THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

63rd Annual Meeting

November 14 - 17, 2001

American Association of Neurological Surgeons

Jointly Sponsored by the American Association of Neurological Surgeons

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2001 Annual Meeting of the

American Academy of Neurological Surgery

GENERAL INFORMATION

REGISTRATION DESK LOCATION AND HOURS:

Wednesday, Nov. 14	South Loggia East	2:00 PM – 8:00 PM
Thursday, Nov. 15	South Ballroom Foyer	6:30 AM – 3:00 PM
Friday, Nov. 16	South Ballroom Foyer	6:30 AM – 2:00 PM
Saturday, Nov. 17	South Ballroom Foyer	8:00 AM – 1:00 PM

SPEAKER READY ROOM

The Speaker Ready Room is located in the South Mezzanine 2 and will be open:

Wednesday, November 14	6:30 AM – 7:00 PM
Thursday, November 15	6:30 AM – 7:00 PM
Friday, November 16	6:30 AM – 7:00 PM
Saturday, November 17	6:30 AM – 1:00 PM

Telephone number for The Breakers: 561-655-6611
Facsimile number: 561-659-8403

PROGRAM SUMMARY

WEDNESDAY, NOVEMBER 14

EVENTS	TIME	LOCATION
Speaker Ready Room	6:30 AM – 7:00 PM	South Mezzanine 2
Registration	2:00 PM – 8:00 PM	South Loggia East
Executive Committee Meeting	2:00 PM – 5:00 PM	South Mezzanine A
OPENING RECEPTION		
Cocktail Dinner (Dressy)	6:30 PM – 9:30 PM	Mediterranean Courtyard (Back-up for weather – Mediterranean Ballroom)

THURSDAY, NOVEMBER 15

EVENTS	TIME	LOCATION
Registration	6:30 AM – 3:00 PM	South Ballroom Foyer
Speaker Ready Room	6:30 AM – 7:00 PM	South Mezzanine 2
Business Breakfast Meeting **For Academy Members Only**	7:00 AM – 8:00 AM	Ponce De Leon I
Breakfast for Guests and Spouses 7:00 AM – 9:00 AM Buffet 9:00 AM – 11:00 AM Coffee and Danish	7:00 AM – 11:00 AM	L'Escalier/Florentine
Scientific Session	8:00 AM – 1:00 PM	Ponce De Leon II
PROGRAM FOR SPOUSES		
Chat with Mr. Ponce	9:15 AM – 9:30 AM	L'Escalier/Florentine
Walking Tour of Hotel with Mr. Ponce	9:30 AM – 10:30 AM	

PROGRAM SUMMARY

OTHER ACTIVITIES

Tennis	Golf
Shopping	Fishing – Transportation to Boat Not Provided

OFFSITE EVENT

Flagler Museum	2:00 PM – 4:30 PM	Bus Transportation Provided (Lunch on your own)
Trolley shuttles to and from Worth Avenue	1:00 PM – 5:00 PM	

DINNER

Caribbean Night (Casual dress)		Ponce De Leon Lawn
Reception	6:30 PM – 7:30 PM	(Back-up for weather –
Dinner	7:30 PM – 10:00 PM	Venetian Room)

FRIDAY, NOVEMBER 16

EVENT	TIME	LOCATION
Registration	6:30 AM – 2:00 PM	South Ballroom Foyer
Speaker Ready Room	6:30 AM – 7:00 PM	South Mezzanine 2
Business Breakfast Meeting **For Academy Members Only**	7:00 AM – 8:00 AM	Ponce De Leon I
Breakfast for Guests and Spouses 7:00 AM – 9:00 AM Buffet 9:00 AM – 11:00 AM Coffee and Danish	7:00 AM – 11:00 AM	Magnolia Room

PROGRAM FOR SPOUSES

Book Review	9:00 AM – 11:00 AM	Magnolia Room

PROGRAM SUMMARY

Scientific Session	8:00 AM – 1:00 PM	Ponce De Leon II
OFFSITE EVENT		
Norton Museum	2:00 PM – 4:30 PM	Bus Transportation Provided (Lunch on your own)
Trolley Shuttles to and from Worth Avenue	1:00 PM – 5:00 PM	
OTHER ACTIVITIES		
Tennis Golf	Shopping Fishing – Transportation to Boat Not Provided	
PRESIDENT'S RECEPTION **All members and guests are invited**	7:00 PM – 8:00 PM	Mediterranean Ballroom
BLACK TIE DINNER	8:00 PM – 11:00 PM	Circle Dining Room

SATURDAY, NOVEMBER 17

EVENT	TIME	LOCATION
Registration	8:00 AM – 1:00 PM	South Ballroom Foyer
Speaker Ready Room	6:30 AM – 1:00 PM	South Mezzanine 2
Breakfast for **All Members and Guests**	7:00 AM – 8:00 AM	Mediterranean Ballroom
Spouse Breakfast 7:00 AM – 9:00 AM Buffet 9:00 AM – 11:00 AM Coffee and Danish	7:00 AM – 11:00 AM	Mediterranean Ballroom
Scientific Session	8:00 AM – 1:00 PM	Ponce De Leon II

SOCIAL ACTIVITIES FOR SPOUSES

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

Wednesday, November 14

6:30 – 9:30 PM	Opening Reception – *Mediterranean Courtyard* Dressy

Thursday, November 15

7:00 – 11:00 AM	Spouse & Guest Breakfast–*L'Escalier/Florentine* 7:00 – 9:00 AM Buffet 9:00 – 11:00 AM Coffee & Danish
9:15 – 9:30 AM	Chat with Mr. Ponce – *L'Escalier Florentine*
9:30 – 10:30 AM	Walking Tour of Hotel with Mr. Ponce
1:00 – 5:00 PM	Shopping – Trolley Leaves for Worth Avenue
2:00 – 4:30 PM	*Flagler Museum – *Bus transportation provided*
6:30 PM	Dinner – Caribbean Night - *Ponce de Leon Lawn* Casual 6:30 – 7:30 PM Reception 7:30 – 10:00 PM Dinner

Friday, November 16

7:00 – 11:00 AM	Spouse & Guest Breakfast – *Magnolia Room* 7:00 – 9:00 AM Buffet 9:00 – 11:00 AM Coffee & Danish
9:00 – 11:00 AM	Book Review – *Magnolia Room*
12:30 – 1:30 PM	**Presidential Address** – "Reflections on Latin America," by Roberto C. Heros, M.D. – *Ponce de Leon II*
1:00 – 5:00 PM	Shopping – Trolley Leaves for Worth Avenue
2:00 – 4:30 PM	*Norton Museum – *Bus transportation provided*
7:00 – 8:00 PM	**President's Reception** – *Mediterranean Ballroom*
8:00 – 11:00 PM	Black Tie Dinner – *Circle Dining Room*

Saturday, November 17

7:00 – 11:00 AM	Spouse & Guest Breakfast – *Mediterannean Room* 7:00 – 9:00 AM Buffet 9:00 – 11:00 AM Coffee & Danish

*Tennis, golf, and fishing (transportation to boat not provided) are also available on Thursday and Friday.

* Activities require prior registration.

DISCLOSURE INFORMATION

The American Association of Neurological Surgeons and *The American Academy of Neurological Surgery* control the content and production of this CME activity and attempt to assure the presentation of balanced, objective information. In accordance with the Standards for Commercial Support established by the Accreditation Council for Continuing Medical Education, speakers and paper presenters are asked to disclose any relationship they or their co-authors have with commercial companies which may be related to the content of their lecture.

Speakers and paper presenters/authors who have disclosed a relationship* with commercial companies whose products may have a relevance to their presentation are listed below.

Faculty Name	Disclosure	Type of Relationship
Adler, J.	Accuray, Inc.	Stock Shareholder
Berger, Mitchel S.	Medtronic	Consultant
	NIH	Grants/Research Support
Canute, Gregory W.	Inclone Systems, Inc.	Stock Shareholder
Chang, S.	Accuray, Inc.	Stock Shareholder
Chen, Peng	Boston Life Science, Inc.	Grants/Research Support
Donaldson, Jill W.	Indiana University	Spinal Cord & Head Injury Res. Grant
Fults, Dan	Ped. Brain Tumor Found.	Grant
Goldman, David E.	Regneron Pharmaceuticals	Collaborative Research Agreement
Gunel, Murat	NIH	Grants/Research Support
Hamilton, Allan J.	Guilford/Aventis	Grants/Research Support
	Guilford/Aventis/ Cook, Inc./Medtronic	Consultant
	Guilford/Aventis	Honorarium
Kondziolka, D.	Elekta Instruments, Inc.	Consultant
Lundsford, L. Dade	Elekta, Inc.	Consultant/Stock Shareholder
Macdonald, R. Loch	NIH/Amer. Heart Assoc.	Grants/Research Support
Markert, James M.	NIH	Grants/Research Support

Newell, David W.	NIH	Grants/Research Support
Park, T.S.	NIH	Grants/Research Support
Spetzler, Robert F.	Zeiss/Medtronics/NMT/ Synergetics/Allegiance	Consultant
Taylor, Michael	Neurosurgery Research and Education Foundation	Grants/Research Support

*Relationship refers to receipt of royalties, consultantship, funding by research grant, receiving honoraria for educational services elsewhere, or any other relationship to a commercial company that provides sufficient reason for disclosure.

Speakers and their paper presenters/authors who have reported they do not have any relationships with commercial companies:

Faculty Name
Bailes, Julian E.
Barbaro, Nicholas M.
Barrow, Daniel
Baskaya, Mustafa K.
Brock, Mario
Burchiel, Kim
Dempsey, Robert
Foley, Kevin T.
Foltz, Eldon L.
Grubb, Robert L.
Gunel, Murat
Hadley, Mark N.
Harbaugh, Robert E.
Harkey, H. Louis
Harsh, Griff
Heileman, Carl B.
Meyer, Fredric
Morcos, Jacques
McComb, J. Gordon
McCormick, Paul C.
Newell, David W.
Pickard, John D.
Rosenow, Joshua
Schindler, Jay
Solomon, Robert

Sonntag, Volker
Tatter, Stephen B.
Traynelis, Vincent
Wharen, Robert E.
Wilson, Charles B.

Speakers and their paper presenters/authors who have refused to disclose whether they have any relationships with commercial companies:

None

Speakers who have disclosed that their presentations will include discussion of an off-label or investigational drug or device:

Faculty Name

Canute, Gregory W.
Hamilton, Allan J.
Markert, James M.
Meyer, Fredric

SCIENTIFIC PROGRAM
AMERICAN ACADEMY
OF NEUROLOGICAL SURGERY
2001 LEARNING OBJECTIVES

Jointly sponsored by The American Association of Neurological Surgeons November 14-17, 2001.

Upon completion of this program, the participants should 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.

