

THE
AMERICAN ACADEMY
OF
NEUROLOGICAL SURGERY



57th Annual Meeting



LOEWS VENTANA CANYON
RESORT

November 2-4, 199~~5~~⁵



Jointly Sponsored by The American
Association of Neurological Surgeons



**THE
AMERICAN ACADEMY
OF
NEUROLOGICAL SURGERY**



57th Annual Meeting



**LOEWS VENTANA CANYON
RESORT**

November 2-4, 1994⁵



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Association of Neurological Surgeons**







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THE AMERICAN ACADEMY OF
NEUROLOGICAL SURGERY

ACTIVITIES PROGRAM

November 1-4, 1995

Tuesday, October 31:

12:00 p.m. - 5:00 p.m. ABNS Exam. Committee
Review

Wednesday, November 1:

8:00 a.m. - 11:00 a.m. ABNS Residency Task
Force

8:00 a.m. - 5:00 p.m. ABNS Exam. Committee
Rincon Room

2:00 p.m. - 5:00 p.m. Registration
Ballroom Foyer

2:00 p.m. - 3:00 p.m. Executive Committee
Executive Board Room

3:00 p.m. - 5:00 p.m. Membership Committee
Executive Board Room

2:00 p.m. - 5:00 p.m. Speaker Ready Room
Sabino Room

6:00 p.m. - 8:00 p.m. Welcome Reception, Western t
Kiva Courtyard

7:00 p.m. - 10:00 p.m. Dinner
Individual arrangements*

* Ventana dining facilities:

Ventana Room - fine dining, tie-jacket
Canyon Cafe - informal

Thursday, November 2:

6:00 a.m.	Speaker Ready Room Sabino Room
7:00 a.m. - 8:00 a.m.	Breakfast/Business Mtg. (Members only)* Salon B
* Guests breakfast available in Canyon Cafe	
8:00 a.m. - 1:00 p.m.	Registration Ballroom Foyer
8:00 a.m. - 1:00 p.m.	General Scientific Session Salon A
10:30 a.m. - 10:50 a.m.	Refreshment Break
1:00 p.m. - 2:30 p.m.	ABNS Advisory Council Rincon Room
2:45 p.m. - 4:00 p.m.	Journal of Neurosurgery Editorial Board Rincon Room
1:00 p.m. - 6:00 p.m.	Tennis/Golf/Free Time (consecutive tee times)
6:00 p.m.	Buses leave for Tucson Country Club
7:00 p.m. - 10:00 p.m.	Dinner Jacket and tie Tucson Country Club

Friday, November 3:

6:00 a.m.	Speaker Ready Room Sabino Room
7:00 a.m. - 8:00 a.m.	Breakfast/Business Mtg. (Members only)* Salon B
* Guests breakfast available in Canyon Cafe	
8:00 a.m. - 1:15 p.m.	Registration Ballroom Foyer
8:00 a.m. - 1:15 p.m.	General Scientific Session Salon A
10:40 a.m. - 11:00 a.m.	Refreshment Break*
* Group Photograph to be taken during break	
12:15 p.m.	Presidential Introduction and Address David L. Kelly, Jr., M.D.
1:00 p.m. - 6:00 p.m.	Golf Tournament sign up at Registration Desk Shotgun Start
1:00 p.m. - 6:00 p.m.	Tennis Tournament sign up at Registration Desk
6:30 p.m. - 7:30 p.m.	Reception Ballroom Foyer
7:30 p.m. - 12:00 p.m.	Banquet - Dance Black tie Grand Ballroom, Salons B &

Saturday, November 4:

6:00 a.m. - ongoing	Speaker Ready Room Sabino Room
7:00 a.m. - 8:00 a.m.	Breakfast/Business Mtg. Members and Guests Salon B
8:00 a.m. - 1:15 p.m.	Registration Ballroom Foyer
8:00 a.m. - 1:00 p.m.	General Scientific Session Salon B
10:30 a.m. - 11:00 a.m.	Refreshment Break
1:00 p.m.	Golf sign up at Registration Desk

Sunday, November 5:

Departures

SCHEDULE OF ACTIVITIES
FOR SPOUSE AND/OR SIGNIFICANT OTHER

November 1-4, 199~~4~~⁵

Wednesday, November 1:

6:00 p.m. - 8:15 p.m. Welcome Reception, Western theme, Kiva Courtyard

7:00 p.m. - 10:00 p.m. Dinner arrangements
Individual arrangements*

* Ventana dining facilities:
Ventana Room - fine dining, tie-jacket
Canyon Cafe - informal

Thursday, November 2:

7:30 a.m. - 9:30 a.m. Ventana Walking Course
3 miles round trip
aerobic stops

7:30 a.m. - 10:30 a.m. Continental Breakfast
Hospitality Suite, Rm. 2205

10:00 a.m. - 12:00 p.m. Book Review
Barbara Kingsolver books
Pigs in Heaven
The Bean Trees
Animal Dream

11:45 a.m. A Book Exchange
Bring the name and author of
a few adored books

1:00 p.m. Tennis/Golf/Free Time
Sign up for tee and court
time at Registration Desk

6:00 p.m. Buses leave for
Tucson Country Club

Thursday, November 2:

7:00 p.m. - 10:00 p.m. Dinner
Tucson Country Club
Jacket and tie

Friday, November 3:

7:30 a.m. - 9:30 a.m. Ventana Walking Course
3 miles round trip
aerobic stops

7:30 a.m. - 10:30 a.m. Continental Breakfast
Hospitality Suite, Rm. 2205

10:45 a.m. Tucson arts & crafts and
shopping

1:00 p.m. - 4:00 p.m. Tennis Tournament
Lakeside Spa and
Tennis Club

1:00 p.m. - 6:00 p.m. Golf Tournament
sign up at Registration Desk

6:30 p.m. - 7:30 p.m. Reception
Ballroom Foyer

7:30 p.m. - 12:00 p.m. Banquet - Dance
Black tie
Grand Ballroom

Saturday, November 4:

7:30 a.m. - 9:30 a.m. Morning Stretch

7:30 a.m. - 10:30 a.m. Continental Breakfast
Room 2205

1:00 p.m. Golf
sign up at Registration Desk

**SCIENTIFIC PROGRAM
AMERICAN ACADEMY OF NEUROLOGICAL
SURGERY
1995 LEARNING OBJECTIVES
November 1-4, 1994⁵**

**Jointly Sponsored by The
American Association of Neurological Surgeons**

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 Surgeons is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

The American Association of Neurological Surgeons designates this continuing medical education activity for 14.7 credit hours in Category I of the American Medical Association.

SCIENTIFIC PROGRAM
**AMERICAN ACADEMY OF NEUROLOGICAL
SURGERY**

November 2-4, 1995

Jointly Sponsored by The American
Association of Neurological Surgery

Thursday, November 2

8:00 AM

Welcome
David L. Kelly, President

8:05 AM

Introduction of Scientific Program

SCIENTIFIC SESSION I
Moderator: **Suzie C. Tindall,**
Scientific Program Chair

8:10 AM

*Surgical Anatomy, Imaging and Results
of The Treatment of the Chiari
Malformation in Adults*
Charles Tator

8:30 AM

Anterior Thoracic Lumbar Plating
Volker Sonntag, Curtis Dickman,
AG Vishteh

8:50 AM

*Value of Cervical Facet Wiring Following
Laminectomy
(A Biomechanical Study)*
Joseph Cusick, Frank Pinter,
Narayan Yoganandan

Thursday, November 2

9:10 AM *The Myelopathy Disability Index - An
Objective Outcome Measure for
Spinal Surgery?*
**H. Alan Crockard, A.T.H. Casey,
M. Bland**

9:30 AM *Mapping Human Sensorimotor Cortex
With Functional MRI*
**G. Rees Cosgrove, BR Buchbinder,
H. Jiang, BK Rosen**

9:50 AM *Integration of Stereotactic Robotic
Guidance in Epilepsy Surgery*
**David Roberts, Terrance Darcey,
Peter Williamson**

10:10 AM *Image Guided Surgery: Experience With the
Zeiss-MKM System*
**James Ausman, Manuel Dujovny,
Fady Charbel, M. Serdar Alp,
Andrew Goldberg**

10:30 AM **Coffee Break**

SCIENTIFIC SESSION II
Moderators: **Richard Morawetz and
John Tew**

10:50 AM *Frameless Radiosurgery*
William Friedman, Frank Bova

Thursday, November 2

11:10 AM *Characterization of the Cerebral Hemodynamic
Phases That Follow Head Injury:
Hypoperfusion, Hyperemia
and Vasospasm*

**Neil Martin, Michael Alexander,
Ravish Patwardhan, Cynthia Zane,
Donald Becker, David Hovda**

11:30 AM *The Role of Reoperation After Radiosurgery
for Glioblastoma Multiform*

**Eben Alexander, III, Marc Schwartz,
Jay Loeffler, Dennis Shrieve,
Patrick Wen, Howard Fine,
Peter McL. Black**

11:50 AM *Radiosurgery for Brain Metastasis: Patient
Outcome and Tumor Response*

**Barton Guthrie, Richard Jenelle,
Randolph Bishop**

12:10 PM *The Kjellberg Experience With Dose
Escalation Proton Radiosurgery for the
Treatment of Large Inoperable Arteriovenous
Malformations*

**Paul Chapman, William Butler,
Christopher Ogilvy**

Thursday, November 2

12:30 PM **Academy Award Presentation**
Richard Morawetz,
Academy Award
Winner Committee Chair

12:35 PM *Neurotrophin Infusion Improves Cognitive*
Deficits and Decreases
Cholinergic Neuronal Cell Loss and Apoptotic
Cell Death Following
Experimental Traumatic Brain Injury

Runner Up Resident

Grant Sinson
University of Pennsylvania
School of Medicine

12:45 PM *Neurons Are Generated in the Adult Brain: In*
Vivo and In Vitro Studies of Cell Phenotype,
Proliferation and Extracellular Matrix in the
Forebrain Subependymal Zone

Resident Award Winner

L. Brannon Thomas
University of Tennessee
College of Medicine

1:00 PM **Adjourn**

Friday, November 3

SCIENTIFIC SESSION III

Moderators: **Julian T. Hoff and
Peter McL Black**

8:00 AM *Therapeutic Internal Carotid Artery Occlusions:
Use and Results in 77 Patients*
**Duke Samson, Thomas Kopitnik,
Hunt Batjer, Phillip Purdy**

8:20 AM *Surgical Treatment of Unruptured Cerebral
Aneurysms: Implications for Future Decisions
Regarding Conservative Treatment and
Endovascular Approaches*
Robert Solomon

8:40 AM *Classification and Treatment of Deep Vascular
Malformations of the Brain*
John Tew

9:00 AM *Urgent Surgery for Poor-Grade
Aneurysm Patients*
**Robert Breeze, Bryan Duke,
Glenn Kindt**

9:20 AM *Treatment of Stroke With Inhibitors
of Polyamine Metabolism*
**Kevin Cockroft, Malcolm Meistrell,
Peter Tonge, Gary Zimmerman,
Anthony Cerami, Kevin Tracey**

9:40 AM *Clips and Coils: Defining
Their Respective Roles*
**Bryce Weir, Nick Hopkins,
Neal Kassel**

Friday, November 3

10:00 AM *Blockade of Nitric Oxide Synthase by L-NAME Attenuates Brain Acidosis and Promotes NAD+Regeneration During Repetitive Focal Cerebral Ischemia*
Fredric Meyer, Robert Anderson

10:20 AM *Requiem for Meningioma Invasive Skull Base Surgery*
**L. Dade Lunsford,
Douglas Kondziolka,
Ann Maita, John Flickinger**

10:40 AM **Coffee Break**

SCIENTIFIC SESSION IV
Moderators: **Roberto Heros and
Ralph Dacey**

11:00 AM *Effect of Difluoromethylornithine Treatment on Ornithine Decarboxylase Activity and Brain Edema After Traumatic Brain Injury*
**Robert Dempsey, Mustafa Baskaya,
A. Muralikrishna Rao,
M. Renuka Prasad**

11:20 AM *Lethal Cranioccephalic Disproportion in Patients With Advanced Anemia and Calvarial Thickening*
**Setti Rengachary, Jeffrey Blount,
Deborah Heros, Susan Bauer,
Charles Truwit**

11:40 AM *The Miami Project--Scientific Achievements*
Barth Green, Richard Bunge

12:00 PM *100 Year Anniversary of X-ray Discovery*
Karl-August Bushe

Friday, November 3

12:20 PM **Presidential Introduction**
 John Van Gilder

12:30 PM **Presidential Address**
 David L. Kelly, Jr., M.D.

