were found to be younger than 35 years of age. Patients with neurofibromatosis were excluded from the study. The selected group of patients included 28 males and 24 females. Analysis of this subgroup of younger patients with acoustic tumors was directed to their clinical presentation, tumor size, histological features and surgical outcome.

Conclusion: Younger individuals with acoustic tumors tend to have larger tumors with a shorter duration of symptoms. Larger tumors of all age groups tend to be more vascular. This study will emphasize the technical difficulties encountered in this selected group of patients with large acoustic tumors and the outcome of facial nerve preservation.

Dendritic Cell Immunotherapy for Patients with Glioblastoma Multiforme and Anaplastic Astrocytoma

Keith L. Black, Christopher Wheeler, Paul Zeltzer, Divina Nacis, Paul Lee, John S. Yu

This Phase I Study was initiated to assess the safety of an immunotherapy trial using peripheral blood dendritic cells to present brain tumor-specific markers to the patient's immune system. Dendritic cells are an extremely small subset of a person's white blood cell population that are exclusively involved in presenting foreign antigens to the body's immune system. Patients in the study with either glioblastoma or anaplastic astrocytoma have undergone phlebotomy or leukapheresis, a method of removing a large number of white cells from the blood. These cells were made into dendritic cells with ex vivo treatment with interleukin 4 and GM-CSF. The patient, after having their tumor removed at surgery, had their tumors grown and tumor antigens eluted. The tumor antigens were then mixed with dendritic cells and then reinjected subcutaneously back into the patient. The goal of this protocol was to reactivate the patient's immune system to recognize and kill remaining tumor cells which have infiltrated the brain.

Fourteen patients have been treated with dendritic cell immunotherapy protocol. Eleven patients had glioblastoma multiforme, five patients had anaplastic astrocytoma. Eight patients were treated after first time diagnosis of their tumor and six patients with recurrent glioblastoma multiforme or anaplastic astrocytoma were retreated. Age ranged from 29-61 years. Two patients progressed during the time of the protocol and died during follow-up. No significant adverse events were noted. One patient had a transient fever with one episode of nausea and vomiting two days after treatment. One patient reported transient headache and the same patient developed significant axillary and inguinal lymph nodes after his second vaccination. Immunologic studies were performed on six patients so far. Four of the six patients developed significant cytotoxicity against brain tumor antigens established through the JAM cytotoxic T-cell assay. This Phase I study shows that vaccination with dendritic cells pulsed with eluted MHC-I associated antigens is safe in the treatment of patients with newly diagnosed or recurrent glioblastoma multiforme and anaplastic astrocytoma. There appears to be significant induction of specific immunity in four out of six patients tested. Further monitoring is required to determine any future adverse events including any autoimmune phenomena.

Tumor Control and Reversal of Cranial Nerve Deficits After Focused Radiation Therapy for Intracavernous Meningiomas

John M. Tew, MD, Abhay Sanan, MD, Rashid M. Janjua, MD, Harry R. Van Loveren, MD, John C. Breneman, MD

The Neuroscience Institute: University of Cincinnati Department of Neurosurgery, Radiation Oncology, and the Mayfield Clinic

Introduction: Appropriate treatment of meningiomas involving the cavernous sinus remains controversial. Direct microsurgical attack on intracavernous meningiomas carries substantial morbidity. Consideration of focused radiation therapy to treat meningiomas of the cavernous sinus raises questions regarding the radiation tolerance of cranial nerves and the ability of radiation to control tumor size. We have adopted a conservative strategy for intracavernous meningiomas, reserving treatment (whether surgery or radiation) for patients whose meningiomas grow and produce increasing symptoms.

Objective: Because of our concern that focused radiation therapy may further injure cranial nerves already compromised by infiltrating tumor, we undertook this study to assess the cranial nerve morbidity of radiation treatment and the ability of radiation to control tumor size.

Methods: All intracavernous meningiomas treated with focused radiation therapy (3-D conformal or stereotactic) at the University of Cincinnati were retrospectively reviewed. Patients who received postoperative radiation were excluded.

Results: Eighteen patients were identified with intracavernous meningiomas that received focused radiation therapy, 11 of whom (our study group) received focused radiation therapy as their initial treatment (vs. a postoperative adjunct). Six patients received stereotactic radiosurgery (mean 1550 cGy) and 5 received 3-D conformal therapy (mean 4932 cGy). Cranial nerve deficits improved in 3, remained unchanged in 7, and worsened in 1. Tumor size was controlled in 11 (100%) patients (mean follow-up 18 months).

Conclusions: Focused radiation therapy can achieve excellent control of intracavernous meningiomas. Cranial nerves of the cavernous sinus seem relatively resistant to injury with the radiation doses used; often preoperative deficits resolve. The short follow-up period limits the long-term predictive value of our conclusions.

The Contemporary Surgical Management of the Chiari I Malformation in Children

Mark D. Krieger, MD, Michael L. Levy, MD and J. Gordon McComb, MD

A wide variety of surgical adjuvants to the standard bony decompression have been advocated in the treatment of the Chiari I malformation, especially when the tonsillar herniation is associated with hydrosyringomyelia. Our practice has been to avoid such adjuvants as duroplasty, obex plugging, cerebellar tonsil resection, and various shunting procedures, and to perform a simple limited occipital craniectomy (<2.5cm in diameter), C1 laminectomy, and dural opening. To evaluate the efficacy of this more limited procedure, a retrospective review was performed of the medical records of 52 consecutive patients treated surgically over a 6-year period. This series includes long-term follow-up of a prior series (4-8 years), as well as 21 additional patients treated in a standard fashion. Included are 27 females and 25 males, ranging in age from 3 months to 18 years (median 11.5 years). Of particular interest is the large number of patients discovered during evaluation for scoliosis (38 patients-73% of the total series), which partially accounts for the large number of patients who harbored a syrinx (44-85%) in this series. All patients had at least one postoperative MRI at 6 months. Syrinx resolution or >50% diminution was seen in 89%. 48 of the patients responded well; 4 patients required subsequent operative procedures: 2 developed progressive hydrocephalus and required ventriculoperitoneal shunting, with symptom resolution. In the other 2 patients the syrinx did not diminish; both received syringopleural shunts. Importantly, no patient who responded satisfactorily at 6 month follow-up subsequently failed either radiographically or clinically. Postoperative morbidity consisted of a 21% incidence of headaches, all of which except 2 resolved within 7 days. Nausea and vomiting occurred in 13%. Four patients did have a postoperative CSF leak; all responded to bedside suturing without further sequelae. This study demonstrates the importance of addressing the Chiari I malformation, especially in the presence of scoliosis with a syrinx, and the effectiveness of a limited surgical approach.

**Emergency Decompression for Cervical Spinal Cord Injury:
Improved Outcome and Reduced Cost**

Stephen M. Papadopoulos, M.D., Nathan R. Selden, M.D., Ph.D.,
Nayna Patel, B.A., Brenda Gillespie, Ph.D., Douglas J. Quint, M.D.,
Susan Grube, R.N.

Immediate pharmacological treatment of acute spinal cord injury has improved neurological outcome in experimental animal models and in clinical trials. The effect of immediate surgical spinal cord decompression on neurological outcome after injury, however, is controversial. Experimental models strongly suggest a beneficial effect of early decompression but there is little supportive clinical evidence.

In order to address this issue, ninety-one consecutive patients with acute, traumatic cervical spinal cord injury (1990-1997) that initially received conventional emergency treatment with cervical immobilization and corticosteroids were prospectively studied. Sixty-six patients (protocol group) underwent emergency magnetic resonance imaging to determine the presence of persistent spinal cord compression followed if indicated, by immediate operative decompression and stabilization. Twenty-five patients were managed outside the protocol because of contraindication to magnetic resonance imaging, need for other emergency surgical procedure, or admitting surgeon preference (reference group). The protocol and reference groups had similar sex and age distributions, admitting Frankel grades, levels of neurological injury, and injury severity scores.

Protocol patients improved an average of 0.7 Frankel grades more than reference patients between admission and most recent follow-up ($p < .01$). Fifty percent of protocol patients, compared to only 24% of reference patients improved from their admitting Frankel grade. Eight protocol patients (12%), but no reference patients, improved from complete motor quadriplegia (Frankel grade A or B) to independent ambulation (Frankel grade D or E). Protocol patients required significantly fewer days of ventilatory support, shorter intensive care unit stays, and shorter total hospital stays than reference patients, representing an average savings per case of approximately \$50,200 in 1997 hospital charges.

We conclude that immediate magnetic resonance imaging to determine the need for emergency operative decompression and stabilization significantly improves neurological outcome and reduces cost in the management of acute traumatic cervical spinal cord injury.

Effect of Timing of Decompression on Neurological Outcome Following Spinal Cord Injury (SCI)

Christopher B. Shields, MD, John R. Dimar, MD, George H. Raque, MD, Y. Ping Zhang, MD, Steven D. Glassman, MD

Optimal timing of decompression of the spinal cord (SC) following SCI with concomitant spinal stenosis (SS) remains controversial. Some surgeons recommend emergent decompression and others believe that timing of decompression has no effect on long term outcome. To answer this question we asked two critical questions: 1) what constitutes a significant spinal stenosis following SCI, and 2) what is the optimal timing of decompression following SCI + concomitant spinal stenosis? In an earlier experiment, we performed a T10 laminectomy in Sprague-Dawley rats and inserted different sized spacers at the level of moderate SCI (25 gm-cm created using a NYU impactor). We observed that narrowing of the spinal canal by a 35% sized spacer was the greatest degree of spinal stenosis that consistently showed neurological improvement ($p < 0.05$). In this experiment, 42 Sprague-Dawley rats underwent a T10 laminectomy, a moderate SCI, and insertion of a 35% spacer which was left in the epidural space for 2, 6, 24, or 72 hours, then removed. All rats were evaluated weekly for 6 weeks using the BBB locomotor score. The neurological outcome was significantly greater the earlier that the decompression was performed. BBB scores for rats in which the decompression was performed at 2 hours was $>$ than at 6 hours, $>$ than at 24 hours, and $>$ than at 72 hours ($p < 0.05$). If extrapolated clinically, the greater the degree of spinal stenosis existing following a spinal cord injury, the poorer the neurological outcome. Furthermore, neurological outcome improve the earlier the spinal cord can be decompressed.

