THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY



61st Annual Meeting

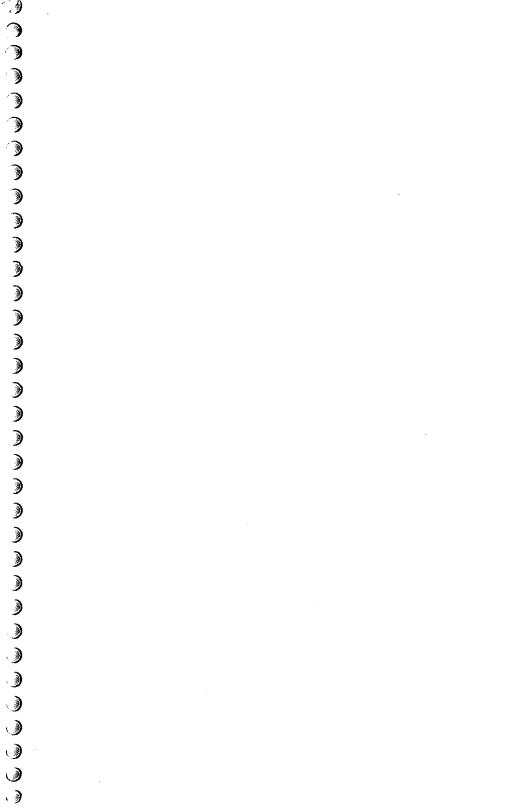


THE RITZ-CARLTON®
AMELIA ISLAND

November 10-13, 1999



Jointly Sponsored by the American Association of Neurological Surgeons



THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY



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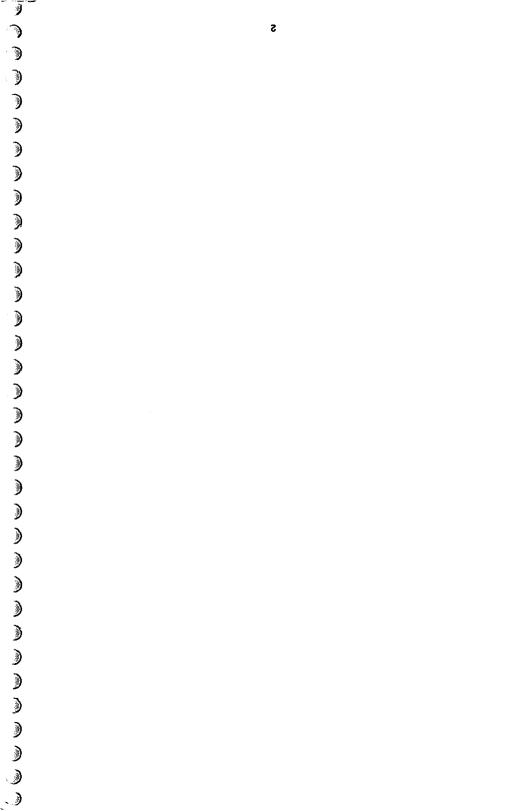
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David G. Piepgras

LOCAL ARRANGEMENTS:

Arthur L. Day

AANS JOINT SPONSORSHIP EDUCATION REPRESENTATIVE:

Richard Morawetz

GENERAL INFORMATION

REGISTRATION

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

REGISTRATION HOURS ARE:

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

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

Friday, November 12 7:00 AM – 5:00 PM

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

SLIDE PREVIEW ROOM

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

MESSAGE CENTER

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

PROGRAM SUMMARY

Tuesday,	November	9	
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12:00 Noon ABNS Primary Exam Committee—
Kings Bay
7:00 PM - 9:00 PM ABNS Dinner—Amelia Room

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Wednesday, November 10

7:00 AM – 8:00 AM 8:00 AM – 4:00 PM	ABNS Breakfast—Kings Bay ABNS Primary Exam Committee— Kings Bay
12:00 PM - 1:00 PM	ABNS Luncheon—Kings Bay
2:00 PM – 3:00 PM	American Academy Executive Committee Meeting—The Ambassador
3:00 PM – 5:00 PM	American Academy Membership Committee—The Ambassador
3:00 PM - 9:00 PM	Registration—Salon III Foyer
4:00 PM - 9:00 PM	Speaker Ready Room—The Boardroom
6:30 PM - 10:00 PM	Welcome Reception—Oceanfront Lawn
7:30 PM – 10:00 PM	American Academy Executive Dinner—The Amelia Room

Thursday, November 11

9:54 AM - 10:15 AM

7:00 AM – 5:00 PM	Registration—Salon III Foyer
7:00 AM – 5:00 PM	Speaker Ready Room—The Boardroom
7:00 AM – 9:00 AM	Buffet Breakfast (Spouses and Guests)—
	The Talbot Room
7:00 AM – 8:00 AM	Buffet Breakfast and Meeting (Members)—Salon II
8:00 AM - 1:00 PM	Scientific Session I—Salon III

Coffee Break-Salon III Foyer

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•	1:00 PM - 2:30 PM	ABNS Advisory Council Luncheon— The Ambassador
•	1:00 PM	Golf—The Golf Club of Amelia Island
)	3:00 PM	Tennis—Ritz-Carlton Tennis Courts
)	3:00 PM – 5:00 PM	Journal of Neurosurgery Meeting— Director's Room
•	6:30 PM - 7:00 PM	Reception—Walkers Landing
•	7:00 PM – 8:30 PM	Dinner—Walkers Landing
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)	Friday, November 12	
)	7:00 AM – 5:00 PM	Registration—Salon III Foyer
)	7:00 AM – 5:00 PM	Speaker Ready Room—The Boardroom
•	7:00 AM - 9:00 AM	Buffet Breakfast
•		(Spouses and Guests)— The Talbot Room
)	7:00 AM - 8:00 AM	Buffet Breakfast and Meeting
)		(Members)—Salon II
)	8:00 AM – 1:00 PM	Scientific Session II—Salon III
)	10:02 AM – 10:22 AM	Coffee Break—Salon III Foyer
)	11:55 PM – 12:30 PM	Presidential Address: J. Charles Rich Introduction by: Donald O. Quest
)	4 00 Pr 4	—Salon III
)	1:00 PM	Golf—The Golf Club of Amelia Island
)	3:00 PM	Tennis—Ritz-Carlton Tennis Courts
)	6:30 PM – 7:30 PM	Cocktails—Ballroom Foyer
))	7:30 PM – 10:30 PM	Black-tie Reception—Salon II
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Saturday, November 13

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

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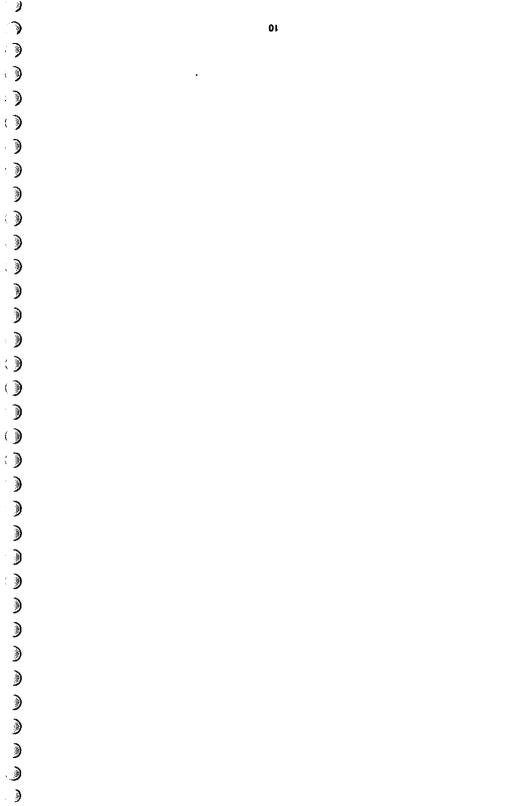
SCHEDULE OF ACTIVITES FOR SPOUSES

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

to all events.	
Wednesday, November 10	
6:30 PM - 10:00 PM	Welcome Reception—Oceanfront Lawn
Thursday, November 11	
7:00 AM – 9:00 AM	Buffet Breakfast—The Talbot Room
7:00 AM - 3:00 PM	Hospitality Suite—The Talbot Room
1:00 PM	Golf & Tennis
6:30 PM - 7:00 PM	Reception—Walkers Landing
7:00 PM – 8:30 PM	Dinner-Walkers Landing
Friday, November 12 7:00 AM - 9:00 AM	Buffet Breakfast —The Talbot Room
7:00 AM – 3:00 PM	Hospitality Suite—The Talbot Room
11:55 PM – 12:30 PM	Presidential Address: J. Charles Rich Introduction by: Donald O. Quest— Salon III
1:00 PM	Golf, Tennis, Shopping, Touring, etc.
6:30 PM - 7:30 PM	Cocktails—Ballroom Foyer
7:30 PM – 10:30 PM	Black-tie Reception—Salon II
Saturday, November 13	
7:00 AM – 9:00 AM	Buffet Breakfast (Spouses)— The Talbot Room
7:00 AM – 3:00 PM	Hospitality Suite—The Talbot Room

