THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

62nd Annual Meeting

A TRADITION OF EXCELLENCE SINCE 1918

THE BROADMOOR
COLORADO SPRINGS

October 11-14, 2000

American Association of Neurological Surgeons

Jointly Sponsored by the American Association of Neurological Surgeons

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Mitchel Berger

Round Robin Editor:

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Local Arrangements:

Glenn Kindt

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GENERAL INFORMATION

REGISTRATION

Meeting Registration will be located in the Main Ballroom Foyer.

REGISTRATION DESK HOURS:

Wednesday, October 11	2:00 PM – 8:00 PM
Thursday, October 12	6:30 AM – 3:00 PM
Friday, October 13	6:30 AM – 2:00 PM
Saturday, October 14	7:00 AM – 1:00 PM

SLIDE PREVIEW ROOM

The Slide Preview Room is located in the Pourtales Room and will be open:

Wednesday, October 11	2:00 PM – 10:00 PM
Thursday, October 12	6:30 AM – 10:00 PM
Friday, October 13	6:30 AM – 10:00 PM
Saturday, October 14	6:30 AM – 1:00 PM

MESSAGE CENTER:

A telephone will be available at the meeting Registration Desk Wednesday, October 11 through Saturday, October 14 to receive messages during official registration hours. The message center has been assigned the following number: (719) 471-6440.

PROGRAM SUMMARY

Tuesday, October 10

2:00 – 5:00 PM	ABNS Primary Exam Committee *Crystal Room*

Wednesday, October 11

7:00 – 8:00 AM	ABNS Breakfast
8:00 – 4:00 PM	ABNS Primary Exam Committee *Gaylord Board Room*
2:00 – 5:00 PM	American Academy Executive Committee Meeting – *Congress A*
2:00 - 8:00 PM	Registration – *Main Ballroom Foyer*
7:00 - 7:00 PM	Speaker Ready Room – *Pourtales*
6:30 – 10:00 PM	Welcome Reception - *Donald Ross*

Thursday, October 12

6:30 AM – 3:00 PM	Registration- *Main Ballroom Foyer*
7:00 AM – 7:00 PM	Speaker Ready Room - *Pourtales*
7:00 – 8:00 AM	Physician's Breakfast - *Penrose*
7:00 – 9:00 AM	Spouse & Guest Breakfast - *Crystal*
8:00 AM – 1:00 PM	Scientific Session - *Main Ballroom*
12:20 – 1:00 PM	***Presidential Address: "The Role of Neurosurgeons in Research"*** George Ojemann, M.D.
1:00 PM	ABNS Advisory Council Lunch *Crystal Room*
1:00 PM	NORAD Tour, golf, tennis
6:30 PM	Cocktails & Dinner –*Mountain View Terrace*

Friday, October 13	
6:30 AM – 3:00 PM	Registration- *Main Ballroom Foyer*
7:00 AM – 7:00 PM	Speaker Ready Room – *Pourtales*
7:00 – 8:00 AM	Physician's Breakfast - *Penrose*
7:00 – 9:00 AM	Spouse & Guest Breakfast - *Crystal Room*
8:00 AM – 1:00 PM	Scientific Session – *Main Ballroom*
12:30 PM	Pike's Peak Cog Railway – *Hotel Main Entrance*
2:00 PM	Golf tournament, tennis tournament
7:00 – 12:00 PM	Cocktails, Banquet – Black Tie *Main Ballroom*
	A special thank you to the following companies for sponsoring the annual Academy Banquet: Boston Scientific/Target; Cordis Neurovascular; and Medtronic Sofamor Danek and Medtronic Neurologic Technologies
Saturday, October 14	
6:30 AM – 1:00 PM	Registration- *Main Ballroom Foyer*
7:00 AM – 7:00 PM	Speaker Ready Room - *Pourtales*
7:00 – 8:00 AM	Physician's Breakfast - *Penrose*
7:00 – 8:00 AM	Spouse & Guest Breakfast – *Penrose*
8:00 AM – 1:00 PM	Scientific Session - *Main Ballroom*

SCHEDULE OF ACTIVITIES FOR SPOUSES

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

Wednesday, October 11	
6:30 - 10:00 PM	Welcome Reception - *Donald Ross*
Thursday, October 12	
7:00 - 9:00 AM	Spouse & Guest Breakfast - *Crystal Room*
7:00 – 3:00 PM	Hospitality Suite - *Crystal Room*
9:30 AM	Book Discussion - "The Poisonwood Bible"
12:20 - 1:00 PM	***Presidential Address: "The Role of Neurosurgeons in Research"*** George Ojemann, M.D.
1:00 PM	NORAD Tour, golf, tennis, shopping
6:30 PM	Western Dinner - *Mountain View Terrace*
Friday, October 13	
7:00 - 9:00 AM	Spouse & Guest Breakfast – *Crystal Room*
7:00 AM - 3:00 PM	Hospitality Suite – *Crystal Room*
9:30 AM	Discussion groups - "
12:30 PM	Pike's Peak Cog Railway – *Hotel Main Entrance*
1:00 PM	Golf tournament, tennis tournament
7:00 PM	Cocktails & Banquet – Black Tie *Lake Terrace Dining Room*

Saturday, October 14

7:00 - 9:00 AM	Spouse & Guest Breakfast - *Crystal Room*
7:00 AM - 1:00 PM	Hospitality Suite – *Crystal Room*
1:00 PM	Meeting adjourns

SCIENTIFIC PROGRAM
AMERICAN ACADEMY
OF NEUROLOGICAL SURGERY
2000 LEARNING OBJECTIVES

Jointly sponsored by The American Association of Neurological Surgeons October 11-14, 2000.

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

American
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Surgeons

This activity has been planned and implemented in accordance with the Essential Areas and policies of the Accreditation Council for Continuing Medical Education through the Joint Sponsorship of American Association of Neurological Surgeons and the American Academy of Neurological Surgeons. The American Association of Neurological Surgeons is accredited by the ACCME to provide continuing medical education for physicians.

The American Association of Neurological Surgeons designates this continuing medical education activity for a maximum of 14 hours in category 1 credit towards the AMA Physician's Recognition Award. Each physician should claim only those hours of credit he/she actually spent in the educational activity

SCIENTIFIC PROGRAM
AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

Thursday, October 12 **Moderator: Howard M. Eisenberg**

8:00 - 8:45	POINT COUNTERPOINT: Treatment of Vestibular Neuroma. Microsurgery or Stereotactic Radiosurgery? Robert Ojemann and Bruce Pollock
8:45 -9:05	Glioma Resection using Intraoperative MRI after Optimal Image-Guided Frameless Stereotactic Resection. Ronald E. Warnick, RJ Bohinski, AK Kokkino, MF Gaskill-Shipley, RR Lukin and JM Tew
9:05-9:25	Cranial Surgery with a Mobile, Low Field Strength MRI. Michael Schulder and Peter W. Carmel
9:25-9:45	Rhamm Mediates Astrocytoma Motility and Invasiveness. James Rutka, Shin Jung and Eva Turley
9:45-10:05	Immunocytochemical Mapping of the PTEN/NMACI Tumor Suppressor Protein in Human Gliomas. Dan Fults and Carolyn Pedone
10:05-10:25	Applications of Magnetic Resonance Spectroscopic Imaging to the Management of Patients with Glioma. Mitchel S. Berger and Sarah Nelson
10:25-10:45	Coffee

10:45-11:05 Transsphenoidal Surgery for Acromegaly - Ultrastructural and Histological Findings and Surgical Complications in a Contemporary Series. Edward R. Laws, J. Kreutzer, MB Lopes and ML Vance

11:05-11:20 A Historic Look at the Academy. William Buchheit

11:20-11:40 ***ACADEMY AWARD PAPER***
Introduction by Robert Ratcheson
Changes in Intrinsic Signals and Phosphorylated Growth-Associated Protein-43 of the Adult Rat Barrel Cortex Following Kainin Acid Induced Central Lesion. Tien T. Nguyen, Takamichi Yamamoto, Karina F. Meiri and Charles J. Hodge

11:40-12:00 ***ACADEMY AWARD HONORABLE MENTION***
In Vivo Optical Mapping of Epileptic Foci and Surround Inhibition in Ferret Cerebral Cortex. Theodore H. Schwartz and Tobias Bonhoeffer

12:00-12:20 ***ACADEMY AWARD HONORABLE MENTION***
Understanding Oligodendrocyte Loss Following Early Ischemic Insult. Shenandoah Robinson, Kasia Petelenz and Robert H. Miller

12:20-1:00 **PRESIDENTIAL ADDRESS**
The Role of Neurosurgeons in Research
George A. Ojemann
Introduction by Howard M. Eisenberg

Friday, October 13 **Moderator: Robert Spetzler**

8:00-8:50 POINT COUNTERPOINT: Management of Chronic Back Pain with Interbody Fusion. Edward Connolly and Charles Branch

8:50-9:10 Prospective Analysis of Preoperative Helical CT Angiography for C1-2 Transarticular Screw Placement: A New Technique. Volker K.H. Sonntag, N Theodore, S Partovi

Friday, October 13, cont.

