THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

60th Annual Meeting

FOUR SEASONS BILTMORE

Santa Barbara

A FOUR SEASONS · REGENT RESORT

November 4 - 7, 1998

Jointly Sponsored by The American Association of Neurological Surgeons

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MEETING OF
THE ACADEMY OF NEUROLOGICAL SURGERY
Schedule of Activities

Tuesday, November 3rd

12:00 noon	ABNS Primary Exam Committee *La Concha*
7:00 - 9:00	ABNS Dinner *La Marina*

Wednesday, November 4th

7:30 AM - 8:00 AM	ABNS Breakfast *La Concha*
8:00 AM - 4:00 PM	ABNS Primary Exam Committee *La Concha*
12:30 PM - 1:30 PM	ABNS Luncheon *La Fonda*
3:00 PM - 9:00 PM	Registration *La Pacifica Ballroom*
4:00 PM - 9:00 PM	Speaker Ready Room *La Fonda*
4:30 PM - 6:00 PM	American Academy Executive Committee Meeting *La Concha*
6:30 PM - 9:00 PM	Opening Reception *La Pacifica Ballroom*

Thursday, November 5th

7:00 AM - 5:00 PM	Registration *El Rincon*
7:00 AM - 1:00 PM	Speaker Ready Room/Editing Room *La Fonda*
7:00 AM - 9:00 AM	Buffet Breakfast (spouses) *La Marina*
7:00 AM - 8:00 AM	Buffet Breakfast and meeting (members) *La Pacifica Ballroom*
8:00 AM - 1:00 PM	Scientific Session I *Loggia Ballroom*
9:58 AM - 10:20 AM	Coffee Break *Loggia Terrace*
12:00 AM - 1:00 PM	ABNS Advisory Council Luncheon *North Patio*
1:00 PM	Golf – *Sandpiper Golf Course*
5:00 PM - 6:00 PM	Meeting of Research Task Force *La Salita*
6:00 PM - 10:00 PM	Buffet Dinner *Recreation Lawn*

Friday, November 6th

7:00 AM - 5:00 PM	Registration *El Rincon*
7:00 AM - 1:00 PM	Speaker Ready Room/Editing Room *La Fonda*
8:00 AM - 9:00 AM	Buffet Breakfast (spouses) *La Marina*
7:00 AM - 8:00 AM	Buffet Breakfast and Business Meeting (members) *La Pacifica Ballroom*
8:00 AM - 1:00 PM	Scientific Session II *Loggia Ballroom*
10:06 AM-10:36 AM	Coffee Break and Group Photograph *Loggia Terrace*
12:34 PM - 1:00 PM	**Presidential Address: Edward S. Connolly** *Introduction by:* Charles H. Tator
1:00 PM	Golf – *Sandpiper Golf Course*
1:00 PM	Tennis – *Four Seasons Biltmore Courts*
7:00 PM - 8:00 PM	Cocktails *Loggia Terrace*
8:00 PM - 11:00 PM	Black-tie Reception *Loggia Ballroom*

Saturday, November 7th

7:00 AM - 1:00 PM	Registration *El Rincon*
7:00 AM - 1:00 PM	Speaker Ready Room/Editing Room *La Fonda*
8:00 AM - 9:00 AM	Buffet Breakfast (spouses) *La Marina*
7:00 AM - 8:00 AM	Buffet Breakfast (members) *La Pacifica Ballroom*
8:00 AM - 1:00 PM	Scientific Session III *Loggia Ballroom*
10:08 AM-10:36 AM	Coffee Break *Loggia Terrace*
1:00 PM	Meeting adjourns

SCHEDULE OF ACTIVITIES
FOR SPOUSES

Wives of American Academy members and guests are welcome to all events.

Wednesday, November 4th

6:30 PM - 9:00 PM	Welcoming Reception *La Pacifica Ballroom*

Thursday, November 5th

7:00 AM - 9:00 AM	Buffet Breakfast *La Marina*
7:00 AM - 3:00 PM	Hospitality Suite *La Marina*
8:30 AM	Buses leave for Getty Museum *Entrance of the Four Seasons Biltmore Hotel*
9:00 AM - 5:00 PM	Tour of the Getty Museum and Lunch
6:00 PM - 9:00 PM	Buffet Dinner *Recreation Lawn (casual)*

Friday, November 6th

8:00 AM - 9:00 AM	Buffet Breakfast *La Marina*
8:00 AM - 3:00 PM	Hospitality Suite *La Marina*
8:30 AM	Book Club Meeting Guest author: Lynn Freed *La Marina*
1:00 PM	Tennis and Golf Bridge Shopping, touring, etc.
7:00 PM - 8:00 PM	Cocktails *Loggia Terrace*
8:00 PM - 11:00 PM	Black-tie Reception *Loggia Ballroom*

Saturday, November 7th

8:00 AM - 9:00 AM	Buffet Breakfast *La Marina*
8:00 AM - 1:00 PM	Hospitality Suite *La Marina*
1:00 PM	Meeting adjourns

SCIENTIFIC PROGRAM
AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
1998 LEARNING OBJECTIVES

Jointly Sponsored by The
American Academy of Neurological Surgery
November 4-7, 1998

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 designates this continuing medical education activity for 13.5 credit hours in Category I of the American Medical Association.

SCIENTIFIC PROGRAM
AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
November 5-7, 1998

THURSDAY, NOVEMBER 5, 1998
Moderator: William Chandler, M.D.

8:00-9:40 am	**SYMPOSIUM: New Technology in Neurosurgery**
8:00-8:25 am	Computer Workstations in the Operating Room Gene Barnett
8:25-8:50 am	The Cyber Knife John Adler
8:50-9:15 am	The Interventional Magnet Peter Black
9:15-9:40 am	The Stereotactic Magnet Ralph Dacey

SCIENTIFIC SESSION I

9:40-9:58 am	*Registration of Head CT Images to Physical Space Using a Weighted Combination of Points and Surfaces* **RJ Maciunas, CR Maurer, JM Fitzpatrick**
9:58-10:20 am	**COFFEE BREAK**
10:20-10:38 am	*Is Radiosurgery Surgery?* **L. Dade Lunsford, D. Kondziolka**

THURSDAY, NOVEMBER 5 (Continued)

10:38-10:56 am — *Surgical Outcomes and Minimizing Complications in VNS Stimulation*
P. Mark Li, Doublas Labar, Richard A.R. Fraser

10:56-11:14 am — *Functional Neuronavigation and Intraoperative MRI in Tumors of the Central Area*
R. Fahlbusch, O. Ganslandt, Ch. Nimsky

11:14-11:32 am — *"Morphing" the Spine: Segmentation and Transformation of Preoperatively Acquired Image Data Sets*
K.T. Foley, R.S. Teichman, K.R. Smith

11:32-11:50 am — *Computer Aided Neurovascular Simulation and Phase Contrast MRI in the Management of Ischemic Cerebrovascular Diseases*
Fady T. Charbel, Meide Zhao, Gabriel Gonzales-Portillo, James I. Ausman

11:50 am-12:08 pm — *A Prospective Randomized Double-Blind Controlled Trial to Evaluate the Efficacy of an Analgesic Epidural Paste Following Lumbar Decompressive Surgery*
Volker K.H. Sonntag, Nicholas Theodore, John Hurlbert, Janine B. Drabier, Andrea M. Magwood

THURSDAY, NOVEMBER 5 (Continued)

12:08 -12:26 pm — *Progressive Post-Traumatic Myelomalacic Myelopathy (PPMM): The Syndrome, Surgical Outcome and New Experimental Models*
Barth A. Green, Thomas T. Lee, Robert P. Yezierski

12:26-12:45 pm — *Resident Award Paper*
Nathan R. Selden, M.D.

12:45-1:00 pm — *Resident Runner-Up Paper*
Gerald A. Grant, M.D.

FRIDAY, NOVEMBER 6, 1998

SCIENTIFIC SESSION II
Moderator: Howard Eisenberg

8:00-8:18 am — *State Funding for Neurotrauma Research*
Christopher B. Shields, Henry D. Garretson

8:18-8:36 am — *Retroviral Vector-Mediated Transfer of Antisense Cell Cycle Control Gene aG1 Inhibits Glioma Cell Invasion*
Mark D. Krieger, Frederick L. Hall, Ann Yee, Erlinda M. Gordon, W. French Anderson, Martin H. Weiss

8:36-8:54 am — *Microglia Trafficking in Gliomas: A Novel Delivery System*
Behnam Badie

8:54-9:12 am — *Endothelial Cell Phenotype in Human Cerebral Cavernous Malformations*
Issam A. Awad, Nikolay I. Baev, Gao Zong, Murat Gunel, John Wong

FRIDAY, NOVEMBER 6 (continued)

9:12-9:30 am	*Genetic Pathways in Glioma Genesis* **Mitchel S. Berger, Sandeep Kunwar, Gayatry Mohapatra, Andrew Bollen, Yuriko Minn, Kathleen Lamborn, Michael Prados, Burt Feuerstein**
9:30-9:48 am	*Effect of Adenoviral-Mediated Nitric Oxide Synthase Gene Transfer on Vasospasm After Experimental Subarachnoid Hemorrhage* **R. Loch Macdonald, Bryce Weir, Ghanashayam Ghadge, Chris Weihl, Marcus Stoodley, Lydia Johns, George Lin, Andrew Kowalczuk, Raymond Roos**
9:48-10:06 am	*Gene Therapy for Brain Tumors* **John S. Yu, Ken Samoto, Yun-hui Liu, Keith Black**
10:06-10:36 am	**COFFEE BREAK**
10:36-10:54 am	*Novel Approaches to Treatment of Malignant Gliomas* **Peter Black, Elizabeth Noll, Jon Strasser, Matthias Kirsch**
10:54-11:12 am	*Searching for the Medulloblastoma Gene* **Corey Raffel**
11:12-11:30 am	*$p57^{KIP2}$ — A Novel Tumor Suppressor Gene for Astrocytomas* **James T. Rutka**

FRIDAY, NOVEMBER 6 (continued)

11:30-11:48 am — *The Role of Re-Investigation by Intracranial Electrodes in Patients Failing Initial Invasive Monitoring*
D.W. Roberts, A.M. Siegel, P.D. Williamson, J. McInerney, V.M. Thadani

11:48 am-12:06 pm — *Deep Brain Stimulation for Parkinson's Disease: Comparison of Pallidal Versus Subthalamic Nucleus Targets*
K.J. Burchiel, V.C. Anderson, J. Hammerstad, J. Favre

12:06-12:24 pm — *The Beneficial Effects of Radiosurgery in an Animal Model of Hippocampal Epilepsy*
Douglas Kondziolka, Y. Mori, J. Balzer, K. Thulborn, L.D. Lunsford

12:24-1:00 pm — **Presidential Address: Edward S. Connolly**
Introduced By: Charles Tator

SATURDAY, NOVEMBER 7

SCIENTIFIC SESSION III
Moderator: Joseph Hahn

8:00-8:18 am — *Pediatric Shunt Death in the 1990's*
Bermans J. Iskandar, Shane Tubbs, Timothy B. Mapstone, Paul A. Grabb, Alfred A. Bartolucci, W. Jerry Oakes

8:18-8:36 am — *Evaluation of Outcome and Cost of Craniotomy for Tumor Performed in Regional Academic Referral Centers*
Don M. Long, Toby Gordon, Anthony Etzel, Gregg Burleyson, Simone Betchen, Soosan Shahrokh, Henry Brem

SATURDAY, NOVEMBER 7, 1998 (continued)

8:36-8:54 am	*Incorporation of Skull Base Techniques into a General Intracranial Microsurgical Practice. A Three Year Experience* **Roberto C. Heros**
8:54-9:12 am	*Intraoperative and Perioperative Electrocorticographic Evidence of Functional Reorganization of* the Homunculus **Paul C. Francel, K.J. Oommen**
9:12-9:30 am	*Re-Operation for Residual/Recurrent Aneurysms of the Basilar Apex* **Duke Samson, H.Hunt Batjer, Thomas Kopitnik, Michael Horowitz**
9:30-9:48 am	*Anterior Cervical Discectomies Without Interbody Fusion* **Paul B. Nelson, Jill Donaldson**
9:48-10:06 am	*Craniopharyngiomas: Experience with Complex Approaches in Children* **Michael L. Levy**
10:06-10:36 am	**COFFEE BREAK**
10:36-10:54 am	*Pathophysiological Changes in Cerebral Vessels Due to Hypertension* **J. Marc Simard, Xing Li**
10:54-11:12 am	*Transsphenoidal Surgery for Cushing's Disease in Patients with a Normal Pituitary MRI* **Patrick L. Semple, Edward R. Laws, Jr.**