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This activity was planned and implemented in accordance with the Essential Areas and policies of the Accreditation Council for Continuing Medical Education through 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 13.5 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, NOVEMBER 15 **Moderator: Robert F. Spetzler**

8:00 - 8:45 AM	POINT COUNTERPOINT: Is there a Time to Retire? Charles B. Wilson and Bennett M. Stein
8:45 - 9:00 AM	Intrathecal Thrombolysis for Aneurysmal Intraventricular Hemorrhage. Julian E. Bailes, MB Medary
9:00 - 9:15 AM	Intraoperative Angiography for Aneurysm Surgery: A Prospective Evaluation of Efficacy and Cost Benefit Analysis. Daniel L. Barrow, G Tang, M Conley, J Dion
9:15 - 9:30 AM	Unruptured Aneurysm: Changing Attitude as a Result of New Data and New Technology. Mustafa K. Baskaya, S Azhari, RC Heros
9:30 - 9:45 AM	Carotid Occlusion Surgery Study (COSS). Preview of a New EC/IC Arterial Bypass Trial. Robert L. Grubb, Jr., WJ Powers, HP Adams, Jr., WR Clarke, RF Woolson, TO Videen, CP Derdeyn
9:45 - 10:00 AM	KRIT1, the CCM1 Protein, is Important in Endothelial Capillary-like Tube Formation During *In Vitro* Angiogenesis. Murat Gunel, M Diluna, D Shin, RP Lifton
10:00 - 10:15 AM	Cavernous Malformation Radiosurgery: The Proof for Patients with Multiple Hemorrhages and the Potential for Patients with One Bleed. J McInerney, Douglas Kondziolka, LD Lunsford
10:15 - 10:30 AM	Metaanalysis of and Effect on Vasospasm of

	Tirilazad Derived from the Tirilazad Database on Aneurysmal Subarachnoid Hemorrhage. R. Loch Macdonald
10:30 - 11:00 AM	Coffee Break
11:00 - 11:15 AM	Modulation of NMDA Receptor Activity during Ischemic Neuronal Damage. David W. Newell, A Emmi
11:15 - 11:30 AM	Matrix Metalloproteinase-9 Mediates Blood-Brain Barrier Breakdown Following Transient Focal Cerebral Ischemia in Mice. TS Park, JM Gidday
11:30 - 11:45 AM	Quantification of Cerebral Ischemia Following Head Injury. DK Menon, JP Coles, TM Fryer, MR Coleman, JN Skepper, JC Matthews, P Smielewski, PS Minhas, DA Chatfield, SPMJ Downey, AK Gupta, F Aigbirihio, EJ Williams, GB Williams, PJ Hutchinson, D Day, IV Kendall, S Boniface, TA Carpenter, JC Clark, John D. Pickard
11:45 - 12:00 PM	Evidence that an Intact Immune System is Neuroprotective in Selective Hippocampal Neuronal Injury. JJ Schindler, RE Anderson, FB Meyer
12:00 - 12:15 PM	Criteria for Primary Endovascular Treatment of Posterior Circulation Aneurysms: Analysis of a Surgical Series. Robert A Solomon, ES Connolly, Jr.
12:15 - 12:30 PM	Medical Evidence-Based Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injuries. Mark N. Hadley, BC Walters, PA Grabb, NM Oyesiku, GJ Przybylski, DK Resnick, TC Ryken
12:30 - 12:45 PM	Floating Lumbar Fusion. WC Huag, RW

Porter, P Detwiler, Volker K.H. Sonntag

12:45 - 1:00 PM — Randomized Controlled Trials: How Tarnished is the Gold Standard? Robert E. Harbaugh

FRIDAY, NOVEMBER 16 **Moderator: Jon Robertson**

8:00 - 8:45 AM — POINT COUNTERPOINT: Subspecialization – Is the Generalist Dead? Arthur Day and Dennis Spencer

8:45 - 9:00 AM — Modulation of Zero-Magnesium Induced Epileptiform Activity in Human Neocortical Slices. MD Smyth, SC Baraban, Nicholas M. Barbaro

9:00 - 9:15 AM — The Impact of Consumerism on Surgical Specialties. Charles B. Wilson

9:15 - 9:30 AM — Selective Microsurgical Amygdalohippocampectomy for Medically Intractable Temporal Lobe Epilepsy. Kim Burchiel, D Spencer, M Salinsky

9:30 - 9:45 AM — The Activity of Anti-EGFR Monoclonal Antibody C225 Against Glioblastoma Multiform. J Eller, S Longe, Gregory W. Canute

9:45 - 10:00 AM — Glial Cell Line-Derived Neurotrophic Factor Protects Hippocampal Neurons after Traumatic Brain Injury in Rats. BT Kim, VLR Rao, KA Sailor, KK Bowen, RJ Dempsey

10:00 - 10:15 AM — MYC Expression Promotes the Proliferation of Neural Progenitor Cells in Culture and *In Vivo*. Dan Fults, C Pedone, C Dai, E Holland

10:15 - 10:30 AM	A Phase III Randomized Double-Blind Placebo-Controlled "Frontline" Clinical Trial of Intracavitary Carmustine (Gliadel) for the Treatment of Malignant Gliomas. M Westphal, E Bortey, D Hilt, Allan Hamilton
10:30 - 11:00 AM	Coffee Break
11:00 - 11:15 AM	Proton Beam and Cyberknife Stereotactic Radiosurgery of Vestibular Schwannomas. GR Harsh, SD Chang, JR Adler, JS Loeffler
11:15 - 11:30 AM	Multimodality Management of Vestibular Schwannomas. CB Heilman, N Blevins, D Poe, D Vernick, J Border
11:30 - 11:45 AM	Acoustic Neuroma Radiosurgery: A Benchmark to Compare Against other Management Modalities. LD Lunsford, D Kondziolka, D Bissonette, JC Flickinger
11:45 - 12:00 PM	Genetically Engineered Herpes Simplex Viruses in the Treatment of Glioma. James M. Markert, Y Gillespie, M Medlock, R Martuza
12:00 - 12:15 PM	***ACADEMY AWARD PAPER*** Inosine Induces Extensive Anatomical Reorganization and Improves Functional Outcome after Cortical Stroke. Peng Chen, David E Goldberg, Larry I. Benowitz
12:15 - 12:30 PM	***ACADEMY AWARD HONORABLE MENTION*** Germline and Somatic Mutations of Suppressor of Fused Predispose to Medulloblastoma Through Failure to Suppress Sonic Hedgehog and Wnt Signaling. Michael D. Taylor, James T. Rutka

12:30 - 1:30 PM	**PRESIDENTIAL ADDRESS** Reflections on Latin America Roberto C. Heros

SATURDAY, NOVEMBER 17 **Moderator: Mitchel S. Berger**

8:00 - 8:45 AM	POINT COUNTERPOINT: Should Endovascular Neurosurgery Fall Within the Department of Neurosurgery? LN Hopkins and Alex Berenstein
8:45 - 9:00 AM	Experimental Treatment of Guinea Pig Sciatic Nerve Injury with Topical Polyethylene Glycol. Jill W. Donaldson, R Shi, R Borgens, SA Shapiro
9:00 - 9:15 AM	Biomechanical Advantage of a Translational Anterior Cervical Plate. Kevin T. Foley, DJ DiAngelo, W Liu, K Olney, L Davidson
9:15 - 9:30 AM	Science for Free? Mario Brock
9:30 - 9:45 AM	Role of RhoA in Experimental Spinal Cord Injury in Rat. JK Sung, L Miao, JW Clavert, HL Harkey, JH Zhang
9:45 - 10:00 AM	Preliminary Results of AANS/CNS Joint Spine Section Pilot Study on Lumbar Disc Herniation Utilizing Internet based Data Collection and Management. Paul C. McCormick
10:00 - 10:15 AM	CSF Pulsatility Analysis: A preliminary Accurate Diagnostic for NPH and Obstructive Hydrocephalus. Eldon L. Foltz
10:15 - 10:30 PM	Multimedia Database. RF Spetzler, S Partovi, J Henn, M Ferreira

10:30 - 11:00 AM	Coffee Break
11:00 - 11:15 AM	Addition of Elemental Iodine to Surgical Irrigation for Shunt Infection Prophylaxis. SH Choi, JG McComb, ML Levy, I Gonzalez, R Bayston
11:15 - 11:30 AM	Neurosurgical Applications of High Resolution Thermal Imaging. FB Meyer, S Goerss, B Kall
11:30 - 11:45 AM	Combined Surgical Approaches through the Temporal Bone: Surgical Anatomy, Pitfalls and Complications. Lessons Learned in a Series of 29 Patients. JJ Morcos, MK Baskaya, IA Abumeri, E Coscarella
11:45 - 12:00 PM	Adenoviral Induction of NG2+ Neural Precursors in the Corpus Callosum. Joshua Rosenow, E Chmielnicki, A Benraiss, SA Goldman
12:00 - 12:15 PM	Diffuse Multilobar Infiltrating Glial Tumors: A Modern Day Account of 22 Cases. Mitchel S. Berger
12:15 - 12:30 PM	An inflatable Balloon Catheter and Liquid I-125 Radiation Source for Treatment of Recurrent Malignant Glioma: The Gliasite Radiation Therapy System. Stephen B. Tatter, CL Branch, EG Shaw, ML Rosenblum, T Mikkelsen, J Weingart, A Olivi, H Brem, JJ Olson, S Brem, DA Vollmer
12:30 - 12:45 PM	Opposite Effects of Cis-Parinaric Acid in Activities of P-38 MAP and c-Jun N-Terminal Kinases in Malignant Rat Astrocytoma Cells. Vincent C. Traynelis, A Zaheer, SK Sahu

12:45 - 1:00 PM

Neurostimulation for Tremor: Functional and Neuropsychological Results. Robert E. Wharen, RJ Uitti, RJ Witte, JA Lucas, A Obwegeser, EG Holker, MF Turk

NOTES:

THURSDAY, NOVEMBER 15 **8:45 - 9:00 AM**

Intrathecal Thrombolysis for Aneurysmal Intraventricular Hemorrhage

Julian E. Bailes, M.D., Max B. Medary, M.D.

Since our initial interest over a decade ago in an aggressive management scheme for poor-grade aneurysm patients, we have continued to be impressed that a certain number of patients will exhibit signs of brainstem dysfunction, yet still be salvageable with intensive therapy. Chief among those are patients presenting with IVH, especially ominous when there is packed IVH, with the ventricular system full of clotted blood, often with dilatation. IVH including the fourth ventricle with dilatation has been shown to be usually untreatable and fatal.

We have noted that some patients presenting in poor-grade condition following aneurysmal subarachnoid hemorrhage (SAH) who exhibit signs of brainstem dysfunction may remain salvageable with intensive therapy. We have utilized the instillation of intrathecal rTPA to effect thrombolysis of intraventricular hemorrhage (IVH) and restore normal cerebrospinal fluid (CSF) circulation dynamics in a series of four poor-grade aneurysmal SAH patients. Following craniotomy for aneurysm clipping, 4 mg divided twice daily is given by intrathecal administration until the IVH is radiographically resolved. All patients survived, with three experiencing a good and one an excellent outcome. This treatment appears to be effective, easy to administer, well tolerated, without deleterious systemic effects on the coagulation system, and allows for continued effective neurosurgical management after securing a ruptured intracranial aneurysm in patients with significant IVH.

Intraoperative Angiography for Aneurysm Surgery: A Prospective Evaluation of Efficacy and Cost Benefit Analysis

Daniel L. Barrow, Gordon Tang, Michael Conley, Jacques Dion

Indications for intraoperative angiography (IOA) during aneurysm surgery remain unclear. To better define its use, we report the results of a prospective study in which intraoperative angiography was used in 517 consecutive patients undergoing surgery for intracranial aneurysms irrespective of location, size or complexity. Additionally, we have performed a cost benefit analysis of intraoperative angiography.

Sixty-four of 517 aneurysms (12.4%) demonstrated IOA findings leading to revision of surgical treatment. Residual aneurysm (52%) was the most frequent finding leading to clip revision. In 45% of cases, IOA disclosed vessel compromise. Aneurysms of the proximal internal carotid artery (ICA) were the most frequently altered with the superior hypophyseal and clinoidal locations having the highest revision rates, 40% (8/20) and 44% (8/18) respectively. Aneurysm size predicted need for revision. Giant aneurysms underwent revision in 29% (9/31) of cases, while aneurysms 15 to 25mm were revised in 22% (12/54) of cases. In a multivariate logistic regression model, factors related to increased revision rates included the superior hypophyseal and clinoidal locations as well as giant and large size. Ninety-five patients underwent both intraoperative and postoperative angiography. Five discrepancies were notes (95% accuracy). Four were flow-related and one demonstrated previously unrecognized residual aneurysm. Complications attributable to IOA occurred in 0.4% of cases. Cost benefit analysis demonstrated substantial benefit to the routine use of intraoperative angiography. Low complication rates, high accuracy, unexpected readjustments and significant cost benefit favor a more indiscriminate use of IOA.

Unruptured Aneurysms: Changing Attitude as a Result of new Data and new Technology

Mustafa K. Baskaya, M.D., Shirzad Azhari, M.D., Roberto C. Heros, M.D.