1:00 PM **Adjourn**

Saturday, November 4

SCIENTIFIC SESSION V
Moderators: **John Van Gilder and**
 Robert Grubb

8:00 AM *Is Thalamic Stimulation Better for Tremor*
 Than Thalamotomy?
 Ronald Tasker, Andres Lozano

8:15 AM *Dysarthria and Dysphasia After Stereotactic*
 Operations on the Thalamus: Case Report and
 Review of the Literature
 Ethan Taub, Andres Lozano,
 Ronald Tasker

8:30 AM *Blinded Assessment of Results of*
 Microelectrode Guided GPi Pallidotomy
 Andres Lozano, A. Lang, Ronald
 Tasker, N. Galvez-Jimenez,
 J. Miyasaki, J. Duff, F. Junn,
 M. Munz, J.O. Dostrovsky,
 W. Hutchison

8:45 AM *The Role of Irradiated Freeze-Drie Dura*
 Allografts in Postoperative
 Neurosurgical Infection
 Frank Rhame, Stephen Haines

Saturday, November 4

9:00 AM *Findings of the Multispecialty Task Force in
Persistent Vegetative State*
Clark Watts

9:15 AM *Management of Petrous Apex Cholesterol
Granulomas*
**Jason Brodkey, Jon Robertson,
Gale Gardner**

9:30 AM *Long-Term Morbidity and Mortality After
Transsphenoidal Surgery*
Nicholas Zervas, Brooke Swearingen

9:45 AM *Repeat Transsphenoidal Surgery in Recurrent
Acromegaly and Cushing's Disease*
Edward Laws, Jr., A.G. Chenelle

10:00 AM *Direct Evidence for Lateralization of
Prosody in the Right Hemisphere*
**Charles Hodge, Andrew Bragdon,
Charles Bradshaw, Mark Smith**

10:15 AM *A Proposed Comprehensive Grading Scale to
Predict Long-Term Outcome After Surgical
Management of Intracranial Aneurysms*
Christopher Ogilvy, Bob Carter

10:30 AM **Coffee Break**

SCIENTIFIC SESSION VI
Moderators: **Suzie C. Tindall and
William Buchheit**

11:00 AM *Biology of Radiation Effect on Vascular
Smooth Muscle*
Marc Mayberg, H. Richard Winn

Saturday, November 4

11:15 AM *Surgically Implanted Biodegradable Polymers
For the Treatment of Malignant Gliomas*
Henry Brem

11:30 AM *Incidence of Malignant Gliomas in Black
and White Patients*
**James Robertson, Brett Gunter, Grant
Somes**

11:45 AM *Genetic Differences Between Familial and
Sporadic Gliomas*
Dan Fufts

12:00 PM *Delivery of Gene Therapy to Brain Tumors*
**Griffith Harsh, IV, N.G. Rainov, R.
Weissleder, E.A. Chiocca,
X.O. Breakefield**

12:15 PM *Extended Subtotal Maxillotomy for
Management of Tumors of the Clivus*
Jon Robertson, Edwin Cocke

12:30 PM *Mechanisms of Leukocyte-Endothelial
Adherence During Postasphyxial Reperfusion*
Tae Sung Park, Jeffrey Gidday

12:45 PM *Bipolar Cutting*
Leonard Malis

1:00 PM *Adjourn*

Thursday, November 2

8:10 AM

Surgical Anatomy, Imaging and Results of the Treatment of the Chiari Malformation in Adults

C. Tator

The author has reviewed the surgical anatomy, imaging and results of treatment of 47 adult patients with Chiari malformation, 20 of whom have had posterior fossa decompression as the primary treatment. In every posterior fossa case, the locations of the brainstem and fourth ventricle as determined by imaging and surgery were compared. At surgery, the locations of the cerebellar tonsils, the obex and the lowermost segment of the fourth ventricle (identified by its ependymal lining) were determined in relationship to the foramen magnum. In all cases, the obex and the lowermost point of the fourth ventricle were found to be located at or below the level of the foramen magnum, proving that the tonsils and the brainstem were herniated through the foramen magnum in every case. Thus, all cases met the criteria of the Chiari II malformation. The obex and lowermost portion of the fourth ventricle were frequently incorrectly located on the basis of imaging. The results suggest that the Chiari malformations in adults are a spectrum of cases all with herniation of the brainstem of varying extent through the foramen magnum and with a variety of associated lesions such as hydrocephalus, syringomyelia and myelodysplasia. The results suggest that the Chiari I malformation does not exist in adults or is very rare. The results of decompression of the brainstem for symptomatic Chiari malformation in adults are excellent with amelioration of symptoms in almost all cases and with minimal morbidity and no mortality. In cases with associated syringomyelia, posterior fossa decompression was also usually effective for treatment of the syringomyelia.

NOTES

Thursday, November 2

8:30 AM

Anterior Thoracic Lumbar Plating

V. Sonntag, C. Dickman, A.G. Vishteh

Anterior thoracic lumbar plating in conjunction with bone grafting is an excellent way to stabilize the spine. The procedure has several advantages: immediate stabilization is achieved, a second posterior procedure is seldom needed, and fewer vertebral segments are fused than in a routine posterior rodding procedure. Twenty patients had an anterior thoracic lumbar plating with a follow-up of 6 to 28 months. Indications for plating were trauma (14), osteoporosis (3), infection (2), and tumor (1). No patient was made worse neurologically and all achieved a solid fusion. Three different systems were employed. The advantage and disadvantage of each will be discussed along with representative examples. From this initial experience, it appears that....In conclusion, anterior thoracic-lumbar plating is an excellent fixation technique for the unstable thoracic-lumbar spine.

NOTES

Thursday, November 2

8:50 AM

Value of Cervical Facet Wiring Following Laminectomy (A Biomechanical Study)

J. Cusick, F. Pintar, N. Yoganandan

Cervical laminectomy may cause increased flexibility and decreased strength of the adult cervical column with subsequent risk for instability or curvature alterations. These considerations suggest that under a variety of clinical situations that corresponding stabilization procedures may offer improved long-term results of laminectomy. To clarify the biomechanical effectiveness of such adjunctive procedures and avoid the controversies of multilevel lateral mass fixation, facet fixation procedures were studied. Methods: Thirteen fresh human cadaver spines from C2-T1 had flexion-compression loads applied through a custom designed apparatus. Kinematics were obtained from retroreflective targets in bony components. Sequential testing was done on intact, three-level laminectomized and fixated specimens. Fixation consisted of four categories of facet fixation (Callahan-Southwick, continuous wire, Luque rectangle and crisscross). Results: Laminectomy (C4-C6) caused decreased stiffness as compared to intact ($p < 0.05$), increased sagittal rotations ($p < 0.05$) with intact at 3.6 deg and laminectomy at 8.0 deg and higher rotations at every level. The classic facet fixation method (Callahan-Southwick) [4.9 0.6] and continuous facet wiring [3.9] failed to restore column strength with decreased stiffness even compared to laminectomized preparations [mean 6.4 0.8]. Luque rectangle (C2-C7) and crisscross wiring which through the joining of individual wires permits crossing of each facet joint significantly increased strength towards intact. [9.2 3.3 fixation to 7.9 0.8 for intact]. Segmental kinematics showed markedly decreased flexion from C4-C7 with the majority of motion at the C3-C4 level. Conclusion: The results show that multilevel laminectomy in the adult causes generalized significant increased flexibility and that the usual methods of facet fixation fails to restore stiffness with resultant potential exaggeration of post-laminectomy flexibility. Luque rectangles and crisscross wiring techniques, however, will significantly restore cervical column strength.

NOTES

Thursday, November 2

9:10 AM

The Myelopathy Disability Index - An Objective Outcome Measure for Spinal Surgery?

H.A. Crockard, A. Casey, M. Bland

Assessing the degree of **neurological** disability objectively in myelopathic patients with rheumatoid involvement of the cervical disease is complicated by the presence of a painful deforming arthritis that makes a conventional neurological examination nearly impossible.

Traditionally neurological grading of these patients has been performed using the Ranawat classification (I-IIIb). This however is a relatively crude system and fails to differentiate adequately between different degrees of disability. Its use as an outcome measure is therefore limited.

We have modified an existing functional disability scoring system (Stanford Health Assessment Questionnaire) using a statistical technique known as principal components analysis. The patient or their carer completes a questionnaire which is scored. The new scale has been mathematically validated and found to be a reliable instrument in a population of 194 patients undergoing surgery for rheumatoid involvement of the cervical spine. It accurately predicts neurological recovery and survival following surgery. This Myelopathy Disability Index may also be used for other conditions such as cervical spondylosis and, if accepted could form the basis of multicentre studies.

NOTES

Thursday, November 2

9:30 AM

Mapping Human Sensorimotor Cortex With Functional MRI

G. Cosgrove, B. Buchbinder, H. Jiang, B. Rosen

In 18 patients with structural lesions (13 tumors, 2 cavernous angiomas, 1 AVM, 1 cortical atrophy, 1 heterotopia) involving the primary sensorimotor cortex, fMRI was performed using a blood oxygenation level dependent (BOLD) contrast technique at 1.5 Telsa on a GE Signa scanner. T-2 weighted echo-planar images were obtained in a standard head coil during alternating epochs of rest and motor task performance. Motor tasks involved simple repetitive movements of the hands, feet or tongue depending on lesion location. Sites of functional localization were identified by image subtraction and co-registration with high resolution 3-D MRI and MRA. The integrated volume rendering of brain surface topography, cortical veins, structural lesion and sites of functional activation were then compared to the results of intraoperative cortical stimulation in all patients.

In each case, fMRI was able to define the somatotopic distribution of motor and sensory function along the central sulcus. fMRI localization was confirmed at surgery by direct electrical stimulation of the cortex to be accurate within 3 to 4 mm in all cases despite the presence of local brain pathology. The magnitude and extent of functional activation was attenuated in the hemisphere ipsilateral to the lesion in over half the cases, however.

We conclude that fMRI can accurately localize human sensorimotor cortex and may be useful in planning surgical resections of cortical lesions. Further studies will be necessary to establish the sources of variance with fMRI mapping techniques and to identify the physiologic causes of blunted activation.

NOTES

Thursday, November 2

9:50 AM

Integration of Stereotactic Robotic Guidance in Epilepsy Surgery

D. Roberts, T. Darcey, P. Williamson

RATIONAL: The identification and subsequent resection of a medically intractable seizure focus is an inherently stereotactic task. Application of co-registration techniques for preoperative planning and intraoperative guidance can facilitate and enhance our surgical capabilities.

METHODS: An established strategy integrating stereotactically placed intracranial electrodes, electrophysiologic studies, and co-registered imaging studies for surgical decision-making has been extended to incorporate a robotic stereotactic operating microscope system. Prospectively planned placement of intracranial subdural strip and grid electrodes, analysis of imaging and physiological findings, and execution of a resective plan have been performed using this frameless stereotactic guidance in a series of ten patients with medically intractable epilepsy. The accuracies achieved in electrode placement and in subsequent resection with respect to preoperative planning were quantitatively assessed by post-operative imaging.

RESULTS: Robotic guidance in the placement of intracranial subdural strip and grid electrodes enabled localization to within specific gyri, and lines of actual surgical resection were achieved with a comparable level of accuracy. Deviation from the surgical plan was attributable to both system error and intraoperative adjustments for gyral pattern and vascular structure. Efficiency in intraoperative localization of both seizure focus and functional cortex was also achieved.

CONCLUSIONS: Stereotactic guidance provided by a frameless, robotic surgical system enhances surgical accuracy in intracranial electrode placement and in execution of a preoperatively determined surgical resection plan.

NOTES

Thursday, November 2

10:10 AM

Image Guided Surgery: Experience with the Zeiss-MKM System

J. Ausman, M. Dujovny, F. Charbel, M. Alp, A. Goldberg

Forty-six (46) patients with pathology included pineal tumor, abscess, aneurysm, intraventricular AVM, interventricular tumors, cavernoma in the insula, glioblastomas, skull base meningiomas, pituitary tumors and seizure foci and optic nerve compression secondary to hyperostosis have been operated using the Zeiss-MKM computer guided microscope system. A variety of fiducial markings from tape applied markings to the scalp, skull implanted markers, to stereotactic fixation have been utilized with error of less than .8 mm. Image input included CT, spiral CT, MR, MRA information either singly or in combination. Targeted approaches to lesions normally felt to be difficult to operate without producing some deficit were utilized. Operations could be performed with progressively smaller craniotomies, utilizing surgical access through the sulcus, small corridors of dissection to reach the targeted lesion. Computer developed imaged sizes of the lesions appeared to be accurate. Projected hospital stays were shortened and morbidity seemed to be reduced. Projected cost savings under fixed payment systems were greater than that under fee for service. Utilization of the Zeiss-MKM system was for a much broader spectrum of neurosurgical pathology than stereotactic approaches to deep seated tumors.

The Zeiss-MKM system will provide a quantum leap in surgical armamentarium, enabling the surgeon to input anatomical, functional and chemical information from imaging modalities reducing morbidity, mortality and length of stay and providing superior results. Examples of a variety of cases in which the system has been utilized will be presented.

NOTES

Thursday, November 2
10:50 AM
Frameless Radiosurgery
W. Friedman, F. Bova

Historically, radiosurgery systems have relied upon rigid skull fixation (i.e. a head rings) for both the acquisition of stereotactic imaging data and for treatment delivery. The discomfort inherent to head ring application has led to an emphasis on single fraction, high dose treatments. From a radiobiological standpoint, single fraction treatment has some merit in the treatment of arteriovenous malformations. Multiple fraction treatment is, however, theoretically advantageous in the treatment of many tumors. This is especially true when the tumors are in close proximity to very radiosensitive structures, such as the optic apparatus. At the University of Florida, a frameless system has been developed for repeatedly positioning patients for fractionated stereotactic radiation treatment. The system is based upon a dental mold - bite plate device, onto which are attached multiple infrared light emitting diodes (IRLEDs). An infrared camera is mounted to the ceiling of the LINAC vault. A computer program has been devised such that the camera is able to detect the position of the IRLEDs. The computer then determines the patient's precise position. A padded thermoplast mask head holder attaches to the standard stereotactic head holder of the radiosurgery system. The computer updates the patient's position in real time as the frame is dialed into the precise target position. Extensive phantom testing, as well as testing of the bite plate positioner in patients undergoing traditional, rigid skull fixation radiosurgery, shows the system to have a mean error of .4 mm. The device is now being used on an investigational basis for patient treatment. Illustrative cases will be presented.

NOTES

Thursday, November 2

11:10 AM

Characterization of the Cerebral Hemodynamic Phases That Follow Head Injury: Hypoperfusion, Hyperemia and Vasospasm

N. Martin, M. Alexander, R. Patwardhan, C. Zane,
D. Becker, D. Hovda

Analysis of cerebral blood flow and transcranial doppler studies in more than 200 patients with acute closed head injury have defined 4 post-traumatic cerebral hemodynamic phases. These phases are characterized by distinct changes in global cerebral blood flow (CBF15), middle cerebral artery velocity (VMCA), and by measurements of cerebral arteriovenous difference in oxygen content (AVDO2).