1:00 PM

Meeting Adjourns



SCIENTIFIC PROGRAM AMERICAN ACADEMY OF NEUROLOGICAL SURGERY 1999 LEARNING OBJECTIVES

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

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

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



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

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

SCIENTIFIC PROGRAM AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

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Thursday, November 1	Moderator—William Chandler, MD
8:00 AM-9:00 AM	Point-Counter-Point - Treatment of an unruptured 8 mm Aneurysm. Robert Spetzler, L. Nick Hopkins, David G. Piepgras
9:00 AM-9:18 AM	Posterior circulation aneurysms: results using combined surgical and endovascular techniques Christopher S. Ogilvy, Christopher Putman, Ronald Budzik, Alex Norbash, In Sup Choi
9:18 AM-9:36 AM	Surgical repair of endovascularly untreatable trasitional cavernous carotid aneurysms Fredric B. Meyer, Jonathan A. Friedman, Douglas A. Nichols
9:36 AM-9:54 AM	Retrospective analysis of carotid revascularization at the Univ. of Buffalo, Department of Neurosurgery Lee R. Guterman, James L. Budny, L. Nick Hopkins
9:54 AM-10:15 AM	Coffee
10:15 AM-10:33 AM	Unilateral acoustic tumors in younger individuals with out neurofibromatosis Jon H. Robertson, Gale Gardner, John J. Shea
10:33 AM-10:51 AM	Dendritic cell immunotherapy for patients with glioblastoma multiforme and anaplastic astrocytoma <u>Keith L.Black</u> , Christopher Wheeler, Paul Zeltzer, Divina Nacis, Paul Lee, John S. Yu

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

Friday, November 12	Moderator—Howard Eisenberg, MD
8:00 AM-8:50 AM	Point-Counter-Point - To plate or not to plate in a single level anterior discectomy Volker Sonntag. Stephen Papadopoulos
8:50 AM-9:08 AM	Surgical experience with an artificial cervical joint <u>James T. Robertson</u> , S. Gill, R. Nelson, N. Metcalf
9:08 AM-9:26 AM	Outcome of 51 cases of unilateral locked cervical facets: interspinous braided cable for lateral mass plate fusion compared with interspinous wire and facet wiring with iliac crest J. Kevin Kaufman, Scott A. Shapiro, Paul B.Nelson
9:26 AM-9:44 AM	Image guidance in spinal surgery: an update Gerald Rodts, Kevin Foley
9:44 AM- 10:02 AM	Developing new technology: An accurate laser targeting system for fluoroscopically assisted procedures Michael K. Landi
10:02 AM-10:22 PM	COFFEE
10:22 AM-10:40 AM	Outcomes studies in carotid endarterectomy Robert E. Harbaugh
10:40 AM-10:58 AM	Improved management of childhood medulloblastoma using proton beam radiotherapy Paul H. Chapman, Nancy J. Tarbell, William E. Butler, Jay S. Loeffler
10:58 AM-11:16 AM	Radical resection of gliomas in functioning brain regions utilizing awake cortical and subcortical mapping and frameless stereotaxis Fredric B. Meyer, Lisa M. Bates, Stephen J. Goerss, Wanda L. Windschitl, Jonathan A. Friedman

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•	11:16 AM-11:34 AM	FasL expression in tumor vessels of
•		patients with glioblastoma multiforme <u>John S. Yu, Yun-hui Liu, Chunren Liu,</u>
•		Ken Samoto, Moneeb Ehtasham,
•		Keith L. Black
)	11:34 AM- 11:52 AM	Stereotactic radiosurgery: the treatment
•		of choice for jugular foramen region
)		tumors? <u>Bruce E. Pollock</u> , Deborah A. Gorman
)	11:55 AM- 12:30 PM	Presidential Address:
•		Athletic Performance Enhancement: The
•		Difference between Sports Science and Sports Medicine J. Charles Rich
•		Sports Medicine d. Charles Rich
)		
)	Saturday, November 13	Moderator: Robert Spetzler, MD
)	8:00 AM-8:50 AM	Point-Counter-Point - Aggressive resec-
)		tion versus biopsy of a low gradeglioma <u>Mitchell L. Berger</u> , L. Dade Lunsford
)	8:50 AM-9:08 AM	Results of a phase I study of the treat-
)	0.50 / MI-7.00 / MI	ment of malignant gliomas with the
•		genetically-engineered herpes simplex
•		virus G207 James A. Markert,
•		Michael D. Medlock, Samuel Rabkin, Yancey Gillespie, Frank Feigenbaum,
)		William D. Hunter, Tomoki Todo,
)		Carlo Tornatore, Robert L. Martuza
)	"9:08 AM-9:26 AM	Clinical and economic consequences of
)		early discharge after stereotactic brain
•		biopsy Gene H. Barnett, Wayel Kaakaji, Diane Bernhard, Kren Valaitis,
)		Sarah Stamp, Narongsak Boonswag
)	9:26 AM- 9:44 AM	An ideal syngeneic mouse glioma for
•		testing immunotherapy strategies
)		Warnick RE, Weiner NE, Pyles RB, Chalk CL, Balko GO, Miller MA,
)		Dyer CA, Parysek LM
)		15
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9:44 AM-10:02 AM	Intraoperative human sensorimotor and language mapping using optical intrinsic signal imaging: comparison with electophysiologic techniques and fMRI in 40 patients Neil Martin, Andrew Cannestra, Nader Pouratian, Donald Becker, Susan Bookheimer, Nancy Sicotte, Arthur Toga
10:02 AM-10:22 AM	COFFEE
10:22 AM-10:40 AM	New neurosurgical perspectives on spontaneous intracerebral hemorrhage Issam A. Awad
10:40 AM-10:58 AM	3-D computer modeling of the cerebral vasculature <u>Paul S. Larson</u> , Chuck Sites, Ashraf Mohamed, Ayman M. Eldeib, Todd Vitaz, Aly A. Faraq, Thomnas M. Moriarty, Christopher B. Shields
10:58 AM-11:16 AM	Instrumentated fusion in the management of post-laminectomy lumbar stenosis Charles L. Branch, David Jones
11:16 AM-11:34 AM	Early moderate hyperventilation does not reduce cerebral metabolism following severe traumatic brain injury Robert L. Grubb, Thomas Videen, Allyson R. Zazulia, Ellen Deibert, Venkadesh Aiyagari, Ralph G. Dacey, Michael N. Diringer, William J. Powers
11:34 AM-11:52 AM	Intracranial hypertension and cerebral perfusion pressure: their influences on neurological deterioration and outcome in severe head injury N. Juul, G.F. Morris, S. B. Marshall, The Executive Committee of the Interna-

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tional Selfotel Trial, L. F. Marshall

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•	Saturday, November 13	1
)	11:52 AM-12:10 PM	Flourescence-guided surgery of malignant gliomas utilizing 5-ALA-
•		induced porphyrins. Experience with
• • • • • • • • • • • • • • • • • • •		66 consecutive patients. <u>H.J. Reulen</u> , R. Baumgartner, W. Stummer
)	13.10 DM 13.30 DM	_
	12:10 PM-12:28 PM	Genetically engineered cytotoxic T lymphocytes targeted against angiogen-
)		esis: A novel anti-glioma strategy Zoher
)		Ghogawala, Robert Carter, Thomas Niederman,
)		Richard C. Mulligan
))	12:28 PM-12:46 PM	Elevation of the internal auditory canal
)		pressure by vestibular schwannomas
		Behnam Badie, Mark Pyle, Peter Nguyen
)		Nguyen
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NOTES:		

THURSDAY PROGRAM

THURSDAY, NOVEMBER 11

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9:00-9:18 AM

Posterior Circulation Aneurysms: Results Using Combined Surgical and Endovascular Techniques

<u>Christopher S. Ogilvy,</u> Christopher Putman, Ronald Budzik, Alex Norbash, In Sup Choi

Massachusetts General Hospital, Harvard Medical School, Boston, MA

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

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

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

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Surgical Repair of Endovascularly Untreatable Transitional Cavernous Carotid Aneurysms

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

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

THURSDAY, NOVEMBER 11	9:36-9:54 AM
Retrospective Analysis of Carotid Revasculariz University of Buffalo, Department of Neurosur May 1, 1995 — July 1, 1998	
Lee R. Guterman PhD MD, James L. Budny MD, a	nd L. N. Hopkins MD
Purpose:	
To compare carotid endarterectomy (CEA) to carotic as a method for carotid revascularization in 228 corpresented to the neurosurgery service study.	
 Retrospective chart Angiographic film review Primary Endpoints Major stroke 30 d Death 30 d MI 30 d Secondary Endpoints Minor Stroke 30d TIA 30d 	
Goals	
Compare medical comorbidity in each population is state of health for this population. Prior to this re- patients in the stent group had a higher morbidity group.	eview it was assumed
Abstract	
There were 228 patients treated at Millard Fillmore 1, 1995 and July 1, 1999 who presented with cervid disease. There were 94 patients revascularized usin followed by stent placement and 134 revascularized The group was predominantly male (61.4%) with	cal carotid bifurcation ag carotid angioplasty using endarterectomy.

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

years. In the stent group 71.3% were male. The majority of patients

were Caucasian (88% stent, 97% CEA).

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

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

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

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

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

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

Procedural Complications

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

THURSDAY, NOVEMBER 11

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Unilateral Acoustic Tumors in Younger Individuals Without Neurofibromatosis

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

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

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

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

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Dendritic Cell Immunotherapy for Patients with Glioblastoma Multiforme and Anaplastic Astrocytoma

<u>Keith L. Black</u>, Christopher Wheeler, Paul Zeltzer, Divina Nacis, Paul Lee, John S. Yu

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

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

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

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

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

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

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

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

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

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

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The Contemporary Surgical Management of the Chiari I Malformation in Children

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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Academy Award Paper

Reduction of Post-traumatic Spinal Cord Injury by Inhibition of the Caspase Cascade

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

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

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Academy Award Paper-Runner up

Fas Upregulation in High-grade Gliomas Results in Increased Apoptosis and Survival

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

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

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

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

D N Finally, the effect of Fas upregulation on survival was studied in immune competent rats with intacranial malignant gliomas. Survival length was assessed after the implantation of 36BIO- and 36BIO-Fas. The median survival increased significantly from 14 days (36BIO-) to 24.5 days (3 6BIO-Fas), representing a 75% increase in survival in the higher Fas-expressing group (p = .0005). In conclusion, it appears that the overall low AI (range: 0-2.0%) seen in high-grade astrocytomas is related to low cell surface expression of Fas. By increasing surface Fas expression, rates of Fas-mediated apoptosis increase, as does survival.