9:10-9:30	Dynamic Weight Bearing Cervical MRI. Christopher B. Shields, TW Vitaz, GH Raque, S Hushek, T Moriarty
9:30-9:50	Thoracic Pedicle Screws are Superior to the Existing Stabilization Techniques. Robert F. Heary, RP Schlenk, C Vaicys, TJ Sernas, M Black
9:50-10:10	Preoperative Detection of Trigeminal Neurovascular Compression by MRI Imaging In Patients with Typical and Atypical Trigeminal Neuralgia. Kim Burchiel
10:10-10:30	Analysis of Posterior Fossa Volumes In Patients with Chronic Fatigue Syndrome. Peter W. Carmel, R Raab, G Lange, BH Natelson
10:30-10:50	Coffee
10:50-11:10	Clinical and PET Imaging Results after Neurotransplantation for Basal Ganglia Stroke. Douglas Kondziolka, L Wechsler, C Meltzer, V Villemagne, S Goldstein, J Gebel, S DeCesare, K Thulborn, P Janetta, E Eiler
11:10-11:30	Deep Brain Stimulation for Parkinson's Disease: Which Target? Philip Starr, Chadwick Christine, Marsha Melnick, Heidi Clay, Susan Heath and William Marks, Jr.
11:30-11:50	Localization of Language Function in Children: Results of Electrical Stimulation Mapping. Steven G. Ojemann, MS Berger, E Lettich and GA Ojemann
11:50-12:10	Possible Functional Consequences of Neurogenesis in Temporal Lobe Epilepsy. Guy M. McKhann II and Helen Scharfman

12:10-12:30 Experience with a Statewide Carotid Endartectomy Registry in New York. A. John Popp and Edward L. Hannan

12:30-12:50 Operative Management of Post-AVM Resection Hemorrhage in the Absence of Residual AVM. Duke Samson, Thomas Kopitnik and Hunt Batjer

Saturday October 14 **Moderator: Mitchel Berger**

8:00-8:50 POINT COUNTERPOINT: The Management of Very Large AVMs. Robert Spetzler and Hunt Batjer

8:50-9:10 Identifying Patients at Risk for Post-Procedural Morbidity Following Treatment of Incidental Intracranial Aneurysms: The Role of Aneurysm Size and Location. V Janardhan, R Friedlander, S Dagen and Philip E. Stieg

9:10-9:30 Regulation of Vascular Endothelial Growth Factor (VEGF) by Hemodynamic Forces in Brain Microvascular Endothelium. Adel M. Malek, I Lee, S Izumo and SL Alper

9:30-9:50 Ornithine Decarboxylase and Normal Adaptive Responses to Transient Cerebral Ischemia. Robert J. Dempsey, VLR Rao, A Dogan, KK Bowen, AM Rao, JF Hatcher

9:50-10:10 Multifractorial Analysis of Surgical Outcome In-Patients with Unruptured Middle Cerebral Artery Aneurysms. Eugene S. Flamm, AA Grigorian, A Marcovici

10:10-10:30 Remote Cerebellar Hemorrhage Complicating Pterional Craniotomy - Further Perspectives from the International Study on Unruptured Intracranial Aneurysms. David G. Piepgras and James Torner

Saturday, October 14, cont.

10:30-10:50	Coffee
10:50-11:10	Approaches to Anterior Inferior Cerebellar Artery: Experience with 38 Cases. Robert F. Spetzler, MJ Alexander and LF Gonzalez
11:10-11:30	The Toxicity of Intracerebral Hemorrhage. Julian T. Hoff
11:30-11:50	Inhibition of HMG-CoA Reductase by Simvastatin Reduces Infarct After Embolic Stroke in Mice. Sepideh Amin-Hanjani, M Asahi, N Stagliano, S Thomas, EH Lo, JK Liao, MA Moskowitz
11:50-12:10	Functional Gap Junction Communication between Malignant Glioma Cells and Vascular Endothelial Cells. William T. Couldwell, W Zhang and M Nedergaard
12:10-12:30	A Nitric Oxide Donor Reverses the Attenuation of Cerebrovascular Reactivity to Hypercapnia by Traumatic Brain Injury in a Rat Model of Controlled Cortical Impact. Z Zhang, S Sprague, MN Henry, MG Son, Dennis G. Vollmer
12:30-12:50	Vasoreactivity After Head Injury: A Transcranial Doppler Study. Daniel F. Kelly, JH Lee, M. Oertel, DL Arthur, TC Glenn, P Vespa, WJ Boscardin, NA Martin

NOTES:

THURSDAY, OCTOBER 12 8:45 -9:05

Glioma Resection Using Intraoperative MRI after Optimal Image-Guided Frameless Stereotactic Resection

Ronald E. Warnick, MD, Robert J. Bohinski, MD, PhD, Andrew K. Kokkino, MD, Mary F. Gaskill-Shipley, MD, Robert R Lukin, MD, John M. Tew, MD

This study sought to determine the contribution of intraoperative MRI to tumor resection after first performing frameless stereotactic tumor resection. Following a routine frameless stereotactic resection, the patient was moved to an adjoining low-field strength open MRI unit for imaging. If indicated, additional tumor resection was performed either just outside the MRI gantry or in the adjoining conventional neurosurgical operating room. Thirty-nine patients with gliomas were enrolled in this study. Gross total resection was confirmed in 14 patients (36%) by the initial intraoperative MRI scan and the craniotomy was then closed. In the remaining 25 patients residual tumor was detected. In 5 of these patients no further resection was performed because of proximity to eloquent cortex. The remaining 20 patients underwent additional tumor resection. Gross total resection was subsequently achieved in 16 of these 20 patients. Four patients underwent significant additional tumor debulking, but were subtotally resected because eloquent cortex was eventually encountered. No patient experienced an immediate complication that could be attributed to the procedure. One patient who had received prior radiation and chemotherapy developed a subgaleal empyema. Postoperative MRI using a conventional 1.5-T magnet was performed in all patients and showed no discrepancies with the intraoperative images. In this series, the use of intraoperative MRI after image-guided frameless stereotactic resection of gliomas resulted in an improved rate of gross total resection from 36% to 74%. The impact of this on patient outcome is under investigation.

Cranial surgery with a mobile, low field strength MRI

Michael Schulder, Peter W. Carmel
Department of Neurosurgery, New Jersey Medical School, Newark, NJ, USA

Introduction. We describe the use of a mobile, compact, intraoperative MRI unit.

Methods. The system contains a permanent magnet with a field strength of 0.12 Tesla (Odin Medical Technologies, Yokneam, Israel). The 5-Gauss line is at 1.5 meters from the center of the magnetic field, which allows use of the device in a regular operating room environment. The magnet docks to a standard operating table, and has two poles 24 cm apart. Surgery can be done with the magnetic poles raised; with the poles lowered no special instrumentation is required. The instrument contains a surgical navigation capability with optical or magnetic probes. Accuracy of these tools was assessed using a composite water-covered phantom.

Results. 18 patients have undergone MRI-guided surgery. Operations included a variety of supratentorial, infratentorial, and transsphenoidal procedures. Images were acquired before, during, and at the conclusion of surgery. The number of scans ranged from 2 to 8 per surgery (average 3.6); image quality was excellent in 42%, adequate in 43%, and poor in 15%. In 3 patients imaging revealed additional tumor that was resected; in 5 others visual examination of the operative field was inconclusive but complete removal was confirmed by imaging. In 11 patients early postoperative MRI scans were obtained, and in all patients intraoperative MRI findings were corroborated. In one patient residual craniopharyngioma was not resected due to misinterpretation of the image.

On the phantom, 120 data points were measured. The accuracy of the magnetic probe averaged 1.3 mm and 2.1 mm in coronal and axial planes, respectively; the optical probe accuracy was 2.1 mm and 1.8 mm in those planes. Navigational accuracy tended to be higher in the center of the field.

Conclusions. High quality intraoperative MR imaging and accurate surgical navigation were achieved with this device, with minimal changes to a standard neurosurgical operating room.