Department of Neurosurgery, University of Miami, FL

During the first half of his career, the senior author had an aggressive policy of recommending microsurgical clipping of most unruptured aneurysms (UAs) provided the patient was relatively healthy and not elderly. The main factors that have affected our current approach are the development of endovascular treatment alternatives, new knowledge about the natural history of UAs and an introspective review of our surgical results, which revealed significant morbidity with clipping of basilar tip aneurysms. One notable change in practice is a more "conservative" surgical approach that has resulted in abandonment of the attempt to clip the UA in 15 (6%) of a group of 236 patients with UAs operated from July 1995 to December 2000. The findings at surgery that led to the decision not to clip will be discussed and some of the cases will be illustrated.

Our current approach to patients with UAs will be discussed. In general: 1) We recommend no treatment, except for periodic follow up of UAs less than 5 mm. 2) UAs between 5 and 10 mm are considered for treatment depending on multiple factors, of which age of the patient and risk of the treatment are the most important. 3) All UAs more than 10 mm are considered for treatment unless the patient is elderly or in poor medical condition. 4) The decision of whether to treat surgically or endovascularly is made by the neurosurgeon after appropriate consultation with endovascular colleagues and is very influenced by the configuration of the UA; but under equal circumstances, we select as first treatment endovascular occlusion for basilar tip aneurysms and clipping for aneurysms in other locations. 5) Treatment approach, whether to clip or to occlude endovascularly, is generally conservative with readiness to "back off" if any difficulties are encountered.

Carotid Occlusion Surgery Study (COSS). Preview of a New EC/IC Arterial Bypass Trial.

Robert L. Grubb, M.D., William J. Powers, M.D., Harold P. Adams, M.D., William R. Clarke, Ph.D, Robert F. Spetzler, M.D. Woolson, Ph.D., Rom O. Videen, Ph.D., Colin P. Derdeyn, M.D.

Ipsilateral increased oxygen extraction fraction (OEF) measured by positron emission tomography (PET) is a powerful independent risk factor for subsequent stroke in patients with symptomatic carotid occlusion. The risk for ipsilateral ischemic stroke at two years was 5.3% in 42 patients with normal OEF and 26.5% in 39 patients with increased OEF in the St. Louis Carotid Occlusion Study. In patients with hemispheric symptoms within 120 days, the two year ipsilateral stroke rates were 12% in 27 patients with normal OEF and 50% in 18 patients with increased OEF. Previous PET studies have demonstrated that superficial temporal artery – middle cerebral artery (STA-MCA) anastomosis can restore OEF to normal. We will test the hypothesis that STA-MCA anastomosis when added to best medical therapy can reduce subsequent ipsilateral ischemic stroke by 40% at two years in patients with recent ($\leq$ 120 days) symptomatic internal carotid artery occlusion and increased OEF. Clinically eligible patients will have a PET measurement of OEF and only patients with increased OEF will be randomized to STA-MCA bypass surgery or medical management. The primary endpoint will be all stroke and death from randomization until 30 days post-operatively (with an equivalent period in the non-surgical group) plus subsequent ipsilateral ischemic stroke within two years. It is estimated that 186 patients in each treatment group will provide 90% power to detect a treatment difference. Assuming 40% of PET scans will demonstrate increased OEF, this will require enrolling 930 clinically eligible subjects. There are 25 participating clinical centers in the trial.

KRIT1, The CCM1 Protein, Is Important in Endothelial Capillary-like Tube Formation During *In Vitro* Angiogenesis

Murat Gunel, M.D., Michael Diluna, Dana Shin, Richard P. Lifton, M.D., Ph.D.

Yale University School of Medicine, Department of Neurovascular Surgery

Mutations in KRIT1 (Krevl Interaction Trapped gene 1) gene have been shown to cause cerebral cavernous malformation, a disease featuring malformation of cerebral capillaries resulting in cerebral hemorrhage and leading to strokes and seizure disorders. The normal function of KRIT1 and the mechanism through which its mutation causes CCM remain unknown. This gene was initially identified and cloned in yeast two-hybrid screen that was intended to study Krevl/rap1A gene, a small GTPase with significant sequence homology to Ras. The histology of cavernous malformations suggests that the KRIT1 may play a central role in normal vascular development or maintenance of vascular integrity. As CCMs contain abnormally dilated channels and consist only of endothelial cells without any supporting smooth muscle cells, it is conceivable that KRIT1 is involved in the formation of normal capillaries and/or communication between endothelial and mesenchymal cell compartments. In order to address this question, we have investigated KRIT1 expression in endothelial cell lines using RT-PCR and polyclonal anti-KRIT1 antibodies. Our results demonstrate that endothelial cells express KRIT1 and as these cells reach confluence and start forming capillary-like tubes, KRIT1 expression significantly increases. This temporal induction of KRIT1 expression with endothelial tube formation, coupled with the phenotype of CCM suggest that KRIT1 is important in determination of cell shape leading to the formation and/or stabilization of vascular channels formed by endothelial cells in vitro.

Cavernous Malformation Radiosurgery: The Proof for Patients with Multiple Hemorrhages and the Potential for Patients with One Bleed

James McInerney, M.D., Douglas Kondziolka, M.D., L. Dade Lunsford, M.D.

Introduction: This study examines 1) the long-term hemorrhage rate after radiosurgery of multi-hemorrhagic, high-risk cavernous malformations (CM); and 2) uses a decision and cost-benefit analysis for patients with one symptomatic bleed.

Methods: Eighty-two symptomatic CM patients had Gamma knife radiosurgery between 1987 and 2000. The baseline risk of symptomatic hemorrhage (4.5%) from untreated CM, the latency period (2 years), the bleed risk post-latency (0.76%) and the risk of major (2.4%) and minor (11%) adverse radiation effects were determined. We assumed the risk of hemorrhage during the latency equaled baseline risk. Costs were set as follows from prior cerebrovascular studies: hemorrhage $5000, radiosurgery $8,000, major neurological deficit $35,800 per year, minor neurological deficit $9500 per year. Utilities were set as follows: death 0, major neurological deficit 0.39, minor neurological deficit 0.95, and well 0.95. We applied a 3-month disutility for minor neurological deficits and hemorrhages and a 3% discount rate. We then constructed a decision analysis model using this data.

Results: Observation prior to radiosurgery averaged 4.33 years (range 2-216 months) for a total of 354 patient years. During this period, 195 hemorrhages were observed for an annual hemorrhage rate of 31.6%, excluding the first hemorrhage. After radiosurgery, patient follow-up averaged 5 years (range, 0.6-12 yrs). During this period, 18 hemorrhages were identified, 16 in the first two years post radiosurgery and two after two years. The annual hemorrhage rate was 11.7% per year for years 0-2 and 0.80% per year from years 2-12. No major adverse radiation effects occurred after 1992. In analysis, the radiosurgery cohort acquired 29.09 quality adjusted life years (QALYs) per patient or $4,600 per QALY. The natural history cohort acquired 25.36 QALYs per patient or $7,000 per QALY. This relationship persisted through a full range of sensitivity analyses.

Conclusion: Radiosurgery confers a dramatic reduction in the risk of hemorrhage for high-risk cavernous malformations, especially after two years (thirty-fold). This decision analysis model suggests that radiosurgery offers a cost-effective benefit to patients harboring cavernous malformations after a single symptomatic hemorrhage.

THURSDAY, NOVEMBER 15 **10:15 – 10:30 AM**

Meatanalysis of and Effect on Vasospasm of Tirilazad Derived from the Tirilazad Database on Aneurysmal Subarachnoid Hemorrhage

R. Loch Macdonald, M.D., Ph.D.

Section of Neurosurgery, Department of Surgery, University of Chicago Medical Center and Pritzker School of Medicine

Data collected on 3449 patients entered into randomized, blinded studies of tirilazad were analyzed to determine by metaanalysis whether tirilazad affects outcome after subarachnoid hemorrhage (SAH) and determine the effect of tirilazad on vasospasm. Uni- and multivariate analyses were used to create logistic regression and proportional hazards models using outcome 3 months after SAH classified by the Glasgow outcome score and using a clinical/radiological definition of vasospasm.

Metaanalysis showed that tirilazad had no significant effect on outcome. Tirilazad use was associated with a significantly less likelihood of use of Atriple-H therapy@ and with significantly less vasospasm although there was no effect on the incidence of cerebral infarction at 3 months. There was significant heterogeneity in outcome between studies that could not be explained by known prognostic factors or by anticonvulsant use. Multivariate logistic regression showed that vasospasm was associated with younger age, female gender, thicker clot on computed tomography, worse neurological grade, larger aneurysm, intraventricular hemorrhage, preexisting hypertension, use of prophylactic induced hypertension and Dilantin use.

In summary, tirilazad had no effect on outcome assessed 3 months after SAH. The decreased use of Atriple-H therapy@ in treated patients is intriguing but cannot be definitely inferred to be due to a beneficial effect of tirilazad. The effect of Dilantin warrants further study.

Modulation of NMDA Receptor Activity During Ischemic Neuronal Damage

David W. Newell, M.D., Adriana Emmi, M.D., Ph.D.

The predominant mechanisms which cause ischemic neuronal injury in clinical situations can differ depending on the insult. We have found that the presence of extracellular glucose above 60 mg % prevents NMDA receptor blockade from providing protection against CA1 neuronal damage caused by anoxic injury (severe oxygen deprivation) in organotypic hippocampal slice cultures (control = 77.2% +/- 10.6, treated 74.8 +/- 6.3 % p =0.8 at 100mg % glucose.) Damage caused by combined oxygen/glucose deprivation however, is reduced by NMDA receptor blockade (control = 70.1% +/- 9.7, treated = 14.1 +/- 6.8 % p<.001 at 0% glucose). We also examined the effect of glucose on ischemia-induced neuronal hyperexcitability. During *in-vitro* anoxia (glucose present) slightly decreased (-10 to -20%) population spike amplitude (PSA) was observed in CA1 and CA3; in contrast, the same anoxic insult, but without glucose, increased PSA by 10 and 30% in CA1 and CA3 respectively. During anoxia-aglycemia, afterdischarge activity and spontaneous bursting spreading from CA3 to CA1 was frequently observed; however neuronal hyperexcitability did not occur during anoxia with glucose present.

Modulation of NMDA receptor activity and hyperexcitability by glucose probably through a pH effect, may prove to be a very important mechanism which could explain the variable protective effect of NMDA antagonists observed in brain ischemia. Neuroprotective compounds which act through blockade of NMDA receptors may therefore not be effective in all clinical settings depending on the nature and severity of the insult.

Matrix Metalloproteinase-9 Mediates Blood-Brain Barrier Breakdown Following Transient Focal Cerebral Ischemia in Mice

T.S. Park, M.D., Jeffrey M. Gidday, Ph.D.

Department of Neurosurgery, St. Louis Children's Hospital, Washington University, St. Louis, MO

Results of recent studies suggest a causal relationship between cerebral production of matrix metalloproteinase-9 (MMP-9) and blood-brain barrier (BBB) breakdown following focal stroke, although conclusive evidence is still lacking. In the present study, we used MMP-9 knockout mice and pharmacologic inhibition of MMPs to test this hypothesis. Mice were subjected to transient focal cerebral ischemia by occluding the middle cerebral artery for 2 h with an intraluminal filament. Vasogenic edema was assessed at 8 h of reperfusion by Evan's blue extravasation and MMP-9 activity by zymography. The resultant volumes of infarction were determined at 24 h. Pro- and active MMP-9 levels were upregulated at 8 h of reperfusion in wild-type animals; no compensatory upregulation of MMP-2 was noted in the knockouts. Vasogenic edema was reduced to a similar extent in MMP-9 knockout mice and in normal mice treated with a hydroxamate-based, nonselective MMP inhibitor. The reduction in vasogenic edema was associated with comparable and significant neuroprotection in both the MMP-9 knockouts and the MMP inhibitor-treated mice. Conversely, MMP-9 knockout mice were not protected against brain injury in permanent focal stroke. These results implicate activated MMP-9 in the loss of BBB integrity during early reperfusion following focal stroke, secondary to degradation of the basal lamina and extracellular matrix. The reperfusion-dependent protection in the knockout mice is consistent with the production of MMP-9 by infiltrating neutrophils. Prevention of MMP-9–mediated proteolysis could serve as a therapeutic approach to reduce vasogenic edema and secondary brain injury following focal cerebral ischemia-reperfusion.

