THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

65th Annual Meeting

Colonial Williamsburg
Williamsburg, Virginia

October 29 – November 1, 2003

American Association of Neurological Surgeons

Jointly Sponsored by the American Association of Neurological Surgeons

FUTURE MEETINGS

2004

October 3–8
Joint Meeting
Germany

2005

September 21–24
Ritz-Carlton Half Moon Bay
Half Moon Bay, CA

2006

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

Mark your calendars now!

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GENERAL INFORMATION

HOTEL INFORMATION

Colonial Williamsburg
310 S. England Street
Williamsburg, VA 23185

Telephone: 757-220-7976
Facsimile: 757-565-8243

A fee of $1.00 per page is charged for incoming faxes. A fee of $3.00 for the first page and $1.00 for each additional page is charged for outgoing faxes. These fees may be posted to a room account.

REGISTRATION

Meeting Registration will be located in Williamsburg Lodge of Colonial Williamsburg each day.

REGISTRATION DESK HOURS:

Wednesday, October 29	East Gallery, North Wall	12:00 PM – 6:30 PM
Thursday, October 30	Lower Level	6:30 AM – 12:30 PM
Friday, October 31	Lower Level	6:30 AM – 12:00 PM
Saturday, November 1	Lower Level	6:30 AM – 12:00 PM

PROGRAM SUMMARY

All events will occur in the Williamsburg Lodge unless otherwise noted.

WEDNESDAY, OCTOBER 29

EVENT	TIME	LOCATION
Registration	12:00 PM – 6:30 PM	East Gallery, North Wall
Executive Committee Meeting	2:00 PM – 5:00 PM	Room E
OPENING RECEPTION		
Cocktail Dinner (Dressy Attire)	6:30 PM – 9:30 PM	Oval Garden of the Abby Aldrich Rockefeller Folk Art Museum

PROGRAM SUMMARY

All events will occur in the Williamsburg Lodge unless otherwise noted.

THURSDAY, OCTOBER 30

EVENT	TIME	LOCATION
Registration	6:30 AM – 12:30 PM	Lower Level
Business Breakfast Meeting For Academy Members Only	6:30 AM – 7:30 AM	North Ballroom
Breakfast for Spouses and Guests 7:00 AM – 9:00 AM Buffet 9:00 AM – 11:00 AM Continental Breakfast	7:00 AM – 11:00 AM	Rooms A & B
Scientific Session	7:30 AM – 12:30 PM	Tidewater Room
PROGRAM FOR SPOUSES		
Introduction to Colonial Williamsburg	9:00 AM	Rooms A & B
Book Review	10:00 AM	Rooms A & B
OTHER ACTIVITIES		
Golf Tee Time*	12:40 PM	Gold Course
Walking Tour of Williamsburg*	1:00 PM – 4:00 PM	Meet in East Gallery
DINNER		
Reception & Dinner (Casual Attire)	6:30 PM – 9:30 PM	
Reception	6:30 PM – 7:30 PM	West Terrace
Drum & Fife Walk to Shields Tavern	7:30 PM	Meet in East Gallery
Dinner	7:45 PM – 9:30 PM	

*Activities that require prior registration

PROGRAM SUMMARY

All events will occur in the Williamsburg Lodge unless otherwise noted.

FRIDAY, OCTOBER 31

EVENT	TIME	LOCATION
Registration	6:30 AM – 12:00 PM	Lower Level
Business Breakfast Meeting **For Academy Members Only**	6:30 AM – 7:30 AM	North Ballroom
Breakfast for Spouses And Guests 7:00 AM – 9:00 AM Buffet 9:00 AM – 11:00 AM Continental Breakfast	7:00 AM – 11:00 AM	Dominion Room
Scientific Session	7:30 AM – 11:15 AM	Tidewater Room
Presidential Address David G. Piepgras, M.D.	11:15 AM – 12:00 PM	Tidewater Room
PROGRAM FOR SPOUSES		
Conversation with Meg Whitman, CEO of eBay	10:00 AM	Dominion Room
OTHER ACTIVITES		
Golf Tee Time*	12:30 PM	Gold Course
Lunch and Tour of Yorktown*	12:30 PM – 5:00 PM	Meet in East Gallery
DINNER		
PRESIDENTIAL RECEPTION **All members, spouses, and guests are invited**	7:00 PM – 8:00 PM	North Gallery
BLACK TIE DINNER & DANCE	8:00 PM – 11:00 PM	Virginia Room

* Activities that require prior registration

PROGRAM SUMMARY

All events will occur in the Williamsburg Lodge unless otherwise noted.

SATURDAY, NOVEMBER 1

EVENT	TIME	LOCATION
Registration	6:30 AM – 12:30 PM	Lower Level
Breakfast for **All Members, Spouses, & Guests**	6:30 AM – 11:00 AM	Rooms A, B, & C
Scientific Session	7:30 AM – 12:30 PM	Tidewater Room
Meeting Adjourns	12:30 PM	

SCHEDULE OF ACTIVITES FOR SPOUSES

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

Wednesday, October 29

6:30 – 9:30 PM	Opening Cocktail Dinner – *Oval Garden of the Abby Aldrich Rockefeller Folk Art Museum*-Dressy Attire

Thursday, October 30

7:00 - 11:00 AM	Spouse & Guest Breakfast – *Williamsburg Lodge Rooms A & B* 7:00 – 9:00 AM Breakfast 9:00 – 11:00 AM Continental
9:00 AM	Introduction to Colonial Williamsburg *Williamsburg Lodge Rooms A & B*
10:00 AM	Book Review – Bel Canto *Williamsburg Lodge Rooms A & B*
1:00 – 4:00 PM	Walking Tour of Williamsburg *Meet in East Gallery of Williamsburg Lodge**
12:40 PM	Golf Tee Time - *Gold Course**
6:30 PM	Dinner – *Shields Tavern* 6:30 – 7:30 PM Reception – *West Terrace* 7:30 PM – *Drum and Fife Walk to Shields Tavern – Meet in East Gallery of Williamsburg Lodge* 7:45 – 9:30 PM-Dinner-Casual Attire

SCHEDULE OF ACTIVITES FOR SPOUSES (continued)

Friday, October 31	
7:00 - 11:00 AM	Spouse & Guest Breakfast – *Dominion Room of Williamsburg Lodge* 7:00 – 9:00 AM Breakfast 9:00 – 11:00 AM Continental
10:00 AM	Conversation with Meg Whitman, CEO of eBay – *Dominion Room of Williamsburg Lodge*
11:15 AM – 12:00 PM	**Presidential Address** by David G. Piepgras, M.D. – *Tidewater Room of Williamsburg Lodge*
12:30 PM	Golf Tee Time - *Gold Course**
12:30 – 5:00 PM	Lunch and Tour of Yorktown – *Meet in East Gallery of Williamsburg Lodge**
7:00 – 8:00 PM	Presidential Reception – *North Gallery of Williamsburg Lodge*
8:00 PM	Black Tie Dinner and Dance – *Virginia Room of Williamsburg Lodge*
Saturday, November 1	
6:30 – 11:00 AM	Members, Spouse & Guest Breakfast – *Rooms A, B, & C of Williamsburg Lodge* 6:30 – 9:00 AM Breakfast 9:00 – 11:00 AM Continental
12:30 PM	Meeting Adjourns

**Activities that require prior registration.*

SCIENTIFIC PROGRAM
AMERICAN ACADEMY
OF NEUROLOGICAL SURGERY
2003 LEARNING OBJECTIVES

Jointly sponsored by The American Association of Neurological Surgeons October 29 – November 1, 2003.

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

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

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

American
Association of
Neurological
Surgeons

This activity has been planned and implemented in accordance with the Essentials and Standards of the Accreditation Council for Continuing Medical Education through the joint sponsorship of the American Association of Neurological Surgeons and the American Academy of Neurological Surgery. The American Association of Neurological Surgeons is accredited by the by the Accreditation Council for Continuing Medical Education (ACCME) to sponsor continuing medical education for physicians.

The American Association of Neurological Surgeons designates this educational activity for a maximum of 13.75 category 1 credits towards the AMA Physician's Recognition Award. Each physician should claim only those hours of credit that he/she actually spent in the educational activity.

DISCLOSURE INFORMATION

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

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

Faculty Name	Disclosure	Type of Relationship
Freeman TB	Provisional Patent	Other Financial or Material Support
Fults, DW	Brain Tumor Society	Grants/Research Support
Hopkins, LN	Boston Scientific, Cordis, BSC/EPI, Endotex, Guidani, Micrus	Grants/Research Support, Consultants
	BSC/EPI	Stock Shareholder
	Cordis, Guidant	Honorarium
	EPI, Endotex, Guidant Micrus	Other Financial or Material Support
Horowitz, M	Target	Grants/Research Support, Consultants
	Micrus Corporation	Other Financial or Material Support
Kondziolka, D	Electa Instruments	Consultants
Rosenwasser, RH	Boston Scientific	Consultants, Stock Shareholder
Sonntag, VKH	Medtronic Sofamor Danek	Consultants
	Metronic, Johnson & Johnson, Acromed Depuy	Other Financial or Material Support
Yu, JS	NIH	Grants/Research Support
	Reneuron, Inc.	Consultants

*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

Aghi, M
Al-Mefty, O
Badie, B
Barbaro, NM
Barker, FG
Barnett, GH
Berger, MS
Boop, FA
Burchiel, KJ
Curry, W
Dempsey, RJ
Fenstermaker, RA
Foley, KT
Harbaugh, RE
Harkey, HL
Howard, MA
Krieger, MD
Laws, ER
Levy, CL
Lunsford, LD
Mapstone, TB
Morgan M
Moriarty, TM
Neimat, JS
Newell, DW
Ogilvy, CS
Pollock, BE
Rutka, JT
Sawaya, R
Shields, CB
Simeone, FA
Souweidane, MM
Tamargo, RJ
Zadeh G

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

Faculty Name

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

Faculty Name

Horowitz, M
Freeman, TB
Yu, JS

SCIENTIFIC PROGRAM
AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

THURSDAY, OCTOBER 30 **Moderator: Fredric B. Meyer, M.D.**

VASCULAR

POINT COUNTERPOINT
The ISAT and ISUIA Studies Demonstrate that Coiling is the Treatment of Choice for Intracranial Aneurysms.

7:30 – 7:45 AM — POINT: L. Nelson Hopkins

7:45 – 8:00 AM — COUNTERPOINT: Robert E. Harbaugh

8:00 – 8:15 AM — Audience Participation

PAPER PRESENTATIONS

8:15 – 8:30 AM — Endovascular Reconstruction for Wide Neck Cerebral Aneurysms with a New Intracranial Stent. Robert H. Rosenwasser, RP Benitez, E Veznedaroglu

8:30 – 8:45 AM — Thirty-Day Procedure-Related Morbidity Associated with Endovascular Treatment of Intracranial Aneurysms. Michael Horowitz, A Kassam, H Park, C Jungreis

8:45 – 9:00 AM — Computed Tomographic Angiography in Place of Catheter Angiography as the Only Diagnostic and Pretreatment Planning Study for Ruptured and Unruptured Cerebral Aneurysms by a Combined Neurovascular Team. Christopher S. Ogilvy, BL Hoh, AC Cheung, JD Rabinov, JC Pryor, BS Carter

THURSDAY, OCTOBER 30 (continued)

9:00 – 9:15 AM	Aneurysm Shape May be a Better Predictor of Rupture than Aneurysm Size. Robert E. Harbaugh, ML Raghavan
9:15 – 9:30 AM	What Makes a Carotid Atherosclerotic Plaque Symptomatic? Microarray Analysis of Gene Expression. Robert J. Dempsey, R Vemuganti, VK Dhodda, T Varghese, S Salamat
9:30 – 9:45 AM	Anti-CD11/CD18 Monoclonal Antibody Prevents Vasospasm after Subarachnoid Hemorrhage in Rabbits. Rafael J. Tamargo, G Pradilla, PP Wang, FG Legnani, L Ogata, GN Dietsch
9:45 – 10:00 AM	Surgical Risks Associated with the Management of Grade I and II Brain Arteriovenous Malformations. Michael K. Morgan
10:00 – 10:15 AM	Beverage Break
SESSION II	**PAPER PRESENTATIONS**
10:15 – 10:30 AM	Software Solutions for Softer Hours: Compliance with the 80-Hour Work Week. David W. Newell
10:30 – 10:45 AM	Human Fetal Tissue Transplantation for the Treatment of Parkinson's Disease (PD): A Surgical Placebo Controlled Trial. Thomas B. Freeman, CG Goetz, JH Kordower, AJ Stoessl, MF Brin, KM Shannon, D Perl, J Godbold, CW Olanow
10:45 – 11:00 AM	GABA Transmission in Human Focal Cortical Dysplasia. Nicholas M. Barbaro, ME Calcagnotto, SC Baraban