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FRIDAY PROGRAM

FRIDAY, NOVEMBER 12

8:00-8:50 AM

Surgical Experience with an Artificial Cervical Joint

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

University of Tennessee, Memphis

847 Monroe, Suite 427

Memphis, Tennessee 38163

INTRODUCTION

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

AIM

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

METHODS

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

RESULTS

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

SIGNIFICANCE AND CONCLUSIONS

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

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

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

5 consecutive patients with unilateral looked facets of the cervical spine underwent treatment over an 11-year period. With the development of internal fixation devices, the authors compared the procedure of using interspinous wire and facet wiring of iliac crest to fix Linilateral locked facets with that in which interspinous braided cable and lateral mass plates were used. Thirty-seven patients (73%) presented with radiculopathy\eight (16%) -with neck pain only, and six (12%) with spinal cord injuries (Sets). Plain x-ray films demonstrated subluxation in only 44 (86%) of 5 1 cases. All patients underwent cervical computerized tomography (CT) scanning, and in all patients with SCI, a magnetic resonance (MR) image was obtained. Fracture in addition to facet locking was seen on 24 (47%) of 51 CT scans. Disc disruption with cord compression was seen in five cases (10%). Based on CT and/or MR imaging findings, a closed reduction procedure was believed to be contraindicated in 11 cases (22%). Of the, remaining 40 patients, 13 (33%) underwent closed reduction procedures. Two patients who underwent a closed reduction procedure were placed in a halo brace but experienced resubluxation and were surgically treated. Forty-six patients underwent posterior reduction and/ or internal fixation alone (in 24 cases spinous process fixation with facet wiring and connected to struts of iliac crest, and in 22 cases interspinous braided cable for lateral mass plating was used).

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

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

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Image-Guided Surgery of the Spine: An Update

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

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

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

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

Michael K, Landi, MD

University at Buffalo Neurosurgery

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

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

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

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

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Outcomes Studies in Carotid Endarterectomy

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

Professor of Surgery (Neurosurgery)

Dartmouth-Hitchcock Medical Center

ABSTRACT

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

However, numerous studies analyzing the database maintained by the United States Health Care Financing authority (HCFA) indicate that the morbidity and mortality associated with carotid endarterectomy is considerable highter in general practice than was the case in the prospective randomized studios. This raises the question as to whether or not &0 results of the prospective, randomized studies can be generally applied to patients undergoing carotid endarterectorny for stroke prevention in the United States.

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

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Improved Management of Childhood Medulloblastoma Using Proton Beam Radiotherapy

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

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

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

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10:58-11:16 AM

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

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

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

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FasL Expression in Tumor Vesels of Patients with Glioblastoma Multiforme

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

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

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

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

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

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

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

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

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SATURDAY PROGRAM

SATURDAY, NOVEMBER 13

8:50-9:08 AM

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

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

Affiliations:

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

Abstract

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

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Clinical and Economic Consequences of Early Discharge after Stereotactic Brain Biopsy

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

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

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

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

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

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

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

An Ideal Syngeneic Mouse Glioma Model for Testing Immunotherapy Strategies

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

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

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

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

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

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

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

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

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

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New Neurosurgical Perspectives on Spontaneous Intracrebral Hemorrhage

Issam A. Awad, MD

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

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

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

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

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

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

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3-D Computer Modeling of the Cerebral Vasculature

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

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

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

Instrumented Fusion in the Management of Post Laminectomy **Lumbar Spinal Stenosis**

Charles L. Branch, David Jones

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

Methods/Results: Over a ten-year period, 43 patients with chronic low back and/or leg pain underwent repeat decompressive lamiectomy an instrumented fusion for recurrent and/or residual lumbar spinal stenoiss. These patients had undergone a total of 51 previous decompressive laminectomies without fusion for lumbar stenosis. The mean interval between the preceding decompressive procedure and subsequent fusion was 2.67 years. The average age of the patients was 66.4 years, and the average number of levels fused was 2.8. Outcome in terms of pain relief and functional imporvement was determined by chart review. With a mean follow-up of 1.46 years, an overall improvement was achieved in 77% of patients. Additionall, 35 patients responded to a questionnaire (mean follow-up 3.26 years) in which they graded their outcomes as much imporved (51.4%) somewhat improved (37.1%), unchanged (11.4%), somewhat worse (0%), and much worse (0%). Five (11.6%) patients required an additional 7 lumbar spine operations for adjacent level stenosis at a mean interval of 1. 93 years after fusion.

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

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

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

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

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

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

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

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Influences on Neurological Deterioration and Outcome in Severe Head Injury N. Juul, GF Morris, SB Marshall, The Executive Committee of the

International Selfotel Trial, L F Marshall

Intracranial Hypertension and Cerebral Perfusion Pressure: Their

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

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

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

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

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

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

Department of Neurosurgery, Laser Research Laboratory, Klinikurn Grosshadern Ludwig-Maixmilians University, Munich Germany

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

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

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

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

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

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

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

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

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

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Elevation of the Internal Auditory Canal Pressure by Vestibular Schwannomas

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

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

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

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

Placement of the pressure monitor into the IAC revealed a biphasic waveform in every patient with a mean ICaP of 20 mm Hg (range 1-45 mm Hg). The ICaP directly correlated with the proportion of tumor in the IAC (r2=0.57, p=0.001) but not with the total tumor size (r2=0.16, p=0.13). Furthermore, 8 patients with class A preoperative hearing (American Academy of Otolaryngology-Head and Neck Surgery classification) tended to have lower ICaP's as compared to 5 patients with class B hearing (16+5 Vs 28+ 4). Although this observation suggested an inverse correlation between the ICaP and hearing function, the difference between the two groups was not statistically significant (p=0.14).

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

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SPECIAL GUESTS			
GUESTS	SPONSORS		
David Andrews, M.D. Philadelphia, PA	Robert Rosenwasser, M.D.		
Armand Awad, M.D. New Haven, CT	Issam Awad, M.D.		
Behnam Badie, M.D. Madison, WI	Robert Dempsey, M.D.		
Gene Barnett, M.D. Cleveland, OH	Joseph F. Hahn, M.D.		
Joshua Dowling, M.D. St. Louis, MO	Ralph Dacey, Jr., M.D.		
Kevin Kaufman, M.D. Indianapolis, IN	Paul Nelson, M.D.		
Carl Lauryssen, M.D. St. Louis, MO	Ralph Dacey, Jr., M.D.		
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Robert Maciunas, M.D. Rochester, NY	Warren Selman, MD		
James Markert, M.D. Birmingham, AL	Julian Hoff, M.D.		
Stephen Papadopoulos, M.D.	William Chandler, M.D.		
Ann Arbor, MI Bruce Pollock, M.D.	Fred Meyer, M.D.		
Rochester, MN Charles C. Rich, M.D.	J. Charles Rich, M.D.		
Salt Lake City, UT Daniel Robertson, M.D.	James Robertson, M.D.		
Olive Branch, MS Gerald Rodts, M.D.	Suzie Tindall, M.D.		
Atlanta, GA Philip Stieg, M.D.	Peter M. Black, M.D., Ph.D		
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Bruce Sorensen, M.D. Salt Lake City, UT	·		
Ronald Warnick, M.D Cincinnati, OH	Harry VanLoveren, M.D.		
Christopher Wolfla, M.D. Edmond, OK	Christopher Loftus, M.D.		
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ACADEMY AWARD WINNERS

Paul M. Lin	1955
Hubert L. Rosomoff	
Byron C. Pevehouse	
Norman Hill	
Jack Stern	
Robert Ojemann	
Lowell E. Ford	
Charles H. Tator	1963
Earle E. Crandall	1964
Stephen Mahaley, Jr.	
Chun Ching Kao	
John P. Kapp	1967
Yoshio Hosobuchi	
Gary G. Ferguson	1970
Richard L. Pressley	
David G. McLone	1972
Arden F. Reynolds, Jr.	1973
Richard L. Rapport	1974
Andrew G. Shetter	
John R. Howe	
Howard W. Blume	
Howard J. Senter	
Elisabeth M. Post	
David Dubuisson	
Dennis A. Turner	
Marc R. Bayberg	
David S. Baskin	
Kevin J. Kiwak	
Terry Lichtor	
Michael G. Nosko	
Joseph R. Madsen	
James T. Rutka	
Christopher D. Heffner	
Scott I. Gingold	
Mary Louise Hlavin	1001
Adam P. Brown	
Michael Tymianski	1772
David Garrett, Jr	1 ファン
L. Brannon Thomas	1 ブブサ 1 ハハチ
John S. Yu	
Gregory Canute	
— ·	
Nathan R. Selden	1998