Quantification of Cerebral Ischaemia following Head Injury

David K. Menon, Jonathan P. Coles, Tim D. Fryer, Martin R. Coleman, Jeremy N, Skeeper, Julian C. Matthews, Piotr Smieleweski, Pawan S. Minhas, Doris A. Chatfield, Stephen PMJ Downey, Arun K. Gupta, Franklin Aigbirihio, Emma J. Williams, Guy B. Williams, Peter J. Hutchinson, Diana Day, Iona V. Kendall, Simon Boniface, T. Adrian Carpenter, John C. Clark, John D. Pickard

Despite the many studies that demonstrate early reductions in cerebral blood flow and post-mortem evidence of cerebral infarction, there has been recent debate over the interpretation of positron emission tomography studies as to when and where in the brain cerebral ischaemia occurs following head injury. It is difficult to define critical ischaemia in head injury using cerebral blood flow and cerebral oxygen consumption thresholds since both the head injury itself and concurrent sedation may reduce oxygen consumption and hence coupled-perfusion in the injured brain.

We have used a combination of triple ^{15}O PET to determine regional cerebral blood volume, oxygen consumption and oxygen extraction fraction, neurophysiology and brain tissue pO_2 measurements to investigate the incidence and mechanisms of tissue hypoxia in twenty-six patients within 7 days of head injury. Region of interest and voxel-based analyses have been complemented by statistical analysis of OEF histograms.

Significant ischaemia is common within the first 24 hours following head injury and may be observed later, particularly in the presence of hypocapnia. However, the effects of hyperventilation were not restricted to the cerebrovascular effects of hypocapnia but also included effects on regional oxygen consumption in some patients.

Evidence that an Intact Immune System is Neuroprotective in Selective Hippocampal Neuronal Injury

Jay Schindler, R. E. Anderson, F. B. Meyer

Background. Inflammation is thought to be deleterious following cerebral ischemia. Many laboratories, investigating specific immune parameters including interleukins, adhesion molecules, and complement pathways, have proposed that the immune system exacerbates pannecrosis and cerebral infarction volume. In this study, we investigated the differences in selective, hippocampal neuronal vulnerability between immune-normal and immune-compromised mice.

Methods. A murine model of reproducible forebrain ischemia was first developed to examine hippocampal ischemia vulnerability. Severe combined immune-deficient (SCID) mice are deficient in functional T- and B- lymphocytes. Thirty-four animals were divided into two groups comprising 17 immune-normal Balb/c and 17 SCID Balb/c mice. Experimental animals were subjected to 15 minutes of temporary bilateral common carotid artery occlusion. Appropriate non-ischemic controls were used. Mice were sacrificed three days post-ischemia and neuronal cell death was measured. Systemic parameters including depth of anesthesia, core body temperature, arterial blood gasses, and duration of ischemia were carefully controlled.

Results. The percentage of neuronal cell death was significantly increased ($p<0.0001$) in the immune-compromised animals (56.7 •••• % Balb/c vs. 93.6 •••• % SCID Balb/c; mean •• SD).

Conclusions. This study supports the hypothesis that an intact immune system, replete with functional T- and B- lymphocytes, is neuroprotective in attenuating selective hippocampal ischemic neuronal injury. This data raises the intriguing question as to whether enhancement of certain immune factors might be neuroprotective.

Criteria for Primary Endovascular Treatment of Posterior Circulation Aneurysms: Analysis of a Surgical Series

Robert A. Solomon, M.D., E. Sander Connolly, Jr., M.D.

Columbia University College of Physicians and Surgeons, New York, NY

Endovascular techniques offer the surgeon a viable alternative to surgical treatment of difficult aneurysms. We sought to identify a subset of patients with posterior circulation aneurysms that had successful surgical clipping with surgical morbidity rates comparable to the best available data on endovascular treatment of cerebral aneurysms.

Literature review suggests that endovascular treatment can be completed without significant neurological deficit in 82% of aneurysm patients. Review of a prospectively collected surgical aneurysm database (single surgeon - RAS) revealed 181 operations for posterior circulation aneurysms. 91% of aneurysms were completely clipped and 69% of patients had an excellent outcome (no complication or deficit other than temporary third nerve palsy). If one excludes patients with giant aneurysms (n=35), patients over the age of 70 (n=8), and H&H grade IV/V patients (n=7), 95% of aneurysms were completely clipped and excellent outcome was achieved in 81%. An additional 7% suffered minor deficits but were able to function independently. Only 6% of patients were dead or severely disabled.

Until randomized data exists, information such as contained in this study will help guide patient management. Since the long term results of clipping appear superior to coiling, clipping should be considered when appropriately safe. These data suggest that satisfactory clipping and outcomes comparable to immediate endovascular results can be realized in patients with non-giant posterior circulation aneurysms, under the age of 70, in H&H grades 0 to 3. Poor grade patients, patients with giant aneurysms, and patients over 70 might appropriately be referred for primary endovascular treatments.

Medical Evidence-Based Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injuries

Mark N. Hadley, M.D., Beverly C. Walters, M.D., Paul A. Grabb, M.D., Nelson M. Oyesiku, M.D., Greg J. Przybylski, M.D., Daniel K. Resnick, M.D., Timothy C. Ryken, M.D.

Medical evidence-based guidelines serve patients and physicians by providing succinct management recommendations on specific medical issues derived from peer-reviewed studies and experiences of clinician-investigators. Properly generated guidelines serve as a resource of treatment options for a given disease entity, and also identify what is yet unknown about that entity, highlighting the need for further investigation.

The AANS/CNS Spine Section set out to generate guidelines for the management of patients with acute cervical spine and cervical spinal cord injuries. Twenty-three topics were identified and over six-dozen critical questions were developed. Committee members immersed themselves in the world literature and reviewed hundreds of articles on a given topic to determine the potential relevance of each to the specific issue. Manuscripts germane to the issue were categorized according to the weight of medical evidence each provided and were incorporated into the scientific foundation of the individual guideline.

Twenty-three guidelines have been completed. Three address issues of immobilization, transport and neurological assessment. Two address the radiographic assessment of acute injury patients. Six address acute management issues of ICU care, blood pressure management, pharmacology, DVT, nutrition and closed reduction of cervical fractures. Two address pediatric spine injuries and SCIWORA. The final ten guidelines are on the management of specific injury types including AOD, condylar fractures, os odontoideum, atlas fractures, axis fractures, C1-C2 combination injuries, sub-axial vertebral fractures, facet dislocation injuries, central cord syndrome and vertebral artery injuries due to cervical fracture-dislocation. An overview of management recommendations will be presented including specific medically justified treatment standards, guidelines and options for each topic.

Floating Lumbar Fusion

W. C. Huang, M.D., Randall W. Porter, M.D., Paul Detwiler, M.D., Volker K. H. Sonntag, M.D.

Object: To determine the efficacy of a new modification of lumbar pedicle screw fixation, termed floating fixation.

Method: Thirty-eight adults (24 men, 14 women; mean age, 51.6 years; range, 31 to 75 years) with lumbar spinal instability or a previous failed back surgery were treated with floating lumbar pedicle screw fixation and reviewed retrospectively. When the configuration of patients' lumbar spine made it difficult to insert adjacent lumbar pedicle screws, one involved vertebral segment was skipped and the adjacent upper and lower levels were fixated instead. The fusion rate was 94.7% at a mean follow-up of 14.4 months. The hardware failed in only one patient who sustained a severe fall several months after surgery. At late follow-up, symptoms had resolved completely in 22 (59%) patients.

Conclusion: When patients with a hyperlordotic lumbar curvature, need pedicle screw fixation, this "floating pedicle screw" technique appears to be as efficacious as placing screws at sequential levels. Fusion rates, complication rates, and outcomes do not appear to be adversely affected by this technique.

Randomized Controlled Trials: How Tarnished is the Gold Standard

Robert E. Harbaugh, M.D.

Randomized controlled trials (RCTs) are viewed as the gold standard for determining the efficacy of medical and surgical treatment. The results of RCTs often supplant other data and have profound effects on practice. Although an indispensable tool for clinical investigation RCTs are not infallible. Because of their profound effects on practice, it is essential that the results of RCTs be interpreted with a thorough understanding of their potential limitations.

In this presentation I will introduce the rationale of Bayesian analysis for interpreting diagnostic test results and propose an analogy between the interpretation of diagnostic studies and the interpretation of the results of RCTs. A Bayesian analysis of the EC-IC bypass trial will be used to demonstrate the effects of patient selection on the results and predictive value of RCTs. This analysis suggests that lax patient selection criteria make the negative predictive value of the EC-IC bypass trial very low. Data from the North American Symptomatic Carotid Endarterectomy Trial (NASCET), the Asymptomatic Carotid Atherosclerosis Study (ACAS) and outcomes analyses of perioperative mortality following carotid endarterectomy using the Medicare database will demonstrate the potential for adverse public health effects from misapplication of RCT results. Other inherent problems with surgical RCTs will be delineated and methods to improve the reliability and applicability of clinical data collection will be suggested.

NOTES:

FRIDAY, NOVEMBER 16 **8:45 - 9:00 AM**

Modulation of Zero-Magnesium Induced Epileptiform Activity in Human Neocortical Slices

M.D. Smyth, S.C. Baraban, Nicholas M. Barbaro

Introduction: *In vitro* cortical slice techniques have long been used to investigate mechanisms of epileptogenesis in animal models of epilepsy, but little data is available confirming that epileptiform activity can be established and modulated in human neocortex. In this study, neocortex obtained from patients undergoing epilepsy surgery was used to analyze epileptiform activity and its response to standard anticonvulsants and novel agents.

Materials and Methods: Slices were prepared (450 μm) from tissue acquired from 6 patients undergoing epilepsy surgery (4 temporal, 2 frontal). Slices were perfused with zero-magnesium artificial CSF at 33.5 °C. Field recordings were used to monitor epileptiform activity. Responses to four agents were tested: Phenobarbital (100μM); baclofen (50μM); valproate (1mM); and carbenoxolone (100μM).

Results: Two types of epileptiform activity were seen: slow, seizure-like events with a sustained depolarization underlying multiple high frequency spikes lasting 20 to 100 seconds, occurring approximately 5 minutes apart (14/24 slices: 58%) and high frequency (1-2 Hz) bursting (10/24 slices: 42%). Baclofen consistently produced a complete block of all epileptiform activity (5/5 slices), and carbenoxolone produced a dramatic attenuation of activity (4/4 slices). By contrast, phenobarbital and valproate had almost no effect on either type of activity (4 and 5 slices, respectively). Nearly identical results were seen in rat neocortical slices.

Conclusions: The zero-magnesium model in the human neocortical slice preparation provides reproducible epileptiform activity similar to that seen in rat neocortex. In both models standard anticonvulsants have little effect, while a variety of other agents produce significant blockade of this activity. These data confirm the relevance of the zero-magnesium neocortical slice model to human epilepsy.

FRIDAY, NOVEMBER 16 9:00 – 9:15 AM

The Impact of Consumerism on Surgical Specialties

Charles B. Wilson, M.D.

By 2005, more than half of the nation's adults will qualify as "New Consumers"---college experience, discretionary income, and connected to the Internet. By then, they will have changed almost every aspect of health care as we have known it over the past quarter century: the signs are evident today and the direction is clear. They will demand, not request, accountability from every segment of health care industry. One component of this accountability will be transparency of medical practice including profiles of insurers, hospitals and health care professionals. For hospitals and physicians this transparency will translate into patient mix, volume and outcomes in a form that will be as readily accessible and understandable as Consumer Reports is today.

How will this change practice patterns? The trend that is gaining momentum incorporates concentration of resources, segmentation of surgical conditions, and an overarching focus on volume-driven excellence communicated through publicized outcomes. This will not mean the disappearance of general neurosurgeons, and the move toward concentration and "micro-specialization" will be evolutionary rather than revolutionary. This change, however, seems inevitable as well as major, and it will require reconfiguration of neurosurgical training and practice.