THURSDAY, OCTOBER 30 (continued)

11:00 – 11:15 AM	Correlation of Preoperative MR Imaging and Intraoperative Observations of Neurovascular Compression in Patients with Trigeminal Neuralgia. Kim J. Burchiel, MA Sandquist
11:15 – 11:30 AM	Local Cortical Cooling as a Functional Mapping Tool. Matthew A. Howard, III
11:30 – 11:45 AM	Magnetic Resonance Spectroscopy of Atypical Diffuse Pontine Masses. Mark D. Krieger, S Bluml, JG McComb
11:45 AM – 12:00 PM	EGFR Intron Recombination in Gliomas: Oncogenesis via Corrupt Immune Diversification? Robert A. Fenstermaker, MJ Ciesielski
12:00 – 12:15 PM	Identification of Differentially Expressed and Developmentally Regulated Genes in Medulloblastoma Using Suppression Subtractive Hybridization. N Yokota, M Loreto, T Mainprize, S Ueda, James T. Rutka
12:15 – 12:30 PM	Neurosurgery: An Expanding Field With a Declining Work Force? L. Dade Lunsford, A Kassam

FRIDAY, OCTOBER 31 **Moderator: James T. Rutka, M.D., Ph.D.**

TUMOR

POINT COUNTERPOINT
Glioblastoma Multiforme: Progress in the Last Thirty Years

7:30 – 7:45 AM — POINT: Edward R. Laws

7:45 – 8:00 AM — COUNTERPOINT: Mitchel S. Berger

8:00 – 8:15 AM — Audience Participation

PAPER PRESENTATIONS

8:15 – 8:30 AM — The Influence of Inferior Petrosal Sinus Sampling on the Outcome of Transsphenoidal Surgery for Cushing's Disease
S Aytug, ML Vance, Edward R. Laws

8:30 – 8:45 AM — Intraoperative Subcortical Stimulation Mapping During Resection of Hemispheric Peri-Rolandic Gliomas Located Within or Adjacent to the Descending Motor Pathways: Evaluation of Morbidity and Assessment of Functional Outcome in 294 Patients.
Mitchel S. Berger, D Lundin, GA Ojemann, K Lamborn, E Keles

8:45 – 9:00 AM — Management of Oligodendroglioma Based on Molecular Characteristics: Four Year Experience. Gene H. Barnett, AA Kanner, S Staugaitis, O Chernova, R Prayson, S Lee, D Peereboom, BH Cohen, G Stevens, MA Vogelbaum, SA Toms

9:00 – 9:15 AM — Generation of Brain Tumor Neovasculature from Transplanted Bone Marrow. Manish Aghi, K Cohen, DT Scadden, EA Chiocca

9:15 – 9:30 AM	G207 Infection of Dendritic Cells: Effects on Maturation and Generation of Antitumor Immunity. William T. Curry, RL Martuza, SD Rabkin
9:30 – 9:45 AM	Results of a Phase II Trial of Resection and Conformal Radiation Therapy for Pediatric Patients with Localized Ependymoma. Frederick A. Boop, RA Sanford, TE Merchant, MJ Krasin, LE Kun, T Williams, C Li, X Xiong, RK Mulhern, RB Khan, RH Lustig, RK Danish
9:45 – 10:00 AM	Radiosurgery for Brain Metastasis Neurological Outcome Versus Tumor Location. M Maldaun, F Lang, D Suki, Raymond Sawaya
10:00 – 10:15 AM	Beverage Break
	PAPER PRESENTATIONS
10:15 – 10:30 AM	Malignant Progression in Meningioma; Documentation of a Series and Analysis of Cytogenetic Findings. Ossama Al-Mefty, P Kadri, S Pravdenkova, J Sawyer, M Husain
10:30 – 10:45 AM	Long-term Results after Radiosurgery for Benign Intracranial Tumors. Douglas Kondziolka, N Nathoo, JC Flickinger, A Niranjan, AH Maitz, LD Lunsford
10:45 – 11:00 AM	***ACADEMY AWARD PAPER*** Targeting the Tie2/Tek Receptor in Astrocytomas. Gelareh Zadeh, B Qian, N Sabhai, A Okhowati, CD Kontos, A Guha

11:00 –11:15 AM ***ACADEMY AWARD RUNNER UP***
Survival but not Migration of Engrafted Schwann Cells Expressing Alkaline Phosphatase Marker Gene after Transplantation into Contused Spinal Cord of Adult Fisher Rats. Charles Levy, J Pei, L Wirthlin, G Perez-Abadia, C Maldonado, XM Xu

11:15 AM – 12:00 PM ***PRESIDENTIAL ADDRESS***
"Perspectives on Life and Health in 18th Century Virginia as Reflected in the Diaries of Landon Carter"
David G. Piepgras

SATURDAY, NOVEMBER 1 **Moderator: Jon H. Robertson, M.D.**

SPINE

POINT COUNTERPOINT
Lumbar Fusion: Which is the Better Approach?

7:30 – 7:45 AM — POINT: Volker Sonntag – Posterolateral Transverse Process Fusion

7:45 – 8:00 AM — COUNTERPOINT: Kevin Foley – The Case for Lumbar Interbody Fusion

8:00 – 8:15 AM — Audience Participation

PAPER PRESENTATIONS

8:15 – 8:30 AM — The Effect of the Malpractice Litigation Crisis on Academic Neurosurgery. Frederick A. Simeone

8:30 – 8:45 AM — Clinical Evolution of a Metal-on-Metal Cervical Disc Prosthesis. R Haid, Vincent Traynelis, T Zdeblick, J Robertson

8:45 – 9:00 AM — Randomized, Prospective, Controlled Clinical Trial of Pulsed Electromagnetic Field Stimulation for Cervical Fusion. Kevin Foley

9:00 – 9:15 AM — Low Dose Irradiation Following Spinal Cord Injury Reduces Inflammatory Cell Infiltration and Lesion Severity. Christopher B. Shields, RA Gray, YP Zhang, Y Han, DN Loy, MD Mills, L Fajardo, D Sun, S Whittemore

9:15 – 9:30 AM — The Management of Tethered Spinal Cord in The Adult Population. Timothy B. Mapstone

9:30 – 9:45 AM	Lumbar Microhemilaminoforamenotomy for Spinal Stenosis. H. Louis Harkey
9:45 – 10:00 AM	Gilliatt-Sumner Hand Revisited. Gabriel C. Tender, AJ Thomas, N Thomas, DG Kline
10:00 – 10:15 AM	Beverage Break
	PAPER PRESENTATIONS
10:15 – 10:30 AM	A Prospective Study Comparing Outcomes after Vestibular Schwannoma Resection and Radiosurgery: Design, Patient Enrollment, and Early Results. Bruce E. Pollock, CLW Driscoll, DA Gorman, MJ Link, JN Mandrekar, SG Harner, MJ Ebersold, RL Foote, KN Krecke, CH Johnson
10:30 – 10:45 AM	MRI Measurement of CBF and CSF Flow Dynamics: Supine vs. Upright Changes in Cerebrovascular Physiology. Thomas M. Moriarty, N Alperin, SG Hushek, A Sivaramakrishnan, RF Moser, NM Hoerter, CB Shields
10:45 – 11:00 AM	Microglia Activation in Brain Tumors. Behnam Badie
11:00 – 11:15 AM	Craniotomy for Resection of Pediatric Brain Tumors in the United States, 1988-2000: The Effect of Progressive Centralization and Specialization of Care. Fred G. Barker
11:15 – 11:30 AM	Hemorrhagic Complications of Endoscopic Neurosurgery for Brain Tumors. Mark M. Souweidane
11:30 – 11:45 AM	IGF2 Enhances Sonic Hedgehog-Induced Medulloblastoma in Mice. G Rao, CA Pedone, Daniel W. Fults

11:45 AM – 12:00 PM	Vaccination of Patients with Malignant Glioma with Tumor Lysate-Pulsed Dendritic Cells Elicits Antigen-Specific Cytotoxicity. John S. Yu, CJ Wheeler, G Liu, H Ying, WH Yong, A Das, KL Black
12:00 – 12:15 PM	Computer-Assisted Anatomic Analysis of Transoral Surgical Approaches to the Clivus. V Balasingam, GJ Anderson, N Gross, C-M Cheng, PE Anderson, A Noguchi, A Dogan, SO McMenomey, Johnny B. Delashaw, Jr. Johnny B. Delashaw, Jr.,
12:15 – 12:30 PM	The Role of the Basal Ganglia in Voluntary Movement Selection. Joseph S. Neimat, EN Eskandar, JA Assad

THURSDAY, OCTOBER 30 **8:15 – 8:30 AM**

Endovascular Reconstruction for Wide Neck Cerebral Aneurysms with a New Intracranial Stent

Robert H. Rosenwasser, M.D., Ronald P. Benitez, M.D., Erol Veznedaroglu, M.D.

Object. The long-term durability of endovascular occlusion of cerebral aneurysms is one of the major limiting factors for more widespread use of this technique. We report the use of a new intracranial stent, the Neuroform ™ microstent (Boston Scientific, Fremont CA), for the treatment of wide neck cerebral aneurysms.

Methods. After institutional IRB approval was obtained, patients identified as harboring a wide neck intracranial aneurysm were considered for stent assisted coiling. After appropriate anticoagulation, depending on whether the aneurysm was ruptured or unruptured, the Neuroform ™ stent was delivered across the neck of the aneurysm and deployed with a coil pusher. Following stent placement, standard coil occlusion of the aneurysm was achieved in the majority of cases.

Results. Fifty-seven patients were identified as having wide neck intracranial aneurysms suitable for stent assisted coiling. A total of 50 aneurysms in 49 patients were treated with stent assisted coiling. There were 8 stent deployment failures. Forty two of the aneurysms were initially stented followed by coil placement. Six aneurysms were stented only and one aneurysm was initially coiled followed by stent placement. There were five mortalities (8.8%), with one secondary to the procedure (1.8%). Four patients experienced thromoembolic events (7%); three were considered to be secondary to the procedure (5.3%). In addition there were two femoral pseudoaneurysms. The overall complication rate was 10.5%. Five patients are also available for follow up angiographic evaluation and will be discussed.

Conclusion. Intracranial stenting may overcome important technical limitations in current endovascular therapy by improving occlusion of wide-necked aneurysms.

Thirty-Day Procedure-Related Morbidity and Mortality Associated with Endovascular Treatment of Intracranial Aneurysms

Michael Horowitz, M.D.. Amin Kassam, M.D., Haekwan Park, M.D., Charles Jungreis, M.D.

Department of Neurosurgery, University of Pittsburgh Medical Center, Pittsburgh, PA

A retrospectice analysis of procedure related endovascular complications between October 1998 and October 2002 (49 months) in 180 patients with 190 aneurysms (118 ruptured, 72 unrutpured) was carried out. 210 procedures were analyzed including three failures. Immediate complete occlusion was achieved in 61%, near complete occlusion was achieved in 22%, partial occlusion was achieved in 16%, and failure occurred in 2.8% of all lesions. A total of 37 procedure related complications were recorded (17.6%) including cerebral thromboembolism (10.4%), aneurysm perforation (4.2%), coil migration (1%), parent vessel injuries (1%), post procedure aneurysm rupture (0.5%), and cranial nerve palsy (0.5%). Morbidity and mortality from perforation was 0% and 33%, respectively. The single post procedure rupture resulted in death. Of the 22 thromboembolic events, ten resulted in persistent neurologic deficits at discharge (45%) and five resulted in death (23%). For the entire series of 210 aneurysms, morbidity and mortality were each 4.8%. Procedure related complications in ruptured aneurysms was higher than the overall group (22.9% vs 17.6%). Procedure related morbidity and mortality for 118 ruptured aneurysms was also higher at 5.9% and 7.6% respectively. Procedure related complications for unruptured aneurysms was lower than for the group as a whole or for ruptured lesions. Overall complication rate in unruptured lesions was 8.3% with morbidity and mortality of 1.4% each. Complications were highest for re-embolized aneurysms with a 20% incidence, each of which was thromboembolic. 50% of these complications lead to persistent neurologic changes at discharge.

One of the most important findings of this review was that 59.5% of all complications were related to thromboembolism with a morbidity and mortality of 4.8% and 2.4%, respectively. More importantly, thromboembolism was responsible for 100% of the series' morbidities and 40% of the series' mortalities. All thromboembolic events occurred within 24 hours of the procedure's completion. Use of IIb/IIIa inhibitors in 28 recent cases has not seemed to reduce the incidence of such thromboembolic events.

Computed Tomographic Angiography in Place of Catheter Angiography as the Only Diagnostic and Pre-Treatment Planning Study for Ruptured and Unruptured Cerebral Aneurysms by a Combined Neurovascular Team

Christopher S. Ogilvy, M.D., Brian L. Hoh, M.D., Arnold C. Cheung, B.A., James D. Rabinov, M.D., Johnny C. Pryor, M.D., Bob S. Carter, M.D., Ph.D.