Controlled Cortical Impact Injury to Reat Brain Up-reglated Peripheral-type Benzodiazapine Receptor Expression in the Thalamus

Robert J. Dempsey, V.L. Raghavendra Roa, A. Dogan, K.K. Bowen

In mammalian CNS, the peripheral-type benzodiazepine receptor (PTBR) is localized on the outer mitochondrial membrane of astrocytes and microglia. PTBR transports cholesterol across mitochondrial membranes to the site of neurosteroid biosynthesis. Several neurodegenerative disorders were reported to be associated with increased PTBR density. In the present study, we evaluated the changes in the PTBR density and gene expression in the brains of rats as a function of time (6h to 14 days) after controlled cortical impact injury. Moderate grade injury was induced in adult, male, Sprague-Dawley rats under halothane anesthesia. Sham-operated rats served as control. Between 3 to 14 days after TBI, there was a significant increase in the binding of the PTBR antagonist [3H]PK11195 (by 106 to 185%, $p<0.01$; as assessed by quantitative autoradiography and in vitro filtration binding) and PTBR mRNA expression (by 2 to 3.4 fold, $p<0.01$; as assessed by RT-PCR) in the ipsilateral thalamus. At 14 days after the injury, the neuronal number decreased significantly (by 85 to 90%, $p<0.01$) in the ipsilateral thalamus. At the same time point, the ipsilateral thalamus also showed increased numbers of GFAP positive cells (reactive astrocytes; by ~3.5 fold) and the ED-1 positive cells (activated microglia/macrophages; by ~36 fold), the two cell types known to be associated with PTBR. Increased PTBR expression following brain injury is possibly an adaptive response to cellular injury and may play a role in the pathophysiology of TBI. Such studies explore the basic mechanisms of injury and repair after brain trauma and may result in future trails of gene therapy to enhance healing after traumatic brain injury. Supported by NIH, VA, AHA and the UW-Madison Medical School.

*Academy Award Paper***Reduction of Post-traumatic Spinal Cord Injury by Inhibition of the Caspase Cascade**

M. Li (1), V.O. Ona (1), M. Chen (1), M. Kaul (2), L. Tennti (2), P.E. Stieg (1), S. A Lipton, and R.M. Friedlander (1)

Evidence indicates that both necrotic and apoptotic cell death contribute to tissue injury and neurological dysfunction following spinal cord injury (SCI). Caspases have been implicated as important mediators of apoptosis following acute central nervous system insults. We investigated whether caspase-1 and caspase-3 are involved in SCI-mediated cell death, and whether caspase inhibition may reduce tissue damage and improve outcome following SCI. We demonstrate a 17-fold increase in caspase-1 activity in traumatized spinal cord samples when compared to samples from sham operated mice. Caspase-1 and caspase-3 activation was also detected by Western blot following SCI, which was significantly inhibited by the broad caspase inhibitor zVAD-fmk. By immunofluorescence or in situ fluorogenic substrate assay, caspase-1 and caspase-3 expression was detected in neuronal and non-neuronal cells following SCI zVAD-fmk treated mice, and transgenic mice expressing a caspase-1 dominant negative mutant, demonstrated a significant improvement of motor function and a reduction of lesion size compared to vehicle-treated mice. Our results demonstrate for the first time that caspase-1 is activated following SCI, and that caspase inhibition reduces post-traumatic lesion size and improves motor performance. Caspase inhibition may be a strategy for the treatment of SCI.

*Academy Award Paper-Runner up***Fas Upregulation in High-grade Gliomas Results in Increased Apoptosis and Survival**

Bruce Frankel, M.D., Sharon L. Longo, B.S., Michele Kyle, B.A., and Timothy C. Ryken, MD

Although a majority of high-grade gliomas express Fas (APO-1, CD95), a cell surface receptor that mediates apoptosis, when it reacts with Fas ligand (FasL) or Fas antibody, little is known about its effects on glioma viability in vivo. In this study, we used in situ labeling of DNA breaks to estimate the proportion of cells undergoing apoptosis in 51 high-grade human astrocytomas (18 WHO grade III and 33 grade IV tumors). A significant correlation between apoptotic index (AI), tumor grade and the degree of Fas expression was demonstrated. The mean AI significantly increased from .39% in grade III astrocytomas to .82 % grader IV tumors ($p=0.003$). In addition, high-grade astrocytomas, (grade III and IV combined) expressing high levels of Fas had a significantly greater AI than those expressing low levels of Fas (. 81 % vs. 43%)($p= 0.017$).

Despite a trend toward longer median survival for patients with Tumors exhibiting high Fas expression, statistical significance was not achieved. Patients with grade III astrocytomas demonstrated a median survival of 20 months vs. 18 months for tumors with high vs. low Fas expression, respectively ($p = .51$). Patients with grade IV astrocytomas demonstrated a median survival of 9 months vs. 7.4 months for tumors with high vs. low Fas expression, respectively ($p= .77$). It was subsequently determined that the cell surface expression of Fas in several human glioblastoma (GBM) cell lines was low, explaining the limited susceptibility of these cells to

Fas-mediated cytotoxicity. Through the use of Fas receptor upregulation by gene transfer in a rat glioma cell line (36B 10), a correlation was demonstrated between increased Fas cell surface expression, and Fas-mediated apoptosis. In fact, the percent of cells undergoing apoptosis after exposure to a FasL producing cell line increased from 4% in a sham transfected line (36B10-) to 27% in a Fas transfected line (36B10 Fas).

Finally, the effect of Fas upregulation on survival was studied in immune competent rats with intracranial malignant gliomas. Survival length was assessed after the implantation of 36BIO- and 36B10-Fas. The median survival increased significantly from 14 days (36B10-) to 24.5 days (36BIO-Fas), representing a 75% increase in survival in the higher Fas-expressing group ($p = .0005$). In conclusion, it appears that the overall low AI (range: 0-2.0%) seen in high-grade astrocytomas is related to low cell surface expression of Fas. By increasing surface Fas expression, rates of Fas-mediated apoptosis increase, as does survival.

NOTES:

FRIDAY PROGRAM

FRIDAY, NOVEMBER 12

8:00-8:50 AM

Surgical Experience with an Artificial Cervical Joint

J. T. Robertson, S. Gill, R. Nelson, N. Metcalf

University of Tennessee, Memphis

847 Monroe, Suite 427

Memphis, Tennessee 38163

INTRODUCTION

After anterior cervical fusion, a two to three per cent incidence per year of adjacent disc disease occurs. With multiple levels of fusion, additional fusion limits neck motion. Maintenance of motion by a cervical joint may prevent adjacent disc disease and enhance motion.

AIM

To determine the clinical utility of an artificial stainless steel cervical joint placed in the intervertebral space after discectomy.

METHODS

After ethical committee approval at Frenchay Hospital, Bristol, England, a 15 patient pilot study was done in patients with radiculopathy and/or myelopathy due to disc disease at the level adjacent to previously acquired congenital or surgical fusion or for patients with a single level symptomatic disc with adjacent cervical disc degeneration. The patients underwent anterior radical discectomy and removal of the appropriate osteophytes with the implantation of the cervical joint. Visual analog scales for pain, neurological examinations, neck disability and SF36 scores pre operatively and at six week, three months, six month and one year visits were done.

RESULTS

All patients have maintained joint function as demonstrated by flexion/extension cervical spine x-rays. All observational scores demonstrated significant improvement. There have been no neurological or wound complications.

SIGNIFICANCE AND CONCLUSIONS

This artificial stainless steel cervical joint placed in the intervertebral space after discectomy maintains motion segment function, has been without complication, and is effective in improving patient results. The ability to preserve motion by the joint is anticipated to reduce adjacent disc degeneration and to insure preservation of preoperative cervical motion.

**Outcome of 51 Cases of Unilateral Locked, Cervical Facets;
Interspinous Braided Cable for Lateral Mass Plate Fusion Com-
pared with Interspinous Wire and Facet Wiring with Iliac Crest.**

J. Kevin Kaufman, M.D., Scott A. Shapiro, M.D., Paul B. Nelson, M.D.

5 consecutive patients with unilateral locked facets of the cervical spine underwent treatment over an 11-year period. With the development of internal fixation devices, the authors compared the procedure of using interspinous wire and facet wiring of iliac crest to fix unilateral locked facets with that in which interspinous braided cable and lateral mass plates were used. Thirty-seven patients (73%) presented with radiculopathy\ eight (16%) -with neck pain only, and six (12%) with spinal cord injuries (Sets). Plain x-ray films demonstrated subluxation in only 44 (86%) of 51 cases. All patients underwent cervical computerized tomography (CT) scanning, and in all patients with SCI, a magnetic resonance (MR) image was obtained. Fracture in addition to facet locking was seen on 24 (47%) of 51 CT scans. Disc disruption with cord compression was seen in five cases (10%). Based on CT and/or MR imaging findings, a closed reduction procedure was believed to be contraindicated in 11 cases (22%). Of the, remaining 40 patients, 13 (33%) underwent closed reduction procedures. Two patients who underwent a closed reduction procedure were placed in a halo brace but experienced resubluxation and were surgically treated. Forty-six patients underwent posterior reduction and/ or internal fixation alone (in 24 cases spinous process fixation with facet wiring and connected to struts of iliac crest, and in 22 cases interspinous braided cable for lateral mass plating was used).

Initial surgery, regardless of technique, was successful in 45 (98%) of 46 cases. One patient experienced a resubluxation and underwent reoperation in which anterior cervical fusion and plating were performed. Four of six patients with SO underwent an emergency combined anterior-posterior compressive procedure in which internal fixation was performed. Overall there were no cases of neurological worsening or death, and there were three cases of wound infection. At 1 year postsurgery, all deficits had improved. Of 37 cases of radiculopathy, three patients (8%) experienced persistent 4/5 weakness, and the remaining patients were normal.,

including four patients in. whom diagnosis was delayed.