Selective Microsurgical Amygdalohippocampectomy for Medically Intractable Temporal Lobe Epilepsy

Kim Burchiel, David Spencer, Martin Salinsky

Departments of Neurological Surgery (KB) and Neurology (DS, MS), Oregon Health and Science University, Portland, OR

Introduction: The outcome of Selective Microsurgical Amygdalohippocampectomy (SMA) was analyzed in 41 consecutive patients (16-56 years old) with medically intractable partial complex seizures of medial temporal lobe origin.

Methods: A small temporal craniotomy was used to provide access to the middle temporal gyrus (T2), and then the tip of the temporal horn was entered transcortically using frameless stereotactic navigation. The uncus was resected, including most of the amygdala, and the hippocampus and parahippocampal gyrus were removed to the level of the colliculi on axial imaging as confirmed by frameless stereotactic localization.

Results: Thirty-five patients had mesial temporal sclerosis and hippocampal atrophy with (n=16) or without (n=19) high T2 signal on MRI. Engel Class I + II outcomes were achieved in 97% and 94% of patients at one and three years, respectively. There was only one instance of major neurological complication postoperatively (2% morbidity), and no mortalities. There were no instances of clinically apparent aphasia postoperatively.

Conclusion: Outcome following SMA for medically intractable Temporal Lobe Epilepsy compares favorably to outcome following standard anterior temporal lobectomy.

The Activity of Anti-EGFR Monoclonal Antibody C225 Against Glioblastoma Multiform

Jorge Eller, Sharon Longe, Gregory W. Canute

Inroduction: Overexpression of epidermal growth factor receptor (EGFR) in glioblastoma multiforme (GBM) secondary to EGFR gene amplification is associated with a more aggressive tumor phenotype and a worse clinical outcome. The purpose of this study was to determine whether blocking this receptor with the anti-EGFR chimeric monoclonal antibody C225 would decrease proliferation and increase apoptosis in GBM cells.

Methods: EGFR expression and amplification were determined for several human GBM cell lines. These GBM lines were then exposed to different concentrations of C225 for 48 hours, 72 hours and 7 days, after which time cytotoxicity, VEGF expression and apoptosis were assessed in vitro. An EGFR amplified human GBM was implanted in flanks of nude mice and the animals received C225 intraperitoneally for 5 weeks. Tumor volumes and survival were compared to sham treated mice.

Results: EGFR gene amplification was demonstrated in 3 of our primary GBM cell lines. C225 treatment produced significant cytotoxicity in all 3 EGFR-amplified GBM lines, but not in unamplified lines. Flow cytometry demonstrated increased apoptosis in C225-treated EGFR-amplified GBM lines, but not in unamplified lines. VEGF expression was also decreased in C225-treated EGFR-amplified GBM lines. C225-treated mice had 200% increase in survival plus a significant decrease in tumor volume.

Conclusions: Our findings demonstrated that blocking EGFR in GBM cells that overexpress this receptor significantly changes tumor cell biology by promoting apoptosis while decreasing proliferation and VEGF expression. The in vivo results suggest that this approach holds great promise for treatment of human GBMs.

Glial Cell Line-Derived Neurorophic Factor Protects Hippocampal Neurons after Traumatic Brain Injury in Rats

B. T. Kim, V. L. R. Rao, K. A. Sailor, K. K. Bowen, R. J. Dempsey

Objective: To study whether glial cell line-derived neurotrophic factor (GDNF) can protect the hippocampal neuronal death after traumatic brain injury (TBI).

Methods: Male Sprague-Dawley rats were subjected to moderate TBI with a controlled cortical impact device under halothane anesthesia and compared to Sham-operated rats. In 8 brain injured and 8 sham-operated rats, GDNF was infused continuously for 7 days (i.c.v., 200 ng/day at a rate of 8.35 ng/0.5 μl/hr) into the frontal horn of left lateral ventricle. An equal volume of vehicle (aCSF) was infused into 8 brain injured and 8 sham-operated rats. Seven days after the injury, all rats were sacrificed. Hippocampal neuronal loss was microscopically evaluated with cresyl violet in the CA2 and CA3 regions. A parallel set of sections from each brain was immunoreacted with antibodies against the astroglial marker GFAP. In the aCSF treated group, TBI resulted in a significant neuronal loss in the CA2 (by 60%, $p<0.05$) and CA3 (by 68%, $p<0.05$) regions compared to the sham-operated control. Compared to aCSF infused control, GDNF infusion significantly decreased the TBI-induced neuronal loss in both CA2 (by 58%, $p<0.05$) and CA3 (by 51%, $p<0.05$) regions. There is no difference in the GFAP positive astroglial cell number in the GDNF infused TBI and sham-operated groups, compared with their respective vehicle-treated groups.

Conclusions: GDNF infusion significantly decreased the TBI-induced hippocampal CA2 and CA3 neuronal death without altering the astrogliosis. This suggests that therapeutic strategies based on pharmacologic protection of neurons with TBI may be possible.

MYC Expression Promotes the Proliferation of Neural Progenitor Cells in Culture and In Vivo

Dan Fults, Carolyn Pedone, Chengkai Dai, Eric C. Holland

Department of Neurosurgery and Huntsman Cancer Institute, University of Utah School of Medicine (DF, CP), Salt Lake City, UT, and Departments of Cell Biology, Neurosurgery, and Neurology Memorial Sloan-Kettering Cancer Center (CD, EH), New York, NY

Primitive neuroectodermal tumors (PNETs) are pediatric brain tumors that result from defects in signaling molecules governing the growth and differentiation of neural progenitor cells. We used the RCAS-TVA system to study the growth effects on neural progenitor cells of three genetic alterations implicated in human PNETs: (1) overexpression of the cellular oncoprotein, MYC, (2) activation of transcription factor, β-catenin, and (3) haploinsufficiency of *Ptc*, the hedgehog receptor gene. The RCAS-TVA system utilizes an avian retroviral vector, RCAS, to target gene expression to specific cell types in transgenic mice. To express exogenous genes in neural progenitor cells we used *Ntv-a* mice. In these mice the *Nestin* gene promoter drives expression of TVA, the cell surface receptor for the virus. Ectopic expression of MYC, but not activated β-catenin, promoted the proliferation of neural progenitor cells in culture and in the cerebral leptomeninges *in vivo*. These effects were equally penetrant in mice with $Ptc^{+/-}$ and $Ptc^{+/+}$ genetic backgrounds. Although overexpression of MYC is not sufficient to cause intraparenchymal tumors, it may facilitate PNET formation by sustaining the growth of undifferentiated progenitor cells.

A Phase III Randomized Double-Blind Placebo-Controlled "Frontline" Clinical Trial of Intracavitary Carmustine (Gliadel) for the Treatment of Malignant Gliomas

Manfred Westphal, M.D., Enoch Bortey, Ph.D., Dana Hilt, M.D., Allan Hamilton, M.D.

Department of Neurosurgery, University Hospital Eppendorf, Hamburg, Germany; Division of Neurosurgery, Department of Surgery, University of Arizona Health Sciences Center, Tucson, AZ

A frontline evaluation of 240 newly diagnosed malignant brain tumors was undertaken to establish the post-surgical effectiveness of sustained release carmustine (Gliadel®) wafer. There was no statistically significant difference in tumor type or post-operative radiotherapy dose delivery between the experimental or placebo groups. There were 101 GBM patients in the experimental and 106 in the placebo arm. A median survival of 13.9 months in the Gliadel® group and 11.6 months in placebo was statistically significant (p=0.03). Treatment effect analysis yields significance with or without adjustment for Known Prognostic Factors p=0.02 and p=0.03 respectively. Median time deterioration was 11.9 for the experimental and 10.4 months in the placebo control group (p=0.05). In 10 of 11 Neuroperformance measures the Gliadel® arm maintained a statistically significant higher status. Visual status, however, remained same between the groups.

To eliminate re-operation effect thought to prolong patient status decline, subjects from either group were statistically censured from the study at re-operation. Under these conditions median survival was 14.8 months (Gliadel®) compared to 11.4 months for placebo control (p=0.01). Unlike the recurrent trial, the frontline study produced no statistical difference in wound healing, convulsions (number or time to onset) or infection rate between the groups. There was a documented increase in post-operative edema in the Gliadel® group and a slightly elevated rate of CSF leak 5% for the Gliadel® arm compared to 1% for the placebo control.

In summary, intracavitary chemotherapy (Gliadel® wafer) significantly improves median survival and sustains neurologic status longer than placebo in a randomized clinical trial and will prove a valuable addition to "frontline" surgical resection of malignant gliomas.

Proton Beam and Cyberknife Stereotactic Radiosurgery of Vestibular Schwannomas

G.R. Harsh, S.D. Chang, J.R. Adler, J.S. Loeffler

Department of Neurosurgery, Stanford Medical Center, Stanford, CA; Radiation Oncology Service, Massachusetts General Hospital, Boston, MA

Aim: High rates of permanent facial and trigeminal neuropathy (10% each) and of hearing loss (>40%) follow single fraction gamma/x-ray stereotactic radiosurgery of vestibular schwannomas with marginal doses of 16-20 Gy. This report compares the reduction of this morbidity achieved with two different strategies: proton beam radiosurgery with only 12 Gy and tri-fractionated Linac doses of 18-21 Gy.

Method: Sixty-eight tumors (mean volume of 2.49 cc) were treated with MGH's proton beam (from 1992 to 1998) with 12 Gy to the tumor margin (70 % IDL). Seventy-seven tumors (mean volume of 1.14 cc) were treated with Stanford's Cyberknife (n=40) or Varian Linac (n=34) with three fractions (at 24 or 12 hour intervals, respectively) of 6 or 7 Gy to (80% IDL). Prospectively specified follow-up consisted of neurologic evaluation and MR imaging at 6, 12, 24, and 36 months.

Results: After mean follow-up of 44 months in 64 proton beam patients, 35 (54.7%) tumors were smaller, 25 (39.1%) were unchanged, and four tumors enlarged (actuarial control rate at 2 years of 94% and at 5 years of 84%). Three patients (4.7%) developed persistent facial weakness; three (4.7%) developed persistent facial hypesthesia, and two of six (33%) retained useful hearing. After a mean follow-up of 24 months in 72 fractionated Linac patients, 29 (40.3%) tumors were smaller, 42 (58.3%) were unchanged, and one (1.4%) tumor enlarged (actuarial control rate at 2 years of 95%). One patient (1.4%) developed persistent facial weakness; six (8.3%) developed persistent facial hypesthesia, and 40 of 46 (87%) retained useful hearing.

Conclusion: Proton beam stereotactic radiosurgery of vestibular schwannomas safely controls tumor growth even at low marginal doses to relatively large tumors. Tri-fractionated Cyberknife treatment may offer high rates of hearing preservation.

Multimodality Management of Vestibular Schwannomas

Carl B. Heilman, M.D., Nik Blevins, Dennis Poe, M.D., David Vernick, M.D., Jon Border, M.D.

Department of Neurosurgery (CBH, JB) and Otolaryngology (NB, DP) Tufts New England Medical Center, Division of Otolaryngology (DV) Beth Israel Deaconess Medical Center

The optimal management of a patient with a vestibular schwannoma is controversial. Treatment options include watchful waiting with serial imaging, radiation therapy, radiosurgery, and surgical excision. The surgical options include the retromastoid approach (RM), translabyrinthine approach (TL) and the middle fossa (MF) approach. The management of 139 consecutive patients with a vestibular schwannoma, treated over a six-year period will be presented. 96 patients were treated surgically (66 RM, 23 TL, 7 MF). In 33 patients, the initial management was watchful waiting with serial imaging studies – 28 patients continue to be followed. Fifteen patients were treated by radiation (2 Linac, 1 IMRT, 12 Gamma Knife). Total tumor removal was performed in 95% (91/96) of the patients treated surgically. In patients with tumors < 3 cm, total tumor removal was performed in 98% (69/70) of the patients. 94% (66/70) of the surgically treated patients with tumors < 3 cm had post operative facial function of House Brackman Grade 1 (59 pts.) or 2 (7 pts.). Although 26% (25/96) of the surgical patients suffered a temporary complication of some kind, there were only 5 permanent complications (other than cranial nerve 7 or 8) in the surgically treated group (3 chronic headaches, 1 contralateral lower cranial nerve palsy, 1 corneal scar). Preoperative trigeminal nerve sensory loss was present in 22 patients and resolved completely postoperatively in 17. Preservation of Gardner Robertson Grade 1 or 2 hearing in the surgical group was possible in 38% overall (15/40), 42% in tumors < 2.0 cm and 70% (7/10) in intracanalicular tumors. There were no cases of postoperative cerebellar ataxia, no strokes, no cerebellar hemorrhages and no deaths. The advantages and disadvantages of surgery versus radiosurgery/radiation therapy will be discussed.