SATURDAY, NOVEMBER 7, 1998 (continued)

11:12-11:30 am	*Microvascular Failure Following Reperfused Stroke: Experimental Evidence and Implications for Clinical Cerebral Protection* **E. Sander Connolly, Jr.**
11:30-11:48 am	*Carotid Revascularization Using Open Surgery or Endovascular Techniques. Definition of Surgical Risk by Quantifying Medical Comorbidity* **L.R. Guterman, L.N. Hopkins**
11:48 am-12:06 pm	*Carotid Angioplasty and Stenting for Recurrent and Radiation-Induced Stenosis* **Robert H. Rosenwasser, Rocco A. Armonda, Ronald P. Benitez**
12:06-12:24 pm	*Moyamoya Disease in Adults: Clinical Characteristics and Outcome After Encephaloduroarteriosynangiosis (EDAS)* **Dae Hee Han, Tae Ho Kim, Young Seob Chung**
12:24-12:42 pm	*Neurological Manifestations of Cowden Disease: Characterization of a New Phakomatosis* **Alan R. Cohen, Shenandoah Robinson**
12:42-1:00 pm	*Leukocyte-Endothelial Adherence in Penumbral Cortex 24 Hours After Permanent Middle Cerebral Artery Occlusion in Mice* **T. S. Park, Jeffrey M. Gidday, Stuart S. Kaplan**
1:00 pm	**ADJOURN**

Thursday, November 5
9:40-9:58 AM

Registration of Head CT Images to Physical Space Using a Weighted Combination of Points and Surfaces

RJ Maciunas, CR Maurer, JM Fitzpatrick

Most previously reported registration techniques that align three-dimensional image volumes by matching geometrical features such as points or surfaces use a single type of feature. We recently reported a hybrid registration technique that uses a weighted combination of multiple geometrical feature shapes. In this study we use the weighted geometrical feature (WGF) algorithm to register CT images of the head to physical space using the skin surface only, the bone surface only, and various weighted combinations of these surfaces and one fiducial point (centroid of a bone implanted marker). We use data acquired from twelve patients that underwent temporal lobe craniotomies for the resection of cerebral lesions. We evaluate and compare the accuracy of the registrations obtained using these various approaches by using as a reference gold standard the registration obtained using three bone-implanted markers. The results demonstrate that a combination of geometrical features can improve the accuracy of CT-to-physical space registration. Point-based registration requires a minimum of three non-colinear points. The position of a bone-implanted marker can be determined much more accurately than that of a skin-affixed marker or an anatomic landmark. A major disadvantage of using bone-implanted markers is that an invasive procedure is required to implant each marker. By combining surface information, the WGF algorithm allows registration to be performed using only one or two such markers. One important finding is that the use of a single very accurate point (a bone-implanted marker) allows very accurate surface-based registration to be achieved using very few surface points. Finally, the WGF algorithm, which not only allows the combination of multiple types of geometrical information but also handles point-based and surface-based registration as degenerate cases, could form the foundation of a "flexible" surgical navigation system that allows the surgeon to use what he considers the method most appropriate for an individual clinical situation.

NOTES

Thursday, November 5
9:58-10:38 AM

Is Radiosurgery Surgery?

L. Dade Lunsford, M.D., FACS,
D. Kondziolka, M.D., FACS, FRCS

Background: Controversy surrounds the incorporation of minimally invasive techniques into more traditional neurosurgery management strategies. An analysis of the effects of a radiosurgical project into an academic health center was performed.

Methods & Materials: A retrospective review of the entire practice of gamma knife radiosurgery during 11 years was conducted to assess growth patterns, roles, and the impact of diversion of more traditional neurosurgery to radiosurgery management.

Results: Radiosurgery (n=2674) grew from 7% to 17% of all annual neurosurgery volume. Case volume grew from 187 in year one to 487 in year 11. The neurosurgeon's responsibilities remained stable and included pre- and post -operative patient management, intraoperative stereotactic frame placement and imaging, computer dose planning, and delivery of the effective dose. Appropriate responsibilities were performed in consultation with radiation oncologists and medical physicists. Radiosurgery was incorporated into the academic teaching mission with formal fellowships and designated resident rotations.

Discussion: Radiosurgery is a subfield of contemporary neurosurgery. It requires special surgical training and relies on non-traditional skills that include computer proficiency, three dimensional volumetric thinking, and precision manual skills enhanced by stereotactic technology. An increasing number of cases (e.g. metastatic tumors, trigeminal neuralgia) were diverted from other neurosurgery techniques to radiosurgery. Failure of the neurosurgical community as a whole to embrace the role of radiosurgery has profound adverse effects pertaining to the future role and training of neurosurgeons. Minimally invasive strategies in general appear to be the future of a large segment of neurosurgical practice. These techniques must enter the mainstream in order to assure the future of our field.

NOTES

Thursday, November 5
10:38-10:56 AM

Surgical Outcomes and Minimizing Complications in VNS Stimulation

P. Mark Li, M.D., Ph.D., Douglas Labar, M.D., Ph.D., & Richard A.R. Fraser, M.D.
The New York & Presbyterian Hospitals, 525 E. 68th Street, Starr 651, New York, NY 10021

The combined experience with vagal nerve stimulation during the E01-E05 multicenter clinical trials show that VNS can be an effective adjunctive therapy for the intractable epilepsies. The results of these clinical trials (published elsewhere) show that VNS decreases seizure frequency 50% or more in approximately 30% of patients. As many as 66% of patients have some improvement in seizure frequency. Furthermore, careful study of cardiac, gastric, pulmonary, and vocal cord function shows long term VNS to be a safe therapeutic option. To date there have been over 2500 VNS devices implanted worldwide, with a burgeoning experience in both efficacy and complication rates. We will present the neurological and surgical outcomes from the multicenter clinical trials along with our personal series of VNS stimulators.

The combined surgical experience with vagal nerve stimulation (VNS) includes 454 patients implanted during the E01-E05 clinical studies. In this group of patients, the most common observed complication was infection of either the generator site or lead implantation site (2.86%). Of the patients who presented with evidence of infection, roughly half (1.7% of total) were treated successfully with antibiotic therapy, whereas roughly half (1.10% of total) required antibiotic therapy and removal of the device. The other common complications include hoarseness or temporary vocal cord paralysis in 0.7% of patients, and lower facial hypesthesia or paralysis in 0.7% of patients presumably due to injury of the inferior laryngeal nerve(s) or the mandibular division of the facial nerve.

The authors's personal surgical series now totals 88 patients implanted, 34 in the last academic year. The outcome of the New York hospital series of VNS patients correlates with the results of the previous multicenter trials. In our series 34% of patients had a 50% or greater seizure reduction. In addition, we report a small series of children in whom this procedure was performed for Lennox-Gastaut syndrome. Although the patient numbers are small, the dramatic effect of VNS on some of these patients suggests that VNS may be a method of treating these patients.

In summary, VNS is a novel treatment for intractable epilepsy. Although the specific mechanisms of this therapy remain unclear, the clinical results of VNS for intractable epilepsy suggest that it can be a good adjunctive therapy. We therefore feel that careful surgical technique, combined with a short surgical duration can minimize complications and make VNS implantation a safe and relatively simple procedure.

NOTES

Thursday, November 5
10:56-11:14 AM

Functional Neuronavigation and Intraoperative MRI in Tumors of the Central Area

R. Fahlbusch, O. Ganslandt, Ch. Nimsky
Department of Neurosurgery, University Erlangen-Nuremberg, Schwabachanlage 6, D-91054 Erlangen, Germany

Patients with tumors in the pre- and postcentral area (Gyrus prae- and postcentralis) offer special problems for indication of surgery and its radicality. Within the Erlangen concept of image-guided surgery functional neuronavigation and intraoperative resection control are available in order to achieve improved conditions.

Patients and Methods: We have operated 46 patients with tumors in the central area among 224 patients operated in our 'twin-OR' since the end of 1995. Our concept is based on an installation of a MR scanner (0.2 Tesla Magnetom Open, Siemens AG, Germany) in combination with two neuronavigation systems (MKM, Zeiss, Germany and Stealth, Sofamor Danek, USA), furthermore functional data such as magnetoencephalography (MEG) (74-channel biomagnetometer MAGNES II, BTI, USA) are integrated into the image data for navigation. The best dipole fit of somatosensory or motor evoked fields were matched to the MR images leading to functional neuronavigation. For intraoperative imaging a 3D-flash MR-sequence (slab 168mm, slice 1.5mm, TR 16ms, TE 7 ms, FOV 250mm) was used, allowing image data update for neuronavigation.

Results: In all 46 patients with lesions adjacent to the motor cortex MEG was confirmed by intraoperative electrocorticography. In 32 patients a macroscopic total tumor resection could be accomplished, in 14 cases only biopsy, or subtotal resection was performed, due to the infiltration of the motor cortex. 6 patients experienced temporary neurological deterioration.

Intraoperative MRI for resection control was performed in a total of 37 glioma patients. In 8 of the 19 low grade gliomas intraoperative resection control showed significant tumor remnants, so in a second look tumor resection was completed in 6 of them allowing complete tumor removal, guided by an update of neuronavigation.

Conclusion: We conclude that the combination of functional imaging and neuronavigation is a helpful technique for surgery around functional important brain areas. Our preliminary experience suggests that intraoperative MRI is a convenient tool to evaluate the extent of tumor resection intraoperatively, furthermore it provides an update of image data for neuronavigation to compensate the effects of tumor removal and brain shift. Despite the open question whether the long-term patient-outcome is improved by a more radical tumor resection, intraoperative imaging gives a chance for more radical resections with less complications.

NOTES

Thursday, November 5
11:14-11:32 AM

"Morphing" the Spine: Segmentation and Transformation of Preoperatively Acquired Image Data Sets

K.T. Foley, R.S. Teichman, K.R. Smith

Purpose: The purpose of this study was to investigate the feasibility of updating a three-dimensional image data set to reflect intersegmental spinal motion occurring between the time of image acquisition and surgery.

Methods: Atlantoaxial CT scans were obtained in 5 patients using 1 mm slice thickness and 14 cm FOV. The scan data were then segmented into the individual C1 and C2 vertebrae in an automated fashion through mapping from a digitized, annotated cervical spine atlas. The atlas data set was deformed utilizing a proprietary algorithm that has previously been used for brain mapping (IntellX), resulting in the automated generation of labeled segmentations and 3D surface models of the patient's vertebrae. The C1 and C2 segments were reduced and immobilized using sublaminar cables, then individually registered utilizing the StealthStation. Based upon the segmentation of the preoperative data set as well as the segmental registration data from surgical space, the image space data set was transformed.

Results: The transformed image space data set display rendered C1 and C2 in their "new" relative positions, reflecting their relationship in surgical space, post-reduction.

Optimal transarticular screw pathways could then be plotted and tracked in real-time through both C1 and C2.

Conclusions: We have shown that a segmented spinal image data set can be transformed based upon intraoperative registration, allowing computerized display of the "new" spatial relationships of the vertebrae at the time of surgery. We conclude that it is feasible to update a preoperatively acquired spinal image data set to reflect changes in intervertebral spatial relationships brought about by patient positioning and/or surgical manipulation.

NOTES

Thursday, November 5
11:32-11:50 AM

Computer Aided Neurovascular Simulation and Phase Contrast MRI in the Management of Ischemic Cerebrovascular Diseases

Fady T. Charbel, MD, Meide Zhao, Ph.D.,
Gabriel Gonzales-Portillo, MD, James I Ausman, MD, Ph.D.
Department of Neurosurgery, University of Illinois at Chicago, IL

Introduction: The indications for either surgery, endovascular treatment, or medical treatment in patients with ischemic cerebrovascular disease (viz. carotid artery disease, subclavian steal syndrome, intracraneal stenosis) continue to be controversial. The use of the Computer Aided NeuroVascular Simulation and the Phase Contrast MR has allowed us to determine the blood flow in the cerebral vasculature and to model different treatment options.