The six patients with SO improved significantly. 'Persistent neck pain was seen in nine cases (18%). Although the lateral mass plates and interspinous cable are stronger, easier to place, and significantly lessened the amount of resultant kyphosis ($p < 0.02$), the results of chi-square analysis demonstrated only a slight trend for improved clinical outcome compared with the use of wire and iliac crest ($P = 0, 1$)

NOTES:

Image-Guided Surgery of the Spine: An Update

Gerald Rodts, M.D., Kevin Foley, M.D.

The use of intraoperative image-guidance in spinal surgery has historically relied upon plain radiographs or live fluoroscopy. With the increasing use of internal fixation, there is a greater need for technology that assists in the accurate placement of spinal implants. The limitations of radiography and fluoroscopy are significant. Recently, stereotactic technology has been applied successfully for use in spinal surgery. There are specific clinical situations in which this new technology offers unequaled ability to navigate through difficult 3-dimensional anatomy. As a result, stronger, more biomechanically-sound constructs can be assembled.

One of the drawbacks of intraoperative frameless spinal stereotaxy is the need to obtain a pre-operative data set. Another drawback is the current inability to update that data set in the operating room after changing a patient's alignment, reducing a fracture fragment, distracting or compressing the spine, etc. Additionally, frameless stereotaxis requires intraoperative registration of the anatomy. A new technology based on conventional fluoroscopy and incorporating stereotactic strategies allows for simple acquisition of a data set with a single x-ray, automatic registration/calibration, and virtual navigation of common spinal instruments based on the acquired fluoroscopic image. Virtual fluoroscopy eliminates repeated (excessive) radiation exposure to a surgeon over time, eliminates many of the time-consuming steps involved in frameless spinal stereotaxis, but allows the surgeon to navigate virtual representations of real instruments in-hand through an acquired fluoroscopic image (e.g. lateral lumbar view). In addition to defining these two forms of image-guidance technology, we will review our clinical experience with spinal stereotaxis and virtual fluoroscopy.

Developing New Technology: An Accurate Laser Targeting System for Fluoroscopically Assisted Procedures

Michael K. Landi, MD

University at Buffalo Neurosurgery

Objective: A laser targeting system was developed for fluoroscopically assisted procedures. It positions a laser beam collinear with a line of x-ray radiation from the source to the detector. The system allows the operator to image and target a deep tissue structure, turn off the x-ray irradiation and use the laser as a guide to the target.

Methods: The device mounts on the x-ray source of a mobile c-arm fluoroscope. X-rays pass through a radiolucent calibration chamber containing radio-opaque cross hairs. A laser beam is reflected, from a radiolucent mirror, along the axis of the chamber through the centers of the cross hairs. The x-ray image is undistorted and displays the two cross hairs. A remote control drives the chamber into calibration such that the images of the two cross hairs are superimposed, providing a radiographic image of the laser axis.

Results: The FDA accepted device has been used for percutaneous vertebroplasty, vertebral body biopsy, trigeminal radiofrequency rhizotomy, bone tumor biopsy, far lateral disc localization, pedicle screw placement, facet blocks and rhizotomy, brain tumor biopsy/localization, distal locking screw placement, and lung biopsy. The device demonstrated accurate localization.

Conclusion: The laser targeting system provides a simple method for localizing a deep internal structure using fluoroscopy. It provides an accurate surface point of entry and angle of approach to deep internal structures eliminating the need for x-ray irradiation during target approach. The process of developing technology conceived by clinicians from concept to commercial product is reviewed.

Outcomes Studies in Carotid Endarterectomy

Robert E. Harbaugh M.D., F.A.C.S.

Professor of Surgery (Neurosurgery)

Dartmouth-Hitchcock Medical Center

ABSTRACT

The efficacy of carotid endarterectomy for stroke prevention in patients with high grade symptomatic and asymptomatic carotid stenosis has been extensively studied. Large, Multicenter, prospective, randomized studies have documented the benefit of this operation for reducing the risk of stroke if the surgery can be done with acceptably low perioperative morbidity and mortality. Because these studies documented a substantial benefit from surgery, the number of patients undergoing carotid endarterectomy in the United States has increased substantially during the last five years.

However, numerous studies analyzing the database maintained by the United States Health Care Financing authority (HCFA) indicate that the morbidity and mortality associated with carotid endarterectomy is considerably higher in general practice than was the case in the prospective randomized studies. This raises the question as to whether or not the results of the prospective, randomized studies can be generally applied to patients undergoing carotid endarterectomy for stroke prevention in the United States.

This presentation will discuss the limitations of both prospective randomized studies and large database outcomes studies. The use of a disease-specific searchable, computerized database for patients undergoing carotid endarterectomy will be reviewed and the efforts of the Outcomes Committee of the American Association of Neurological Surgeons and Congress of Neurological Surgeons to prospectively evaluate the outcomes of patients undergoing carotid endarterectomy using an on-line outcomes reporting system, will be discussed.

Improved Management of Childhood Medulloblastoma Using Proton Beam Radiotherapy

Paul H. Chapman, MD; Nancy J. Tarbell, MD; William E. Butler, MD; and Jay S. Loeffler, MD

Division of Neurosurgery and Department of Radiation Oncology
Massachusetts General Hospital; Boston, MA.

Radiation therapy is an integral part of the management of medulloblastoma. This typically consists of craniospinal axis irradiation for potential CSF dissemination, and post-operative x-ray boost therapy to the posterior fossa. In children, such treatment may result in failure of normal somatic growth, serious neurocognitive deficits, and disordered hypothalamic-pituitary function. Delivering enough photon radiation for adequate tumor control can be problematic in these patients. By virtue of its superior dose distribution, the proton beam offers a fundamentally new way of optimizing therapy for childhood medulloblastoma, while minimizing the risk of injury to normal tissues. Posterior fossa boost therapy excludes the temporal lobes, hypothalamus-pituitary axis, and the inner ears. The whole brain skin dose is substantially reduced; and the spinal axis can be treated without irradiating thoracic and abdominal viscera, including the ovaries in females. This approach holds great promise, not only for the treatment of medulloblastoma, but for other disseminating CNS malignancies in children as well.

Radical Resection of Gliomas in Functioning Brain Regions Utilizing Awake Cortical and Subcortical Mapping and Frameless Stereotaxis

Fredric B. Meyer, M.D., Lisa M. Bates, B.S., Stephan J. Goerss, B.S., Wanda L. Windschitl, R.N., Jonathan A. Friedman, M.D.

Analyzed here are the surgical techniques utilizing frameless stereotaxis combined with awake cortical and subcortical mapping to facilitate radical resection of gliomas located in functioning brain regions. Forty consecutive patients underwent awake stereotactic resection of gliomas located in eloquent cortical and subcortical regions. The goal of surgery was to resect the maximum neurologically permissible tumor volume as defined by the T2 margins on preoperative stereotactic imaging. Cytoreduction was determined by measuring preoperative and postoperative residual tumor volumes as defined by both T2 and T1 with gadolinium utilizing novel imaging software. All patients underwent postoperative detailed neurological examinations. In Grade 2 gliomas, a greater than 90% resection of the T2 signal was achieved in 8/10 patients. In Grade 3 gliomas, a greater than 90% reduction of T2 was achieved in 6/21. In Grade 4 patients, a 90% reduction in T1 gad was achieved in 7/8 but only 2/8 had greater than a 90% reduction of T2 because of location and large T2 volumes. Many lesions which on preoperative imaging were thought to be low grade gliomas proved to contain islands of higher grade neoplasm suggesting dedifferentiation. There were no perioperative deaths. One patient suffered an intraoperative seizure without sequelae. Fifteen patients suffered a neurological deterioration secondary to the radical surgery out of which 8 have recovered completely and 3 have made a significant partial recovery. Aggressive glioma resections may be associated with an improved prognosis. Often these neoplasms are infiltrative through eloquent brain regions. Combining computer guided stereotaxis with awake cortical and subcortical monitoring facilitates a radical resection with an acceptable postoperative neurological morbidity.

FasL Expression in Tumor Vesels of Patients with Glioblastoma Multiforme

John S. Yu, Yun-hui Liu, Chun-ren Liu, Ken Samoto, Moneeb Ehtasham, Keith L. Black

Tumors have evolved multiple mechanisms for evading the immune system. These range from passive failure to express costimulatory and major histocompatibility complex molecules to active processes such as expressing immunosuppressive cytokines such as TGF-beta. We are measuring potential inhibition of T-cell migration in glioma by assessing the expression of FasL in glioma and specifically in the endothelial cells of intracranial tumors. Preliminary data suggests colocalization of FasL on endothelial cells in GBM patients and less so in meningioma patients. By performing immunohistochemistry using FasL and Factor VIII we have compared these data with already established data of FACS of tumor infiltrating lymphocytes in gliomas and meningiomas. There is an inverse correlation in the number of CD8+ and CD4+ lymphocytes in tumor with FasL expression on tumor endothelial cells.

Coculturing of endothelial cells with primary GBM cells was shown to upregulate FasL on endothelial cells by Western and FACS analysis. TNF-alpha is known to downregulate FasL expression on endothelial cells. We have delivered adenovirus containing the TNF-alpha gene and demonstrated FasL downregulation on human endothelial cells. Our goal is to develop gene transfer strategies to counteract the T-cell inhibition of FasL expression on endothelial cells in patients with GBM. THURSDAY,

Stereotactic Radiosurgery: The Treatment of Choice for Jugular Foramen Region Tumors?

Bruce E. Pollock, M.D., Deborah A. Gorman, R.N.

Although many tumors affecting the jugular foramen region are histologically benign, complete tumor resection frequently results in lower cranial nerve dysfunction. As an alternative to surgical resection, thirty-two patients with tumors involving the jugular foramen underwent radiosurgery at our center between 1990 and 1998.

Pathology included glomus tumor (n=19), schwannoma (n=7), and meningioma (n=6). The mean patient age was 56 years (range, 28-83). Fourteen patients (44%) had undergone one or more prior tumor resection. Of the 14 patients having prior surgery, thirteen (93%) had lower cranial nerve deficits, three (21%) had facial palsy, and five were deaf (36%). Four of 18 patients (22%) having radiosurgery as primary management had lower cranial nerve deficits preoperatively. Multiple isocenter dose planning was used in all patients, and the mean marginal and maximum radiation doses were 18 Gy and 36 Gy, respectively. At a mean follow-up interval of 37 months (range, 12-100 months), 22 tumors were unchanged in size and 10 tumors were smaller. No patient has required any additional treatment of their tumor. Morbidity was limited to one patient having a decline in hearing (Gardner-Robinson Class II); no patient developed new or worsened lower cranial nerve function postoperatively.