Objective: Many centers are performing CTA but also performing DSA in the same patients, which negates most of CTA's advantages and makes the risks and disadvantages of the two techniques additive. There have been previous reports in the literature assessing the sensitivity and specificity of CTA compared to DSA; however, these have not evaluated the clinical implications of a protocol of CTA replacing DSA as the only diagnostic and pre-treatment planning study for cerebral aneurysms.

Methods: Since late 2001-early 2002, we have adopted a prospective protocol of CTA in place of DSA as the only diagnostic and pre-treatment planning study for cerebral aneurysms (ruptured and unruptured) at the combined neurovascular unit of the Massachusetts General Hospital. We report the results of a 12 month period, January 2002 to January 2003.

Results: During the study period, 223 patients with cerebral aneurysms had their initial diagnostic evaluation for cerebral aneurysm by the combined neurovascular team of the Massachusetts General Hospital: 109 patients with confirmed SAH (Group A) and 114 without SAH (Group B). All of these patients were included in the prospective CTA protocol. Cerebral aneurysm treatment was initiated based on CTA alone in 93 Group A patients (86%), 89 Group B patients (78%), and 182 patients (82%) overall. Treatment consisted of surgical clipping in 152 patients (68%), endovascular coiling in 56 patients (25%), endovascular parent artery balloon occlusion in 4 patients (2%), and EC-IC bypass and carotid artery surgical occlusion in 2 patients (1%). Nine patients (4%) did not undergo treatment. The cerebral aneurysm detection rate by CTA was 100% for the presenting aneurysm (ruptured aneurysm in Group A or symptomatic/presenting aneurysm in Group B) in both Group A and Group B. the detection rate by CTA for total cerebral aneurysms including incidental multiple aneurysms was 96.4% in Group A, 98.8% in Group B, and 97.7% overall. The overall morbidity associated with DSA (pre-treatment or as intraoperative or postoperative clip evaluations) was one patient (1.3%) with minor non-neurological complication, one patient (1.3%) with minor neurological complication, and no patients (0%) with major neurological complications.

Conclusions: We have had promising results with a prospective protocol of CTA in place of DSA as the only diagnostic and pre-treatment planning study for ruptured and unruptured cerebral aneurysms. It appears safe and effective to make decisions regarding treatment based on CTA without

performing DSA in the majority of patients with ruptured and unruptured cerebral aneurysms.

Aneurysm Shape May Be a Better Predictor of Rupture Than Aneurysm Size

Robert E. Harbaugh, M.D. and Madhavan L Raghavan, Ph.D.

Introduction: The predictive value of the size of cerebral aneurysms in regard to risk of rupture has received considerable attention. Less attention has been paid to aneurysm shape and risk of rupture. We used differential geometry techniques to quantify the size and shape of intracranial aneurysms and statistically compared the predictive value of these parameters in regard to risk of aneurysmal subarachnoid hemorrhage.
Methods: CT Angiography was performed on patients who presented with ruptured (N=9) and unruptured (N=19) cerebral aneurysms. Three dimensional geometry of the aneurysm and surrounding vasculature was reconstructed. Differential geometry techniques were employed to calculate indices of size and shape for all aneurysms. The size indices used were aneurysm volume and aneurysm surface area. The shape indices calculated were the isoperimetric ratio (IPR), the convexity ratio (CR), the mean curvature index (MCI), and the gaussian curvature index (GCI).
Results: Unpaired two-tailed student-t tests revealed that IPR (5.0 versus 4.5, p=0.002), CR (96.7% versus 98.4%, p=0.008), and MCI (0.38 versus 0.33, p=0.03) were statistically significantly different between ruptured and unruptured aneurysms while the GCI showed a trend towards statistical significance (2.7 versus 2.0, p=O.067). Size indices, however, did not show a statistically significant difference between ruptured and unruptured aneurysms. The results for volume (146 versus 82 cubic-mm, p=0.29) and for surface area (122 versus 85 sq-mm, p=0.26) did not reliably differentiate between ruptured and unruptured aneurysms. A receiver operating characteristic curve analysis showed that the best predictors of aneurysm rupture are IPR, CR, MCI, GCI, volume and surface area, in that order.
Conclusion: Quantified aneurysm shape indices may be better predictors of aneurysm rupture than aneurysm size.

What Makes A Carotid Atherosclerotic Plaque Symptomatic? Microarray Analysis of Gene Expression

R.J. Dempsey, R. Vemuganti, V.K. Dhodda, T. Varghese, S. Salamat

Carotid atherosclerosis is far more common than carotid embolic disease. The question is what makes a plaque to become symptomatic is of great clinical importance involving proliferation of smooth muscle cells, formation of connective tissue, cholesterol deposition, calcification and extravasation of inflammatory cells. The molecular mechanisms underlying plaque maturation, rupture and embolization are largely unknown. GeneChips/microarrays enable the identification of the cellular events and predict the functional consequences of plaque dynamics at the transcriptional level. Using Affymetrix Human GeneChip set, the present study analyzed the gene expression patterns of 44,862 mRNA transcripts in surgically removed carotid artery plaques from 6 symptomatic stroke patients and 4 non-symptomatic patients. Differential data analysis showed a statistically significant higher abundance (>2 fold, $p<0.05$ in each case) of 104 transcripts in symptomatic plaques over the asymptomatic plaques. Of these, 44% of the transcripts code for proteins which promote cell proliferation and differentiation. These include 25 transcripts that control DNA/RNA maintenance, transcription, splicing, replication and neoplasia, 10 transcripts that control amino acid metabolism, protein translation, structural maintenance and protein transport and 9 transcripts related to second messenger signal transduction. Neuropathological and biophysical examinations showed no significant differences in the amount of calcification, cholesterol levels, percent hemosiderin, inflammatory cell number and stiffness between the symptomatic and non-symptomatic plaques. Thus, the plaques from symptomatic stroke patients show the molecular fingerprints to develop more actively and in a neoplastic-like fashion compared to plaques from non-symptomatic patients. This study further suggests that histologically similar looking plaques can be different at the molecular level based on the stroke status. The implications of these findings suggest that both diagnosis and prevention of stroke symptoms may become possible at the genetic level. These studies were supported by NIH, AHA and the UW-Madison.

Anti-CD11/CD18 Monoclonal Antibody Prevents Vasospasm after Subarachnoid Hemorrhage in Rabbits

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Background and Purpose: Adhesion and transendothelial migration of leukocytes into the periadventitial space may be a determinant event in the pathophysiology of vasospasm after subarachnoid hemorrhage (SAH). Cell-adhesion-molecules (CAMs) involved in this process are LFA-1 (CD11a/CD18) and Mac-1 (CD11b/CD18), present on the surface of neutrophils and macrophages, and ICAM-1 (CD54) present on endothelial cells. A humanized monoclonal antibody, Hu23F2G, targets CD1 1/CD 18 and prevents leukocyte adhesion to endothelial cells. In this study, systemic administration of Hu23F2G prevented chronic vasospasm in the rabbit model of SAH.

Methods: New-Zealand-white rabbits (n=26) were injected with autologous blood in the cisterna magna to induce a SAH, and randomized to receive injections of either Hu23F2G (n=10) or of placebo at 30 minutes, 24, and 48 hours after SAH (n=6). Control animals underwent sham operations (n=4) or SAH alone (n=6). The animals were euthanized 72 hours after SAH, perfused-fixed, and their basilar arteries processed for morphometric analysis. Peripheral white-blood-cell counts were measured at 72 hours. Percentage lumen patencies were compared using the Student's t-test.

Results: Treatment with Hu23F2G resulted in significant prevention of vasospasm. Animals treated with Hu23F2G had 90 ± 7% lumen patencies compared to 65 ± 7% in the placebo group (p=0.025). Lumen patencies in the SAH only group were *59* ± 10%. Mean WBC counts were 16300 ± 710 in the treatment group compared to 7000 ± 386 in the control group (p=0.02). Administration of Hu23F2G produced elevation of WBC counts in 70% of the animals treated.

Conclusions: This study supports the concept that leukocyte-endothelial cell interactions play an important role in the pathophysiology of chronic vasospasm after SAH. Systemic therapy with an anti-CD11/CD18 monoclonal antibody prevents vasospasm after SAH by inhibiting the adhesion-migration of neutrophils and macrophages into the periadventitial space.

Surgical Risks Associated with the Management of Grade I and II Brain Arteriovenous Malformations

Michael Kerin Morgan M.D., BS, FRACS

Grade 1 and 2 AVMs have been considered very safe lesions to resect and the preferred treatment option. However, this is based on series that have not taken into consideration unoperated cases. The aim of this series is to examine all cases, both operated and unoperated, and to identify any characteristics of low grade AVMs that would form a sub-group of relatively "high risk".

A prospectively enrolled arteriovenous malformation database included 248 Spetzler-Martin grade 1 and 2 cases. These were analyzed by: demographic characteristics, angiographic and MRI features, clinical presentation, method of treatment, and outcome.

Management of the Spetzler-Martin grade 1 and 2 cases was by surgery in 229 patients. Seventeen cases did not undergo treatment because of poor neurological condition (6 cases), patient refusal (9 cases), and perceived operative difficulty (despite their grade) (2 cases). These last 2 cases were considered to be "high risk" grade 2 AVMs. The surgical morbidity was 0.9% and the mortality was 0.5%. All 228 patients surviving surgery underwent post-operative angiography confirming cure. There have been 1,143 patient years of follow-up (average of 5.3 years) without post-operative hemorrhage.

Surgery in non-eloquent locations was associated with 0.5% permanent morbidity and 5% morbidity in eloquent locations. However, if the eloquent "high-risk" Spetzler-Martin grade 2 cases had undergone surgery the risk of morbidity may have increased to a maximum of 9.3% for eloquent locations. Extrapolating the chance of adverse outcomes to all grades 1 and 2 AVMs suggest there may be a small group of eloquent AVMs that need special consideration to treatment options.

Software Solutions for Softer Hours: Compliance with the 80-Hour Work Week

David W. Newell, M.D.

Implementation of the eighty-hour-work week for Neurosurgery Residency training is a task which will affect a large number academic medical centers throughout the country. A number of solutions are being implemented to comply with these regulations:

Methods: At the University of Washington, Department of Neurological Surgery, four hospital services comprise the training program. Resident coverage involves implementation of clinically assigned residents, as well as on-call coverage from selected residents during lab rotations, as well as pre-residency and post-residency fellows. In order to collect and maintain quality data on resident work hours while allowing for conferences, vacation and CME activities, a computerized web-based scheduling system has been implemented.

Results: The web-based on call system allowed complex schedules to be constructed in accordance with the new 80/88 hour requirements. The schedule can be accessed by any location via the web and requests for changes in the existing schedule can be made, and approved or denied, on a timely basis. This system also allows instantaneous reports to be generated, indicating the number of hours worked by all the providers.

Conclusions: The system is valuable in structuring a system of on call schedules for residents and attendings, and provides valuable information and recording of hours worked to be compliant with new working hour requirements. A demonstration and features will be presented.

Human Fetal Tissue Transplantation for the Treatment of Parkinson's Disease (PD): A Surgical Placebo Controlled Trial

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Fetal nigral dopaminergic transplants provide benefit to subjects with PD in preliminary open-label clinical trials. We performed a 24-month variable dose double blind placebo controlled trial of human fetal nigral transplantation in 34 subjects with advanced PD. Subjects were randomized to receive bilateral transplants of one or four donors per side, or an imitation operation. The predetermined primary endpoint was the change in UPDRS motor scores. There was no significant treatment effect (P=0.24). Subjects in the placebo and one-donor groups deteriorated by 9.4 +/- 4.25 and 3.5 +/- 4.23 points respectively, whereas those in the four-donor group improved by 0.72 +/- 4.05 points. Comparison of the four-donor versus placebo groups yielded a P-value of 0.096. Post hoc stratification based on disease severity demonstrated a treatment effect in milder subjects. Those receiving four donors per side improved by 1.5 +/- 4.2 points while those in the placebo group deteriorated by 21.4 +/- 4.3 (P=0.005). Initial benefit observed at 6 months in transplant groups became attenuated after discontinuation of immunosuppression. 56% of transplanted subjects developed off period dyskinesia. Striatal fluorodopa uptake was significantly increased following transplantation and robust graft survival was observed post-mortem. Fetal nigral transplantation did not significantly improve motor function in advanced PD subjects despite robust graft survival. This study suggests that transplantation may provide benefit for patients with more moderate disease, and that long-term immunosuppression may be required in future studies. The etiology of transplant-induced dyskinesia must be better understood before further transplant trials can be recommended.

GABA Transmission in Human Focal Cortical Dysplasia

Nicholas M. Barbaro, M.D., Maria Elisa Calcagnotto, M.D., and Scott C. Baraban, Ph.D.