Acoustic Neuroma Radiosurgery: A Benchmark to Compare Against Other Management Modalities

L. Dade Lunsford, M.D., Douglas Kondziolka, M.D., David Bissonette, PA-C, John C. Flickinger, M.D.

Background: Management options for acoustic neuroma have expanded during the last decade. Confusion appears to be increasing about the outcomes of various radiation techniques. In order to provide a benchmark for gamma knife stereotactic radiosurgery (GKSR), we reviewed our 13 year experience.

Methods and Materials: 668 patients underwent GKSR. 109 (16%) had recurrent tumors after surgery. Multiple isocenter radiosurgery (mean 5.8 per patient) was used. After 1993, the marginal dose averaged 13 Gy and has not changed in eight years. 134 had follow-up greater than ten years.

Results: With minimum five year follow-up, the tumor control rate was 98%. Preservation of useful hearing, Gardner-Robertson I or II was 75%. Transient adverse radiation effects including early temporary enlargement developed in 19 (2.8%). During the past seven years, the risk of developing a temporary facial nerve dysfunction was $\leq$1%. Patients returned to their pre-procedure lifestyle in 24 to 48 hours.

Discussion: By the year 2001, radiation modality management may be applied to more than one-half of newly diagnosed acoustic neuroma patients. Our experience supports the benefit of GKSR in properly selected patients. Such results should not be generalized to emerging fractionated radiation modalities in the absence of long-term outcome data. We believe that patients should select therapeutic options based on a thoughtful, long-term outcome analysis rather than hype or hubris.

Genetically Engineered Herpes Simplex Viruses in the Treatment of Glioma

James Markert, M.D., Yancey Gillespie, Ph.D., Michael Medlock, M.D., Robert Martuza, M.D.

G207 is a conditionally replicating derivative of herpes simplex virus (HSV) type-1 engineered with deletions of both • ¡34.5 loci and a lacZ insertion disabling the U_L39 gene. We have demonstrated the efficacy of G207 in treating malignant glial tumors in athymic mice, as well as the safety of intracerebral G207 inoculation in mice and in *Aotus nancymai*. We sought to determine the safety of G207 inoculation into cerebral malignant glial tumors in humans. Criteria for inclusion into this dose-escalation study were the diagnosis of histologically proven malignant glioma, Karnofsky score $\geq$ 70, recurrence despite surgery and radiation therapy, and an enhancing lesion greater than 1 cm in diameter. Serial magnetic resonance images were obtained for volumetric analysis. The trial commenced at a dose of 10^6 plaque-forming units (pfu) inoculated at a single enhancing site and was completed when the 21st patient was inoculated with $3x10^9$ p.f.u. at five sites. No toxicity developed that was ascribed to G207. No patient developed HSV encephalitis. We found radiographic and neuropathologic evidence suggestive of anti-tumor activity and long-term presence of viral DNA in some cases.

Because of these promising findings, a phase Ib/II trial was designed to examine safety of increasing doses of G207 and provide preliminary efficacy data. In this trial, Phase Ib patients undergo stereotactic inoculation of G207 followed by resection and re-inoculation; Phase II patients will undergo resection followed by inocuation of G207. Preliminary results will be reported.

ACADEMY AWARD PAPER

Inosine Induces Extensive Anatomical Reorganization and Improves Functional Outcome after Cortical Stroke

Peng Chen, M.D., David E. Goldberg, M.S., Larry I. Benowitz, Ph.D.

Department of Neurosurgery/Neuroscience, Children's Hospital, Harvard Medical School, Boston, MA

Introduction: We have investigated anatomical and molecular mechanisms that enable mature cortical neurons to reorganize their connections after cerebral infarct and to enhance functional recovery. An intracellular, purine-sensitive kinase, N-kinase, is activated when neurons undergo neurite outgrowth. Inosine acts as an N-kinase agonist, and in cell culture, it causes neurons to regenerate their axons and to express a number of genes that are associated with axon growth, e.g., GAP-43, alpha-1 tubulin, etc. This study examined whether inosine can activate this same intracellular signaling pathway to cause neurons to revert to a growth state and to reorganize their connections after stroke.

Methods: Inosine or normal saline was administrated intraventricularly into rats with a unilateral middle cerebral artery occlusion. Comprehensive neuro-behavioral testing, e.g., forepaw placement, swimming, and food retrieval, was carried out for 6 weeks on a blinded basis. Biotin Dextran Amine (BDA), an anterograde axonal tracer, was then injected into the contralateral sensorimotor cortex to visualize axon trajectories arising from the intact hemisphere. Infarct volumes were analyzed to evaluate potential neuroprotective effects.

Results: Inosine-treated animals showed significantly greater recovery compared to vehicle-treated controls, most strikingly in free use of the denervated forepaw. Along with this we observe unprecedented levels of brain reorganization after inosine treatment, i.e., massive growth of axonal collaterals from the spared corticorubral and corticospinal tracts, extending across the midline into the denervated areas of the brainstem and spinal cord. The absence of differences in infact volume between groups suggests that inosine exerts its effect primarily by inducing CNS re-organization.

Conclusion: The structural re-organization after stroke demonstrated in this study is unprecedented. The strong effects of inosine on re-organizing cortical projections and improving functional outcome in an animal model may have significant clinical applications in victims of stroke or CNS trauma. A clinical trial is planned for the near future.

ACADEMY AWARD HONORABLE MENTION

Germline and Somatic Mutations of Suppressor of Fused Predispose to Medulloblastoma Through Failure to Suppress Sonic Hedgehog and Wnt Signaling

Michael D. Taylor, James T. Rutka

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The high incidence of medulloblastoma in children with Gorlin's syndrome (Nevoid Basal Cell Carcinoma Syndrome) and Turcot's syndrome has shown the importance of developmental signaling pathways in the pathogenesis of this malignant childhood brain tumor, and may suggest why some children with brain tumors also have developmental anomalies. These syndromes (Gorlin and Turcots) are due to over-activation of the Sonic Hedgehog and Wnt signaling pathways respectively. Both of these signaling pathways are known to be powerful mitogens for cells of the developing external granule cell layer of the cerebellum which is believed to be the cell of origin for many medulloblastomas. Somatic mutations of genes in these two pathways that result in pathway activation have been found in medulloblastoma, stressing their importance in sporadic medulloblastomas as well.

We show for the first time that a subset of medulloblastomas, (predominantly the desmoplastic subtype) undergo truncating mutations of *Human Suppressor of Fused* on chromosome 10q24.3, accompanied by loss of the wild type allele. This suggests that HSUFU acts as a classical Knudsen type tumor suppressor gene in some medulloblastomas. We further show that some children with medulloblastoma (with and without developmental anomalies) have germline mutations of HSUFU that may predispose them to develop medulloblastomas. Whereas wild type HSUFU can bind oncogenic Gli transcription factors (the effectors of Shh signaling) and export them from the nucleus thus blocking Shh signaling, this ability is lost in tumor derived mutants of HSUFU. While wild type HSUFU can also bind ß-catenin (the effector of Wnt signaling), export it from the nucleus, thus blocking Wnt signaling, medulloblastoma derived mutant HSUFU cannot. These findings suggest that germline mutations of HSUFU predispose to the development of a novel genetic syndrome with developmental and neoplastic manifestations in which affected patients develop medulloblastoma through failure to suppress excessive Shh and Wnt signaling.

NOTES:

SATURDAY, NOVEMBER 17 8:45 - 9:00 AM

Experimental Treatment of Guinea Pig Sciatic Nerve Injury with Topical Polyethylene Glycol

Jill W. Donaldson, M.D., Riyi Shi, Ph.D., Richard Borgens, Ph.D., Scott A. Shapiro, M.D.

Objective: To use polyethylene glycol (PEG) to restore physiological function in a severely injured guinea pig peripheral nerve.

Methods: A guinea pig sciatic nerve segment was placed into a sucrose gap chamber. Following baseline recordings, the nerve was transected and was treated with a 2 minute application of PEG solution. Recording following PEG was done for 30 minutes. Controls were treated with Krebs' solution. The *in situ* experiments were done by exposing the gastrocnemius muscle and the sciatic nerve in anesthetized 300-425 gram guinea pigs. Simultaneous measurements of compound motor endplate potentials, contraction displacement of the hind paw, and muscle contraction force in response to square wave stimulation of the sciatic nerve were made before and after a constant displacement crush injury was made. The crushed site was treated topically with 0.15 to 0.30 cc of 1800 MW PEG (50% w/w in dH2O) immediately after the crush for 2 minutes and then irrigated with Krebs' solution (n=8). A second group had PEG treatment delayed to 1 hour post crush (n=6), and a third group had PEG applied at 4 hours post injury (n=6). Controls were treated with Krebs' solution (n=12).

Results: *In vitro* studies showed recovery of action potential propagation through a transected nerve following PEG application (2 of 3 PEG treated vs. 0 of 3 control). The proportion of immediately PEG treated animals showing a recovery of at least one of the three functions was significantly improved over control spontaneous recoveries (7 of 8 PEG treated vs. 3 of 13 control; P=0.007, Fishers exact test). Delayed PEG application showed improved recovery over controls (8 of 12 PEG treated vs. 4 of 12 control); however, the results did not reach statistical significance.

Conclusion: Some degree of functional recovery of severely injured mammalian peripheral nerves can be accomplished using PEG.

Biomechanical Advantage of a Translational Anterior Cervical Plate

Kevin T. Foley, M.D., Denis J. DiAngelo, Ph.D., Weiqiang Liu, Ph.D., Kristine M. Olney, B.Sc., Larry Davidson, M.D.

School of Biomedical Engineering and Department of Neurosurgery, The University of Tennessee, Memphis, TN

INTRODUCTION: Clinical experience has shown that anterior cervical plating does not prevent construct failure in multi-level cervical corpectomy. The design of the anterior cervical plate may contribute to this phenomenon.

METHODS: Ten fresh cadaveric cervical spines (C2-T1) were evaluated in the following conditions: harvested (H), (C4-C6) corpectomy (C), strut-graft alone (SGA), strut-graft with constrained anterior cervical plate (CACP, Orion™), and strut-graft with translational anterior cervical plate (TACP, Premier™). Measurements included vertebral motion, applied load and moment, and load transferred through the strut-graft. Vertebral rotation and applied moment data were combined to calculate global spine stiffness. A one-way ANOVA ($p<0.05$) was used for statistical comparisons.

RESULTS: Application of both anterior cervical plates increased the global (C2-T1) stiffness and decreased the local (C3-C7) motion. Flexion of the SGA spine loaded the strut-graft, whereas extension unloaded the strut-graft. With both plates, these load transfer patterns were reversed. The CACP construct produced significantly higher graft loads in extension than the TACP construct ($p<0.001$) and SGA spine ($p<0.001$). Importantly, there was no significant difference in graft loads in extension between TACP and SGA constructs. In flexion, there were no significant differences in graft loads among the CACP, TACP, and SGA constructs.

CONCLUSIONS: This study is the first to report the *in vitro* effects of "dynamized" cervical instrumentation on multi-level graft loads. Application of either a constrained or a semi-constrained, translational anterior cervical plate to a strut-grafted, multi-level cervical corpectomy model decreased the local motion and increased the stiffness of the instrumented levels. Clinically, this would serve to promote fusion. However, the constrained plate significantly increased strut-graft loads in extension, which would promote graft pistoning and may lead to construct failure. While stabilizing the corpectomy model, the semi-constrained, translational plate did not produce significant changes in strut-graft loads. Thus, this plating system would be considered biomechanically advantageous in this setting.