Radiosurgery provides tumor growth control with low morbidity for the majority of patients with benign tumors of the jugular foramen. For patients with symptoms related to local cranial nerve involvement and not brainstem mass effect, radiosurgery may be preferred over surgical resection to minimize the risk of postoperative lower cranial nerve dysfunction.



SATURDAY PROGRAM

SATURDAY, NOVEMBER 13

8:50-9:08 AM

Results of a Phase I Study of the Treatment of Malignant Gliomas with the Genetically-Engineered Herpes Simplex Virus G207

James M. Markert (1), Michael D. Medlock (3), Samuel Rabkin (3), Yancey Gillespie (1,2) Frank Feigenbaum (3), William D. Hunter (3), Tomoki Todo (3), Carlo Tornatore (4) Robert L. Martuza (3)

Affiliations:

University of Alabama at Birmingham Department of Surgery, Division of Neurosurgery (1) and Department of Microbiology (2) Georgetown University Medical Center Departments of Neurosurgery (3) and Neurology (4)

Abstract

G207 is an conditionally replication-competent derivative of Herpes Simplex Virus type-1 engineered to contain deletions of both (134.5 neurovirulence loci and a disabling lacZ sequence insertion into the UL39 gene (ribonucleotide reductase, large subunit). We have previously demonstrated that G207 efficaciously treats malignant glial tumors in mice. We sought to establish the safety of G207 inoculation into high grade glial tumors in humans. Criteria for inclusion into this multicenter dose-escalation study were the presence of a histologically proven malignant glioma, Karnofsky score >70, recurrence despite surgical and radiation therapy, and an enhancing lesion greater than one centimeter in size. Patients with isolated brainstem involvement, a history of other CNS disease, or increasing steroid dependence were excluded from the trial. Pre- and post-treatment cerebral magnetic resonance imaging studies were obtained for volumetric analysis. Each patient received a stereotactic intratumoral inoculation of G207 followed by four days of close neurologic monitoring in an in-patient setting. In February, 1998, the first patient in the study underwent inoculation with 1x10⁶ plaque forming units (pfu) of G207. In May, 1999, the trial was closed to further enrollment after twenty-one patients had been inoculated with doses up to 3 x 10⁹ pfu. No serious adverse events clearly related to G207 inoculation have been observed to date. Data accrual is continuing on surviving patients. Phase II studies are planned for treatment of newly-diagnosed patients. Future trials will likely employ novel HSV-1-vectors expressing foreign genes to increase the efficacy of treatment. These vectors will also be discussed.

Clinical and Economic Consequences of Early Discharge after Stereotactic Brain Biopsy

Gene H. Barnett, Wayel Kaakaji, Diane Bernhard, Kren Valaitis, Sarah Stamp, Narongsak Boonswag

Objective: To determine the clinical and economic consequences of early discharge after stereotactic brain biopsy (SBB).

Methods: The records of patients undergoing SBB in 1994 and 1995 (Group A) were reviewed for the nature and timing of perioperative (<48 hours) clinical and radiological complications. Guidelines for early discharge after SBB (below) were applied for patients from January 1996 through July 1998 (Group B). Hospital financial records for patients undergoing SBB in 1997 and 1998 were reviewed for net revenue by discharge status.

Results: Group A: 130 biopsies were performed. There were five serious complications (3.8 per cent), of which one was sustained, and one death (0.7 per cent). All complications occurred within 6 hours of surgery. Guidelines for early discharge (<8 hours) were absence of: excessive intraoperative bleeding, new postoperative deficit and clot on a delayed postoperative CT scan.

Group B: 139 biopsies were performed. There were three serious complications (2.2 per cent) of which one was sustained. All occurred within six hours of surgery.

Financial analysis: Hospital financial records were available on 96 patients of which 22 were early discharges (<8 hours), 11 were extended outpatient observations (8 - <24 hours), and the remainder inpatient (> 24 hours). Net hospital income were \$946, \$261 and \$382, respectively across the two years, but declined to \$671, -\$1,339, and -\$889 for 1998.

Conclusions: Extended outpatient observation is not clinically necessary in the absence of excessive intraoperative bleeding, new postoperative deficit and clot on delayed postoperative CT, and may be economically prohibitive in a teaching hospital setting.

An Ideal Syngeneic Mouse Glioma Model for Testing Immunotherapy Strategies

Warnick RE, Weiner NE, Pyles RB, Chalk CL, Balks GO Miller MA, Dyer CA, Parysek LM

Animal brain tumor models serve a vital role in the development and testing of new anticancer therapies. Since the immune system is likely to play an essential role in tumor eradication, there is a particular need for modeling brain tumors in immunocompetent hosts. Few glioma models have been developed in immunocompetent mice and none of these tumors have the histological and antigenic characteristics of glioblastoma. We have used a cell line, 4C8, derived from a spontaneous glioma that arose in a transgenic mouse, to develop a new syngeneic glioma model. The intracranial injection of 4C8 cells into immunocompetent syngeneic B6D2F1 mice resulted in tumors that were densely cellular, developed a pseudopallisading pattern of necrosis, and expressed GFAP; all important features of human glioblastoma. The average neurological endpoint was 51 days after intracranial injection. The 4C8 cells also grew rapidly in the flank reaching an average volume of 100 mm³ by 34 days postinjection. The 4C* cell line was found to be highly motile and invasive in standard Matrigel and wound filling assays. Further studies of the 4C8 cell line using green fluorescent protein are underway to better characterize its invasiveness in vivo. Overall, our results suggest that the 4C8 mouse glioma has the histological, antigenic, and growth characteristics of human glioblastoma and represents an ideal system it). which to test new therapies, especially those that rely on an immune response (e.g., gene therapy, immunotherapy),

Intraoperative Human Sensorimotor and Language Mapping Using Optical Intrinsic signal Imaging: Comparison with Electrophysiologic Techniques and fMRI in 40 Patients

Neil Martin, MD, Andrew Cannestra, PhD, Nader Pouratian, BS, Donald Becker, MD, Susan Bookheimer, PhD, Nancy Sicotte, MD, Arthur Toga, PhD

Division of Neurosurgery, Laboratory of Neuroimaging, and Ahmanson-Lovelace Brain Mapping Center, UCLA

By measuring changes in light reflectance off the cortex, optical intrinsic signal (OIS) imaging detects and maps optically-active processes coupled to neuronal activity (Haglund et al, 1992; Toga et al, 1995). This study reports the method for OIS imaging of cortical activity, and describes correlation with established techniques for functional mapping.

Methods: Intraoperative finger and face sensory stimulations were administered using a 110 Hz vibrator in awake or anesthetized patients; language stimulations (naming, reading, and speaking) were performed in awake patients. During stimulation trials, baseline and activated OIS images were obtained using a slow-scan CCD camera mounted on the operating microscope. Functional maps were generated by pixel-by-pixel subtraction of control images from stimulation images. OIS images were compared with preoperatively acquired fMRI maps or with intraoperatively acquired maps obtained using SSEP recording or direct cortical electrical stimulation. After IRB approval, forty patients with cortical tumors or AVMs were studied.

Results: OIS mapping was successful even in tumor patients with mild neurological deficits or with high-flow AVMS. OIS maps provided topographical specificity for somatosensory and language functions, and the localization of peak optical responses in individuals correlated well with electrophysiologic mapping. The agreement between OIS maps and fMRI was generally good, but while OIS mapped activation to gyral surfaces, fMRI localized signal changes more over venous structures in adjacent sulci.

Conclusion: This study suggests that OIS offers a novel, valid technique for mapping somatosensory and language function over a wide area of exposed cortex, without the need for cortical contact or repetitive electrical stimulation.

New Neurosurgical Perspectives on Spontaneous Intracerebral Hemorrhage

Issam A. Awad, MD

The Neurovascular Surgery Program, Department of Neurosurgery
Yale University School of Medicine, New Haven, Connecticut

There are many facets to spontaneous intracerebral hemorrhage (SICH) with neurosurgical considerations reflecting underlying etiology, patient age, clinical condition and hematoma size and location. We discuss novel neurosurgical perspectives from three ongoing research projects.

In elderly patients with SICH we describe an ongoing protocol of minimally invasive bedside hematoma thrombolysis and aspiration procedure. The stereotactic thrombolysis and aspiration of cerebral hematoma (STACH) protocol is currently being prepared for Phase I-II multi-institutional study of safety and feasibility, including thrombolytic dose escalation. We describe preliminary safety data from our institution in 12 cases, and the design of the proposed collaborative trial.

In younger patients with SICH there is concern about underlying etiology of hemorrhage. We present data from the Yale based Hemorrhagic Stroke Project including a population based cohort of 39 cases with SICH who survived and received complete diagnostic evaluation for underlying etiology. In 18 of these 39 cases (46%) underlying etiology remained occult to complete diagnostic evaluation. We hypothesize that hematoma evacuation with microsurgical exploration is safe and might reveal underlying etiology of SICH which might cause recurrent stroke.

We present clinical experience with 16 consecutive neurologically stable patients with SICH who underwent hematoma evacuation and microsurgical exploration after initial diagnostic evaluation failed to reveal an underlying etiology. In ten of the 16 cases (62.5%) an underlying pathology not suspected by pre-operative imaging was discovered (9 occult vascular malformation, 1 occult neoplasm). There were no surgical complications and all patients recovered fully.

We conclude that novel indications for neurosurgical intervention merit further study in individual subgroups of patients with SICH. We propose further study of minimally invasive evacuation of SICH in elderly patients, and open microsurgical exploration for etiology in younger patients even when neurologically stable.

3-D Computer Modeling of the Cerebral Vasculature

Paul S. Larson, M.D., Chuck Sites, B.S., Ashraf Mohamed, M.S.,
Ayman M. Eldeib, Ph.D., Todd Vitaz, M.D., Aly A. Farag, Ph.D.,
Thomas M. Moriarty, M.D., Ph.D. and Christopher B. Shields, M.D.

