The American Academy Of Neurological Surgery

67th Annual Meeting

Ritz-Carlton Half Moon Bay, California
September 21-24, 2005

American Association of Neurological Surgeons

Jointly Sponsored by the American Association of Neurological Surgeons

FUTURE MEETINGS

2006
October 18–21
Ritz-Carlton Lodge, Reynolds Plantation
Greensboro, GA

2007
October 31-November 3
Ritz-Carlton, Lake Las Vegas
Henderson, NV

Mark your calendars now!

American Association of Neurological Surgeons

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2004 Annual Meeting of the

American Academy of Neurological Surgery

General Information

REGISTRATION DESK LOCATION AND HOURS

Wednesday, September 21 — 12:00pm – 6:30pm
Business Center Foyer

Thursday, September 22 — 6:30am – 12:30pm
Ballroom Pre-function

Friday, September 23 — 6:30am – 12:00pm
Ballroom Pre-function

Saturday, September 24 — 6:30am – 12:00pm
Ballroom Pre-function

HOTEL INFORMATION

Ritz-Carlton, Half Moon Bay
One Miramontes Point Road
Half Moon Bay, California 94019

Telephone: 650-712-7000
Facsimile: 650-712-7070

Program Summary - Wednesday 9/21

Registration — 12:00pm – 6:30pm
Business Center Foyer

Executive Committee Mtg — 2:00pm – 5:00pm
Boardroom

DINNER

Opening Reception – Cocktail Dinner — 6:00pm – 8:30pm
Miramontes, Observatory Montara, El Granada
Casual Attire

Program Summary - Thursday 9/22

Registration 6:30am – 12:30pm
Ballroom Pre-function

Business Breakfast - **Members Only** 6:30am – 7:30am
Miramar Room

Spouse & Guest Breakfast 7:00am – 9:30am
Observatory

Scientific Session 7:30am – 12:30pm
Ballroom

PROGRAM FOR SPOUSES

Guest Speaker (Laura Carstensen, PhD) 10:00am – 11:30am
Observatory

OTHER ACTIVITIES

Ano Nuevo Elephant Seal Habitat 2:00pm – 5:00pm
Meet in Hotel Lobby

Kayaking on the Bay 2:00pm – 5:00pm
Meet in Hotel Lobby

Golf Tournament Tee times from 12:45pm
Old Course

Tennis Tournament 2:00pm – 5:00pm
Meet at Tennis Club

DINNER

Reception 6:30pm – 7:30pm
Dinner 7:30pm – 9:30pm
Gazebo Lawn *Casual Attire*

Program Summary - Friday 9/23

Registration 6:30am – 12:00pm
Ballroom Pre-function

Business Breakfast – **Members Only** 6:30am – 7:30am
Miramar Room

Spouse & Guest Breakfast 7:00am – 9:30am
Observatory

Scientific Session 7:30am – 11:15am
Ballroom

Presidential Address – **Martin B. Camins** 11:15am – 12:00pm
Ballroom

PROGRAM FOR SPOUSES

Book Discussion **– The Kite Runner** 9:30am – 11:00am
Observatory

Presidential Address – **Martin B. Camins** 11:15am – 12:00pm
Ballroom

OTHER ACTIVITES

Filoli Mansion & Gardens Tour 12:30pm – 4:00pm
Meet in Hotel Lobby

Bicycle Tour along Coastal Access Road 2:00pm – 4:00pm
Meet at the Ocean Colony Club

Redwood Forest Trek 2:00pm – 5:00pm
Meet in Hotel Lobby

Golf Tee times from 12:45pm
Ocean Course

Tennis Clinic 2:00pm – 5:00pm
Meet at Tennis Club

Program Summary - Friday 9/23

DINNER

Presidential Reception *Ballroom Pre-function*	6:30pm – 7:30pm
BLACK TIE Dinner & Dance *Ballroom*	7:30pm – 11:00pm

Program Summary - Saturday 9/24

Registration *Ballroom Prefunction*	6:30am – 12:30pm
Breakfast for Members, Guests & Spouses *Miramar Room*	6:30am – 9:30am
Scientific Session *Salon III, IV*	7:30am – 12:30pm

SOCIAL ACTIVITIES FOR SPOUSES

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

Sunday, October 3

6:30pm – 9:30pm Opening Reception at Four Seasons Hotel, Rooms Gontard & Langhans (drinks and heavy hor d'oeuvres)

Monday, October 4

1:00pm – 4:30pm *Guided bus tour of Berlin. Tour will depart from the lobby of the Four Seasons Hotel, Casual clothes are recommended

Individual evening plans

Tuesday, October 5

10:30am *Bus transportation to Dresden (3 hours)
1:30pm Arrival at Kempinski Hotel Taschenbergpalais
2:30pm Afternoon tea at Bel Etage
7:00pm – 10:00pm Opening reception at Bel Etage

Wednesday, October 6

7:30am – 9:00am Guest/spouse breakfast at Bel Etage
8:30am – 1:00pm *Guided bus tour to the 1000-year-old town of Meissen (includes a guided tour of the renowned Meissen Porcelein Factory) Departs from hotel lobby
1:30pm *Golf at Elbflorence Course or Tennis at Tennis Club "Blau Weiß Dresden e.V."
2:00pm – 4:30pm *Guided walking tour through historical heart of Dresden, including Royal Palace, the Zwinger, and the Catholic Court Church. Departs from hotel lobby.
5:00pm – 6:30pm Snack buffet at Bel Etage
7:00pm – 9:45pm *Semper Opera Performance of Beethoven's "Fidelio"

Thursday, October 7

7:30am – 9:00am Guest/spouse breakfast at Bel Etage
9:00am – 11:00am *Guided walking tour of the Old Masters Gallery of Arts
or *Guided walking tour of the Museum of the Green Vault Treasury. Departs from hotel lobby.
12:15pm – 1:00pm Presidential Address
1:30pm *Golf at Elbflorence Course or Tennis at Tennis Club "Blau Weiß Dresden e.V."
1:30pm – 4:30pm *Guided bus tour to the baroque hunting lodge, Schloss Moritzburg, 18th century residence of King August the Strong during hunting season. Departs from hotel lobby.
7:00pm – 7:30pm Cocktail reception at Residence Castle (within walking distance)
7:30pm – 10:00pm Black Tie Dinner and Dance at Residence Castle

Friday, October 8

7:30am – 10:00am Breakfast for all members, guests, spouses at Bel Etage

* Activities require prior registration.

SCIENTIFIC PROGRAM
AMERICAN ACADEMY
OF NEUROLOGICAL SURGERY
2004 LEARNING OBJECTIVES

Jointly sponsored by Theamerican Association of Neurological Surgeons, October 3-8, 2004.

Upon completion of this program, the participants should be able to:

1. Critique the value of surgical and non-surgical options presented in the scientific papers.

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

This activity was planned and implemented in accordance with the essentials and standards of the Accreditation Council for Continuing Medical Education (ACCME) through Joint Sponsorship of theamerican Association of Neurological Surgeons and theamerican Academy of Neurological Surgery. Theamerican Association of Neurological Surgeons is accredited by the ACCME to sponsor continuing medical education for physicians.

Theamerican Association of Neurological Surgeons designates this educational activity for a maximum of 14 category 1 credits toward theamA Physician's Recognition Award. Each physician should claim only those credits actually spent in the educational activity.

DISCLOSURE INFORMATION

Theamerican Association of Neurological Surgeons and Theamerican 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 ACCME 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
Canute, GW	Inclone Systems, Inc.	Stock Shareholder
Fessler, RG	Medtronic Sofamor Danek	Consultant
Haid, R	MDT; Johnson & Johnson	Stock Shareholder
Kalfas, IH	Z-KAT	Consultant
Levy, EI	Boston Scientific;	
	Cordis Corporation	Grants/Research Support
	Cordis Corporation	Consultant
Macdonald, RL	Corporate Organization	Affiliation/Financial Interest
	NIH;american Heart Assoc.	Grants
	Actelion Pharmaceuticals, Brainsgate	Consultant
Rosenwasser, RH	Corporate Organization	Affiliation\Financial Interest
Spetzler, RF	Medtronic, Zeiss	Consultant
	Synergetics	Stock Shareholder
	Allegiance, Portal Vision	Financial/Material 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

Barnett, GH
Black, P
Bricolo, A
Brotchi, J
Carmel, PW
Chandler, WF
Couldwell, WT
Dempsey, RJ
Flamm, ES
Harbaugh, RE
Hodge, C
Hudson, AR
Krauss, WE
Lawton, MT
Leuthardt, E
Lonser, RR
Olivi, A
Oyesiku, NM
Robertson, JH
Shaffrey, ME
Shields, CB
Young, AB
Zager, EL

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

Faculty Name

None

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

Faculty Name

Canute, GW
Haid, R

SCIENTIFIC PROGRAM
American Academy of Neurological Surgery Annual Meeting
Jointly Sponsored by theamerican Association of Neurological Surgeons
Berlin, Germany
October 3–5, 2004

MONDAY, OCTOBER 4	**BERLINam*erican Academy Members***
8:00-10:00am	**Scientific Paper Presentations,** Moderator: Jim Rutka
8:00-8:15am	Recent Developments in Image-Guided Minimally Invasive Neurosurgery. Peter Black, R Kinkinis, E Claus, A Golby, D Oh, F Jolesz
8:15-8:30am	Molecular Targeting, and Imaging of Nonfunctional Pituitary Tumors. Nelson M. Oyesiku
8:30-8:45am	Cancer Care Ontario Policies Regarding Malignant Brain Tumors. Alan R Hudson, T Sullivan, JT Rutka, H-P Richter
8:45-9:00am	The Treatment of Advanced Sinonasal Malignancies with Preoperative Intra-arterial Cisplatin and Concurrent Radiation. Jon H Robertson, LM Michael, S Samant
9:00-9:15am	Surgery of Brainstem Gliomas. Albino Bricolo
9:15-9:30am	Surgical Strategy in Spinal Cord Hemangioblastomas. Experience in 30 Cases. Jacques Brotchi, F Lefranc

MONDAY, OCTOBER 4 (continued)

9:30-9:45am	Cyclooxygenase 2 (COX-2) is Ubiquitously Expressed in Meningiomas and Treatment in Vitro with the COX-2 Inhibitor Celecoxib Decreases Cell Growth: Potential Therapeutic Application. BT Ragel, RL Jensen, William T Couldwell
9:45-10:00am	Deficiency in Low-density Lipoprotein Receptor-related Protein (LRP) Expression Favors a More Invasive Glioblastoma Growth Phenotype. M Mut, MD Brown, JE Carpenter, GT Redpath, SR Vanderberg, M-B S Lopez, IM Hussaini, Mark E Shaffrey
10:00–10:30am	*Beverage Break*
10:30-11:00am	**Special Academy Lecture** Berlin: 14 Years After Unification and 767 Years After Its Foundation. Ambassador Leopold-Bill von Bredow, *to be introduced by Mario Brock*
11:00am –12:00pm	**Scientific Paper Presentations,** Moderator: Steve Papadopoulos
11:00-11:15am	Mechanisms of Cortical Reorganization: An Experimental Study. Charles Hodge, A Siddiqui, J Dubrof, R Stevens, J McCasland
11:15-11:30am	Avoiding Mount Everest - Assessing the Risks of Cerebral Aneurysms and Their Treatment. Eugene S Flamm
11:30-11:45am	Unilateral Intraputamenal GDNF Improves Bilateral Motor Functions in Patients with Parkinson's Disease. JT Slevin, GA Gerhardt, CD Smith, DM Gash, R Kryscio, A Byron Young

MONDAY, OCTOBER 4 (continued)

11:45am-12:00pm	Human Dorsal Root Ganglion Neurons as Live Nerve Constructs: Implications for Transplantation. Eric L Zager, JH Huang, EM Wilensky, BJ Pfister, DH Smith

SCIENTIFIC PROGRAM
Joint Meeting of theamerican Academy of Neurological Surgery, the German Academy of Neurosurgery and the German Society of Neurosurgery, Dresden, Germany
October 5–8, 2004

TUESDAY, OCTOBER 5	**DRESDEN**
2:30–2:45pm	Reflections on Joint Meetings Between theamerican Academy of Neurological Surgery and German Neurosurgeons. Rudolf Fahlbusch
2:45–4:30pm	**Scientific Paper Presentations,** Moderators: Jim Rutka, Hans-Peter Richter
2:45-3:00pm	Cortical and Striatal Migration of Endogenous Adult Stem Cells from the Sub-ventricular Zone and Posterior Peri-ventricular Region in Response to Stroke. Robert J Dempsey, KA Sailor
3:00-3:15pm	Genetic Variants of Matrix Metalloproteinase Genes and Their Inhibitors in Patients with Intracranial Aneurysms. Dietmar Krex, IR König, A Zieglar, HK Schackert, G Schackert
3:15-3:30pm	Downregulation of Potassium Channels After Subarachnoid Hemorrhage: A Possible Mechanism for Cerebral Vasospasm. BS Jahromi, Y Aihara, G Weyer, E Nikitina, M Agbaje-Williams, D Ryan, R Yassari, Robert Loch Macdonald
3:30-3:45pm	Prospective Evaluation of Surgical Microscope Based Indocyanine Green Video Angiography in Aneurysm Surgery. Andreas Raabe, P Nakaji, J Beck, J Kamerman, V Seifert, RF Spetzler

TUESDAY, OCTOBER 5 (continued)

3:45-4:00pm	Multivariate Analysis of a Consecutive Prospective Series of Carotid Endarterectomies. Robert E Harbaugh, A Agarwal, V Chinchilli
4:00-4:15pm	A Phase IIa, Multicenter, Double-blind, Placebo-controlled, Randomized Study to Assess the Safety and Tolerability of the Selective Endothelin A (ETA) Receptor A Antagonist Clazosentan (AVX-034343) for the Prevention of Cerebral Vasospasm, Following Severe Aneurysmal Subarachnoid Hemorrhage (aSAH). Bernhard Meyer for the Axovan Study Group
4:15-4:30pm	Endovascular Management of Intracranial Aneurysms: An Experience of 1321 Aneurysms in 1101 Patients: Single Center Experience. Robert H Rosenwasser, E Veznedaroglu, S Whang
4:30–4:45pm	*Beverage Break*
4:45–6:45pm	**Scientific Paper Presentations,** Moderators: Gabriele Schackert, Steve Papadopoulos
4:45-5:00pm	Susceptibility of Murine Neural Stem Cells and of Tumor Cells to Respond to Regenerative Signals of Axolotl Tissue. Matthias Kirsch, E Schnapp, S Lydia, E Tanaka, P Weigel, G Schackert, HK Schackert
5:00-5:15pm	Local Delivery of a Fragment of Human Endostatin Combined with Systemic Administration of BCNU Prolongs Survival in a Rodent Experimental Glioma Model. Alessandro Olivi, FG Legnani, G Pradilla, BM Tyler, F Chillemi, F Di Meco

TUESDAY, OCTOBER 5 (continued)

5:15-5:30pm	Expression of Hypoxia-inducible Carbonic Anhydrases in Brain Tumors. Martin Proescholdt, C Mayer, M Merril, A Brawanski
5:30-5:45pm	Anti-EGFR Monoclonal Antibody Cetuximab Augments Radiation and Chemotherapeutic Effects in Glioblastoma Multiforme *in vitro* and *in vivo*. JL Eller, SL Longo, MM Kyle, D Bassano, DJ Hicklin, Gregory W Canute
5:45-6:00pm	Multicentric Phase III Study, Fluorescence-guided Resection of Malignant Gliomas with 5-ALA: Preliminary Results on Interim Analysis of 270 Assessable Patients. Walter Stummer, T Meinel, the ALA-Glioma Study Group, U Pichlmeier, OD Wiestler, FE Zanella, HJ Reulen
6:00-6:15pm	Is Gross Total Resection Sufficient Treatment for Posterior Fossa Ependymoma? L Rogers, J Pueschel, Robert Spetzler, W Shapiro, S Coons, T Thomas, B Speiser
6:15-6:30pm	Management of Optic Nerve Sheath Meningiomas. U Schick, Werner Hassler
6:30-6:45pm	The Role of Endogenous Growth Hormone-Releasing Hormone (GHRH) in Acromegaly. William F Chandler, EV Dimaraki, AL Barkan, MB Brown, V Padmanabhan, SY Kim, R Taussig

WEDNESDAY, OCTOBER 6	**DRESDEN**
8:30–9:15am	**Point - Counterpoint Debate** Intraoperative MRI, Gimmick or Godsend? Robert Spetzler and Rudolf Fahlbusch
9:15-10:30am	**Scientific Paper Presentations** Moderators: William Couldwell, Johannes Schramm
9:15-9:30am	Selection of the Optimal Entry Zone to the Brain Stem for Removal of Intraaxial Cavernous Angiomas. Helmut Bertalanffy
9:30-9:45am	The Synthetic ACDF: Experience with Rh-BMP-2 and Peek Cages. Regis Haid, P Mummaneni, G Rodts, M Boakye
9:45-10:00am	Role of EphB4 Signaling in the Regulation of Vascular Morphogenesis and Vascular Permeability in Malignant Glioma. Peter Vajkoczy, R Erber, U Eichelsbacher, V Powajbo, P Knyazev, A Ullrich
10:00-10:15am	Gamma Knife Radiosurgery in the Management of Glomus Tumors—A Volumetric Study of 17 cases. Gene H Barnett**,** A Varma, JH Suh, J Ross
10:15-10:30am	Intraoperative Functional MRI. First Results and Technical Considerations. Thomas Gasser, D Stolke, R Fahlbusch, C Nimsky
10:30–10:45am	*Beverage Break*
10:45–12:30pm	**Scientific Paper Presentations** Moderators: James Rutka, Dietmar Stolke
10:45-11:00am	Sirolimus-Eluting Stents in the Canine Cerebral Vasculature**:** Assessment of Safety Profile and Vessel Response. Elad I Levy, RA Hanel, AS Boulos, FO Tio, AM Paciorek, KS Kagan-Hallet, MD Fronckowiak, LR Guterman, LN Hopkins