Rationale: Patients with focal cortical dysplasia (FCD) frequently have pharmaco-resistant epilepsy. Animal models of dysplasia including freeze lesion and MAM have specific abnormalities in synaptic function. We examined the kinetic properties of inhibitory postsynaptic currents (IPSCs) in tissue slices obtained from patients undergoing epilepsy surgery for FCD.
Methods: Visualized whole-cell voltage-clamp recordings were performed on cortical slices (300μm). To isolate GABAergic synaptic currents, slices were perfused with aCSF containing 10μM CNQX/50 μM APV. IPSCs were evoked at 0.1Hz using a monopolar electrode placed adjacent to the dysplasia or in white matter. Bicuculline (10μM) abolished spontaneous and evoked IPSCs confirming a role for $GABA_A$ receptors. Recordings were obtained from "dysplastic" and "control" pyramidal cells.
Results: "Dysplastic" pyramidal cells exhibited sIPSCs with amplitude similar to "control" cells (25.5±3.7; n =18). Interestingly, pyramidal cells from FCD exhibited sIPSCs with a slow decay time constant (13.2 ± 2.4 ms) vs. controls (8.3+0.3 ms; $p > 0.05$) and lower frequency (FCD: 1.3+0.3Hz, n =18; control: 2.7+0.3Hz, n =18; $p > 0.05$). eIPSCs on neurons from FCD also exhibited a slow decay time constant (76.9+9.1ms; n= 10) vs. controls (20.4+1.1ms; n =20).
Conclusion: $GABA_A$ receptor mediated currents on neocortical pyramidal cells in FCD exhibit low frequency and slow decay kinetics. These changes in synaptic inhibition may represent enhanced GABA release, decreased GABA re-uptake, or altered GABA receptor function. Thus, studies of tissue obtained from human epilepsy surgery can validate animal models and provide insight on the mechanisms of epilepsy in FCD.

Correlation of Preoperative MR Imaging and Intraoperative Observations of Neurovascular Compression in Patients with Trigeminal Neuralgia

Kim J. Burchiel and Michael A. Sandquist

Introduction: This study evaluated the utility of MRI in predicting the presence, source, location and degree of neurovascular compression (NVC) in patients with trigeminal neuralgia (TN) who subsequently underwent microvascular decompression (MVD).

Methods: 54 patients (17 males and 37 females) with medically intractable trigeminal neuralgia were prospectively studied. A high resolution MRA (TOF) and gadolinium-enhanced T1 (FSE) MRI centered on the trigeminal nerve, was obtained in all cases. Our thesis was that MRA would show only arteries, and the enhanced MRI would show both arteries and veins. Bilateral analysis of the MRA source images, and the enhanced T1 MRI was performed by a neuroradiologist blinded to the patient's side of pain, and to the surgical findings. The neuroradiologist determined the presence or absence of trigeminal NVC on the MRA/MRI, whether an artery or vein compressed the nerve, and both the location and severity of NVC, if any. Independently, the surgical findings, as documented by operative videotape of the microdissection, were similarly assessed by a neurosurgeon who was not a member of the operative team. The imaging and surgical assessments were then compared for each patient.

Results: 46 patients (85.2%) had MRI evidence of a vessel on or near the symptomatic nerve, and 40 (74.1%) showed a vessel in proximity to the asymptomatic nerve ($p < .2$). MRI imaging was able to correctly identify the source of NVC (artery or vein) in 43 patients (94.5%), when compared with the surgical findings. Localization of the NVC (superior, inferior, medial, lateral) by imaging was highly correlated with the surgical findings ($p< .001$). More importantly, the degree of NVC (none, simple, compression, dislocation) was found to be significantly greater on the symptomatic nerve ($p<.01$).

Conclusions: MRI is an effective means of preoperative determination of the presence of, source, location and severity of NVC in patients with TN. Bilateral trigeminal neurovascular relationships are common in TN, but the degree of NVC is significantly greater on the symptomatic side.

Local Cortical Cooling as a Functional Mapping Tool

Matthew A. Howard, III, M.D.

Electrical stimulation mapping (ESM) of cerebral cortex is used routinely in neurosurgical practice to guide critical clinical decision-making. Although safe and effective, one confounding attribute of this method relates to the conduction of stimuli along activated axons to distant sites. When stimuli are delivered to one site, functionally connected brain regions are influenced as well. In order to avoid this drawback, basic neuroscientists make use of an alternative method for causing reversible cortical dysfunction: localized cooling. Results of animal studies indicate that temporary cortical cooling causes reversible, localized synaptic dysfunction. In this report we set out to determine whether this same approach could be applied to human brain mapping. A saline cooled, thermocouple regulated cooling probe was designed and built in the medical device machine shop. Engineering safety studies were conducted to insure the mechanical, electrical and thermal safety of the device. All protocols were IRB approved. Intra-operative experiments have been carried out in 19 epilepsy surgery subjects to date. In all cases chamber temperatures were maintained between 0-5? C during active cooling. Surface EEC recordings revealed statistically significant localized suppression of stimulus evoked and spontaneous field potential activity. When language critical sites, defined using the ESM method, were locally cooled, a qualitatively different pattern of language disruption was observed. EEG and neurological functions returned to normal with rewarming. Our findings indicate that local cortical cooling is a safe and effective method for causing reversible synaptic dysfunction in human cerebral cortex.

Magnetic Resonance Spectroscopy of Atypical Diffuse Pontine Masses

Mark D. Krieger, M.D., Stephan Bluml, Ph.D., and J. Gordon McComb, M.D.

Children's Hospital, Los Angeles Department of Neurological Surgery, Keck School of Medicine of the University of Southern California, Los Angeles, CA

Diffuse pontine gliomas account for 10-15% of childhood brain tumors. The majority of cases are high-grade gliomas which carry a dismal prognosis, with mean survival of less than one year despite therapy. The diagnosis is made by the characteristic changes seen on traditional magnetic resonance imaging, commonly without histologic diagnosis. However, 6-10% of pontine masses with MRI appearances consistent with diffuse pontine gliomas do not behave in the expected fashion and have prolonged survival, calling the original diagnosis into question. The present study evaluates the role of proton magnetic resonance spectroscopy (MRS) as a means to gain additional information regarding the biologic activity of diffuse pontine masses.

A retrospective review was conducted of 42 children (age 6 months-13 years) followed at our institution with the diagnosis of diffuse pontine glioma. Five of these patients (12%) survived longer than 18 months, exceeding expected survival time. These patients did not differ in terms of demographics, presentation, traditional imaging data, or treatment from the group as a whole. Iiowever, MR spectroscopy displayed 2 distinct patterns not seen in typical diffuse pontine gliomas. Two patients showed elevated lipid and lactate levels, with decreased levels of choline, myo-inositol, and NAA. The other 3 patients showed strikingly elevated choline:creatinine ratios and myo-inositol levels when compared with typical pontine tumors. These results suggest that MR spectroscopy data can yield information that differentiates among lesions in the pons, and may carry prognostic significance.

Epidermal Growth Factor Receptor (EGFR) Intron Recombination in Gliomas: Oncogenesis via Corrupt Immune Diversification?

Fenstermaker, Robert A. and Ciesielski, Michael J.

The epidermal growth factor receptor (EGFR) is a membrane-anchored, 170 kDa, protein tyrosine kinase that has been implicated in tumorigenesis. Recent sequence data from the Human Genome Project has led to a revision in the structure of the EGFR gene, as well as an improved understanding of its mutations in tumor cells. The exons and introns of the EGFR gene are contained within 168 kilobases of DNA, including a completely sequenced 123-kilobase first intron. The EGFR gene is frequently amplified and rearranged in malignant gliomas with expression of oncogenic deletion (DM) and tandem duplication (TDM) mutants. The most common mutant is EGFRvIII, which arises from recombination between introns 1 and 7 with deletion of intervening sequences. Some human gliomas express 185 kDa and 190 kDa EGFR tandem duplication mutants with constitutive functional activity. These tumors contain EGFR genes with an in-frame tandem duplication of exons 18 through 25 or exons 18 through 26 respectively. The TDM also arise from recombination between flanking introns 17 and either 25 or 26. DM and TDM have been found in the same tumors, suggesting that the mechanisms responsible for both types of mutants may be closely related. Each of the introns involved in tumor-specific recombination contain sequences with homology to the recombination signal sequence (RSS) heptamers present in the V(D)J region of the immunoglobulin and T lymphocyte antigen receptor genes. These observations suggest a possible mechanism for oncogenic EGFR gene recombination in malignant gliomas.

Identification of Differentially Expressed and Developmentally Regulated Genes in Medulloblastoma Using Suppression Subtractive Hybridization

Naoki Yokota, M.D., Ph.D., Michael Loreto, MSc, Todd Mainprize, M.D., Shigeo Ueda, M.D., Ph.D., and James T. Rutka, M.D., Ph.D.

Arthur and Sonia Labatt Brain Tumour Research Centre, The Division of Neurosurgery, The University of Toronto

To increase our understanding of the molecular pathogenesis of medulloblastoma (MB), we utilized the technique of suppression subtractive hybridization (SSH) to identify genes that are dysregulated im MB when compared to normal cerebellum. After the subtractive hybridization of cDNA from corresponding normal cerebellar tissue, SSH libraries from both human and $Ptch^{+/-}$ heterozygous murine MBs were generated. Through differential screening of the libarics, over 100 up-regulated tumor cDNA fragments were isolated, sequenced and identified with the NGBI BLAST program. From these, we selected genes involved in cellular proliferation, anti-apoptosis, and differentiation of the cerebellum for further analysis. Upregulated genes identified in the human MB library included Unc33-like protein (ULIP), SOX4, neuronatin, the mammalian homologue of Drosophila BarH-like 1(BARHL1), the nuclear matix protein NRP/B (ENC1), and the homeobox OTX2 gene. Genes found to be up-regulated in the murine MB library included cyclin D2, thymopoietin, Musashi-1, protein phosphatase 2A inhibitor-2(I-2FP2A), and Unc5H4(D). Using semi-quantitative RT-PCR and western blot analysis the expression level of these genes were assayed in an array of tumor cell lines and human MB specimens. The expression levels for many of these genes were markedly higher in the MB specimens than in normal cerebellum. The role of these overexpressed, developmentally regulated genes in the transformation of normal cells of the cerebellum in the pathogenesis of medulloblastoma will be discussed.

Neurosurgery: An Expanding Field With A Declining Work Force?

L. Dade Lunsford, M.D., FACS, Amin Kassam, M.D.

Background: Neurosurgery is an expanding specialty with an increased demand for molecular, endovascular, radiosurgical and spinal surgery skills. Approximately 20% of the neurosurgical work force has retired or left practice in selected areas. In order to assess the impact of these issues, we surveyed all current U.S. program directors. In addition, we estimated the need for neurosurgeons trained in radiosurgery and molecular techniques.

Methods and Materials: Seventy-two of 91 program directors responded to a 31-question survey (79% response rate). Incidence data for brain metastases and neurodegenerative disorders was reviewed.

Results: When asked about the total number of practicing neurosurgeons in the U.S., 60% of respondents felt that the number was too low. Sixty-four percent felt that the number of practicing neurosurgeons in the U.S. had declined 5-20% in the last seven years. Eighty percent felt that at least one additional neurosurgeon was needed in their community. Twenty-five percent indicated that 4-5 neurosurgeons were needed in their region. Thus, 400-500 neurosurgical jobs annually exist in the U.S.

Training skills in endovascular, radiosurgery and molecular techniques may greatly expand the need for trained neurosurgeons in the U.S. As one example, it is estimated that between 250,000 and 400,000 patients each year in the United States are eligible for radiosurgery for management of metastatic brain cancer. By the year 2030, as many as 30 million Americans may suffer from neurodegenerative disorders; such patients may potentially benefit from emerging molecular, growth factor, or cellular therapies. A decline in neurosurgeons in Western Pennsylvania (20% loss in two years) is thought to be related to declining reimbursement, lack of medical malpractice tort reform, increasing burden of paperwork, and general job dissatisfaction.

Discussion: This analysis suggests that despite the growing need for neurosurgical intervention, our workforce is shrinking because of concerns related to malpractice, reimbursement, and job dissatisfaction. Compensation models for academic neurosurgeons may no longer be competitive with offers provided by hospitals willing to compensate neurosurgeons based on combined professional and technical revenues. This has profound implications for recruitment to academic neurosurgery. Neurosurgical societies, academic leaders, and hospitals should further analyze the potential impact of a declining neurosurgical workforce in the midst of expanded demand.