Department of Neurological Surgery, School of Medicine and Computer
Vision and Image Processing (CVIP) Laboratory, Department of Engi-
neering, University of Louisville, Louisville, KY

INTRODUCTION: We describe a method to create fully interactive 3-D
computer models of cerebral arterial anatomy using preoperative MRI
data. **METHODS:** Software was developed using Silicon Graphics UNIX
workstations. Volunteers were recruited to undergo MRI for building the
computer models and magnetic resonance arteriography (MRA) for com-
parative analysis. **RESULTS:** A single T1-weighted axial MRI slice is
viewed by the surgeon, and a computer program previously developed in
our lab is used to identify arterial flow voids. A mathematical paradigm
is then employed to identify areas in the adjacent MR slices that are
connected to the flow voids in the original MR image. The process is
automatically repeated throughout the image volume, and the final 3-D
model is displayed. The entire process can be accomplished in under 30
seconds. The operator can use a mouse to navigate around the model from
any distance or perspective. 3-D models were created from MRI scans of
volunteers and compared with MRA of the same individuals. The 3-D
models were found to be anatomically accurate and had a high degree of
correlation with the subject's MRA. **CONCLUSION:** This experimental
technique is rapid, accurate and provides the surgeon with unique
information regarding the geometry of aneurysms and other vascular
malformations. It has potential application in the planning and execution
of traditional craniotomy, intraoperative MR-guided craniotomy and
radiosurgery for a variety of vascular lesions.

Instrumented Fusion in the Management of Post Laminectomy Lumbar Spinal Stenosis

Charles L. Branch, David Jones

Objective: While the indications for and the effectiveness of decompressive laminectomy have been well documented, there is uncertainty regarding the use of fusion and instrumentation in degenerative spinal disorders. This retrospective review was designed to assess the outcome of repeat decompression and instrumented fusion for post-laminectomy lumbar spinal stenosis.

Methods/Results: Over a ten-year period, 43 patients with chronic low back and/or leg pain underwent repeat decompressive laminectomy and instrumented fusion for recurrent and/or residual lumbar spinal stenosis. These patients had undergone a total of 51 previous decompressive laminectomies without fusion for lumbar stenosis. The mean interval between the preceding decompressive procedure and subsequent fusion was 2.67 years. The average age of the patients was 66.4 years, and the average number of levels fused was 2.8. Outcome in terms of pain relief and functional improvement was determined by chart review. With a mean follow-up of 1.46 years, an overall improvement was achieved in 77% of patients. Additionally, 35 patients responded to a questionnaire (mean follow-up 3.26 years) in which they graded their outcomes as much improved (51.4%), somewhat improved (37.1%), unchanged (11.4%), somewhat worse (0%), and much worse (0%). Five (11.6%) patients required an additional 7 lumbar spine operations for adjacent level stenosis at a mean interval of 1.93 years after fusion.

Conclusions: Long-term improvement was attained in approximately 80% of patients who underwent repeat lumbar decompression and instrumented fusion for post-laminectomy spinal stenosis. However, fusion may accelerate degeneration at adjacent levels necessitating additional lumbar spine operations.

Early Moderate Hyperventilation does not Reduce Cerebral Metabolism Following Severe Traumatic Brain Injury.

Robert L. Grubb, Thomas Vidcen, Allyson R. Zazulia, Ellen Deibert, Venkadesh Aiyagari, Ralph G. Dacey, Michael N. Diringner, William J. Powers

Hyperventilation (HV) has been used for many years in managing patients with traumatic brain injury (TBI). Several studies have reported reduced cerebral blood flow (CBF) early after severe TBI. This has led to concerns that HV could cause cerebral ischemia, especially if used early after TBI. We tested the hypothesis that moderate HV early after TBI would not produce a reduction in CBF severe enough to reduce cerebral oxygen metabolism.

Nine patients were studied with positron emission tomography (PET) 11.2 ((1.6 SD) (range 8-14) hours after TBI. Glasgow Coma Scale (GCS) was 5.6 ((1.8 SD), and age was 27 ((9 SD) years. Eight patients were males. Intracranial pressure (ICP), mean arterial blood pressure (MABP) and jugular venous oxygen content were monitored. Cerebral perfusion pressure (CPP) was maintained at >70 mm Hg, using vasopressors when needed. Measurements of CBF, cerebral blood volume (CBV), cerebral oxygen utilization (CMRO₂), oxygen extraction fraction (OEF) and cerebrovenous oxygen content (CvO₂) were made before and after thirty minutes of HV to a PaCO₂ of 30 ((2 SD) mm Hg. Ten healthy age-matched volunteers were used as normocapnic controls.

There were no differences in global CBF, CBV, and CvO₂ between the TBI and control groups. In the TBI patients global CMRO₂ and OEF were reduced (1.59 (0.44 SD ml/100g/min, p<0.01 and 0.31 (0.06 SD, p<0.0001 respectively). During HV global CBF fell 25.5 ((8.7 SD) ml/100g/min (p<0.0009), CBV fell to 2.8 ((0.56 SD) ml/100g (p<0.001), OEF rose to 0.45 ((0.13 SD) (p<0.02), and CvO₂ fell to 8.3 ((3.0 SD) vol% (p<0.02). CMRO₂ did not change.

Early, brief, moderate HV does not appear to impair cerebral metabolism in patients with severe TBI and thus is not likely to cause further neurologic injury. Additional studies are necessary to assess the effect of more severe HV and the effect of HV in patients with increased ICP.

Intracranial Hypertension and Cerebral Perfusion Pressure: Their Influences on Neurological Deterioration and Outcome in Severe Head Injury

N. Juul, GF Morris, SB Marshall, The Executive Committee of the International Selfotel Trial, L F Marshall

Background: Recently management of severe head injury has placed a renewed emphasis on elevating cerebral perfusion pressure (CPP) defined as the mean arterial pressure (MAP) minus intracranial pressure (ICP). Some have suggested that CPP is more important in influencing outcome than intracranial hypertension.

Methods: We examined the relative contribution to outcome of these two parameters in a trail of 427 patients prospectively studied in an international multi-center randomized double blind trail of the NMDA antagonist drug Selfotel.

Results: Excluding 18 patients lost to follow up, mortality rose from 9.6% in patients who had no objectively defined episodes of neurologic deterioration (n=292) to 56.5% in patients who suffered one or more episodes of neurologic deterioration (n=117). Correspondingly, favorable outcome,, defined as good or moderate on the Glasgow Outcome Scale (GOS) at six months, fell from 67.8% to 29.1 % in the presence of neuroworsening. In patients who had objective evidence of neurological deterioration, the relative influence on outcome of ICP and CPP was assessed. The most powerful predictor of neurological worsening was the presence of intracranial hypertension (ICP > 20) either initially or during neurologic deterioration. There was no correlation with the CPP as long as the CPP was > 60 mmHg.

Conclusion: Treatment protocols for the management of severe head injury should emphasize the immediate reduction of increased ICP to below 20 mmHg if possible. CPP above 60 mmHg appears to have little influence on the outcome of patients with severe head injury.

Fluorescence-guided Surgery of Malignant Gliomas Utilizing 5-ALA-induced porphyrins. Experience with 66 consecutive patients.

H.-J. Reulen, R Baurgartner, W, Stummer

Department of Neurosurgery, Laser Research Laboratory, Klinikum Grosshadern Ludwig-Maximilians University, Munich Germany

Background: Prognosis of patients suffering from malignant gliomas depends on the completeness of tumor resection. However, malignant glioma borders are often difficult to distinguish intra-operatively. We have demonstrated 5-aminolevulinic acid (5-ALA) to induce the accumulation of fluorescent protoporphyrin IX in malignant gliomas, a phenomenon exploitable for enhancing resection of this tumor entity. We now analyze the influence of fluorescence-guided resection on post-operative magnetic resonance imaging (MRI) and survival.

Methods: Sixty-six consecutive patients with malignant glioma operated on in our department received 20 mg 5-ALA/kg b.w. orally 3 hours prior to anesthesia. Intra-operatively, tumor fluorescence was visualized using a modified operating microscope. Visible fluorescence was removed whenever considered safely possible. Post-operative MRI was obtained within 72 hours for assessment of residual contrast enhancement. Patient survival was analyzed using the Kaplan-Meier method and multivariate analysis considering Karnofsky status, age, histology and degree of resection, as determined from early post-operative MRI. The present series was compared to 89 consecutive patients operated on between 1990 and 1992.

Results: Complete resection of enhancing tumor was accomplished in 62 % of patients. In 35 %, residual enhancement on MRI was predicted by residual intra-operative tissue fluorescence intentionally left un-resected. Karnofsky status, residual fluorescence and abscesses of contrast-enhancement on MRI were independent explanatory factors for survival. Compared to the historical series, overall survival using 5-ALA was significantly prolonged. No peri-operative mortality and only one case of permanent morbidity were encountered.

Conclusions: our observations demonstrate that resection guided by 5-ALA-induced tumor fluorescence enhances resection safely, and prolongs survival in patients suffering from malignant gliomas.

**Genetically Engineered Cytotoxic T Lymphocytes Targeted
Against Angiogenesis: A Novel Anti-Glioma Strategy**

Zoher Ghogawala, MD, Bob Carter, MD, Tom Niederman, MD, PhD,
and Richard C. Mulligan, PhD

This study joins two previously unrelated areas of research in order to redirect the cytotoxic T cell against an important marker of angiogenesis. This work raises the novel concept that a cytotoxic T cell genetically engineered to express a recombinant T cell receptor (TCR) bearing vascular endothelial growth factor (VEGF) as its extracellular ligand might recognize angiogenic endothelial cells by binding a receptor called Flk-1, which is expressed almost exclusively on dividing endothelial cells.

A VEGF-chimeric TCR was created and cloned into the CMMP retroviral vector. Mouse T cells were transduced with retroviral particles produced after transient transfection of 293 GPG (VSV G) viral producing cells. T cells expressing the VEGF-TCR were able to kill Flk-1 expressing cells without any toxic effect upon control cells in cytotoxicity studies. Adoptive transfer of genetically transduced murine T cells in mouse tumor models demonstrated a significant anti-tumor effect in three tumor models: GL 261 (murine glioma), Lewis lung carcinoma, and B16 melanoma. The effect of the VEGF-T cells in these models appeared to wane with time, but the ability to prolong survival and to inhibit tumor growth was very encouraging in all three models.