MEETINGS OF THE ACADEMY

Hotel Netherland Plaza, Cincinnati, Ohio	
Roosevelt Hotel, New Orleans, Louisiana	
Tudor Arms Hotel, Cleveland, Ohio	
Mark Hopkins Hotel, San Francisco, California	
Ambassador Hotel, Los Angelas, California	
The Palmer House, Chicago, Illinois	
Hart Hotel, Battle Creek, Michigan	September 17-18, 1943
Ashford General Hospital,	
White Sulphur Springs, West Virginia	
The Homestead, Hot Springs, Virginia	
Boardmoor Hotel, Colorado Springs, Colorado	October 9-11, 1947
Windsor Hotel, Montreal, Canada	September 20-22, 1948
Benson Hotel, Portland, Oregon	October 25-27, 1949
Mayo Clinic, Rochester, Minnesota	September 28-30, 1950
Shamrock Hotel, Houston, TX	October 4-6, 1951
Waldorf-Astoria Hotel, New York CitySep	otember 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	
Del Monte Lodge, Pebble Beach, California	· ·
Copley Sheraton Plaza, Boston, Massachusetts	
Royal Orleans, New Orleans, Louisiana	
El Mirador, Palm Springs, California	October 23-26, 1963
The Key Biscayne, Miami, Florida	November 11-14, 1964
Terrace Hilton Hotel, Cincinnati, Ohio	
Fairmont Hotel & Towers, San Francisco, California	
The Key Biscayne, Miami, Florida	November 8-11, 1967
Broadmoor Hotel, Colorado Springs, Colorado	October 6-8, 1968
St. Regis Hotel, New York City	September 21, 1969
Camino Real, Mexico City	November 18-21, 1970
Sahara-Tahoe Hotel, Stateline, Nevada	September 26-30, 1971
New College, Oxford, England	September 4-7, 1972
Huntington-Sheraton Hotel, Pasadena, California	
Southampton Princess Hotel, Bermuda	
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The Wigwam (Litchfield Park), Phoenix, Arizona	*
Mills Hyatt House, Charleston, South Carolina	
Mauna Kea Beach Hotel, Kamuela, Hawaii	
Hotel Bayerischer Hof, Munich, Germany	October 22-25, 1978
Hyatt Regency, Memphis, Tennessee	
Waldorf Astoria, New York City	October 1-4, 1980
Sheraton Plaza, Palm Springs, California	November 1-4, 1981
Ritz-Carlton Hotel, Boston, Massachusetts	October 10-13, 1982
The Lodge at Pebble Beach, California	October 23-26, 1983
The Homestead, Hot Springs, Virginia	October 17-20, 1984
The Lincoln Hotel Post Oak, Houston, Texas	
The Cloister, Sea Island, Georgia	November 5-8, 1986
Hyatt Regency, San Antonio, Texas	October 7-10, 1987
Omni Netherland Plaza, Cincinnati, Ohio	September 13-17, 1988
Loews Ventana Canyon, Tucson,	•
Arizona	September 27-October 1, 1989
Amelia Island Plantation, Amelia Island, Florida	October 2-7, 1990
Salishan Lodge, Gleneden Beach, Oregon	September 22-26, 1991
Ritz-Carlton Hotel, Naples, Florida	October 21-25, 1992
The Wigwam, Phoenix, Arizona	October 27-30, 1993
The Cloister, Sea Island, Georgia	November 3-6, 1994
Loewis Ventana Canyon Resort, Tucson, Arizona	November 1-5, 1995
The Greenbrier, White Sulphur Springs,	
West Virginia	September 18-22, 1996
Rimrock Resort, Banff, Alberta, Canada	September 10-14, 1997
Four Seasons Biltmore, Santa Barbara, California	November 4-7, 1998
Ritz Carlton, Amelia Island, Florida	November 10-13, 1999
FUTURE MEETINGS:	
The Broadmoor, Colorado Springs, Colorado	October 11-14, 2000
Colonial Williamsburg Williamsburg,	
Virginia	. October 28-November 3, 2001

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PAST PRESIDENTS

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

Francis Murphey1941	John R. Green1972
William S. Keith1942	George J. Jayes1973
John Raaf1943	Richard L. DeSaussure 1974
Rupert B. Raney1944	Ernest W. Mack1975
Arthur R. Elvidge1946	Frank E. Nulsen1976
John Raaf1947	Robert S. Knighton1977
Arthur R. Elvidge1948	Robert G. Fisher 1978
F. Keith Bradford1949	H.T. Ballantine, Jr 1979
David L. Reeves1950	George Ehni1980
Henry G. Schwartz1951	Courtland H. Davis, Jr 1981
J. Lawrence Pool1952	John F. Mullan 1982
Rupert B. Raney1953	Hugo Rizzoli1983
David L. Reeves1954	James W. Correll1984
Stuart N. Rowe1955	E. Bruce Hendrick1985
Jess D. Herrmann1956	Griffith R. Harsh III1986
George S. Baker 1957	Ellis B. Keener1987
Samuel R. Snodgrass 1958	Robert Grossman1988
C. Hunter Shelden1959	Jim Story1989
Edmund Morrissey1960	John Jane1990
Donald F. Coburn 1961-62	Stewart Dunsker1991
Deben Alexander, Jr 1963	Burton Onofrio1992
George L. Maltby1964	Martin Weiss1993
Robert Pudenz1965	John M. Tew, Jr1994
Francis A. Echlin1966	John Van Gilder1995
Benjamin Whitcomb1967	Edward Connolly1996
Homer S. Swanson1968	George Ojemann 1997
Augustus McCravey 1969-70	Charles H. Tator 1998
Edward W Davis 1071	

PAST SECRETARY-TREASURERS Eben Alexander, Jr. 1954-57 Francis Murphey 1938-40 Robert L. McLaurin 1958-62 A. Earl Walker 1941-43 Edward W. Davis...... 1963-65 Theodore C. Erickson ... 1944-47 Robert G. Fisher 1966-68 Wallace B. Hamby 1948-50 Byron C. Pevehouse 1969-72 Theodore B. Rasmussen 1951-53 PAST SECRETARIES Byron C. Pevehouse 1973 Nicholas T. Zervas...... 1987-89 Russell H. Patterson, Jr., 1974-76 William A. Buchheit 1990-92 Phanor L. Perot. Jr. 1977-80 Julian T. Hoff 1992-95 John T. Garner 1981-83 Roberto C. Heros 1995-98 James T. Robertson 1984-86 **PAST TREASURERS** William A. Buchheit 1987-89 Russell H. Patterson, Jr...... 1973 Julian T. Hoff 1990-92 Phanor L. Perot. Jr. 1974-76 Roberto C. Heros 1992-95 John T. Garner 1977-80 James T. Robertson 1981-83 David G. Piepgras 1995-98 Nicholas T. Zervas 1984-86

HONORARY MEMBERS

GUY LAZORTHES (Annick)	Elected 1973
VALENTINE LOGUE (Anne)	1974
BERNARD PERTUISET Hospital de la Pitie 83 Boulevard de liHopital 75651 Paris, Cedex 13 FRANCE	1986
KEIJI SANO (Yaeko)	1975

SENIOR MEMBERS

•

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

W. KEMP CLARK (Fern)
WILLIAM COLLINS, JR. (Gwendolyn)
EDWARD CONNOLLY (Elise)
JAMES CORRELL (Cynthia)
COURTLAND DAVIS, JR. (Carrie Chamberlain) 1967 2525 Warwick Road Winston-Salem, NC 27104
RICHARD DESAUSSURE, JR. (Phyllis)
DONALD DOHN (Carolyn)
WILLIAM FEINDEL (Faith)
ROBERT FISHER (Constance)

ŧ

)	
•	
•	ELDON FOLTZ (Catherine)
•	UCI Medical Center
_	Bldg. 3, Rm. 313, Route 81 101 The City Drive South
•	Orange, CA 92868
•	06-1, 0 / 2
)	RICHARD FRASER (Sara Ann)1976
)	525 East 68th Street
	New York, NY 10021
)	LYLE FRENCH (Gene) 1954
3	P.O. Box 1007
•	Pauma Valley, CA 92061-1007
•	·
)	JOHN GARNER (Candace)
	67 Hillsdale Drive Newport Beach, CA 92660-4235
)	Newport Beach, CA 92000-4233
)	HENRY GARRETSON (Marianna Schantz) 1973
)	Neurological Surgery, Suite 1102
)	University of Kentucky
	210 East Gray Street
)	Louisville, KY 40202-3907
)	SIDNEY GOLDRING (Lois) 1964
)	Neurosurgery, CB-8057
)	Washington University
	660 South Euclid
)	St. Louis, MO 63110-1094
)	PHILIP GORDY (Silvia) 1968
•	3601 Carmel Drive
)	Casper, WY 82604-4949
)	
	ROBERT GROSSMAN (Ellin) 1984
)	Department of Neurosurgery
)	Baylor College of Medicine One Baylor Place
)	Houston, TX 77030
•	
)	GRIFF HARSH, III (Craig) 1980
	P.O. Box 232
)	Sweetwater, TN 37874-0232
)	73
)	

MAJOR GEN. GEORGE HAYES1962
Apartment 113
221 Booth Street
Gathersburg, MD 20878
MARK PETER HEILBRUN (Robyn)1984
Neurosurgery, #313406
University of Utah
50 North Medical Drive
Salt Lake City, UT 84132
E. BRUCE HENDRICK (Gloria) 1968
63 Leggett Avenue
Toronto, Ontario M9P 1X3
CANADA
JULIAN T. HOFF (Diane)
Neurosurgery, TC 2128
University of Michigan
1500 East Medical Center Drive
Ann Arbor, MI 48109-0338
Allii Albol, WI 40109-0330
HAROLD HOFFMAN (Jo Ann) 1982
The Hospital for Sick Children
555 University Avenue
Toronto, Ontario M5G 1X8
CANADA
EDGAR HOUSEPIAN (Marion Grace Lyon)
The Neurological Institute
710 West 168th Street
New York, NY 10032-2603
New Tork, N 1 10032-2003
ALAN HUDSON (Susan) 1978
The Toronto Hospital, Bell Wing 1-658
585 University Avenue
Toronto, Ontario M5G 2C4
CANADA
JOHN JANE, SR. (Noella)
Neurosurgery, Box 212
University of Virginia
Health Science Center
Charlottesville, VA 22908