Methods and Results: A computer simulation model was developed which creates a virtual replica of the circle of Willis , the intracranial and the extracranial vessels. The model has been validated in the human. Flow and pressure can be determined in normal, stenotic vessels, grafts, and cerebral aneurysms using PCMR data. With this data an animated qualitative and quantitative flow profile is developed illustrating the hemodynamics in each vessel of interest in real time. Information about the magnitude and direction of flow in the cerebral vasculature is obtained from these animated flow profiles. The computer simulation model and PCMR has been used in over 40 patients. We will present an animated version of two representative cases and discuss the role of these in the outcome of our patient series

Conclusions: The computer simulation model software can predict the flow in the vessels of the patients with cerebrovascular diseases. This information plus the quantitative evaluation of the hemodynamic changes using the PCMR is key in the management of ischemia.

NOTES

Thursday, November 5
11:50 AM - 12:08 PM

A Prospective Randomized Double-Blind Controlled Trial to Evaluate the Efficacy of an Analgesic Epidural Paste Following Lumbar Decompressive Surgery

Volker K. H. Sonntag, MD, Nicholas Theodore, MD, R. John Hurlbert, MD, Ph.D., Janine B. Drabier, RN, and Andrea M. Magwood, RN.

Introduction: A prospective, randomized, double-blind study was performed to evaluate the efficacy of a morphine-based analgesic "paste" in the control of pain after lumbar surgery.

Methods: Sixty patients undergoing elective lumbar decompressive surgery with a single surgeon were randomized to either an active or placebo group. The paste was applied to the exposed dura just before wound closure. Patients were given postoperative analgesics as requested. All patients were followed for 3 months after surgery. Primary outcome measures included the McGill Pain Questionnaire and analgesic use. Secondary outcome measures included vital signs, length of stay, physician assessments, and patient-reported health (SF-36 and Aberdeen Back Pain Index).

Results: There were no significant postoperative differences between groups with respect to demographic data, neurological deficits, or perceived disability. Patients receiving the active paste had a significantly lower consumption of narcotics in the hospital (p=0.007) and at home (p=0.044). They also had significantly better pain control immediately postoperatively and significantly better general health perception (p=0.015). These beneficial effects lasted as long as 6 weeks postoperatively. No adverse effects were attributable to the paste.

Conclusions: Administration of a morphine-based analgesic paste directly to the epidural space during lumbar decompressive surgery significantly improves postoperative pain control, reduces prescribed analgesic consumption, and improves overall perception of health for as long as 6 weeks after surgery. This postoperative pain control strategy may provide a new gold standard for pain management. Long-term follow up is ongoing.

NOTES

Thursday, November 5
12:08-12:26 PM

Progressive Post-traumatic Myelomalacic Myelopathy (PPMM); the Syndrome, Surgical Outcome and New Experimental Models.

Barth A. Green, MD, Thomas T. Lee, MD and Robert P. Yezierski, PhD
Department of Neurological Surgery and The Miami Project
University of Miami School of Medicine, Miami, FL 33136

Progressive post-traumatic myelomalacic myelopathy (PPMM) and progressive post-traumatic cystic myelopathy (PPCM) can occur in patients after spinal cord injury, spinal or spinal cord surgery, or other causes of arachnoiditis. All of these cases have in common an alteration of CSF flow dynamics, either from a tethered cord with scar tissue or extrinsically induced blockage from soft tissue and/or bony canal compression. Patients have been reported to become symptomatic anywhere from two months to 37 years after injury. Magnetic resonance imaging (MRI) especially with CINE' software, has made the diagnosis, monitoring and treatment evaluation non-invasive and increasingly accurate. Frustrations associated with surgical failures have encouraged these authors to develop treatment protocols including untethering, cord covering and expansile duraplasty.

New experimental models have been developed to better understand the pathophysiology and optimize treatment. One experimental model of spinal cord tethering and syrinx formation is created with intrathecal injection of kaolin which causes intense arachnoiditis, cord tethering and progressive cyst formation. Injection of the AMPA receptor agonist quisqualic acid into the grey matter of the rat spinal cord also causes syrinx formation. The post-injury inflammatory changes and other secondary injury cascades including those resulting from elevated levels of excitatory amino acids lead to the pathognomonic microscopic and macroscopic changes seen in a traumatically injured spinal cord. These authors believe that the macrocystic or large confluent cyst (which may be singular or multiloculated and complex) is simply a progression of what, in most cases, starts out to be myelomalacic or microcystic changes.

Our clinical experience includes 41 patients with PPMM who underwent untethering of the spinal cord and nerve roots with an expansile duraplasty. Lysis of dorsal and lateral adhesions of the spinal cord and nerve roots were performed. Intra-operative ultrasonography was used to detect the presence of residual cord deformation or cysts, thereby assessing the success of untethering. The mean postoperative follow up was 35 months for the 37 patients available for analysis with the range being 15 to 57 months.

Trauma was the most common etiology, accounting for 31 out of 41 patients. The interval between the causative event and surgical treatment was from seven months to 37 years in this group. Motor function deterioration was the most frequent presenting symptom occurring in 31 patients. Improvements in more than 60% of the patients were noted in motor function, autonomic dysreflexia and pain on the most recent follow up. Two patients experienced retethering of the spinal cord and one underwent repeat operation. Details relating to the diagnosis, surgical selection perioperative protocol and morbidity will be presented as well as the experimental models developed to further our understanding of the pathophysiology.

NOTES

Friday, November 6
8:00-8:18 AM

State Funding For Neurotrauma Research

Christopher B. Shields, Henry D. Garretson
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The success of funding neuroscience research grants by the NIH, NSF, and other federal agencies has diminished over the past several years. Local funding has also been curtailed as a result of alterations in reimbursement patterns to hospitals and neurosurgical practices caused by managed care. These changes raise concern that advances in neurotrauma research will be severely curtailed in the future. Another source of innovative funding that may be possible at the state level should be considered.

The Kentucky Head and Spinal Cord Injury Trust (KSCHIRT) serves as a model for other states to emulate. In 1994, legislation was passed under the guidance of Senator Tim Shaughnessy whereby a $12.50 surcharge is added to the fine of all motorists convicted of speeding. Such fines are morally justified as they penalize the very individuals that are often involved in major neurotrauma. This legislation has raised between $2-2.5 million per year in Kentucky, which is distributed on a competitive basis to research scientists at the two state universities. Over the past three years there has been over $3,500,000 distributed to fund 41 research projects. Over the past two years there have been four additional grants of $500,000 each to provide seed money to fund endowed research chairs. To date there have been 3 endowed research chairs named in Kentucky by this mechanism. In addition, KSHIRT has helped fund three annual research meetings in Kentucky.

The goals of KSCHIRT have been to 1) further basic research towards finding a cure for spinal cord and head injuries, 2) recruit nationally prominent research scientists to Kentucky, and 3) provide seed money for important research projects which will lead to federal funding in neurotrauma. The program in Kentucky has been followed in several other states in the United States, particularly in Florida and Virginia. The legislative mechanisms which were followed to pass this legislation will be discussed. All neurosurgeons, because of their intimate involvement in both the treatment and the tragedy of neurotrauma, should participate in efforts to enact similar legislation in every state. If all states could raise the same amount as in Kentucky, an additional $100 million would be available for neurotrauma research in the United States.

NOTES

Friday, November 6
8:18-8:36 AM

Retroviral Vector-Mediated Transfer of Antisense Cell Cycle Control Gene aG1 Inhibits Glioma Cell Invasion

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Rapid cell cycle progression and malignant tissue invasion are indicators of the malignancy of a tumor cell line. Unfortunately, the mechanisms which underly these malignant behaviors are poorly understood. The cyclins and their dependent kinases have attracted attention in this regard as an inducible class of proteins which are present at critical cell cycle phase transitions. Cyclin G1, a member of this class, is upregulated during the G1 to S phase transition. Previous in vitro and in vivo studies have shown a growth inhibitory effect of retroviral vector-mediated transfer of an antisense gene to cyclin G1on a malignant glioma cell line. Additionally, this decreased growth rate was associated with increased production of GFAP, an indicator of malignancy. The present study evaluates the role of cyclin G1 in regulating invasion of an aggressive glioma cell line by blocking expression of cyclin G1 with an oligonucleotide in an antisense orientation to cyclin G1 (aG1).

Invasion chamber analysis was performed using Boyden chambers and Matrigel substrate. 9L glioblastoma cells were plated in triplicate at a density of 1000 cells/mm3. 9L cells transduced with viral supernatant containing the aG1 gene or a control gene were also plated in triplicate. After a 12 hour period of migration, with dextrose-enriched medium used as a chemoattractant, the aG1 transduced cells migrated at 10% of the rate of the other 2 cell types (21 vs 170 cells/mm3, $p<.001$).

These studies demonstrate a significant role for cyclin G1 in tumor progression, growth and invasion in a malignant glioma cell line.

NOTES

Friday, November 6
8:36-8:54 AM

Microglia Trafficking in Gliomas: A Novel Delivery System?

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Objective: Although gene therapy is currently being evaluated as a possible treatment approach for malignant gliomas (MG), preliminary studies using herpes simplex thymidine kinase gene delivery have indicated a lack of response in large tumors. One explanation for this unexpected result is poor diffusion of virus-producing cells (fibroblasts) within invasive gliomas. As immune effector cells of the CNS, microglia, on the other hand, are capable of rapid migration within the CNS. Because microglia represent a major component of the inflammatory cells in MG, we hypothesized that their trafficking in these tumors is more efficient than fibroblasts. In order to evaluate microglia as a possible delivery system for gliomas, and to determine the chemokines involved, we studied microglia migration in glioma models.

Methods: *In vivo* migration of BV-2 murine microglia cells was examined by injecting fluorescently labeled (PKH-26) BV-2 and NIH 3T3 fibroblasts (as control) into GL261 tumor-bearing mice. Animal brains were sectioned serially, and the extent of BV-2 and 3T3 diffusion was evaluated. *In vitro*, the effect of glioma cells in augmenting BV-2 motility was studied using a Boyden chamber migration assay. The role of hepatocyte growth factor/scatter factor (HGF/SF) and monocyte chemotactic protein-1 (MCP-1) on microglia migration was evaluated by using neutralizing monoclonal antibodies. Immunoblotting was used to test the expression of HGF/SF by glioma cells, while the expression of its receptor, cMet, by BV-2 cells was examined by flow cytometry.

Results: Intratumoral injections of BV-2 cells led to diffusion of these cells throughout the gliomas within 2 days. 3T3 fibroblasts, on the other hand, remained at the site of the injections and did not migrate even after 1 week. *In vitro*, BV-2 migration was noted within 7 hrs of incubation with both human (U251MG, U373MG, U87MG) and murine (GL261) glioma cell lines, but not with MCF-7 breast carcinoma cells. This migration corresponded to HGF/SF secretion by glioma cells and was completely inhibited by neutralizing mAb against HGF/SF, but not MCP-1. Exposure of BV-2 cells to recombinant HGF/SF, but not MCP-1, resulted in their migration and down-regulation of cMet in a dose-dependent fashion.

Conclusion: Microglia are more efficient than fibroblasts in trafficking within gliomas. This active microglia infiltration into gliomas may be mediated through release of HGF/SF, a known glioma mitogen and motility factor. Considering their role as CNS immune effector cells and their trafficking within gliomas, microglia may be useful in development of future glioma therapies.

NOTES

Friday, November 6
8:54-9:12 AM

Endothelial Cell Phenotype in Human Cerebral Cavernous Malformations

Issam A. Awad, M.D., M.Sc., F.A.C.S., Nikolay I. Baev, M.D., Ph.D., Gao Zong, M.D., Ph.D., Murat Gunel, M.D., John Wong, M.D., F.R.C.S.(C)

Background: The cerebral cavernous malformation (CCM) consists of blood filled caverns lined by endothelial cells (EC) and lacking mature vessel elements. Errors in vascular genesis or proliferation, both EC related, are postulated to play a role in this disease. Yet, EC from CCM have not been successfully isolated previously and the EC phenotype in CCMs has not been carefully analyzed.