NOTES:

FRIDAY, OCTOBER 31 8:15 – 8:30 AM

The Influence of Inferior Petrosal Sinus Sampling on the Outcome of Transsphenoidal Surgery for Cushing's Disease

S. Aytug, M.L. Vance, E.R. Laws

Between 1992 and 2002, 231 patients were treated with initial transsphenoidal surgery (TS) for Cushing's disease (CD) resulting from a presumed pituitary microadenoma. Endocrine diagnosis was assisted by inferior petrosal sinus sampling (IPSS) with CRH administration in 133 patients (58%), and anatomic diagnosis of a pituitary microadenoma was assisted by MRI as interpreted by the surgeon (positive for microadenoma in 70%).

Remission of CD after TS surgery (Cortisol $\leq$ 3 ng/ml) was achieved in 71% of the IPSS positive patients and in 79% of the non-IPSS patients. The cohorts were well matched. Logistic regression analysis showed that only the presence of a microadenoma on MRI was significantly associated with remission ($p=0.024$).

We conclude that although IPSS provides valuable confirmatory information in the diagnosis of CD, suggestive information on lateralization, and a rationale for continued treatment directed at the pituitary source (operative re exploration, radiosurgery), this test has little detectable influence on outcome of surgery performed at a Pituitary Center. The importance of MRI is noteworthy, and should encourage further attempts to detect microadenomas with sophisticated imaging.

Intraoperative Subcortical Stimulation Mapping During Resection of Hemispheric Peri-rolandic Gliomas Located Within or Adjacent to the Descending Motor Pathways: Evaluation of Morbidity and Assessment of Functional Outcome in 294 Patients

Berger MS, Lundin D, Ojemann GA, Lamborn K, Keles E

Introduction

In this study, we report our experience using intraoperative stimulation mapping to locate subcortical motor pathways in 294 patients who underwent surgery for hemispheric gliomas within or adjacent to the Rolandic cortex.

Materials and Methods

Data were collected regarding intraoperative cortical and subcortical stimulation mapping results, along with the patients' neurological status pre- and postoperatively. For those patients who had an additional motor deficit (MD) postoperatively, we examined the evolution of this deficit.

Results

Of the 294 patients, 60 had an additional postoperative MD (20.4%). Of those patients who had an additional MD, 38% (23/60) recovered to their preoperative baseline status within the first postoperative week. Another 20% (12/60) recovered from their postoperative MD by the end of the 4th postoperative week. Eleven additional patients with postoperative MD recovered to their baseline status by the end of the 3rd postoperative month. Thus, 76.7% (46/60) of patients with postoperative MD regained their baseline function within the first 90 days following surgery. The remaining 14 patients (4.8% of the entire study population, 14/294) had persistent MD after 3 months. Patients whose subcortical pathways are identified with stimulation mapping were more prone to develop an additional (temporary or permanent) MD than those patients in whom subcortical pathways could not be identified (27.5% vs 13.1%, respectively) (P=0.003). This was also true when additional (permanent) MDs lasted more than 3 months (7.4% when subcortical pathways are found vs 2.1% when subcortical pathways are not found) (P=0.041).

Conclusion

In patients with gliomas that are located within or adjacent to the Rolandic cortex, and thus, the descending motor tracts, stimulation mapping of subcortical pathways is a useful and dependable surgical technique enabling the surgeon to identify these descending motor pathways during tumor removal and to achieve minimal permanent morbidity in these high risk functional areas.

Management of Oligodendroglioma Based on Molecular Characteristics: Four Year Experience

Gene H. Barnett, Andrew A. Kanner, Susan Staugaitis, Olga Chernova, Richard Prayson, Shih-Yuan Lee, David Peereboom, Bruce H. Cohen, Glen Stevens, Michael A. Vogelbaum, Steven A. Toms

Objective: The chemosensitivity of oligodendrogliomas is predicted by the allelic loss of chromosome 1p and 19q. We reviewed our four-year experience using testing of 1p status by polymerase chain reaction (PCR) or fluorescent in situ hybridization (FISH) to guide management of patients with low-grade or anaplastic oligodendrogliomas (LGO, AO).
Methods. Since February 1999, testing for allelic loss of chromosome 1p by PCR or FISH has been available at our institutuion. Allelic loss of 19q and EGFR amplification became routinely available in 2001. Patients with pure LGO or AO (not mixed oligo-astrocytomas) known to have loss of 1p were preferentially treated initially with chemotherapy (procarbazine, lomustine, vincristine [PCV] or temozolomide) over radiotherapy or surgery unless entered into a clinical trial. Patient data was retrospectively reviewed from February 1999 to the present in this IRB-approved study.
Results: 100 patients with the histological diagnosis of LGO (65 patients) or AO (34 – 1 of unknown grade) were treated during this period. Of these, 72 had assessment of 1p, the remainder having had surgery elsewhere and whose tissue was not available or evaluable. 68% of LGOs and 64% of AOs had LOH of 1p. Median follow up period was 19.7 months (0.1-172.8). 13 (13%) patients have expired; of those only 2 had 1p LOH and 9 were AOs. Surgical practice patterns were modified to obtain biopsy of lesions suspected to be an oligodendroglioma rather than the previous practice of immediately proceeding to craniotomy for operable tumors. Representative management algorithms will be shown.
Conclusions: The increasing acceptance of predictive tests for chemosensitivity has altered the management of oligodendroglioma at our institution. Extended follow up of these patients will better characterize the durability of response and potential benefit of sparing patients more neurotoxic treatments.

Generation of Brain Tumor Neovasculature from Transplanted Bone Marrow

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Introduction. Because viral vectors transduce inefficiently when inoculated intracranially, glioma gene therapy may improve using a cellular vehicle that can be transduced *ex vivo* then reimplanted. Bone marrow cells are accessible, transducible, and sometimes transplanted into glioma patients receiving chemotherapy. However, their role in glioma angiogenesis needs further characterization.

Methods. Bone marrow from β-galactosidase transgenic Rosa-26 mice was donated to irradiated wild-type mice. Eight weeks later, KR158/ΔEGFR astrocytoma cells were implanted intracranially. Four weeks later, tumors were stained immunoflourescently for von Wellibrand factor (vWF[3]), β-galactosidase, and leukocyte antigen CD45. Transgenic mice with NF1 and p53 deletions and subcutaneous tumors received Rosa-26 bone marrow, with tumors stained eight weeks later.

Results. In glioma-bearing mice receiving bone marrow transplants (n=4), 26.2% of vWF-positive cells near glioma expressed β-galactosidase, compared to 1.7% in tumor-free transplanted mice ($p < 0.0005$). Glioma neovasculature therefore specifically stimulates bone marrow differentiation into endothelium, reducing toxicity if therapeutic genes were expressed by endothelial promoters. In glioma-bearing transplanted mice, 28.7% of β-galactosidase positive cells were vWF-positive, compared to 2.1% in tumor-free transplanted mice ($p < 0.0005$). Half of non-endothelial β-galactosidase positive cells were microglia, with 34.7% of β-galactosidase positive cells expressing CD45 (SD 3.1%). To determine if bone marrow could vascularize pre-existing tumors, transgenic mice with p53 and NF1 deletions with subcutaneous tumors were irradiated and given Rosa-26 bone marrow, after which 19.3% of vWF-positive cells near tumor expressed β-galactosidase.

Conclusions. Transplanted bone marrow contributes significantly to the neovasculature of pre-existing intracranial and spontaneously formed subcutaneous tumors, making bone marrow a potential cellular vehicle for glioma gene therapy. Oncolysis may result from *ex vivo* transduction of bone marrow cells with tumoricidal genes such as herpes thymidine kinase expressed by endothelial promoters.

G207 Infection of Dendritic Cells: Effects on Maturation and Generation of Antitumor Immunity

Wiiliam T Curry, M.D., Robert L Martuza, M.D., Samuel D Rabkin, Ph.D.

Introduction: G207 is a multimutated oncolytic herpes simplex virus (HSV) that replicates conditionally in tumor cells and has demonstrated efficacy against murine and human gliomas. Safe use in humans with recurrent malignant gliomas has been demonstrated in a Phase I dose-escalation trial. We sought to study the effects of in vitro G207 infection of tumor cells on dendritic cell (DC) maturation and subsequent DC-mediated generation of antitumor immunity in mice.

Methods: Bone marrow was harvested from the long bones of 6-8 week old female AJ mice and cultured in GM-CSF and IL-4, with media exchange every 2 days. After 6 or 7 days of culture, floating and loosely adherent cells were collected and expression of CDllc, MHC I, MHC II, B7-l, arid B7-2 examined by FACS. In parallel, murine N18 neuroblastoma cells were infected with G207 at a multiplicity of infection (MDI) of 1.0 for 2 hours, and then treated overnight, either with or without heat inactivation of virus at 39.5 degrees. Immature dendritic cells, collected from bone marrow culture, were then pulsed with infected N18 cells. DC expression of MHC and costimulatory molecules were again determined by FAGS, and these cells were used in intraperitoneal treatments of mice bearing established subcutaneous N18 tumors.

Results: Pulsing of DC's with G207-infected G18 cells leads to high rates of infection of DC's themselves, which corresponded with a lack of maturation and down-regulation of MHC and co-stimulatory molecules. Correspondingly, these DC's lost effectiveness at inhibiting subcutaneous tumor growth in mice. When infection of dendritic cells themselves was prevented by heat-inactivation of virus, maturation was regained.

Conclusions: HSV infection of dendritic cells prevents their maturation and may depress anti-tumor immunity, an important consideration as we explore the immune effects of viral oncolytic therapies for malignant gliomas.

Results of a Phase II Trial of Resection and Conformal Radiation Therapy for Pediatric Patients with Localized Ependymoma

Boop FA, Sanford, RA, Merchant, TE, Krasin MJ, Kun LE, Williams T, Li C, Xiong X, Mulhern RK, Khan RB, Lustig RH, Danish RK

St. Jude Children's Research Hospital and LeBonheur Children's Hospital
Memphis, Tennessee

Ependymomas comprise 8-10% of childhood brain tumors. The authors present the results of a prospective trial of resection followed by conformal radiation therapy (CRT) in 88 children with ependymoma treated between 7/97 and 1/03. Median age was 2.8 ± 4.5 years. Patients were characterized by extent of resection [gross total resection (GTR)=74, near total resection (NTR)=6, sub-total resection (STR)=8], prior chemotherapy (n=16), tumor grade (anaplastic=35, differentiated=53), and tumor location (infratentorial=68, supratentorial=20). Children were treated with clinical target volumes of 10mm to doses of 59.4 Gy (n=73) or 54.0 Gy (age<18 mo and GTR). Patients were evaluated before and after adjuvant therapy to determine endocrine, cognitive and audiometric effects of therapy.

At present, with a median survival of 35.8 months (±19.8 months), the 3 year actuarial event-free survival and local control estimates are 80.6% ± 6.8% and 89.5% ± 5.6%, respectively. Thirteen patients experienced local (n=3), distant (n=7) or local plus distant failure (n=3). IQ estimates (IQ ± SD) at baseline (93±18), 6 months (97±19), 12 months (97±19), 24 months (94±18), 36 months (98±19) and 48 months (97±19) were within the range of normal. Pre-CRT GH deficiency was observed in 29 of patients based upon provocative testing. This increased to 50% of patients by 12 months post-CRT, and correlated with hypothalamic dosimetry. CRT related hearing loss was not observed in patients who did not received pre-irradiation chemotherapy.

These results demonstrate that, in the face of aggressive surgery, radiation volumes may be safely reduced, thus limiting treatment related side-effects without affecting disease control for localized ependymoma.

Radiosurgery for Brain Metastasis: Neurological Outcome Versus Tumor Location

Marcos Maldaun, Frederick Lang, Dima Suki, Raymond Sawaya

Stereotactic radiosurgery (SRS) is being used with increasing frequency in the treatment of brain metastasis. The predominant measures of outcome have been related to patient's survival and local control of the tumors. It is generally accepted that the volume of the tumor influences the overall response, with larger tumors being more likely to fail. The location of the tumor relative to the functional anatomy of the brain has not received sufficient attention as a predictor of the neurological outcome of the patients, even though patients with tumors in eloquent parts of the brain are more likely to be treated with SRS. In this study, we have retrospectively analyzed the complication rate following SRS at several interval periods, with particular attention being paid to the neurological outcome in rapport to the functional localization of the tumor.

Results: 213 patients and 261 tumors were treated with SRS. Two thirds of the patients were symptomatic at presentation, and 52% had progressive systemic disease. 58 tumors (22%) were in non-eloquent brain, 110 tumors (42%) were in near eloquent brain, and 93 tumors (36%) were in eloquent brain. Median tumor volume was 1.8cm^3, and median dose was 18 GY. The complications encountered were varied in frequency and in seriousness. Seizures were the most common (16%) followed by necrosis (13%), cognitive deficits (11%), motor deficits (8%), etc. 30% of the tumors failed SRS, but these were not considered as complications. When only significant complications are considered (i.e., seizures excluded), tumors in non-eloquent brain had a 17% complication rate compared to 23% for tumors in near-eloquent brain, and 44% for tumors in eloquent brain (P=0.004). The two functional areas that represent the best examples are the brain stem and the Rolandic area. When compared to tumors in non-functional areas where the complication rates at 6 months and 12 months are 19% and 28%, those for brain stem tumors are 28% and 81%, and for the Rolandic area, 37% and 53% respectively (corresponding P values of 0.01 and 0.008). As expected, tumors greater than 1.46 cm^3 were associated with a higher complication rate (P=0.0006). Finally, on multivariate analysis, the functional location of the tumor, and the volume >1.46 cm^3 remained as significant predictors of poorer outcome (P=0.001 and P=0.008 respectively).