€

Ę

•	PETER JANNETTA (Diana) 1994
	Neurosurgery, Suite B-400
•	Presbyterian University Hospital
•	230 Lothrop Street Pittsburgh, PA 15213-2582
•	1 100001g., 1.1 10210 0000
•	ELLIS KEENER (Ann) 1978
	915 East Lake Drive, N.W.
•	Gainesville, GA 30508-1729
•	DAVID KELLY, JR. (Sarah {Sally})
•	Department of Neurosurgery
_	Wake Forest University
•	Medical Center Boulevard
•	Winston-Salem, NC 27157-1029
•	WILLIAM KELLY (Joan)1977
)	Apartment B102
	16925 Inglewood Road NE
)	Bothell, WA 98011
)	
•	ROBERT KING (Molly Gibbs) 1958
	Department of Neurosurgery State University of New York
•	750 East Adams Street
)	Syracuse, NY 13210-2306
)	59140436, 111 15210 2500
)	WOLFF KIRSCH (Marie-Claire) 1971
	1360 Prospect
)	Redlands, CA 92373
•	ROBERT KNIGHTON (Louise) 1966
)	9288 Avenida San Timoteo
)	Cherry Valley, CA 92223-4314
)	THEODORE KURZE (Joan)1967
)	Suite D
	510 31st Street
)	Newport Beach, CA 92663-3806
)	THOMAS LANGFITT (Carolyn) 1971
)	Glenmede Corporation
)	One Liberty Place, Suite 1200 1650 Market Street
•	Philadelphia, PA 19103-7391
)	75
<i>9</i>	·-

SANFORD LARSON (Jackie) 1989	J
Department of Neurosurgery	
Medical College of Wisconsin	
9200 West Wisconsin Avenue	
Milwaukce, WI 53226	
RAEBURN LLEWELLYN (Carmen Rolon) 1963	
Unit 6D	
3 Poydras Street	
New Orleans, LA 70130-1665	
DON LONG (Harriett) 1983	
Neurosurgery, Meyer 7-109	
Johns Hopkins Medical School	
600 North Wolfe	
Baltimore, MD 21287-7709	
WILLIAM LOUGHEED 1962	
178 Klempenfeld Drive	
Barrie, Ontario L4M 1C3	
CANADA	
JOHN LOWREY (Catherine {Katy})1965	
Box 6989	
Kamuela, HI 96743-6989	
ALFRED LUESSENHOP (Frances) 1977	
Department of Neurosurgery	
Georgetown University Hospital	
3800 Reservoir Road	
Washington, DC 20007	
ERNEST MACK 1956	
235 Juniper Hill Road	
Reno, NV 89509-2935	
LEONARD MALIS (Ruth)	
219-44 Peck Avenue	
Hollis Hills, NY 11427-1122	

9	
•	
)	ROBERT MCLAURIN (Sarah) 1955 900 4th & Vine Tower
•	Cincinnati, OH 45202
)	
	JOHN MULLAN (Vivian Dunn)
)	5844 Stony Island Avenue Chicago, IL 60637-2022
•	•
)	BLAINE NASHOLD, JR. (Irene)
•	Division of Neurosurgery Box 3807
•	Duke University Medical Center
)	Durham, NC 27710-0001
	GUY ODOM1946
)	2812 Chelsea Circle
)	Durham, NC 27707-5133
•	DODEDT OFFICENCY (I)
)	ROBERT OJEMANN (Jean) 1968 Neurosurgery Service
)	Massachusetts General Hospital
)	Fruit Street
)	Boston, MA 02114
	BURTON ONOFRIO (Judith) 1975
)	1105 Tenth Street SW
)	Rochester, MN 55905
)	RUSSEL PATTERSON, JR. (Julie) 1971
)	Apartment #65A
)	146 West 57th Street
)	New York, NY 10019-3301
	SYDNEY PEERLESS (Ann) 1977
)	Neurosurgery, Suite 209
)	Mercy Neuroscience Institute 3661 South Miami Avenue
)	Miami, FL 33133-4206
)	
)	PHANOR PEROT, JR
)	Neurosurgery, 428 CSB Medical Univ. of South Carolina
y N	96 Jonathan Lucas Street
)	Charleston, SC 29425
)	
	77

BYRON CONE PEVEHOUSE (Lucy)
J. LAWRENCE POOL
ROBERT PORTER (Dean)
JOHN RAAF (Lorene)
AIDEN RANEY
JOSEPH RANSOHOFF, II (Lori Cohen, DDS)
THEODORE RASMUSSEN
ALBERT RHOTON, JR. (Joyce)
HUGO RIZZOLI (Helen)

•	
•	THEODORE ROBERTS (Joan) 1976
•	Neurosurgery, CH-50 University of Washington
	P.O. Box 5371
)	Seattle, WA 98105
)	
)	JAMES ROBERTSON (Valeria) 1971 Sofamor Danek
•	1800 Pyramid Place
)	Memphis, TN 38132
	•
	EDWARD SELJESKOG (Peggy) 1992
•	Neurosurgery, Suite 110
•	2805 Fifth Street South Rapid City, SD 57701-7306
•	Rapid City, 3D 37701-7300
)	C. HUNTER SHELDEN 1941
•	Huntington Medical Research Institute
	10 Rico Street
)	Pasadena, CA 91105-3201
)	WILLIAM SHUCART (Laura)
•	Department of Neurosurgery
)	New England Medical Center
)	750 Washington Street
	Boston, MA 02111
)	JAMES SIMMONS (Vanita) 1975
)	190 South Grove Park Road
•	Memphis, TN 38117
)	VENDETTI CMITTI ID (Mariania) 1007
)	KENNETH SMITH, JR. (Marjorie) 1987 Division of Neurosurgery
	St. Louis University
)	3635 Vista Avenue at Grand Boulevard
)	St. Louis, MO 63110-0250
)	ROBERT SMITH 1989
)	Neurosurgery, Suite 230
)	5903 Ridgewood Road
	Jackson, MS 39211
)	
)	
þ	79

)

BENNETT STEIN (Bonita) 1970
Neurosurgery, Room 204
Columbia University
710 West 168th Street
New York, NY 10032-2603
JIM STORY (Joanne)
Suite 1240
315 North San Saba
San Antonio, TX 78207-3154
ANTHONY SUSEN (Patricia)1965
193 Old Glebe Point Road
Burgess, VA 22432-9801
NULLIAN ON FETT (FULLIA)
WILLIAM SWEET (Elizabeth)
309 Goddard Avenue
Brookline, MA 02445
RONALD TASKER (Mary) 1971
Neurosurgery, McL. 2-431
Toronto Hospital, Western Division
399 Bathurst Street
Toronto, Ontario M5T 2S8
CANADA
OHADI ES TATOR (CI)
CHARLES TATOR (Carol)
Neurosurgery, McL. 2-435
Toronto Hospital, Western Division
399 Bathurst Street
Toronto, Ontario M5T 2S8
CANADA
GEORGE TINDALL (Wendy) 1968
Department of Neurosurgery
Emory University Clinic
1327 Clifton Road NE
Atlanta, GA 30322-1013
1 manua
JOHN TYTUS (Virginia)
3827 East Crockett Street
Seattle, WA 98112

(

•	
•	JOHN VAN GILDER (Kerstin)
•	University of Iowa Hospitals
)	200 Hawkins Drive
•	Iowa City, IA 52242
)	EXUM WALKER (Nellie)1938
)	735 Peachtree Battle Avenue, NW Atlanta, GA 30327-1250
•	·
•	CLARK WATTS (Patricia) 1975 5922 Northwest Place
)	Austin, TX 78731
)	LOWELL WINES ID (Mar.)
	LOWELL WHITE, JR. (Margie)
)	Mukilteo, WA 98275
)	ROBERT WILKINS (Gloria) 1973
)	Neurosurgery, Box 3807
)	Duke University Medical Center
)	Durham, NC 27710-0001
)	CHARLES WILSON (Francie Petrocelli)
)	Neurological Surgery, Room U-125 Univ. of California, San Francisco
)	533 Parnassus Avenue
)	San Francisco, CA 94143-0112
)	DAVID YASHON (Christine) 1972
)	Suite 1201
•	1492 East Broad Street Columbus, OH 43205-1546
)	
)	HAROLD YOUNG (M. Theresa)
)	Medical College of Virginia Station
)	Richmond, VA 23298
<i>)</i>	NICHOLAS ZERVAS (Thalia) 1972
	Neurosurgery, White 502
)	Massachusetts General Hospital Fruit Street
)	Boston, MA 02114-2698
)	
.)	81

ACTIVE MEMBERS

Ę

MICHAEL APUZZO (Helene) Neurosurgery, Suite 5046 University of Southern California 1200 North State Street Los Angeles, CA 90033-4525	Elected 1988
ISSAM AWAD (Catherine) Neurosurgery, TMP 405	1996
Yale University 333 Cedar Street	
New Haven, CT 06520-3206	
DANIEL BARROW (Mollie Winston)	1993
H. HUNT BATJER (Janet)	1996
MITCHEL BERGER (Joan)	1997
KEITH BLACK (Carol Bennett) Neurosurgery, Suite 490W Cedars-Sinai Neurological Institute 8635 West Third Street Los Angeles, CA 90048	1995