Science for Free

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The world wide exchange of scientific information has recently been exposed to a deep conflict of interests, and to a strategic dilemma.

On the one hand, there is a legitimate interest of the scientific community to ensure adequate quality of published data. This appears to be best achieved by peer review (which, however, necessarily leads to a delay in publication).

On the other hand, the internet now offers the unlimited possibility to publish scientific data world wide at literally no expense, with no delay, and with no control.

The conflict between quality control and unrestricted publication has triggered a controversy about the "three f's": free, fast, faultless.

The battle is not without economic interests. Since almost every piece of scientific information is freely available on the internet, who is willing to carry the costs of scientific publication?

Not only this. Scientific research data, usually obtained with the help of public funding, is forwarded gratis by the scientists (as manuscripts) to private institutions (publishers), who earn a considerable amount of money with this valuable "scientific merchandise". Why?

In addition, in order to have a scientific manuscript accepted, the scientist must transfer the copyright to the publisher. Why?

Publication of a manuscript in one journal excludes publication of the same paper in any other journal. Why?

Nevertheless, the scientist is at the mercy of publishers, since funding of scientific projects is still governed by the "impact factor", a form of evaluation exposed to numerous sources of error.

Two internet sites are engaged in debating the above deficiency and in searching for solutions:

www.free-science.com and www.publiclibraryofscience.org.

The problems exposed above require thorough and prompt discussion by the scientific world.

Role of RhoA in Experimental Spinal Cord Injury in Rat

J.K. Sung, M.D., Ph.D., L. Miao, M.D., Ph.D., J.W. Clavert, B.S., H. Louis Harkey, M.D., J. H. Zhang, M.D., Ph.D.

Object: The small GTPase RhoA belongs to the Rho subfamily of GTPases within the Ras superfamily. RhoA regulates the organization of the actin cytoskeleton, gene expression, and cell proliferation. This study has undertaken to investigate the involvement of the RhoA signaling pathway in the secondary injury that follows traumatic spinal cord injury in rats.

Method: Female Sprague-Dawley rats (n=90) weighing 250-300-gm were used. A moderately contused spinal cord injury was effected by the weight drop method (a 10-gm weight x 1.25-cm height) at T9 or T10 level after one level laminectomy. The injured segment of spinal cord was collected at 1 hour, 3 hours, 1 day, 3 days, 1 week, and 3 weeks post-injury and the expression of RhoA was measured with competitive RT-PCR, Western blot, and immunohistochemistry. RhoA mRNA and protein expressions were enhanced significantly in the injured spinal cord 1 week after surgery ($p<0.05$, ANOVA).

In another series, C3 exozyme (RhoA inhibitor) and fasudil (Rho kinase inhibitor) were administered after spinal cord injury, and the subjects were evaluated for 5 weeks as per BBB locomotor score. Poor rat response interrupted the C3 experiment. Fasudil significantly improved the BBB score ($P<0.05$, ANOVA). The levels of Rho-kinase (ROKα ROKβ) proteins decreased significantly in the group of fasudil-treated rats.

Conclusion: Spinal cord injury activates the RhoA/Rho-kinase α, β associated pathway. RhoA/Rho-kinase α, β might be involved in the secondary injury. Fasudil might exert a cytoprotective effect by inhibiting Rho-kinase α, β.

Preliminary Results of AANS/CNS Joint Spine Section Pilot Study on Lumbar Disc Herniation Utilizing Internet Based Data Collection and Management

Paul C. McCormick, M.D., M.P.H.

Study Design: A prospective multicenter observational outcomes study of patients treated surgically for herniated lumbar disc.

Objective: To gain organizational experience with online data collection, storage, and analysis.

Methods: 40 neurosurgeons from academic and private practice were recruited to prospectively enroll 10 consecutive patients undergoing surgery for single level lumbar disc herniation into the study. Each patient completed a baseline assessment instrument preoperatively and at 6 weeks, 3 months, and 1 year postoperatively. The patient instrument includes the SF-36, the Oswestry pain/disability scale, a neurogenic symptom scale, comorbidity assessment, and various demographic and socioeconomic items known to be associated with spinal surgery outcomes. A two page surgeon operative questionnaire and 1 page 6 week follow-up form was completed on each patient by the surgeon. The instruments were available online through the Neurosurgery On/Call website.

Results: Over 350 patients from 25 surgeons have been enrolled to date. Not surprisingly, significant improvements were noted in the physical functioning, role functioning, and bodily pain subscales of the SF-36 as well as the Oswestry index and the neurogenic symptom scale. Patient satisfaction with treatment and outcome was high but did identify opportunity for improvement, particularly at the hospital level.

Conclusions: This pilot study demonstrates the feasibility of online data collection for outcomes studies, practice management, and best practice benchmarking. The preliminary experiences, possible implications and utilization, and potential obstacles for Internet based data collection and management will be discussed.

CSF Pulsatility Analysis: A Preliminary Accurate Diagnostic for NPH and Obstructive Hydrocephalus

Eldon L. Foltz, M.D.

For 12 years, CSF pulsatility studies, laboratory and clinical, have proved:

1. The normal CSF pulsatility (CSFp) is a cardiac systole generated brain pulse, damped by cerebral venus volume venting with each systole into dural sinuses. As per Monroe Doctrine concerning compartment spaces within a relatively closed compartment, compensation for increasing volume/pressure in one compartment occurs, causes compensating changes (volume reduction etc.) in the other compartments which have a common physical component – i.e., an effort to maintain total intracranial stability in this case.
2. All progressing hydrocephalus has been shown associated with undamping of this normally damped pulsatility, secondary to exhaustion of venus volume venting due to excessive venus volume loss. Brain pulsations are progressively lost. Presumptive conclusion is that compliance is being lost. The brain therefore must deform if the enlargement in the one compartment (CSF) continues – i.e. the ventricles enlarge, herniation of the brain occurs, etc.
3. In cases of developing volume increase in one cranial compartment, initiation and progression of the undamped high amplitude, short latency CSFp proceeds and PRECEEDS main pressure elevation as compliance (cranial venus volume loss) is exhausted.
4. This is a progressive event, not necessarily rapid. CSFp undamping, therefore, may start brain deformation before mean ICP elevation is recognized.
5. Since underlying CSFp is characteristic of hydrocephalus in any compartment, such also represents loss of CSF absorption capacity in that compartment.
6. Two clinical applications have been initiated for the use of this CSFp undamping as a diagnostic aid, preliminary to proposed larger studies:
    a) 22 patients: differential diagnosis of NPH vs atrophy by lumbar puncture and analysis of the CSFp; undamped wave indicates hydrocephalus is the diagnosis.
    b) 4 patients: L.P. recording of CSFp in aqueduct stenosis with a ventricle shunt indicates that the subarachnoid space is normal for absorption of

CSF, and a third ventriculostomy may succeed since communicating hydrocephalus will not be produced instead of obstructive. If CSFp shows undamping, third ventriculostomy is contra-indicated.

This initial report is designed to stimulate interest in this relatively simple but apparently effective diagnostic aid in two problem areas of neurosurgery where more accurate preoperative diagnoses are important.

Multimedia Database

Robert F. Spetzler, M.D., Shahram Partovi, M.D., Jeffrey Henn, M.D., Mauro Ferreira, M.D.

Our multimedia database is composed of two segments. One housing textual medical record data, and the other archiving video and still image data on neurosurgical operative procedures. These databases are cross-linked such that viewing a patient's medical record can lead the user to the operative digital photographs and videos for that individual. A full search engine aids in identifying target cases. The retrieval interface is platform independent and browser based. Data entry occurs at three key locations: first in the operating theatre, secondly from our radiology digital Picture Archiving and Communication Systems (PACS) environment, and thirdly via a link to our Information Systems (IS) gateway. Our operative scopes are equipped with digital cameras for still image acquisition and digital video recorders to store the video output. The still images are edited and optimized prior to entry into the multimedia database, and the videos are digitally edited down to three versions – short, medium and long duration – and added to same database. The pre-operative and post-operative radiographic images, such as Magnetic Resonance (MR) images and cerebral angiograms, are copied from our PACS system onto our multimedia database to accompany the operative photos. Finally, the IS gateway retrieves relevant patient information and this data is linked to the multimedia information on our back end Structured Query Language (SQL) server. When a user selects a patient, not only can radiographic and operative images be viewed, but the actual edited video footage can be streamed over the network to the viewer for review on demand. Anatomical, computer modeling and operative procedures are compiled for 2D and 3D and interactive PC presentations.

Addition of Elemental Iodine to Surgical Irrigation for Shunt Infection Prophylaxis

SooHo Choi, J. Gordon McComb, Michael L. Levy, Ignacio Gonzalez, Roger Bayston

Objective: Elemental iodine has excellent ability to kill a broad spectrum of bacteria, fungi and viruses. Furthermore, it is inexpensive, bacterial resistance is unknown, and allergic reaction is rare. Because of these properties and in an attempt to further reduce shunt infections, we have undertaken to determine the concentration of elemental iodine that will kill Staphylococcus epidermidis and aureus, the most common organisms to cause shunt infections, without causing injury to the CNS.

Methods: Bacterial kill studies using Staphylococcus epidermidis and aureus were performed using Ringer's lactate alone or containing iodine at a concentration of 5, 10, 20, 50, 100 or 1000 ppm and compared with Cefazolin (1 mg/ml) and Bacitracin (50 units/ml).

21 Adult males Wistar rats consisting of 7 groups underwent a fronto-parietal craniotomy. Brains were irrigated for one hour with Ringer's lactate alone or containing iodine of the same concentration as noted above. After 72 hours of observation, the animals were sacrificed. The brains were then fixed with formalin and stained with hematoxylin/eosin and examined.

Results: Even with exposure for as little as 15 seconds to an iodine solution of 20 ppm, zero growth was detected following an inoculum of 100 million of either bacteria. In contrast, the two antibiotics were not nearly as effective as the iodine, with kill rates ranging from 19% to 93%.

Examination of the rat brains showed no histologic changes at 5, 10, 20, 50 ppm. However, concentration of 100 and 1000 ppm, necrosis was observed.

Conclusion: Elemental iodine can be added to an irrigating solution in sufficient concentration to be bactericidal without causing any CNS injury.

Neurosurgical Applications of High Resolution Thermal Imaging

F.B. Meyer, S. Goerss, B. Kall

Background: Infrared or thermal brain imaging has been attempted in the past without significant success. Advances in optics combined with improved understanding of quantum tunneling have resulted in the development of superior novel thermal imaging technology that utilized quantum well infrared detectors (QWIP). In addition novel imaging software has been developed to analyze modulations in temperature of tissue being imaged.

Objective: QWIP infrared imaging was combined with temperature modulation software to access surface brain temperature changes in various neurosurgical procedures to determine if this technology might have a future role in examining cortical surface blood flow to outline tumor margins, seizure foci, and hyperemic conditions.

Results: A variety of patients were imaged intraoperatively. In two patients undergoing extracranial-intracranial bypass, there were increases in cortical blood flow over the exposed brain surface which correlated well with doppler blood flow through the bypass graft. In one patient the imaging data provided evidence for hyperemia in adjacent brain following AVM resection which led to aggressive control of blood pressure postoperatively to help prevent postoperative hemorrhage. The imaging software nicely depicted metastatic tumor below the cortical surface. In gliomas there was reasonable correlation with the tumor margins outlined by the imaging with MRI stereotactic coordinates.

Conclusions: This preliminary data indicates that new infrared imaging technology and software may prove to be a valuable addition to intraoperative imaging techniques. Specifically, it appears to be able to nicely demonstrate significant cortical blood flow changes which may prove valuable in managing cerebrovascular patients. Furthermore, it may also prove to be an intraoperative technique to outline tumor margins and perhaps allow the surgeon to account for intraoperative brain shift. However, this preliminary data needs to be substantiated by a large number of intraoperative imaging experiments.

Combined Surgical Approaches Through the Temporal Bone: Surgical Anatomy, Pitfalls and Complications

Jacques J. Morcos, M.D., Mustafa K. Baskaya, M.D., Imad A. Abumeri, M.D., Ernesto Coscarella, M.D.