WEDNESDAY, OCTOBER 6 (continued)

11:00-11:15am	Brain Tumor Surgery in the Vicinity of Short-Term Memory Representation—Results of Neuronavigation using fMRI Images. Veit Braun, A Albrecht, A Wunderlich, H-P Richter
11:15-11:30am	Fiber Tract Deformation as Visualized with Sequential Acquisitions of 3D-Ultra-Sound (3DUS) During Intracranial Surgery. Volker A Coenen, T Krings, J Weidemann, FJ Hans, P Reinacher, JM Gilsbach, V Rohde
11:30-11:45am	**German Society First Prize Award - (Wilhelm Tönnis Preisträger)** Mechanical Response of a Cervical Spine Motion Segment on Removing a Local Autograft. Tobias Pitzen, CJ Kempf, WI Steudel, TR Oxland
11:45am-12:00pm	**American Academy Honorable Mention Award** Selective Ablation of Vanilloid Receptor 1 Positive Nociceptive Neurons for Elimination of Hyperalgesia and Neuropathic Pain. Russell R Lonser, GC Tender, S Walbridge, Z Olah, L Karai, M Iadarola, EH Oldfield
12:00-12:15pm	**German Academy First Prize Award** Identification of Brain Tissue Necrosis by MRI: Validation by Histomorphometry. Michael Stoffel, C Blau, H Reinl, J Breidt, K Gersonde, A Baethmann, N Plesnila
12:15-12:30pm	**American Academy Award** Achieving a Multidimensional Brain Computer Interface in Humans Using Electrocorticographic Signal. Eric Leuthardt, G Schalk, JR Wolpaw, JG Ojemann, DW Moran

THURSDAY, OCTOBER 7 **DRESDEN**

8:30–9:15am	**Point - Counterpoint** The Fate of the Academic Neurosurgeon, Viewpoints from the United States: Peter Black, and Germany: Hans-Peter Richter
9:15–10:45am	**Scientific Paper Presentations** Moderators: Steve Papadopoulos, Hansdetlef Wassman
9:15-9:30am	Clinical Outcome Following Transforaminal Lumbar Interbody Fusion (TLIF): One Year Follow-up of Prospective Data. Richard G Fessler
9:30-9:45am	Transgenic Arteriovenous Fistula in the Rat: An Experimental Model of Gene Therapy for Brain Arteriovenous Malformations. Michael T Lawton, C Stewart, AWulfstat, N Derugin, T Hashimoto, W Young
9:45-10:00am	Application of Image-Guided Spinal Navigation to Decompression and Internal Fixation of the Upper Cervical Spine. Iain H Kalfas
10:00-10:15am	Laboratory Testing of an Implantable Microsensor System for Intraoperative 3D-Computer Animation of Vertebral Body Motion in Cervical Spinal Surgery. Olaf Süss, T Kombos, S Schönherr, S Mularski, B Kühn, M Brock
10:15-10:30am	Transthoracic Discectomy Without Interbody Fusion. D Edwards, A Cohen-Gadol, William E Krauss
10:30-10:45am	Treatment of Spinal Cord Injury Via Topical Perfusion with an ATP Solution. Christopher B Shields, YP Zhang, S Chien, Y Han, LBE Shields, M Li, B Chiang, DA Burke

THURSDAY, OCTOBER 7 (continued)

10:45–11:00am	*Beverage Break*
11:00am –1:00pm	**Scientific Paper Presentations** Moderators: Joachim Gilsbach, James Rutka
11:00-11:15am	IOMaster 7D, a New Device for Virtual Neuroendoscopy. Christos Trantakis, J Meixensberger, G Strauβ, E Nowatius, D Lindner, HK Cakmak, H Maaβ, C Nagel, U Kühnapfel
11:15-11:30am	Fedor Krause, The Krause Operations, and Krause's Impact onamerican Neurosurgeons. Peter W Carmel, M Buchfelder
11:30-11:45am	Diffusion Tensor Imaging for 3-D Fiber Tract Visualization in Navigated Glioma Surgery. Christopher Nimsky, O Ganslandt, AG Sorensen, R Fahlbusch
11:45am -12:00pm	Changes of the Astrocytic Matrix and Reappearance of Radial Glia in Hippocampi of Patients Suffering From Temporal Lobe Epilepsy. Thomas M Freiman, J Eismann, M Frotscher, J Zentner
12:00-12:15pm	Surgical Treatment of Hemispheric Intractable Epilepsy in Childhood With Functional Hemispherectomy or Peri-insular Hemispherectomy. Falk Oppel, V Zountsas, HW Pannek
12:15 – 1:00pm	**Presidential Address:** Volker Sonntag**,** *to be introduced by Bill Chandler*
1:00pm	**Concluding Remarks:** Rudolf Fahlbusch, Volker Sonntag, Dietmar Stolke

Meeting Adjournment

MONDAY PROGRAM

MONDAY, OCTOBER 4 **8:00 – 8:15am**

Recent Developments in Image-guided Minimally Invasive Neurosurgery

Peter Black, R Kikinis, E Claus, A Golby, D Oh, F Jolesz

Image-guided minimally invasive techniques have significantly changed the approach to brain tumor surgery at our institution. Diffusion tensor imaging has allowed the routing anatomical mapping of fibre tracts. Functional MR with new paradigms especially created for pre-surgical planning has extended our understanding of brain function. Mathematical models have created new understandings of what factors are responsible for deficits after surgery for low-grade gliomas. Outcome analysis has begun to define what lesions are best for what technique.

These new developments now can begin to define the appropriate role of intraoperative imaging opposed to image-guided surgery in the traditional operating room. Using an experience of 240 frameless stereotactic cases and 640 intraoperative MR cases, the senior author will develop these themes to begin to present a comprehensive scheme for the use of image-guided techniques in neurosurgical oncology.

MONDAY, OCTOBER 4 **8:15 – 8:30am**

Molecular Targeting and Imaging of Nonfunctional Pituitary Tumors

Nelson M. Oyesiku

Introduction/Objectives: Our long-term goal is to determine molecular pathogenesis of pituitary tumors and to exploit these insights for diagnostic and therapeutic benefit. We have identified new molecular targets in these tumors and have developed a novel molecular-targeted imaging agent – Folatescan, to identify them.

Methods: Tumor specimens from surgery were used in the molecular and cell-culture studies. GeneChip arrays were performed and verified by RT-qPCR. Western analysis, immunochemistry and folate-binding studies were performed to determine folate expression. Cell culture and cytotoxicity studies were performed with MTT assays. In vivo imaging was performed by injection of Folatescan followed by planar and SPECT/CT imaging.

Results: Several genes, including GATA3, GATA2, PITX2, HLF, SMARCD3, and Histone deacetylase HD1 were upregulated in non-functional pituitary adenomas. The cyclin-dependent kinase inhibitor 1C (p57, Kip2), insulin-like growth factor binding protein 3 (IGFBP3), and frizzled homolog 7 (FZD7) were strongly downregulated in non-functional pituitary adenomas. In particular the folate-receptor (FR) was significantly and uniquely upregulated in nonfunctional pituitary adenomas. Folatescan successfully imaged folate receptor expressing tumors and was validated by Westerns. Similarly, non-FR expressing tumors were not imaged by Folatescan. Folate-targeted liposomes containing doxorubicin induced cell-death in folate-receptor expressing pituitary tumors in vitro, as measured by MTT assays.

Conclusions: These preliminary data demonstrate that Folatescan can successfully target FR+ pituitary tumors and provide preliminary evidence that folate can be used as a delivery system for successful tumor-targeted delivery of drugs into non-functional pituitary tumors opening the possibility of novel chemotherapy and molecular imaging of these tumors.

MONDAY, OCTOBER 4 **8:30 – 8:45am**

Cancer Care Ontario Policies Regarding Malignant Brain Tumors

Alan R Hudson, T Sullivan, JT Rutka, H-P Richter

Canada spends 10% of GDP on health. Ontario has the largest population of any Province (State) in Canada. The total Ontario health budget is $27 Billion.

The Ontario Provincial Government pays for all its hospitals, physicians, nurses, drugs and radiotherapy from its tax income. Cancer Care Ontario (CCO) advises the Provincial Government on spending $2 Billion on Oncology Services in Ontario. The Ontario Cancer Registry compiles data on all patients suffering from malignancy. No consent is required. CCO has access to all Ontario surgical billing and, hence, has the ability to link the billing data base with the Cancer Registry, and other data bases in the Province, provided privacy legislation is not contravened.

Cancer surgery is practiced in a dispersed system throughout Ontario, in non-teaching and teaching hospitals. Neurosurgery is an exception, with concentration of service provision to a limited number of facilities. Radiation of malignant neurosurgical disease is confined to specific centres in the Province and CCO has recently integrated these units with their adjacent general hospitals. Drug therapy for malignancy is provided at a wide array of sites.

This paper will present the Provincial policies underlying provision of neurosurgical treatment of malignancies in virtual regional networks, and will provide detailed numerical information concerning neurosurgical malignancy traffic patterns in a population of 12 million people. These policies and delivery systems will be compared to cancer policies and practices in Germany.

MONDAY, OCTOBER 4 **8:45 – 9:00am**

The Treatment of Advanced Sinonasal Malignancies with Preoperative Intra-arterial Cisplatin and Concurrent Radiation

Jon H. Robertson, LM Michael, S Samant

Malignancies of the nasal and paranasal sinuses are uncommon tumors, accounting for only 3% of all aerodigestive tract neoplasms and less than 1% of all malignant diseases. For many years, the treatment of paranasal carcinomas consisted of surgery via a transfacial approach +/- radiation therapy. Despite significant procedural progress since the 1950's, 5-year survival for sinonasal tumors remains less than 50%. Even less encouraging results are found in more advanced disease (T3 and T4 stage), with 5-year survival being 7% to 29% for T4 cancers. The major cause of death is locoregional failure with a small minority of patients succumbing to distant metastases.

Between July 1996 and August 2003, ele3ven patients with advanced sinonasal malignancies (T3 and T4 stage) underwent treatment utilizing neoadjuvant selective intra-arterial cisplatin with concurrent radiation therapy (RADPLAT) followed by a planned surgical resection via a craniofacial approach at the University of Tennessee Health Science Center in Memphis. Patient charts, operative notes, histopathological studies, follow-up clinic notes, and pre- and post-operative imaging studies were reviewed in detail for each patient.

Histopathological analysis of the tumors revealed 7 squamous cell carcinomas (64%), 2 adenocarcinomas (18%), 1 adenoid cystic carcinoma (9%), and 1 sinonasal undifferentiated carcinoma (9%). There was a clear sex predilection, with a higher incidenceamong males (81%).

T4N0M0 disease was present in 9 patients (81%), and two patients had T3N0M0 disease (19%). Staging was performed using theamerican Joint Committee Classification for Cancer Staging and End Result Reporting (5^{th} Edition, 1997). No patients harbored node-positive disease, as this was felt to be a relative contraindication for surgical resection.

Nine of the eleven patients (81%) included in the study are alive at present and eight (72%) have no evidence of disease. Mean follow-up is 58.7 months ranging from 10 months to 94 months. Only one patient (9%) developed a major complication as a result of radiation necrosis. The only minor complication was related to the chemotherapeutic regimen. Excellent functional status was preserved in all of the patients.

The treatment of advanced sinonasal malignancies with pre-operative intra-arterial cisplatin and concurrent radiation results in significant improvement in survival. This can be done safely with superb cosmetic results, high response rates, and excellent loco-regional control in T3 and T4 disease. Although our results are encouraging, there is a need for a cooperative, multi-institutional, prospective study.

MONDAY, OCTOBER 4 **9:00 – 9:15am**

Surgery of Brainstem Gliomas

Albino Bricolo

Background: For many years gliomas growing inside the brainstem were considered malignant in themselves and managed homogeneously by radiation therapy with disappointing results. In the last two decades a series of favorable concomitant factors made this pathology more accessible for direct surgery. Based on MRI brainstem glioma can now be categorized in four groups (focal, cervico-medullary, dorsally exophytic and diffuse) which reflect different growth patterns and assist in selecting thoseamenable for surgery.

Method and results: Since 1982 we have treated 230 brainstem gliomas (1, 2, 3) by using direct surgery designed for extensive or possibly total excision. The majority (163) were focal and benign astrocytomas (128), whereas almost all the 67 diffuse tumors (92%) were malignant. A gross total removal was achieved in 74% of the 163 focal low-grade gliomas, while in only 4% of the 67 diffuse high-grade gliomas. Overall results were favorable: 85% of the patients with focal low-grade astrocytomas had a good outcome and resumed normal life; 70% of the patients with malignant gliomas died during the follow-up period (mean 7.1 years). The main problem in direct surgery is how to reach and remove the tumor avoiding damage in highly critical places such as corpora quadrigemina, facial colliculi, medial longitudinal fascicules and calamus scriptorius. Suitable safer entry zones for entering the brainstem have been identified and their introduction correlated with a decrease in surgically-related morbidity.

Conclusions: (a) a number of brainstem gliomas are focal masses that dislocate nervous structures without invading them; (b) the brainstem can be surgically violated with low risk by entering it through safer entry zones; and (c) direct microsurgery can obtain complete removal of many brainstem gliomas with good results and even permanent cure.

MONDAY, OCTOBER 4 **9:15 – 9:30am**

Surgical Strategy in Spinal Cord Hemangioblastomas. Experience in 30 Cases

Jacques Brotchi, F Lefranc

The complete removal of spinal cord hemangioblastomas (SCH) must be a neurosurgeon's ultimate goal. We have tried to draw up a simple topographic classification in order to achieve this goal with respect to patients' preoperative neurological status.

We have classified the lesions in three topographical categories:
-A. Subpial posterior and postero-lateral SCH (n=15)
-B. Small subpial lateral or anterior-sided SCH (9 lateral, 1 anterior)
-C. Pure intramedullary SCH (n=5)

Different surgical approaches related to topographic classification were used:

A. Classical "en-bloc" resection with careful dissection of the lesion from spinal cord.

B. A tense syringomyelic cyst, which was present in all the cases, was aspirated with a 22G needle, so deflating the spinal cord and giving an easy access to the solid nodular tumor either when it was lateral or even anterior.

C. When the hemangioblastoma was not visible we opened the posterior *sulcus* along the midline using the same procedure as for any other intramedullary spinal cord tumor.

Complete surgical removal was achieved in all cases except in one previously operated upon twice and irradiated in another institution. Improvement was observed in 18, stabilization in 10 and worsening in 2 patients. The results were closely related to the pre-operative status. Patients with a good pre-operative neurological status or harboring a large cystic cavity had a better prognosis than others.

MONDAY, OCTOBER 4 **9:30 – 9:45am**

Cyclooxygenase 2 (COX-2) Is Ubiquitously Expressed in Meningiomas and Treatment in Vitro with the COX-2 Inhibitor Celecoxib Decreases Cell Growth: Potential Therapeutic Application

BT Ragel, RL Jensen, William T Couldwell

Introduction: Meningiomas are the second most common primary central nervous system tumor in adults. Epidemiologic data suggest a causal relationship with a history of head trauma. Cyclooxygenase-2 (COX-2), an inducible inflammatory enzyme, converts arachidonic acid to prostaglandins, which have angiogenic, anti-apoptotic, and cell proliferative effects. Human meningioma tumors show extensive immunohistochemical staining for COX-2. The authors investigated the effects of celecoxib, a COX-2 inhibitor, on meningioma cell growth in vitro.

Methods: Human meningioma surgical specimens were immunohistochemically stained for COX-2, HIF-1 alpha, and vascular endothelial growth factor (VEGF). Six fields in each tumor were graded from 0 to 4. Human meningoma cells grown in cell culture were treated with vehicle (0.1% DMSO) or celecoxib (0.25mM – 1.00mM). Cell viability assays (MTT), TUNEL apoptosis assays, and FACS analysis were performed.

Results: Eighty-seven percent (111/128) of benign meningiomas and 86% (6/7) of atypical meningiomas had high COX-2 immunoreactivity (grade 4). COX-2 was not correlated with HIF-1alpha or VEGF. Celecoxib inhibited cell growth in a dose-dependent fashion and induced apoptosis by day 2. FACS analysis showed that 74% of treated cells were in G1 cell-cycle phase versus 67% of the control group.

Conclusion: COX-2 is universally expressed in the cytoplasm of meningiomas but not correlated with markers of hypoxia (HIF-1 alpha) or angiogenesis (VEGF). The absence of correlative findings is probably a result of the overall high level of COX-2 staining. Celecoxib inhibits meningioma growth in vitro in a dose-dependent fashion, with evidence of apoptosis and a G1 cell-cycle arrest. COX-2 inhibitors may have a role in the treatment of recurrent meningiomas.

Deficiency in Low-density Lipoprotein Receptor-related Protein (LRP) Expression Favors a More Invasive Glioblastoma Growth Phenotype

M Mut, MD Brown, JE Carpenter, GT Redpath, SR Vanderberg, M-B S Lopez, IM Hussaini, Mark E Shaffrey

Low-density lipoprotein receptor–related protein (LRP) mediates the endocytosis of a wide variety of ligands that are upregulated with anaplasia (including urokinase and urokinase-type plasminogen activator receptors) and is implicated in cellular growth, adhesion, and proteinase catabolism. Epidermal growth factor treatment in astrocytomas significantly reduces total and cell surface LRP. Functional effects of LRP elimination and overexpression in U-1242 human glioblastoma multiforme cells were assessed. LRP-deficient GBM cells (Lα-42) were prepared from U-1242, which were transfected with the antisense constructs. LRP in Lα-42 was completely eliminated (Western blot). LRP-expressing U-1242 cells were transfected with the LRP gene (Lδ-32 clones) and 3-4 times greater expression of LRP was confirmed (Western blot). Non-transfected U-1242 cells (ntU-1242) have intermediate LRP expression. In vitro, Lα-42 invaded fibrin-coated transwell membranes more than ntU-1242, and urokinase neutralizing antibody reduced the invasiveness of the Lα-42 to control levels. Gelfoam® three-dimension cultures of Lα-42, ntU-1242 and Lδ-32 clones were implanted in the brains of mice (n=30) and tumor growth were assessed at 5 and 9 weeks. At 5 weeks, Lα-42 presented a faster growth pattern, ntU-1242 cells showed an intermediate growth pattern without apparent invasion. Lδ-32 did not have apparent tumor growth. At 9 weeks, Lα-42 showed a diffusely invasive pattern with comparably larger tumors. LRP expressing clones (ntU-1242 and Lδ-32) were also found to be invasive, but showed only focal invasion with “pushing” borders and more prominent surrounding gliosis. These results suggest that LRP expression may be critical in determining the invasive growth phenotype of glioblastomas.