We have created the first functional chimeric TCR directed against an endothelial receptor (Flk-1), which is specifically upregulated in angiogenesis. The ability to target cells mediating angiogenesis raises the intriguing possibility that T cells directed against angiogenesis might function to inhibit tumor growth and prolong survival in angiogenic tumors including glioblastoma.

Elevation of the Internal Auditory Canal Pressure by Vestibular Schwannomas

Behnam Badie, MD, Mark Pyle, MD, Peter Nguyen, MD

Department of Neurological Surgery and Division of Otolaryngology (MP) University of Wisconsin School of Medicine, Madison, Wisconsin 53792.

The exact mechanism of hearing loss, the most common presenting symptom in patients with vestibular schwannomas, remains unclear. To test whether mechanical injury from tumor growth in the internal auditory canal (IAC) is responsible for this clinical finding, we measured the intracanalicular pressure (ICaP) in patients undergoing a retrosigmoid approach for tumor excision.

Before drilling of the IAC, the ICaP in 15 consecutive patients was measured using a Codman pressure sensor. The pressure readings were then correlated to the tumor size and patient's preoperative hearing status.

Placement of the pressure monitor into the IAC revealed a biphasic waveform in every patient with a mean ICaP of 20 mm Hg (range 1-45 mm Hg). The ICaP directly correlated with the proportion of tumor in the IAC ($r^2=0.57$, $p=0.001$) but not with the total tumor size ($r^2=0.16$, $p=0.13$). Furthermore, 8 patients with class A preoperative hearing (American Academy of Otolaryngology-Head and Neck Surgery classification) tended to have lower ICaP's as compared to 5 patients with class B hearing (16+5 Vs 28+ 4). Although this observation suggested an inverse correlation between the ICaP and hearing function, the difference between the two groups was not statistically significant ($p=0.14$).

Pressure on the cochlear nerve as a result of intracanalicular tumor growth may be responsible for hearing loss in patients with vestibular schwannomas. Modification of surgical techniques to address the elevated ICaP during tumor resection, such as early drilling of the IAC, may be beneficial in improving postoperative hearing function in these patients.

SPECIAL GUESTS

GUESTS

David Andrews, M.D.
Philadelphia, PA

Armand Awad, M.D.
New Haven, CT

Behnam Badie, M.D.
Madison, WI

Gene Barnett, M.D.
Cleveland, OH

Joshua Dowling, M.D.
St. Louis, MO

Kevin Kaufman, M.D.
Indianapolis, IN

Carl Lauryszen, M.D.
St. Louis, MO

Paul Larson, M.D.
Louisville, KY

Robert Maciunas, M.D.
Rochester, NY

James Markert, M.D.
Birmingham, AL

Stephen Papadopoulos, M.D.
Ann Arbor, MI

Bruce Pollock, M.D.
Rochester, MN

Charles C. Rich, M.D.
Salt Lake City, UT

Daniel Robertson, M.D.
Olive Branch, MS

Gerald Rodts, M.D.
Atlanta, GA

Philip Stieg, M.D.
Boston, MA

Bruce Sorensen, M.D.
Salt Lake City, UT

Ronald Warnick, M.D.
Cincinnati, OH

Christopher Wolfla, M.D.
Edmond, OK

SPONSORS

Robert Rosenwasser, M.D.

Issam Awad, M.D.

Robert Dempsey, M.D.

Joseph F. Hahn, M.D.

Ralph Dacey, Jr., M.D.

Paul Nelson, M.D.

Ralph Dacey, Jr., M.D.

Christopher Shields, MD

Warren Selman, MD

Julian Hoff, M.D.

William Chandler, M.D.

Fred Meyer, M.D.

J. Charles Rich, M.D.

James Robertson, M.D.

Suzie Tindall, M.D.

Peter M. Black, M.D., Ph.D.

J. Charles Rich, M.D.

Harry VanLoveren, M.D.

Christopher Loftus, M.D.

ACADEMY AWARD WINNERS

Paul M. Lin	1955
Hubert L. Rosomoff	1956
Byron C. Pevehouse	1957
Norman Hill	1958
Jack Stern	1959
Robert Ojemann	1960
Lowell E. Ford	1962
Charles H. Tator	1963
Earle E. Crandall	1964
Stephen Mahaley, Jr.	1965
Chun Ching Kao	1966
John P. Kapp	1967
Yoshio Hosobuchi	1968
Gary G. Ferguson	1970
Richard L. Pressley	1971
David G. McLone	1972
Arden F. Reynolds, Jr.	1973
Richard L. Rapport	1974
Andrew G. Shetter	1975
John R. Howe	1976
Howard W. Blume	1977
Howard J. Senter	1978
Elisabeth M. Post	1979
David Dubuisson	1980
Dennis A. Turner	1981
Marc R. Bayberg	1982
David S. Baskin	1983
Kevin J. Kiwak	1984
Terry Lichtor	1985
Michael G. Nosko	1986
Joseph R. Madsen	1987
James T. Rutka	1988
Christopher D. Heffner	1989
Scott I. Gingold	1990
Mary Louise Hlavin	1991
Adam P. Brown	1992
Michael Tymianski	1993
David Garrett, Jr	1994
L. Brannon Thomas	1995
John S. Yu	1996
Gregory Canute	1997
Nathan R. Selden	1998

MEETINGS OF THE ACADEMY

Hotel Netherland Plaza, Cincinnati, Ohio	October 28-19, 1938
Roosevelt Hotel, New Orleans, Louisiana	October 27-19, 1939
Tudor Arms Hotel, Cleveland, Ohio	October 21-22, 1940
Mark Hopkins Hotel, San Francisco, California	November 11-15, 1941
Ambassador Hotel, Los Angeles, California	November 11-15, 1941
The Palmer House, Chicago, Illinois	October 16-17, 1942
Hart Hotel, Battle Creek, Michigan	September 17-18, 1943
Ashford General Hospital, White Sulphur Springs, West Virginia	September 7-9, 1944
The Homestead, Hot Springs, Virginia	September 9-11, 1946
Boardmoor Hotel, Colorado Springs, Colorado	October 9-11, 1947
Windsor Hotel, Montreal, Canada	September 20-22, 1948
Benson Hotel, Portland, Oregon	October 25-27, 1949
Mayo Clinic, Rochester, Minnesota	September 28-30, 1950
Shamrock Hotel, Houston, TX	October 4-6, 1951
Waldorf-Astoria Hotel, New York City	September 29-October 1, 1952
Biltmore Hotel, Santa Barbara, California	October 12-14, 1953
Broadmoor Hotel, Colorado Springs, Colorado	October 21-23, 1954
The Homestead, Hot Springs, Virginia	October 27-29, 1955
Camelback Inn, Phoenix, Arizona	November 8-10, 1956
The Cloister, Sea Island, Georgia	November 11-13, 1957
The Royal York Hotel, Toronto, Canada	November 6-8, 1958
Del Monte Lodge, Pebble Beach, California	October 18-21, 1959
Copley Sheraton Plaza, Boston, Massachusetts	October 5-8, 1960
Royal Orleans, New Orleans, Louisiana	November 7-10, 1962
El Mirador, Palm Springs, California	October 23-26, 1963
The Key Biscayne, Miami, Florida	November 11-14, 1964
Terrace Hilton Hotel, Cincinnati, Ohio	October 14-16, 1965
Fairmont Hotel & Towers, San Francisco, California	October 17-19, 1966
The Key Biscayne, Miami, Florida	November 8-11, 1967
Broadmoor Hotel, Colorado Springs, Colorado	October 6-8, 1968
St. Regis Hotel, New York City	September 21, 1969
Camino Real, Mexico City	November 18-21, 1970
Sahara-Tahoe Hotel, Stateline, Nevada	September 26-30, 1971
New College, Oxford, England	September 4-7, 1972
Huntington-Sheraton Hotel, Pasadena, California	November 14-17, 1973
Southampton Princess Hotel, Bermuda	November 6-9, 1974

The Wigwam (Litchfield Park), Phoenix, Arizona	November 5-8, 1975
Mills Hyatt House, Charleston, South Carolina	November 10-13, 1976
Mauna Kea Beach Hotel, Kamuela, Hawaii	November 2-5, 1977
Hotel Bayerischer Hof, Munich, Germany	October 22-25, 1978
Hyatt Regency, Memphis, Tennessee	November 7-10, 1979
Waldorf Astoria, New York City	October 1-4, 1980
Sheraton Plaza, Palm Springs, California	November 1-4, 1981
Ritz-Carlton Hotel, Boston, Massachusetts	October 10-13, 1982
The Lodge at Pebble Beach, California	October 23-26, 1983
The Homestead, Hot Springs, Virginia	October 17-20, 1984
The Lincoln Hotel Post Oak, Houston, Texas	October 27-30, 1985
The Cloister, Sea Island, Georgia	November 5-8, 1986
Hyatt Regency, San Antonio, Texas	October 7-10, 1987
Omni Netherland Plaza, Cincinnati, Ohio	September 13-17, 1988
Loews Ventana Canyon, Tucson, Arizona	September 27-October 1, 1989
Amelia Island Plantation, Amelia Island, Florida	October 2-7, 1990
Salishan Lodge, Gleneden Beach, Oregon	September 22-26, 1991
Ritz-Carlton Hotel, Naples, Florida	October 21-25, 1992
The Wigwam, Phoenix, Arizona	October 27-30, 1993
The Cloister, Sea Island, Georgia	November 3-6, 1994
Loewis Ventana Canyon Resort, Tucson, Arizona	November 1-5, 1995
The Greenbrier, White Sulphur Springs, West Virginia	September 18-22, 1996
Rimrock Resort, Banff, Alberta, Canada	September 10-14, 1997
Four Seasons Biltmore, Santa Barbara, California	November 4-7, 1998
Ritz Carlton, Amelia Island, Florida	November 10-13, 1999

FUTURE MEETINGS:

The Broadmoor, Colorado Springs, Colorado	October 11-14, 2000
Colonial Williamsburg Williamsburg, Virginia	October 28-November 3, 2001