-	
•	
•	PETER BLACK (Katharine) 1988
•	Department of Neurosurgery
	Childrens Hospital
•	300 Longwood Avenue Boston, MA 02115
•	Boston, WA 02113
•	LAWRENCE BORGES (Susan) 1993
)	Neurosurgery, White 1205
	Massachusetts General Hospital
•	32 Fruit Street
•	Boston, MA 02114
•	CHARLES BRANCH, JR. (Lesa) 1996
)	Department of Neurosurgery
	Wake Forest University
•	Medical Center Boulevard
•	Winston-Salem, NC 27157-1029
•	HENRY BREM (Rachel)
)	Neurosurgery, Carnegie 466
	Johns Hopkins Hospital
)	600 North Wolfe Street
)	Baltimore, MD 21287-0001
)	
}	WILLIS BROWN, JR. (Ann)
	Division of Neurosurgery Univ. of Texas Health Science Center
)	7703 Floyd Curl Drive
•	San Antonio, TX 78284-7843
)	
)	DEREK BRUCE (Frances) 1984
	Neurosurgery, Suite B308
)	7777 Forest Lane
•	Dallas, TX 75230-2505
)	KIM BURCHIEL (Debra)
	Neurosurgery, L-472
)	Oregon Health Sciences University
)	3181 S.W. Sam Jackson Park Road
)	Portland, OR 97201-3098
)	
)	

MARTIN CAMINS (Joan) 1995	
Neurological Surgery, Suite T 1-C	
205 East 68th Street	
New York, NY 10021-5735	
PETER CARMEL (Jacqueline Bello) 1991	
Neurosurgery, Suite 7300	
New Jersey Medical School	
90 Bergen Street	
Newark, NY 07103-2499	
WILLIAM CHANDLER (Susan) 1989	
2124D Taubman Health Center	
University of Michigan	
1500 East Medical Center Drive	
Ann Arbor, MI 48109-0338	
PAUL CHAPMAN (Tansy) 1983	
Neurosurgery, GRB502	
Massachusetts General Hospital	
55 Fruit Street	
Boston, MA 02114	
PAUL COOPER (Leslie)	
Department of Neurosurgery	
New York University Medical Center	
550 First Avenue	
New York, NY 10016-6481	
REES COSGROVE (Karen) 1997	
Neurosurgery, Suite 331	
Massachusetts General Hospital	
15 Parkman Street	
Boston, MA 02114-2696	
RALPH DACEY, JR. (Corinne Mears) 1990	
Neurosurgery, CB 8057	
Washington University	
660 South Euclid	
St. Louis, MO 63110	

,	
•	
)	ARTHUR DAY (Dana) 1990
)	Department of Neurosurgery
	University of Florida P.O. Box 100265
•	Gainesville, FL 32610-0265
•	
•	ROBERT DEMPSEY (Diane) 1996
	Neurosurgery, H4/338
)	University of Wisconsin
3	600 Highland Avenue
•	Madison, WI 53792-0001
•	STEWART DUNSKER (Ellen) 1975
)	Suite 441
	Mayfield Clinic & Spine Institute
•	2123 Auburn Avenue
)	Cincinnati, OH 45219-2970
•	MICHAEL EDWARDS (Linda) 1992
)	Neurosurgery, Suite 340
	2800 L Street
)	Sacramento, CA 95816
)	
)	HOWARD EISENBERG (Janet) 1985
)	Neurosurgery, S12D10A University of Maryland
	22 South Greene Street
•	Baltimore, MD 21201-1734
•	·
)	MEL EPSTEIN (Lynn) 1992
)	Suite 100
	Neurosurgery Foundation, Inc. 55 Claverick Street
)	Providence, RI 02903
)	1104100100, 11 02703
)	EUGENE FLAMM (Susan) 1979
)	Department of Neurosurgery
	Beth Israel North
)	170 East End Avenue
)	New York, NY 10128
•	
)	
	n-

ALLAN FRIEDMAN (Elizabeth Bullitt)	
WILLIAM FRIEDMAN (Ransom) 1995	
Department of Neurosurgery	
University of Florida Health Sciences Center	
P.O. Box 100265	
Gainesville, FL 32610-0265	
DANIEL FULTS, III (Carol) 1997	
Department of Neurosurgery	
University of Utah	
50 North Medical Drive	
Salt Lake City, UT 84132-0001	
STEVEN GIANNOTTA (Sharon)	
Neurosurgery, Suite 5046	
University of Southern California	
1200 North State Street	
Los Angeles, CA 90033-4525	
ROBERT GRUBB, JR. (Julia) 1985	
Neurosurgery, Campus Box 8057	
Washington University	
660 South Euclid Avenue	
St. Louis, MO 63110-1010	
JOSEPH HAHN (Andrea)	
Neurosurgery/S80	
Cleveland Clinic Foundation	
9500 Euclid Avenue	
Cleveland, OH 44195-1004	
STEPHEN HAINES (Jennifer Plambon) 1994	
Neurosurgery, Suite 428	
Medical Univ. of South Carolina	
96 Jonathan Lucas Street	
Charleston, SC 29425	

•

•	
•	ROBERTO HEROS (Deborah)1985
	Department of Neurosurgery
•	University of Miami
)	1501 NW 9th Avenue
•	Miami, FL 33136
•	CHARLES HODGE, JR
	Department of Neurosurgery
•	State University Hospital
•	750 East Adams Street
•	Syracuse, NY 13210
)	L. NELSON (NICK) HOPKINS, III (Ann {Bonnie}) 1992
	Department of Neurosurgery
•	State University of New York
•	3 Gates Circle
)	Buffalo, NY 14209-1194
)	DATDICK RELIAN (Compl) 1000
	PATRICK KELLY (Carol)
)	New York University
)	550 First Avenue
)	New York, NY 10016
)	
)	GLENN KINDT (Charlotte)
	Neurosurgery, Box C307 University of Colorado
)	4200 East 9th Avenue
)	Denver, CO 80262-0001
)	,
	DAVID KLINE (Nell) 1971
)	Department of Neurosurgery
•	Louisiana State Univ. Medical Center
)	1542 Tulane Avenue New Orleans, LA 70112
)	New Officialis, LA 70112
	DOUGLAS KONDZIOLKA (Susan)
)	Neurosurgery, B-400
•	Univ. of Pittsburgh Medical Center
•	200 Lothrop Street
)	Pittsburgh, PA 15213-2582
)	
3	87

EDWARD LAWS, JR. (Margaret) 1983	
Department of Neurosurgery	
University of Virginia	
Box 212 Health Science Center	
Charlottesville, VA 22908-0001	
CHRISTOPHER LOFTUS (Sara) 1992	
Neurosurgery, #206	
University of Oklahoma	
711 Stanton L. Young Boulevard	
Oklahoma City, OK 73104	
L. DADE LUNSFORD (Julianne) 1992	
Neurosurgery, B-400	
Univ. of Pittsburgh Medical Center	
200 Lothrop Street	
Pittsburgh, PA 15213-2582	
1110001611, 111 10010 2002	
NEIL MARTIN 1997	
Department of Neurosurgery	
UCLA Medical Center	
Box 957039	
Los Angeles, CA 90025-7039	
Los Aligoids, CA 70025-7057	
ROBERT MARTUZA (Jill) 1989	
Neurosurgery/PHC 1	
Georgetown University Medical Center	
3800 Reservoir Road NW	
Washington, DC 20007-2197	
174511116ton, 2 = 2000 / 217 /	
ROBERT MAXWELL (Karen) 1992	
Neurosurgery, Box 96	
University of Minnesota	
420 Delaware Street SE	
Minneapolis, MN 55455-0374	
MARC MAYBERG (Terry)1995	
Neurosurgery/S80	
The Cleveland Clinic	
9500 Euclid Avenue	
Cleveland, OH 44195	
•	

€

•

•

)	J. GORDON MCCOMB (Rhoda) 1998
	Neurosurgery, #906
	1300 North Vermont Avenue
	Los Angeles, CA 90027
•	PAUL MCCORMICK (Doris) 1998
•	Department of Neurosurgery
)	Columbia Presbyterian Medical
	710 West 168th Street
)	New York, NY 10032
•	J. MICHAEL MCWHORTER (Barbara) 1989
•	Carolina Neurosurgical Associates
)	2810 North Maplewood Avenue
	Winston-Salem, NC 27103-4151
)	EDEDDIC MEVED (I-ma Mairren) 1005
•	FREDRIC MEYER (Irene Meissner)
•	Mayo Clinic
)	200 First Street SW
)	Rochester, MN 55905
	DICHARD MODAWETT (Many Lane) 1000
}	RICHARD MORAWETZ (Mary Jean)
)	University of Alabama
)	1600 Seventh Avenue South
)	Birmingham, AL 35294-3295
)	PAUL NELSON (Teresa)
)	Neurosurgery, Emerson #139 Indiana University
•	545 Barnhill Drive
•	Indianapolis, IN 46202-5124
)	• • • • • • • • • • • • • • • • • • • •
	GEORGE OJEMANN (Dr. Linda M.) 1975
)	Neurological Surgery, Box 359924
)	University of Washington
)	1959 N.E. Pacific Street Seattle, WA 98195-9924
	Scattic, WA 90193-3924
)	
)	
)	
à	on.