THURSDAY, OCTOBER 12 9:25-9:45 AM

Rhamm Mediates Astrocytoma Motility and Invasiveness

James T. Rutka, MD, PhD, FRCSC, Shin Jung, MD, Eva Turley, PhD
Division of Neurosurgery, The Hospital for Sick Children, The University of Toronto, And The Arthur and Sonia Labatt Brain Tumour Research Centre

The extracellular matrix (ECM) of the central nervous system (CNS) is enriched in hyaluronic acid (HA). A ubiquitous ligand for HA is the *R*eceptor for *HA-M*ediated *M*otility known as RHAMM. In the present study, we have investigated the potential role of RHAMM in the motility of human astrocytoma cells. RHAMM expression in astrocytomas was determined by immunochemistry, western blot, northern blot and RT-PCR analyses. Astrocytoma cells were pulsed with HA (10 ng/ml) to examine the effects of HA-stimulation on cell motility by time lapse videomicroscopy. Two human astrocytoma cell lines of differing basal cell motilities, U87 and U343, were transfected with a RHAMM*v4* cDNA and a green fluorescent protein (GFP) tag in both sense and antisense orientations to determine whether modulation of RHAMM expression would alter astrocytoma motility and invasion in a novel human brain slice model. We found that HA was a strong stimulus for astrocytoma motility. Astrocytoma cell lines and tumor specimens expressed RHAMM by immunostaining, western, northern blot and RT-PCR analyses. RHAMM expression was greater in the anaplastic astrocytomas than in low great astrocytomas or human brain specimens. The basal motility of astrocytoma cells was significantly inhibited by anti-RHAMM antibodies and by antisense RHAMM transfection. Stimulation of basal cell motility was observed following treatment with purified HA and following RHAMM transfection. Finally, GFP-tagged invasive astrocytoma cells could be readily tracked in the human brain slice model by laser confocal microscopy. These experiments suggest that RHAMM is an important mediator of astrocytoma motility. RHAMM should also be taken into consideration as an important molecule regulating astrocytoma invasiveness along with matrix metalloproteinases and serine proteinases.

Immunocytochemical Mapping of the PTEN/MMAC1 Tumor Suppressor Protein in Human Gliomas

Dan Fults and Carolyn Pedone, Department of Neurosurgery, University of Utah School of Medicine, Salt Lake City, Utah

PTEN/MMAC1 is a tumor suppressor gene whose inactivation is an important step in the progression of gliomas to end-stage glioblastoma multiforme (GBM). We examined the distribution of PTEN protein in 49 primary human gliomas by immunocytochemistry using polyclonal antibodies that we raised against PTEN-glutathione S-transferase fusion proteins expressed in *E. Coli*. The study group consisted of 6 low-grade astrocytomas, 7 anaplastic astrocytomas, 21 GBMs, 4 low-grade oligodendrogliomas, 6 malignant oligodendrogliomas, and 5 malignant mixed oligoastrocytomas. For each tumor we determined the percentage of tumor cells showing PTEN immunoreactivity in the most cellular regions of the tumor specimen. In both astrocytomas and oligodendrogliomas there was an inverse relationship between the percentage of $PTEN^+$ cells and malignancy grade, consistent with a role for *PTEN* as a tumor suppressor gene whose expression declines during glioma progression. In non-neoplastic tissue, PTEN was expressed in human fetal brain at 16, 23, and 27 weeks gestation, but not in adult brain, indicating that PTEN is developmentally regulated in the central nervous system. In 21 GBMs we correlated PTEN protein expression with *PTEN* gene sequence. Although *PTEN*-mutant tumors showed significantly diminished PTEN protein expression compared to wild-type cases, suppressed expression of PTEN is more prevalent than predicted from mutation frequencies.

THURSDAY, OCTOBER 12 10:05-10:25 AM

Applications of Magnetic Resonance Spectroscopic Imaging to the Management of Patients with Gliomas

Mitchel S. Berger, M.D. and Sarah Nelson, M.D.
Departments of Neurological Surgery and Neuroradiology, University of California, San Francisco, CA

The definition of tumor grade and spatial extent are critical factors for management of patients with gliomas. Although Gadolinium enhanced and T2-weighted MR images are widely used for evaluation of such lesions, they are not specific for tumor and may provide ambiguous or misleading information. At UCSF, we have addressed this problem using MR Spectroscopic Imaging (MRSI) and have performed over 1000 examinations on patients with gliomas. These studies have provided compelling evidence for differences in the metabolic profiles of normal brain tissue, tumor and necrosis. Comparison of in vivo MRSI data with histological analysis of image guided biopsies indicated that regions containing tumor were more likely to have elevated levels of choline containing compounds and reduced levels of the neuronal marker N-acetylaspartate. Levels of creatine were variable, both within and between tumors of different grades and the presence of lactate or lipid peaks was correlated with higher grade. There was also considerable spatial hetereogeneity in the metabolite levels in individual lesions, the regions with highest choline frequently being on the border or outside the Gadolinium enhancing lesion and comprising a subset of the T2 abnormality. These findings suggest an important role for MRSI in directing biopsies and planning focal therapy. Preliminary studies using MRSI to evaluate gamma knife radiosurgery for patients with gliomas have indicated that the cohort with MRSI lesions larger than the radiation target had significantly reduced time to further treatment and survival. Further applications that are being investigated include response to external beam radiation and brachytherapy.

Transsphenoidal Surgery for Acromegaly – Ultrastructural and Histological Findings and Surgical Complications in a Contemporary Series

E. Laws, J. Kreutzer, MB Lopes, ML Vance

A rigorous analysis of transsphenoidal surgery for acromegaly has been performed in a consecutive series of 89 patients operated upon in the past eight years and followed for at least 12 months. Favorable outcome (remission – normal IGF-1 and OGTT nadir GH < 1.0 μg/ml) was achieved in 73.7%.

There was 10 complications in the 98 patients (11.1%), the most common being transient diabetes insipidus and transient SIADH.

Systemic analysis of immunohistochemical and ultrastructural characteristics was performed, and was correlated with outcome. Remission rates were lowest in invasive macroadenomas, GH-PRL tumors and the mammosomatotroph and acidophil stem cell tumors. The management of patients who do not achieve remission is also considered. Gamma knife radiosurgery was effective in salvaging 2/10 such patients.

The data support the safety and efficacy of transsphenoidal surgery for definitive initial therapy in acromegaly, and provide guidelines for management when surgical remission is not achieved.

THURSDAY, OCTOBER 12 11:20-11:40

ACADEMY AWARD PAPER

Robert Ratcheson Changes in Intrinsic Signals and Phosphorylated Growth-Associated Protein-43 of the Adult Rat Barrel Cortex Following Kainin Acid Induced Central Lesion. Tien T. Nguyen, Takamichi Yamamoto, Karina F. Meiri and Charles J. Hodge

In order to study the function and morphological changes of the adult brain following a central lesion, we used repeated intrinsic signal optical imaging studies and immunohistochemical staining of phosphorylated growth-associated protein (GAP)-43 to examine the rat barrell cortex following a kainic acid induced central lesion. The functional representation of the elicited principal whisker (PW) corresponding to the injected barrel in the repeat study relocated to the perimeter of the lesion. By the third day and until the second week after injection, the quantified area of the signals of the PW and of adjacent whiskers were significantly larger than those obtained before injection and were occasionally ameboid instead of the usual oval shape. This resulted in a significant increase overlapping of adjacent signals and shift in the signals' geometric centers. Increased immunoreactivity of the phosphorylated GAP-43 immunoreactivity protein, which is concentrated in the distal tip and growth cones of axons that have grown near its target, was present 3 days after KA injection and peaked in the first or second week. GAP-43 immunoreactivity decreased by the third week, presumably because the axons have completed growth and formed new synapse. After the second week, even though the PW signal remains relocated, the shapes and sizes of quantified whisker signals returned to those seen at baseline. We propose that the changes in functional representation in the first two weeks result from disinhibition and unmasking of latent synapses. After the second week, the functional reorganization was replaced by strengthened by physical remodeling by axon growth and new synapse formation.

ACADEMY AWARD RUNNER UP

In Vivo Optical Mapping of Epileptic Foci and Surround Inhibition in Ferret Cerebral Cortex.

Theodore H. Schwartz and Tobias Bonhoeffer

Abstract

Electrophysiological mapping of epileptic foci is the gold standard, both for animal research and human surgical resections. Despite its indisputable strengths there are significant sampling limitations inherent to the technique. Here we show that optical imaging of intrinsic signals, which can record the optical signals associated with neuronal activity from large cortical areas with sub-millimeter resolution, can be used to generate maps of pharmacologically-induced interictal, ictal and secondary mirror foci. Interictal foci are usually circular, with a sharp border, and each spike elicits a clearly discernible focal increase in blood flow and metabolism. A large region of a negative optical signal, correlating well with electrophysiologically recorded inhibition, can be seen in the surrounding cortex. Ictal onset zones were localized to regions as small as 1-2 mm^2 and when non-propagating were also surrounded by a strong inverted optical signal. Optical epilepsy maps can be generated within a matter of seconds with spike-triggered image division, making it a potentially very useful tool for guiding human epilepsy surgery.

THURSDAY, OCTOBER 12 12:00-12:20 PM

ACADEMY AWARD RUNNER UP

Understanding Oligodendrocyte Loss Following Early Ischemic Insult.

Kasia Petelenz, Robert H. Miller and Shenandoah Robinson

Objective: Cerebral palsy is strongly correlated with white matter lesions in the neonatal brain that reflect primarily disruption of oligodendrocyte development. Oligodendrocytes arise in the human brain during the third trimester and neonatal period, the time when the insults that cause cerebral palsy occur. Normal development of the oligodendrocyte lineage is dependent upon precise spatial and temporal regulation of complex cellular interactions involving growth factors and cytokines. Our hypothesis was that the abnormal expression of cytokines due to perinatal insults disrupts oligodendrocyte development by disturbing the expression of growth factors and cytokines critical for normal white matter development.