MONDAY, OCTOBER 4 **11:00 – 11:15am**

Mechanisms of Cortical Reorganization: An Experimental Study

Charles Hodge, A Siddiqui, J Dubrof, R Stevens, J McCasland

Reorganization of cortex after injury is an important and under-studied aspect of trauma and ischemia. Understanding the phenomenon of cortical plasticity will allow manipulation of these mechanisms for patient benefit. We used the rodent sensory cortex representing the contralateral snout whiskers in this study. Small excitotoxic lesions were made in a single whisker representation and the reorganization that ensued studied using determination of somatosensory cortical maps using optical imaging techniques and a variety of anatomic techniques.

We present evidence indicating sprouting and alterations in excitatory/ inhibitory balance occur as concomitants of cortical plasticity. Retrograde transport of fluoro ruby was used to demonstrate that, after a small cortical lesion, there is dramatic increase in apparent afferent input to the damaged area. There was nearly a two fold increase in input to the reorganizing area from adjacent cortex, the thalamus, the second somatosensory area and the homologous sensory cortex. Additional evidence of sprouting was an increase in GAP-43 and BDNF, both associated with neurite outgrowth. Microarray analysis indicates that there is up regulation of these compounds as well as change in the balance of inhibitory and excitatory transmitters. The excitatory/inhibitory balance change is distinctly different when plasticity is induced by peripheral perturbation as opposed to small central lesions.

Demonstration of sprouting and alterations in transmitter and receptor messages point to the dynamic responses that result from even small cortical lesions. Understanding how to maximize these responses through pharmacologic means might allow better recovery of our patients from a variety of nervous system injuries.

MONDAY, OCTOBER 4 **11:15 – 11:30am**

Avoiding Mount Everest – Assessing the Risks of Cerebral Aneurysms and Their Treatment

Eugene S Flamm

Case reports, personal experience, institutional series, national and international studies all provide data, often conflicting and non-intuitive, about when, if and how to manage cerebral aneurysms. With improved screening methods and several alternative methods of therapy available, it is important to determine not only the methods to be used but what the risk-benefit ratios for a given case. We must avoid the Mount Everest syndrome of treating an aneurysm simply because it is there.

Based on personal experience with over 1700 treated cerebral aneurysms, the author has used several methods to analyze the risks of treatment and how to apply these outcome measures on a case by case basis. While this approach cannot completely resolve the question, it is helpful to define the risks, determine the factors that contribute to the risk, and incorporate these data into an algorhythm on which to base therapeutic recommendations.

We have used epidemiological techniques such as comparisons with large studies of demographics and natural history as well as analyses of outcome measures such as Quality Adjusted Life Years (QALYs). While helpful in determining generalized trends, these methods are difficult to apply on a case by case basis. More recently we have utilized a Factor Accumulation Index (FAI), the sum of 7 different factors observed in a given patient to determine potential risk factors that might adversely affect to outcome in patients with unruptured aneurysms. In patients with more that 3 of these factors, an unexpected neurological deficit was observed. Finally we have developed an algorhythm that takes into account the age of the patient and known natural history factors as well as the anticipated treatment risks based on location, size and experience with the site specific aneurysms. These techniques seem applicable to retrospective data and are currently being applied prospectively to determine their true utility.

MONDAY, OCTOBER 4 **11:30 – 11:45am**

Unilateral Intraputamenal GDNF Improves Bilateral Motor Functions in Patients with Parkinson's Disease

JT Slevin, GA Gerhardt, CD Smith, DM Gash, R Kryscio, A Byron Young

Objective: Glial cell line-derived neurotrophic factor (GDNF) has significant antiparkinsonian actions in several animal models of Parkinson's disease (PD) and in a recent pilot study in five patients in England, of which four received bilateral putamenal delivery. Here, we present results from unilateral intraputamenal GDNF infusion in six advanced PD patients receiving a dose-escalation regimen of GDNF for six months.

Methods: PD patients were evaluated in a functionally defined OFF and ON state at -1, +1 and +4 weeks after intraputamenal catheter implantation contralateral to the most affected side. Subjects then received a dose-escalation regimen of GDNF: 3 mcg/day, 10 mcg/day and 30 mcg/day at successive eight week intervals, followed by a five-week washout period.

Results: Total UPDRS OFF and ON scores were significantly improved (34% and 29%, respectively) at 24 weeks (OFF = 47 +/- 7; ON = 36 +/- 6) compared to baseline (OFF = 72 ± 7; ON = 51 ± 5). Measures using CAPSIT-PD criteria showed significant bilateral improvements (40-50%) in fine motor control and speed. The bilateral effects included improved postural stability and gait. All motoric improvements continued through washout. The only side effect observed was transient Lhermitte's symptoms in 2 subjects.

Conclusions: This open label study of unilateral intraputamenal GDNF demonstrates safety of the procedure and its potential efficacy. There were significant sustained bilateral effects from unilateral administration of the protein.

Human Dorsal Root Ganglion Neurons as Live Nerve Constructs: Implications for Transplantation

Eric L Zager, JH Huang, EM Wilensky, BJ Pfister, DH Smith

Introduction: We sought a clinically applicable source of neurons that could be engineered into transplantable nerve constructs for neural repair. We chose dorsal root ganglion (DRG) neurons due to both their accessibility and their robustness in culture. These neurons can be harvested from a patient for reimplantation as an autograft or from organ donors to serve as allografts.

Methods: Human DRGs were harvested from patients during C2 ganglionectomy for intractable occipital neuralgia. Additionally, we harvested DRG neurons from thoracic ganglia of organ donor patients within 2 hours post-aortic clamping. Harvested ganglion cells were dissociated and cultured on two adjacent membranes in a bio-elongator. We then used a microstepper motor system to progressively separate the two membranes, elongating the axons at a maximal rate of 2 mm/day.

Results: These cultures were maintained for up to three months and surviving DRG neurons were positively identified using antibodies NF 100, SMI-254, SMI-32 and CGRP (calcitonin-gene related peptide). In the bio-elongator, bundles of axons grew across the membranes, integrating with the DRGs on the other side. By 10 days of stretch, axons spanning the two membranes had grown to 10mm in length, creating a living nerve construct. These constructs were implanted in a rat sciatic nerve injury model and a spinal cord injury model. Preliminary results of behavioral, histological, and electrophysiological studies will be presented.

Conclusions: The viability of adult human DRG neurons coupled with their capacity to grow under continuous mechanical tension underscores their promise as a novel means of repairing spinal cord and nerve injuries.

Supported by NIH grants NS46170-01 (JHH), AG 21527(DHS) and NS38104 (DHS), and an AANS Codman Award (JHH).

NOTES:

TUESDAY, OCTOBER 5 **2:45 – 3:00pm**

Cortical and Striatal Migration of Endogenous Adult Stem Cells from the Sub-ventricular Zone and Posterior Peri-ventricular Region in Response to Stroke.

Robert J Dempsey, KA Sailor

The proliferation of endogenous adult stem cells represents a potential therapy for repair after a stroke. Adult neurogenesis is a phenomenon that occurs in the subventricular zone (SVZ) at the lining of the lateral ventricles and in the dentate gyrus (DG) of the hippocampus, and has been shown to be stimulated by various interventions including stroke. Migration of these cells is necessary for them to repopulate a region of stroke damage. Normally the adult stem cells located in the SVZ are only seen migrating anteriorly to the olfactory bulbs where they differentiate into olfactory interneurons. After 1 hour of focal cerebral ischemia in rats, and at 2, 4, 6 and 8 days of reperfusion, we observed these cells also migrating laterally, extending their axons in their path of migration toward the infarct, into the striatum. These cells were labeled with the immature migrating neuronal marker doublecortin (DCX) and the extent of the migration was correlated with theamount of injury to the striatum. In addition to this, we have observed cells in the posterior peri-ventricular region (pPV), a posterior extension of the SVZ, migrating directly to the ipsilateral cortex. By imaging these regions at two-day time intervals we were able to observe an abrupt increase in proliferation in the ipsilateral pPV at day 2 after ischemia, with localized migration along the corpus callosum. By days 8 and 12, we observed DCX positive cells that had migrated laterally into the ischemic penumbra with their axons extending into the ischemic core. This study demonstrates that there is an endogenous response to injury by these cells. By understanding the factors that attract such cells, future therapies may enhance this migration for possible restoration of function to the damaged region of the brain.

Genetic Variants of Matrix Metalloproteinase Genes and their Inhibitors in Patients with Intracranial Aneurysms

Dietmar Krex, IR König, A Ziegler, HK Schackert, G Schackert

Department of Neurosurgery (D.K., K.K., G.S.) and Department of Surgical Research (H.K.S.), University Hospital Carl Gustav Carus, University of Technology, Dresden, and Institute of Medical Biometry and Statistics (I.R.K., A.Z.), University at Lübeck, Germany

Objective: There are several lines of evidence that remodeling of the extracellular matrix is a crucial event in the pathogenesis of cerebral aneurysms. Matrix-metalloproteinases (MMPs) are the most important degrading enzymes in the extracellular matrix. Their activity is predominantly controlled by tissue inhibitors of metalloproteinases (TIMPs). To investigate the possible impact of genetic variants within the genes encoding *TIMP-1, -2*, and *-3*, and *MMP-2, -3, -9*, and *-14*, respectively, we conducted a case-control study.

Methods: The entire coding regions and parts of the promoter sequences of the referring genes were investigated using the automated laser fluorescence (A.L.F.) technique. Genotypes and allele frequencies were determined in a primary study sample comprising 44 well defined aneurysm patients and 40 controls. Those being in Hardy-Weinberg disequilibrium were analyzed in another sample of 40 cases and 40 controls, respectively. Odds ratios (ORs) and exact 95% confidence intervals (CIs) were calculated to compare allele frequencies and genotype frequencies using the Cochrane-Armitage trend test.

Results: A total of 38 single nucleotide polymorphism (SNPs) were identified; 3, 4, and 2 in *TIMP-1*, *-2*, and *-3* genes, and 10, 4, 11, and 4 in *MMP-2, -3, -9*, and *-14* genes, respectively. SNPs -621C>T, -596A>C, -261G>A of the *TIMP-2* gene were new identified polymorphisms. Deviations from Hardy-Weinberg equilibrium were particularly found for *MMP-2* and *MMP-9* SNPs, however, there were no significant differences in genotype and allele frequencies in those or between any of the other groups.

Conclusions: Our analysis of the entire coding region of three *TIMPs* and four *MMPs*, which are main contributors to extracellular matrix remodeling in vessel walls, failed to show an association of genetic polymorphisms with an intracranial aneurysm. However, deviations from Hardy Weinberg equilibrium found for *MMP-2*, and *MMP-9*, but also for *MMP-3*, and *TIMP-1* SNPs suggest that there might be additional more distantly located genetic variants of functional impact, which are the subject of ongoing studies.

Downregulation of Potassium Channels After Subarachnoid Hemorrhage: A Possible Mechanism for Cerebral Vasospasm

BS Jahromi, Y Aihara, G Weyer, E Nikitina, M Agbaje-Williams, D Ryan, R Yassari, Robert Loch Macdonald

Cerebral vasospasm remains a significant adverse prognostic factor for outcome after subarachnoid hemorrhage (SAH) and is a significant cause of morbidity and mortality in such patients. The pathogenesis remains ill-defined but it is clear that the narrowing is primarily due to abnormally sustained contraction of arterial smooth muscle cells. Cerebrovascular arterial constriction is regulated by membrane potential which in turn is determined by potassium conductance.

These studies examined expression of the major potassium channels by real time polymerase chain reaction and Western blotting and their function using whole cell and perforated patch clamp electrophysiology in normal dog basilar artery smooth muscle cells and in these cells obtained during vasospasm 7 days after SAH.

There were no significant changes in messenger ribonucleic acid and protein of the large-conductance, calcium-activated potassium channel (BK, alpha and beta subunits). There was significant downregulation of Kv2 class of voltage-gated potassium channels. Furthermore, of the two dominant potassium conductances in cerebrovascular smooth muscle cells, BK currents were unaffected by SAH whilst Kv2 class currents were nearly halved after SAH. Immunohistochemistry confirmed localization of Kv 2 (Kv2.1, 2.2) class channels to normal dog basilar smooth muscle and showed that immunoreactivity was reduced after SAH. Vasospastic myocytes were depolarized and showed decreased contribution of potassium conductance towards maintenance of membrane potential. Pharmacological block of Kv but not BK channels in control myocytes mimicked the depolarization observed in vasospastic myocytes and contracted basilar artery rings.

We propose that decreased Kv channel function contributes to the pathogenesis of cerebral vasospasm after SAH.

Prospective Evaluation of Surgical Microscope Based Indocyanine Green Video Angiography in Aneurysm Surgery

Andreas Raabe, P Nakaji, J Beck, J Kamerman, V Seifert, RF Spetzler[1]

Department of Neurosurgery, Neurocenter, Johann Wolfgang Goethe University Frankfurtam Main, Frankfurtam Main, Germany and [1]Division of Neurological Surgery, Barrow Neurological Institute, St. Joseph's Hospital and Medical Center, Phoenix, Arizona

Objective: We report about the final results of a prospective trial comparing a new technique of surgical microscope based indocyanine green video angiography (ICGA) with intraoperative or postoperative DSA.

Method: ICGA was performed during aneurysm surgery using a newly developed setup with the Zeiss microscope (Zeiss, Oberkochen, Germany). A microscope integrated light source containing infrared excitation light illuminates the operating field. After intravenous injection of the indocyanine dye (25 mg), intravascular fluorescence of ICG from within vessels is imaged by a microscope video camera. Patency of parent, branching and perforating arteries and clip occlusion of the aneurysm as found by ICGA were compared to intraoperative or postoperative DSA findings. The study is expected to close in May after inclusion of 100 patients.

Results: After including 90% of cases, 153 ICGA procedures were performed. The results of ICGA were concordant with postoperative DSA in 36 of 38 cases. One case of mild stenosis and one case of a 4 mm residual aneurysm were missed by ICGA. Compared with intraoperative DSA, the results of ICGA were concordant in 36 of 41 cases. In 4 cases a mild stenosis and in one case a severe stenosis was missed by ICGA. All false negative ICG findings were inconsequential. ICGA led to clip intraoperative clip correction in 7% of cases. None of the cases had clip correction after intraoperative or postoperative DSA when performed after ICGA. The final results will be reported at the meeting.

Conclusions: Microscope based ICGA is simple and provides real-time information about the patency of arterial and venous vessels of all relevant diameters and about the aneurysm sac. ICGA may fill the gap between intraoperative DSA and intraoperative Doppler. It may be used as an alternative to intraoperative DSA in many cases.

TUESDAY, OCTOBER 5 **3:45 – 4:00pm**

Multivariate Analysis of a Consecutive Prospective Series of Carotid Endarterectomies

Robert E Harbaugh, A Agarwal, V Chinchilli

Objective**:** We performed a multivariate analysis to determine predictors of adverse clinical outcomes following carotid endarterectomy (CEA). Numerous variables, including gender, age, medical comorbidities, contralateral internal carotid artery occlusion, choice of anesthetic, presentation and resident or faculty surgeon were evaluated in regard to the incidence of neurological and non-neurological perioperative complications.

Methods: A prospective series of nearly 1200 CEAs were analyzed. All patients were operated upon under the direction of one neurosurgeon (REH). Clinical outcomes measures evaluated were any stroke, death or myocardial infarction within 30 days of operation. All patients were followed to a clinical endpoint and/or six weeks postoperatively. All outcomes were adjudicated by third party observers.

Results: The ipsilateral stroke rate within 30 days of CEA was 2.25%. None of the commonly cited risk factors such as advanced age, medical co-morbidities or contralateral carotid occlusion were associated with a significantly ($P<0.05$) increased risk of perioperative morbidity or mortality. The use of regional anesthesia was associated with a significantly decreased risk of non-neurological perioperative complications. Other details of the multivariate analysis will be presented.

Conclusion: CEA using regional anesthesia can be performed in patients with advanced age, diabetes mellitus, atherosclerotic coronary vascular disease and contralateral ICA occlusion with acceptably low perioperative morbidity.

A Phase IIa, Multicenter, Double-blind, Placebo-controlled, Randomized Study to Assess the Safety and Tolerability of the Selective Endothelin A (ETA) Receptor Antagonist Clazosentan (AXV-034343) for the Prevention of Cerebral Vasospasm, Following Severe Aneurysmal Subarachnoid Hemorrhage (aSAH)

Bernhard Meyer for the Axovan Study Group

Department of Neurosurgery, University of Bonn, Germany

Objective: To assess whether the endothelin A (ETA) receptor antagonist clazosentan can reduce the incidence and severity of angiographic vasospasm on Day 9 post rupture (primary endpoint) in a selected high-risk population of patients with aSAH (Fisher Grade ≥ 3 / Hunt & Hess Grade III to IV) compared to placebo.