Phase I (hypoperfusion phase) occurs during the first 24 hours after injury, and is defined by low cerebral blood flow, and normal middle cerebral artery velocity. The AVDO2 is normal or high. The hypoperfusion of this initial phase appears to be due to an increase in microcirculatory resistance, and not due to large artery spasm. Phase II (hyperemia phase, days 1-3) is characterized by increasing CBF and middle cerebral artery velocity, and falling AVDO2. There is, apparently, a degree of uncoupling between oxygen metabolism and cerebral blood flow during this hyperemia phase. During phase III (vasospasm phase, days 4-22) there is again a fall in cerebral blood flow, accompanied by a pronounced rise in middle cerebral artery velocity. The hemodynamic characteristics of this phase appear, in part, to be related to post-traumatic arterial spasm. Phase IV (resolution phase) is characterized by a gradual normalization of CBF and middle cerebral artery velocities. The effects of age, injury severity, and the presence or absence of intracranial hypertension on the post-traumatic hemodynamic patterns will be described.

This is the first study to combine an analysis of CBF and TCD measurements in order to delineate the distinct cerebral hemodynamic phases that follow craniocerebral trauma. The unique pathophysiology of each phase suggests the need for distinct, time-dependent treatment strategies tailored to the patients' prevailing hemodynamic condition.

NOTES

Thursday, November 2

11:30 AM

The Role of Reoperation After Radiosurgery for Glioblastoma Multiforme

E. Alexander, IV, P. Wen, H. Fine, P. Mcl. Black

As part of their treatment for glioblastoma, a total of 214 patients underwent stereotactic radiosurgery (SRS). Overall median actuarial survival for this group was 18 months post-diagnosis and 11 months post SRS. Following SRS, patients underwent enhanced CT or MRI every 3 months. 66 patients (30.8%) with exacerbation of neurologic symptoms related to increased edema and mass effect on CT and/or MRI, usually dependent on steroids, underwent reoperation. The median time between SRS and reoperation was 5.5 months (1 to 27). Actuarial risk for reoperation was 36.9% at 12 months and 47.7% at 24 months. Postdiagnosis and posttreatment median actuarial survivals for the 66 patients who underwent reoperation were 24 and 16 months, while median survivals for the 148 who did not undergo reoperation were 16 and 9 months ($p=0.046$ & 0.009).

Of the 66 patients who underwent reoperation, 50 were operated upon at our institution using stereotactic guidance, usually with dual isotope (Tl-201 and Tc-99m) SPECT analysis to assist in discriminating between tumor growth and necrosis. At the time of surgery, 14% (7) were neurologically intact, 30% (15) had mild deficits, 32% (16) had moderate deficits and 24% (12) had severe deficits or depressed level of consciousness. The average daily preoperative dexamethasone dose for these patients was 16.1 mg. Immediately following surgery, 23.4% of patients were neurologically improved and 21.2% of patients deteriorated. At follow-up 3 months after surgery, 70.3% of the surviving patients were neurologically at or above their preoperative status, with 9 deaths having occurred (4 patients alive with < 3 months of follow-up). The average daily dexamethasone dose for all surviving patients was 7.2 mg. at this time. At follow-up 6-12 months after surgery, 56.2% of living patients remained at or above their preoperative neurologic status, with an average daily dexamethasone dose for all

surviving patients of 4.5 mg. There were 26 deaths within 1 year and 8 patients alive with < 12 months follow-up.

Prolongation of survival following stereotactic high-dose radiation techniques for selected patients with glioblastoma has been previously reported. A significant number of patients deteriorate clinically following treatment. Our data suggest that reoperation is associated with and may, in fact, confer additional survival benefit for this group of patients. In general, patients undergoing reoperation enjoyed an improvement in quality of life and were able to be maintained on lower doses of steroids. We conclude that reoperation plays an integral part in the management of patients with glioblastoma who undergo SRS.

NOTES

Thursday, November 2

11:50 AM

Radiosurgery for Brain Metastasis: Patient Outcome and Tumor Response

B. Guthrie, R. Jenelle, R. Bishop

Linear accelerator stereotactic radiosurgery was used to treat 75 intracranial metastatic tumors in 56 consecutive patients. Treatment criteria included a KPR greater than 75%, and tumor size of 3cm or less. Followup was by clinical and imaging assessment at three month intervals. Outcome was assessed versus characteristics of patient (age, sex, KPR), tumor (histology, number, volume) and treatment (radiodosimetry), the number of tumors treated, use of WBRT, use of open surgery.

Patient diagnoses were non-small cell lung (22/56), melanoma (17/56), renal cell (5/56), other (12). Tumor location was frontal (23/75), parietal (15/75), temporal (14/75), occipital (7/75), cerebellar (7/75), other (9/75). The median radiosurgical dose was 20 cGy to the isocenter (80% isoline to the contrast margins). The median collimator size was 2.25cm (mean 2.34). Open craniotomy was used in 7 patients.

Currently, 57% of patients are alive, with a median survivor followup of 42 weeks. Causes of death included: radiosurgical failure (1/24), local surgical failure (2/24), carcinomatous meningitis (5/25), systemic disease (16/24). Four of the 5 patients with carcinomatous meningitis had craniotomies. Followup of tumor response indicates a mean decrease in volume by 28%. Thirty percent developed surrounding edema, half of whom required steroids. The patient who failed locally and all who required steroids had a tumor diameter of larger than 2.25cm.

We conclude that, in these patients, radiosurgical treatment significantly reduces the chance of death from cerebral disease. We also conclude that open surgery increases the risk of meningeal spread, therefore should be avoided if possible. Details of treatment and outcome will be discussed.

NOTES

Thursday, November 2
12:10 PM

**The Kjellberg Experience with Dose Escalation
Proton Radiosurgery for the Treatment of Large
Inoperable Arteriovenous Malformations**

P. Chapman, W. Butler, C. Ogilvy

The Harvard Cyclotron Laboratory (HCL) was completed in 1949. In 1961 Dr. Raymond Kjellberg treated the first patient at HCL, a 2_ year-old girl with a hypothalamic tumor. Subsequently, proton radiosurgery using the Bragg peak became commonplace. In 1965 he performed the first radiosurgical treatment of an AVM. Between 1972 and 1993 Dr. Kjellberg treated more than 1300 AVMs. In addition to demonstrating the usefulness of radiosurgery for the treatment of AVMs, a major contribution of his pioneering work was to explore the relationships between radiation dose, treatment volume, efficacy and complications. One of the more difficult problems he encountered was the management of large AVMs where the usual radiation doses were ineffective. In an effort to alter the natural history of these lesions, he explored the effect of dose escalation. The results of this experience will be described and their significance for present efforts to treat large inoperable AVMs by radiosurgery discussed.

NOTES

Friday, November 3

8:00 AM

Therapeutic Internal Carotid Artery Occlusions: Use and Results in 77 Patients

D. Samson, T. Kopitnik, H. Batjer, P. Purdy

CAROTID ARTERY SACRIFICE

Over an 11 year period, 77 patients have undergone iatrogenic internal carotid artery sacrifice for the management of a variety of cerebral vascular conditions. Over 90% of these patients harbored intracavernous or extradural aneurysms of the internal carotid artery and were subjected to a strict protocol of radiographic and hemodynamic evaluation, to include trial balloon occlusion, prior to elective sacrifice of the involved carotid artery. The risk of cerebral infarction associated with trial balloon occlusion in this patient population was 5%. Seven of the patients have undergone abrupt unplanned sacrifice of the internal carotid artery secondary to intraoperative complications during the management of ruptured intradural carotid aneurysms. None of these patients had preliminary trial balloon occlusion.

Cerebral infarctions related to carotid sacrifice occurred in six patients, all within 48 hours of the time of occlusion, and have been fatal in three. One half of these infarctions were in patients undergoing unplanned abrupt carotid artery ligation without preliminary testing or prior revascularization. The risk of unanticipated sacrifice of the internal carotid artery in the absence of preliminary testing, was very high (43%) and could not be predicted by preoperative angiographic findings. The risk of iatrogenic carotid occlusion in patients who tolerated preliminary trial balloon occlusion testing was low (5%) but three patients developed cerebral infarctions in the initial 48 hours following occlusion. None of these patients were anticoagulated, and in all the ischemic event appeared to have been embolic. None of the 67 patients available for follow-up have developed symptomatic ischemia in the distribution of the occluded artery following hospital discharge.

NOTES

Friday, November 3

8:20 AM

Surgical Treatment of Unruptured Cerebral Aneurysms: Implications for Future Decisions Regarding Conservative Treatment and Endovascular Approaches

R. Solomon

Recent advances in neuroimaging have made the identification of unruptured cerebral aneurysms common. Controversy abounds regarding the natural history and proper treatment of these lesions. This study was undertaken to determine the risks of surgical treatment of unruptured aneurysms and discuss appropriate cases for conservative treatment or endovascular approach.

A personal series of 809 aneurysm operations was reviewed: 307 operations were performed for unruptured cerebral aneurysms above the cavernous sinus. Mode of presentation, patient age, aneurysm size, aneurysm location and 3-month outcome were tabulated for each patient.

In 133 operations for aneurysms <11 mm in diameter, there were 4 patients (3%) that were dead or disabled at 3 month follow-up. For 99 operations for aneurysm 11-24 mm in diameter, there were 9 patients (9%) that were dead or disabled at 3 month follow-up. For 78 operations for aneurysms >24 mm in diameter, there were 17 patients (22%) that were dead or disabled at 3 month follow-up. Nine of these latter 17 patients had giant basilar aneurysms, and only 8 of 66 aneurysm operations (12%) done for aneurysms >24 mm not located on the basilar artery resulted in death or disability.

The conclusion of the study is that aneurysms <2.5 cm in diameter can be treated with operative morbidity significantly lower than the natural history of the disease left untreated. In these cases, location of the aneurysm and age of the patient do not seem to play a role in predicting poor outcome. The results of giant basilar aneurysm surgery have been poor, and improved methodology needs to be developed.

NOTES

Friday, November 3

8:40 AM

Classification and Treatment of Deep Vascular Malformations of the Brain

J. Tew, Jr.

Vascular malformations of the brain are classified according to pathology, location, and clinical grading. Pathology. Malformations classified according to pathology are arteriovenous malformations (AVMs), cavernous angiomas, capillary telangiectasis, or venous angiomas. Of these types, AVMs and cavernous angiomas cause most of the neurologic deficits in patients. Location. Vascular malformations classified according to location are either superficial (convexity) or deep. Deep malformations occur in the ventricles, basal ganglia, thalamus, and brain stem. Clinical grading. This grading is helpful in selecting appropriate therapies, which may be observation, embolization, surgical excision, or radiosurgery. We have developed a new clinical grading system, which uses magnetic resonance imaging (MRI) and neurologic examination, that more accurately predicts outcome. Results of treatment for deep vascular malformations will be discussed. In most circumstances, MRI is preferred to determine the lesion's pathology and location. Digital angiography is valuable to intraoperatively document the total removal of most vascular lesions, except cavernous malformations.

NOTES

Friday, November 3

9:00 AM

Urgent Surgery for Poor-Grade Aneurysm Patients

R. Breeze, B. Duke, G. Kindt

The practice of operating on sacular cerebral aneurysms early has become well established, particularly with good-grade patients. The timing of surgery in poor-grade patients, however, remains controversial. At the University of Colorado , we advocate emergency angiography followed immediately by definitive surgery in all cases of aneurysmal subarachnoid hemorrhage, regardless of grade, aneurysm size or location (moribund patients who fail to respond to aggressive resuscitation efforts are excluded). We have analyzed our results for the three-year period from 1992 to 1994. During that time 175 aneurysmal hemorrhages were managed, of which 27 were classified as grade IV on admission. Aneurysm location was distributed as follows: ACA -12, ICA -8, MCA -4 and VB -3. Fourteen aneurysms were less than 10mm in size, 11 were 10-25mm and two were greater than 25mm. Outcome was assessed at six months. Seven patients experienced a good outcome and six experienced a fair outcome, giving a total of 13 patients (48%) who returned to an independent lifestyle. Five patients had a poor outcome and nine (33%) died. Four of the deaths occurred prior to surgery and were due to rebleeding. One occurred in the immediate postoperative period and was due to an intraoperative stroke. Two deaths were ascribed to vasospasm, while two were due to the late complications of a vegetative state. These results compare favorably to those of other authors who advocate early surgery in poor-grade patients, and are vastly superior to previously reported results with delayed surgery.

NOTES

Friday, November 3

9:20 AM

Treatment of Stroke with Inhibitors of Polyamine Metabolism

K. Cockroft, M. Meistrell, P. Tonge, G. Zimmerman,
A. Cerami, K. Tracey

In stroke, cytotoxic factors produced in the ischemic brain kill neurones. Identification of candidate neurotoxins (glutamate, nitric oxide, and platelet-activating factor) has led to the development of experimental therapeutics to minimize stroke damage. The present studies now identify a class of neurotoxins formed by the enzymatic metabolism of polyamines. Polyamines, ubiquitous intracellular molecules (e.g. spermine), are released from dying ischemic cells. Diamine oxidase (DAO), an enzyme found in brain, serum, and other tissues, oxidizes polyamines into aldehydes which are cytotoxic. In studies of neuronal death in stroke, we initially discovered that aminoguanidine, a small molecule in clinical trials for diabetic complications, is neuroprotective. Aminoguanidine prevented neuronal death and significantly limited infarct size, even when given up to 2 hr after the onset of ischemia. Since aminoguanidine is a potent inhibitor of DAO, we hypothesized that its neuroprotective mechanism in stroke is attributable to inhibition of DAO enzyme activity. Our results now demonstrate that spermine, when stereotactically administered into the brain cortex, is neurotoxic; administration of aminoguanidine (320 mg/kg, i.p.) prevents brain necrosis after i.c. spermine. In cultured primary neurones, spermine toxicity depends upon DAO activity in the media, and two structurally distinct DAO inhibitors (aminoguanidine and chloroquine) prevent spermine-mediated neurotoxicity in vitro. Each of these DAO inhibitors (aminoguanidine and chloroquine) is effective in attenuating experimental stroke damage, even when administered after the onset of ischemia. These results give evidence that neurotoxins formed by DAO-catalyzed oxidation of spermine participate in the cytotoxicity of stroke. A clinical trial of the DAO inhibitor aminoguanidine in stroke is planned.