Methods: To test this hypothesis, a prenatal model of ischemia in rats was used to examine the disruption of oligodendroglial lineage cell development and the expression of growth factors induced by the prenatal insult with *in vitro* and *in vivo* immunohistochemistry, migration, proliferation and apoptosis assays. As part of this project, data from the animal model will be correlated with postmortem specimens from human infants that suffered perinatal insults. Results: The prenatal insult in the rat model did not affect the number of oligodendrocyte precursor cells, but caused a significant reduction in the number of mature oligodendrocytes. Preliminary data suggest the decrease in oligodendrocyte cell number was due to diminished survival rather than limited migration, proliferation, or differentiation of oligodendrocyte precursor cells. The perinatal insult appears to induce aberrant expression of cytokines that disrupts the secretion of survival factors by astrocytes and neurons that are necessary for oligodendrocyte survival.

Conclusions: The current study was designed to define the cellular and molecular mechanisms that mediate oligodendrocyte cell loss and white matter lesions following early injury to the CNS. Together these data will furnish comprehensive information on the response of oligodendrocyte lineage cells to a perinatal insult, and provide insights into potential interventions that could be administered in the neonatal period to minimize the development of white matter lesions. The disruption of oligodendrocyte development prevents normal myelination of axons, and thus hinders neuronal development. By restoring oligodendrocyte development in the neonate, we hope to minimize the incidence of cerebral palsy and associated neurologic deficits

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FRIDAY, OCTOBER 13 8:50 -9:05

Prospective Analysis of Preoperative Helical CT Angiography for C1-2 Transarticular Screw Placement: A New Technique

Volker K.H. Sonntag, MD, Nicholas Theodore, MD, and Shahram Partovi, MD*

Division of Neurological Surgery, and *Division of Neuroradiology
Barrow Neurological Institute, St. Joseph's Hospital and Medical Center
350 West Thomas Road, Phoenix, AZ

Introduction: Placing C1-2 transarticular screws is fraught with numerous potential complications, of which injury to the vertebral artery is one of the most devastating. Preoperative CT of the atlantoaxial complex is the standard imaging study but gives no information about the location of the vertebral artery in relationship to the foramen transversarium.

Methods: In addition to standard bone-windowed sagittal reconstruction CT, a helical CT scan (1.25 mm x 1 mm slices with an overlap of 0.25 mm) of the craniocervical junction was obtained prospectively from 12 patients (mean age, 44.1 years; range, 19-75) before C1-2 transarticular screws were placed. Visipaque 320 was administered intravenously, with a delay based on the patient's age and cardiac status. Post-processing included 3-D reconstructions of the vertebral arteries and sagittal and coronal reformatted images of the bony structures. Combining these images accurately represented anatomic relationships. Three patients had a chronic dens fracture; one had os odontoideum, and one had posttraumatic atlantoaxial instability.

Results: Bilateral screws were placed in 8 patients and unilateral screws in 4. CT angiography helped define 3 patients' unsuitability for transarticular placement, which was not readily evident on plain sagittal CT. In one patient CT angiography demonstrated an absent vertebral

artery, allowing successful screw placement. CT angiography was helpful in all cases.

Conclusion: CT angiography is an extremely useful preoperative adjunct prior to transarticular screw placement and should be considered in all cases. Further studies are required to maximize the usefulness of this innovative technique.

FRIDAY, OCTOBER 13 9:10-9:30 AM

DYNAMIC WEIGHT BEARING CERVICAL MRI

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Introduction: Conventional MR imaging of cervical spine disease can be limiting because patients are imaged while supine in a neutral position. Dynamic myelography provides good imaging studies in flexion and extension, but this requires an invasive procedure. The development of open configuration interventional MRI units has provided new opportunities in dynamic MR imaging.

Method: We reviewed a series of 10 patients who underwent diagnostic dynamic weight bearing images performed in our interventional GE Signa SP system. A removable chair that fits in the center of the gantry and a flexible cervical RF transmit and receive coil allows dynamic imaging. Patients are imaged in a total of 5 different positions including the neutral, fully flexed, and fully extended and two intermediate positions. Conventional supine imaging was then performed for comparison evaluations.

Results: This technique illustrates a greater degree of compressive pathology when the weight bearing images in extended positions are compared with standard supine images. In addition, evaluation of images performed with the spine in flexed or extended positions illustrate the changes in spinal cord compression and spinal column alignment that occur under normal physiologic stresses. The MR images taken in hyper-extension reveal greater degrees of spinal cord compression than in the flexed position.

Conclusion: Dynamic weight bearing MR imaging is a new and exciting technique for evaluating cervical spine pathology. This imaging modality is superior to conventional supine cervical MRI and its non-invasive nature and excellent image quality will likely allow it to replace cervical myelography.

Thoracic Pedicle Screws are Superior to the Existing Stabilization Techniques

Robert F. Heary, MD, Richard P. Schlenk, MD, Ceslovas Vaicys, MD, Thomas J. Sernas, PA-C, Margaret Black, ANP

Screws placed in the pedicles of the thoracic spine provide a biomechanically superior form of stabilization when compared to the existing alternative techniques. Standard thoracic stabilization techniques (hooks, rods, cables) immobilize the posterior column of the spine. Thoracic pedicle screws provide for stabilization of the posterior, middle, and anterior columns of the spine.

Twenty one patients (8 males, 13 females) with a mean age of 42 years (range 22-76 years) had placement of thoracic pedicle screws over a 2 year period. Surgery was performed for trauma (12), infection (5), congenital (2), degenerative (1), and neoplastic (1) conditions. Revision surgery to correct an iatrogenic spinal deformity from a prior failed procedure was performed in 7 patients. Nine patients had ASIA class A spinal cord injuries and 12 had incomplete lesions (ASIA classes C-E). A total of 141 screws were placed (T1-11, T2-14, T3-10, T4-8, T5-9, T6-12, T7-12, T8-10, T9-6, T10-14, T11-17, T12-18). Image guidance (Stealth) was utilized in 12 patients. Posterior surgeries were performed in 13 patients and 8 patients had AP surgeries. Real-time fluoroscopy was utilized in all cases; however, the images were often suboptimal in the upper thoracic spine. Postoperatively, plain film radiographs were performed on all patients and CT scans were obtained in the majority of patients. The mean duration of clinical and radiographic follow-up was 6 months. There were no nerve root, spinal cord, or vascular injuries resulting from inaccurate screw placement. A single patient required revision surgery for a laterally placed left T5 screw which was in close proximity, but did not violate, the aorta. There were no postoperative spine infections.

Pedicle screws offer numerous advantages to the destabilized thoracic spine. Thoracic pedicle screws: allow for shorter construct lengths; do not diminish the space available for the spinal cord; immobilize all 3 columns of the spine; effectively correct kyphotic deformity; frequently eliminate the need for anterior surgery; and decrease the need for postoperative use of external orthoses. Pedicle screws, used throughout the thoracic spine, are a safe and effective alternative compared to existing stabilization techniques.

Preoperative Detection of Trigeminal Neurovascular Compression by MRI Imaging in Patients with Typical and Atypical Trigeminal Neuralgia

Kim J. Burchiel, M.D.

In a prospective trial, a sequential series of patients with trigeminal neuralgia (typical and atypical), underwent T2 and 3D-TOF MRI imaging (MRA source images) to determine the rate at which neurovascular compression (NVC) of the trigeminal nerve could be detected by pre-operative imaging. MRI studies were evaluated by a neuroradiologist who was unaware of either the nature or side of the patient's pain. Twentyfive patients had MRI imaging, and 21 of these patients had a retromastoid craniectomy for microsurgical exploration of the region of the trigeminal nerve. Of the four patients who did not have surgery, one had a positive MRI indication of NVC, but remains on medical management; one patient had prior MVD and no demonstrable NVC; one showed no NVC, but later proved to have a neurotropic nasopharyngeal tumor of the mandibular nerve root; and one is awaiting surgery. Twenty of the 21 patients who had surgery proved to have NVC at the time of surgical exploration. The one exception was a patient with occipital neuralgia with an element of facial pain, who had negative MRI imaging and a completely negative exploration of the trigeminal nerve (true negative rate = 1/1 = 100%). Of the patients that showed NVC at surgery, 13 showed arterial NVC, and 12 of these were detected by MRI (12/13 = 92% arterial NVC detection rate). Seven patients proved to have venous NVC at surgery. Six of these patients had preoperative MRI imaging (one patient had severe claustrophobia and the MRI study was aborted). A total of 2/6 were detected by MRI pre-operatively (2/6 = 33% venous NVC detection rate). Thus, of the 19 patients with surgically-proven NVC who also had preoperative imaging, 14 instances of NVC were detected pre-operatively by MRI (true positive rate =14/19 = 74%). 5/19 patients had surgically-proven NVC and negative MRI imaging (false negative rate =26%), and in 4/5 cases the NVC was found to be venous in origin. 9/11 (82%) patients with typical TN were found

to have NVC by MRI (9/12 patients proved to have had arterial NVC at exploration). 4/6 (66%) patients with atypical TN were found to have NVC by MRI (4/6 had venous NVC at surgery). The two patients in this series with MS were both found to have NVC, and NVC was confirmed at surgery in both cases. This preliminary study demonstrates that trigeminal NVC may be reliably imaged in patients with trigeminal neuralgia. This technique may not only aid in decision making in patients with typical TN, but also in cases of atypical TN and symptomatic TN secondary to MS.