Methods: The study was divided into a double-blind Part A (randomized continuous iv. Infusion of clazosentan 0.2 mg/kg/h versus placebo starting within 48h post aSAH up to Day 14 post rupture) and an open-label Part B (clazosentan 0.4 mg/kg/h for 12 h followed by 0.2 mg/kg/h up to Day 14 for patients with persistent vasospasm as documented by DSA on Day 9). The primary endpoint was determined in comparison to baseline-DSA via central evaluation, secondary variables included daily blood flow velocities by TCD, incidence of infarcts on CT and outcome according to NIH-SS/mGCS score on Day 14. 34 patients were recruited over a 7-month period in 5 centers (safety population: 16 clazosentan, 18 placebo, analyzed post-hoc according to ITT). 32 patients remained in the intent-to-treat A (ITT-A) and per protocol (PP) population (15 clazosentan, 17 placebo), 19 patients entered Part B and were analyzed according to ITT (ITT-B; 7 clazosentan, 12 post-placebo). Two-sided Fisher's exact test was used to compare the incidence of vasospasm. Differences in severity of vasospasm were tested with the Wilcoxon test.

Results: The ITT-A population showed a significant reduction in the occurrence and severity of angiographic vasospasm in the clazosentan group ($p=0.008$, relative risk reduction: 55%; from 88.2% to 40.0% and odds ratio of 0.089). Even after conservative analysis (i.e., counting deaths and cerebral vasospasms during Part B of the study as treatment failures in the ITT post-hoc population), there was still a trend towards a lower incidence ($p=0.052$) and severity ($p=0.061$) of vasospasm (relative risk reduction of 37%; from 88.9% to 56.3%). A lower incidence of new infarcts was observed in patients randomized to

clazosentan group (15.4% vs. 43.8%) and TCD measurements yielded lower median Lindegaard indices for patients randomized to clazosentan suggesting a milder form of vasospasm.

Conclusions: Clazosentan showed a statistically significant reduction in the frequency and severity of vasospasm compared with placebo. When subsequent analysis incorporating imputation rules for missing data was performed there was still a statistical trend in favor of patients treated with clazosentan. The incidence/severity of adverse events observed with clazosentan was comparable to placebo.

TUESDAY, OCTOBER 5 **4:15 – 4:30pm**

Endovascular Management of Intracranial Aneurysms: An Experience of 1321 Aneuryms in 1101 Patients: Single Center Experience

Robert H Rosenwasser, E Veznedaroglu, S Whang

Introduction/Purpose: Endovascular management of intracranial aneurysms has become an option in certain patient populations based on age, neurologic condition, and anatomical location.

Clinical Materials/Methods: At TJUH from July 1994 through December, 2003, 2294 patients were treated for intracranial aneurysms. Age range was 9 – 89 years. Out of this group, 1192 patients (52%) underwent transcranial surgery and 1101 patients (48%) underwent Endovascular treatment. Eight hundred and sixty-nine patients (78%) presented with SAH. Patient selection for endovascular occlusion was based on physiologic age, Neurologic grade, and anatomy. Patients were treated under general anesthesia with neurophysiologic monitoring and full heparinization.

Results: A technical failure rate of 16% was noted and 175 patients underwent transcranial surgery when the lesion could not be endovascularly treated. The rate of intraprocedural rupture was 1.4%. The rate of embolic events were 2.9% acutely (intraprocedural) and 5.9% delayed. Mortality related to the procedure was 2.5%. The complete occlusion rate was 64% in the first 5 years and was 205 in the last 4 years. To date 4.7% of patients have required retreatment due to recurrence and longer followup is demonstrating that this number is increasing. Thirty-six percent were G1,2 and had a good/fair outcome (GOS), 64% were G3, 4; 87% good/fair; 6% fair; 7% dead; G4; 28% fair, 52% poor, 20% dead. No patient was treated as a G5.

Conclusions: Endovascular occlusion of intracranial aneurysms is a reasonable alternate to microsurgical intervention, particularly in elderly patients and patients in poor neurological grade. Incomplete occlusion and durability remain problematic.

TUESDAY, OCTOBER 5 **4:45 – 5:00pm**

Susceptibility of Murine Neural Stem Cells and of Tumor Cells to Respond to Regenerative Signals of Axolotl Tissue.

Matthias Kirsch, E Schnapp, S Lydia, E Tanaka, P Weigel, G Schackert, HK Schackert

Neurosurgery, Carl Gustav Carus Universitätsklinikum, Technische Universität Dresden and Max-Planck-Institute for Cell Biologie and Genetics, Dresden

Objective: Regeneration of the nervous system in humans and other vertebrates is not sufficient to allow functional recovery. In contrast, urodele and axolotlamphibians are able to regrow organs, such as the limbs and the tail including the peripheral nerves and spinal cord representing central nervous system tissue. Several reports indicate that axolotl do not reject foreign tissue. In-vitro fusion of axolotl with murine cells were successful. The purpose of this study was to evaluate the ability of regenerative axolotl tissue to recruit transplanted vertebrate (mouse) cells to follow the regenerative process.

Methods: Cross-species xenotransplantation was evaluated, both the transplantation of murine neural progenitor cell precursors and of murine brain tumor cells into the regenerating tail of an axolotl and the transplantation of axolotl cell spheroids, spinal cord, or regenerating tail tissue into the chronic cranial window of nude mice.

Results: Temperature-adapted cells and tissues were transplanted. The regenerating axolotl blastema was invaded by murine neural progenitor cells which remained within the growth cone. Three days post implantation, no living murine cells were observed. In the cranial window of mice, axolotl spinal cord and blastema pieces, as well as spheroids from cultured myotubes were implanted. None of the transplants showed invasion of single cells into the surrounding parenchyma. A steadily increasing number of cells were observed that underwent apoptotic cell death. On day four post-implantation, no viable axolotl cells remained in the murine environment. However, in vitro confrontation studies using spheroids and blastema tissue, revealed a shift of the vertebrate cells to a regenerative phenotype.

Conclusion: Xenotransplantation of axolotl cells into nude mice and murine pluripotent neural progenitor cells into the axoltol blastema did not induce a regenerating phenotype. In vitro experiments confronting tumor tissue and blastema revealed a phenotypic change revealing an increase of precursor markers.

TUESDAY, OCTOBER 5 **5:00 – 5:15pm**

Local Delivery of a Fragment of Human Endostatin Combined with Systemic Administration of BCNU Prolongs Survival in a Rodent Experimental Glioma Model

Alessandro Olivi, FG Legnani, G Pradilla, BM Tyler, F Chillemi, F Di Meco

Introduction: Endostatin is an anti-angiogenic agent currently tested for several solid malignancies. A fragment of endostatin (43amino acids), easily synthesized with high stability and water solubility was incorporated into controlled-release polymers and tested to determine its efficacy when combined with conventional chemotherapy for treatment of experimental gliomas.

Methods: Cytotoxicity of the endostatin fragment against 9L gliosarcoma, and F98 glioma was determined *in vitro*. The fragment was incorporated into polyanhydride-poly-[bis-(carboxiphenoxy-propane)-sebacic-acid] (pCPP:SA) at different loading concentrations and its pharmacokinetics were analyzed *in vitro*. The rat cornea micropocket assay was used to evaluate the anti-angiogenic activity of the fragment/polymer formulations. Systemic toxicity and efficacy of locally delivered endostatin fragment/polymers combined with systemic BCNU were determined in Fischer 344 rats(n=64) using the intracranial 9L gliosarcoma model.

Results: Endostatin fragment showed significant cytotoxicity *in vitro* against 9L and F98. Comparable sustained release was seen by day 19 in all polymer formulations. Corneas implanted with 40% endostatin fragment/pCPP-SA polymers had a mean angiogenesis index of 4.5±0.7 when compared to 8.5±1.3 in controls ($p=0.01$). Survival was prolonged in animals treated with the combination of intracranial 40% endostatin fragment/pCPP:SA polymers and systemic BCNU (mean survival of 120 days) when compared to controls (mean survival 11 days, $p<0.001$, with 33% long term survivors).

Conclusions: Controlled release polymers can effectively deliver a biologically active fragment of endostatin in a sustained fashion. This novel endostatin fragment inhibits angiogenesis *in vitro* and *in vivo*, and exhibits a synergistic effect when combined with systemically delivered with BCNU in the intracranial 9L gliosarcoma model.

TUESDAY, OCTOBER 5 **5:15 – 5:30pm**

Expression of Hypoxia-inducible Carbonic Anhydrases in Brain Tumors

Martin Proescholdt, C Mayer, M Merrill, A Brawanski

Introduction: Malignant Gliomas are the most frequent intrinsic brain tumors in adults. Metabolically, these tumors display a distinct pattern: First, they show a high glucose utilization rate combined with significant levels of lactate production, probably due to aerobic glykolysis. Surprisingly, the intracellular pH of malignant brain tumors has been found to be significantly more alkaline compared to normal brain, whereas the extracellular space displays an acidic pH as demonstrated by *in vivo* studies using pH microelectrodes. Secondly, malignant gliomas display large areas of intratumoral hypoxia as shown *in vivo* by polarographic measurements as well as by PET studies using ^{18}F-misonidazole as hypoxia specific tracer. Carbonic anhydrase (CA) IX and XII are transmembrane isoenzymes which are induced by tissue hypoxia. They participate in the regulation of pH homeostasis by catalyzing the reversible hydration of carbon dioxide. We hypothesize, that CA IX and XII may be overexpressed in malignant brain tumors due to intratumoral hypoxia. Induction of these enzymes in brain tumors might contribute to an aggressive phenotype by improved acid clearance. The aim of our study was to investigate, whether brain tumors of different histology and grade of malignancy express elevated levels of CA IX and XII compared to normal brain.

Methods: We analyzed 112 fresh frozen tissue samples from primary and metastatic brain tumors for CA IX and XII expression by immunohistochemistry. 8 brain tissue samples obtained from patients who died from extracerebral disease were used as normal brain control. The staining was semiquantitatively graded by three independent investigators blinded to the underlying histology. To confirm the immunostaining results, Western Blot analysis was performed from total protein extract of the tumor tissue. The CA IX and XII mRNA expression was investigated by *in situ* hybridization. To correlate CA IX and XII with tissue hypoxia, immunohistochemical staining for hypoxia inducible factor (HIF-1□) was performed on adjacent sections.

Results: While normal brain tissue showed minimal CA IX and XII expression, we found a profound upregulation of both enzymes in the brain tumor tissue both by immunohistochemistry and Western blot. The levels of CA IX and XII induction correlated with the grade of malignancy. CA IX and XII mRNA levels were elevated particularly around areas of micronecrosis. Accordingly, the highest levels of CA IX and XII protein expression was found to be in

perinecrotic pseudopallisading cells indicating hypoxic induction. Also, comparison of CA IX and XII staining and HIF-1□ staining revealed a similar microanatomical distribution, confirming hypoxia as the main factor of induction.

Conclusion: The results demonstrate that CA IX and XII are upregulated in intrinsic and metastatic brain tumors compared to normal brain tissue. Hypoxia seems to be the major mechanism of induction highlighting the importance of hypoxia as a pathogenetic factor in brain tumors. This may contribute to maintain the pH gradient with alkaline intracellular and acidic extracellular pH observed in brain tumors. Since powerful inhibitors of carbonic anhydrases are clinically available, CA IX and XII may be an important target for future treatment of brain tumors.

TUESDAY, OCTOBER 5 **5:30 – 5:45pm**

Anti-EGFR Monoclonal Antibody Cetuximab Augments Radiation and Chemotherapeutic Effects in Glioblastoma Multiforme *in vitro* and *in vivo*.

JL Eller, SL Longo, MM Kyle, D Bassano, DJ Hicklin, Gregory W Canute

Objective: We previously demonstrated that anti-EGFR monoclonal antibody Cetuximab was effective against EGFR-amplified glioblastoma multiforme (GBM) cells *in vitro* and *in vivo*. This work examines the combination of Cetuximab with radiation or chemotherapy.

Methods: Cells were implanted intracranially to verify whether Cetuximab crosses the blood brain barrier. To determine the efficacy of Cetuximab on increasing tumor burden, mice with different size flank GBM were used. Finally, mice with flank GBM were exposed to Cetuximab alone, or in combination with radiation. EGFR-amplified GBM lines were also exposed to Cetuximab and chemotherapeutic agents *in vitro*.

Results: Cetuximab treated-mice had a significant increase in median survival when compared to sham-treated mice for both intracranial and flank tumor models. The inhibition of tumor growth obtained with Cetuximab in mice with flank GBM was inversely proportional to tumor size at the beginning of treatment. Tumor cells undergoing apoptosis was greater upon exposure to Cetuximab and radiation than with either treatment alone. The combination of Cetuximab and radiation treatment increased median survival in mice with flank GBM compared to either treatment alone. The combination of Cetuximab and chemotherapeutic agents increased cytotoxicity *in vitro* when compared to either treatment alone.

Conclusions: The effectiveness of Cetuximab as a solo agent is dependent upon tumor burden. Cetuximab was effective when administered systemically for mice harboring intracranial GBM. Cetuximab plus radiation was more effective against EGFR-amplified GBM than either treatment alone. Cetuximab in combination with chemotherapeutic agents had an additive effect *in vitro*. These results confirm EGFR blockade as a potential chemotherapeutic treatment against human GBM.

TUESDAY, OCTOBER 5 **5:45 – 6:00pm**

Multicentric Phase III Study, Fluorescence-guided Resection of Malignant Gliomas with 5-ALA: Preliminary Results on Interim Analysis of 270 Assessable Patients

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Objective: 5-Aminolevulinic acid (5-ALA) leads to the accumulation of fluorescent porphyrins in malignant gliomas, a phenomenon under investigation for the enhancement of tumor resection. In order to determine patient benefit in terms of resection radicality, progression-free survival and morbidity, a pivotal two-armed, randomized, group-sequential, blinded-observer trial was designed together with the sponsor Medac. This paper gives preliminary results of the interim analysis on the first 270 patients in the full analysis set (intent-to-treat principle population).

Methods: Patients with suspected malignant gliomas bearing the potential for complete resection of contrast-enhancing tumor were randomized to receive either 20 mg 5-ALA/kg body weight for fluorescence-guided resection (ALA-group) or nothing for conventional microsurgery (white light group). Surgery was performed using identical microscopes (Zeiss OPMI Neuro Fl) available to each participating center. Primary study aim was to determine the number of patients without contrast-enhancing tumor on early postoperative MRI and progression free survival at six months.

Results: Postoperative MRI was devoid of residual, contrast-enhancing tumor in 65 % of patients in the ALA group compared to 36 % in the white light group ($p < 0.001$). Preliminary Kaplan-Meier analyses revealed prolonged progression-free survival in ALA patients ($p < 0.01$ compared to white light) with cumulative 6-months progression-free survival rates of 41% and 21%, respectively. Median survival was significantly prolonged in patients devoid of contrast-enhancing tumor (16 vs. 12 months, $p < 0.001$). Postoperative neurological status and Karnofsky Index did not differ between ALA or white light patients.

Conclusions: Fluorescence-guided resections using 5-ALA allow a larger number of complete resections without endangering patients. Complete resections have a direct impact on overall survival.

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TUESDAY, OCTOBER 5 **6:00 – 6:15pm**

Is Gross Total Resection Sufficient Treatment for Posterior Fossa Ependymoma?

L Rogers, J Pueschel, Robert Spetzler, W Shapiro, S Coons, T Thomas, B Speiser

All newly diagnosed intracranial ependymoma patients (n=45) treated at our institution from 1983 to 2002 were identified to determine whether gross total resection (GTR) alone serves as definitive treatment and to evaluate the role of radiation therapy (RT) after either GTR or subtotal resection (STR). Patients with supratentorial primaries, subependymomas, or neuraxis dissemination were excluded. GTR was accomplished in 32 (71%) and STR in 13 (29%). RT was delivered to 25 patients: 13 after GTR and 12 after STR. RT fields were craniospinal followed by a posterior fossa boost in 6, and posterior fossa or local only in 19. With a median follow-up of 66 months, the median local control (LC) was 73.5 months with GTR alone, but has not yet been reached for GTR+RT patients. Median LC after STR+RT was 79.6 months. Ten-year actuarial LC (10y-LC) was 100% for GTR+RT, 50% for GTR alone, and 36% STR+RT, representing significant differences between GTR+RT and GTR alone ($p=0.018$) and between GTR+RT versus STR+RT ($p=0.003$). There was no significant difference in 10y-LC between GTR alone and STR+RT ($p=0.370$). The 10-year overall survival rate of GTR+RT was 83%, 67% for GTR alone, and 43% for STR+RT. RT, tumor grade, and resection extent were significant predictors of LC. GTR should be the intent of surgery when feasible with acceptable morbidity. Even after GTR confirmed with postoperative imaging, adjuvant RT significantly improved LC. We recommend postoperative RT after both gross total or subtotal resection.

Management of Optic Nerve Sheath Meningiomas

U Schick, Werner Hassler

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Object: The management of optic nerve sheath meningiomas (ONSM) remains controversial, but includes surgery, radiotherapy and plain observation. We introduce a new classification scheme and derive treatment modalities from the different types and subtypes.

Methods: A retrospective analysis was performed on 73 patients with optic nerve sheath meningiomas who underwent surgery between 1991 and 2002. The follow-up period ranged from between 6 to 144 months (mean 45.4 mo). Our classification system differentiates between intraorbital (type 1), intracanalicular or intrafissural (type 2), and intraorbital and intracranial (type 3) types of ONSMs. 32 tumors demonstrated extension through the optic canal (type 2a). 29 further tumors reached the chiasm (type 3a) or contralateral side (type 3b). Intraorbital flat tumors (type 1a) should be radiated with visual decline. Type 1b tumors with a large intraorbital mass should be observed, radiated with visual decline, and operated on without useful vision. Only type 1c tumors with exophytic tumor growth should undergo excision. Type 2 tumors areamenable to decompression to save vision and type 3 tumors areamenable to resection of the intracranial part. The intraorbital part should be radiated. The visual acuity was not significantly influenced by surgery. Visual acuity became worse with longer duration of preoperative symptoms and longer follow-up period. Location in the optic canal was another negative factor. Radiotherapy could preserve vision in 5 out of 10 cases.

Conclusions: Loss of vision in optic nerve sheath meningiomas is a question of time. The role of radiotherapy has to be reevaluated and offered to adults once mild vision loss develops in intraorbital ONSMs. Surgery with decompression of the optic canal and intracranial tumor resection is favored for tumors with intracanalicular and intracranial extension.