Introduction: "Combined" surgical approaches through the temporal bone provide access to more than one intra/extra cranial fossa or compartment. Though their general usefulness has not been questioned, their safety and specific applications have varied widely. Our aim is to discuss the surgical anatomy, applications, pitfalls and complications of these approaches.

Methods and Results: We analyzed retrospectively a series of 29 patients with mass lesions who had undergone combined surgical approaches through the temporal bone between November 1995 and October 2000. Ages range from 8 to 69 years with a mean of 41 years. Male to female ratio was 16 to 13. Pathologies included 14 meningiomas, 6 chondrosarcomas, 2 acoustic neuromas, 2 glomus jugulare tumors, 1 trigeminal schwannoma, 1 craniopharyngioma, 1 epidermoid tumor, 1 hemangiopericytoma, and 1 mucocele of the petrous apex. Surgical approaches were divided into the following groups: a) anterior petrosal or Kawase approach (n=3); b) posterior petrosal combined with subtemporal approach (n=15); c) combined posterior and anterior petrosal approach (n=3); d) other combinations (n=8). Posterior petrosal combined with subtemporal approach included retrolabyrinthine, partial translabyrinthine, translabyrinthine, transcochlear and transotic approaches. The "other combinations" included the following approaches: combined retrolabyrinthine with jugular foramen; combined-combined; combined translab with retrosigmoid and transsigmoid; combined translab or transotic with neck dissection; and combined retrolab with jugular foramen approach. Clinical outcome and complications are analyzed and presented in detail as they relate to specific approaches. Anatomical dissections in the laboratory were also performed to rationalize the use of specific approaches for specific pathologies at specific locations. We present our decision-making paradigm.

Conclusion: Combined surgical approaches through the temporal bone enable surgical treatment of various pathologies in the petroclival region in a safe and effective manner. A better understanding of the interaction between surgical anatomy and nature and extent of the lesion, along with refinements of each approach are of great importance in improving outcome and complication avoidance.

Adenoviral Induction of NG2+ Neural Precursors in the Corpus Callosum

Joshua Rosenow, M.D., Eva Chmielnicki, Abdellatif Benraiss, Ph.D., Steven A. Goldman, M.D., Ph.D.

Introduction: Neural progenitor cells persist in multiple locations within the adult mammalian brain and respond to many diffusible factors, such as brain-derived neurotrophic factor (BDNF) and noggin, with increased survival or maturation. This pilot study attempted to induce endogenous precursors using adenoviral delivery of neurotrophin genes.

Methods: Adenovirus (AdBDNF) containing the BDNF gene under CMV control in tandem with the gene for human green fluorescent protein (hGFP) was injected (2• L) into the cortex (n=3), corpus callosum (n=3), and striatum (n=3) of adult rats. Contralateral sites in each animal received AdCMV:Null:hGFP or saline. Rats were injected for 18 days with bromodeoxyuridine (BrdU), 100 mg/kg intraperitoneal, to label mitotically active cells prior to sacrifice. Other animals also received intraventricular injections of AdNoggin with (n=1) or without (n=3) focal delivery of AdBDNF to the corpus callosum. Confocal microscopy was used to analyze sections for colocalization BrdU and markers of either neuronal (• III-tubulin) or oligodendroglial (NG2) lineages.

Results: Neurogenesis was not observed in either the cortical or callosal sites. A few • III-tubulin+/BrdU+ cells were noted in the striatum of one animal. AdBDNF produced a marked increase in the number of NG2+/Brdu+ oligodendroglial precursors in the callosum as compared to AdCMV- (2.78-fold) and saline- (5-fold) injected animals. Intraventricular AdNoggin injections did not significantly alter this effect.

Conclusions: Adenoviral delivery of neurotrophin genes increase the number of proliferating oligodendroglial, but not neuronal, precursors in the callosum.

Diffuse Multilobar Infiltrating Glial Tumors: A Modern Day Account of 22 Cases

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Diffuse multilobar infiltrating glial tumors, also known as gliomatosis cerebri, involve extensive portions of the cerebral hemispheres and underlying structures. Because of its rarity, little is known about factors that influence its natural course, or about the value of treatment. We reviewed 22 cases of this entity, diagnosed at our institution by MRI and biopsy; half the patients were male, and the median age at onset was 49 years (range 7-79). The median time from onset of symptoms to diagnosis was 3 months, with most patients presenting mental status changes (77%), seizures (49%) or headaches (41%). All patients had MRI, and we found that T2 weighted images and FLAIR best showed the extent of disease. The majority (55%) of patients had more than two lobes involved, almost all showed deep gray matter involvement (95%), and most patients showed bilateral disease (77%). Twelve patients had a dominant tumor mass (55%), twelve patients (55%) showed gadolinium enhancement, and nine patients (41%) had both. Seven patients also had MR spectroscopy, although only one had MRS before biopsy. Eighteen patients had tissue specimens diagnostic of astrocytoma; only one patient had tissue also showing oligodendroglial features. One patient had grade 2 tumor, seven had grade 3 tumor, and ten had grade 4 tumor. The other four patients showed gliosis, although two had neoplasm at autopsy. Kaplan-Meier analysis showed median length of survival (LOS) was two months in patients who did not receive treatment (n=4), 28 months in patients treated with radiation, and 29 months in patients treated with radiation and chemotherapy. Cox proportional hazards multivariate regression showed that initial KPS <70 and higher tumor grade all correlated with worse outcome ($p < .95$); in addition, treatment with radiation (with or without chemotherapy) significantly prolonged LOS 9 $p < .01$). Age and extent of disease on MRI did not correlate with LOS. Our findings suggest that aggressive, modern day treatment can improve length of survival, contrary to previous reports, and that biopsy is essential to not only diagnose but also to prognosticate about LOS. MRS may prove useful in guiding biopsy and in describing the full extent of disease.

An Inflatable Balloon Catheter and Liquid I-125 Radiation Source for Treatment of Recurrent Malignant Glioma: The Gliasite Radiation Therapy System

Stephen B. Tatter, M.D., Ph.D., Charles L. Branch, Jr., M.D., Edward G. Shaw, M.D. (Wake Forest, Winston-Salem, NC), Mark L. Rosenblum, M.D., Tom Mikkelsen, M.D. (Henry Ford, Detroit, MI), Jon Weingart, M.D., Alessandro Olivi, M.D., Henry Brem, M.D. (Johns Hopkins, Baltimore, MD), Jeffrey J. Olson, M.D. (Emory, Atlanta, GA), Steven Brem, M.D. (Moffitt Cancer Center, Tampa, FL), Dennis A. Vollmer, M.D. (University of Texas, San Antonio, TX) for the New Approaches to Brain Tumor Therapy (NABTT) CNS Consortium

INTRODUCTION: The GliaSite RTS is an inflatable balloon catheter that is placed in the resection cavity at the time of tumor debulking. Internal radiation is delivered with an aqueous solution of organically-bound I-125 (Iotrex). A multi-institutional study of 21 patients (GBM=15, AA=5, AO=1 at initial diagnosis) has been completed.

METHODS: Adults with recurrent malignant gliomas (enhancing tumor diameter = 2-5 cm, maximum ratio of major axes <=1.5) and KPS >=60 underwent resection and implantation of a 2, 3, or 4cm diameter GliaSiteRTS catheter. 1-2 weeks later, the device was filled with Iotrex and saline for 3-6 days, whereupon the Iotrex was retrieved and the device explanted.

RESULTS: Implant, brachytherapy and explant procedures were well tolerated by all patients. Radiation delivery lasted 3-6 days achieving prescription doses of at least 40-60 Gy to all tissues within the target volume (5-10 mm depth). In most cases, the prescription dose encompassed the entire residual enhancing tumor volume. There were no serious adverse events during brachytherapy. Two patients had craniotomy infections and two had aseptic meningitis. No radiation necrosis has been identified in 18.1 patient years of follow-up. Median survival in the entire cohort of already aggressively-treated patients is extremely encouraging: 394 (95%CI=210-455) days.

CONCLUSIONS: The GliaSite RTS performs safely and efficiently. This device preserves quality-of-life and delivers a readily-quantifiable radiation dose to the tissue at highest risk for recurrence. The current results lead to FDA approval of Gliasite RTS for the treatment of malignant gliomas April 2001. They justify additional dose-escalation trials.

Opposite Effects of CIS-Parinaric Acid in Activities of P-38 MAP and c-Jun N-Terminal Kinases in Malignant Rat Astrocytoma Cells

Vincent C. Traynelis, Ayesha Zaheer, Shailendra K. Sahu

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Cis-Parinaric acid (cPNA) is a natural, conjugated polyunsaturated fatty acid. Micromolar concentrations of cPNA are preferentially toxic to malignant glial cells compared to cultured normal astrocytes. Our lab has demonstrated that exposure of malignant glioma cells to cPNA is followed by increased production of free radicals and that antioxidants diminish the cytotoxicity of cPNA. This study was designed to investigate the molecular mechanism of cPNA-induced oxidative stress on gliomas.

Members of mitogen-activated protein (MAP) kinases superfamily, p38 MAP kinase (p38 MAPK) and c-Jun N-terminal protein kinase (JNK) are involved in the signal transduction of oxidative stress. There are data which indicate that p38 MAPK may have a "protective" function while JNK is associated with apoptosis. We measured the activities of p38 MAPK and JNK in 36B10 rat astrocytoma cells using Western Blot analysis and specific phospho-antibodies against each of the two kinases. These studies revealed that *in vitro* glioma exposure to cPNA results in a greater than 50 percent decrease in p38 MAPK activity. JNK activity increases three-fold following treatment with cPNA. The observed decrease in p38 MAP kinase activity by cPNA differs from the known effects of other compounds such as H_2O_2, which activates both kinases. It may be related to the preferential toxicity of cPNA for malignant glioma cells. Future investigations will focus on the use of specific kinase inhibitors to determine if the inhibition of p38 MAPK is required with a persistent activation of JNK for cytotoxicity of cPNA in glial cells.

Neurostimulation for Tremor: Functional and Neuropsychological Results

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Objectives: We studied outcome measures for the first 41 of 125 consecutive patients following unilateral and bilateral thalamic stimulation for disabling tremor from essential tremor and Parkinson's disease. Surgical technique, qualitative and quantitative tremor assessments, stimulation parameters, location of active electrodes, complications and side effects, and neuropsychological evaluations are described and analyzed.

Methods: Preoperative qualitative and quantitative tremor measures were compared to those following unilateral and bilateral surgery with activated and deactivated stimulators. Stimulation parameters and side effects were recorded and outcome measures statistically analyzed. The neuropsychological effects of deep brain stimulation on cognition, tremor, and mood were assessed at presurgical baseline and again three months after surgery.

Results: Qualitative and quantitative measurements showed a significant improvement of contralateral arm ($P<0.001$), leg ($P<0.01$), midline ($P<0.001$, and ipsilateral ($P<0.01$) arm tremor after unilateral surgery. Activities of daily living improved after unilateral ($P<0.001$) and additionally after bilateral ($P<0.05$) surgery. Stimulation related side effects were reversible in all patients. Stimulation parameters did not change significantly over time. ANOVA testing demonstrated significant changes of letter fluency ($P<.01$), semantic fluency ($P<.05$) , and the Stroop test ($P<.01$), following surgery regardless of the stimulation condition. Learning and memory performances were similar to baseline with the stimulator on, but declined significantly with the stimulator off ($P<0.5$). These results are consistent with a possible "microthalamotomy" effect of surgery on specific aspects of cognition.

Conclusion: Unilateral and bilateral thalamic stimulation are safe and effective procedures leading to qualitative and quantitative improvements in resting, postural, and kinetic tremor. Thalamic stimulation-related side effects are mild and reversible. Changes could be observed three months after surgery on neuropsychological testing.

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	Elected	Deceased
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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
ROBERT S. BOURKE Rockville, Maryland (Senior)	1983	1996
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
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HOWARD BROWN ..... 1939.....1990
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Houston, Texas
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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

2002 – October 16-19
The Phoenician – Scottsdale, AZ

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2003 – October 29–November 1
Colonial Williamsburg – Williamsburg, VA

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