In conclusion, the location of a brain metastasis is clearly influencing the neurological outcome of patients treated with SRS. This variable should be included in the analysis of SRS series, and should be taken into consideration more critically when selecting patients for such a therapy.

Malignant Progression in Meningioma: Documentation of a Series and Analysis of Cytogenetic Findings

Ossama Al-Mefty, M.D., Paulo A. S. Kadri, M.D., Svetlana Pravdenkova, M.D., Ph.D., Jeffrey Sawyer, Ph.D. Muhammed Husain, M.D.

Object: Malignant progression of benign tumors, explained by the model of clonal evolution, is well documented in gliomas and others systemic tumors. Some meningiomas take a progressively malignant course despite their initial benign pathology. The data of cytogenetic abnormalities implicated in this malignant progression are derived from different tumors with different grades in different patients. We study here a group of patients harboring meningioma that showed clear histopathological progression toward a higher grade of malignancy.

Methods: Among 175 recurrent meningiomas, 11 cases showed histopathological progression toward a higher grade associated with aggressive clinical course, six to malignant and 5 to atypical over a period averaged 112 months. In four patients the results of KI 67, P53 and cytogenetic study by FISH technique were studied in successive specimens. The KI 67 showed increase in value in the subsequent sample. The cytogenetic analysis by FISH showed deletions of 22, lp and l4 q.

In all of the cases but one, these aberrations were present on the previous specimen despite the lower grade histopathology.

Conclusion: We document progression of meningiomas from benign to a higher histological grade. These tumors were associated by complex karyotype that included deletions of 22, lp and 14 q present in a prior histologically lower grade tumor and have not progressed in stepwise fashion as the histopathology did. FISH technique, although limited to the tested probes appears to be more accurate than the standard cytogenetic technique to detect these alterations. Tumors that present with complex genetic alteration, even with a benign histological grade are potentially aggressive and require closer follow-up.

Long-Term Results after Radiosurgery for Benign Intracranial Tumors

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Background
Stereotactic radiosurgery is the principal surgical alternative to resection of benign intracranial tumors. The goals of radiosurgery are long term prevention of tumor growth, maintenance of patient function, and prevention of new neurologic deficits or adverse radiation effects. Long-term outcomes more than 10 years after radiosurgery are needed.
Methods
We evaluated 285 consecutive patients who underwent radiosurgery for benign intracranial tumors between 1987 and 1992. Serial imaging studies and clinical evaluations were performed. Our series included 157 patients with acoustic neuromas, 85 with meningiomas, 28 with pituitary adenomas, 10 with other cranial nerve schwannomas, and 5 with craniopharyngiomas. Prior surgical resection had been performed in 44% of these patients, and prior radiation therapy administered to 5%.
Results
Overall, 95% of the 285 patients in this series had imaging-defined local tumor control (63% had tumor regression and 32% had no further tumor growth). The actuarial tumor control rate at 15 years was 93.7%. In 5% of patients, delayed tumor growth was identified. Resection after radiosurgery was performed in 13 patients (5%). No patient developed a radiation-induced tumor. Eighty-one percent of patients were still alive. Median follow-up was 10 years. Normal facial function was maintained in 95% of patients who had it before treatment for their acoustic neuroma.
Conclusions
Stereotactic radiosurgery provided high rates of tumor growth control, often with tumor regression, and low morbidity rates for patients with benign intracranial tumors when evaluated over the long term. This study supports the increasing reliance on radiosurgery as an alternative to surgical resection for benign intracranial tumors.

ACADEMY AWARD PAPER

Targeting the Tie2/Tek Receptor in Astrocytomas

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Tie2 is the second group of endothelial cell specific receptor tyrosine kinase and its ligands are Angiopoietins. Angiopoietin mediated modulation of Tie2 contributes significantly to normal vessel development and stability, however, the functional consequence of this angiogenesis specific pathway in tumors is not well known. We have investigated the role of Tie2 activation in malignant astrocytomas, a common and highly vascularized primary human brain tumor, with hopes of increasing our understanding of the molecular regulators of tumor angiogenesis and improving our therapeutic approach to these lethal tumors. We have found that Tie2 expression and activation increases with increasing malignancy grade of astrocytomas. Furthermore, inhibition of Tie2 using a kinase deficient Tie2 construct, which acts as a dominant-negative mutant, decreases growth of malignant astrocytoma xenografts. Inhibition of Tie2 disrupted tumor vascularity as evidenced by a significant decrease in microvascular density, increased presence of abnormally dilated vessels and loss of interaction between endothelial cells and surrounding smooth muscle cells, collectively resulting in increased tumor cell apoptosis. Overall, our findings strongly suggest that Tie2 activation contributes significantly to astrocytoma tumor angiogenesis and growth. We postulate that targeting Tie2 activation, either independently or in conjunction with other anti-angiogenic therapies, is of potential clinical interest.

ACADEMY AWARD RUNNER-UP

Survival but not Migration of Engrafted Schwann Cells Expressing Alkaline Phosphatase Marker Gene After Transplantation into Contused Spinal Cord of Adult Fisher Rats

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We investigated whether purified Schwann cells (SCs) isolated from sciatic nerves of adult transgenic Fisher rats in which the marker gene human placental alkaline phosphatase (hPAP) is linked to the ubiquitously active R26 gene promoter, were detectable in vivo after being grafted into contused adult Fisher rat spinal cord. Moderate contusive spinal cord injury (SCI) was performed at the 10"' thoracic vertebral level using an JH impacter. Ten days post-injury, hPAP-positive SCs were engrafted either into the injury epicenter (EC group, n = 6) or into the adjacent cord parenchyma at 1.5 mm rostral and caudal to the epicenter (R-C group, n = 5). Control injured rats received only medium injection into the injury epicenter (Ctrl, n = 3). One month post-transplantation, a well defined gliotic boundary area (GBA) was found encircling the area of injury in all three groups. Within the defined GBA, grafted hPAP-SCs were found to survive and promote axonal regeneration, evidenced by the association of SCs with the regenerating axons. Surprisingly, in the R-C group, grafted hPAP-SCs filled almost the entire area of the defined GBA whereas in the EC group only half of the GBA was occupied by grafted hPAP-SCs. Conversely, the EC group contained more cavities than the R-C one. The percentage of the GBA filled with grafted hPAP-SCs was 80% in the R-C group versus *45%* in the EC group ($p < 0.05$). Conversely, the percentage of the GBA taken up by the cavity was only *5%* in the R-C group versus *35%* in the EC group ($p < 0.01$) and 57% in the Ctrl group ($p < 0.01$). In both SC injections, substantial migration of grafted hPAP-SCs through the GBA into the surrounding host spinal cord tissue was not found although overlapping areas of hPAP-immunoreactivity (IR) and glial fibrillary acidic protein(GFAP) JR was found throughout the GBA. Thus, the current study demonstrated that 1) transgene expression of hPAP is robust for up to one month in grafted hPAP-SCs in vivo; 2) engrafted hPAP-SCs survived and supported axonal regeneration in a moderate contusive rat SCI model; 3) substantial migration out of the GBA into surrounding spinal cord tissue by engrafted hPAP-SCs did not occur in the contusion SCI model; and 4) better SC survival, axonal regeneration, and cavity obliteration can be achieved by injecting hPAP-SCs into rostral and caudal cord areas adjacent to the injury as compared to the injections made directly into the lesion epicenter.

NOTES:

SATURDAY, NOVEMBER 1 8:15 – 8:30 AM

The Effect of the Malpractice Litigation Crisis on Academic Neurosurgery

Frederick A. Simeone, M.D.

Malpractice litigation in America has had well publicized detriments to access to medical care, cost of defensive medicine and physicians' attitudes.

Less emphasized is the role of a menacing malpractice climate on academic pursuits. Furthermore, the state-to-state variation of this problem ranges from overpowering to some professors and barely noticeable to others.

Neurosurgical departments from diverse state wide regions will be sampled to compare malpractice coverage rates, neurosurgeons' perceived tensions, and the effects of these on resources available for research, teaching and recruitment.

Clinical Evolution of a Metal-on-Metal Cervical Disc Prosthesis

Regis Haid, M.D., Vincent Traynelis, M.D., Tom Zdeblick, M.D., James Robertson, M.D.

Purpose: Cervical arthroplasty is an intriguing and rapidly developing surgical treatment option. It is important to understand the clinical history and design progression of the devices as they become incorporated into standard clinical practice. The most extensive clinical experience with a cervical disc prosthesis involves a metal-on-metal design which was first implanted in 1991. This device has progressed to a 4th generation and valuable clinical information has been learned at each stage of development. This paper presents the development history and clinical outcomes of a metal-on-metal cervical disc prosthesis.

Methods: The first articulating cervical disc prosthesis, a metal-on-metal ball in socket design known as the Bristol/Cummins disc, was implanted in February 1991. In 1998 the first formal clinical trail for cervical arthroplasty was initiated with an improved Bristol/Cummins device known as the Prestige™ I disc. In 2000, additional improvements were made to the device (Prestige™ II disc) and a randomized study vs fusion was initiated. In 2002, the US FDA approved an IDE study for the further refined device known as Prestige™ SS.

Results: The Bristol/Cummins disc was implanted in 20 end-stage patients. Patients continue to be followed at up to 12 years post-operative. Of the 12 patients available for long-term follow-up, 8 showed maintenance of motion. Device motion is shown to be related to implant design and technique. No device related complications or revisions were seen in long-term follow-up.

The Prestige™ I disc was implanted into 17 end stage-patients. Patients have been prospectively followed to 4 years post-operative. An independent radiographic analysis of 2-year motion films shows all discs maintain motion at the treated level.

The Prestige™ II disc has been implanted in 25 primary indication patients to date. Clinical and radiographic comparisons to the control group are favorable at up to 2 years post-op.

The Prestige™ SS disc has been implanted in 8 primary indication patients to date in the FDA approved IDE study. Limited short-term data are available.

Conclusions: As cervical arthroplasty develops and becomes an accepted treatment option, it is important for the spine surgeon to understand the clinical development of these implants. The evolution of this metal-on-metal cervical disc prosthesis shows a design progression based on clinical experience. Updated data will be presented.

Randomized, Prospective, Controlled Clinical Trial of Pulsed Electromagnetic Field Stimulation for Cervical Fusion

Kevin T. Foley, M.D.

Semmes-Murphey Clinic and Department of Neurosurgery, University of Tennessee, Memphis, TN

Purpose: This is an interim report of a multi-center, prospective, randomized, controlled clinical trial of the safety and efficacy of pulsed electromagnetic field stimulation (PEMF) as an adjunct to cervical spine fusion.

Methods: Three hundred twenty-three patients with symptomatic radiculopathy and correlating radiographic evidence of cervical nerve root compression were enrolled in the study. All patients, who were either smokers or required multi-level surgery, underwent anterior cervical discectomy and Smith-Robinson fusion using allograft bone and anterior cervical plating. Patients were randomized to either the PEMF group or non-PEMF (control) group on a 1:1 basis. They were assessed preoperatively and at 1, 3, 6, and 12 months postoperatively. Parameters included a focused neurological exam, a visual analogue pain scale, the Oswestry Neck Disability Index (NDI), and radiographs. Radiographs were evaluated in a blind fashion by an independent orthopedic surgeon and rated as either "fused" or "not fused". All patients were followed for adverse events to assess safety.

Results: Two-hundred-thirty-five of the 323 patients were available for six-month postoperative evaluation. The fusion rate for the PEMF group was 79.5% (97/122); the fusion rate for the non-PEMF group was 65.5% (74/113) ($p=0.0158$). Both groups showed a significant decrease in pain and neck disability. The incidence of adverse events was comparable in both groups.

Conclusions: The interim results of this multi-center, prospective, randomized, controlled clinical trial indicate that PEMF is a safe and effective adjunct to cervical fusion in a patient population at high risk for nonunion (smokers, multi-level fusion). The fusion rate for the PEMF group was significantly higher than that of the non-PEMF group six months after surgery, with no difference in adverse events.