TUESDAY, OCTOBER 5 **6:30 – 6:45pm**

The Role of Endogenous Growth Hormone-Releasing Hormone (GHRH) in Acromegaly

William F Chandler, EV Dimaraki, AL Barkan, MB Brown, V Padmanabhan, SY Kim, R Taussig

There is indirect evidence suggesting hypothalamic control of GH secretion in acromegaly. To examine whether GH secretion in acromegaly is dependent on GHRH, we studied 8 patients with classic untreated acromegaly due to a GH producing pituitary tumor. All patients were admitted to the General Clinical Research Center and underwent an intravenous infusion of normal saline for 24 hours and GHRH-antagonist at 50 μg/kg/h for 7 days. GH was measured every 10 min for 24h during the normal saline infusion and on the last day of the GHRH- antagonist infusion. A group of 9 different patients with untreated acromegaly served as the control group and underwent blood sampling for GH every 10 min for two consecutive 24h periods to assess the day-to-day variability of GH secretion. All 8 study patients underwent transsphenoidal surgery and RNA was extracted from the each tissue specimen and tested for *gsp* mutations.

In 6 of 8 experimental subjects 24h mean GH decreased by 5.8-30.0 % during GHRH-antagonist infusion and in 3 of 8 subjects the decline of the 24h mean GH was greater than twice the average Coefficient of Variation in the control group. The probability of this magnitude of change to occur in 3/8 subjects by chance is 0.0058. Although 3 of 8 subjects were found to have *gsp* mutations (2 at site R201, 1 at site R227), no relationship was noted between the presence of *gsp* mutation and GH suppression with GHRH- antagonist.

We conclude that in a subset of patients with acromegaly due to a pituitary adenoma, GH secretion is under partial control by GHRH.

NOTES:

WEDNESDAY, OCTOBER 6 **9:15 – 9:30am**

Selection of the Optimal Entry Zone to the Brain Stem for Removal of Intraaxial Cavernous Angiomas

Helmut Bertalanffy

Objective: Cavernous malformations located within the brainstem show a substantially higher propensity for bleeding than those in other locations. Since radiosurgery appears to be inappropriate for prevention of bleeding, microsurgical removal of brainstem cavernomas is the favored therapeutic option. To completely remove an intraaxial cavernoma without additional morbidity, however, an individually tailored specific entry zone to the brainstem is required, particularly in those cases, in which the lesion is not visible on the surface of the brainstem.

Patients and Methods: During the past 7 years we have treated surgically 56 individuals who harbored - with one exception – a symptomatic brainstem cavernoma. Fourteen of the lesions were located in the midbrain, 36 in the pons and 6 in the medulla. The patient´s records, pre- and postoperative images and surgical video tapes were analyzed. To obtain an optimal surgical window and viewing trajectory to the brainstem, we carefully selected the surgical approach in each single case. Intraoperative electrophysiological monitoring was used in all, neuronavigation in the majority of patients.

Results: The surgical approaches included the pterional/orbitozygomatic, the subtemporal transtentorial, the retrosigmoid, the suboccipital lateral transcondylar, the supracerebellar infratentorial and the suboccipital median telovelomedullary routes. The lesion itself or the bulging hematoma with xanthochromic coloration was seen in 30 cases, while an apparently normal brainstem was found in 26 cases. In the latter, the brainstem was incised at a site where the shortest distance to the lesion was suspected and confirmed by neuronavigation. The exact site, however, was slightly modified when direct electrical stimulation dictated a different safe entry zone to the brainstem. Postoperatively, 9 patients (16 %) showed transient additional neurological deficits whereas 6 individuals (11 %) experienced permanent neurological morbidity, of whom four are completely independent. One patient died from a disease unrelated to the cavernous malformation. The remaining patients of this series were either in the same neurological condition or improved.

Conclusions: A carefully planned and individually tailored approach was essential to obtain a specific exposure of the brainstem which depended upon the size and exact location of the lesion. The optimal entry zone to the brainstem was based on precise anatomical criteria, on the local vascular pattern, on electrophysiological mapping and neuronavigation. In combination with modern neuroanesthesia and advanced microsurgical techniques, excellent results have been achieved with this method.

WEDNESDAY, OCTOBER 6 9:30 – 9:45am

The Synthetic ACDF: Experience with Rh-BMP-2 and Peek Cages

Regis Haid, P Mummaneni, G Rodts, M Boakye

We evaluated the fusion status and clinical outcomes following ACDF using polyetheretherketone (PEEK) spacers filled with recombinant bone morphogenetic protein (rhBMP-2) and covered with a cervical plate.

Methods: 26 patients (13 men and 13 women)(mean age 54 years, range 36-71 years) were retrospectively evaluated. 4 of the 26 patients were smokers. Single level ACDF was performed in 13 patients, 2 level ACDF in 10 patients, and 3 level ACDF in 3 patients. The patients' pathologies included 11 with disc herniations, 10 with spondylosis, 2 with OPLL, 1 with pseudoarthrosis from prior ACDF, 2 with instability. Clinical outcomes were assessed using Odom's criteria. Radiographic outcomes were evaluated using flexion-extension x-rays, and CT was utilized in cases where fusion as questionable.

Results: Of the 26 patients, 1 was lost to follow-up, 2 died of complications unrelated to surgery, and 23 were followed for a mean of 13 months (range 10 months to 2 years). Clinical outcomes (Odom's) were rated as good-excellent in 20 patients, fair in 4 patients. All patients achieved solid radiographic fusion by three months postop. Complications included recurrent laryngeal nerve palsy (1 patient), transient dysphagia (3 patients), CSF leak (1 patient), and transient C5 paresis (1 patient). Evaluation of radiographs showed asymptomatic posterior osteophyte formation in 7 patients.

Conclusion: ACDF with PEEK and BMP with cervical plate fixation affords a fusion rate equivalent to autograft in our experience. Unlike autograft, bone graft harvest site morbidity is avoided. Transmission of disease is not a concern with PEEK spacer and BMP (unlike allograft). New osteophytes may form with PEEK and BMP use, but they were not clinically significant.

A Role for EphB4 Signaling in the Regulation of Vascular Morphogenesis and Vascular Permeability in Malignant Glioma

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Objective: Several endothelial-specific receptor tyrosine kinases (RTKs) and their ligands have been implicated in glioma angiogenesis. Recently, members of the Eph-group of RTKs (Ephs) and their ligands (ephrins) have been suggested to be involved in developmental angiogenesis and embryonal vascular differentiation. The role of ephrin/Eph signaling in adult angiogenesis, however, has remained unknown so far. The objective of the present study was to assess the role of ephrin/Eph signaling for glioma biology.

Methods: Expression of ephrins and Ephs was assessed in glioma cell lines, human glioma xenografts as well as low- and high-grade human astrocytoma using c-DNA-array analysis, RT-PCR and in-situ hybridization. To further study the role of EphB4 signaling for glioma angiogenesis we used Phoenix E cells producing (i) ecotropic retroviral constructs for EphB4-WT, (ii) EphB4-DN, i.e. a truncated dominant-negative mutant form of EphB4 lacking the kinase domain, and (iii) Phoenix E cells producing an empty vector (mock). Phoenix E cells were co-implanted with glioma cells into flanks or the dorsal skinfold chamber preparation of nude mice. Glioma angiogenesis and glioma growth were assessed by immunohistochemistry and intravital multi-fluorescence videomicroscopy.

Results: Expression of several ephrins (EphrinA1, EphrinB1, EphrinB2) and Ephs (EphA2, EphA5, EphB2, EphB3, EphB4) could be detected in human gliomas. Compared to normal brain, expression of EphB4, EphB3 and EphrinB2 mRNA was increased in glioma, whereas expression of EphA5 and EphA2 mRNA was reduced in glioma. Interestingly, expression of ephrins and Ephs in glioma xenografts was detected on both blood vessels and tumor cells. In situ hybridization of human astrocytoma specimens further demonstrated that, while ephrinB2 was homogeneously expressed by tumor cells and tumor blood vessels, EphB4 mRNA was predominantly expressed by perinecrotic tumor cells and tumor blood vessels. Based on these results we next set out to study the role of EphB4 signaling for glioma angiogenesis in detail. *In vivo* manipulation of endothelial EphB4 signaling with EphB4-WT and EphB4-DN

constructs did not affect initial sprouting tumor angiogenesis, but markedly altered the vascular phenotype of the tumors. Compared to mock tumors, both EphB4 WT- and EphB4-DN tumors were characterized by a rather circumferential than branching vessel growth, resulting in extremely large (>200µm) tumor vessels, and by a significant reduction in tumor vessel permeability.

Conclusions: Our results suggest a role for ephrin/Eph signaling in glioma tumorigenesis and angiogenesis through a complex interaction between ephrins and Ephs expressed by tumor cells and endothelial cells. EphB4 signaling in endothelial cells seems to play a central role in the regulation of glioma vascular morphogenesis and vascular permeability. Our results contribute to a better understanding of basic mechanisms underlying glioma angiogenesis and uncover further steps in glioma vascularization beyond the sprouting angiogenesis mediated by VEGF, the angiopoietins and their receptors.

Gamma Knife Radiosurgery in the Management of Glomus Tumors – A Volumetric Study of 17 Cases

Gene H Barnett, A Varma, JH Suh, J Ross

Twenty patients with glomus jugulare tumors underwent Gamma Knife radiosurgery (GKRS) at the Cleveland Clinic during a 6-year period from 1997 to 2002. Three patients with follow up of less than 10 months were excluded. Clinical and radiological data from 17 patients were retrospectively analyzed. MRI tumor volumes at treatment and follow-up were assessed.

There were 15 women and 2 men, with an average age of 63.1 years (range 22 to 87). Follow up was 10 to 79 months (average 44.5 months). Six patients had undergone previous surgical resection of the same tumor, two had multiple tumors and one had a functional chemodectoma. Symptoms were tinnitus (n=12), hearing loss (n=12), lower cranial nerve paresis (n=6) and otalgia (n=5). Tumor margins received 13 to 18 Gy (average = 15 Gy, median = 15 Gy) at 48% to 63% (average = 53%, median = 50%) isodose line. Eight patients clinically improved, 2 patients worsened and 7 patients were unchanged. One patient experienced transient hearing impairment. Tinnitus and otalgia responded most favorably to GKRS. Initial tumor volumes ranged from 0.4 to 26.1 cm^3. Tumor volume showed a transient increase in 7 patients though ultimately decreased in 8 patients (4 clinically improved, 3 unchanged & 1 worse), increased in 4 patients (2 clinically unchanged, 1 improved, 1 worse) and was unchanged in 5 patients (3 clinically improved and 2 unchanged).

GKRS is a safe, effective treatment for glomus jugulare tumors, particularly in elderly patients and patients with serious medical conditions.

WEDNESDAY, OCTOBER 6 **10:15 – 10:30am**

Intraoperative Functional MRI. First Results and Technical Considerations.

Thomas Gasser*, D Stolke*, R Fahlbusch°, C Nimsky°

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Objective: The preservation of eloquent cortex during a neurosurgical intervention is essential for the functional outcome. Functional MRI represents an established concept to map functional units. However, fMRI paradigms are generally active in nature, limiting the method to awake and cooperative patients and to the preoperative period. However, a novel passive fMRI paradigm for localization analysis of the sensorimotor cortex allows functional analysis of neurologically impaired or anaesthetized patients. The paradigm is based on peripheral electrical nerve stimulation during echo-planar image acquisition. Preceding studies have already established clinical applications of this fMRI-paradigm.

The present prospective study evaluates the feasibility of true intraoperative fMRI utilizing this paradigm in anaesthetized patients undergoing brain tumour resection. The study focused as well on the technical setup and the intraoperative management.

Methods: In 3 anaesthetized patients with centrally localized lesions intraoperative fMRI scans were acquired at 3 distinct stages of the surgical procedure employing an intraoperative 1.5 Tesla MR scanner. The data were analyzed statistically and co-registered with the Talairach space. Additionally the data were validated by electrophysiology.

Results: Utilizing this passive fMRI paradigm, the sensorimotor cortex could be identified intraoperatively in 2 of the 3 patients. We observed a significant change in signal intensity in the course of the operation and detected regularly an inverted BOLD-signal response, which may be caused by an inhibition of cerebrovascular autoregulation under total intravenous anaesthesia (TIVA). Susceptibility artefacts influenced image quality marginally.

Conclusion: We could demonstrate the feasibility of this method to identify the sensorimotor cortex in anesthetized patients in the surgical setup. Further studies will have to evaluate the BOLD-signal characteristics under anaesthesia and the clinical impact of intraoperative fMRI.

Sirolimus-Eluting Stents in the Canine Cerebral Vasculature: Assessment of Safety and Vessel Response

Elad I Levy, RA Hanel, AS Boulos, FO Tio,am Paciorek, KS Kagan-Hallet, MD Fronckowiak, LR Guterman, LN Hopkins

Purpose**:** The treatment of intracranial atherosclerosis with bare-metal stents results in excessive restenosis rates. Neurotoxicity effects and vessel injury following implantation of drug-eluting stents are unknown in the cerebrovasculature. To assess the safety and vascular effects of sirolimus-coated stents, we conducted a prospective comparison study of drug-eluting and bare metal stent implantation in the canine basilar artery.

Method**:** Sixteen mongrel dogs were randomized to receive either bare metal 1.5- x 8-mm (6-cell) stents or sirolimus-eluting stents of the same dimensions. Serial peripheral blood samples were obtained during the first week after stent implantation to determine the time-dependent serum sirolimus concentration. Angiograms were performed 30 days post-implantation to assess the effects of stent placement on the basilar artery and brainstem perforators. Stent and basilar artery explantation was performed immediately after angiography. Histological and computer-assisted morphometric analyses of specimens obtained were performed.

Results**:** No sirolimus was detected in peripheral blood samples obtained later than 24 hours post-treatment. On follow-up angiography, no evidence of parent vessel damage or pseudoaneurysm formation was seen. Explanted vessels and brainstem sections showed no evidence of neurotoxicity. No significant differences were found in the time to endothelialization of bare-metal and sirolimus-coated stents. Smooth-muscle cell proliferation was lower in animals receiving sirolimus-coated stents ($p = 0.003$). Additionally, intimal fibrin density was increased in the group treated with sirolimus-coated stents ($p < 0.0001$). Histological evidence of an inflammatory response indicated a trend toward a reduced response in the sirolimus group.

Conclusion**:** These findings suggest that sirolimus-coated stents may inhibit in-stent stenosis. Further studies with longer-term follow-up are required to assess the restenosis rates of sirolimus-coated stents implanted in intracranial vessels.

WEDNESDAY, OCTOBER 6 **11:00 – 11:15am**

Brain Tumor Surgery in the Vicinity of Short-Term Memory Representation –Results of Neuronavigation Using fMRI Images.

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Objective: Functional informations concerning the surrounding brain are mandatory for a good clinical outcome in brain tumour surgery. The value of fMRI to detect the motorcortex and the Broca area is widely accepted today. If an appropriate paradigm is used, short-term memory areas can be visualized as well. Obviously these informations must be integrated into cranial neuronavigation for an appropriate intraoperative use. We report our first experiences with the direct integration of short-term memory fMRI into cranial neuronavigation.

Method:. From January 2001 to March 2002 14 patients were operated on for intracranial tumors with short-term memory fMRI imaging, using the „two-back-paradigm“. Both pre- and postoperatively, the short-term memory of all patients was tested additionally by a standardized test battery including 16 different verbal and visuo-spatial items. FMRI was repeated postoperatively as well.

Results: In all 14 patients the general level of working memory capacity was preserved after surgery and we found an improvement in verbal short-term memory items. In contrast, the visuo-spatial performance kept unchanged or deteriorated slightly and the alertness slightly worsened as well.

Conclusion: Functional fMRI basing on a short-term memory paradigm is able to improve the outcome after surgery for cortical and subcortical brain tumors localized in the frontal and precentral area. The so-called two-back paradigm mainly serves for verbal memory tasks. For visuo-spatial items, we are going to design a new paradigm. Unfortunately it has to be mentioned, that irradiation may deteriorate this capacity as well.

WEDNESDAY, OCTOBER 6 **11:15 – 11:30am**

Fiber Tract Deformation as Visualized with Sequential Acquisitions of 3D-Ultrasound (3DUS) During Intracranial Surgery

Volker A Coenen, T Krings, J Weidemann, FJ Hans, P Reinacher, JM Gilsbach, V Rohde

Objective: We present a technique that intraoperatively allows displaying brain shift and its effects on fibre tracts that preoperatively were studied with diffusion-weighted imaging (DWI). With DWI the pyramidal tract and the optic radiation can be displayedamong other fiber tracts. Intraoperative 3D – ultrasound (3DUS) has the ability to directly compare ultrasound and MRI (or DWI) anatomy. 3DUS cannot directly visualize fiber tracts in the white substance of the brain. However, it is possible to estimate a fiber tract's position by defining an environment of ultrasound landmarks on the basis of the US – DWI comparison.

Methods: In three patients intracranial lesions (1 malignant glioma, 1 metastasis, 1 cavernoma) with contact to either the corticospinal or the geniculostriate tracts were removed microneurosurgically. Preoperatively, diffusion weighted magnetic resonance imaging (DWI) was obtained to visualize the fiber tract at risk. DWI data were fused with the anatomical T1-weighted magnetic resonance (MR) image. A single rack 3D - ultrasound (3DUS) neuronavigation system, which displays simultaneously the MR image and the corresponding ultrasound image was used intra-operatively for 1) navigation 2) definition of fixed and shiftable ultrasound-landmarks in vicinity to the fiber tract 3) sequential image update at different steps of resection resulting in time dependent brain deformation data. Using a standard personal computer equipped with Windows®-based image software, the brain shift-associated fiber tract deformation was assessed by sequential landmark registration. After surgery, DWI was obtained to directly confirm the predicted fiber tract deformation.