Analysis of posterior fossa volumes in patients with chronic fatigue syndrome, Chiari Type I malformations, and healthy controls.

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Introduction: Chronic fatigue syndrome (CFS) is a specific entity as defined by Center for Disease Control criteria (CDC - 1994). Decompression of the posterior fossa has been suggested for treatment of CFS, implying that these patients have "tight" posterior fossae or borderline cerebellar ectopia. We have analyzed posterior fossa volumes in patients with CFS, Chiari I malformations and healthy controls.

Methods: MRI studies were performed on a well-characterized cohort of CFS patients (n=20), as well as a gender and age matched group of healthy patients (n=16). Details of characterization criteria are published (Lange et al; J Neuro Sci. 1999). A group of consecutively operated Chiari I adult patients (N=30) were also analyzed. Volumetric analysis was performed using the method of Cavalieri. Readers were blinded as to diagnostic group, and controls of reader variability and consistency were performed.

Results: Posterior fossa volumes were 160.3 ($\pm$ 12.5) cm^3 for CSF subjects; 149.9 ($\pm$ 16.6) cm^3 for healthy controls; and 130 ($\pm$ 26.8) cm^3 Chiari I patients. The posterior fossa volumes for Chiari I patients were significantly smaller than both the healthy controls ($p=0.008$) and the CFS patients ($p<0.001$). Posterior fossa volumes of healthy controls and CFS patients were not significantly different ($p=.09$)

Conclusion: These studies indicate that CFS is not associated with an abnormally small posterior fossa; operations to treat CFS by posterior fossa enlargement are not supported by this data.

FRIDAY, OCTOBER 13 10:50-11:10

Clinical and PET Imaging Results after Neurotransplantation for Basal Ganglia Stroke

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Objective: Transplantation of cultured neuronal cells was shown to be safe in different animal models and effective at improving motor and cognitive deficits in rats with stroke. This study tested the safety and feasibility of human neuronal cellular transplantation in patients with substantial fixed motor deficits associated with basal ganglia stroke.
Methods: 12 patients with basal ganglia infarcts received stereotactic implants of neurons (Layton BioScience, Atherton, CA), randomized to either 2 or 6 million cells placed into and around the stroke. Inclusion criteria were age 40-75 years, stroke of 6 months to 6 years duration and a fixed motor deficit that was stable for at least 2 months. Assessments included the NIH stroke scale (NIHSS), European stroke scale (ESS), Barthel Index, SF-36 and were performed at 1,2,4,8,12,16,24, and 36 weeks, and then yearly. MRI and FDG-PET were used to assess brain metabolism and structure (1 cm area of stroke, cerebellum, and cortical lobes).

Results: All twelve patients (ages 44-75) underwent uncomplicated surgeries. Serial evaluations (12-18 months) showed no cell-related adverse serologic or imaging-defined effects. One patient suffered a single seizure and another had worsening of pre-existing renal insufficiency. Two patients had new remote strokes. The total ESS improved in 6 patients (range, 3-10 points), with an overall mean change from baseline of 2.9 points for the 12 patients ($p=.046$). The mean change at 24 weeks was 1.75 in the 2-million group and 5.25 in the 6-million group (p• .139). The ESS-Motor score improved by 2.5 points ($p=.026$). In the 6 million cell group, the mean BI improved 6.25 points and the SF-36, 6.5 points ($p\geq .172$). At baseline, PET showed marked hypometabolism at the stroke and throughout the hemisphere. Cerebellar diaschesis was frequent. An increase of $\geq$15% in the uptake of [F-18]fluorodeoxyglucose (FDG) at the implant site or in adjacent brain was noted in 6 of 11 patients. Changes in relative FDG uptake at 6 and 12 months post-surgery in the stroke ($p=.02$) and penumbra ($p=.006$) were correlated to performance on the motor subscale of the ESS.
Conclusions: The implantation of neuronal cells was feasible and accomplished without adverse effects. A greater number of patients had improvements in neurologic scale scores than had worsening or no change, and a trend to a better result with more cells was noted. Changes in FDG uptake on PET imaging correlated with clinical measures of neurologic function. A multi-center dose-response trial is planned.

Deep brain stimulation for Parkinson's disease: Which target?

Philip Starr, Chadwick Christine, Marsha Melnick, Heidi Clay, Susan Heath, and William Marks Jr.

University of California, San Francisco and San Francisco Veteran's Affairs Hospital

Introduction: Deep brain stimulation (DBS) is a promising technique for the treatment of advanced Parkinson's disease (PD). There are now 3 possible target sites in the brain: The thalamic ventralis intermedius nucleus (Vim), the globus pallidus internus (GPi), and the subthalamic nucleus (STN). We are performing a prospective randomized trial of DBS of the GPi or STN to assess which target provides superior symptomatic relief.

Methods: Implants are placed into the motor territory of the GPi or STN using MRI-based stereotaxy, microelectrode recording, and intraoperative test stimulation. Implants are staged, with 3-6 months between sides. Patients are evaluated preoperatively and at regular intervals postoperatively by a standardized rating scale of motor function, the Unified Parkinson's Disease Rating Scale (UPDRS); as well as computerized gait testing. All patients had a postoperative MRI to confirm lead location.

Results: Within our randomized study, we have implanted 62 leads in 43 patients. There were 2 small asymptomatic hematomas (<5 ml) detected on postoperative MRI. There was 1 lead fracture requiring replacement, and 1 pulse generator infection requiring replacement. In both the GPi and STN stimulation groups, all cardinal signs of PD improved with both unilateral and bilateral stimulation, as did gait. Thus far, the clinical outcomes are not different between the two groups.

Conclusions: Deep brain stimulation of the GPi or STN are safe and effective for improving all cardinal signs of PD. With short-term follow-up in a randomized study design, there is no difference in efficacy for the two target sites.

FRIDAY, OCTOBER 13 11:30-11:50 AM

Localization of Language Function in Children: Results of Electrical Stimulation Mapping

Steven G. Ojemann, M.D., Mitchel S. Berger, M.D., Ettore Lettich, R.E.E.G.T., George A. Ojemann, M.D.

Abstract: Electrical stimulation mapping has provided substantial information about the cortical organization of language in adults, and can be applied to the study of language acquisiton in children. The localization of sites where electrical stimulation produced significant naming errors in 30 children ages 3-16 are presented here. Mapping was performed in the intraoperative setting in 8 patients, and in the extraoperative setting by stimulating across a subdural grid in 22 patients. Considerable variability was found in the localization of language sites. The surface area of contiguous sites of stimulation that generated significant error rates was categorized as less than 1 cm^2, sites around 1 cm^2 but with indeterminate boundaries, and sites greater than 2.5 cm^2. The location and surface area of language sites, as well as the presence of multiple, noncontiguous sites was compared to patient age, gender, date of seizure onset, and preoperative verbal IQ. No significant relationship between language localization and patient age emerged, but females were more likely to have stimulation-evoked errors in the superior temporal gyrus ($P<0.005$), while patients with lower VIQs were more likely to have sites in the middle temporal gyrus ($P<0.04$). Patients in whom the surface area over which stimulation produced errors was greater than 2.5 cm^2 were more likely to have a lower VIQ ($P<0.03$). Surface area was not significantly related to age. These findings are relevant to theories of the intrahemispheric and interhemispheric organization of cortex devoted to language function, and support the concept that increasing facility with language is represented by a smaller area of cortex. However, language representation by age 4 appears no different in terms of surface area or location within the dominant hemisphere than is found in older age groups.