Methods. We describe in situ analysis of EC phenotype is excised CCM lesions including immunohistochemical expression of a panel of angiogenesis receptors and EC specific antigens, and transmission electron microscopy (EM) of EC morphology and inter-EC junctions. We report for the first time successful isolation, culture and phenotypic characterization of EC from CCM lesions.

Results: The CCM EC layer expresses in situ the EC specific vWF antigen, eNOS, and angiogenesis receptors Flk1, Flt1, TGF-ß RI and RII, and Tie. On EM, the CCM EC exhibits increased cytoplasmic vesicles suggestive of trans-EC transport and abnormal inter-EC junctions. CCM EC lines were established after the excised specimens with 0.3% trypsin-1% EDTA, selective cloning, and growth in MCDB107, 0.3 g/l heparin, 0.15 g/l ECGS, and 15% FBS. Those lines consisted of a morphologically heterogeneous population of cells larger than brain and skin EC. CCM EC showed contact inhibition and a rounded cobble-stone appearance, and expressed CD31, CD105, vWF, and binding sites for UEA-1 and Dil-Ac-LDL. They showed low levels of Flt-1, Flk-1, TGF-ß RII expression, but stained strongly with antibodies against Tie-1 and Tie-2. CCM EC did not express smooth muscle actin or GFAP.

Conclusion: This information is essential for phenotype-genotype correlations in this disease. Cultured EC will assist in further identification of CCM genes, possible somatic mutations or loss of heterozygocity in CCM lesions, and in vitro assays of EC proliferation, inter EC junctions and tube formation, which may contribute to lesion genesis and progression.

NOTES

Friday, November 6
9:12-9:30 AM

Genetic Pathways in Glioma Genesis

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Although current diagnoses of gliomas are based primarily on histopathology which correlates with prognosis, clinical outcome within a given histology varies greatly and pathologists often disagree in the classification of individual tumors. Recently, activation of oncogenes and inactivation of tumor suppressor genes have been linked to the behavior of glial tumors. Thus, genetic markers may predict clinical outcome, supplement current histologic classification and define molecular pathways that lead to the development of gliomas. We analyzed 175 primary gliomas: 24 oligodendrogliomas, 43 oligoastrocytomas (OA), 36 anaplastic astrocytomas (AA), and 72 glioblastoma multiforme (GBM), using comparative genomic hybridization (CGH), a technique which scans the entire tumor genome for DNA copy number abnormalities (CNA). Tumors with oligodendroglial components had aberrations involving loss of chromosome 1p and 19q (>50% of oligodendrogliomas and >35% of OAs). Among these tumors, -18q occurred specifically in OAs but not in oligodendrogliomas, while -14q was present in both tumors. -9p, +10p, and -19q comprised a second subset of OAs. Similarly, there were specific aberrations in AAs not present in GBMs (-4q, +8q, +10p), whereas a set of aberrations were noted in both of these malignant gliomas (+19p, +7p, +7q, -10). We correlated CNAs that occurred in >10% of tumors with age, sex, time to progression and survival within diagnostic subtypes. Aberrations correlated with both age and survival in each histologic group. Among OAs, -14q correlated with a longer progression free survival (PFS) ($p<0.05$) and +10p had shorter PFS ($p<0.05$). In AAs, aberrations infrequent among higher grade GBMs were associated with longer survival (-4q, $p<0.05$) while aberrations common to both tumors had shorter PFS and survival (+7p, +7q, $p<0.01$). +19p among AAs were associated with shorter survival, whereas among GBMs, +19p was associated with longer survival, suggesting these GBMs are "secondary" tumors, arising from lower grade lesions. We present a schema for genetic pathways involved in glioma genesis based on tumor specific aberrations and their relationships to clinical parameters. These results indicate that cytogenetic markers will prove useful in predicting clinical behavior and provide an objective genetic basis for diagnosis. Supported by CA13525, CA61147, CA64898.

NOTES

Friday, November 6
9:30-9:48 AM

Effect Of Adenoviral-Mediated Nitric Oxide Synthase Gene Transfer On Vasospasm After Experimental Subarachnoid Hemorrhage

R. Loch Macdonald, Bryce Weir, Ghanashayam Ghadge, Chris Weihl, Marcus Stoodley, Lydia Johns, George Lin, Andrew Kowalczuk, Raymond Roos, Departments of Surgery and Neurology, University of Chicago, Chicago, Illinois, USA

Vasospasm is vasoconstriction of the cerebral arteries that occurs after subarachnoid hemorrhage (SAH) and that contributes to morbidity and mortality in this condition. This study tested whether intracisternal injection of replication-defective adenovirus expressing endothelial nitric oxide synthase (eNOS) would decrease vasospasm in a dog model. 16 dogs underwent baseline cerebral angiography and then SAH by 2 injections of blood into the cisterna magna. They were randomized (n=8 per group) to intracisternal injection of adenovirus expressing lacZ or eNOS given immediately after the first blood injection. Angiography was repeated on day 7. L-arginine (50 mg) then was given intracisternally and angiography was repeated. Dogs were euthanized and the basilar arteries were removed and studied pharmacologically. The brains were analyzed for transgene expression by staining with 5-bromo-4-chloro-3-indolyl-ß-galactopyranoside for ß-galactosidase activity and by reverse-transcriptase polymerase chain reaction for eNOS messenger ribonucleic acid. Angiography showed significant vasospasm in each group (lacZ: 54 ± 16% reduction in basilar artery diameter, eNOS: 53 ± 16%) with no significant difference between groups. Injection of virus into dogs (n = 2 per group) without SAH showed that viral injection itself caused some arterial narrowing (13 ± 14% [lacZ] and 14 ± 51% [eNOS] reductions in basilar artery diameter). Injection of L-arginine caused an insignificant increase in arterial diameter in each group. Histologic examination and ß-galactosidase staining of dogs injected with lacZ virus showed staining in inflammatory cells in the subarachnoid space and in the adventitia of the cerebral vessels and in the liver and lungs. Under isometric tension, basilar arteries from each group showed minimal relaxation to L-arginine but arteries exposed to eNOS showed significantly greater relaxation to L-arginine plus tetrahydrobiopterin compared to arteries exposed to lacZ. These results show that adenoviral vectors can be used to transfer genes to cells in the subarachnoid space of dogs, that they cause inflammation that is associated with arterial narrowing and that NO plus cofactors can dilate arteries *in vitro* but enough NO was not generated in the subarachnoid space to prevent vasospasm.

NOTES

Friday, November 6
9:48-10:06 AM

Gene Therapy for Brain Tumors

John S. Yu, Ken Samoto, Yun-hui Liu, Keith Black

The excitement engendered by gene therapy is tempered by the clinician's observation of the perilous complexity of the human nervous system. The sanctum of the brain within the skull and blood-brain barrier hinders access to the central nervous system for gene transfer. The goal of gene therapy for brain tumors is to eradicate all neoplastic cells while sparing the normal surrounding brain tissue. Gene therapy for brain tumors is not a single therapeutic strategy, but consists of several approaches using a variety of vectors and therapeutic genes from its armamentarium. Gene therapy approaches to be discussed include selective prodrug activating systems, replication-conditional viral cytolysis, immunomodulation, tumor suppressor genes, and apoptosis induction. Furthermore, we will discuss the benefits and limitations of different viral vector systems and means of delivery including blood-brain barrier disruption. These preclinical data have provided a rationale for the development of a clinical gene therapy program.

NOTES

Friday, November 6
10:36-10:54 AM

Novel Approaches to Treatment of Malignant Gliomas

Peter Black, Elizabeth Noll, Jon Strasser, Matthias Kirsch

The management of malignant gliomas remains unsatisfactory. In our laboratory we are exploring two novel methods of treating these lesions.

The first is anti-angiogenic therapy, using angiostatin, a fragment of plasminogen. This agent has been shown to block new vessel formation in solid tumors. In a series of experiments in rats, we have demonstrated that angiostatin inhibits tumor growth of C6, 9L, and U87 cells implanted intracranially in nude mice. In these experiments intraperitoneal administration of angiostatin was able to effectively limit glioma growth significantly.

The second approach we are exploring makes use of a naturally occurring apoptosing agent. This molecule, called GM3, is ganglioside that acts during embryonic development to limit cell number in mitotically active cells. Using an in vitro assay, we have demonstrated that GM3 can selectively induce apoptosis in a variety of glioblastomas.

Using a murine model and the rat 9L gliosarcoma cell line, we were able to demonstrate that intracranial application of GM3 was able to significantly increase symptom free survival. These are examples of novel and potentially important treatments.

NOTES

Friday, November 6
10:54-11:12 AM

Searching for the Medulloblastoma Gene

Justin S. Smith, Issei Imoto, Cory Brown, Hyun K. Lee, Robert B. Jenkins, and Corey Raffel

Medulloblastoma is the most common malignant pediatric brain tumor. The most common cytogenetic abnormality in this tumor is an iso 17q, in which one short arm is lost and a long arm duplicated in one chromosome 17. This abnormality is found in about 40% of tumors. The molecular genetic equivalent of this cytogenetic abnormality, loss of heterozygosity for 17p, has been documented to occur in a similar percentage of tumors. Much rarer is the loss of a distal marker on 17013.3, but no other 17p marker. In one case reported by our laboratory, one entire 17p was deleted, while a rearrangement of the remaining short arm was detected in 17p13.3. We reported that this rearrangement consisted of a 9 kb deletion and identified markers on both sides of the deletion. These probes have now been used to isolate a BAC that contains both markers and the intervening genomic DNA. We have used the technique of a hybrid selection to look for genes contained within this BAC. Screening of 120 clones from the initial selection has resulted in the identification of 3 clones containing poly-A tails and 11 contiguous open reading frames containing 2 or more overlapping clones (contigs). The sequences of these contigs have been used to search the BLAST database; all of the open reading frames appear to encode potential genes that have not been previously identified. These coding regions are now being examined for their potential as the medulloblastoma tumor suppressor gene located on chromosome 17p.

NOTES

Friday, November 6
11:12-11:30 AM

p57^{KIP2} — A Novel Tumor Suppressor Gene for Astrocytomas

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Division of Neurosurgery, The Hospital for Sick Children,
The University of Toronto

Astrocytic tumors frequently exhibit defects in the expression or activity of proteins which control cell cycle progression. Inhibition of kinase activity associated with cyclin/cyclin-dependent effects of growth signals are down-regulated. These are two main families of CDKIs; INK4 and CIP/KIP. Recently, a new p57^{KIP1}-like CDKI, p57^{KIP2}, was isolated and cloned. The human gene for p57^{KIP2} is situated on chromosome 11p15.5 in close proximity to the IGF@ gene. We demonstrate here that the inducible expression of p57^{KIP2} in the astrocytoma cells using the tetracycline repressor system potently blocks the proliferation of cells in G_1 as determined by growth curve and flow cytometric analyses. To determine if the induction of p57^{KIP2} was associated with alterations in the expression of downstream cell cycle regulatory proteins, we also performed western blot analyses of pRB/E2F family proteins. The expression levels of pRB and p107 were sharply reduced with a shift to hypophosphorylated forms following p57^{KIP2} induction, while p130 expression levels increased. E2F-1 was also repressed while E2F-4 levels were unchanged. Interestingly, the induction of p57^{KIP2} in the p53 mutant U373 astrocytoma cell line led to an apoptotic cell death in a high percentage of cells. Our data demonstrate for the first time that the proliferative block imposed by p57^{KIP2} on these malignant astrocytoma cells results in changes in the expression of a number of cell cycle regulatory factors and in cell morphology. Our results further implicate stimulation of a p53-independent apoptotic pathway among p57^{KIP2}-induced astrocytoma cells.

NOTES

Friday, November 6
11:30-11:48 AM

The Role of Re-Investigation by Intracranial Electrodes in Patients Failing Initial Invasive Monitoring

D.W. Roberts, A.M. Siegel, P.D. Williamson, J. McInerney, V.M. Thadani

Objective: Intracranial electrode recording often provides localization of the site of seizure onset sufficient to enable epilepsy surgery. In patients in whom evaluation fails to localize seizure origin, the utility of further invasive monitoring is unknown. This study was undertaken to explore the hypothesis that a second intracranial investigation warrants consideration and can lead to successful epilepsy surgery.