NOTES

Friday, November 3

9:40 AM

Clips and Coils: Defining Their Respective Roles

B. Weir, N. Hopkins, N. Kassel

It is anticipated that sometime in 1995 the Food and Drug Administration will authorize the introduction of detachable platinum coils as treatment for intracranial aneurysms. It is possible, because of the apparent ease of coil placement and the lack of long term follow-up data, that the clip may be displaced by market forces rather than scientific evidence. We feel that it is essential to have contemporary hard data on the safety and efficacy of these modalities in the treatment of all types of ruptured aneurysms. To this end, a data bank will be established similar to the one which was done for the cooperative timing of aneurysm surgery study. The analysis of this data may be used to plan a prospective randomized trial of clip versus coil in those circumstances where it would appear to be appropriate. The study and its rationale will be described.

NOTES

Friday, November 3

10:00 AM

Blockade of Nitric Oxide Synthase by L-NAME Attenuates Brain Acidosis and Promotes NAD⁺ Regeneration During Repetitive Focal Cerebral Ischemia

F. Meyer, R. Anderson

Techniques to provide intraoperative cerebral protection during neurovascular procedures which require temporary vessel occlusion are controversial. This study examined the effects of nitric oxide synthase (NOS) inhibition by N^w-nitro-L-arginine methyl ester (L-NAME) during repetitive focal cerebral ischemia with ischemic times chosen to mimic the neurosurgical setting. Forty-two New Zealand rabbits under halothane anesthesia were divided into 6 groups of 7 each: 1) non-ischemic controls; 2) ischemic controls; and 4 drug groups receiving 3) 0.1 mg/kg L-NAME; 4) 1 mg/kg L-NAME; 5) 10 mg/kg L-NAME; and 6) 1.0 mg/kg L-NAME + 5 mg/kg L-arginine. The ischemic paradigm was four 15-minute periods of MCA and ACA occlusion each separated by 5 minutes of reperfusion. Following the fourth ischemic insult, there was a final 180 minute period of reperfusion. Intracellular brain pH (pH_i), cortical blood flow (CBF), and NAD⁺/NADH were measured with in vivo fluorescence imaging. Administration of 0.1 and 1.0 mg/kg l-NAME significantly prevented brain acidosis and facilitated NAD⁺ regeneration during ischemia (p<0.05). In the 10 mg/kg group and in the combined l-NAME + L-arginine group, pH_i declined significantly during the first ischemic insult and remained acidotic. These effects were independent from cortical blood flow changes. In conclusion, low dose l-NAME prevents the development of brain acidosis and promotes NAD⁺ regeneration during repetitive focal cerebral ischemia. This observation suggests that nitric oxide is involved in pH_i regulation during focal cerebral ischemia and that the use of l-NAME may represent a pharmacological manipulation to provide intraoperative cerebral protection during neurovascular procedures.

NOTES

Friday, November 3

10:20 AM

Requiem for Meningioma Invasive Skull Base Surgery

L. D. Lunsford, D. Kondziolka, A. Maitz, J. Flickinger

Appropriate outcome goals for management of difficult skull base meningiomas include tumor control, minimal morbidity, rapid return to work, and reduced hospital stay. We assessed the role of radiosurgery instead of or after microsurgery (65%) for invasive skull base meningiomas. In an eight year interval, 200 patients (mean age=57) underwent stereotactic radiosurgery for meningiomas, the majority of which were located in the skull base (cavernous sinus = 51, petrous apex = 29, petroclival = 13, foramen magnum = 3, parasellar = 4, sphenoid wing = 8). Radiosurgical principles included volumetric conformal dose planning and reliance on high resolution intraoperative and postoperative imaging. At two years, the prevention of further tumor growth control rate was 95% with 35% of patients undergoing definitive volumetric tumor regression. In contrast to patients (who had a more than 60% chance of developing a new neurological deficit after skull base microsurgery) fewer than 5% of patients developed new neurological deficits after radiosurgery. Skull base radiosurgery represents a primary management strategy for patients with relatively small volume meningiomas located in difficult skull base regions, and a secondary management strategy done in conjunction with initial surgical cytoreductive efforts. Staged surgical and radiosurgical procedures offer a patient option that significantly reduces risks and enhances long term outcomes.

NOTES

Friday, November 3

11:00 AM

Effect of Difluoromethylornithine Treatment on Ornithine Decarboxylase Activity and Brain Edema After Traumatic Brain Injury

R. Dempsey, M. Baskaya, A. Rao, M. Prasad

We examined the effect of difluoromethylornithine (DFMO) on regional activities of ornithine decarboxylase (ODC) and edema formation in cortices and hippocampi after a lateral controlled cortical-impact (CCI) injury in rats. To measure the activity of ODC, the brains of injured and control rats were frozen in situ at 30 min, 6, and 24 h after CCI brain injury. Regional specific gravity, an indicator of edema formation, was examined in decapitated animals at corresponding time points. Brain injury induced significant increases of ODC in the ipsilateral hippocampus, adjacent and injury-site cortices as well as contralateral cortex and hippocampus at 6 hours after injury. ($p < 0.05$) No significant edema formation was found in any brain region at 30 min after injury. A significant edema formation was found in all regions ipsilateral to the injury-site at 6 hours which persisted at 24 hours. ($p < 0.05$) A significant but less severe edema was also found in the contralateral cortex and hippocampus at 24 hours. DFMO abolished the increase in ODC in all regions, it attenuated edema formation in the adjacent cortex, and the contralateral cortex and hippocampus. ($p < 0.05$) These findings indicate that polyamines may play a role in traumatic brain edema formation, particularly in brain regions remote from the injury-site.

NOTES

Friday, November 3

11:20 AM

Lethal Craniocephalic Disproportion in Patients With Advanced Anemia and Calvarial Thickening

S. Rengachary, J. Blount, D. Heros, S. Bauer, C. Truwit

The Monroe Kellie hypothesis dictates that any increase in the volume of intracranial contents such as from a hematoma or neoplasm is compensated by a corresponding decrease in the volumes of spinal fluid and venous blood. This compensatory mechanism ultimately fails leading to profound increase in intracranial pressure and ultimately death.

Rare causes of increased intracranial pressure include primary diseases of the skull. A notable example familiar to all neurosurgeons is craniosynostosis.

Recently we encountered two patients with severe advanced anemia with extensive hypertrophy of the diploic space of the skull leading to thickening of the cranium and a corresponding decrease of the volume of the cranial cavity. We encountered these patients in the terminal stages of their disease when the intracranial volume had reached critical proportions resulting in severe increase in the intracranial pressure and brain herniation resulting ultimately in death. Such a clinical phenomenon in advanced anemia has never been described in the past.

Detailed analysis of clinical features, brain imaging studies and autopsy findings will be presented.

NOTES

Friday, November 3

11:40 AM

The Miami Project--Scientific Achievements

B. Green, R. Bunge

The Miami Project to Cure Paralysis was established in 1985 with support from the University of Miami Department of Neurological Surgery, generous philanthropic gifts, and with a determination to find more effective treatment for patients with spinal cord injury. The Project has grown to encompass 15 laboratory groups, with expertise ranging from molecular biology to clinical physiology and rehabilitation, and including a comprehensive study of human spinal cord pathology and physiology. This unique group of basic and clinical scientists concentrating on a single clinical entity has:

- 1) shown that central cord syndrome may result from bilateral damage to the lateral corticospinal tract at the cervical level, rather than central cord cavitation;
- 2) developed a new method for stimulus-evoked EMG monitoring during transpedicular lumbosacral spine instrumentation that reliably detects screw misplacement and decreases complications;
- 3) documented that assisted ejaculation and fertilization can allow many spinal cord injured men to successfully father children;
- 4) demonstrated that a central pattern generator for locomotion can be detected in selected cases of human spinal cord injury;
- 5) established that axons of the long tracts of the spinal cord can be induced to regenerate by a favorable cellular environment at the site of injury (for example, transplantation of Schwann cells to a midthoracic spinal cord injury);
- 6) devised reliable methods for the isolation and cultivation of Schwann cells from adult human nerve;
- 7) generated neuronal cell lines by genetic manipulation and established that some lines can express neuronal morphology appropriate to the brain region into which they are transplanted. This work and other studies will be presented.

NOTES

Saturday, November 4

8:00 AM

Is Thalamic Stimulation Better for Tremor Than Thalamotomy?

R. Tasker, A. Lozano

Thalamotomy is a well established treatment for Parkinsonian tremor. Since stimulation-induced tremor arrest is a good indicator for the lesion site, chronic stimulation has also been used, more extensively since good equipment has become available. Our experience with approximately 30 cases will be presented. It is our conclusion that stimulation is more effective than thalamotomy in Parkinson's disease since tremor recurrence (20% of thalamotomies) and the 3 "cerebellar" complications (dysarthria, ataxia, gait disturbance significant in 5% of thalamotomies) can usually be circumvented by changing the choice of electrode pole stimulated and/or the parameters used. The risk of hematoma remains the same and that associated with chronic implant are added. Limited experience with cerebellar tremor will also be presented.

NOTES

Saturday, November 4

8:15 AM

Dysarthria and Dysphasia After Stereotactic Operations on the Thalamus: Case Report and Review of the Literature

E. Taub, A. Lozano, R. Tasker

Stereotactic thalamotomy and thalamic stimulation are being performed with increasing frequency for the treatment of movement disorders and intractable pain. It is important to characterize the potential adverse effects of these operations and how they correlate with the location, size and uni- or bilaterally of the lesions, so that surgery may be performed as safely as possible and with realistic expectations of outcome. In particular, post-thalamotomy dysarthria and dysphasia have been reported with incidence as high as 60% and 15%, respectively. We present illustrative cases from The Toronto Hospital and review more than 20 previous neurosurgical reports. Dysarthria is more common with bilateral than with unilateral lesions, and dysphasia (generally of the transcortical motor type) is more common with lesions in the dominant hemisphere. Both tend to improve over time, but the residual deficits may be severe. The localization of speech and language functioning to specific thalamic nuclei is only partially understood.

NOTES

Saturday, November 4

8:30 AM

Blinded Assessment of Results of Microelectrode Guided GPi Pallidotomy

A. Lozano, A. Lang, R. Tasker, N. Galvez-Jimenez, J. Miyasaki, J. Duff, F. Junn, M. Munz, J. Dostrovsky, W. Hutchinson

The major motor disturbances in Parkinson's disease are thought to be caused by an overactivity of the internal segment of the globus pallidus (GPi), in large part due to excessive drive from the subthalamic nucleus. The excessive inhibitory activity of GPi is thought to "brake" the motor thalamus and the cortical motor system to produce the slowness, rigidity and poverty of movement characteristic of parkinsonian states. To test the hypothesis that directly reducing the activity of GPi in patients with Parkinson's disease can improve motor function, we have studied the effect of GPi pallidotomy in 50 patients and present the blinded 6 month follow up results in the first 14 consecutive patients. Patients were evaluated with the Unified Parkinson's Disease Rating Scale (UPDRS) and timed motor tests preoperatively and at 3 month intervals post-operatively. The location of the GPi nucleus was confirmed using microelectrode recording criteria prior to lesioning. Standardized video tape evaluations were randomized and scored by a "blinded" evaluator. 6 months after surgery, the total UPDRS motor score while "off" had improved by 30% and the total akinesia score by 33%. The gait score in the off state, improved 15%. After the surgery there was an almost total elimination in drug induced involuntary movements (dyskinesias), with a 92% reduction on the side contralateral to the pallidotomy. There were no statistically significant improvements in motor function in the "on state". No patient suffered significant visual or corticospinal complications. We conclude that lesioning the GPi in patients with Parkinson's disease improves motor performance, reduces akinesia, improves gait and eliminates the neural elements responsible for L-Dopa induced dyskinesias.

NOTES

Saturday, November 4

8:45 AM

The Role of Irradiated Freeze-Dried Human Dura Allografts in Postoperative Neurosurgical Infection

F. Rhame, S. Haines

Background: Freeze-dried irradiated human dura is often used as a dural substitute. Because it is a foreign body, there is concern that its use may predispose to infection at the operation site. This study was carried out to test the hypothesis that the use of this dural substitute predisposes to postoperative wound infection.

Method: To carry out this case-control study, all patients undergoing neurosurgical operations with the implantation of freeze-dried irradiated dura between January 1986 and September 1993 were identified from operating room records. Two controls matched for procedure surgeon and year of surgery were identified from surgical logs. Crude infection rates in cases and controls were calculated and then adjusted for factors potentially influencing infection rate.

Results: 285 cases utilizing dural substitute and 570 controls were identified. Matching produced groups comparable in procedure, year of surgery, gender, age and duration of operation. Although there was a statistically significant difference in crude infection rate suggesting an increased risk when dural substitute was used (3.7% vs 1.2%, less than .005), adjustment for age and ASA class eliminated significant difference (relative risk 2.0, 95% CI .90-4.3).

Conclusions: The use of freeze-dried irradiated human cadaver dura does not significantly increase the risk of postoperative infection in neurosurgery.

NOTES

Saturday, November 4

9:00 AM

**Findings of the Multi-Specialty Task Force on
Persistent Vegetative State**

C. Watts

The author was a member of the Multi-Specialty Task Force on Persistent Vegetative State as a representative of the American Association of Neurological Surgeons. Other societies represented on the Task Force were the American Academy of Neurology, Child Neurology Society, American Neurological Association and the American Academy of Pediatrics. Through a process of literature search, consensus conferences and review of outside consultants, the task force examined the current knowledge of the medical aspects of the persistent vegetative state in adults and children and reached a consensus position. Defined were such matters as epidemiology, causes and clinical course, pathologic features, prognosis for recovery, survival, the issue of pain and suffering and treatment. The findings of the Task Force will be summarized, and the recommendations the neurosurgeon may use in counseling will be provided.