Possible functional consequences of neurogenesis in temporal lobe epilepsy

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Recent studies have shown that granule cell neurogenesis occurs normally in the adult mammalian brain and is increased after seizures, ischemia, and exercise in rats. However, the functional consequences of this cell birth remain largely unknown. We are investigating granule cell neurogenesis in the rodent pilocarpine and kainic acid models of temporal lobe epilepsy (TLE) and in human TLE. Following kainic acid or pilocarpine seizures, there is a dramatic increase in neurogenesis in the dentate granule cell layer, as shown by BRDU and calbindin double labeling. A subpopulation of these neurogenic cells aberrantly migrate into the hilar region, forming a plexus of calbindin positive cells at the CA3/hilar border. This plexus is GABA, GAD, NPY, somatostatin, calretinin and parvalbumin-negative. Immunohistochemical staining of resected human hippocampal specimens similarly shows rare granule-like cells that have migrated into the hilar region. Simultaneous electrophysiological recordings from neurogenic hilar cells and CA3 pyramidal neurons reveal spontaneous epileptiform burst activity that is synchronized between these populations of cells. Studies are underway in rodent and human epileptic tissue to further characterize the physiological properties of neurogenic granule cells and to compare the single cell molecular phenotype of mature dentate gyrus granule cells, neurogenic dentate gyrus granule cells, and aberrantly migrated hilar granule cells. In addition, we are investigating whether increasing neurogenesis alters seizure susceptibility and/or seizure related cell death. The abnormal migration and synaptic connectivity of neurogenic granule cells following seizures may contribute to the development and maintenance of temporal lobe epilepsy.

FRIDAY, OCTOBER 13 12:10-12:30 PM

Experience with a Statewide Carotid Endartectomy Registry in New York

A. John Popp, M.D. and Edward L. Hannan, PhD.

A statewide registry to collect data for assessment of outcome after Carotid Endartectomy (CE) was established with funding from the New York State (NYS) Clinical Guidelines and Medical Technology Assessment Program. The study ran for 2 years (1997-1999) and used a data collection instrument designed by an advisory group composed of neurosurgeons and vascular surgeons. The accuracy of information collected by the registry was monitored by comparison with New York's acute care hospital database, the Statewide Planning and Research Cooperative System (SPARCS).

60 surgeons (7%) performing CE in NYS submitted 3928 records to the registry representing 23% of all CE's performed in NYS during the period the registry was active. When patients' records were compared with SPARCS only one death was not coded in the registry. The in-hospital mortality rate in the registry was 0.69% (27 deaths) compared to a mortality rate of 1.10% for all patients in NYS undergoing CE. Patient and process of care characteristics were analyzed using a stepwise logistic regression model; those significantly associated with in-hospital mortality included CABG surgery during same admission; Age > 79; Female Gender; Congestive heart failure; Diabetes; Cresendo TIA, RIND or Stroke in evolution; non use of shunt; and procedure performed by a general surgeon.

In conclusion: 1) Collecting accurate outcome data submitted voluntarily by the operating surgeon over a broad geographic area is feasible. 2) In this population of patients certain patient characteristics, comorbidities, processes of patient care and surgeon type were associated with increased in-hospital mortality.

Operative Management of Post-AVM Resection Hemorrhage in the Absence of Residual AVM

Duke Samson, MD, Thomas Kopitnik, Jr., MD, H. Hunt Batjer, MD

Over a nineteen year period, four hundred ninety-seven patients underwent resection of intra-parenchymal arteriovenous malformations. In that patient population, forty-four procedures were performed to remove residual malformation (35) or to evacuate acute post-resection hemorrhage within the resection bed and adjacent brain tissue (9).

The majority of reoperations (21) occurred in the immediate post- resection period and were targeted at residual malformation identified on acute post-operative angiography while the patient was still under the operative anesthetic. Nine patients underwent delayed operations for residual malformation, eight of whom were referred from other centers following their initial resections. Five patients having undergone steroetaxic-focused radiation as the preliminary mode of therapy have been operated at Southwestern, three in the presence of progressive neurological deficit secondary to radio-necrosis.

Nine patients, all with large symptomatic lesions that underwent preliminary embolization, developed hemorrhage and or hemorrhagic edema/ infarction at the site of the resected AVM. In all nine this occurred following uncomplicated surgical resection and post-operative angiography documenting no AV shunting. The complication was ictal in nature in three patients, came on over several hours in two and evolved over a longer period in the remaining four. All had focal neurological deficits related to the delayed hemorrhage and four patients had evidence of major intracranial mass effect. Repeat angiography showed persistant dilatation of feeding vessels and leptomeningeal collaterals and no other abnormality. Barbiturate coma was unsuccessful in preventing progressive evidence of edema, mass effect and hemorrhage in three patients and all nine were eventually returned to the operating room for re-exploration.

Intraoperatively, in each patient a tenacious clot was found to occupy the almost-effaced resection bed; the surrounding brain was uniformly hemorrhagic, inordinately swollen and friable, and uniformly contained multiple small arterial branches which bled copiously. In no instance was simple clot removal and minimal debridement possible - a major revision of the entire resection bed was always necessary, with circumferential identification of healthy pial and parenchymal margins and establishment of secure hemostasis at every level. This procedure was in every case a major operative undertaking requiring several hours and significant blood replacement. Routine use of an intracranial pressure monitor and barbiturate coma was necessary in the early postoperative period, and one patient required a second reoperation for recurrent bleeding despite a second unremarkable angiogram. One patient died, and five had significant exacerbation of their pre-operative neurological deficits. Despite protracted critical illnesses, a total of five patients made functional neurological recoveries.

The exact etiology of post–resection hemorrhage in the angiograhic absence of residual AVM is uncertain. Obviously in some situations, small amounts of malformation can be occult or unrecognized on the initial post-resection angiogram. In others, this phenomena probably represents a hyperperfusion abnormality ("normal perfusion pressure breakthrough"), and in others inadvertent surgical compromise of venous return may play an important role. Prompt recognition of the identity and gravity of the complication and early, aggressive operative management coupled with rigorous post-resection hemodynamic control may offer the best resolution of this fortunately infrequent occurrence.

NOTES:

Identifying patients at risk for post-procedural morbidity following treatment of Incidental Intracranial Aneurysms: the role of aneurysm size and location.

Vallabh Janardhan, MD[1,2] Robert Friedlander, MD[3-6] Sarajune Dagen, RN[3-5] Philip Edwin Stieg, Phd, MD[3-6]

[1]Department of Neurology, [2]Boston University School of Medicine, [3]Cerebrovascular Center, [4]Division of Neurological Surgery, [5]Brigham and Women's Hospital, [6]Harvard Medical School, Boston, Massachusetts.

Objective: A decision to treat Incidental Intracranial Aneurysms (IIAs) relies on understanding the risks of treatment and weighing them against the risks of aneurysm rupture. While the natural history of IIAs is currently being studied, the morbidity and mortality associated with treating IIAs and factors associated with poor outcome need to be clearly established.

Methods: One hundred and sixty IIAs were treated either surgically (n=152) or endovascularly (n=8) in a consecutive series of 125 patients. Aneurysms were graded based on size into small (<13 mm) and large (≥13mm) and based on location into anterior and posterior circulation aneurysms. Outcomes were assessed at 1-month and 6-months post-operatively. Post-procedural morbidity was defined as a new neurologic deficit associated with a score ≥3 on the Modified-Rankins Scale or ≤24 on the Mini-Mental Status Examination. Logistic regression analysis was used to identify predictors of post-procedural morbidity from retrospectively collected data on demographic, clinical and angiographic characteristics of the patients.

Results: Treatment of IIAs was not associated with any mortality and was associated with post-procedural morbidity in 13.6% (17/125) and 6.4% (8/125) of patients at 1-month and 6-months respectively. Treatment of aneurysms (≥13 mm) [odds ratio (OR) 0.30; 95% CI 0.09-0.96] and posterior circulation aneurysms [OR 0.24; 95% CI 0.06-0.95] were independently associated with post-procedural morbidity. Subgroup analysis of patients with poor outcome (n=8) showed that 75% (6/8) and 38% (3/8) of patients

had aneurysms with broad and calcified necks respectively. Age, co-morbidities, multiple aneurysms, specific aneurysm location and history of sub-arachnoid hemorrhage from a different aneurysm were not significantly associated with poor outcome.

Conclusion: We have shown that IIAs can be safely and effectively treated without any mortality and the associated morbidity is less than previously reported. A combination of angiographic variables can be helpful in identifying patients at risk for post-procedural morbidity.

SATURDAY, OCTOBER 14 9:10 – 9:30 AM

Regulation of Vascular Endothelial Growth Factor (VEGF) By Hemodynamic Forces In Brain Microvascular Endothelium

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Hemodynamic factors have long been proposed to play a role in the regulation of blood vessel growth and structure, as manifested by the increased caliber of high-flow arterial feeders to AVMs and, conversely, by the regression of arteries in low-flow states. Vascular Endothelial Growth Factor (VEGF) is a potent autocrine growth stimulator known to play a crucial role in embryonic vascular development and blood brain barrier permeability.

We hypothesized that the endothelial production of VEGF may be regulated by prevailing hemodynamic factors. In order to reproduce physiologic flow *in vitro*, we exposed brain microvascular endothelial cells (BMEC) to hemodynamic shear stress using a specially designed cone-plate device.