Material and Methods: A series of 100 consecutive patients suffering from partial epilepsy who had undergone intracranial evaluation (by strip-, grid-, and depth electrodes) between 1992 and 1998 was retrospectively analyzed. Seventy-nine of these patients were directly operated upon. Nine of the 21 remaining patients underwent re-investigation.

Results: the nine patients underwent initial implantation at a mean age of 26.9 years and re-evaluation was performed a mean of four months later. In seven patients satisfactory seizure onset localization was achieved and re-evaluation resulted in epilepsy surgery (mean age at operation 27.5 years). Three frontal, two temporal, and one occipital resection as well as one multiple subpial transection were performed. Four patients have become seizure-free (Class I seizure outcome by Engel's classification), one now has rare seizures (Class II), and two were not significantly improved (Class III and IV), with a mean follow-up of 1.5 years. There was no permanent morbidity.

Conclusions: In selected patients in whom invasive monitoring fails to identify the site of seizure origin, re-investigation by intracranial electrodes can successfully achieve satisfactory localization and enable worthwhile surgical treatment.

NOTES

Friday, November 6
11:48 AM - 12:06 PM

Deep Brain Stimulation for Parkinson's Disease: Comparison of Pallidal versus Subthalamic Nucleus Targets

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Parkinson's disease is typically a condition which produces a bilateral movement disorder. Bilateral pallidotomy is effective in relieving "off" symptoms (bradykinesia, rigidity, tremor, walking dysfunction, imbalance, akinesia, dystonia) and "on" symptoms (dyskinesias). However, bilateral pallidotomy also has an unacceptably high rate of hypophonia, dysarthria, dysphagia, drooling, writing problems, and occasionally, severe cognitive dysfunction. The clinical dilemma is how to choose a surgical procedure in medically intractable cases of Parkinson's disease.

We describe results of a prospective, randomized, double-blind pilot study of Deep Brain Stimulation (DBS) of globus pallidus internus (GPi) and subthalamic nucleus (STN) for the bradykinesia, rigidity and associated motor disturbances of idiopathic Parkinson's disease (PD). Ten subjects with advanced PD (56± 13 years old; 3 female), off-levodopa Hoehn and Yahr stage ≥3 were enrolled. A majority of the subjects suffered from dose-limiting dyskinesia/dystonia. Pre-operative neurological assessment included Unified Parkinson's Disease Rating Scale (UPDRS) on and off levodopa and dyskinesia rating. Neuropsychological function was assessed using a variety of standard instruments. Response to DBS was assessed at 10 days, 3, 6 and 12 months after implant. Complete 12 month data are available for 7 subjects.

UPDRS scores were improved by both GPi and STN stimulation immediately after implant and remained improved for at least 12 months (UPDRS on-levodopa 54.9±24.2 [baseline] → 33.6±12.2 [12 mo]; off-levodopa 95.8±29.5 [baseline] → 56.6±19.5 [12 mo]). Subjects in both groups sowed reduced dyskinesia after 12 months of stimulation. Mean levodopa dose was not changed by stimulation at either site. Although subjects numbers are small, the data suggest that some symptoms respond more to stimulation at GPi or STN, dependent on levodopa phase: (1) Motor UPDRS scores were more improved over baseline by GPi than STN stimulation, with differences being more pronounced while a levodopa (31%{GPi} vs. 7%{STN}); (2) Gait was also more improved by GPi stimulation on levodopa (35% {STN} vs 78% {GPi}), with little site-dependent difference when levodopa was off; (3) Bradykinesia, rigidity and tremor were more improved by STN than GPi stimulation, especially when off levodopa. Our results indicate that DBS at both GPi and STN alleviates parkinsonian signs and symptoms and suggest that off-levodopa motor symptoms respond more to STN stimulation, while on symptoms are more improved by stimulation at GPi.

DBS devices provided by Medtronic, Inc.

NOTES

Friday, November 6
12:06-12:24 PM

The Beneficial Effects of Radiosurgery in an Animal Model of Hippocampal Epilepsy

Douglas Kondziolka, MD, Y. Mori, MD, J. Balzer, PhD, K. Thulborn, MD, L.D. Lunsford, MD

Background: Stereotactic radiosurgery (SR) has been used in patients with lesion-related or idiopathic epilepsy. The radiation dose necessary to eliminate epileptogenesis is unknown and the effects of radiosurgery undefined. Injection of kainic acid into the rat hippocampus produces a reliable model of chronic epilepsy. We hypothesized that radiosurgery could significantly reduce seizures without causing brain injury.

Methods: Kainic acid (8 mcg) was injected stereotactically into the rat hippocampus. Focal seizures were created and identified with scalp and depth EEG. Rats were randomized to a control group (n=20) and SR groups of 20, 40, 60, 80, and 100 Gy (8-9 animals per dose). Over 42 days, rats were observed daily and seizures counted. Scalp EEG was performed weekly. Functional MR images (T1, T2, and total sodium concentration) were performed on days 7, 21, and 42.

Results: Significant reductions in seizures were noted during each successive week after 20 Gy radiosurgery (p=0.01-.002). From weeks 4-6, we found significant reductions in seizures after 20 Gy (p=.007), 40 Gy (p=.03), 60 Gy (p=.03), and 100 Gy (p=.03) compared to control. Seizure reduction on EEG correlated with increasing SR dose. MR determined total Na+ concentration in the injected hippocampus was 49.8 mM compared to 42.8 mM in the contralateral side (within normal limits). This increase in [Na+] was due to kainic acid and did not change with increasing SR dose. No histological parenchymal effects from radiosurgery were identified after 20, 40, and 60 Gy, and only 2 rats had necrosis at 100 Gy. All animals showed CA-1 neuron loss from kainic acid.

Conclusions: In a model of hippocampal epilepsy, radiosurgery was followed by a significant reduction in the number of observed and EEG-defined seizures. This reduction correlated with increasing radiosurgery dose. These effects were not due to radiation-induced brain injury or change in Na+ metabolism. Radiosurgery may prove to be a new, non-destructive approach for the management of idiopathic epilepsy.

NOTES

Saturday, November 7
8:00-8:18 AM

Pediatric Shunt Death in the 1990's

Bermans J. Iskandar, M.D., Shane Tubbs, S.A.,
Timothy B. Mapstone, M.D., Paul A. Grabb, M.D.,
Alfred A. Bartolucci, Ph.D., W. Jerry Oakes, M.D.

We conducted a population based study of children and adolescents in Jefferson County, Alabama (Birmingham) who died from shunt failure from January 1990 to July 1996. We excluded patients that were reasonably thought to have died from some other process (brain tumor, bronchopulmonary dysplasia, etc.). A total of 28 patients met these criteria. Twenty-six of the twenty-eight had adequate data for analysis. Ten of the twenty-six had symptoms suggestive of shunt malfunction from 1-14 days prior to their demise but had no neurosurgical evaluation. These children died from a lack of education of either the family or the non-neurosurgical medical community. Five of the twenty-six died in neurosurgical care and could be considered deaths from technical errors or poor surgical judgements. Eleven of the twenty-eight had their first shunt insertion performed at the Children's Hospital of Alabama during this time frame. This was from a total of 357 initial shunt insertions. When viewed from a probability standpoint we continue to loose 1-2% of the shunted population each year despite vigorous educational and teaching efforts as well as improvements in shunt products and shunting techniques.

NOTES

Saturday, November 7
8:18-8:36 AM

Evaluation of Outcome and Cost of Craniotomy for Tumor Performed in Regional Academic Referral Centers

Don M. Long, Toby Gordon, Anthony Etzel,
Gregg Burleyson, Simone Betchen, Soosan Shahrokh,
Henry Brem

This study examines the effects of regionalization of tertiary care by analyzing cost and outcome for cranitomy for tumors and compares academic medical centers versus community based hospitals. Outcomes and charges were analyzed for all adult patients undergoing cranitomy for tumor in Maryland hospitals.

In-hospitals mortality, length of stay, and charges for all adult patients operated upon during a seven year period (1990-1996), were compared between tertiary care centers (>50 cases a year), and community based hospitals (≤50 cases a year). Mortality statewide was 3.5%. Mortality at tertiary care centers was 2.5%, and in community -based hospitals was 4.9% with a relative risk of 4.3 ($p<0.001$). If all patients in the state were treated at centers with survival rates equal to the tertiary care centers then an additional 46 patients would be alive or 48.6% fewer patients would have died. Average length of stay statewide was 11.0 days. Average length of stay in tertiary care centers as 9.9 and 12.5 in community based hospitals ($p<0.001$). Average total charges were $18,383 statewide and adjusted for age, race, comorbidity score, payment source, and diagnosis were $15,867 at tertiary care centers and $14,049 at community based hospitals ($p<0.001$).

High volume regional medical centers are capable of providing tertiary care services with significantly improved mortality and less hospital days although at adjusted costs slightly higher than community based hospitals.

NOTES

Saturday, November 7
8:36-8:54 AM

Incorporation of skull base techniques into a general intracranial microsurgical practice. A three year experience.

Roberto C. Heros, M.D.
University of Miami

An experienced neurosurgeon, with his knowledge of anatomy, additional pertinent reading and short periods of focused practice in the dissecting laboratory, should be able to incorporate basic "skull base" approaches into his general neurovascular and tumor practice. I have found the four basic approaches and occasional modifications to be discussed sufficient to handle almost all the "operable" tumors and vascular lesions seen in my practice during the last three years.

1. The low basal bifrontal approach without or with orbitonasal osteotomy. This approach has been used for 16 craniofacial tumors (mostly esthesioneuroblastomas, invasive meningiomas and low grade malignancies), 18 meningiomas of the olfactory groove, and the tuberculum, and 2 craniopharyngiomas.

2. The combined pterional/subtemporal approach without or with orbito-zygomatic or zygomatic osteotomy. This approach has been used for 22 aneurysms, mostly of the upper basilar (138 other aneurysm of the anterior circle of Willis have been operated through a standard pterional craniotomy) and for 26 tumors (mostly medial-basal meningiomas, trigeminal neurinomas and clival chordomas and chrondosarcomas). Kawase's anterior petrosectomy has been added to this approach in 2 cases.

3. The combined subtemporal/pre-sigmoid approach. This approach was used for 3 basilar origin and basilar trunk aneurysms, 5 meningiomas, 2 cholesteatomas and 3 acoustic neuromas (during this time 58 other acoustic neuromas were operated by the standard suboccipital or subtemporal approaches). A translabrynthine corridor was added in 2 cases.

4. The far lateral suboccipital approach. This approach was used for 6 vertebral and vertebrobasilar aneurysms and for 8 tumors, mostly meningiomas of the foramen magnum, lower clivus or jugular fossa.

The techniques used by the author with some examples of their application will be briefly illustrated.

NOTES

Saturday, November 7
8:54-9:12 AM

Intraoperative And Perioperative Electrocorticographic Evidence Of Functional Reorganization Of The Homunculus.