NOTES

Saturday, November 4

9:15 AM

Management of Petrous Apex Cholesterol Granulomas

J. Brodkey, J. Robertson, G. Gardner

Cholesterol granulomas of the petrous apex represent a unique class of temporal bone lesions. Different from cholesteatomas, which are epithelial lined cysts containing desquamated keratin, and epidermoid cysts which are considered an embryonal tumor, a cholesterol granuloma is a lesion thought to arise secondary to interference with drainage of the pneumatized petrous apex. These trapped air cells, by a cascade of events including hypoxia with resultant mucosal edema, hemorrhage and release of hemorrhagic contents (e.g. cholesterol crystals) produces the granuloma. In this paper we will present a series of 17 cholesterol granulomas managed over the past fourteen years by the two senior authors (J.R., G.G.). Of these, 5 were managed non-operatively by us with no progression of symptoms. One patient went to another center for surgery. We managed eleven surgically by drainage procedures. Approaches depended on location of the lesion and the presence, or absence of hearing. Eight patients had a transmastoid/infralabyrinthine procedure with simple drainage into the mastoid cavity. Other procedures included infracochlear, translabyrinthine and transcanal/transcochlear drainage procedures. In no case did we attempt removal of the granuloma. One case was previously operated at another center where an attempt at removal, through a middle fossa approach, was unsuccessful.

We will discuss presentation, otological data, surgical decision making, follow-up data and complications. Though some advocate removal of the granuloma, our data supports drainage with resultant aeration of the lesion is an optimal management strategy.

NOTES

Saturday, November 4

9:30 AM

Long-Term Morbidity and Mortality After Transsphenoidal Surgery

N. Zervas, B. Swearingen

We have attempted to determine the long term morbidity, early and late mortality, and employment disability following transsphenoidal procedures in over 1000 patients. There was one immediate postoperative death. The majority of patients died from causes unrelated to their pituitary disease, most commonly cardiac or neoplastic. Deaths related to pituitary disease were infrequent, but occurred in cases of giant invasive macroadenomas and one case of pituitary carcinoma.

The vast majority (over 80%) of patients were able to continue in their previous employment. Few patients were disabled by their disease: primarily from persistent headaches, pre-existing visual deficits, or from complications of Cushing's disease.

These results confirm that the morbidity and mortality from transsphenoidal surgery over the long-term is low; precise statistics will be summarized. The low disability rates suggest that, with appropriate endocrine management, pituitary disease is compatible with a full and productive life.

NOTES

Saturday, November 4

9:45 AM

Repeat Transsphenoidal Surgery in Recurrent Acromegaly and Cushing's Disease

E. Laws, A. Chenelle

Although transsphenoidal (TS) surgery is the procedure of choice for the initial management of acromegaly and Cushing's disease, recurrence of endocrinopathy occurs in 8-12% of patients followed for 10 years. Because failure to obtain a remission after transsphenoidal surgery is generally thought to represent incomplete tumor removal, some patients in this category are considered to be candidates for transsphenoidal re-exploration.

We retrospectively reviewed 29 patients with acromegaly and 29 patients with Cushing's disease who underwent repeat transsphenoidal surgery for recurrent or residual tumor. New remissions were seen in 48% of acromegalics and 41.4% of patients with Cushing's disease. Failures were attributed to the inability to remove all invasive tumor cells. The complication rate for second TS surgery was slightly higher than for initial attempts.

Secondary TS surgery for recurrent or persistent endocrine active pituitary tumors requires:

- 1) an anatomic tumor target on MRI scan
- 2) no obvious signs of tumor invasion
- 3) meticulous operative techniques

The principles of secondary TS surgery, methods of optimizing results, and long-term follow-up and management are also discussed.

NOTES

Saturday, November 4

10:00 AM

Direct Evidence for Lateralization of Prosody in the Right Hemisphere

C. Hodge, Jr., A. Bragdon, C. Bradshaw, M. Smith

Prosody is the emotional, non-verbal content of speech conveyed by facial expression as well as the rate, rhythm and inflection of verbal delivery. Much of the meaning speech is found within the prosody of a phrase, rather than the words of the phrase. The current study was undertaken to determine if prosodic aspects of speech and facial expression are lateralized to the right hemisphere. Eight patients undergoing temporal lobectomy for seizure disorder, under local anesthesia, were studied. Four of the patients underwent right and four patients left sided craniotomy. The patients were presented first with previously recorded phrases with clear prosodic components of anger, happiness, sadness and surprise, and then with cartoons depicting facial expressions showing the same emotions. The patients were asked to characterize the emotional content of the speech or facial expression. A variety of right and left perisylvian areas were stimulated during this task. In none of the left sided patients was stimulation able to disrupt the correct interpretation of emotional content. Stimulation of the right posterior superior temporal gyrus and temporal parietal junction disrupted prosodic interpretation in three right handed but not in the single left handed patient. The topography of the active sites suggests that prosody is organized in the right hemisphere in a manner analogous to organization of verbal speech in the left hemisphere. Consideration should be given to disruption of prosody during right sided cranial procedures.

NOTES

Saturday, November 4

10:15 AM

A Proposed Comprehensive Grading Scale to Predict Long-Term Outcome After Surgical Management of Intracranial Aneurysms

C. Ogilvy, B. Carter

In reviewing clinical outcome of aneurysm patients treated using modern microneurosurgical techniques and intensive care unit management, we have found that classification systems based only on the clinical assessment of the patient (e.g. Hunt and Hess grading scale or WFNS grading scale), groups outcome into a bimodal distribution without significant stratification or outcome between low (0,1,2,3) and high (4,5) grade patients. Furthermore, patients with unruptured aneurysms are inadequately stratified using these grading systems. A more comprehensive grading system which is easy to use yet incorporates age of patient, size of aneurysm, location of aneurysm, as well as severity of subarachnoid hemorrhage (if present) along with clinical condition has been developed.

We performed multivariate logistic regression of several factors suspected to be associated with long-term outcome in a series of 318 aneurysm surgeries performed in patients with both unruptured and ruptured aneurysms. We found four factors that were each independently associated with long-term outcome in the multivariate model: Hunt and Hess grade (grade 0,1,2,3 vs 4,5), Fisher Score (0,1,2 vs 3,4), patient age and size of aneurysm. In this initial review, location of lesion (anterior vs posterior circulation) was not found to influence outcome significantly. This information was used to develop a four point scale which accurately predicts (ANOVA overall and between group differences; highly significant) long-term outcome (follow-up 6 months-5 years). This new grading scale is easy to apply, separates patients into groups with markedly different outcomes, and is comprehensive, allowing for prediction of outcome in both unruptured and ruptured cerebral aneurysms. A test of the scale on a larger number of patients is currently in progress.

NOTES

Saturday, November 4

11:00 AM

Biology of Radiation Effect on Vascular Smooth Muscle

M. Mayberg, H. Winn

Restenosis due to intimal hyperplasia complicates 20%-50% of vascular procedures including endarterectomy, bypass grafting and angioplasty. To demonstrate the effect of gamma radiation on intimal hyperplasia, a single dose of 0-2000 cGy gamma radiation was delivered to the right carotid artery in rats at intervals from 5 days before to 5 days after bilateral carotid balloon catheter injury. At 20 days after injury, radiation produced a dose-dependent reduction in intimal area, with 50% decrease at approximately 750 cGy. There was a time-dependent radiation inhibition of intimal hyperplasia for radiation administered before or after injury, with the greatest effect noted within 24 hours before or after injury. For rats sacrificed at 3-12 months after injury, there was persistent inhibition of intimal hyperplasia without histologic evidence of radiation injury. To determine the cellular mechanisms by which radiation inhibits smooth muscle cell (SMC) proliferation, quiescent rat aortic SMCs in plasma were fed with whole blood serum to stimulate synchronous proliferation and irradiated with doses of 0-2000 cGy from 1 to 7 days after stimulation. Radiation caused a dose-dependent inhibition of SMC growth, with an ED50 at 500 cGy. Similar to in vivo experiments, the growth inhibition was time-dependent; growth rates in irradiated cells approached control values by 4 days after treatment. A micronucleus assay demonstrated a dose-dependent increases in chromosomal damage which correlated with cell killing determined by clonogenic assay. Inhibition of SMC growth by radiation did not correlate with changes in intra- or extracellular mitogenic activity, and there was no DNA ladder formation, suggesting that apoptosis did not occur. These data suggest that external gamma radiation persistently inhibits intimal hyperplasia in a time- and dose-dependent manner, probably through DNA damage and repair in SMC's.

NOTES

Saturday, November 4

11:15 AM

Surgically Implanted Biodegradable Polymers for the Treatment of Malignant Gliomas

H. Brem and the Polymer-Brain Tumor Treatment Group

Prolonged direct exposure of chemotherapeutic agents to brain tumors has been achieved by incorporating the drugs into sustained release biodegradable polymers. The drug-impregnated polyanhydride implants are placed directly at the tumor site. A prospective, randomized, placebo-controlled study demonstrated that treating recurrent malignant gliomas with biodegradable polymers impregnated with carmustine was safe and effective (LANCET, 345:1008-12, 1995). A subsequent Phase I trial demonstrated that interstitial chemotherapy with these polymers was safe when used as the initial therapy for newly diagnosed malignant gliomas. Biodegradable polymers can be utilized to deliver a wide spectrum of chemotherapeutic agents. We have found that chemotherapeutic drugs such as carboplatin, 4-hydroxycyclophosphamide, camptothecin and taxol can be effectively delivered intracranially in rats. Furthermore, steroids, antiangiogenic agents, immunotoxins and cytokines are effectively delivered by polymers.

NOTES

Saturday, November 4

11:30 AM

Incidence of Malignant Gliomas in Black and White Patients

J. Robertson, B. Gunter, G. Somes

A retrospective review of the adult (age > 15) glioma patients seen at our institution from July 1, 1984 to June 30, 1994, an era of modern neuroimaging, was performed. 823 patients were identified with histologically confirmed disease. 480 (58%) patients had grade IV astrocytoma, 228 astrocytoma I-III, 3 pilocytic astrocytoma, 8 astroblastoma, 66 oligodendroglioma, 15 ependymoma, 12 ganglioglioma and 10 gliosarcoma. The mean age was 53.6 years with an age range of 15.01 to 92.9 years and standard deviation of 17.7 years. The female:male ratio was 1.0:1 with 412 female patients and 411 male patients. 729 patients were white and 94 patients were black. The overall white:black case ratio was 7.7:1. Using U.S. census statistics and the home zip codes for the patients the population for the referral area was estimated to have a white:black population ration of 1.6:1. Kaplan-Meier survival curves were created for patients with glioblastoma multiforme. Using the Wilcoxin method, $p = 0.01$ when a comparison of the survival curves for white and black patients was made. The man survival for black patients with glioblastoma was 9.5 months and 11.5 months for white patients. The man age of diagnosis for black patients with glioblastoma was 60.7 years and 60.2 years for white patients. The white:black case ratio for glioblastoma multiforme was 9:1, oligodendroglioma 8.4:1, astrocytoma 6.8:1 and ependymoma 4:1. We conclude that the incidence of gliomas in the black population of this area is lower than would be predicted from the general population. In addition, the difference in the survival curves for black and white patients with glioblastoma multiforme is statistically significant, which may indicate that the biology of the tumors is different in white and black patients.

NOTES

Saturday, November 4

11:45 AM

Genetic Differences Between Familial and Sporadic Gliomas

D. Fults

Tumors arising in cancer-prone families result from inherited, germ-line mutations in various genes controlling cell growth. DNA markers that detect sequence polymorphism within the human population have enabled cancer geneticists to track variant markers in these cancer families and eventually to isolate the disease-causing genes. Success stories include the P53 gene in Li-Fraumeni syndrome, NF1 in von Recklinghausen's neurofibromatosis, APC in familial adenomatous polyposis coli and MTS1 in familial melanoma. Although the fundamental cause of CNS cancer is also genetic, few brain tumor cases appear to be inherited. An important question is whether brain tumors result from somatic mutations in the same genes that segregate in cancer families or in a different set of cell growth control genes. To address this question, we carried out DNA sequence analysis of four genes known to cause hereditary cancer syndromes (P53, NF1, APC, MTS1) in patients with sporadic gliomas. We found various types of gene mutations in tumor cells but not in peripheral blood leukocytes from affected patients. Mutation frequencies were much lower in these sporadic tumors than reported in familial cases. These results indicate that the majority of brain tumors in adults result from acquired, somatic mutations in cell growth control genes and not from inherited, germ-line mutations. In addition, the specific chromosome deletions that occur in sporadic gliomas indicate that other genes participate in brain tumor formation. Whether these genes are involved in both familial and sporadic cancers must await their isolation.

NOTES

Saturday, November 4

12:15 PM

Extended Subtotal Maxillectomy for Management of Tumors of the Clivus

J. Robertson, E. Cocke

From 1988 to 1995 an extended subtotal maxillectomy was utilized in the management of 29 patients having pathology which involved the clival area of the skull base. This series of cases encountered included: chordoma (9), meningioma (6), prolactin-secreting tumor (2), chondrosarcoma (2), juvenile angiofibroma (2), chondromyxoid fibroma (1), fibrosarcoma (1), epidermoid carcinoma (1), fibrous dysplasia (1), cranial cervical junction abnormality (3), and meningoencephalocele (1).

There were 17 males and 12 females in the patient population with an age range of 15-69 years. The mean duration of follow-up for this group of patients was 3.4 years.

In the patient group with tumors of the clivus, gross total resection was accomplished in 19/24 cases (79%). The cases of fibrous dysplasia and the meningoencephalocele were easily managed. Those individuals (3) with brain stem or upper cervical spinal cord compression from craniocervical junction abnormalities were decompressed anteriorly followed by posterior stabilization.