Results: The lesions were removed without any morbidity. The comparison of 3DUS with DWI and T1-weighted MRI allowed to define fixed and shiftable landmarks close to the respective fiber tract. Postoperative DWI confirmed, that the actual fiber tract position corresponded to the sonographically predicted fiber tract position at the end of surgery.

Conclusion: By definition and sequential intraoperative registration of ultrasound landmarks in vicinity to a fiber tract, brain shift associated deformation of the sonographically not visible tract can be correctly assessed. This approach appears to be helpful to identify and avoid eloquent brain during intracranial surgery.

GERMAN SOCIETY FIRST PRIZE AWARD
(Wilhelm Tönnis Preisträger)

Mechanical Response of a Cervical Spine Motion Segment on Removing a Local Autograft

Tobias Pitzen[1], CJ Kempf[1], TR Oxland[2], WI Steudel[1]

[1]Neurochirurgische Klinik, Universitätskliniken des Saarlandes, 66421 Homburg/ Saar. [2]Division of Orthopaedic Engineering Research, Departments of Orthopaedics and Mechanical Engineering, University of British Columbia, Vancouver, Canada

Objective: There is evidence, that filling a cervical spine fusion cage with either bone substitute or autologous bone graft from the iliac crest is beneficial with respect to speed and rate of the bony fusion. However, both bone substitutes and the iliac crest autograft have specific disadvantages. Using a local autograft from the cervical spine vertebral bodies to fill a cervical spine fusion cage might be an alternative. However, removing the local autograft from the vertebral bodies of the cervical spine could result in reduced strength of the spinal segment, thus again leading to severe complications. Therefore, mechanical testing of such a procedure should be performed before the method is to be used in clinical practice. The objective of the study was to investigate, if removing a local autograft from the cervical spine vertebral bodies significantly reduces compression strength of a cervical spine segment.

Methods: 14 human cervical spine segments (C4-C5 and C6-C7) were used. According to bone mineral density they were assigned to either control or test group. Each specimen underwent anterior discectomy and insertion of a titanium cage (Rabea, Signus Medizintechnik, Alzenau). Within the specimen, assigned to the test group, a cylindrical bone dowel of 5 mm depth and 5 mm diameter was removed from the anterior aspect of each vertebra. The specimens were then loaded in flexion-compression. We looked at the force resulting in the first failure, at the force resulting in 4 mm displacement of the construct and at the correlation of these parameters to the bone density of the segment.

Results: Then mean force resulting in initial failure was 1149 N for the test and 1647 N for the control group ($p = 0{,}252$). The force resulting in 4 mm displacement was 1064 N for the test and 1574 N for the control group ($p = 0{,}155$). There was a strong and significant correlation of these parameters versus bone density for the control group, however, the correlation was moderate and not significant within the test group.

Conclusion: Removing a cylindrical dowel of 5 x 5 mm from the anterior aspect of the cervical spine vertebral bodies does result in a not significant reduction of strength in flexion-compression.

AMERICAN ACADEMY
HONORABLE MENTION AWARD

Selective Ablation of Vanilloid Receptor 1 Positive Nociceptive Neurons for Elimination of Hyperalgesia and Neuropathic Pain

Russell R Lonser, GC Tender, S Walbridge, Z Olah, L Karai, M Iadarola, EH Oldfield

Objectives. Neuropathic pain is mediated by nociceptive neurons that selectively express the vanilloid receptor 1 (VR1). Resiniferatoxin, an excitotoxic VR1-agonist, destroys VR1-positive neurons. To determine if resiniferatoxin can selectively ablate VR1-positive neurons and eliminate neuropathic pain without affecting tactile sensation and motor function, we infused it unilaterally into primate trigeminal ganglia**.**

Methods. We perfused resiniferatoxin (experimental=3) or vehicle (control=1) into the right trigeminal ganglia of Rhesus primates and tested tactile and hyperalgesic sensation in trigeminal-innervated territories (corneal application of saline and capsaicin, respectively) at 1, 4 and 7 weeks postoperatively. Number of blinks, eye wipes, and duration of squinting were recorded after application. Neurogenic inflammation, mediated by VR1-positive neurons, was tested by facial application of capsaicin cream. Immunohistochemical analyses were performed after euthanization.

Results. Consistent with elimination of hyperalgesia on the side of resiniferatoxin-treated ganglia, there was a dramatic reduction in the number of blinks (mean±S.D.; 25.7±4.4 versus 106±20.8, and 112.8±19.7), eye wipes (1.4±0.8 versus 19.3±5.0 and 16.7±4.5), and duration of squinting (1.4±0.7 seconds versus 11.4±1.6 and 14.8±1.7) in response to corneal application of capsaicin, compared to the vehicle and untreated sides, respectively ($p<0.01$). Tactile sensation (mean blinks after saline application; 18.8±2.3, versus 18.0±2.6, and 18.4±3.3, respectively; $p>0.05$) and motor function (mastication) remained intact. Neurogenic inflammation was blocked on the resiniferatoxin-treated side. Animals showed no adverse clinical effects. Immunohistochemical analysis revealed selective ablation (mean decrease, 79.3±2.8%; $p<0.001$) of VR1-positive neurons in the resiniferatoxin-treated ganglia.

Conclusions. VR1-positive nociceptive neurons can be safely and selectively ablated by intraganglionic resiniferatoxin infusion, eliminating hyperalgesia and neurogenic inflammation while maintaining intact tactile sensation and motor function. Intraganglionic resiniferatoxin infusion could provide a new site-specific, mechanism-based treatment approach for neuropathic pain.

GERMAN ACADEMY FIRST PRIZE AWARD

Identification of Brain Tissue Necrosis by MRI: Validation by Histomorphometry

Michael Stoffel[1,2], C Blau[3], H Reinl[3], J Breidt[3], K Gersonde[3], A Baethmann[2], N Plesnila[2]

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The volume of an experimental necrotic lesion of the cortex expands up to 400 % of its initial size within the first 24 hrs after the insult. Lesion expansion, a clinically well known phenomenon, is often accompanied by perifocal brain edema and consequently difficult to image and to analyze by magnetic resonance imaging (MRI). Therefore we aimed to validate a T_2-weighted spin echo sequence upon its ability to distinguish necrotic from edematous brain tissue.

Male Sprague-Dawley rats (n=5 per group) were subjected to a cortical freezing lesion leading to immediate tissue necrosis with subsequent perifocal vasogenic brain edema. Immediately, 4, 12, and 24 hrs after the lesion the maximal area of necrosis was quantified longitudinally by coronal T_2-weighted spin echo MRI-scans. After the last scan, animals were sacrificed for direct comparison of the lesion area obtained by MRI and histomorphometry. In parallel groups of animals, lesion expansion was quantified by histology.

The acquired T_2-maps clearly distinguish the cortical necrosis from perifocal edema and healthy brain. Focal freezing led to a cortical lesion of 5.24 ± 0.36 mm^2 immediately after trauma (0 h; 100 %) which expanded progressively to a maximum of 6.82 ± 0.34 mm^2 after 24 hrs (131 %; * $p<0.01$ vs. 0h). Lesion expansion quantified by histology was almost identical (132 % within 24 hrs). Histological assessment resulted in smaller absolute lesion areas compared to MRI, most likely due to shrinking during tissue processing (4.72 ± 0.26 mm^2 vs. 6.82 ± 0.34 mm^2, $p<0.01$).

The current study shows that necrotic brain tissue can be distinguished from surrounding brain edema by T_2-mapping. The technique is sensitive enough to detect small changes in necrosis expansion in vivo as validated by histology. The presented technique may be a useful future tool for the non-invasive identification of necrotic brain tissue following brain injury, e.g. from trauma or ischemia.

WEDNESDAY, OCTOBER 6 **12:15 – 12:30pm**

AMERICAN ACADEMY AWARD

Achieving a Multidimensional Brain Computer Interface in Humans Using Electrocorticographic Signal

Eric C Leuthardt, G Schalk, JR Wolpaw, JG Ojemann, DW Moran

Brain-computer interfaces (BCIs) enable users to control devices with electroencephalographic (EEG) activity from the scalp or with single-neuron activity from within the brain. Both methods have disadvantages: EEG has limited resolution and requires extensive training, while single-neuron recording entails significant clinical risks and has limited stability. This research demonstrates that electrocorticographic (ECoG) activity recorded from the surface of the brain can enable users to control a computer cursor rapidly and accurately. Previously, over brief training periods (several minutes), four patients mastered control and achieved high success rates in one dimensional tasks. In additional experiments, we found that ECoG signals at frequencies up to 180 Hz accurately reflected the direction of two-dimensional joystick movements. These techniques derived to achieve one dimensional control were then used to translate this to two dimensional control in one patient with success rates of 70-94%. These results suggest that an ECoG-based BCI could provide people with severe motor disabilities a non-muscular multidimensional communication and control option that is more powerful than EEG-based BCIs and is significantly easier and safer than BCIs that use electrodes penetrating the brain.

NOTES:

THURSDAY, OCTOBER 7 **9:15 – 9:30am**

Clinical Outcome Following Transforaminal Lumber Interbody Fusion (TLIF): One Year Follow-up of Prospective Data

Richard G Fessler

METHODS**:** Two studies were completed using a prospective, non-randomized design.

Study 1: 50 patients underwent either open TLIF or minimal access TLIF (MAST TLIF). 25 patients were in each group. All patients receiving open TLIF were performed by one surgeon, and all MAST TLIFs were performed by a second surgeon. Surgeries were performed over the same period of time. Data collected included age, sex, levels operated, operative time, blood loss, transfusions, pain medications received, and length of stay. Results were analyzed using Wilcoxon Signed Rank analysis and Student's t-test analysis.

Study 2: 56 patients who were scheduled to undergo MAST TLIF were pre-operatively administered VAS, Oswestry Disability Scale, and SF-36 questionnaires. Surgery was then performed and the same tests were re-administered at 6 wks, 4 ½ months, and 1 year post-operatively. Data at 1 year was compared to the pre-operative data using the Wilcoxon Signed rank analysis for VAS scores and repeated measures ANOVA for the Oswestry Disability Scores and SF-36 scores.

RESULTS:
Study 1: Patient demographics, including age and levels operated were not significantly different. More males were done through mast technique than open. Operative time was not significantly different between the groups. Patients undergoing MAST TLIF lost significantly less blood (200 v 1100 cc, $p < .002$), required significantly fewer transfusions (0 v 33 %, $p < .001$), and took significantly less pain medicine (130 v 180 MSO4 equivalents, $p < .05$). Length of stay was less for MAST TLIF patients, but did not reach statistical significance.

Study 2: Patients followed for 1 year after MAST TLIF experienced a 98 % fusion rate. Visual analog scores decreased from 5.5 to 3.5 ($p < .008$). Oswestry disability scores decreased from 45 to 30 ($p < .0001$), and SF-36 scores for physical functioning increased from 35 to 46 ($p < .01$).

Discussion: The MAST TLIF technique for performing lumbar interbody fusion and stabilization is a safe and effective procedure. In similar groups of patients it can be performed in the same or less time than open procedures, while causing less blood loss, fewer blood transfusions, and requiring less pain medicine and possibly shorter hospital stays. Long term follow-up demonstrate significant improvement in pain, disability, and overall satisfaction with life compared to pre-operative values.

THURSDAY, OCTOBER 7 **9:30 – 9:45am**

Transgenic Arteriovenous Fistula in the Rat: An Experimental Model of Gene Therapy for Brain Arteriovenous Malformations

Michael T Lawton, C Stewart, A Wulfstat, N Derugin, T Hashimoto, W Young

Objective**:** To introduce the transgenic arteriovenous fistula (AVF) model in the rat, constructed by interposing mouse aorta in a fistula between the common carotid artery and external jugular vein in a nude rat; to describe the model's technical feasibility, long-term patency, and expression of reporter genes.

Methods**:** Carotid-jugular fistulas were surgically created in 112 rats. In 25 immunodeficient nude rats, wild-type mouse thoracic aorta (TAo) was interposed in the fistula; in 10 immunocompetent rats, TAo was interposed; in 19 nude rats, transgenic TAo with reporter genes for beta-galactosidase (lac-Z) or green fluorescent protein (GFP) was interposed; in 18 nude rats, wild-type mouse ascending aorta (AAo) was interposed; and in 40 rats, a simple fistula was constructed without an interposition graft. Host tolerance and graft viability were determined by histopathology and immunohistochemistry for CD31 (mouse endothelial cell marker), endothelial nitric oxide synthase (eNOS), smooth muscle actin, fibronectin, beta-galactosidase, and GFP.

Results**:** The transgenic AVF was technically feasible and immunologically tolerated in nude rats, but not in immunocompetent rats. The overall angiographic patency rate was 41% with TAo grafts and 56% with AAo grafts, both lower than the 98% patency rate in fistulas with a single anastomosis and no interposition graft. Mouse endothelium survived on the graft for 3 months according to CD31 staining, but longer survival by transgenic smooth muscle cells resulted in continued expression of lac-Z for 6 months and GFP for 4 months. Endothelium and smooth muscle in the fistula were functional, with normal expression of eNOS, and smooth muscle actin and fibronectin, respectively.

Conclusion**:** The transgenic arteriovenous fistula model enhances other carotid-jugular fistula models by integrating transgenic tissue, thereby creating an experimental system for investigating the molecular biology of and gene therapies for arteriovenous malformations.

THURSDAY, OCTOBER 7 **9:45 – 10:00am**

Application of Image-Guided Spinal Navigation to Decompression and Internal Fixation of the Upper Cervical Spine

Iain H Kalfas

The techniques for decompression and internal fixation of the upper cervical spine have evolved significantly. This has resulted from an improved understanding of spinal biomechanics, improvements in spinal imaging, the refinement of surgical exposure techniques as well as continued advances in the design of internal fixation devices.

Despite these advances, the anatomic complexity of the upper cervical region can present difficulties for even the most experienced spinal surgeons. In particular, orientation to the pertinent neural, vascular and bony structures is critical to optimizing the outcome of surgery. While intraoperative fluoroscopy can help facilitate anatomic orientation, it has several technical limitations.

Computed tomography-based image-guided spinal navigational technology has evolved over the past decade. Unlike, fluoroscopy, it provides the surgeon with multiplanar images through the upper cervical region. These images can be manipulated intraoperatively to provide enhanced orientation to the surgical anatomy.

Forty patients underwent surgery for decompression and internal fixation of the upper cervical spine using image-guided spinal navigation. Thirty-two patients underwent posterior C1-2 screw fixation, 5 patients underwent transoral surgery with posterior C1-2 screw fixation and 3 patients underwent anterior odontoid screw fixation. Fluoroscopy was also used on a limited basis during each case to validate the navigational information.

Satisfactory screw placement was achieved in all patients. There were no incidences of neural of vascular injury due screw placement. The technique of using image-guided technology for upper cervical surgery will be reviewed.

THURSDAY, OCTOBER 7 **10:00 – 10:15am**

Laboratory Testing of an Implantable Microsensor System for Intraoperative 3D-Computer Animation of Vertebral Body Motion in Cervical Spinal Surgery

Olaf Süss, T Kombos, S Schönherr*, S Mularski, B Kühn, M Brock

Neurochirurgische Klinik, Charité-Universitätsmedizin Berlin, Campus Benjamin Franklin* Institut für Informatik, Freie Universität Berlin

Objective: Degenerative diseases of the cervical spine may not only cause neurological deficits due to direct compression of the spinal cord or the spinal nerves, but also alter its biomechanical function and statics. In such cases, the aim of surgery is double: to decompress neural structures and to provide the highest possible stability and the best possible preservation of function. Regardless of the surgical technique used, intraoperative prediction of the functional (neurological and biomechanical) results of surgery, might represent a major progress.

Materials & Methods: Navigationsystems have been used in spinal surgery for specific surgical planning, optimization of anatomical orientation and improvement of surgical accuracy. In the present research project (sponsored by the Deutsche Arthrose Hilfe e.V.), reference sensors of an electromagnetic navigation system (ACCISS II™, Schaerer Mayfield Technologies) have been miniaturized for direct implantation into one or more vertebral bodies with the aim of providing precise additional spatial online information on the location of the vertebral bodies and their orientation in space. In laboratory tests, up to three microsensors were implanted into several cervical vertebrae of cadaver specimens.

Results: Five different surgical techniques (ventral fusion, dorsal fixateur implantation, ventrolateral plate osteosynthesis, foraminotomy, cage implantation) were performed in a total of ten cervical spine models using different segments (C1-3, C5/6, C6-T1). The isolated vertebral body movements were registered and visualized online on the navigation screen.

Conclusion: The implanted reference sensors allow computer-animated, real-time visualization of isolated movements of individual vertebral bodies or of entire motion segments. This means that motion can be visualized immediately

during surgery without the need for additional intraoperative imaging procedures such as fluoroscopy or computed tomography. These results indicate that reliable information on the expected postoperative biomechanical outcome can be obtained by continuous intraoperative three-dimensional animation.

Transthoracic Discectomy Without Interbody Fusion

D Edwards, A Cohen-Gadol, William E Krauss

Object: Transthoracic discectomy is an established surgical procedure for the treatment of thoracic disc disease. Most authors advocate interbody fusion following transthoracic discectomy. The purpose of this study was to determine if there were adverse consequences in forgoing interbody fusion following transthoracic discectomy.

Methods: From 1996 to 2002, the senior author performed transthoracic discectomy without fusion on eighteen patients. There were eleven women and seven men in this series. Their mean age was 54 years (SD=17, range 28-84). Surgical indications were radiculopathy in 1 patient, myelopathy in 14 patients, and radiographic cord compression in 3 patients. We obtained postoperative CT scans to evaluate the decompression in most patients. We obtained followup data from clinic visits and telephone surveys. We obtained and compared pre and postoperative radiographs to determine the incidence of postoperative kyphosis at the operated level. Mean followup was 2.3 years. No patients reported the new onset of axial spine pain postoperatively. Fourteen of 18 patients had significant improvement in neurological symptoms and signs. The only three non-ambulatory patients regainedambulation. One 83 year-old patient had a slight increase in gait difficulties postoperatively. There were three major complications: 1 wound infection, 1 sympathetic pleural effusion requiring pleurodesis, and 1 case of disabling postthoracotomy pain. No patients developed a clinically significant kyphotic deformity.