Paul C. Francel, K. J. Oommen, Department of Neurosurgery and the University Presbyterian Neurologic Institute, University of Oklahoma Health Sciences Center, Oklahoma City, OK 73104

There is clinical evidence that a functional reorganization of the cerebral cortex occurs following various forms of cerebral injury. This is particularly apparent in patients who have had stroke events or traumatic brain injury, but can be seen in other forms of cerebral injury. Actual intraoperative and perioperative recording of functional reorganization, however, has not been previously well studied. We report the functional reorganization and sometimes even duplication of the motor homunculus in patients presenting with intractable seizures induced by various pathologic entities. Perioperative and intraoperative electrocorticography was performed in all of these patients in order to successfully remove an epileptogenic focus in the motor cortex and the offending lesion without causing permanent hemiplegia or hemiparesis. These surgeries were performed by the primary author in conjunction with a principle epileptologist (2nd author) at our quaternary level epilepsy center. In each patient, the cortex was mapped following placement of one or more subdural grids which enabled both intraoperative and postoperative EEG recording, stimulation capability, and evaluation of motor, speech, sensory and other neurologic functions in the adjoining area. Stimulation was performed using a Grass Model S12 stimulator. Patient's stimulus artifact, EEG, and after discharges were recorded on both paper EEG for immediate visual characterization and also digital recording with both direct observation of the patient and closed circuit video surveillance. The motor and sensory cortices were mapped in each case and the relationship of the epileptogenic focus was correlated to the derived homunculus and this was then used for intraoperative planning. The pattern of change could not be predicted preoperatively and could not be directly correlated with age. For example, one patient with a very well circumscribed oligodendroglioma distinctly within the motor cortex showed minimal reorganization of the cortex with little translocation of function (and with a distinct area of lack of cortical function over the region of the oligodendroglioma) whereas another older patient who had multiple head injuries and subdural hematomas showed a major cortical reorganization with the motor homunculus being distinctly organized in a nonsequential pattern and being localized in a nontraditional format. One teenage child showed a complete duplication of his motor cortical function 3 cms posterior to the region of his dysembryoplastic neuroepithelial tumor which enabled, in that case, a full resection of his tumor and of the epileptogenic focus with minimal postoperative morbidity and with long term follow-up of normal neurological motor function. Some patients showed no change in the cortical structure even within the tumor (i.e. showing normal function within a more diffuse primary brain tumor) in which case resection of the tumor was not possible without significant risk to the patient. Age did not appear to be a complete criteria as significant reorganization was noted both in several pediatric case examples and also in patients of middle age or older. We conclude the functional reorganization occurs both in adults as well as in pediatric patients to a significant extent and that translocation may be secondary either to the extent of cortical injury, the acuteness versus chronicity of that injury, and potentially to the period of time of clinical seizures. We also conclude that both intraoperative and perioperative extensive mapping through both recording and stimulation parameters plays a significant role in intraoperative planning and resection of both tumors and epileptogenic foci.

NOTES

Saturday, November 7
9:12-9:30 AM

Re-operation for Residual/Recurrent Aneurysms of the Basilar Apex

Duke Samson MD, H.Hunt Batjer MD, Thomas Kopitnik MD, Michael Horowitz MD

The surgical management of previously operated intracranial aneurysms is recognized to be a technically challenging endeavor with high operative morbidity/ mortality. Giannotta has pointed out this enhanced surgical risk relates in part to the length of elapsed time between surgical procedures and to the location and size of the residual lesion. To clarify the operative problems and anatomical features of basilar apex aneurysms of this type, and to investigate the management morbidity of such lesions, a retrospective review of 346 basilar aneurysms operated at one institution over a nineteen year time span was conducted.

Criteria for inclusion in the study were the post-operative presence of a basilar apex aneurysm (or aneurysm remnant) which was either symptomatic or was shown by sequential angiography to undergo enlargement. Excluded from consideration were patients undergoing immediate surgical re-exploration to correct errors in clip placement demonstrated on intra- or post-operative angiography.

Nineteen patients having undergone a total of twenty-one prior operative procedures were identified. Four had undergone surgical explorations without treatment, followed in one case by re-operation and attempted clipping and in the other by re-operation and wrapping. Four patients had basilar aneurysms initially invested with foreign material (acrylic-1, muscle-1, muslin-2) . The eleven remaining patients had undergone primary clip-ligation. Only four patients had undergone post-operative angiography immediately following their initial operation; none of those studies were available for our review. Formal radiology reports in three of these described no residual aneurysm.

Eleven patients (58%) presented with documented SAH occurring from one week to eight and one-half years after initial treatment, four developed symptoms related to aneurysm mass effect and four were diagnosed on investigation of severe headache. The patients came to clinical attention at periods from one week to fourteen years after the original surgical procedure - patients tended to present with sub-arachnoid bleeding earlier following initial treatment, and to be more prone to the development of mass effect-related symptoms remote from original operation.

Six aneurysms were giant in size at the time of representation (two of which had been described as giant at the original operation) and the remainder were all greater than 7mm in diameter. The site of aneurysm residual or regrowth appeared to be the aneurysm neck in nine of the twelve having undergone previous clip-ligation.

Re-treatment involved temporary arterial occlusion in ten cases, hypothermic cardiac arrest in two, mandatory removal of the previously placed aneurysm clips in eight and was complicated by intra-operative aneurysm rupture in seven. Definitive clip placement with aneurysm obliteration was possible in eighteen of nineteen cases, but post-operative angiographic conformation was obtained in only eleven.

Management morbidity and mortality was high. Ten patients were able to return to full vocational and avocational activity, although oculomotor paresis and/or mild pyramidal weakness was present in four of these patients at six-months follow-up. Following surgery five patients were neurologically disabled (26%); in three deficits were related to operative intervention. Four patients died (21%), three of whom presented with SAH and one with brainstem compression. The primary causes of morbidity/mortality were the effects of SAH, protracted intra-operative ischemia and aneurysmal mass effect.

This small surgical series documents the difficulty of successful management of residual/ recurrent aneurysms of the basilar apex, and confirms the area of aneurysm neck as the locus of re-growth and ultimate (re)hemorrhage. It suggests that hemorrhage from these basilar apex aneurysms is more prone to occur in the first months and years after initial treatment, and that symptoms of mass effect secondary to aneurysmal enlargement may be more common at times more remote from original operation. By virtue of the findings at angiography and operation, it also suggests that residual/recurrent basilar aneurysms generally must reach a size of some 7mm before producing symptoms . Finally, its results endorse the benefits of routine early post-operative angiography in patients undergoing surgical treatment of basilar artery lesions even though a normal post-operative study may not offer complete reassurance that a symptomatic re-occurrence may not develop.

NOTES

Saturday, November 7
9:30-9:48 AM

Anterior Cervical Discectomy without Interbody Fusion

Paul B. Nelson, M.D., Jill Donaldson, M.D.

Sixty four consecutive patients who had anterior cervical discectomies (ACD) without interbody fusion from 4/97 to 2/98 were reviewed retrospectively. There were 35 males and 29 females. The mean age was 48.8 years and the mean follow-up was 8.6 months. Forty seven (68.8%) patients presented with a radiculopathy. Fifteen (23.4%) patients presented with a radiculopathy and myelopathy. Five (7.8%) patients presented with a myelopathy. All patients had failed consecutive therapy and were evaluated pre-operatively with cervical MRI's. At surgery, all disc and osteophytes were removed. The patients were placed in a cervical collar for 3 months. Forty two (63%) had surgery at 1 level, sixteen (25%) had surgery at 2 levels, and 1 (27%) had surgery at 3 levels.

Fifty eight (91%) of the patients had excellent or good results. Six (9%) had satisfactory results. There were no poor results. 91% of the patients who had jobs returned to work. Most patients went on to have a solid boney fusion at the operative level. The most common post-operative complication was 8 (12.5%) patients who had transient increased neck/interscapular pain.

The difference in charges for patients with ACD with and without interbody fusion will be discussed.

NOTES

Saturday, November 7
9:48-10:06 AM

Craniopharyngiomas: Experience with complex approaches in children

Michael L. Levy, MD

Craniopharyngiomas are benign intracranial tumors, which can represent complicated management problems due to the invasive nature of the lesion and the poor response to adjuvant therapy. Given that optimal cure rates are related to the completeness of resection, considerations of surgical approach are significant determinants of potential outcome. An additional consideration is the manner in which such approaches are practiced and perfected.

We evaluated our series of children presenting with craniopharyngioma and the anatomical patterns and decision making process utilized in determining the operative approach. Approaches reviewed included the transphenoidal (6/16), subfrontal (5/16), orbitozygomatic (2/16), and transbasal/interhemispheric (3/16). Patient ages ranged from two years and seven months at the date of surgery to 16 years with an average of 8.1 years ($\pm$ 4.4yrs). Four of the patients were female and 12 male.

We will discuss the advantages and disadvantages of each approach in addition to the potential and real complications. The attendant limitations of each surgical approach will be discussed with regard to tumor extension and injury to eloquent structures. Correlates of experience and landmarks established in the neurosurgical pediatric skull base laboratory utilizing 3 dimensional endoscopy and concurrent picture in picture imaging for education will also be discussed. We will further discuss the facility of use and the importance of the integration of concurrent 3-D endoscopic images into display systems to potentially increase the facility of microsurgical manipulation and dissection.

NOTES

Saturday, November 7
10:36-10-54 AM

Pathophysiological Changes in Cerebral Vessels Due to Hypertension

J. Marc Simard, M.D., Ph.D. and Xing Li, Ph.D., Neurosurgery Department, University of Maryland, Baltimore, MD

Hypertensive intracerebral hemorrhage remains a devastating cause of stroke. Hemorrhage is ascribed to arteriolosclerosis of penetrating arterioles, with advanced disease accompanied by focal loss of arteriolar smooth muscle and formation of Charcot Bouchard aneurysms. We hypothesized that loss of smooth muscle was due to hypertension-induced elevation of intracellular calcium, leading to calcium-mediated apoptotic cell death. To elucidate hypertension-induced physiological changes that could lead to smooth muscle apoptosis, we studied calcium channels in cerebrovascular smooth muscle cells isolated from hypertensive rats. In a series of 104 cells, we found that the density of functional calcium channels correlated strongly with blood pressure, but that the biophysical properties of the channels were not altered. We then showed that these effects were identical to removal of nitric oxide, and thus hypothesized that the effects in hypertension were due to endothelial dysfunction with loss of nitric oxide production. To test this, we studied another series of cells from animals pretreated with agents to protect from mechanical stimulation of endothelium-derived nitric oxide production and showed that the changes in calcium channels in hypertension were consistent with endothelial dysfunction. Finally, in separate experiments on human tissue, we obtained evidence for apoptosis in lenticulostriate arterioles from hypertensive patients. Our data provide evidence for a new hypothesis unifying hypertension-induced endothelial dysfunction, calcium channel dysregulation in smooth muscle, and calcium mediated apoptosis of smooth muscle as a cause for arteriolosclerosis predisposing to intracerebral hemorrhage.

Supported by grants from the National Heart, Lung and Blood Institute (HL42646) and the American Heart Association.

NOTES

Saturday, November 7
10:54-11:12 AM

Transsphenoidal Surgery for Cushing's Disease in Patients with a Normal Pituitary MRI

Patrick L. Semple, MD, Edward R. Laws, Jr., MD
Department of Neurosurgery, University of Virginia Health System, Box 212, Charlottesville, Virginia 22908, USA

The diagnosis and surgical management of patients with Cushing's disease and a normal pituitary imaging study continues to be a great challenge. It is recognized that somewhere between 20% and 40% of patients with Cushing's disease will have a normal sella and normal pituitary MRI imaging. In these cases, the diagnosis is made by careful and systematic endocrine testing which frequently includes Inferior Petrosal Sinus Sampling.

We reviewed a consecutive series of 105 patients operated upon for Cushing's disease within the last five years. In this group were 21 patients with normal pituitary MRI studies as interpreted both by neuroradiologist and the operating surgeon. Sixteen of the patients had IPS Sampling, the endocrine diagnosis of Cushing's disease was made using a series of laboratory tests which will be described with regard to their reliability and the sequence in which they should be performed. At surgery, 17 patients had pituitary microadenomas, and one patient had diffuse involvement with the pituitary tumor cells throughout a pituitary gland that was removed by hypophysectomy. Follow-up averaging 21.6 months revealed that 16 patients were in remission.

The method of diagnosis of this difficult group of patients will be described along with the techniques of surgery. The decision for or against hypophysectomy and the management of those patients when surgical cure is not achieved, will be discussed along with current recommendations based on this review.

NOTES

Saturday, November 7
11:12-11:30 AM

Microvascular Failure following Reperfused Stroke: Experimental Evidence and Implications for Clinical Cerebral Protection

E. Sander Connolly, Jr. Assistant Professor of Neurological Surgery, Columbia University, New York, NY
guest of: Edward S. Connolly, M.D., The Ochsner Clinic, New Orleans, LA

Cerebral ischemia is the third leading cause of death, the leading cause of disability and a persistent cause of perioperative morbidity in patients undergoing cerebrovascular surgery. Although timely reperfusion can rescue jeopardized brain tissue, delayed reperfusion may actually worsen outcome by exposing neurons to toxic enzymes and reactive oxygen intermediates. In our laboratory we have demonstrated that this ischemia-reperfusion injury is in large part mediated by proinflammatory cytokines and endothelial adhesion receptor molecuses which together lead to progressive microvascular failure and diminished regional flow.