The outcome of treatment in this patient population is listed: A. perioperative mortality 2/29 (7%), one patient died of a massive pulmonary embolus, and the other of an acute myocardial infarction, B. new cranial nerve deficits 2/29 (7%), transient VI cranial nerve palsies, C. CSF leakage 2/29 (7%) each managed by lumbar drainage only, D. meningitis 1/29 (3.5%) managed with antibiotic therapy, E. other neurologic deficits 2/29 (7%), a transient motor paresis, transient worsening of preoperative quadriparesis, F. eustachian tube dysfunction 2/29 (7%), managed with ventilation tube placement, G. gingival breakdown 1/29 (3.5%), healed secondarily, H. loss of teeth 1/29 (3.5%), two teeth lost required correction with a dental bridge.

This presentation represents the largest reported series of cases involving the clival area of the skull base managed with an extended subtotal maxillotomy. The major advancement recognized in this approach has been the significant reduction of complications and improved functional outcome. A discussion of surgical technique, recurrence-free survival rates for tumors treated, and the use of radiotherapy in selected cases is planned.

NOTES

Saturday, November 4

12:30 PM

Mechanisms of Leukocyte-Endothelial Adherence During Postasphyxic Reperfusion

T. Park, J. Gidday

Leukocytes may contribute to brain injury in stroke. To evaluate mechanisms underlying the adherence of leukocytes to cerebral pial venules, newborn piglets were outfitted with closed cranial windows. Leukocytes were labelled with rhodamine 6G and venular endothelial adherence of leukocytes was quantified by videomicroscopy during reperfusion following 9 min asphyxia. In this study, we tested hypothesis that pretreatment 0.5 h before asphyxia with superoxide dismutase and catalase would reduce leukocyte adherence during reperfusion following asphyxia.

Results are as follows:

	1 h reperfusion	2 h reperfusion
Non-asphyxia (n=4)	-1±1	-1±2
Asphyxia (n=6)	29±5*	56±8*
SOD + CAT (n=5)	11±3*†	20±9†

* p<0.05 vs. baseline and non-asphyxic control group;

†p<0.05 vs. asphyxia

These results indicate that the cerebroprotective effects of free radical scavengers in cerebral ischemia may result in part from their inhibition of leukocyte adherence and subsequent parenchymal injury.

Supported by 2R01NINDS21045-12.

NOTES

Saturday, November 4
12:45 PM
Bipolar Cutting
L. Malis

Since 1982 I have been using bipolar cutting current for coring of tumors, first with my CMC-2 instrument using sharp pointed regular bipolar forceps. Since 1991 I have used my CMC-3 generator, which has significant advantages over the CMC-2. Ring bipolar forceps were then introduced, and this technique became my main method of tumor resection. The method had reduced operative time on relatively equivalent tumors by more than one third. Due to its characteristic hemostasis it has virtually obviated the need for transfusions even in major removals.

Unlike unipolar cutting, there is virtually no tissue heating and no current spread. It may be used in spinal intramedullary neoplasms or adjacent to vessels or the brain stem without damage. The electrosurgical principles will be briefly discussed and a four minute surgical videotape will be shown.

NOTES

GUEST

**Eben Alexander, III
Boston, MA**

**Charles Branch
Winston-Salem, NC**

**Robert E. Breeze
Denver, Colorado**

**Henry Brem
Baltimore, MD**

**Jason A. Brodkey
Memphis, TN**

**Rees Cosgrove
Boston, MA**

**Joseph Cusick
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Paul M. Lin	1955
Hubert L. Rosomoff	1956
Byron C. Pevehouse	1957
Norman Hill	1958
Jack Stern	1959
Robert Ojemann	1960
Lowell E. Ford	1962
Charles H. Tator	1963
Earle E. Crandall	1964
Stephen Mahaley, Jr.	1965
Chun Ching Kao	1966
John P. Kapp	1967
Yoshio Hosobuchi	1968
Gary G. Ferguson	1970
Richard L. Pressley	1971
David G. McLone	1972
Arden F. Reynolds, Jr.	1973
Richard L. Rapport	1974
Andrew G. Shetter	1975
John R. Howe	1976
Howard W. Blume	1977
Howard J. Senter	1978
Elisabeth M. Post	1979
David Dubuisson	1980
Dennis A. Turner	1981
Marc R. Mayberg	1982
David S. Baskin	1983
Kevin J. Kiwak	1984
Terry Lichtor	1985
Michael G. Nosko	1986
Joseph R. Madsen	1987
James T. Rutka	1988
Christopher D. Heffner	1989
Scott I. Gingold	1990
Mary Louise Hlavin	1991
Adam P. Brown	1992
Michael Tymianski	1993
David Garrett, Jr.	1994
L. Brannon Thomas	1995

The Neurosurgeon Award Winners

Edwin B. Boldrey	1955
Georgia and John Green	1956
Dean Echols	1957
Arthur R. Elvidge	1958
John Raaf	1959
Rupert B. Raney	1960
R. Glen Spurling	1961
Hannibal Hamlin	1962
Frank H. Mayfield	1963
Francis Murphey	1964
The Ladies	1965
David L. Reeves	1966
Eben Alexander, Jr.	1967
Donald D. Matson	1968
Henry Schwartz	1969
Guy L. Odom	1970
William F. Meacham	1971
Richard L. DeSaussure, Jr.	1972
James G. Galbraith	1973
Lyle A. French	1974
Charles G. Drake	1975
Robert Pudenz	1976
William Sweet	1977
Robert B. King	1978
C. Hunter Shelden	1979

Meetings of the Academy

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

Southampton Princess Hotel, BermudaNovember 6-9, 1974
 The Wigwam (Litchfield Park), Phoenix,
 ArizonaNovember 5-8, 1975
 Mills Hyatt House, Charleston,
 South CarolinaNovember 10-13, 1976
 Mauna Kea Beach Hotel, Kamuela, Hawaii.....November 2-5, 1977
 Hotel Bayerischer Hof, Munich, GermanyOctober 22-25, 1978
 Hyatt Regency, Memphis, Tennessee November 7-10, 1979
 Waldorf Astoria, New York City October 1-4, 1980
 Sheraton Plaza, Palm Springs, CaliforniaNovember 1-4, 1981
 Ritz-Carlton Hotel, Boston, MassachusettsOctober 10-13, 1982
 The Lodge at Pebble Beach, CaliforniaOctober 23-26, 1983
 The Homestead, Hot Springs, VirginiaOctober 17-20, 1984
 The Lincoln Hotel Post Oak, Houston,
 TexasOctober 27-30, 1985
 The Cloister, Sea Island, GeorgiaNovember 5-8, 1986
 Hyatt Regency, San Antonio, TexasOctober 7-10, 1987
 Omni Netherland Plaza, Cincinnati, Ohio September 13-17, 1988
 Loews Ventana Canyon, Tucson,
 ArizonaSeptember 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, FloridaOctober 21-25, 1992
 The Wigwam, Phoenix, Arizona,October 27-30, 1993
 The Cloister, Sea Island, GeorgiaNovember 3-6, 1994
 Loew's Ventana Canyon Resort, Tucson, AZNovember 1-5, 1995

FUTURE MEETINGS:

The Greenbrier, White Sulphur Springs, WV September 18-22, 1996
 Rimrock Hotel, Banff, Alberta, Canada..... September 10-14, 1997

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

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John Raaf	1943	John R. Green	1972
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Arthur R. Elvidge.....	1946	Richard L. DeSaussure	1974
John Raaf	1947	Ernest W. Mack.....	1975
Arthur R. Elvidge.....	1948	Frank E. Nulsen.....	1976
F. Keith Bradford.....	1949	Robert S. Knighton.....	1977
David L. Reeves.....	1950	Robert G. Fisher	1978
Henry G. Schwartz	1951	H.T. Ballantine, Jr.....	1979
J. Lawrence Pool.....	1952	George Ehni	1980
Rupert B. Raney	1953	Courtland H. Davis, Jr. ...	1981
David L. Reeves.....	1954	John F. Mullan.....	1982
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George S. Baker.....	1957	E. Bruce Hendrick.....	1985
Samuel R. Snodgrass	1958	Griffith R. Harsh III.....	1986
C. Hunter Shelden	1959	Ellis B. Keener	1987
Edmund Morrissey.....	1960	Robert Grossman	1988
Donald F. Coburn	1961-62	Jim Story	1989
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George L. Maltby.....	1964	Stewart Dunsker.....	1991
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Theodore C. Erickson.....	1944-47	Edward W. Davis.....	1963-65
Wallace B. Hamby.....	1948-50	Robert G. Fisher	1966-68
Theodore B. Rasmussen ...	1951-53	Byron C. Pevehouse ...	1969-72

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John T. Garner.....	1981-83		

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John T. Garner.....	1977-80	Julian T. Hoff.....	1990-92
James T. Robertson.....	1981-83		

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Elected

GUY LAZORTNES, (Annick)

1973

26 Rue D. Aurlol
31400 Toulouse
FRANCE 61528334

VALENTINE LOGUE (Anne)

1974

16 Rowan Road
London, W6 7DU
ENGLAND

BERNARD PERTUISET

1986

Hopital de la Pitie
83 Bernard de l'Hopital
75651 Paris Cedex13
FRANCE

KELJI SANO (Yaeko)

1975

Dept. of Neurosurgery
Teikyo Univ. Hospital
2-11-1 Kaga, Itabashi-ku
Itabasji-ku
Tokyo 173 JAPAN

SENIOR MEMBERS	Elected
EBEN ALEXANDER JR. (Betty) Wake Forest School of Medicine 300 S. Hawthorne Winston-Salem, NC 27157-1002	1950
H. THOMAS BALLANTINE, JR. (Elizabeth) Massachusetts General Hospital Fruit Street Boston, MA 02114-2696	1951
GILLES BERTRAND Montreal Neurological Institute 3801 University Street Montreal, QUEBEC H3A 1B4 CANADA	1967
E. HARRY BOTTERELL (Margaret) 2 Lakeshore Boulevard Kingston, Ontario CANADA	1938
HARVEY CHENAULT (Billee) 6340 Brier Hill Road Paris, KY	1949
SHELLEY CHOU (Jolene) Box 96-Univ. of Minnesota Hospital 420 Delaware Street S.E Minneapolis, MN 55455	1974
GALE CLARK 12621 Brookpark Road Oakland, CA 94619	1970

- W. KEMP CLARK** (Fern) 1970
3909 Euclid Avenue
Dallas, TX 75205
- WILLIAM COLLINS, JR.** (Gwendolyn) 1963
Yale University School of Medicine
333 Cedar Street
New Haven, CT 06510
- JAMES CORRELL** (Cynthia) 1966
249 Olde Pointe Rd.
Hampstead, NC 28443
- COURTLAND DAVIS, JR.** (Carrie) 1967
2525 Warwick Road
Winston-Salem, NC 27104
- RICHARD DESAUSSURE JR.** (Phyllis) 1962
4290 Heatherwood Lane
Memphis, TN 38117-2302
- DONALD DOHN** (Carolyn) 1968
P.O. Box 998
Pt. Clear, AL 36564
- CHARLES DRAKE** (Ruth) 1958
University Hospital
339 Windermere Road
London, ONT N6A 5A5
CANADA
- WILLIAM FEINDEL** (Faith) 1959
Montreal Neurological Institute
3801 University Street
Montreal, Quebec FH3A 2B4
CANADA

- ROBERT FISHER** (Constance) 1955
 Department of Neurosurgery
 DHMC
 Lebanon, NH 03756
- ELDON FOLTZ** (Catherine) 1960
 UCI Medical Center
 Division of Neurosurgery
 P.O. Box 14091
 Orange, CA 92613-4091
- LYLE FRENCH** (Gene F.) 1954
 Dept. of Neurosurgery
 University of MN Hospital
 420 Delaware Street, S.E.
 Minneapolis, MN 55455
- JAMES GALBRAITH** (Marguerite {Peggy}) 1947
 Division of Neurosurgery
 Room 515, M.E.B.
 University Station
 Birmingham, AL 35294
- JOHN GARNER** (Candace) 1971
 50 Allesandro Place, Suite 400
 Pasadena, CA 91105
- HENRY GARRETSON** (Marianna) 1973
 University of Louisville
 Dept. of Neurological Surgery
 210 E. Gray Street
 Louisville, KY 40202
- SIDNEY GOLDRING** (Lois) 1964
 #1 Barnes Hospital Plaza
 Neurosurgery
 St. Louis, MO 63110

- PHILIP GORDY** (Silvia) 1968
3601 Carmel Drive
Casper, WY 82604
- EVERETT GRANTHAM** (Mary) 1942
Gray Street Medical Bldg.
210 Gray Street
Louisville, KY 40202
- WALLACE B. HAMBY** (Ellen) 1941
Apt. #306/Eastlake
601 S.W. 6th Street
Pompano Beach, FL 30060
- JOHN W. HANBERY** (Shirley) 1959
750 Welch Road, Suite 215
Palo Alto, CA 94304
- GRIFF HARSH, III** (Craig) 1980
P.O. Box 232
Sweetwater, TN 37874
- MAJOR GEN. GEORGE HAYES** 1962
303 Skyhill Road
Alexandria, VA 22314
- E. BRUCE HENDRICK** (Gloria) 1968
63 Leggett Ave.
Weston, Ontario M9P1X3
CANADA
- EDGAR HOUSEPIAN** (Marion) 1976
The Neurological Institute
710 West 168th Street
New York, NY 10032

- WILLIAM HUNT** (Carole A. Miller) 1970
 1000 Urlin Ave., #2205
 Columbus, OH 43212
- JOHN A. JANE** (Noella) 1982
 Dept. of Neurosurgery
 University of Virginia
 Charlottesville, VA 22908
- ELLIS KEENER** (Ann) 1978
 434 Academy Street, NE
 Gainesville, GA 30501
- WILLIAM KELLY** 1977
 16925 Englewood
 Bothell, WA 98011
 (206) 488-7981
- ROBERT KING** (Molly) 1958
 State Univ. of NY Health Science Ctr.
 750 East Adams Street
 Syracuse, NY 13210
- WOLFF KIRSCH** (Marie-Claire) 1971
 Loma Linda University Med. Ctr.
 Division of Neurosurgery
 11234 Anderson Street, Rm. 2539
 Loma Linda, CA 92354
- ROBERT KNIGHTON** (Louise) 1966
 9388 Avenida
 San Timoteo
 Cherry Valley, CA 92223