Low Dose Irradiation Following Spinal Cord Injury Reduces Inflammatory Cell Infiltration and Lesion Severity

CB Shields[1,2,3], RA Gray[1,2], YP Zhang[1,3], Y Han[1,3], DN Loy[3], MD Mills[4], L Fajardo[4], D Sun[5], S Whittemore[1,2,3]

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Introduction: Elucidation of the immune response following spinal cord injury is essential to the development of successful therapeutic strategies that target the secondary injury thought to be initiated by an evolving inflammatory cascade. Our laboratory studied the effects of low dose single fraction x-irradiation on the secondary immune response following contusion injury.

Methods: Rats received moderate contusion injuries (12.5 gm-cm) at T10 and the spinal cords were treated with 2 or 5Gy dose of lateral photon x-irradiation 48 hours following injury. Six weeks post-injury, cavity volume and spared white matter were calculated using the Neurolucida (MicrobrightField, Inc., Williston, VT) software.

Results: No adverse effects were observed following either a 2Gy or 5Gy dose of irradiation. Cavity volume was 70-90 percent smaller 6 weeks following treatment. Inflammatory cells were visualized using immunohistochemical techniques 2 days, 5 days, and 6 weeks subsequent to 5Gy irradiation. Inflammatory cell infiltration was quantified using FACS techniques 2 days and 5 days after irradiation. T cell infiltration and macrophage infiltration was significantly reduced at 4 and 7 days post-injury, respectively.

Conclusion: Our data suggest that low dose irradiation safely alters the temporal progression of inflammatory cascades and the pattern of immune cells infiltrating following contusive spinal cord injury and significantly reduces the extent of traumatic cystic cavity formation. Thus, low dose irradiation may prove to be a valuable component of therapeutic strategies to treat spinal cord injury.

The Management of Tethered Spinal Cord in the Adult Population

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The management of tethered spinal cord (TCS) in the pediatric population is fairly uniform in North America. There remains controversy as to the ideal to management of TCS in the adult (over 18 years) population both in unoperated and previously operated patients. This paper discusses 29 adult patients who have been treated by a single surgeon over the past 48 months for TCS.

Of these 9 were the first operation and 20 were reoperations. Two patients had two separate tethering lesions. A total of 32 procedures for TCS were undertaken. Four patients required reoperation for pseudomeningocele (1), infection (2), and syrinx/retethering (1) . Median f/u is 24 months.

27 of 29 patients the primary complaint was worsening pain of a nature which was nondermatomal. Two patients primary complaint was a painless myelopathy. Only two patients had a completely normal exam and no complaints of bladder or bowel dysfunction (both were unoperated diastematomyelia).

Of the 9 patients undergoing their first operation none had complications and all had significant relief of pain. All had some improvement of any pre-existing neurological function but none returned to normal.

Of the 20 patients undergoing reoperation there were 3 minor complications and 2 serious complications (one significant worsening and one moderate worsening). All patients had an improvement in pain although only 14 felt they were virtually pain free. 13 patients showed objective neurological improvement.

Operation in adults with TCS is useful especially in previously untreated patients and should be considered for relief of pain and improvement of function.

Lumbar Microhemilaminoforamenotomy for Spinal Stenosis

H. Louis Harkey, M.D.

Introduction: Wide laminectomy is the traditional therapy for lumbar spinal stenosis secondary to spondylosis. However, this condition frequently affects the elderly who are less tolerant of a large open spine procedure. Selective decompression through a minimally invasive approach can potentially decrease the morbidity of lumbar laminectomy while providing satisfactory symptomatic relief.

Materials and Methods: Twenty-nine patients averaging 68 years of age (range 45 to 84) with symptomatic lumbar stenosis were decompressed using the Caspar microlumbar technique (unilateral, intrafascial exposure with a speculum retractor and microsurgical decompression). Twenty-six patients had single level decompression and 3 had complete hemilaminectomy with two level decompression.

Results: Follow up was available on all 29 patients. Twenty-five (86 %) had good to excellent relief of preoperative leg pain. One patient had improvement in back pain only and three had poor relief of leg pain. One patient returned 18 months later with pain in a new lumbar distribution and one patient developed recurrence of pain 6 months after surgery. Of the 20 patients with preoperative back pain, 15 experience significant or complete relief of back pain. Two patients with preoperative motor deficits experienced some progression despite pain relief. Complications included. No patient has developed post operative instability.

Discussion and conclusion: Minimally invasive spine procedures significantly reduce the soft tissue injury associated with traditional spinal decompression potentially decreasing surgical morbidity and postoperative recovery time. Despite limited soft tissue exposure, significant decompression can be achieved even crossing midline to remove ligamentum flavum and bone. Neurologic outcomes are comparable to traditional wide laminectomy but recovery is much quicker. Preservation of the contralateral facets and soft tissue may decrease the chance of postoperative instability. Perhaps in properly selected patients, minimally invasive surgery for spinal stenosis can improve overall outcomes.

Gilliatt-Sumner Hand Revisited

Gabriel C. Tender, Ajith J. Thomas, Najeeb Thomas, David G. Kline

Object: Thirty three patients with true neurogenic thoracic outlet syndrome (NTOS), or Gilliatt-Sumner hand, underwent surgical treatment at Louisiana State University over a 25-year period. This study retrospectively evaluates the outcome referable to pain and motor function in these patients.
Methods: All patients had the typical Gilliatt-Sumner hand, secondary to compression of C8, T1 and/or lower trunk. 19 patients had an anterior supraclavicular approach and 15 patients had a posterior subscapular approach to the brachial plexus. Nerve action potential recordings showed plexus involvement close to the spine, at the level of spinal nerves to lower trunk junction.
Results: Pain, present in 22 cases, improved in 21. Mild motor deficit improved in 12 of 14 patients. Severe motor deficit improved partially in 14 of 20 patients.
Conclusions: The diagnosis of true NTOS provides a clear operative indication. Surgical decompression needs to involve the medial portion of the plexus, and especially the spinal nerves. An anterior supraclavicular approach is preferred in most cases. If there is a large cervical rib or there has been a prior anterior operation, then a posterior subscapular approach is indicated.

A Prospective Study Comparing Outcomes after Vestibular Schwannoma Resection and Radiosurgery: Design, Patient Enrollment, and Early Results.

Bruce E. Pollock, Colin L.W. Driscoll, Deborah A. Gorman, Michael J. Link, Jayawant N. Mandrekar, Stephen G. Harner, Michael J. Ebersold, Robert L. Foote, Karl N. Krecke, Craig H. Johnson.

Introduction: The best management for patients with small- to medium-sized vestibular schwannomas (VS) is controversial.
Methods: Prospective cohort study of patients with unilateral, unoperated VS less then 3 cm having surgical resection or radiosurgery at the Mayo Clinic. Blinded, independent observers determined tumor size, hearing loss, facial function, symptoms, and performed the data analysis.
Results: From June 2000 to July 2002, 153 patients with VS were managed at our center. Eighty-two of 88 eligible patients (93%) agreed to participate in the study. Thirty-six underwent surgical resection; 46 had radiosurgery. Patients having resection were younger (48.2 yrs vs. 53.9 yrs, P=0.03). No difference was noted between groups with respect to hearing loss, associated symptoms, tumor size, or activities of daily living (by the Health Status Questionnaire, a modification of the SF-36). Three months after surgery, patients having radiosurgery more frequently had normal facial movement and preserved hearing (P<0.01). The radiosurgical group had higher physical function (P<0.01), role-physical (P<0.001), energy/fatigue (P=0.01), and overall physical component (P<0.01) scores compared to the resection group. Data at the 1-year follow-up interval will be available at the time of presentation.
Conclusions: The study design permitted similar patients to be enrolled into the surgical resection and radiosurgery groups without the need for randomization. In the early follow-up period, VS patients having radiosurgery had better outcomes compared to the surgical resection group. Ongoing evaluation and analysis of these patients will continue to determine if these differences persist at longer follow-up intervals and to determine if tumor control is equivalent.

SATURDAY, NOVEMBER 1 **10:30 – 10:45 AM**

MRI Measurement of Cerebral Blood Flow and CSF Flow Dynamics: Supine vs. Upright Changes in Cerebrovascular Physiology

Thomas M. Moriarty, M.D., Ph.D.[1], Noam Alperin, Ph.D.[2], Stephen G. Hushek, Ph.D.[3], Anusha Sivaramakrishnan[2], Robert F Moser[3], Neil M Hoerter[3], Christopher B. Shields, M.D.

Department of Neurological Surgery, University of Louisville[1], Department of Radiology, University of Illinois at Chicago[2] and iMRI Department, Norton Hospital, Louisville KY[3]

MRI can be used to quantify cerebral blood flow (CBF) and CSF flow dynamics. The unique upright design of the GE Signa SP MRI scanner allows imaging of the brain both supine and upright. We report here the first ever results of using MRI as a noninvasive tool to study the effects of posture on cerebrovascular physiology including total CBF, the distribution of the cerebral venous drainage, and cerebral vascular compliance.

Method: Healthy volunteers were studied at upright and supine positions in a SP *0.5T* MRI scanner (GE Medical Systems, Milwaukee). An 8"X 10" flexible transmit/receive coil was used. Retrospectively gated cine phase contrast scans were used to measure arterial inflow, venous outflow, and CSF flow between the cranium and the spinal canal. An MRV of the neck was obtained with 2DTOF for visualization of the venous drainage system.

Results: The distribution and the flow dynamics of venous drainage were different between the two postures. In the upright position, the venous drainage shifted from the jugular veins to the epidural and the deep neck veins. Venous flow becomes less pulsatile in the upright position. A 3-to 4-fold increase in cerebral vascular compliance was found in the upright position. A lower total CBF and a large (2-3 fold) increase in the intracranial compliance (decrease in ICP) were also measured in the upright position. A lower cerebral vascular compliance was found in the supine posture.

This ability to non.-invasively study cerebral physiology in both upright and supine postures may provide an important diagnostic and research tool to study cerebral regulation in normal and diseased states.

SATURDAY, NOVEMBER 1 10:45 – 11:00 AM

Microglia Activation in Brian Tumors

Behnam Badie, M.D.

Department of Neurological Surgery, University of Wisconsin, Madison

Enhancement of the CNS innate immune response may have therapeutic potential in the management of malignant brain tumors. As specialized macrophages, microglia have been shown to become activated in CNS pathological processes such as inflammation and trauma. To better understand their function in brain tumors, we have isolated microglia from experimental rodent gliomas and have studied their cytokine and surface antigen expression both in vitro and in vivo. Our studies indicate that microglia activation state is directly related to the immunogenicity of brain tumors, i.e. microglia in less immunogenetic glioma models expressed low levels of B7-1 and MHC-II surface molecules. Although microglia could be stimulated in vivo, their activation did not lead to regression of established tumors. Interestingly, activation of these cells in vitro not only resulted in the release of pro-inflammatory cytokines such as IL-12 or TNF-a, but also anti-inflammatory cytokines and mediators such as IL-10 and PGE_2 were also over-expressed. These findings suggest that the expression of anti-inflammatory mediators may suppress microglia activation in CNS tumors. These findings should be considered when delivery of local immune mediators is considered for treatment of such tumors.

Craniotomy for Resection of Pediatric Brain Tumors in the United States, 1988-2000: The Effect of Progressive Centralization and Specialization of Care

Fred G. Barker, M.D.

Introduction. We have previously shown lower mortality rates and better discharge disposition after pediatric brain tumor craniotomy with higher-volume hospitals and surgeons, as well as a 56% relative decrease in mortality rates nationwide for this procedure between 1988 and 2000. In the present study, we investigate trends toward centralization and specialization of pediatric brain tumor craniotomy in the US, 1988-2000.
Methods. Cross-sectional and longitudinal cohort study using the Nationwide Inpatient Sample, 1988-2000. Multivariate analyses adjusted for age, sex, geographic region, admission type (emergency, urgent, elective), and tumor location and malignancy.
Results. About 5% of US hospitals performed pediatric craniotomies for brain tumor during this period, and there was no significant temporal trend in the number of hospitals where the surgery was performed. Per-hospital median caseload increased from 1.7 to 3/yr and 90th percentile hospital volume increased from 11 to 17/yr, accounting for about one-third of the decrease in mortality observed during this period. Care shifted toward teaching hospitals, from 58% of cases in 1988-90 to 85% in 1997-2000 ($P<0.001$), and toward surgeons whose practice was predominantly pediatric (median percent of practice age<19 increased from 12% in 1988-90 to 27% in 1997-2000, $P=0.05$). These changes indicate both progressive centralization and specialization of US pediatric brain tumor surgery in 1988-2000.
Conclusions. For pediatric brain tumor craniotomy in the US, 1988-2000, there were trends toward greater centralization of surgery and more specialization of surgeons. The increased per-hospital caseload explained a substantial fraction of the observed decrease in mortality rates during this period.

Hemorrhagic Complications of Endoscopic Neurosurgery for Brain Tumors

Mark M. Souweidane, M.D.