Specifically we have demonstrated the critical role of acute leukocyte influx as mediated by endothelial ICAM-1, P-selectin and E-selectin in promoting progressive flow failure and neurological demise, and have shown that mice either deficient in these receptors or exposed to functionally blocking antibodies are dramatically protected. We have also demonstrated the role of the leukocyte counterligand CD18, especially in the setting of reperfused stroke, as well as that of the proinflammatory cytokine IL-1, a potent upregulator of ICAM-1. In addition it appears that Pselectin dependent protection is in part mediated by reductions in delayed microvascular fibrin deposition and thrombosis, and that novel strategies which block P-selectin binding and complement fixation are more protective than those which block the former alone.

However, while we have been able to obtain convincing evidence of progressive microvascular failure excacerbating experimental murine stroke, simultaneously conducted human trials of anti-ICAM-1 antibody strategy have yielded disappointing results. Some have suggested that this failure is due to the relative irrelevance of microvascular failure in human stroke, while others have suggested that greater physiological redundancy in primates necessitates a cocktail approach.

To address these questions we have established two novel models of reperfused stroke, one in baboons and one in humans to explore this question. In the former we have demonstrated the upregulation of P- and E- selectin in serum and have demonstrated for the first time a correlation between their levels, infarct volume and neurological outcome. We have also demonstrated in humans undergoing CEA that subclinical neurological injury as evidenced by elevated neuron-specific enolase levels and neuropsychological dysfunction seem to correlated with proinflammatory markers as well.

Together these data suggest that an anti-P/E- selectin strategy may be useful in ameliorating the reperfusion injury in humans and we will present the preliminary results of a placebo blinded trial of a humanized anti-P/E-selectin antibody in the treatment of primate stroke as well as ongoing studies designed to elucidate the effect of this treatment on microvascular failure.

NOTES

Saturday, November 7
11:30-11:48 AM

Carotid Revascularization Using Open Surgery or Endovascular Techniques

Definition of Surgical Risk by Quantifying Medical Comorbidity

L. R. Guterman, PhD, MD and L.N. Hopkins MD

The use of carotid angioplasty and stent for revascularization of the cervical carotid bifurcation remains controversial. The NACSET and ACAS studies have demonstrated that carotid endarterectomy (CEA) is safe and efficacious in a selected patient population. Morbidity and Mortality (M&M) associated with CEA in this patient population is well defined. The M&M for patient who were not candidates for NASCET and ACAS should not be extrapolated from this data. Patients with restenosis after CEA, radiation induced stenosis, unstable angina awaiting coronary revascularization, high cervical bifurcations with short necks, multiple medical comorbidites, or unacceptable risk for general anesthesia were considered "high risk" for CEA at our institution.

From May 1995 to present the authors performed over 100 cervical carotid revascularization procedures using angioplasty and stent in a "high risk" population. During that time our group performed a similar number of CEA.

A retrospective review of medical and surgical risk factors is underway to stratify the risk profile of patients treated by CEA and angioplasty and stent based on the taxonomy of medical and surgical disease. Comparison of patients revascularized using the two techniques will quantify the degree of infirmary and the surgical risk in each group. Comparison of perioperative morbidity and mortality for the two procedures will include the medical and surgical comorbidity in each group.

NOTES

Saturday, November 7
11:48 AM - 12:06 PM

Carotid Angioplasty and Stenting for Recurrent and Radiation-Induced Stenosis

Robert H. Rosenwasser, Rocco A. Armonda, Ronald P. Benitez

Introduction: Recurrent and radiation-induced stenosis may carry an increased morbidity and mortality associated with surgical repair. In this patient population, carotid angioplasty and stenting may have a role in the treatment of this disease process.

Materials and Methods: This analysis includes 27 patients treated for high grade carotid stenosis; 7 of these were radiation-related, 14 were post-surgical with recurrent stenosis, and 6 were high Sundt and Goldman classification. All patients treated were Sundt classification III or IV, or Goldman classification III or IV. The protocol involved local standby anesthesia with temporary transvenous cardiac pacing via transfemoral route. Patients underwent angioplasty and pre-dilation followed by stent deployment of either the Wallstent or Palmaz stent. Anticoagulation was continued for 24 hours, at which time the sheath was removed and patients were begun on Aspirin and Ticlopidine.

Results: 26.27 patients had no transient ischemic episodes in the perioperative period, with a minimum of three month follow-up. One patient developed vascular insufficiency of the right lower extremity which resolved with Heparin. One patient presented one week later with complete carotid occlusion, which was reopened with urokinase; however, in the subsequent 24 hours, the patient developed an intracranial hemorrhage and succumbed. In 26/27 patients, the length of stay was less than or equal to 36 hours. The cost of the angioplasty and stenting was approximately $28,000.00 per case, as compared to $20,000.00 for carotid endarterectomy.

Conclusions: Carotid angioplasty and stenting may be a reasonably alternative to carotid endarterectomy in certain high risk patients. Rationale and methodology will be further discussed.

NOTES

Saturday, November 7
12:06-12:24 PM

Moyamoya Disease in Adults: Clinical Characteristics and Outcome after Encephaloduroarteriosynangiosis (EDAS)

Dae Hee Han, Tae Ho Kim, Young Seob Chung
Department of Neurosurgery, Seoul National University College of Medicine, Seoul, Korea

To determine the clinical characteristics and effectiveness of encephaloduroarteriosynangiosis (EDAS) in adulthood-onset moyamoya disease (MMD), the authors retrospectively reviewed 37 patients suffering from MMD who were admitted to Seoul National University Hospital between 1987 and 1997, presenting with major symptoms over the age of 16 years; three patients were with probable MMD.

The most common presenting symptom was intracranial hemorrhage, found in 20 patients(54%); others were transient ischemic attack and/or infarction in 15(41%) and seizures in 2(5%). The Suzuki angiographic stage 3 and less than 3 accounted for 73% of all 71 hemispheres. Twenty-seven patients underwent single photon emission computed tomography (SPECT) preoperatively. In terms of cerebral perfusion based on four SPECT grades(SG), 55% of their hemispheres revealed normal (SG 1) or localized decreased-perfusion (SG 2). The other 45% had extensive decreased-perfusion or localized perfusion defect (SG 3). There was no case that had extensive perfusion defect (SG 4). Twenty-four patients underwent EDAS operations (EDAS group) and thirteen did not undergo any operations (no-op group). The EDAS group showed significantly better clinical outcomes than the no-op group after a 18-month mean follow-up period ($p=0.03$). On the angiographic and SPECT follow-up studies, there was also satisfactory angiographic revascularization in 12 of 13 patients and improvement in cerebral perfusion in 11 of 14 patients.

It is concluded that the involvement of posterior circulation of MMD is not frequent and cerebral perfusion is preserved in adulthood-onset MMD patients. These findings may explain the reason why hemorrhages are frequent in adulthood-onset MMD as the late onset of symptoms. Surgical treatment with EDAS seems to be effective in adulthood-onset MMD and enable to make clinical improvement.

NOTES

Saturday, November 7
12:24-12:42 PM

Neurological Manifestations of Cowden Disease: Characterization of a New Phakomatosis

Alan R. Cohen, M.D., and Shenandoah Robinson, M.D.
Case Western Reserve University School of Medicine, Cleveland, Ohio

We report five patients at the Case Western Reserve university Medical Center who had an unusual association of Lhermitte-Duclos disease and Cowden disease. The association of these rare disorders suggests that they may be part of a single phakomatosis.

Lhermitte-Duclos disease, or dysplastic cerebellar gangliocytoma, is a peculiar hamartoma arising from the cerebellar cortex in young adults, although age at presentation ranges from birth to the sixth decade. There is no sex predilection. The clinical presentation is one of gradually progressive headache, gait disturbance and cranial nerve dysfunction. Magentic resonance imaging shows a non-enhancing lesion of the cerebellar hemisphere with a gyriform pattern and, sometimes, hydrocephalus. The only effective therapy for symptomatic Lhermitte-Duclos disease is surgical excision. Coincident with advances in neurodiagnostic imaging, the prognosis for patients with Lhermitte-Duclos disease has improved.

Cowden disease, also called the multiple hamartoma/neoplasia syndrome, is a rare autosomal dominant disorder characterized by mucocutaneous lesions including facial papules, gingival papillomas and acral keratoses. Other systemic hamartomas are often present, and there is a high incidence of associated breast, genitourinary and thyroid carcinoma.

We report five patients with manifestations of both Lhermitte-Duclos disease and Cowden disease. All were women who presented with signs of increased intracranial pressure and subsequently underwent surgical resection of a dysplastic cerebellar gangliocytoma. Each patient also had systemic hamartomas, along with a variety of benign and malignant neoplasms, leading to the diagnosis of Cowden disease.

We believe that Lhermitte-Duclos disease is a neurologic manifestation of the phakomatosis, Cowden disease. The association between Lhermitte-Duclos disease and Cowden disease has been under-recognized and under-reported. Recognition of this association has direct clinical bearing: diligent long-term follow-up of individuals with Lhermitte-Duclos disease and the Cowden spectrum may lead to the early detection of malignancy.

NOTES

Saturday, November 7
12:42-1:00 PM

Leukocyte-Endothelial Adherence In Penumbral Cortex 24 Hours After Permanent Middle Cerebral Artery Occlusion in Mice

T.S. Park, M.D., Jeffrey M. Gidday, Ph.D., Stuart S. Kaplan, M.D.
Department of Neurosurgery, St. Louis Children's Hospital
Washington University School of Medicine, St. Louis, MO

Recent studies in temporary occlusion models of cerebral ischemia in transgenic mice support the hypothesis that leukocytes contribute to ischemic brain injury; however, based on studies in rats, leukocytes may play a less significant role in permanent MCAO injury models. The present study was performed in adult male chloral hydrate-anesthetized Swiss-Webster mice to assess leukocyte-endothelial interactions in pial venules in the penumbral cortex in a model of permanent focal MCAO produced by temporal craniotomy and MCAO distal to the internal cerebral vein. Occlusion was confirmed by visual inspection and reduction of laser Doppler CBF to 10% of baseline. Rectal temperature was controlled until the animals regained full consciousness. At 24 h post-occlusion, animals were equipped with closed cranial windows over the right parietal, parasagittal cortex, a region which at 24 h exhibited significantly lower flow than that measured in sham-operated controls. Penumbral leukocyte dynamics imaged by epifluorescence videomicroscopy of rhodamine-labeled leukocytes revealed a significant increase in the number of adherent leukocytes at this time (473±108 leukocytes/mm^2 endothelial surface; n=6) compared to sham controls (60±4/mm^2; n=5; $p<0.003$). Infarct volume determined by TTC staining in these animals was 25±3 mm^3; the area of infarction was always lateral to the location of the cranial window. Our results suggest that anti-leukocyte interventions may find utility in ameliorating vascular and parenchymal injury in penumbral regions following permanent focal ischemia. Supported by NIH grant 1R01 NS 21045

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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
Loew's Ventana Canyon Resort,
Tuczon, 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

FUTURE MEETINGS:

Amelia Island, FL
Ritz Carlton ..... November 10-13, 1999
The Broadmoor
Colorado Springs, CO ..... October 11-14, 2000
Colonial Williamsburg
Williamsburg, Virginia ..... October 28-Nov. 3, 2001

Past Presidents

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

Francis Murphey 1941
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Henry G. Schwartz 1951
J. Lawrence Pool 1952
Rupert B. Raney 1953
David L. Reeves 1954
Stuart N. Rowe 1955
Jess D. Herrmann 1956
George S. Baker 1957
Samuel R. Snodgrass 1958
C. Hunter Shelden 1959
Edmund Morrissey 1960
Donald F. Coburn 1961-62
Deben Alexander, Jr. 1963
George L. Maltby 1964
Robert Pudenz 1965
Francis A. Echlin 1966
Benjamin Whitcomb 1967
Homer S. Swanson 1968
Augustus McCravey 1969-70
Edward W. Davis 1971
John R. Green 1972
George J. Jayes 1973
Richard L. DeSaussure 1974
Ernest W. Mack 1975
Frank E. Nulsen 1976
Robert S. Knighton 1977
Robert G. Fisher 1978
H.T. Ballantine, Jr. 1979
George Ehni 1980
Courtland H. Davis, Jr. 1981
John F. Mullan 1982
Hugo Rizzoli 1983
James W. Correll 1984
E. Bruce Hendrick 1985
Griffith R. Harsh III 1986
Ellis B. Keener 1987
Robert Grossman 1988
Jim Story 1989
John Jane 1990
Stewart Dunsker 1991
Burton Onofrio 1992
Martin Weiss 1993
John M. Tew, Jr. 1994
John Van Gilder 1995
Edward Connolly 1996
George Ojemann 1997