- THEODORE KURZE** 1967
1936 Palisades Drive
Pacific Palisades, CA 90272
- THOMAS LANGFITT (Carolyn)** 1971
Glenmede Corporation
229 South 18th Street
Philadelphia, PA 19103
- SANFORD LARSON (Jackie)** 1989
Department of Neurosurgery
9200 W. Wisconsin Ave.
Milwaukee, WI 53226
- RAEBURN LLEWELLYN (Carmen Rolon)** 1963
5640 Read Boulevard, Suite 840
New Orleans, LA 70127
- WILLIAM LOUGHEED** 1962
15086 Victoria Avenue
White Rock, BC V4B 1G3
CANADA
- JOHN LOWREY (Catherine {Katty})** 1965
Box 44369
Kawai Hae, Hawaii 96743
- ALFRED LUESSENHOP (Frances)** 1977
Georgetown University Hospital
3800 Reservoir Road
Washington, D.C. 20007
- ERNEST W. MACK (Bobbie)** 1956
505 Arlington, South, Suite 106
Reno, Nevada 89505

- LEONARD MALIS** (Ruth) 1973
 1148 Fifth Avenue
 New York, NY 10128
- ROBERT L. MCLAURIN** (Sarah) 1955
 250 Wm. Hwd. Taft Rd., Suite 205
 Cincinnati, OH 45219
- WILLIAM MEACHAM** (Alice) 1952
 709 St. Thomas Medical Plaza East
 Nashville, TN 37205
- JOHN F. MULLAN** (Vivian) 1963
 5841 S. Maryland Ave. MC3026
 Chicago, IL 60637
- BLAINE NASHOLD, JR.** (Irene) 1967
 Duke University Medical Center
 Department of Surgery
 Division of Neurosurgery
 Durham, NC 27710
- GUY ODOM** (Mataline) 1946
 2812 Chelsea Circle
 Durham, NC 27707
- ROBERT G. OJEMANN** (Jean) 1968
 Neurosurgery Service
 Massachusetts General Hospital
 Fruit Street
 Boston, MA 02114
- BURTON ONOFRIO** (Judith) 1975
 Mayo Clinic
 Department of Neurosurgery
 Rochester, MN 55902

- RUSSEL H. PATTERSON, JR.** (Julie) 1971
 New York Hospital
 525 East 68th Street
 New York, NY 10021
- PHANOR PEROT, JR.** 1970
 Dept. of Neurosurgery
 Med. Univ. of South Carolina
 171 Ashley Avenue
 Charleston, SC 29425-2272
- BYRON CONE PEVEHOUSE** (Lucy) 1964
 135 Mountain Spring Avenue
 San Francisco, CA 94114
- J. LAWRENCE POOL** 1940
 41 Cherry Hill Road
 Westcornwall, CT 06796
- ROBERT W. PORTER** (Dean) 1962
 5301 E. 7th Street
 Long Beach, CA 90815
- ROBERT H. PUDENZ** (Rita) 1943
 Huntington Medical Research Institute
 734 Fairmount Avenue
 Pasadena, CA 91105
- JOHN RAAF** (Lorene) Founder
 1120 N.W. 20th Avenue, #100
 Portland, OR 97209
- AIDEN A. RANEY** 1946
 125 N. Las Palmas Avenue, Suite 203
 Los Angeles, CA 90004

- JOSEPH RANSOHOFF II** (Lori) 1965
 James A. Haley Veteran's Hospital
 13000 Bruce B. Downs Blvd.
 Tampa, FL 33612
- THEODORE RASMUSSEN** (Catherine) 1947
 29 Surry Drive
 Montreal, Quebec H3P 1B2
 CANADA
- HUGO V. RIZZOLI** (Helen) 1973
 2150 Pennsylvania Avenue, N.W.
 Washington, D.C. 20037
- THEODORE ROBERTS** (Joan) 1976
 University of Washington/Dept. of Neuro.
 University Hospital RI-20
 Seattle, WA 98105
- JAMES T. ROBERTSON** (Valeria) 1971
 University of Tennessee
 College of Medicine
 847 Monroe Ave., Suite 427
 Memphis, TN 38163
- HENRY G. SCHWARTZ** 1942
 #1 Barnes Hosp. Plaza, Neurosurgery
 St. Louis, MO 63110
- C. HUNTER SHELDEN** 1941
 Huntington Medical Research Inst.
 10 Pico Street
 Pasadena, CA 91105

- JAMES C. SIMMONS** (Vanita) 1975
 190 S. Grove Park Road
 Memphis, TN 38117
- ROBERT R. SMITH** (Helen) 1989
 University of Miss. Med. Ctr.
 Department of Neurosurgery
 Jackson, MS 39216
- BENNETT M. STEIN** (Bonita) 1970
 The Neurological Institute
 710 West 168th Street
 New York, NY 10032
- JIM STORY** (Joanne) 1972
 Univ. of TX, HSC, Neurosurgery
 7703 Floyd Curl Drive
 San Antonio, TX 78284-7843
- ANTHONY F. SUSEN** (Patricia) 1965
 504 Remora Circle
 Fripps Island, SC 29921
- WILLIAM H. SWEET** (Elizabeth) 1950
 Massachusetts General Hospital
 Fruit Street
 Boston, MA 02114
- RONALD R. TASKER** (Mary) 1971
 Toronto Western Hospital
 399 Bathurst Street
 Toronto, ON M5T 2S8,
 CANADA

- GEORGE T. TINDALL** 1968
 Emory Univ. School of Medicine
 1327 Clifton Road
 Atlanta, GA 30322
- JOHN TYTUS (Virginia)** 1967
 1100 9th Ave.
 Seattle, WA 98101
- EXUM WALKER (Nellie)** 1938
 490 Peachtree Street, N.E
 Atlanta, GA 30308
- ARTHUR A. WARD, JR. (Janet)** 1953
 Dept. of Neurological Surgery, Univ. of WA
 Seattle, WA 98104
- W. KEASLEY WELCH (Elizabeth)** 1957
 25 Gould Road
 Waban, MA 02168
- BENJAMIN B. WHITCOMB (Peggie)** 1947
 RDI Box 124
 Surrey, ME 04684
- LOWELL E. WHITE JR. (Marsie)** 1971
 5750 Huffman Dr., N.
 Mobile, AL 36693
- CHARLES B. WILSON (Francie Petrocelli)** 1966
 Dept. of Neurological Surgery
 Univ. of California - San Francisco
 U125 Box 0350
 San Francisco, CA 94143-0350

NICHOLAS T. ZERVAS (Thalia)
Massachusetts General Hospital
32 Fruit Street
Boston, MA 02114

1972

ACTIVE MEMBERS

Elected

MICHAEL APUZZO (Helene)
1200 N. State Street, Ste. 5046
Los Angeles, CA 90033

1988

JAMES AUSMAN (Carolyn)
Univ. of Il, Chicago
Dept. of Neuro/ M/C 799
912 S. Wood St.
Chicago, IL 60612

1979

DANIEL BARROW (Molly)
Dept of Neurological Surgery
Emory Clinic
1365 Clifton Ave., N.E.
Atlanta, Georgia 30322

1993

DONALD BECKER (Maria)
UCLA, Division of Neurosurgery
10833 La Conte Avenue
Los Angeles, CA 90024

1990

PETER MCL. BLACK (Katharine)
Brigham and Women's Hospital
75 Francis Street
Boston, MA 02115

1988

LAWRENCE F. BORGES (Susan)
Massachusetts General Hospital
Div. of Neurosurgery, White 1205
32 Fruit Street
Boston, MA 02114

1993

- JERALD BRODKEY** (Arielle) 1977
24755 Chagrin Blvd., Suite 205
Beachwood, OH 44122
- WILLIS BROWN, JR.** (Ann) 1984
Division of Neurosurger
Univ. of Texas Health Science Ctr.
7703 Floyd Curl Drive
San Antonio, TX 78284-7843
- DEREK BRUCE** (Frances) 1984
1935 Motor Street
Dallas, TX 75235
- WILLIAM BUCHHEIT** (Christa) 1980
Thomas Jefferson University
Department of Neurosurgery
1015 Chestnut, #1400
Philadelphia, PA 19107
- KIM J. BURCHIEL** (Debra) 1992
Division of Neurosurgery
Oregon Health Sciences University
3181 S.W. Sam Jackson Park Rd.
Portland, OR 97201-3098
- PETER W. CARMEL** (Jacqueline Bello) 1991
Neurological Institute
710 W. 168th Street
New York, NY 10032
- WILLIAM CHANDLER** (Susan) 1989
2128 Taubman Health Ctr., 0338
University of Michigan
1500 E. Medical Center Drive
Ann Arbor, MI 48109-0338

- PAUL CHAPMAN (Tansy)** 1983
 Department of Neurosurgery
 Massachusetts General Hospital
 32 Fruit Street
 Boston, MA 02114
- EDWARD CONNOLLY (Elise)** 1972
 Ochsner Clinic
 Department of Neurosurgery
 1514 Jefferson Highway
 New Orleans, LA 70121
- ROBERT CROWELL (Mary)** 1990
 Neurosurgery/ACC #31
 510 North Street
 Pittsfield, MA 01201
- RALPH DACEY, JR. (Corinne)** 1990
 Washington Univ. School of Med.
 CB #8057/Dept. of Neurosurgery
 660 S. Euclid
 St. Louis, MO 63110
- ARTHUR L. DAY (Dana)** 1990
 University of Florida Health Cente
 Neurosurgery/Box 100265
 Gainesville, FL 32610
- STEWART DUNSKER (Ellen)** 1975
 Mayfield Neurological Institute
 2123 Auburn Avenue
 Cincinnati, OH 45219
- MICHAEL S.B. EDWARDS (Linda)** 1992
 UCSF, Neurosurgery
 533 Parnassus Ave., U-126
 San Francisco, CA 94143

- HOWARD EISENBERG** (Janet) 1985
Division of Neurosurgery
University of Maryland
22 S. Greene Street
Baltimore, MD 21201
- MEL H. EPSTEIN** (Lynn) 1992
Brown University
Department of Neurosurgery
110 Lockwood Street
Providence, RI 02903
- EUGENE S. FLAMM** (Susan) 1979
Hospital of Univ. of Pennsylvania
3400 Spruce Street
Philadelphia, PA 19104
- RICHARD A. R. FRASER** (Sara Ann) 1976
525 East 68th Street
New York, NY 10021
- ALLAN FRIEDMAN** (Elizabeth Bullitt) 1994
Division of Neurosurgery
Duke University Hospital
P.O. Box 3807
Durham, NC 27710
- STEVEN GIANNOTTA** (Sharon) 1992
LAC/Univ. Southern California Medical Ctr.
1200 N. State, Box 239
Los Angeles, CA 90033
- ROBERT GROSSMAN** (Ellin) 1984
Department of Neurosurgery
Baylor College of Medicine
One Baylor Place
Houston, TX 77030

- ROBERT L. GRUBB, JR.** (Julia) 1985
 Dept. of Neurological Surgery, Box 8057
 Wash. Univ. Schl. of Med.
 660 S. Euclid Avenue
 St. Louis, MO 63110
- JOSEPH F. HAHN** (Andrea) 1993
 Cleveland Clinic
 9500 Euclid Ave.
 Cleveland, OH 44195
- STEPHEN J. HAINES** 1994
 Box 96, UMHC
 420 Delaware St., S.E.
 Minneapolis, MN 55455
- PETER HEILBRUN** (Robyn) 1984
 Division of Neurosurgery #3B409
 Univ. of Utah Medical Center
 50 North Medical Drive
 Salt Lake City, UT 84132
- ROBERTO C. HEROS** (Deborah) 1985
 Department of Neurosurgery
 University of Miami
 1501 NW Ninth Ave.
 Miami, Fl 33136
- CHARLES HODGE, JR.** 1982
 750 East Adams Street
 Syracuse, NY 13210
- JULIAN T. HOFF** (Diane) 1975
 2128 Taubman Health Ctr., 0338
 1500 E. Medical Ctr. Drive
 Ann Arbor, MI 48109-0338

- HAROLD HOFFMAN** (Jo Ann) 1982
Hospital for Sick Children
555 University Avenue
Toronto, ONTARIO M5G 1X8
CANADA
- L. N. HOPKINS** (Ann {Bonnie}) 1992
3 Gates Circle
Buffalo, NY 14209
- ALAN HUDSON** (Susan) 1978
585 University Avenue, Suite BW1-658
Toronto, Ontario M59 2C4
CANADA
- DAVID KELLY, JR.** (Sarah {Sally}) 1975
Department of Neurosurgery
Bowman Gray School of Medicine
Medical Center Blvd.
Winston-Salem, NC 27157-1029
- PATRICK KELLY** (Carol) 1992
New York University Medical Center
550 First Avenue
New York, NY 10016
- GLENN KINDT** (Charlotte) 1977
Div. of Neurosurgery
Univ. of Colorado Med. Ctr., Box C-307
4200 East 9th Avenue
Denver, CO 80262
- DAVID KLINE** (Nell) 1971
Department of Neurosurgery
Louisiana State University Medical Center
1542 Tulane Avenue
New Orleans, LA 70112