PAST PRESIDENTS

Dean H. Echols	1938-39	William B. Scoville	1971
Spence Braden	1940	Robert L. McLaurin	1972
Joseph P. Evans	1941	Lyle A. French	1973
Francis Murphey	1942	Benjamin B. Whitcomb	1974
Frank H. Mayfield	1943	John R. Green	1975
A. Earl Walker	1944	William H. Feindel	1976
Barnes Woodhall	1946	William H. Sweet	1977
William S. Keith	1947	Arthur A. Ward	1978
Howard A. Brown	1948	Robert B. King	1979
John Raaf	1949	Eben Alexander, Jr.	1980
E. Harry Botterell	1950	Joseph Ransohoff II	1981
Wallace B. Hamby	1951	Byron C. Pevehouse	1982
Henry G. Schwartz	1952	Sidney Goldring	1983
J. Lawrence Pool	1953	Russell H. Patterson, Jr.	1984
Rupert B. Raney	1954	Thomas Langfitt	1985
David L. Reeves	1955	Phanor L. Perot, Jr.	1986
Stuart N. Rowe	1956	Shelley N. Chou	1987
Arthur R. Elvidge	1957	James T. Robertson	1988
Jess D. Herrmann	1958	Thoralf Sundt, Jr.	1989
Edwin B. Boldrey	1959	Robert Ojemann	1990
George s. Baker	1960	Nicholas Zervas	1991
C. Hunter Shelden	1961-62	Henry Garretson	1992
Samuel R. Snodgrass	1963	George Tindall	1993
Theodore B. Rasmussen	1964	William A. Buchheit	1994
Edmund J. Morrissey	1965	David L. Kelly, Jr.	1995
George Maltby	1966	John M. Tew, Jr.	1996
Guy L. Odom	1967	Julian T. Hoff	1997
James G. Galbraith	1968	Edward Connolly	1998
Robert H. Pudenz	1969-70		

PAST VICE-PRESIDENTS

Francis Murphey	1941	John R. Green	1972
William S. Keith	1942	George J. Jayes	1973
John Raaf	1943	Richard L. DeSaussure	1974
Rupert B. Raney	1944	Ernest W. Mack	1975
Arthur R. Elvidge	1946	Frank E. Nulsen	1976
John Raaf	1947	Robert S. Knighton	1977
Arthur R. Elvidge	1948	Robert G. Fisher	1978
F. Keith Bradford	1949	H.T. Ballantine, Jr.	1979
David L. Reeves	1950	George Ehni	1980
Henry G. Schwartz	1951	Courtland H. Davis, Jr.	1981
J. Lawrence Pool	1952	John F. Mullan	1982
Rupert B. Raney	1953	Hugo Rizzoli	1983
David L. Reeves	1954	James W. Correll	1984
Stuart N. Rowe	1955	E. Bruce Hendrick	1985
Jess D. Herrmann	1956	Griffith R. Harsh III	1986
George S. Baker	1957	Ellis B. Keener	1987
Samuel R. Snodgrass	1958	Robert Grossman	1988
C. Hunter Shelden	1959	Jim Story	1989
Edmund Morrissey	1960	John Jane	1990
Donald F. Coburn	1961-62	Stewart Dunsker	1991
Deben Alexander, Jr.	1963	Burton Onofrio	1992
George L. Maltby	1964	Martin Weiss	1993
Robert Pudenz	1965	John M. Tew, Jr.	1994
Francis A. Echlin	1966	John Van Gilder	1995
Benjamin Whitcomb	1967	Edward Connolly	1996
Homer S. Swanson	1968	George Ojemann	1997
Augustus McCravey	1969-70	Charles H. Tator	1998
Edward W. Davis	1971		

PAST SECRETARY-TREASURERS

Francis Murphey	1938-40	Eben Alexander, Jr.	1954-57
A. Earl Walker	1941-43	Robert L. McLaurin	1958-62
Theodore C. Erickson ...	1944-47	Edward W. Davis	1963-65
Wallace B. Hamby	1948-50	Robert G. Fisher	1966-68
Theodore B. Rasmussen	1951-53	Byron C. Pevehouse	1969-72

PAST SECRETARIES

Byron C. Pevehouse	1973	Nicholas T. Zervas	1987-89
Russell H. Patterson, Jr.	1974-76	William A. Buchheit	1990-92
Phanor L. Perot, Jr.	1977-80	Julian T. Hoff	1992-95
John T. Garner	1981-83	Roberto C. Heros	1995-98
James T. Robertson	1984-86		

PAST TREASURERS

Russell H. Patterson, Jr.	1973	William A. Buchheit	1987-89
Phanor L. Perot, Jr.	1974-76	Julian T. Hoff	1990-92
John T. Garner	1977-80	Roberto C. Heros	1992-95
James T. Robertson	1981-83	David G. Piepgras	1995-98
Nicholas T. Zervas	1984-86		

HONORARY MEMBERS

- | | Elected |
|---------------------------------|---------|
| GUY LAZORTHES (Annick) | 1973 |
| 26 Rue D. Aurlol | |
| 31400 Toulouse | |
| FRANCE | |
| VALENTINE LOGUE (Anne) | 1974 |
| 16 Rowan Road | |
| London, England W6 7DU | |
| UNITED KINGDOM | |
| BERNARD PERTUISET | 1986 |
| Hospital de la Pitie | |
| 83 Boulevard de l'Hopital 75651 | |
| Paris, Cedex 13 | |
| FRANCE | |
| KEIJI SANO (Yacko) | 1975 |
| Fuji Brain Institute | |
| 270-12 Sugita | |
| Fujinomiya, 4180021 | |
| JAPAN | |

SENIOR MEMBERS

- Elected
- EBEN ALEXANDER, JR. (Betty) 1950
Wake Forest School of Medicine
Medical Center Boulevard
Winston-Salem, NC 27157-1002
- JAMES AUSMAN (Carolyn) 1979
Neurosurgery, MC799
Univ. of Illinois at Chicago
912 South Wood Street
Chicago, IL 60612-7329
- DONALD BECKER (Maria)..... 1990
Neurosurgery, Box 957039
UCLA Medical Center
10833 Le Conte Avenue
Los Angeles, CA 90095-7039
- GILLES BERTRAND (Louise)..... 1967
Montreal Neurological Institute
3801 University Street
Montreal, Quebec H3A 2B4
CANADA
- JERALD BRODKEY (Arielle) 1977
P.O. Box 18090
Cleveland, OH 44118-0090
- WILLIAM BUCHHEIT (Christa) 1980
Am Nordtor 21
Espelkamp 32339
GERMANY
- HARVEY CHENAULT (Billee) 1949
6340 Briarhill Road
Paris, KY 40361-9063
- SHELLEY CHOU (Jolene) 1974
183 Galtier Place
Shoreview, MN 55126

- W. KEMP CLARK (Fern) 1970
 3909 Euclid Avenue
 Dallas, TX 75205-3103
- WILLIAM COLLINS, JR. (Gwendolyn) 1963
 Neurosurgery, Box 208082
 Yale University
 333 Cedar Street
 New Haven, CT 06520-8082
- EDWARD CONNOLLY (Elise) 1972
 Ochsner Clinic
 1514 Jefferson Highway
 New Orleans, LA 70121-2429
- JAMES CORRELL (Cynthia) 1966
 249 Olde Point Road
 Hampstead, NC 28443
- COURTLAND DAVIS, JR. (Carrie Chamberlain) 1967
 2525 Warwick Road
 Winston-Salem, NC 27104
- RICHARD DESAUSSURE, JR. (Phyllis) 1962
 4290 Heatherwood Lane
 Memphis, TN 38117-2302
- DONALD DOHN (Carolyn) 1968
 P.O. Box 998
 Moss Point
 Pt. Clear, AL 36564-0998
- WILLIAM FEINDEL (Faith) 1959
 Montreal Neurological Institute
 3801 University Street
 Montreal, Quebec H3A 2B4
 CANADA
- ROBERT FISHER (Constance) 1955
 87 Shore Drive North
 Bristol, NH 03222

- ELDON FOLTZ (Catherine) 1960**
 UCI Medical Center
 Bldg. 3, Rm. 313, Route 81
 101 The City Drive South
 Orange, CA 92868
- RICHARD FRASER (Sara Ann) 1976**
 525 East 68th Street
 New York, NY 10021
- LYLE FRENCH (Gene) 1954**
 P.O. Box 1007
 Pauma Valley, CA 92061-1007
- JOHN GARNER (Candace) 1971**
 67 Hillsdale Drive
 Newport Beach, CA 92660-4235
- HENRY GARRETSON (Marianna Schantz) 1973**
 Neurological Surgery, Suite 1102
 University of Kentucky
 210 East Gray Street
 Louisville, KY 40202-3907
- SIDNEY GOLDRING (Lois) 1964**
 Neurosurgery, CB-8057
 Washington University
 660 South Euclid
 St. Louis, MO 63110-1094
- PHILIP GORDY (Silvia) 1968**
 3601 Carmel Drive
 Casper, WY 82604-4949
- ROBERT GROSSMAN (Ellin) 1984**
 Department of Neurosurgery
 Baylor College of Medicine
 One Baylor Place
 Houston, TX 77030
- GRIFF HARSH, III (Craig) 1980**
 P.O. Box 232
 Sweetwater, TN 37874-0232

- MAJOR GEN. GEORGE HAYES 1962
 Apartment 113
 221 Booth Street
 Gathersburg, MD 20878
- MARK PETER HEILBRUN (Robyn) 1984
 Neurosurgery, #313406
 University of Utah
 50 North Medical Drive
 Salt Lake City, UT 84132
- E. BRUCE HENDRICK (Gloria) 1968
 63 Leggett Avenue
 Toronto, Ontario M9P 1X3
 CANADA
- JULIAN T. HOFF (Diane) 1975
 Neurosurgery, TC 2128
 University of Michigan
 1500 East Medical Center Drive
 Ann Arbor, MI 48109-0338
- HAROLD HOFFMAN (Jo Ann) 1982
 The Hospital for Sick Children
 555 University Avenue
 Toronto, Ontario M5G 1X8
 CANADA
- EDGAR HOUSEPIAN (Marion Grace Lyon) 1976
 The Neurological Institute
 710 West 168th Street
 New York, NY 10032-2603
- ALAN HUDSON (Susan) 1978
 The Toronto Hospital, Bell Wing 1-658
 585 University Avenue
 Toronto, Ontario M5G 2C4
 CANADA
- JOHN JANE, SR. (Noella) 1982
 Neurosurgery, Box 212
 University of Virginia
 Health Science Center
 Charlottesville, VA 22908