Past Secretary-Treasurers

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

Past Secretaries

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

Past Treasurers

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

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GUY LAZORTHES, (Annick) — 1973
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VALENTINE LOGUE (Anne) — 1974
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ENGLAND

BERNARD PERTUISET — 1986
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Paris, Cedex 13
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KEIJI SANO (Yaeko) — 1975
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EBEN ALEXANDER JR. (Betty) 1950
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RAEBURN C. LLEWELLYN (Carmen Rolon) 1963
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RUSSEL H. PATTERSON, JR. (Julie) 1971
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SYDNEY JOHN PEERLESS (Ann) 1977
Mercy Neuroscience Institute
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PHANOR PEROT, JR. 1970
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BYRON CONE PEVEHOUSE (Lucy) 1964
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J. LAWRENCE POOL 1940
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AIDEN A. RANEY 1946
125 N. Las Palmas Avenue, Suite 203
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JOSEPH RANSOHOFF II (Lori Cohen, DDS) 1965
James A. Haley Veteran's Hospital
13000 Bruce B. Downs Blvd.
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THEODORE RASMUSSEN (Catherine) (deceased) 1947
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Montreal, Quebec H3P 1B2
CANADA

ALBERT RHOTON, JR. (Joyce) 1984
Department of Neurological Surgery
College of Medicine, P.O. Box 100265
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HUGO V. RIZZOLI (Helen) 1973
2150 Pennsylvania Avenue, N.W. #7-420
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THEODORE ROBERTS (Joan) 1976
Childrens Hospital
4800 Sand Point Way
Seattle WA 98105

JAMES T. ROBERTSON (Valeria) 1971
University of Tennessee
College of Medicine
847 Monroe Ave., Suite 427
Memphis, TN 38163-0001

EDWARD L. SELJESKOG (Peggy) 1992
2805 Fifth Street South
Suite 110
Rapid City, SD 57701-7306

C. HUNTER SHELDEN 1941
Huntington Medical Research Institute
744 Fairmont Avenue
Pasadena, CA 91105-3105

WILLIAM SHUCART (Laura) 1989
Department of Neurosurgery
New England Medical Center
750 Washington Street
Boston, MA 02111

JAMES C. SIMMONS (Vanita) 1975
190 S. Grove Park Road
Memphis, TN 38117

BENNETT M. STEIN (Bonita) 1970
The Neurological Institute
710 West 168th Street
New York, NY 10032

JIM STORY (Joanne) 1972
315 N. Saba, Suite 1240
San Antonio, TX 78207-3154

ANTHONY F. SUSEN (Patricia) 1965
193 Old Glebe Point Road
Burgess, VA 22432-9801

WILLIAM H. SWEET (Elizabeth) 1950
Massachusetts General Hospital
Fruit Street
Boston, MA 02114

RONALD R. TASKER (Mary) 1971
Toronto Western Hospital
399 Bathurst Street, 2-431 McL
Toronto, Ontario M5T 2S8
CANADA

GEORGE T. TINDALL (Wendy) 1968
Emory Univ. School of Medicine
Department of Neurosurgery
1327 Clifton Road
Atlanta, GA 30322

JOHN S. TYTUS (Virginia) 1967
P.O. Box 279
Arlington, WA 98223-0279

EXUM WALKER (Nellie) 1938
735 Peachtree Battle Avenue, NW
Atlanta, GA 30327-1250

CLARK WATTS (Patricia) 1975
4314 Medical Parkway #101
Austin, TX 78756

LOWELL E. WHITE JR. (Margie) 1971
5750 Huffman Dr., N.
Mobile, AL 36693-3013

ROBERT H. WILKINS (Gloria) 1973
Duke University Medical Center, Box 3807
Durham, NC 27710-0001

CHARLES B. WILSON (Francie Petrocelli) 1966
Dept. of Neurological Surgery
Univ. of California in San Francisco
533 Parassus Ave., U125 Box 0350
San Francisco, CA 94143-0350

DAVID YASHON (Christine) 1972
#1201 1492 E. Broad Street
Columbus, OH 43205-1546

NICHOLAS T. ZERVAS (Thalia) 1972
Massachusetts General Hospital
Fruit Street, White 502
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ACTIVE MEMBERS

Elected

MICHAEL L. J. APUZZO (Helene) — 1988
1200 N. State Street, Ste. 5046
Los Angeles, CA 90033-4525

JAMES I. AUSMAN (Carolyn R.) — 1979
Univ. of Illinois at Chicago
912 S. Wood St.
Neuro. MC799
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ISSAM A. AWAD (Catherine) — 1996
Department of Neurosurgery
Yale University
333 Cedar Street, TMP 405/Neuro
New Haven, CT 06520-3206

DANIEL LOUIS BARROW (Mollie Winston) — 1993
Dept. of Neurological Surgery
The Emory Clinic, Suite 2200
1365 Clifton Road N.E.
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H. HUNT BATJER (Janet) — 1996
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MITCHEL S. BERGER (Joan)
UCSF/NEUROSURGERY
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KEITH L. BLACK (Carol Bennett) — 1995
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10833 Le Conte Ave., Box 957039
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PETER M. BLACK (Katharine) 1988
Children's Hospital/Neuro.
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LAWRENCE F. BORGES (Susan) 1993
Massachusetts General Hospital
Division of Neurosurgery, White 1205
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CHARLES L. BRANCH, JR. (Lesa) 1996
Dept. of Neurosurgery
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HENRY BREM (Rachel F. Brem) 1996
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John Hopkins Hospital
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WILLIS E. BROWN, JR. (Ann) 1984
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Univ. of Texas Health Science Ctr.
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7777 Forest Lane, Suite B308B
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KIM J. BURCHIEL (Debra) 1992
Oregon Health Sciences University/
Neurosurgery L-472
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MARTIN B. CAMINS (Joan) 1995
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PETER W. CARMEL (Jacqueline Bello) 1991
New Jersey Med. Sch./Neuro.
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WILLIAM F. CHANDLER (Susan) 1989
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Department of Neurosurgery
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DAVID G.T. THOMAS (Hazel) 1995
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DECEASED MEMBERS

	Deceased	Elected
JAMES R. ATKINSON Phoenix, Arizona (Active)	1978	1970
PERCIVAL BAILEY Evanston, Illinois (Honorary)	1973	1960
GEORGE BAKER Litchfield Park, AZ (Senior)	1993	1940
H. THOMAS BALLANTINE, JR. Boston, Massachusetts (Senior)	1996	1951
WILLIAM F. BESWICK Buffalo, New York (Active)	1971	1959
EDWIN B. BOLDREY San Francisco, California (Senior)	1988	1941
E. HARRY BOTTERELL Kingston, Ontario CANADA (Senior)	1997	1938
SPENCER BRADEN Cleveland, Ohio (Active)	1969	Founder
F. KEITH BRADFORD Houston, Texas (Active)	1971	1938

HOWARD BROWN San Francisco, California (Senior)	1990	1939
GALE CLARK Oakland, California (Senior)	1996	1970
DONALD COBURN Wilbington, Delaware (Senior)	1988	1938
WINCHELL McK. CRAIG Rochester, Minnesota (Honorary)	1960	1942
EDWARD DAVIS Portland, Oregon (Senior)	1988	1949
PEARDON DONAGHY Burlington, Vermont (Senior)	1991	1970
CHARLES DRAKE London, Ontario (Senior)	1958	1998
FRANCIS ECHLIN New Poaltz, New York (Senior)	1988	1944
DEAN ECHOLS New Orleans, Louisiana (Senior)	1991	Founder
GEORGE EHNI Houston, Texas (Senior)	1986	1964
ARTHUR ELVIDGE Montreal, Quebec, Canada (Senior)	1985	1939

THEODORE C. ERICKSON Madison, Wisconsin (Senior)	1986	1940
JOSEPH P. EVANS Kensington, Maryland (Senior)	1985	Founder
JOHN FRENCH Los Angeles, California (Senior)	1989	1951
JAMES GALBRAITH Birmingham, Alabama (Senior)	1997	1947
EVERETT GRANTHAM Louisville, KY 40202	1997	1942
JOHN GREEN Phoenix, Arizona (Senior)	1990	1953
JAMES GREENWOOD, JR. Houston, Texas (Senior)	1992	1952
WESLEY A. GUSTAFSON Jensen Beach, Florida (Senior)	1975	1942
HANNIBAL HAMLIN Providence, Rhode Island (Senior)	1982	1949
JOHN W. HANBERY Palo Alto, CA (Senior)	1996	1959
JESS HERRMANN Oklahoma City, OK (Senior)	1994	1938

HENRY L. HEYL Hanover, New Hampshire (Senior)	1975	1951
OLAN HYNDMAN Iowa City, Iowa (Senior)	1966	1942
KENNETH G. JAMIESON Brisbane, Australia (Corresponding)	1976	1970
SIR GEOFFREY JEFFERSON Manchester, England (Honorary)	1961	1951
RICHARD JOHNSON Cheadle Hulme, England (Senior Corresponding)	1974	1997
WILLIAM S. KEITH Toronto, Canada (Senior)	1987	Founder
HUGO KRAYENBUHL Zurich, Switzerland (Honorary)	1985	1974
KRISTIAN KRISTIANSEN Oslo, Norway (Senior corresponding)	1993	1967
WALPOLE S. LEWIN Cambridge, England (Corresponding)	1980	1973
HERBERT LOURIE Syracuse, New York (Senior)	1987	1965
WILLEM LUYENDIJK Oegstgeest, the Netherlands (Senior Corresponding)	1995	1973

M. STEPHEN MAHALEY Birmingham, Alabama (Active)	1992	1972
GEORGE L. MALTBY Scarsborough, Maine (Senior)	1988	1942
FRANK MARGUTH Munich, Germany (Senior Corresponding)	1991	1978
DONALD D. MATSON Boston, Massachusetts (Active)	1969	1950
FRANK MAYFIELD Cincinnati, Ohio (Senior)	1991	Founder
AUGUSTUS McCRAVEY Chattanooga, Tennessee (Senior)	1990	1944
KENNETH G. McKENZIE Toronto, Ontario, Canada (Honorary)	1964	1960
JAMES M. MEREDITH Richmond, Virginia (Active)	1962	1946
J. DOUGLAS MILLER Edinburgh, Scotland (Corresponding)	1995	1988
W. JASON MIXTER Woods Hole, Massachusetts (Honorary)	1968	1951
EDMUND J. MORRISSEY San Francisco, California (Senior)	1986	1941

FRANCIS MURPHEY Naples, FL (Senior)	1994	Founder
GOSTA NORLEN Goteborg, Sweden (Honorary)	1985	1973
FRANK NULSEN Naples, Florida (Senior)	1994	1956
SIXTO A. OBRADOR Madrid, Spain (Honorary)	1978	1973
PIETRO PAOLETTI Milan, Italy (Corresponding)	1991	1989
HANS-WERNER PIA Giessen, West Germany (Corresponding)	1986	1978
WILDER PENFIELD Montreal, Canada (Honorary)	1976	1960
HELMUT PENZHOLZ Heidelberg, West Germany (Corresponding)	1985	1978
ROBERT H. PUDENZ South Pasadena, CA (Senior)	1943	1998
RUPERT R. RANEY Los Angeles, California (Active)	1959	1939
BRONSON RAY New York, New York (Honorary)	1993	1992

DAVID L. REEVES Santa Barbara, California (Active)	1970	1939
DAVID REYNOLDS Tampa, Florida (Active)	1978	1964
R.C.L. ROBERTSON Houston, Texas (Senior)	1985	1946
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GLEN SPURLING LaJolla, California (Honorary)	1968	1942
C. WILLIAM STEWART Montreal, Quebec, Canada (Corresponding)	1948	1948
THORALF SUNDT, JR. Rochester, Minnesota (Active)	1992	1971

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