EDWARD OLDFIELD (Susan)
ANDRE OLIVIER (Nicole)
TAE SUNG PARK (Hyun Sook Kim)
DAVID G. PIEPGRAS (Jane)
LAWRENCE PITTS (Mary)
KALMON POST (Linda)
DONALD QUEST (Ilona)

((`

(

7	
•	
•	COREY RAFFEL (Kathy)
	Department of Neurologic Surgery
•	Mayo Clinic
•	200 First Street SW
•	Rochester, MN 55905
	BERT RATCHESON (Peggy Steiner) 1986
•	Department of Neurosurgery
•	University Hospitals of Cleveland
•	11100 Euclid Avenuc
	Cleveland, OH 44106-5000
•	
)	J. CHARLES RICH, JR. (Jasmine) 1987
•	Neurosurgery, Suite 111
	Neurosurgical Associates, Inc.
•	370 Ninth Avenue
•	Salt Lake City, UT 84103-2677
•	
	DAVID ROBERTS (Kathryn)
•	Section of Neurosurgery
)	Dartmouth-Hitchcock Medical Center One Medical Center Drive
)	Lebanon, NH 03758-0001
	Ecoulidii, 1111 03/30 0001
•	JON ROBERTSON (Carol Ann) 1992
•	Neurosurgery, Suite 600
•	920 Madison Avenue
	Memphis, TN 38103
•	
•	ROBERT ROSENWASSER (Deborah August) 1996
)	Neurosurgery, Suite 650
	Jefferson Faculty Foundation
•	834 Walnut Street Philadelphia, PA 19107-5102
•	Finaucipina, FA 1910/-5102
•	JAMES RUTKA (Mari)
	Neurosurgery, Suite 1502
)	The Hospital for Sick Children
)	555 University Avenue
)	Toronto, Ontario M5G 1X8
	CANADA
)	
)	

	É
DUKE SAMSON (Patricia) 1994	•
Department of Neurosurgery	()
Univ. of Texas, Southwestern Med. Center	•
5323 Harry Hines Boulevard Dallas, TX 75235-8855	
Danas, 1A 13233-0033	•
R. MICHAEL SCOTT (Susan)1991	(,
Neurosurgery, Bader 319	()
Childrenis Hospital	()
300 Longwood Avenue Boston, MA 02115-5724	•
200001, 1411 02113 3724	
WARREN SELMAN (Diana) 1995	
Neurosurgery, HHS 5042	()
University Hospitals of Cleveland 11100 Euclid Avenue	()
Cleveland, OH 44106	(
	()
CHRISTOPHER SHIELDS (Deborah) 1993	
Neurosurgery, Suite 1102	
University of Louisville 210 East Gray Street	€)
Louisville, KY 40202	()
	€)
FREDERICK SIMEONE 1981	
Department of Neurosurgery	•
Pennsylvania Hospital 909 Walnut Street	(·
Philadelphia, PA 19107	
•	
ROBERT SOLOMON (Barbara)	()
Neurological Institute of New York 710 West 168th Street	()
New York, NY 10032-2603	_
, and the second se	
VOLKER SONNTAG (Lynne)1995	
Division of Neurological Surgery Barrow Neurological Institute	
2910 North Third Avenue	()
Phoenix, AZ 85013	()
	()
	_
92	& .

3	DENNIS SPENCER (Susan) 1989
•	Neurological Surgery, Box 208082
•	Yalc University
•	333 Cedar Street
•	New Haven, CT 06520-8082
	ROBERT SPETZLER (Nancy) 1997
•	Barrow Neurosurgical Assoc., Ltd.
•	2910 North Third Avenue
•	Phoenix, AZ 85013-4496
•	TOTAL TO A CO. NO. NO. NO. NO. NO. NO. NO. NO. NO. N
	JOHN TEW, JR. (Susan)
)	University of Cincinnati
•	231 Bethesda Avenue
)	Cincinnati, OH 45267-0515
•	
	SUZIE TINDALL
)	Department of Neurosurgery
)	Emory University 1365 Clifton Road NE
•	Atlanta, GA 30322-1013
)	Attailat, OA 30322-1013
	RUSSELL TRAVIS 1994
•	Neurosurgical Associates, #485B
•	1401 Harrodsburg Road
•	Lexington, KY 40504-3700
•	HARRY VAN LOVEREN (Judy)
•	Neurosurgery, Suite 110
	3219 Clifton Avenue
)	Cincinnati, OH 45220-3027
•	
)	RAND VOORHIES (Terry) 1996
	Department of Neurosurgery
)	Ochsner Clinic 1514 Jefferson Highway
•	New Orleans, LA 70121-2483
)	Tion Orionio, Dr. 10121-2703
)	
)	
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<i>)</i>	•

	()
	€ ,
BRYCE WEIR (Mary Lou)	()
Section of Neurosurgery, MC 3026 University of Chicago	()
5841 South Maryland Avenue	•
Chicago, IL 60637	
MARTIN WEISS (Debby) 1981	•
Neurosurgery, Box 766	(,
USC Medical Center	€ >
1200 North State Street	€)
Los Angeles, CA 90033	()
H. RICHARD WINN (Debbie) 1993	()
Neurosurgery, Box 359924	
University of Washington	
700 Ninth Avenue Seattle, WA 98195-9924	•
Journal, 1111, 20172 7721	
FREMONT PHILIP WIRTH 1993	
4 Jackson Boulevard	()
Savannah, GA 31405-5810	()
ALLEN WYLER (Lily)1990	()
Epilepsy Center, Box 14999	()
Swedish Medical Center 747 Summit	()
Seattle, WA 98114-0999	•
	()
A. BYRON YOUNG (Judy) 1989	
Neurosurgery, MS101	€ >
University of Kentucky 800 Rose Street	()
Lexington, KY 40536-0084	()
	(
RONALD YOUNG 1986 Northwest Gamma Knife Center, #G-5	Ì
1560 North 115th Street	_
Seattle, WA 98133	()
	€ 1
	()
94	€.
	()

SENIOR CORRESPONDING

Elected	
R. LEIGH ATKINSON (Noela)	
FERNANDO CABIESES	
LUC CALLIAUW (Dora)	
JUAN CARLOS CHRISTENSEN (Diana Poli)	
GUISEPPE DALLE ORE (Guisi)	

7	
•	
•	JACQUES DEVILLIERS (Jeanne Marie Erica) 1986
•	Department of Neurosurgery University of Cape Town
	Observatory 7925
•	Cape Town 7
•	SOUTH AFRICA
•	
•	HANS ERICH DIEMATH (Dr. Karin) 1970
•	Department of Neurosurgery Landesnervenklinik
	Ignaz Harrer-Strasse 79
•	Salzburg, A-5020
•	AUSTRIA
•	
•	HERMANN DIETZ (Elfrun)
•	An Der Trist 10 B Hannover, 30559
•	GERMANY
•	
	F. JOHN GILLINGHAM (Judy) 1962
7	Easter Park House
9	Easter Park Drive
)	Edinburgh, EH4 6SN SCOTLAND
	SCOTLAND
))))	JAIME G. GOMEZ (Lucy) 1975
à	Suite 2-549
<i>9</i>	7040 West Palmetto Park Road
3	Boca Raton, FL 33433
•	CALVADOR CONTALEZ CORNEIO (Para) 1000
•	SALVADOR GONZALEZ-CORNEJO (Rosa) 1982 Av. Chapultepec Sua 130-204
)	Guadalajara, Jal. 44630
	MEXICO
)	
)	ERNST H. GROTE (Juliana)
•	Department of Neurosurgery University Kliniks Schnarrenberg
)	Hoppe Seyler-Str. 3
	72076 Tubingen
)	GERMANY
)	
)	

	£ '
HAJIME HANDA (Hiroko) 1985	(
Takeda General Hospital	()
26-1 Moriminami-cho, Ishida Fushimi-ku	
Kyoto, 601-1495	()
JAPAN	€ 4
JOHN HANKINSON (Nicole)	()
Westacres, Woolsington Hall Newcastle Upon Tyne, England NE13 8DG	()
UNITED KINGDOM	-
	()
FABIAN ISAMAT (Maria Victoria {Marivi}) 1989	(
Clinica Sagrade Familia	()
Neurogrup	€ ,
Torras y Pujalt, I 08022 Barcelona	
SPAIN	()
37 ,	()
SHOZO ISHII (Akiko) 1975	()
Department of Neurosurgery	()
Juntendo Medical College	()
2-1-1 Hongo, Bunkyo-ku Tokyo 113-8421	•
JAPAN	()
	()
HANS-PETER JENSEN 1980	()
Karolinenweg 23	
Kiel, 24105 GERMANY	
GERMAN I	()
KATSUTOSHI KITAMURA (Yoshiko) 1970	()
Neurosurgery Neurologic Institute	()
Kyushu University	()
3-1-1 Maidashi, Higashi-ku	
Fukuoka, 812-8582 JAPAN	()
JAPAN	()
SHIGEAKI KOBAYASHI (Hideko) 1998	()
Department of Neurosurgery	()
Shinshu University	()
Matsumoto 390-8621 JAPAN	
JAFAN	€ .