- PETER JANNETTA (Diana) 1994
 Neurosurgery, Suite B-400
 Presbyterian University Hospital
 230 Lothrop Street
 Pittsburgh, PA 15213-2582
- ELLIS KEENER (Ann) 1978
 915 East Lake Drive, N.W.
 Gainesville, GA 30508-1729
- DAVID KELLY, JR. (Sarah {Sally}) 1975
 Department of Neurosurgery
 Wake Forest University
 Medical Center Boulevard
 Winston-Salem, NC 27157-1029
- WILLIAM KELLY (Joan) 1977
 Apartment B102
 16925 Inglewood Road NE
 Bothell, WA 98011
- ROBERT KING (Molly Gibbs) 1958
 Department of Neurosurgery
 State University of New York
 750 East Adams Street
 Syracuse, NY 13210-2306
- WOLFF KIRSCH (Marie-Claire) 1971
 1360 Prospect
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 London, England WC1N 3BG
 UNITED KINGDOM

DECEASED MEMBERS

	Elected	Deceased
JAMES R. ATKINSON Phoenix, Arizona (Active)	1970	1978
PERCIVAL BAILEY Evanston, Illinois (Honorary)	1960	1973
GEORGE BAKER Litchfield Park, Arizona (Senior)	1940	1993
H. THOMAS BALLANTINE, JR. Boston, Massachusetts (Senior)	1951	1996
WILLIAM F. BESWICK..... Buffalo, New York (Active)	1959	1971
EDWIN B. BOLDREY San Francisco, California (Senior)	1941	1988
E. HARRY BOTTERELL Kingston, Ontario, CANADA (Senior)	1938	1997
SPENCER BRADEN Cleveland, Ohio (Active)	Founder	1969
F. KEITH BRADFORD..... Houston, Texas (Active)	1938	1971
JEAN BRIHAYE Bruxelles, BELGIUM (Senior Corresponding)	1975	1999

- KARL-AUGUST BUSHE** 1972 1999
 Wurzburg, GERMANY
 (Senior Corresponding)
- HOWARD BROWN** 1939 1990
 San Francisco, California
 (Senior)
- JUAN CARDENAS** 1966 1996
 Mexico City, MEXICO
 (Senior Corresponding)
- GALE CLARK** 1970 1996
 Oakland, California
 (Senior)
- DONALD COBURN** 1938 1988
 Wilmington, Delaware
 (Senior)
- WINCHELL McK. CRAIG** 1942 1960
 Rochester, Minnesota
 (Honorary)
- EDWARD DAVIS** 1949 1988
 Portland, Oregon
 (Senior)
- PEARDON DONAGHY** 1970 1991
 Burlington, Vermont
 (Senior)
- CHARLES DRAKE** 1958 1998
 London, Ontario, CANADA
 (Senior)
- FRANCIS ECHLIN** 1944 1988
 New Poaltz, New York
 (Senior)
- DEAN ECHOLS** Founder 1991
 New Orleans, Louisiana
 (Senior)
- GEORGE EHNI** 1964 1986
 Houston, Texas
 (Senior)

- ARTHUR ELVIDGE 1939 1985
 Montreal, Quebec, CANADA
 (Senior)
- THEODORE ERICKSON 1940 1986
 Madison, Wisconsin
 (Senior)
- JOSEPH EVANS Founder 1985
 Kensington, Maryland
 (Senior)
- JOHN FRENCH 1951 1989
 Los Angeles, California
 (Senior)
- JAMES GALBRAITH 1947 1997
 Birmingham, Alabama
 (Senior)
- EVERETT GRANTHAM 1942 1997
 Louisville, Kentucky
 (Senior)
- JOHN GREEN 1953 1990
 Phoenix, Arizona
 (Senior)
- JAMES GREENWOOD, JR. 1952 1992
 Houston, Texas
 (Senior)
- WESLEY GUSTAFSON 1942 1975
 Jensen Beach, Florida
 (Senior)
- WALLACE HAMBY 1941 1999
 Pompano Beach, Florida
 (Senior)
- HANNIBAL HAMLIN 1949 1982
 Providence, Rhode Island
 (Senior)

- JOHN HANBERY** 1959 1996
Palo Alto, California
(Senior)
- JESS HERRMANN** 1938 1994
Oklahoma City, Oklahoma
(Senior)
- HENRY HEYL** 1951 1975
Hanover, New Hampshire
(Senior)
- WILLIAM HUNT** 1970 1999
Columbus, Ohio
(Senior)
- OLAN HYNDMAN** 1942 1966
Iowa City, Iowa
(Senior)
- KENNETH JAMIESON** 1970 1976
Brisbane, AUSTRALIA
(Corresponding)
- SIR GEOFFREY JEFFERSON** 1951 1961
Manchester, ENGLAND
(Honorary)
- RICHARD JOHNSON** 1974 1997
Cheadle Hulme, ENGLAND
(Senior Corresponding)
- WILLIAM KEITH** Founder 1987
Toronto, CANADA
(Senior)
- HUGO KRAYENBUHL** 1974 1985
Zurich, SWITZERLAND
(Honorary)
- KRISTIAN KRISTIANSEN** 1967 1993
Oslo, Norway
(Senior Corresponding)

- WALPOLE LEWIN** 1973 1980
 Cambridge, ENGLAND
 (Corresponding)
- HERBERT LOURIE** 1965 1987
 Syracuse, New York
 (Senior)
- WILLEM LUYENDIJK** 1973 1995
 Oegstgeest, NETHERLANDS
 (Senior Corresponding)
- M. STEPHEN MAHALEY** 1972 1992
 Birmingham, Alabama
 (Active)
- GEORGE MALTBY** 1942 1988
 Scarsborough, Maine
 (Senior)
- FRANK MARGUTH** 1978 1991
 Munich, GERMANY
 (Senior Corresponding)
- DONALD MATSON** 1950 1969
 Boston, Massachusetts
 (Active)
- FRANK MAYFIELD** Founder 1991
 Cincinnati, Ohio
 (Senior)
- AUGUSTUS McCRAVEY** 1944 1990
 Chattanooga, Tennessee
 (Senior)
- KENNETH McKENZIE** 1960 1964
 Toronto, CANADA
 (Honorary)
- WILLIAM MEACHAM** 1952 1999
 Nashville, Tennessee
 (Senior)

- JAMES MEREDITH** 1946 1962
 Richmond, Virginia
 (Active)
- J. DOUGLAS MILLER** 1988 1995
 Edinburgh, SCOTLAND
 (Corresponding)
- W. JASON MIXTER** 1951 1968
 Woods Hole, Massachusetts
 (Honorary)
- EDMUND MORRISSEY** 1941 1986
 San Francisco, California
 (Senior)
- FRANCIS MURPHEY** Founder 1994
 Naples, Florida
 (Senior)
- GOSTA NORLEN** 1973 1985
 Goteborg, SWEDEN
 (Honorary)
- FRANK NULSEN** 1956 1994
 Naples, Florida
 (Senior)
- SIXTO OBRADOR** 1973 1978
 Madrid, SPAIN
 (Honorary)
- PIETRO PAOLETTI** 1989 1991
 Milan, ITALY
 (Corresponding)
- HANS-WERNER PIA** 1978 1986
 Giessen, WEST GERMANY
 (Corresponding)
- WILDER PENFIELD** 1960 1976
 Montreal, CANADA
 (Honorary)

- HELMUT PENZHOLZ 1978 1985
 Heidelberg, WEST GERMANY
 (Corresponding)
- ROBERT PUDENZ 1943 1998
 South Pasadena, California
 (Senior)
- BRONSON RAY 1992 1993
 New York, New York
 (Honorary)
- DAVID REEVES 1939 1970
 Santa Barbara, California
 (Active)
- DAVID REYNOLDS 1964 1978
 Tampa, Florida
 (Active)
- R. C. L. ROBERTSON 1946 1985
 Houston, Texas
 (Senior)
- STEWART ROWE 1938 1984
 Pittsburgh, Pennsylvania
 (Senior)
- RICHARD SCHNEIDER 1970 1986
 Ann Arbor, Michigan
 (Senior)
- HENRY SCHWARTZ 1942 1998
 St. Louis, Missouri
 (Senior)
- WILLIAM SCOVILLE 1944 1984
 Hartford, Connecticut
 (Senior)
- R. EUSTACE SEMMES 1955 1982
 Memphis, Tennessee
 (Honorary)

- SAMUEL SNODGRASS 1939 1975
Galveston, Texas
(Senior)
- GLEN SPURLING 1942 1968
LaJolla, California
(Honorary)
- C. WILLIAM STEWART 1948 1948
Montreal, CANADA
(Corresponding)
- THORALF SUNDT, JR. 1971 1992
Rochester, Minnesota
(Active)
- KENICHIRO SUGITA 1988 1994
Nagoya, Japan
(Senior Corresponding)
- HENDRIK SVIEN 1957 1972
Rochester, Minnesota
(Active)
- HOMER SWANSON 1949 1987
Atlanta, Georgia
(Senior)
- ALFRED UIHLEIN 1950 1990
Rochester, Minnesota
(Senior)
- A. EARL WALKER 1938 1995
Albuquerque, New Mexico
(Senior)
- ARTHUR WARD, JR. 1953 1997
Seattle, Washington
(Senior)
- THOMAS WEAVER, JR. 1943 1985
Dayton, Ohio
(Senior)

W. KEASLEY WELCH 1957 1996
Waban, Massachusetts
(Senior)

BENJAMIN WHITCOMB 1947 1998
Surrey, Maine
(Senior)

BARNES WOODHALL 1941 1985
Durham, North Carolina
(Senior)

FRANK WRENN 1973 1990
Greenville, South Carolina
(Senior)

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