Conclusions: These results indicate that interbody fusion may not be necessary in all patients undergoing transthoracic discectomy.

Treatment of Spinal Cord Injury via Topical Perfusion with an ATP Solution

Christopher B Shields, YP Zhang, S Chien, Y Han, LBE Shields, M Li, B Chiang, DA Burke

ATP depletion damages ATP sensitive ion channels following spinal cord injury (SCI), leading to membrane failure and cell death. These processes may be reversed by the application of exogenous ATP. ATP solution, stabilized in a lecithin suspension, has been shown to protect hepatic tissue and skin following injury (unpublished data). Besides supporting metabolism, ATP may be trophic in neurogenesis and cell survival by acting on its P2X and P2X receptor subunits. We suggest that exogenous administration of ATP solution to the spinal cord following SCI may prevent irreversible damage and improve functional recovery.

Sixteen Sprague-Dawley rats received a 25 gm-cm SCI at T10 and were assigned into one of two groups: 1) injury/vehicle (control: n = 8) and 2) injury/ATP enhanced solution containing 3000 µmol ATP/ml (n = 8). A perfusion well was designed to irrigate ATP solution to the injured spinal cord. Flow rate was 5 ml/hour for 24 hours. Locomotion was tested weekly for 6 weeks after SCI using the BBB score. At week 6, rats were fixed, and their spinal cords are being assessed for lesion size and tissue preservation.

BBB scores were higher in rats irrigated with the ATP enhanced solution compared to the control group. Locomotor function was significantly better in the ATP-treated groups at 2, 4, and 5 weeks ($p \leq 0.05$). Tissue sparing and lesion size of the spinal cords will be analyzed after 6 weeks.

We conclude that topical application of exogenous ATP provides functional and possibly anatomical neuroprotection following moderate SCI.

THURSDAY, OCTOBER 7 **11:00 – 11:15am**

IOMaster 7D – A New Device for Virtual Neuroendoscopy

Christos Trantakis[1], J Meixensberger[1], G Strauß[2], E Nowatius[3], D Lindner[1], HK Cakmak[4], H Maaß[4], C Nagel[1], U Kühnapfel[4]

[1]Department of Neurosurgery, [2]Department of ENT, University of Leipzig
[3]Zwonull Media GmbH, Leipzig [4]Institut für Angewandte Informatik, Research Center Karsruhe

Abstract: Objective: To create a VR training system for neuroendoscopy. A new force feedback device was intended to be developed and evaluated regarding the suitability for the simulation of endoscopic ventriculocisternostomy.

Methods: A VR model for ventriculostomy was generated based on a MRI dataset. Software modules were used for segmentation (VESUV), modelling (KisMo) and visualization (KISMET) and implemented on a WIN32 platform. Real instruments (MINOP, Aesculap, Germany) were adapted to the simulator. The system was evaluated in 79 procedures.

Results: The "IOMaster 7D" offers 7 degrees of freedom. Both the trocar and the acting instruments are captured separately. The trocar`s position determines the view of the camera, the access to the target, and the operating range. An elastodynamic hydrocephalic ventricular system with realistic proportions was modeled. An interactive virtual preparation with force feedback was implemented. The system provides for axial movement or rotation of the instruments, cutting, grasping and realistic elastodynamic deformations of the ventricle wall. First evaluations proved a reduction of the median failure rate and a reduction of the median time to reach the target. Analysis of the total distance of instruments movement showed a reduction, too.

Conclusion: VR systems can simulate realistic and real time surgical procedures and may open new perspectives for the neurosurgical training. Integrating haptic information increases the quality. The definition of no touch areas and targets and the possibility of registration of both kinetic parameters, failure rate and the time course of the procedure provide objective criteria for the appreciation of a learning effect.

THURSDAY, OCTOBER 7 **11:15 – 11:30am**

Fedor Krause, The Krause Operations, and Krause's Impact onamerican Neurosurgeons

Peter W Carmel, M Buchfelder

Fedor Krause (1856-1937), the pioneering German neurosurgeon, is widely known and revered in Germany, but is poorly knownamongamerican neurosurgeons. He is slightly better known for his innovative cranial approaches; the "Krause operations".

We have assessed Krause's impact on contemporaneousamerican neurosurgeons by review of references in over 30 books and monographs by Cushing, Dandy, Elsberg, and Frazier. This informal "citation index" permitted comparison with other contemporaneous authors.

Each of the four authors cited Krause frequently, not only from the English translation of his book (1909-1911), but from his papers in German as well. Indeed, Elsbergs' "Surgical Diseases of the Spinal Cord" (1916) cites Krause more often than any other author. Elsberg and Frazier credited Krause with innovation of two further "Krause operations"; a form of osteoplastic laminectomy, and mid-line cord myelotomy for pain.

This generation of neurosurgeons knew German, (the language of science), and several had spent a *Wanderjahre* in Europe while training. These fouramerican neurosurgeons were clearly highly influenced by Krause's work. Why is he not better known inamerica today? It may be related to the fact that subsequent neurosurgical generations were less likely to read German, and after 1933, less likely to travel to Germany. They only knew of Krause through his 1911 book, which had been through many revisions in German (68 editions!) but not in English.

We will review the career, surgical innovations, and impact of this unique neurosurgical progenitor, with the hope of wider understanding of his contributions.

Diffusion Tensor Imaging for 3-D Fiber Tract Visualization in Navigated Glioma Surgery

Christopher Nimsky, O Ganslandt, AG Sorensen, R Fahlbusch

Department of Neurosurgery, University Erlangen-Nürnberg, Erlangen, Germany (CN, OG, RF), Department of Radiology/Nuclear Magnetic Resonance Center, Massachusetts General Hospital, Boston, USA (AGS)

Objective: To investigate the intraoperative displacement of major white matter tracts during navigated glioma resection by comparing pre- and intraoperative diffusion tensor imaging (DTI) based fiber tracking.

Methods: In 37 patients undergoing glioma surgery pre- and intraoperative DTI was performed with a 1.5 T magnetic resonance scanner applying an echo-planar imaging sequence with 6 diffusion directions. For 3-D tractography we implemented a knowledge-based multiple-ROI (region of interest) approach applying user-defined seed regions in the color-encoded maps of fractional anisotropy (FA). Tracking was initiated in both retrograde and orthograde directions according to the direction of the principal eigenvector in each voxel of the ROI. The tractography results were also assigned color applying the convention used in color-encoded FA maps. In 15 patients DTI data were integrated into the navigation setup.

Results: Pre- and intraoperative fiber tracking was technically feasible in all cases. Fiber tract visualization gave a quick and intuitive overview of the displaced course of white matter tracts in 3-D space. Comparing pre- and intraoperative tractography depicted a marked shifting of major white matter tracts during glioma removal. Maximum white matter tract shifting ranged from -8 to +15 mm (+2.7 ± 6.0 mm; mean ± standard deviation); in 29.7% an inward, in 62.2% an outward shifting was detected. DTI based neuronavigation allowed identification and preservation of deep seated eloquent brain areas.

Conclusion: Comparing pre- and intraoperative fiber tracking visualizes a marked shifting and deformation of major white matter tracts due to tumor removal. This brain shift of deep structures emphasizes the need for an intraoperative update of navigation systems, when navigation is applied during resection of deep-seated tumor portions near eloquent brain areas.

Changes of the Astrocytic Matrix and Reappearance of Radial Glia in Hippocampi of Patients Suffering from Temporal Lobe Epilepsy

[1]Thomas M Freiman, [1,2]J Eismann, [2]M Frotscher, [1]J Zentner

[1]Department of Neurosurgery, **[2]**Institute for Anatomy and Cell Biology, Albert-Ludwigs-University, D-79106 Freiburg in Breisgau

Objective: Temporal lobe epilepsy (TLE) is frequently associated with a sclerosis of the hippocampus, also known asammon's horn sclerosis (AHS). These hippocampi show a significant neuron loss with astrogliosis. In the recent literature most attention was paid to neuronal changes. However these hippocampi show complex remodeling of synapses and neuronal layers, as a widening of the usually very slim granule cell layer (GCL dispersion) which might be influenced by the change of the glial matrix.

Methods: Hippocampal specimens were obtained from hippocampi of patients treated for pharmaceutically intractable TLE (n=12) and from patients with intracerebral tumors, who served as controls (n=3). The GCL dispersion and the back-grown mossy fibres of the two groups were compared with immunohistochemistry against glial fibrillary acid protein (GFAP) and Silver-Impregnations. The astrocytes were reconstructed and the morphology was evaluated by an optical analysis system (Neurolucida).

Results: Astrocytes proximal to the granule cell layer display a change of their processes. The usually star-like shape of the processes changed into longer processes projecting vertical through the dispersed GCL. Some radial glial cells were observed projecting also vertical to GCL.

Conclusion: Hippocampi of patients with AHS demonstrate a change of the glial scaffold and a new appearance of progenitor cells which project vertical through the GCL along the embryonal sprouting direction of neurons in the hippocampus.

Surgical Treatment of Hemispheric Intractable Epilepsy in Childhood with Functional Hemispherectomy or Peri-insular Hemispherectomy.

Falk Oppel, V Zountsas, HW Pannek

Neurochirurgische Klinik Krankenanstalten Gilead Bethel Bielefeld

Objective: To present the surgical and clinical results of the therapy of 71 hemispherical intractable epilepsies in children and juveniles, who were operated on in Bethel Epilepsy Centre between May 1990 and August 2003, undergoing either hemispherectomy or hemispherotomy.

Method: We analyzed retrospectively these 71 cases. The age at the operation ranged from 4 months to about 17 years (30% aged 3-14 years, 33% 1-3 years and 29% infants). The indication for hemispherectomy was set in 44 cases with congenital etiologies (25 hemimegalencephaly, 14 cortical dysplasia and 5 Sturge-Weber cases) as well as in 27 cases with acquired lesions (11 Rasmussen encephalitis, 13 porencephaly cases after MCA-infarct and 3 other encephaloclastic lesions). By the functional hemispherectomy, the big central region tissue resection was followed from the temporal lobectomy,amygdalohippocampectomy, callosotomy and undercutting disconnection of frontal and occipital lobes. By the hemispherotomy we achieved through smaller craniotomies, shorter operating times and less blood loss, the disconnection of the hemisphere from inside the lateral ventricle. The epilepsy outcome is evaluated according to the Engels classification, with mean follow up of 6 years and 4 months.

Results: 28 patients underwent functional hemispherectomy and 43 patients peri-insular hemispherotomy. The advantages of hemispherotomy are obvious in the porencephalic and the atrophic lesions. The epilepsy outcome is 71 % seizure free or almost seizure free and additionally 17% of cases benefited from the operation (Engels III). The rates of incomplete disconnection have been reduced by the hemispherotomy. 7 % of cases needed a shunt implantation. The mortality was 2,8 %.

Conclusion: The peri-insular hemispherotomy can be safely applied in all etiologies of hemispheric intractable epilepsy and comparable to functional hemispherectomy, has better surgical and clinical results. The early operation is beneficial for the development of the children, taking full advantage of the brain plasticity in this age.

NOTES:

SPECIAL GUESTS

GUESTS	SPONSORS
Markus Bittl (fellow) Charlottesville, VA	John Jane
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Richard Fessler Chicago, IL	Hunt Batjer
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Eric Leuthardt (resident) St. Louis, MO	Academy Award Recipient
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Russell Lonser Bethesda, MD	Academy Award Honorable Mention
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David G. McLone 1972
Arden F. Reynolds, Jr. 1973
Richard L. Rapport 1974
Andrew G. Shetter 1975
John R. Howe 1976
Howard W. Blume 1977
Howard J. Senter 1978
Elisabeth M. Post 1979
David Dubuisson 1980
Dennis A. Turner 1981
Marc R. Mayberg 1982
David S. Baskin 1983
Kevin J. Kiwak 1984
Terry Lichtor 1985
Michael G. Nosko 1986
Joseph R. Madsen 1987
James T. Rutka 1988
Christopher D. Heffner 1989
Scott I. Gingold 1990
Mary Louise Hlavin 1991
Adam P. Brown 1992
Michael Tymianski 1993
David Garrett, Jr. 1994
L. Brannon Thomas 1995
John S. Yu 1996
Gregory Canute 1997

MEETINGS OF THE ACADEMY

Hotel Netherland Plaza, Cincinnati, Ohio ..... October 28-29, 1938
Roosevelt Hotel, New Orleans, Louisiana ..... October 27-29, 1939
Tudor Arms Hotel, Cleveland, Ohio ..... October 21-22, 1940
Mark Hopkins Hotel, San Francisco, California ... November 11-15, 1941
Ambassador Hotel, Los Angeles, California..... November 11-15, 1941
The Palmer House, Chicago, Illinois ..... October 16-17, 1942
Hart Hotel, Battle Creek, Michigan..... September 17-18, 1943
Ashford General Hospital, White Sulphur Springs,
West Virginia ..... September 7-9, 1944
The Homestead, Hot Springs, Virginia ..... September 9-11, 1946
Broadmoor Hotel, Colorado Springs,
Colorado ..... October 9-11, 1947
Windsor Hotel, Montreal, Canada ..... September 20-22, 1948
Benson Hotel, Portland, Oregon ..... October 25-27, 1949
Mayo Clinic, Rochester, Minnesota..... September 28-30, 1950
Shamrock Hotel, Houston, Texas ..... October 4-6, 1951
Waldorf-Astoria Hotel, New York City,
New York ..... September 29-October 1, 1952
Biltmore Hotel, Santa Barbara, California ..... October 12-14, 1953
Broadmoor Hotel, Colorado Springs, Colorado ..... October 21-23, 1954
The Homestead, Hot Springs, Virginia ..... October 27-29, 1955
Camelback Inn, Phoenix, Arizona ..... November 8-10, 1956
The Cloister, Sea Island, Georgia ..... November 11-13, 1957
The Royal York Hotel, Toronto, Canada ..... November 6-8, 1958
Del Monte Lodge, Pebble Beach, California..... October 18-21, 1959
Copley Sheraton Plaza, Boston, Massachusetts ..... October 5-8, 1960
Royal Orleans, New Orleans, Louisiana ..... November 7-10, 1962
El Mirador, Palm Springs, California ..... October 23-26, 1963
The Key Biscayne, Miami, Florida ..... November 11-14, 1964
Terrace Hilton Hotel, Cincinnati, Ohio ..... October 14-16, 1965
Fairmont Hotel & Towers, San Francisco,
California ..... October 17-19, 1966
The Key Biscayne, Miami, Florida ..... November 8-11, 1967
Broadmoor Hotel, Colorado Springs, Colorado ..... October 6-8, 1968
St. Regis Hotel, New York City ..... September 21, 1969
Camino Real, Mexico City, Mexico..... November 18-21, 1970
Sahara-Tahoe Hotel, Stateline, Nevada ..... September 26-30, 1971
New College, Oxford, England ..... September 4-7, 1972
Huntington-Sheraton Hotel, Pasadena, California November 14-17, 1973
Southampton Princess Hotel, Bermuda..... November 6-9, 1974
The Wigwam (Litchfield Park), Phoenix, Arizona ... November 5-8, 1975
Mills Hyatt House, Charleston, South Carolina .... November 10-13, 1976

Mauna Kea Beach Hotel, Kamuela, Hawaii ..... November 2-5, 1977
Hotel Bayerischer Hof, Munich, Germany ..... October 22-25, 1978
Hyatt Regency, Memphis, Tennessee ..... November 7-10, 1979
Walforf-Astoria Hotel, New York City, New York ..... October 1-4, 1980
Sheraton Plaza, Palm Springs, California ..... November 1-4, 1981
Ritz-Carlton Hotel, Boston, Massachusetts ..... October 10-13, 1982
The Lodge at Pebble Beach, California ..... October 23-26, 1983
The Homestead, Hot Springs, Virginia ..... October 17-20, 1984
The Lincoln Hotel Post Oak, Houston, Texas ..... October 27-30, 1985
The Cloister, Sea Island, Georgia ..... November 5-8, 1986
Hyatt Regency, San Antonio, Texas ..... October 7-10, 1987
Omni Netherland Plaza, Cincinnati, Ohio ..... September 13-17, 1988
Loews Ventana Canyon, Tucson,
Arizona ..... September 27-October 1, 1989
Amelia Island Plantation,amelia Island, Florida ..... October 2-7, 1990
Salishan Lodge, Gleneden Beach, Oregon ..... September 22-26, 1991
Ritz-Carlton Hotel, Naples, Florida ..... October 21-25, 1992
The Wigwam, Phoenix, Arizona ..... October 27-30, 1993
The Cloister, Sea Island, Georgia ..... November 3-6, 1994
Loews Ventana Canyon Resort, Tucson, Arizona ..... November 1-5, 1995
The Greenbrier, White Sulphur Springs,
West Virginia ..... September 18-22, 1996
Rimrock Resort, Banff, Alberta, Canada ..... September 10-14, 1997
Four Seasons Biltmore, Santa Barbara, California .... November 4-7, 1998
Ritz-Carlton,amelia Island, Florida ..... November 10-13, 1999
The Broadmoor, Colorado Springs, Colorado ..... October 11-14, 2000
The Breakers, Palm Beach, Florida ..... November 14-17, 2001
The Phoenician, Scottsdale, Arizona ..... October 16-19, 2002
Colonial Williamsburg,
Williamsburg, Virginia ..... October 29–November 1, 2003
Four Seasons Berlin and
Taschenbergpalais Dresden, Germany ..... October 3-8, 2004