Physiological flow produced a dramatic cell shape change from polygonal to spindle-like, alignment in the direction of the flow vector, and appearance of short actin fiber bundles. Flow resulted in upregulation of nitric oxide synthase and supression of endothelin-1 gene expression. In addition, flow rapidly increased levels of VEGF mRNA in a biphasic manner with a transient response at venous shear magnitude (4 dyn/cm^2) and a sustained 2.5-fold increase at high arterial shear magnitude (20 dyn/cm^2). Flow similarly augmented the release of VEGF peptide, but was without effect on the expression of the VEGF receptor flk-1. Medium from flow-exposed endothelium partially conferred VEGF increase, while reproduction of endothelial blood-brain barrier phenotype by C6 glioma-conditioned medium was found to suppress both basal and flow-induced VEGF expression.

In conclusion, we have identified physiological flow as a powerful regulator of the angiogenic growth factor VEGF. This finding defines a novel feedback loop of hemodynamic control of angiogenesis and blood-brain barrier permeability that has major implications to the pathophysiology of AVMs, cerebral ischemia, cerebral reperfusion phenomena, and tumor vessel homeostasis.

Ornithine decarboxylase and normal adaptive responses to transient cerebral ischemia.

RJ Dempsey, VLR Rao, A Dogan, KK Bowen, AM Rao, JF Hatcher

Introduction: Cerebral ischemia leads to increased ornithine decarboxylase (ODC) expression. Contradicting studies attributed both neuroprotective and neurotoxic roles for ODC after ischemia. ODC triggers the formation of polyamines essential for cell division and programmed cell death in embryogenesis and neoplasia.

Aim: To understand the functional significance of ODC induction after focal cerebral ischemia by antisense knockdown methods.

Experiments: Transient focal cerebral ischemia was induced in adult, male spontaneously hypertensive rats by an intraluminal middle cerebral artery occlusion (MCAO). At various reperfusion periods, ODC expression was studied by immunohistochemistry and Western blotting. ODC catalytic activity was assessed by estimating the formation of $^{14}CO_2$ from ^{14}C-ornithine. Using antisense oligonucleotides (ODNs), the functional role of ODC in the neuronal events after transient focal cerebral ischemia was evaluated.

Results: Transient MCAO significantly increased ODC immunoreactive protein levels and catalytic activity in the ischemic cortex between 3h to 24h of reperfusion. Infusion of antisense ODNs specific for ODC completely prevented the ischemia-induced increase in ODC protein and activity. Transient MCAO in rats infused with ODC antisense ODNs increased the motor deficits, mortality and infarct volume compared to ODC sense ODN infused controls. The number of viable neurons in the cerebral cortex and striatum of the rats undergoing transient MCAO were significantly lower in the ODC antisense ODN infused rats compared to sense ODN infused rats.

Conclusions: Transient focal cerebral ischemia leads to persistent increase in ODC expression within the ischemic regions of brain. Preventing the ODC induction exacerbates ischemic neuronal death and neurological deficits. Results of the present study suggest an essential neuroprotective role of ODC after transient focal ischemia that can be therapeutically enhanced.

SATURDAY, OCTOBER 14 9:50-10:10 am

Multifactorial Analysis of Surgical Outcome in Patients with Unruptured Middle Cerebral Artery Aneurysms.

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Background: Some of the well known predictors of the clinical outcome of patients with ruptured aneurysms are not useful in forecasting outcome of patients with unruptured aneurysms. The goal of the current study was to develop a predictive tool for assessing both favorable outcome and morbidity in a large series of unruptured aneurysms.

Methods: We analyzed 93 patients harboring a total of 101 unruptured middle cerebral aneurysms who underwent surgical clipping. Intraoperative data was reviewed and 7 factors that might influence outcome were identified. These include 1. aneurysm size above 10 mm, 2. presence of a broad neck, 3. presence of intra-aneurysmal plaque, 4. clipping of more than one aneurysm during the same surgery, 5. temporary occlusion of the middle cerebral artery (MCA), 6. multiple clip applications and repositionings and 7. the use of multiple clips. The entire group of unruptured middle cerebral artery aneurysms was divided into two subgroups on the basis of outcome. Each patient was subsequently analyzed for the Factor Accumulation Index (FAI), the sum of different factors observed in a given patient.

Results: The expected outcome subgroup, those who developed not neurological deficits, was represented by 86 patients, with a total of 92 aneurysms and demonstrated the following results: no factors were found in 6 patients, an FAI of 1 was found in 24 patients, 23 patients had an FAI of two, FAI of 3 was present in 12 patients, FAI of 4 was documented in 11 patients, FAI of 5 in 8 patients, one patient had an FAI of 6 and only one patient had a sum of seven factors. Seven patients who developed post-operative deficits represented the subgroup of unexpected outcomes with total morbidity of 7.5%. There were no deaths. None of the patients in this subgroup accumulated FAI of 0, 1, 2 or 5.
A FAI of 3 was found in 2 patients. Two patients accumulated an FAI of 4. An FAI of 6 was exhibited by one and an FAI of 7 by two patients.

Conclusion: We believe that it is possible to predict the outcome in patients with unruptured middle cerebral artery aneurysm by calculating FAI. The postoperative morbidity increases with FAI of 3 or higher.

SATURDAY, OCTOBER 14 10:10-10:30

Remote/Cerebellar Hemorrhage Complicating Pterional Craniotomy – Further Perspectives from the International Study on Unruptured Intracranial Aneurysms

David G. Piepgras and James Torner

Remote intraparenchymal hemorrhages in the superior cerebellum and sometimes associated hemorrhages in the contralateral cerebral hemisphere, occurring after apparently uncomplicated frontotemporal craniotomy, have been documented in case reports and small series in the past decade.

At our center we have been aware of this complication in approximately 40 cases identified over a 20 year span. Although we and others have suspected positional venous obstruction and secondary venous infarction as the etiology, the exact pathophysiologic mechanism remains unclear.

In the ISUIA prospective study of patients treated for unruptured intracranial aneurysms, 996 patients underwent open intracranial surgery, of which 43 patients suffered major morbidity and mortality related to intra- or post-operative non-aneurysmal intracranial hemorrhage. Seven of these were found to be remote hemorrhages, 5 of them major in degree, and 2 with fatal outcome. These cases will be discussed in the context of the ISUIA series and the larger problem of idiopathic remote intracerebral hemorrhage complicating pterional craniotomy. Further investigations and clarification of the contributing factors and mechanisms of these hemorrhages are necessary to assure their prevention.

Approaches to Anterior Inferior Cerebellar Artery: Experience with 38 Cases

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Anterior inferior cerebellar artery (AICA) aneurysms are among the most rare aneurysms and among the most difficult to treat surgically because they are located adjacent to the brain stem in a compact, critical area. The treatment of these aneurysms requires a facility with several skull base approaches because the preferred exposure depends on the aneurysm's anatomy (size, location, orientation) as well as on the patient's clinical presentation and the surgeon's experience.

Thirty-six patients with 38 AICA aneurysms underwent surgical clipping of their lesion. The surgical approaches to these aneurysms included the retrosigmoid, far-lateral, translabyrinthine, transcochlear, subtemporal, and orbitozygomatic. Deliberate cardiac standstill was used in 17 patients to facilitate clipping. Postoperative angiograms demonstrated that 33 (87%) aneurysms were obliterated. Small residual necks were seen in three patients. One patient had a moderate-sized residual neck that incorporated a perforating vessel. The postoperative 30-day mortality rate was 2.6% (one patient).

A retrosigmoid approach is advocated for small- to medium-sized aneurysms associated with the lower two-thirds of the clivus. An orbitozygomatic approach can be used to access high-riding AICA aneurysms. Large and giant AICA aneurysms, particularly those oriented toward the brain stem, require a transpetrosal approach with or without intraoperative cardiac standstill. As we gained more experience with these aneurysms, the classic retrosigmoid approach became our favorite and most frequently used exposure.

The Toxicity of Intracerebral Hemorrhage

Julian T. Hoff, M.D.

Intracerebral hemorrhage has a variety of causes, most common of which is spontaneous intracerebral hemorrhage usually related to hypertension. The problem causes significant morbidity and mortality around the globe. Clinical investigations over the past fifty years have generally concluded that operative therapy provides no benefit over medical therapy. Wiley McKissock studied the problem first, concluding that surgery did not improve the outcome of patients with intracerebral hematomas. David Mendelow recently initiated the Surgical Therapy Intracerebral Hemorrhage Trial (STICH) involving many centers hoping to reach a conclusion that would provide reliable guidelines for clinicians. That trial allows a 72-hour period from ictus to surgical treatment with follow-up of patients over many months.

While there are uncertainties about the human condition, the entity is clearer in the laboratory. Over the past two decades, experiments with various animal models have shown that an intracerebral hematoma is accompanied by edema in brain adjacent to and remote from the clot in those animals that survive the initial hemorrhage. The formation of edema causes increased mass effect and progressive neurological deficits. Edema is triggered by the coagulation cascade, erythrocyte lysis, releasing toxic hemoglobin byproducts, and activation of the complement cascade, which is part of the inflammatory process, initiated by the insult. These processes begin within an hour of the hemorrhage and continue for several days before resolution begins.