Introduction: Hemorrhagic complications resulting from endoscopic neurosurgery for intraventricular brain tumors is frequently cited as a prohibitive risk. The true incidence of hemorrhagic sequelae from endoscopic neurosurgery for brain tumors is unknown.

Methods: Endoscopic procedures performed by the author since 1995 were reviewed. Endoscopic procedures for intraventricular tumor biopsy or resection provide the basis for this review. Sequelae resulting from hemorrhage were recorded.

Results: Of 238 endoscopic procedures performed during the study interval, 58 were for an intraventricular brain tumor. Forty-one patients underwent tumor biopsy and 17 underwent tumor resection. Site was categorized as lateral ventricular in 11 and third ventricular in 47. The intended surgical goal was met in 57 of 58 procedures (98%). One procedure was aborted due to poor visualization from hemorrhage. No patient underwent conversion to an open craniotomy for uncontrolled hemorrhage. No patient demonstrated a postoperative deficit or seizure due to hemorrhage. Seventeen patients had a ventriculostomy placed during the primary procedure, 6 due to the degree of intraventricular blood. Of these 6 patients, 4 (67%) underwent colloid cyst removal. All patients with a ventriculostomy were weaned based upon pressure dynamics. One patient was readmitted with symptoms of hydrocephalus that necessitated ventriculoperitoneal shunting 13 days after her primary procedure.

Conclusions: Clinically significant hemorrhage associated with primary endoscopic tumor management is rare. Based upon the results of this study, the risk of hemorrhagic complications does not preclude endoscopic neurosurgery for intraventricular brain tumors.

IGF2 Enhances Sonic Hedgehog-Induced Medulloblastoma in Mice

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Introduction: We reported previously that targeted expression of Sonic hedgehog (Shh) in nestin- expressing neural progenitor cells in the cerebellum induces medulloblastoma in mice and that tumor formation is enhanced by coexpression of the oncoprotein, c-Myc. Shh is a crucial determinant of embryonic pattern formation in the CNS and a potent mitogen for neural progenitor cells. Knock-out mice that are heterozygous defective for Patched (Ptc), the Shh receptor, develop medulloblastomas spontaneously, presumably because the Shh/Ptc signaling pathway is activated. Insulin-like growth factor 2 (IGF2), is required for medulloblastoma formation in these Ptc +/- mice (Hahn et al, J. Biol. Chem., 2000). The fact that both normal and neoplastic tissues from Ptc+/- mice show upregulated expression of IGF2 suggests that IGF2 may be a downstream target of activated Shh/Ptc signaling.

Methods: We investigated the effect of IGF2 on Shh-induced medulloblastoma formation in mice using the RCAS/tv-a system. This system utilizes an avian retroviral vector, RCAS, to target gene expression to specific cell types in transgenic mice. To express exogenous proteins in neural progenitor cells, we used Ntv-a mice. In these mice, the Nestin gene promoter drives expression of TVA, the cell surface receptor for the virus. We targeted expression of Shh and IGF2 to nestin-expressing neural progenitor cells by injecting RCAS vectors into the cerebella of newborn Ntv-a mice. We sacrificed the mice after 12 weeks and examined their brains for histopathological changes.

Results: Following injection with RCAS-IGF2 plus RCAS-Shh, 19/49 (39%) mice developed medulloblastomas, a higher rate than with RCAS-Shh alone (3/32= 9%; p=0.004) or with RCAS-Shh plus RCAS-Myc (9/39 = 23%; p=0.116). Mice injected with RCAS-IGF2 alone did not develop tumors. The tumors closely resembled human medulloblastomas histologically. Immunoperoxidase staining showed expression of synaptophysin, beta III tubulin and NeuN in tumor cells, suggesting an origin of the induced tumors from neuronal precursors. Correlation with Ptc genotype showed that the tumor enhancing effect of IGF2 was equal in Ptc+/- mice (51%) compared to Ptc+/+ mice (49%).

Conclusions: We show here that IGF2 enhances Shh-induced medulloblastoma formation. This cooperativity suggests that Shh and IGF2 may not be connected in a simple, linear fashion through a common pathway. Instead, these two molecules may exert independent, mitogenic effects on the cells-of-origin for medulloblastoma.

Vaccination of Patients with Malignant Glioma with Tumor Lysate-Pulsed Dendritic Cells Elicits Antigen-specific Cytotoxicity

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The primary goal of this phase I study was to assess the safety and bioactivity of tumor lysate-pulsed dendritic cell (DC) vaccination to treat patients with glioblastoma multiforme and anaplastic astrocytoma. Parameters measured were adverse events, survival, and cytotoxicity against autologous tumor and tumor-associated antigens. Nine patients with recurrent glioblastoma multiforme and three patients with recurrent anaplastic astrocytoma were treated. One patient with newly diagnosed glioblastoma and one patient with newly diagnosed anaplastic astrocytoma were also included. Patients were vaccinated three times, two weeks apart with autologous DCs isolated through leukapheresis and pulsed with peptides derived from tumor lysate. Peripheral blood mononuclear cells were differentiated into phenotypically and functionally confirmed DCs. Vaccination with tumor lysate-pulsed dendritic cells was safe, and no evidence of autoimmune disease was noted. Ten patients were tested for the development of cytotoxicity through a quantitative PCR based assay. Five of ten patients demonstrated robust systemic cytotoxicity as demonstrated by IFN-γ release by PBMC in response to tumor lysate after vaccination. Using HLA restricted tetramer staining, we identified a significant expansion in CD8+ antigen specific T-cell clones following DC vaccination in 4 patients, all of whom also demonstrated strong post-treatment anti-tumor cytotoxicity, as determined by qPCR measurement of IFN? message in re-stimulated PBMC. Six patients underwent reoperation for recurrent tumor. A significant CD8+ T cell infiltrate was noted intratumorally in three of six patients. Patients with recurrent glioblastoma were included in a survival analysis and compared to patients who underwent craniotomy for recurrent glioblastoma at our institution during the same time period. The median survival for the study (n=8) and control (n=26) groups were 133 and 30 weeks respectively (p =0.0013, log rank). This phase I study demonstrated the feasibility, safety and bioactivity of an autologous tumor lysate-pulsed dendritic cell vaccine for patients with malignant glioma. We demonstrate for the first time the ability of an active immunotherapy strategy to generate antigen-specific cytotoxicity in brain tumor patients.

Computer-Assisted Anatomic Analysis of Transoral Surgical Approaches to the Clivus

Vijayabalan Balasingam, Gregory J. Anderson, Neil Gross, Cheng-Mao Cheng, Peter E. Andersen, Akio Noguchi, Aclan Dogan, Sean O. McMenomey, Johnny B. Delashaw

This paper was designed to quantitate and compare the area of surgical exposure to the periclival region, as well as the surgical freedom available for instrument manipulation, via the following four transoral surgical approaches: 1) transoral (TO); 2) transoral with a palate split (TOPS); 3) Le Fort 1 Osteotomy (LF); and 4) median labio-glosso-mandibulotomy (MM).
Methods: Twelve unembalmed fresh cadaver heads with normal mouth opening were each serially dissected. Care was taken to avoid destructive interference between approaches. Quantitation of periclival exposure and surgical freedom for each approach was accomplished by stereotactic localization.
Results: Periclival exposure (mm^2)/ surgical freedom (mm^2) were as follows: 1) TO = 492 ± 229 / 3164 ± 1900; 2) TOPS = 743 ± 319 / 3478 ± 2363; 3) LF = 689 ± 248 / 2760 ± 1922; and 4) MM 1312 ± 384 / 8074 ± 6451. The differences were further evaluated by calculating the extent of linear midline clival exposure (from the foramen magnum and extending superiorly), together with the percentage of clival linear midline exposure relative to the total linear midline exposure: 1) TO = 0.6 +4.9 mm, 7.8+11%; 2) TOPS = 8.9+5.5 mm, 24.2+16.7%; 3) LF = 32.9+10.2 mm, 85.0+18.7%; and 4) MM = 2.1+4.4 mm, 6.7+11.1%.
Conclusion: The choice of approach is dependent on the location of the pathology. Each approach has different degrees of complexity and associated morbidity. Maximal exposure of the clivus proper was provided by the Le Fort 1 osteotomy.

The Role of the Basal Ganglia in Voluntary Movement Selection

Joseph S Neimat, M.D.; Emad N. Eskandar, M.D.; John A. Assad, Ph.D.

Introduction: Current models suggest that the basal ganglia play a role in selecting desired movements among a number of possible movement choices. This may account for the paucity of movement in Parkinsonism and the inability to suppress movement in cases of ballism or chorea.
Methods: Single-unit electrode recordings were made in macaque monkeys trained to execute movements using a joystick to guide a spot to a target presented on a video monitor. Trials with a single object for movement were interleaved with trials presenting 2 objects and thus requiring a movement selection
Results: One hundred thirty-eight cells (72 in the putamen, 59 in the pallidum, and 7in the sub-thalamic nucleus) were recorded during the selection task. Two-way ANOVA ($p<0.05$) was used to analyze the effect of movement direction and presence or absence of choice on neuronal firing rates. Firing rates were analyzed both before movement initiation and during movement. Of the putaminal cells 31(43%) and 37(51%) were significantly modulated by the direction of movement before and during movement respectively. In the same epochs, 36(27%) and 21(29%) cells were modulated by the situational context in which the movement was made; i.e. by the presence or absence of a competing choice for movement direction.
In the pallidum 24(41%) and 23(39%) were modulated by direction before and during movement, while 8(14%) and 13(22%) were modulated by movement context. Preliminary data on 7 STN cells demonstrated significant context variability in firing of 4(57%) and 5(71%) cells before and during movement respectively.
Conclusions: These data support the proposition that the basal ganglia may play a role in selecting among potential voluntary movement choices.

NOTES:

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Gregory Thompson Ann Arbor, MI	Julian Hoff
John Ward Richmond, VA	Harold Young
John Yu Los Angeles, CA	Keith Black
Gelareh Zadeh (resident) Toronto, Canada	Academy Award Winner
Eric Zager Philadelphia, PA	David Kline

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Gregory W. Canute 1997
Nathan R. Selden 1998

Robert M. Friedlander 1999
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Ganesh Rao 2002
Gelareh Zadeh 2003

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Roosevelt Hotel, New Orleans, Louisiana ..... October 27-29, 1939
Tudor Arms Hotel, Cleveland, Ohio ..... October 21-22, 1940
Mark Hopkins Hotel, San Francisco and
Ambassador Hotel, Los Angeles, California..... November 11-15, 1941
The Palmer House, Chicago, Illinois ..... October 16-17, 1942
Hart Hotel, Battle Creek, Michigan..... September 17-18, 1943
Ashford General Hospital, White Sulphur Springs,
West Virginia ..... September 7-9, 1944
The Homestead, Hot Springs, Virginia ..... September 9-11, 1946
Broadmoor Hotel, Colorado Springs,
Colorado ..... October 9-11, 1947
Windsor Hotel, Montreal, Canada ..... September 20-22, 1948
Benson Hotel, Portland, Oregon ..... October 25-27, 1949
Mayo Clinic, Rochester, Minnesota..... September 28-30, 1950
Shamrock Hotel, Houston, Texas ..... October 4-6, 1951
Waldorf-Astoria Hotel, New York City,
New York ..... September 29-October 1, 1952
Biltmore Hotel, Santa Barbara, California ..... October 12-14, 1953
Broadmoor Hotel, Colorado Springs, Colorado ..... October 21-23, 1954
The Homestead, Hot Springs, Virginia ..... October 27-29, 1955
Camelback Inn, Phoenix, Arizona ..... November 8-10, 1956
The Cloister, Sea Island, Georgia ..... November 11-13, 1957
The Royal York Hotel, Toronto, Canada ..... November 6-8, 1958
Del Monte Lodge, Pebble Beach, California..... October 18-21, 1959
Copley Sheraton Plaza, Boston, Massachusetts ..... October 5-8, 1960
Royal Orleans, New Orleans, Louisiana ..... November 7-10, 1962
El Mirador, Palm Springs, California ..... October 23-26, 1963
The Key Biscayne, Miami, Florida..... November 11-14, 1964
Terrace Hilton Hotel, Cincinnato, Ohio ..... October 14-16, 1965
Fairmont Hotel & Towers, San Francisco,
California ..... October 17-19, 1966
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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
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Mills Hyatt House, Charleston, South Carolina .... November 10-13, 1976
Mauna Kea Beach Hotel, Kamuela, Hawaii ..... November 2-5, 1977

Hotel Bayerischer Hof, Munich, Germany ..... October 22-25, 1978
Hyatt Regency, Memphis, Tennessee ..... November 7-10, 1979
Waldorf-Astoria 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,.....October 29-November 1, 2003
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Dean H. Echols..... 1938-39
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Frank H. Mayfield..... 1943
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Barnes Woodhall..... 1946
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