PAST PRESIDENTS

Dean H. Echols..... 1938-39
Spence Braden..... 1940
Joseph P. Evans..... 1941
Francis Murphey 1942
Frank H. Mayfield..... 1943
A. Earl Walker 1944
Barnes Woodhall..... 1946
William S. Keith..... 1947
Howard A. Brown 1948
John Raaf..... 1949
E. Harry Botterell..... 1950
Wallace B. Hamby 1951
Henry G. Schwartz..... 1952
J. Lawrence Pool..... 1953
Rupert B. Raney..... 1954
David L. Reeves..... 1955
Stuart N. Rowe..... 1956
Arthur R. Elvidge..... 1957
Jess D. Herrmann 1958
Edwin B. Boldrey..... 1959
George S. Baker..... 1960
C. Hunter Shelden.... 1961-62
Samuel R. Snodgrass 1963
Theodore B. Rasmussen..... 1964
Edmund J. Morrissey 1965
George Maltby 1966
Guy L. Odom 1967
James G. Galbraith 1968
Robert H. Pudenz 1969-70
William B. Scoville 1971
Robert L. McLaurin 1972
Lyle A. French 1973
Benjamin B. Whitcomb..... 1974
John R. Green..... 1975
William H. Feindel 1976
William H. Sweet..... 1977
Arthur A. Ward 1978
Robert B. King 1979
Eben Alexander, Jr. 1980
Joseph Ransohoff II 1981
Byron C. Pevehouse 1982
Sidney Goldring 1983
Russel H. Patterson, Jr..... 1984
Thomas Langfitt..... 1985
Phanor L. Perot, Jr..... 1986
Shelley N. Chou 1987
James T. Robertson 1988
Thoralf M. Sundt, Jr. 1989
Robert Ojemann 1990
Nicholas Zervas..... 1991
Henry Garretson..... 1992
George Tindall 1993
William A. Buchheit 1994
David L. Kelly, Jr. 1995
John M. Tew, Jr..... 1996
Julian T. Hoff 1997
Edward S. Connolly 1998
J. Charles Rich 1999
George A. Ojemann..... 2000
Roberto C. Heros..... 2001
Donald O. Quest 2002
David G. Piepgras 2003

PAST VICE-PRESIDENTS

Francis Murphey 1941
William S. Keith.....1942
John Raaf.....1943
Rupert B. Raney.....1944
Arthur R. Elvidge.....1946
John Raaf 1947
Arthur R. Elvidge 1948
F Keith Bradford 1949
David L. Reeves.....1950
Henry G. Schwartz.....1951
J Lawrence Pool.....1952
Rupert B. Raney.....1953
David L. Reeves.....1954
Stuart N. Rowe.....1955
Jess D. Hermann.....1956
George S. Baker.....1957
Samuel R. Snodgrass.....1958
C Hunter Shelden.....1959
Edmund Morrissey.....1960
Donald F. Coburn 1961-62
Eben Alexander, Jr.....1963
George L. Maltby.....1964
Robert Pudenz.....1965
Francis A. Echlin.....1966
Benjamin Whitcomb 1967
Homer S. Swanson.....1968
Augustus McCravey..... 1969-70
Edward W. Davis 1971
John R. Green 1972
George J. Hayes 1973
Richard L. DeSaussure 1974
Ernest W. Mack..... 1975
Frank E. Nulsen..... 1976
Robert S. Knighton..... 1977
Robert G. Fisher 1978
H Thomas Ballantine, Jr..... 1979
George Ehni..... 1980
Courtland H. Davis, Jr..... 1981
John F. Mullan..... 1982
Hugo V. Rizzoli..... 1983
James W. Correll 1984
E Bruce Hendrick 1985
Griffith R. Harsh, III..... 1986
Ellis B. Keener 1987
Robert Grossman..... 1988
Jim Story 1989
John Jane, Sr..... 1990
Stewart Dunsker 1991
Burton M. Onofrio..... 1992
Martin H. Weiss 1993
John M. Tew, Jr..... 1994
John C. VanGilder..... 1995
Edward S. Connolly..... 1996
George Ojemann..... 1997
Charles H. Tator 1998
Donald O. Quest 1999
Howard M. Eisenberg..... 2000
Richard B. Morawetz..... 2001
Martin B. Camins 2002
Arthur L. Day 2003

PAST SECRETARY-TREASURERS

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

PAST SECRETARIES

Byron C. Pevehouse 1973
Russel H. Patterson, Jr. 1974-76
Phanor L. Perot, Jr..... 1977-80
John T. Garner..... 1981-83
James T. Robertson 1984-86
Nicholas T. Zervas..... 1987-89
William A. Buchheit..... 1990-92
Julian T. Hoff 1992-95
Roberto C. Heros 1995-98
David G. Piepgras..... 1998-01

PAST TREASURERS

Russel H. Patterson, Jr. 1973
Phanor L. Perot, Jr 1974-76
John T. Garner 1977-80
James T. Robertson 1981-83
Nicholas T. Zervas 1984-86
William A. Buchheit 1987-89
Julian T. Hoff 1990-92
Roberto C. Heros 1992-95
David G. Piepgras 1995-98
L. Nelson Hopkins..... 1998-01

HONORARY MEMBERS

Elected

GUY LAZORTHES (Annick)..... 1973
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KEIJI SANO (Yaeko)..... 1975
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SENIOR MEMBERS

Elected

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WILLIS BROWN, JR. (Elizabeth Ann {Ann}).....1984
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WILLIAM BUCHHEIT (Christa).....1980
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PAUL CHAPMAN 1983
Massachusetts General Hospital
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HARVEY CHENAULT (Billee).....1949
952 Edgewater Drive
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W. KEMP CLARK (Fern) 1970
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WILLIAM COLLINS, JR. (Gwendolyn).....1963
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12134 Putting Green Row
San Diego, CA 92128
858-673-9025

EDWARD CONNOLLY (Elise).....1972
Department of Neurosurgery, 7th Floor
Ochsner Foundation Clinic
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PAUL COOPER (Leslie) 1995
Department of Neurosurgery/NYU Medical Center
550 First Avenue
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212-263-6514, fax 212-263-8225, paul.cooper@med.nyu.edu

COURTLAND DAVIS, JR. (Carrie) 1967
2525 Warwick Road
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336-723-7296, chdccdmd@triad.rr.com

RICHARD DESAUSSURE, JR. (Phyllis).....1962
4290 Heatherwood Lane
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901-761-5230, desrichard@aol.com

DONALD DOHN (Carolyn) 1968
P.O. Box 998
Point Clear, AL 36564
251-928-7670, fax 251-990-3738

STEWART DUNSKER (Ellen) 1975
551 Abilene Trail
Cincinnati, OH 45215
513-522-0330, fax 513-522-0333, dunsker@aol.com

HOWARD EISENBERG 1985
Department of Neurosurgery
22 S. Greene Street, 12 D South
Baltimore, MD 21201
410-328-3514, fax 410-328-1420, heisenberg@smail.umaryland.edu

MEL EPSTEIN (Lynn) 1992
411 Poppasquash Road
Bristol, RI 02809
401-254-5083, fax 401-253-6422, melepstein@earthlink.net

WILLIAM FEINDEL (Faith) 1959
Montreal Neurological Institute
3801 University Street
Montreal, Quebec H3A 2B4
CANADA
514-398-1939, fax 514-398-1375

ROBERT FISHER (Constance)..... 1955
12419 Woodley Avenue
Granada Hills, CA 91344-1817

EUGENE FLAMM (Susan) 1979
Albert Einstein College of Medicine
Montefiore Medical Center
111 East 210th Street
Bronx, NY 10467
718-920-7400, fax 718-515-8235, eflamm@montefiore.org

ELDON FOLTZ (Catherine)..... 1960
2480 Monaco Drive
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949-494-3422, fax 949-494-8947, eldonlfoltzmd@aol.com

RICHARD FRASER (Anne) 1976
5 Ellis Court
Rye NY 10580
914-967-6910

LYLE FRENCH (Gene) 1954
7501 East Thompson Peak Parkway
Casita 509
Scottsdale AZ 85255-4540
480-659-6286

JOHN GARNER (Candace) 1971
2834 Dove Run Creek Drive
Las Vegas, NV 89135
949-275-2357, jtgrex@aol.com

HENRY GARRETSON (Marianna) 1973
517 Tiffany Lane
Louisville, KY 40207
502-896-8270, fax 502-896-8437
tiffanyhmg2@aol.com

29 Surfsong Road
Kiawah Island, SC 29455
843-768-7260, fax 843-768-7282

SIDNEY GOLDRING (Lois) 1964
11430 Conway Road
St. Louis, MO 63131
314-567-1171

PHILIP GORDY (Silvia) 1968
3601 Carmel Drive
Casper, WY 82604-4949
307-265-7883

ROBERT GROSSMAN (Ellin) 1984
Department of Neurosurgery
Baylor College of Medicine
6560 Fannin, Suite 944
Houston, TX 77030
713-798-4696, fax 713-798-3739, grossman@bcm.tmc.edu

ROBERT GRUBB, JR. (Julia) 1985
Department of Neurological Surgery, Box 8057
Washington University School of Medicine
660 South Euclid Avenue
St. Louis, MO 63110
314-362-3567, fax 314-362-2107, grubbr@nsurg.wustl.edu

JOSEPH HAHN (Andrea)..... 1993
Department of Neurosurgery, S 80
The Cleveland Clinic Foundation
9500 Euclid Avenue
Cleveland, OH 44195
216-444-5802, fax 216-444-1011, hahnj@ccf.org

GRIFFITH HARSH, III (Craig)..... 1980
P.O. Box 232
Sweetwater, TN 37874
423-337-7483, gharsh3@aol.com

M. PETER HEILBRUN (Robyn) 1984
PO Box 920041
Snowbird UT 84092-0041
801-742-0516
mpeterheilbrun@msn.com

115 Barbaree Way
Tiburon CA 94920
415-381-8700

ROBERTO HEROS (Deborah)..... 1985
Department of Neurological Surgery
1095 NW 14th Terrace (D4-6)
Miami, FL 33136
305-243-4572, fax 305-243-3180, rheros@med.miami.edu

CHARLES HODGE, JR. (Catherine)..... 1982
Department of Neurosurgery
611 Jacobsen Hall
750 East Adams Street
Syracuse, NY 13210
315-464-5510/5511, fax 315-464-6384, hodgec@upstate.edu

JULIAN HOFF (Diane)..... 1975
Department of Neurosurgery
University Hospitals
1500 East Medical Center Drive, Box 038
Ann Arbor, MI 48109
734-936-5015, fax 734-936-9294, jhoff@umich.edu

HAROLD HOFFMAN (Jo Ann).....1982
110 Blorr Street West, Apartment 607
Toronto, Ontario M5S 2W7
CANADA
416-964-7775, fax 416-964-7848, j.hoffman@sympatico.ca

EDGAR HOUSEPIAN (Marion Grace).....1976
710 West 168th Street
New York, NY 10032
212-305-5256, fax 212-305-3250, emh4@columbia.edu

ALAN HUDSON (Susan) 1978
Cancer Care Ontario
620 University Avenue
Toronto, Ontario M5G 2L9
CANADA
416-971-5100 x1610, fax 416-971-5940,
alan.hudson@cancercare.on.ca

JOHN JANE, SR. (Noella).....1982
Department of Neurosurgery
University of Virginia Health System
PO Box 800212
Charlottesville, VA 22908
434-982-3244, fax 434-243-2954, kes4a@virginia.edu

PETER JANNETTA (Diana).....1994
Allegheny General Hospital
420 East North Avenue, Suite 302
Pittsburgh, PA 15212
412-359-4810, fax 412-359-4811, pjannett@wpahs.org

ELLIS KEENER (Ann).....1978
915 East Lake Drive
Gainesville, GA 30506
770-532-5616, ebkeener@bellsouth.net

DAVID KELLY, JR. (Sarah {Sally}) 1975
Wake Forest University Baptist Medical Center
Medical Center Boulevard
Winston-Salem, NC 27157-1029
336-716-4049, 336-716-3065, dkelly@wfubmc.edu

PATRICK KELLY (Carol).....1992
530 First Avenue, Suite 8R
New York, NY 10016
212-263-8002, 212-263-8031, patrick.kelly@med.nyu.edu

ROBERT KING (Molly).....1958
S.U.N.Y. Upstate Medical University
750 E. Adams Street
Syracuse, NY 13210
315-464-4470, fax 315-464-5520, kingrb@upstate.edu

WOLFF KIRSCH (Marie-Claire).....1971
Neurosurgery Center for Research, Training, and Education
Loma Linda University
11175 Campus Street, Suite 11113
Loma Linda, CA 92350
909-558-7070, fax 909-558-0472, wkirsch@som.llu.edu

DAVID KLINE (Helen {Nell}).....1971
Department of Neurosurgery
LSUHSC
1542 Tulane Avenue
New Orleans, LA 70112
504-568-2641, fax 504-568-6127, vprout@lsuhsc.edu

THOMAS LANGFITT (Carolyn).....1971
Glenmede Corporation
One Liberty Place, Suite 1200
1650 Market Street
Philadelphia, PA 19103-7391
215-419-6010, fax 215-419-6196,

SANFORD LARSON (Jacquelyn).....1989
Department of Neurosurgery
Medical College of Wisconsin
9200 W. Wisconsin Avenue
Milwaukee, WI 53226
414-805-5407

EDWARD LAWS, JR. (Margaret {Peggy}).....1983
Department of Neurosurgery, Box 800212
University of Virginia
Charlottesville, VA 22908-0212
434-924-2650, fax 434-924-5894, el5g@virginia.edu

RAEBURN LLEWELLYN (Carmen).....1963
3 Poydras Street, Unit 6D
New Orleans, LA 70130
504-581-5252, fax 504-523-1520, carsonllewellyn@yahoo.com

DONLIN LONG (Harriett).....1983
600 North Wolfe Street
Carnegie 466
Baltimore, MD 21287
410-614-3536, fax 410-955-6407, dmlong@jhmi.edu

WILLIAM LOUGHEED.....1962
178 Kempenfelt Drive
Barrie, Ontario L4M 1C3
CANADA
705-728-8521

JOHN LOWREY (Catherine {Katy}).....1965
65-1160 Kokuala Road
Kamuela, HI 96743
808-885-5867

ALFRED LUESSENHOP (Frances).....1977
4524 Foxhall Crescents
Washington, DC 20007
202-338-1914, franhop@aol.com
(May-October)

27160 Oak Knoll Drive
Bonita Springs, FL 34134
239-495-9923
(November-April)

LEONARD MALIS (Ruth).....1973
219-44 Peck Avenue
Hollis Hills, NY 11427
718-479-9326, fax 718-479-9328, nsdoclen@aol.com

ROBERT MAXWELL (Karen).....1992
Department of Neurosurgery, MMC 96
420 Delaware Street SE
Minneapolis, MN 55455
612-626-0137, fax 612-624-0644, rem_kam@yahoo.com

ROBERT MCLAURIN (Sally).....1955
900 Fourth & Vine Tower
5 W Fourth Street
Cincinnati, OH 45202-3688
513-381-9200, fax 513-381-9206, mclaurin@one.net

J. MICHAEL MCWHORTER (Barbara).....1989
Carolina Neurosurgical Associates
2810 North Maplewood Avenue
Winston-Salem, NC 27103-4151
336-768-1811, fax 336-768-0360, jmcwhorter@triad.rr.com

JOHN MULLAN (Vivian) 1963
5844 Stony Island Avenue
Chicago, IL 60637
773-241-6546, fax 773-241-6546 #51

BLAINE NASHOLD, JR. (Irene) 1967
Department of Neurosurgery
Duke University Medical Center, Box 3807
Durham, NC 27710
919-684-2937, fax 919-684-2026, nasho002@aol.com

GEORGE OJEMANN (Linda) 1975
Department of Neurosurgery
University of Washington
Campus Box 356470
1959 N.E. Pacific Street
Seattle, WA 98195
206-543-3570, fax 206-543-8315, gojemann@u.washington.edu

ROBERT OJEMANN (Jean) 1968
Massachusetts General Hospital
Fruit Street
Boston, MA 02114
617-726-2936, fax 617-726-3126, rojemann@partners.org

ANDRE OLIVIER (Nicole Poulin).....1989
Montreal Neurological Hospital
3801 University Street, #109
Montreal, Quebec H3A 2B4
CANADA
514-398-1935, fax 514-398-2811, luisa.birri@mcgill.ca

BURTON ONOFRIO (Judy) 1975
1105 Tenth Street SW
Rochester, MN 55902
507-289-3684, fax 507-529-9469

RUSSEL PATTERSON, JR. (Juliet {Julie}) 1971
146 West 57th Street
Apartment 65A
New York, NY 10019-3301
212-586-9237, fax 212-315-3877, mail49212@pop.net

SYDNEY PEERLESS (Ann) 1977
2721 Hibiscus Court
Punta Gorda, FL 33950
(November-April)

21171 Thames Road
Appin, Ontario NOL 1AO
CANADA (May-October)

941-833-5710, fax (same #), speerless@earthlink.net

PHANOR PEROT, JR. 1970
MUSC Neurosurgery Department
96 Jonathan Lucas Street, Suite 428, PO Box 250 616
Charleston, SC 29425
843-792-2424, fax 843-792-0468, perotplj@musc.edu

BYRON CONE PEVEHOUSE (Lucy) 1964
13623 N.E. 32nd Place
Bellevue, WA 98005-1400
425-881-5531, fax 425-881-5831, byron.pevehouse@verizon.net

DAVID PIEPGRAS (Jane).....1987
Department of Neurologic Surgery
Mayo Clinic, Gonda 8-209
200 First Street SW
Rochester, MN 55905
507-284-2254, fax 507-284-5206, piepgras.david@mayo.edu

ROBERT PORTER (Dean) 1962
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DONALD QUEST (Ilona) 1986
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215-305-5582, fax 212-305-2026, doq1@columbia.edu

ROBERT RATCHESON (Peggy) 1986
University Hospitals of Cleveland
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ALBERT RHOTON, JR. (Joyce) 1984
Department of Neurosurgery
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J. CHARLES RICH, JR. (Jasmine).....1987
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HUGO RIZZOLI 1973
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EDWIN B. BOLDREY San Francisco, California (Senior)	1941	1988
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