It is reasonable to assume that findings in the laboratory are applicable to patients. If the events described above are to be dealt with better in patients, the clot should be removed as soon as possible, preferably within a few hours of ictus. The clinical question, ie, who needs operation and who does not requires a protocol that fits experimental evidence with many matched patients followed to conclusion.

Inhibition of HMG-CoA reductase by simvastatin reduces infarct after embolic stroke in mice.

Sepideh Amin-Hanjani MD, Minoru Asahi MD, Nancy Stagliano PhD, Sunu Thomas Bsc, Eng H Lo PhD, James K Liao MD, Michael A Moskowitz MD.

Introduction. Simvastatin, an HMG-CoA reductase inhibitor (statin), is a widely used cholesterol lowering agent demonstrated to reduce the incidence of myocardial infarction and stroke in clinical trials. Cholesterol independent actions of statins including upregulation of endothelial nitric oxide (eNOS) and tissue plasminogen activator (tPA) have been reported. Previous experiments have established a protective effect of chronic simvastatin pretreatment in mice after filament MCA occlusion, but have not examined infarct protection in the more relevant setting of embolic stroke. In the present study we investigated the protective effects of simvastatin in a mouse model of focal embolic stroke.

Methods. Adult male SV-129 mice (20-30g) treated with 20 mg/kg/day simvastatin or vehicle for 14 days were subjected to permanent focal ischemia by cerebral embolus to the proximal MCA. Simvastatin treated mice were exposed to thrombus derived from either simvastatin (homologous) or vehicle (heterologous) treated mice (n=8-12 per group). Vehicle treated animals were exposed to vehicle derived clots only. Neurological deficits were assessed and cerebral infarct volumes measured at 22 hrs. Arterial blood pressure and gases, regional cerebral blood flow, and cholesterol levels were measured in a subset of animals. Endothelial tPA and PAI-1 (plasminogen activator inhibitor-1) mRNA levels were determined.

Results. Simvastatin reduced neurological deficits and infarct size after embolic stroke. A 35% reduction in stroke size ($p<0.05$) was seen in simvastatin treated animals regardless of the source of the embolic thrombus. Levels of endothelial tPA were upregulated, and PAI-1 downregulated in a time and concentration dependent fashion. Physiologic parameters including cholesterol levels did not differ significantly among groups.

Discussion. Simvastatin upregulates eNOS and mediators of fibrinolysis, and offers infarct protection in an embolic model of stroke. These actions may indicate a strategy for stroke prevention in humans.

Functional gap junction communication between malignant glioma cells and vascular endothelial cells

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Department of Neurosurgery, New York Medical College, Valhalla and New York, NY

The authors have previously described the ability of malignant glioma cells to communicate with and phenotypically modify neighboring non-tumor astrocytes both *in vitro* and *in vivo*. The present study was undertaken to examine the nature of communication between human malignant glioma cells and endothelial cells in tissue culture.

Human umbilical vein endothelial (HUVEC) cells and glioblastoma cell line U87 cells (both obtained from the ATCC) were use in the study. U87 cells were double labeled with gap junction permeable dye, CDCF (green) and DiIC18 (red) before mixing with unlabeled endothelial cells. Dye transfer was measured using Bio-Rad confocal microscopy, in which functional gap junction communication between U87 and HUVEC cells was demonstrated. Furthermore, the HUVEC cells were prelabeled with DiIC18 and co-cultured with U87 cells, and dye transfer was noted.

The confluent cultures were labeled with Fluo-3 and the intercellular calcium signaling was measured using confocal microscopy. After mechanically stimulation of one cell, calcium waves were seen from glioma cells to endothelial cells. These findings suggest that direct bidirectional cellular interaction occurs between malignant glioma cells and endothelial cells *in vitro*, and such gap junction and calcium signaling may represent a potential mechanism for regulation of glioma angiogenesis.

A Nitric oxide donor reverses the attenuation of cerebrovascular reactivity to hypercapnia by traumatic brain injury in a rat model of controlled cortical impact

F Zhang, MD, S Sprague, MN Henry, MD, MG Son, MD, DG Vollmer, MD, Division of Neurosurgery University of Texas Health Science Center at San Antonio

Traumatic brain injury (TBI) attenuates the cerebrovasodilation to hypercapnia. Cortical spreading depressing (CSD) also transiently reduces hypercapnic vasodilation. We sought to determine 1.) whether the CSD elicited by the controlled cortical impact (CCI) injury masks the true effect of the TBI on hypercapnic vasodilation at the early stage after injury, 2.) whether nitric oxide (NO) donor can reverse the attenuation of hypercapnic vasodilation following CCI. Anesthetized rats underwent CCI of 8m/s, 2.5mm and 50ms. The CBF was monitored with laser Doppler flowmetry. In non-injured cortex ipsilateral to the CCI, a single wave of CSD was recorded and CO2 response at this location was significantly attenuated for up to 30 min ($P<0.05$, n=7). At the injured cortex, hypercapnic vasodilation remains attenuated for at least 7 hours. The cerebrovasodilation to CO2 was 37±5% in injured rats (n=6) vs 84±10% in the sham-injured group (n=5), $P<0.05$. After topical superfusion with the NO donor, S-nitroso-N-acetylpenicillamine for 30 min, hypercapnic vasodilation was restored to 74±7% (n=9, $p>0.1$ compared with that of the sham group). In contrast, papaverine, a potent vasodilator independent of the NO pathway, failed to reverse the attenuation of CO2 response by CCI. We conclude that CSD elicited by CCI can mask the true effect of TBI on hypercapnic vasodilation for at least 30 minutes. Exogenous NO, but not papaverine, can reverse the attenuation of cerebrovascular reactivity to CO2 by TBI. This result supports the hypothesis that NO production is reduced after TBI and NO donors have a potential beneficial role in clinical management of head injury.

Vasoreactivity After Head Injury: A Transcranial Doppler Study

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Contemporary management of head injured patients is based on assumptions regarding carbon dioxide (CO_2) reactivity, pressure autoregulation (PA), and vascular reactivity to pharmacological metabolic suppression. In this study, serial assessments of vasoreactivity were performed using transcranial Doppler (TCD) of the middle cerebral artery (MCA).

Twenty eight patients, (mean age 32 ± 13 years, median GCS 7), underwent a total of 61 vasoreactivity testing sessions during post-injury days 0 to 13. CO_2 reactivity, PA, and metabolic suppression reactivity were quantified for each hemisphere by measuring changes in MCA velocity in response to transient hyperventilation, arterial blood pressure elevation or propofol-induced burst suppression, respectively.

One or both hemispheres had below normal vasoreactivity scores in 39.7%, 68.6% and 97.1% of study sessions for CO_2 reactivity, PA, and metabolic suppression reactivity ($p< 0.001$), respectively. Global impairment of CO_2 reactivity, PA and metabolic reactivity was associated with simultaneous intracranial hypertension ($p < .05$ for all 3 comparisons). Other significant correlates of impaired vasoreactivity included hypotension or hypoxia ($p<.05$) and low cerebral perfusion pressure (CPP), ($p<.01$) for CO_2 reactivity; low CPP ($p < .01$) for PA; and ipsilateral MCA territory hemorrhagic brain lesions ($p<.01$) and vasospasm ($p<.05$) for metabolic suppression reactivity. Six month Glasgow Outcome Scale score correlated with overall degree of impaired vasoreactivity ($p < .05$). In summary, during the first 2 weeks after head injury, CO_2 reactivity remained relatively intact, PA was variably impaired and metabolic suppression reactivity remained severely impaired. Elevated ICP appears to affect all three tested components of vasoreactivity. Incorporation of vasoreactivity data may facilitate more injury-specific and time-specific therapies for head injured patients.

Source of support: National Institutes of Neurological Disorders and Stroke, grant # NS30308 and Astra-Zeneca Pharmaceuticals

NOTES:

NOTES:

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L Brannon Thomas 1995
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Gregory Canute 1997
Nathan R Selden 1998
Robert M Friedlander 1999

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Roosevelt Hotel, New Orleans, Louisiana..... October 27-29, 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
Broadmoor 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, Texas ..... October 4-6, 1951
Waldorf-Astoria Hotel, New York City,
New York ..... 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, Cincinnato, 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, Mexico..... 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
Walforf-Astoria Hotel, New York City, New York ..... 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
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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
Loews 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
The Broadmoor, Colorado Springs, Colorado ..... October 11-14, 2000

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ROBERT S. BOURKE Rockville, Maryland (Senior)	1983	1996
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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)

HANS-PETER JENSEN 1980 2000
Kiel, GERMANY
(Senior Corresponding)

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)

JOHN E. RAAF 1938 2000
Portland, Oregon
(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)

FUTURE MEETINGS

2001 – November 12-17
The Breakers – Palm Beach, FL

2002 – October 14-19
The Phoenician – Scottsdale, AZ

2003 – October 27–November 2
Colonial Williamsburg – Williamsburg, VA

Mark your calendars now!