•	LAURI LAITINEN (Kerstin) 1972
)	Dano, FI-22340 Geta
	FINLAND
)	
•	RUEDIGER LORENZ 1998
•	Department of Neurosurgery
•	J. W. Goethe Univ. Clinic Schleusenweg 2-16
•	Frankfurt, Main 60528
)	GERMANY
	DALII MADINO ID (Annala)
•	RAUL MARINO, JR (Angela) 1977 R. Maestro Cardim 808/814
)	Sao Paulo, SP 01323-001
)	BRAZIL
)	
)	JORGE S. MENDEZ (Soledad)
)	Santiago
	CHILE
)	D D 114114 TOTAL (7 II)
)	B. RAMAMURTHI (Indira)
•	Voluntary Health Services Taramani
)	Chennai 600-113
)	INDIA
)	
	HANS-J. REULEN (Ute)
)	Klinikum Grosshadern
•	Marchioninistrasse 15
•	Munich 81377
)	GERMANY
)	MADJID SAMII (Mahschi) 1996
	Neurosurgery Clinic
)	Nordstadt Hospital
)	Haltenhoffstrasse 41
)	Hannover 30167 GERMANY
)	ODIGMEN I
)	
)	99
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	•
KURT-FRIEDRICH SCHURMANN 1978	(
Am Eselsweg 29 D-6500 Mainz 1	
GERMANY	(
CHARAS SUWANWELA 1972 Chulalongkorn Hospital	
Medical School	(
Bangkok	(
THAILAND	()
LINDSAY SYMON (Pauline)1982	()
Department of Neurosurgery	()
The National Hospital	
Queen Square	(;
London, England WC 1 UNITED KINGDOM	
UNITED KINGDOM	(:
KINTOMO TAKAKURA (Tsuncko) 1988	€ ;
Tokyo Womenís Medical University	
8-1 Kawadacho, Shinjukuku Tokyo, 162-8666	()
JAPAN	()
	()
KJELD VAERNET 1970 Gardes Alle 7, 4 TV	()
Hellerup, 2900	()
DENMARK	-
	•
SYDNEY ERIC WATKINS (Susan) 1975 Royal London Hospital	
Whitechapel	()
London, England E1 1BB	()
UNITED KINGDOM	()
M. GAZI YASARGIL (Dianne)	(
Neurosurgery, Slot 507	()
University of Arkansas	
4301 West Markham Little Rock, AR 72205-7199	
Little Ruck, AR 12205-1177	()
	(

CORRESPONDING

)	E	lected
)	H. ALAN CROCKARD (Caroline)	1992
•	Department of Surgical Neurology	
)	National Hospital	
	Queen Square	
•	London, England 1N 3BG	
)	UNITED KINGDOM	
•	NOEL G. DAN (Adrienne)	1989
•	Specialist Medical Center, Suite 302	
)	235-285 New South Head Road	
	Edgecliff, 2027	
•	Sydney, N.S.W.	
•	AUSTRALIA	
)	NICOLAS DE TRIBOLET (Veronique)	1995
)	Service de Neurochirurgie	
	Hopital Cantonal de Geneve	
)	Rue Micheli-du-Crest 24	
•	1211 Geneve 14	
)	SWITZERLAND	
)	VINKO DOLENC	1988
	Department of Neurosurgery	
)	University Hospital Center	
)	Zaloska 7	
)	1525 Ljubljana	
	SLOVENIA	
)	RUDOLPH FAHLBUSCH (Hanna)	1001
•	Neurochirurgische Klinik	. , , ,
)	Universitat Erlangen-Nurnberg	
)	Schwabachanlage 6	
	Erlangen, 91054	
)	GERMANY	
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#28 Cho Sec Sec	HAN (Sung Soon Cho)	91
De _l Kel 35 Tol	KAWASE (Mieko)) 7
De _l Roj Par Me	KAYE (Judith)	96
Dej Kyd 54, Sak	O KIKUCHI	93
Ins Qu Loi	HOMAS (Hazel)	95
	102	

DECEASED MEMBERS

•	Elected Deceased
•	JAMES R. ATKINSON 1970 1978
•	Phoenix, Arizona (Active)
•	
•	PERCIVAL BAILEY 1960
•	(Honorary)
)	GEORGE BAKER 1940 1993
3	Litchfield Park, Arizona
•	(Senior)
)	H. THOMAS BALLANTINE, JR 1951
)	Boston, Massachusetts (Senior)
)	
)	WILLIAM F. BESWICK 1959
)	(Active)
)	EDWIN B. BOLDREY 1941 1988
•	San Francisco, California
)	(Senior)
)	E. HARRY BOTTERELL 1938 1997
)	Kingston, Ontario, CANADA (Senior)
)	(Sellor)
)	SPENCER BRADEN Founder
)	Cleveland, Ohio (Active)
•	E VERTI DE ADECUD. 1020 1020
)	F. KEITH BRADFORD19381971 Houston, Texas
•	(Active)
)	JEAN BRIHAYE 1975 1999
•	Bruxelles, BELGIUM
)	(Senior Corresponding)
)	

		6
KARL-AUGUST BUSHE 1972	1999	(,
(Senior Corresponding)		(
HOWARD BROWN 1939 San Francisco, California	1990	
(Senior)		
JUAN CARDENAS 1966 1966 Mexico City, MEXICO	1996	•
(Senior Corresponding)		· ·
GALE CLARK 1970 Oakland, California	1996	
(Senior)		
DONALD COBURN	1988	
(Senior)		
WINCHELL McK. CRAIG 1942 Rochester, Minnesota	1960	
(Honorary)		()
EDWARD DAVIS 1949	1988	
(Senior)		
PEARDON DONAGHY 1970	1991	· · · · · · · · · · · · · · · · · · ·
(Senior)		(
CHARLES DRAKE 1958	1998	
(Senior)		-
FRANCIS ECHLIN 1944	1988	المراجعة المراجعة
(Senior)		•
DEAN ECHOLS Founder	1991	(
New Orleans, Louisiana (Senior)		ا ا
GEORGE EHNI 1964	1986	
Houston, Texas		•
(Senior)		•

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•	ARTHUR ELVIDGE
•	(Senior)
•	THEODORE ERICKSON 1940 1986
•	Madison, Wisconsin
)	(Senior)
•	JOSEPH EVANS Founder
•	(Senior)
•	JOHN FRENCH 1951
•	Los Angeles, California
•	(Senior)
•	JAMES GALBRAITH 1947 1997
)	Birmingham, Alabama (Senior)
•	
)	EVERETT GRANTHAM 1942 1997 Louisville, Kentucky
)	(Senior)
)	JOHN GREEN 1953
•	Phoenix, Arizona
•	(Senior)
)	JAMES GREENWOOD, JR 1952 1992 Houston, Texas
)	(Senior)
)	WESLEY GUSTAFSON 1942 1975
)	Jensen Beach, Florida
)	(Senior)
)	WALLACE HAMBY 1941 1999
)	Pompano Beach, Florida (Senior)
)	
)	HANNIBAL HAMLIN 1949 1982 Providence, Rhode Island
)	(Senior)
)	
)	
)	105
<i>y</i>	•••

JOHN HANBERY 1959 1996 Palo Alto, California (Senior)	
JESS HERRMANN	
HENRY HEYL	
WILLIAM HUNT 1970 1999 Columbus, Ohio (Senior)	
OLAN HYNDMAN 1942	
KENNETH JAMIESON	
SIR GEOFFREY JEFFERSON	
RICHARD JOHNSON	
WILLIAM KEITH Founder	
HUGO KRAYENBUHL	
KRISTIAN KRISTIANSEN 1967 1993 Oslo, Norway (Senior Corresponding)	

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•	WALPOLE LEWIN 1973
•	(Corresponding)
•	HERBERT LOURIE19651987
•	Syracuse, New York
•	(Senior)
•	WILLEM LUYENDIJK 1973 1995
•	Oegstgeest, NETHERLANDS
•	(Senior Corresponding)
•	M. STEPHEN MAHALEY 1972
)	Birmingham, Alabama (Active)
•	GEODGE MATERY 1942 1999
)	GEORGE MALTBY 1942
)	(Senior)
)	FRANK MARGUTH19781991
)	Munich, GERMANY (Scnior Corresponding)
)	(Senior Corresponding)
)	DONALD MATSON
)	Boston, Massachusetts (Active)
)	EDANIZ MANETELD
)	FRANK MAYFIELD Founder 1991 Cincinnati, Ohio
)	(Senior)
)	AUGUSTUS McCRAVEY 1944
)	Chattanooga, Tennessee
•	(Senior)
)	KENNETH McKENZIE
)	Toronto, CANADA (Honorary)
)	•
•	WILLIAM MEACHAM 1952
•	(Senior)
)	
)	107
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)

JAMES MEREDITH 1946 1962 Richmond, Virginia (Active)
J. DOUGLAS MILLER
W. JASON MIXTER 1951
EDMUND MORRISSEY 1941 1986 San Francisco, California (Senior)
FRANCIS MURPHEY Founder
GOSTA NORLEN
FRANK NULSEN 1956
SIXTO OBRADOR
PIETRO PAOLETTI
HANS-WERNER PIA
WILDER PENFIELD

((

•	HELMUT PENZHOLZ 1978 1985 Heidelberg, WEST GERMANY
)	(Corresponding)
))	ROBERT PUDENZ 1943 1998 South Pasadena, California
•	(Senior)
)	BRONSON RAY 1992 1993
•	New York, New York (Honorary)
•	•
)	DAVID REEVES
)	(Active)
)	DAVID REYNOLDS
)	Tampa, Florida (Active)
)	R. C. L. ROBERTSON 1946 1985
)	Houston, Texas (Senior)
)	
)	STEWART ROWE 1938 1984 Pittsburgh, Pennsylvania
)	(Senior)
)	RICHARD SCHNEIDER 1970 1986
))	Ann Arbor, Michigan (Senior)
<i>)</i>	HENRY SCHWARTZ 1942
)	St. Louis, Missouri
)	(Senior)
)	WILLIAM SCOVILLE 1944 1984 Hartford, Connecticut
)	(Senior)
)	R. EUSTACE SEMMES
)	Mcmphis, Tennessee (Honorary)
))	
Ź	109

)

SAMUEL SNODGRASS 1939
GLEN SPURLING
C. WILLIAM STEWART
THORALF SUNDT, JR
KENICHIRO SUGITA 1988 1994 Nagoya, Japan (Senior Corresponding)
HENDRIK SVIEN
HOMER SWANSON
ALFRED UIHLEIN
A. EARL WALKER
ARTHUR WARD, JR
THOMAS WEAVER, JR 1943

•	
•	
3	W. KEASLEY WELCH19571996
•	Waban, Massachusetts
•	(Senior)
•	BENJAMIN WHITCOMB 1947 1998
)	Surrey, Maine (Senior)
)	(Semor)
•	BARNES WOODHALL
)	Durham, North Carolina (Senior)
)	•
)	FRANK WRENN 1973 1990 Greenville, South Carolina
)	(Senior)
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NOTES:

THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

62nd Annual Meeting October 11-14, 2000

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