- PETER JANNETTA (Diane)** 1994
 Department of Neurological Surgery
 Presbyterian University Hospital
 Pittsburgh, PA 15213
- EDWARD R. LAWS, JR. (Margaret {Peggy})** 1983
 Department of Neurosurgery
 Box 212 HSC
 University of Virginia
 Charlottesville, VA 22908
- DONLIN M. LONG (Harriet)** 1983
 Dept. of Neurological Surgery
 Johns Hopkins Medical School
 600 N. Wolfe, Meyer 7-109
 Baltimore, MD 21287-7709
- CHRISTOPHER LOFTUS (Sara Sim)** 1992
 Div. of Neurosurgery, Univ of Iowa Hosp.
 200 Hawkins Drive, 1844 JPP
 Iowa City, IA 52242
- L. DADE LUNSFORD (Julianne)** 1992
 B-400, Presbyterian University Hospital
 Pittsburgh, PA 15213
- ROBERT L. MARTUZA (Jill)** 1989
 Georgetown University Medical Center
 3800 Reservoir Road, N.W.
 Washington, D.C. 20007
- ROBERT E. MAXWELL (Karen)** 1992
 Department of Neurosurgery, Box 142
 420 Delaware Street, S.E.
 Minneapolis, MN 55455

- JOE MAURICE MCWHORTER** (Barbara) 1989
 2810 N. Maplewood.
 Winston-Salem, NC 27103
- RICHARD MORAWETZ** (Mary Jean) 1990
 University of Alabama
 Division of Neurosurgery
 MEB 512
 Birmingham, AL 35294
- PAUL B. NELSON** (Teresa) 1991
 Indiana University, NS, EM-139
 545 Barnhill Drive
 Indianapolis, In 46202
- GEORGE OJEMANN** (Linda) 1975
 Department of Neurological Surgery RI-20
 University of Washington
 Seattle, WA 98195
- ANDRE OLIVIER** (Nicole) 1989
 Montreal Neurological Hospital
 3801 University Street, Suite #109
 Montreal, Quebec H3A2B4
 CANADA
- SYDNEY JOHN PEERLESS** (Ann) 1977
 3663 S. Miami Ave., Ste. 209
 Miami, FL 33133
- DAVID G. PIEPGRAS** (Jane) 1987
 Department of Neurological Surgery
 Mayo Clinic, 200 First Street, S.W.
 Rochester, MN 55905

- DONALD QUEST** (Ilona) 1986
 Department of Neurological Surgery
 The Neurological Institute - Columbia Univ.
 710 West 168th Street
 New York, NY 10032
- ROBERT A. RATCHESON** (Peggy) 1986
 University Hospitals of Cleveland
 2074 Abington Road
 Cleveland, OH 44106
- ALBERT RHOTON, JR.** (Joyce) 1984
 Department of Neurological Surgery
 College of Medicine, P.O. Box 100265
 University of Florida
 Gainesville, FL 32610
- J. CHARLES RICH, JR.** (Jasmine) 1987
 370 Ninth Ave., Suite 111
 Salt Lake City, UT 84103
- JON H. ROBERTSON** (Carol Ann) 1992
 920 Madison Ave., Suite 600
 Memphis, TN 38103
- DUKE SAMSON** (Patricia) 1994
 Department of Neurosurgery
 University of Texas, Southwestern
 5323 Harry Hines Blvd.
 Dallas, Texas 75235-8855
- R. MICHAEL SCOTT** (Susan) 1991
 Neurosurgery / Bader 3
 Childrens Hospital
 300 Longwood Ave., Neuro
 Boston, MA 02115

- EDWARD L. SELJESKOG (Peggy)** 1992
 2805 Fifth St., South, Ste. 110
 Rapid City, SD 57701
- CHRISTOPHER SHIELDS (Deborah)** 1993
 Department of Neurosurgery
 University of Louisville
 210 E.Gray St., Suite 1105
 Louisville, Ky 40202
- WILLIAM SHUCART (Laura)** 1989
 Department of Neurosurgery
 New England Medical Center
 750 Washington Street
 Boston, MA 02111
- FREDERICK SIMEONE** 1981
 Pennsylvania Hospital
 800 Spruce Street
 Philadelphia, PA 19107
- KENNETH R. SMITH, JR. (Marjorie)** 1987
 St. Louis University Hospital
 3635 Vista Avenue
 St. Louis, MO 63110-0250
- DENNIS D. SPENCER (Susan)** 1989
 Section of Neurological Surgery
 Yale University School of Medicine
 333 Cedar St., P.O. Box 3333
 New Haven, CT 06510
- CHARLES H. TATOR (Carol)** 1991
 Toronto Western Hospital
 399 Bathurst Street
 Toronto, ON M5T 2S8
 CANADA

- JOHN M. TEW, JR. (Susan)** 1971
 Mayfield Neurological Institute
 506 Oak Street
 Cincinnati, OH 45219
- SUZIE C. TINDALL** 1990
 Emory University
 1365 Clifton Road
 Atlanta, GA 30322
- RUSSELL L. TRAVIS** 1994
 1401 Herrodburg Rd., Suite 485B
 Lexington, KY 40504-3700
- JOHN VAN GILDER (Kerstin)** 1980
 Department of Neurosurgery
 University of Iowa School of Medicine
 Iowa City, IA 55242
- CLARK WATTS (Patricia)** 1975
 Ford & Ferraro
 98 San Jacinto Blvd., Suite 2000
 Austin, TX 78701
- BRYCE K. WEIR (Mary Lou)** 1984
 Section of Neurosurgery, MC 3026
 University of Chicago
 5841 S. Maryland Ave.
 Chicago, IL 60637
- MARTIN H. WEISS (Debby)** 1981
 USC Medical Center, Box 786
 1200 North State Street
 Los Angeles, CA 90033

- ROBERT H. WILKINS** (Gloria) 1973
 Duke University Medical Center, Box 3807
 Durham, NC 27710
- H. RICHARD WINN** (Debbie) 1993
 Univ. of WA, Sch. of Medicine
 Dept. of Neurosurgery
 325 Ninth ZA86
 Seattle, WA 98104
- FREMONT P. WIRTH** (Penny) 1993
 4 Jackson Blvd.
 Savannah, Ga 31405
- ALLEN WYLER** (Lily) 1990
 Epilepsy Center, Swedish Medical Center
 747 Summit
 Seattle, WA 98104
- DAVID YASHON** 1972
 #1201 1492 E. Broad Street
 Columbus, OH 43205
- A. BYRON YOUNG** (Judy) 1989
 University of Kentucky Medical Center
 800 Rose Street, MN 268
 Division of Neurosurgery
 Lexington, KY 40536
- RONALD F. YOUNG** (Christina) 1986
 Northwest Hospital
 1560 N. 115th St., #G5
 Seattle, WA 98133

HAROLD F. YOUNG (M. Theresa)
Medical Col. of Virginia Station
P.O. Box 631
Richmond, VA 23298

1994

INACTIVE

ELECTED

ROBERT BOURKE

1983

5802 Nicholson Lane, Ste. 305
Rockville, MD 20852
(301) 881-4567

JOHN KAPP

1985

P.O. Box 448
Galax, VA 24333
(703) 236-2613

RICHARD S. KRAMER (Mollie)

1978

Duke University Medical Center
Box 3255
Durham, NC 27710

SENIOR CORRESPONDING	ELECTED
JEAN BRIHAYE (van Geertruyden) Belgium 98 avenue Des Franciscains Brussels, BELGIUM	1975
KARL AUGUST BUSHE (Eva-Christa) Technische Universitat Dresden Helmholtzstrasse 18 8027 Dresden D-8700 GERMANY	1972
FERNANDO CABIESES Peruano De Formento Educativo Av. Arenales 371, of. 501 Apartado 5254 Lima, PERU	1966
JUAN CARDENAS (Delores) Insurgentes Sur 594 Av. Insurgentes Mexico City, 40 MEXICO	1966
JUAN CHRISTENSEN (Diana Poli) Jose' C. Paz 234 Acassusi (1641) Buenos Aires Province ARGENTINA	1970
HANS ERICH DIEMATH (Karin) Landesnervenklinik, Dept. of Neurosurgery 5020 Salzburg, Ignaz Harrer-StraBe 79 AUSTRIA	1970

- HERMANN DIETZ** (Elfrun) 1970
 Department of Neurosurgery
 Hannover School of Medicine
 30623 Hannover
 GERMANY
- JOHN GILLINGHAM** 1962
 Royal Infirmary
 Lauriston Place
 Edinburgh EH43 PB
 Scotland, United Kingdom
- JAIME G. GOMEZ** (Lucy) 1975
 5353 N. Federal Highway, #210
 Fort Lauderdale, FL 33068
- JOHN HANKINSON** (Nicole) 1973
 Westacres
 Woolsington Hall
 Newcastle-Upon-Tyne, NE13 8DG
 ENGLAND
- SHOZO ISHII** (Akiko) 1975
 Juntendo University
 2-1 Hongo, Bunkyo-ku
 Tokyo 113, JAPAN
- HANS-PETER JENSEN** 1980
 Neurochirurgische
 Universitätsklinik Kiel
 Welmarer Strasse 8
 Kiel D-2300
 GERMANY

- RICHARD JOHNSON** 1974
 Dept. of Neurological Surgery
 Royal Infirmary
 Manchester, ENGLAND
- KATSUTOSHI KITAMURA (Yoshiko)** 1970
 1-3-1 Kanada
 Kokurakita-Ku, Kitakyushu
 803, JAPAN
- LAURI LAITINEN (Kerstin)** 1972
 Sophiahemmet
 Box 5605
 S-114 86, Stockholm
 SWEDEN
- WILLIAM LUYENDIJK** 1973
 2341 KL Oegstgeest
 THE NETHERLANDS
- GUISEPPE DALLE ORE (Guisi)** 1970
 Clinica Neurochirurgica
 Universita di Verona
 Piazzale Stefani
 Verona 37100
 ITALY
- B. RAMAMURTHI (Indira)** 1973
 Voluntary Health Services
 Adyar Madras-600 113
 INDIA
- KURT-FRIEDRICH SCHURMANN** 1978
 Am Eselsweg 29
 D-6500 Mainz 1
 GERMANY

CHARAS SUWANWELA 1972
Chulalongkorn Hospital Medical School
Bangkok
THAILAND

LINDSAY SYMON (Pauline) 1982
Gough-Cooper Dept. of Neurological Surgery
Institute of Neurology, The National Hospital
Queen Square London WC1N 3BG
ENGLAND

KJELD VAENET 1970
Department of Neurosurgery
Rigshospitalet
Copenhagen 2100
DENMARK

SIDNEY WATKINS 1975
The London Hospital
Whitechapel
London E 1
ENGLAND

M. GAZI YASARGIL (Dianne) 1975
Dept. of Neurosurgery
University of Arkansas
Little Rock, AR 72205

CORRESPONDING

Elected

LEIGH ATKINSON

1989

Alexandra, Suite, 2nd, Floor
201 Wickham Terrace, 4000
Brisbane 4000
AUSTRALIA

LUC CALLIAUW (Dora)

1988

Dept. of Neurosurgery, University Hospital
De Pintelaan
Ghent, BELGIUM

H. ALAN CROCKARD (Caroline)

1992

Dept. of Surgical Neurology, National Hosp.
Queen Square
London, WCIN 3BG, ENGLAND

NOEL GEORGE DAN (Adrienne)

1989

Specialist Medical Center, Suite 302
235-285 New South Head Road
Edgecliff, N.S.W. 2027
AUSTRALIA

JACQUES DEVILLIERS (Jeanne Marie Erica)

1986

Department of Neurosurgery
University of Cape Town
Observatory 7925 Cape 7
Republic of SOUTH AFRICA

VINKO DOLENC

1988

Univ. of Ljubljana/Neuro.
Clinical Ctr. Zaloska 7
Ljubljana 61105
YUGOSLAVIA

- RUDOLPH FAHLBUSCH** (Hanna) 1991
 Neurochirurgische Klinik
 Universitat Erlangen-Nurnberg
 Schwabachanlage 6
 91054 Erlangen
 GERMANY
- SALVADOR GONZALEZ-CORNEJO** (Rosa) 1982
 Av. Chapultepec Sur 130-204
 Guadalajara, 44140
 MEXICO
- ERNST GROTE** (Juliana) 1984
 Department of Neurosurgery
 University Kliniks Schnarrenberg
 Hoppe Seyler-Str. 3
 72076Tubingen
 GERMANY
- DAE HEE HAN** (Sung Soon Cho) 1991
 SNU Hospital
 Seoul Nat'l, Univ., School of Medicine
 #28 Yougon-dong
 Chongno-Gu, Seoul 110-744
 KOREA
- HAJIME HANDA** (Hiroko) 1985
 Takeda General Hospital
 28-1 Moriminami-cho Ishida
 Fushimi-ku,
 Kyoto 601-13, JAPAN
- FABIAN ISAMAT** (Maria V. {Marivi}) 1989
 Clinica Sagrade Familia
 Neurogrup, Torras y Pujalt, 1
 08022 Barcelona, SPAIN

RAUL MARINO, JR. 1977
Rua Maestro Cardim, 808
Instituto Nueurologico de S. Paulo
S. Paulo-SP
01323-100, BRAZIL

HARUHIKO KIKUCHI 1993
Dept. of Nueorsurgery
Kyoto Univ., Medicine
5-1 Kawahara-cho
Shogoin Skyo-ku 606
Kyoto, JAPAN

KINTOMO TAKAKURA 1988
Dept. of Neurosurgery
Neurological Institute
Tokyo's Women's Medical College
8-1, Kawadacho, Shinjuku-ku
Tokyo 162, JAPAN

DECEASED MEMBERS

	Deceased	Elected
SIXTO O. ALCALDE Madrid, Spain (Honorary)	1978	1973
JAMES R. ATKINSON Phoenix, Arizona (Active)	1978	1970
GEORGE BAKER Litchfield Park, AZ (Senior)	1993	1940
PERCIVAL BAILEY Evanston, Illinois (Honorary)	1973	1960
WILLIAM F. BESWICK Buffalo, New York (Active)	1971	1959
EDWIN B. BOLDREY San Francisco, California (Senior)	1988	1941
SPENCER BRADEN Cleveland, Ohio (Active)	1969	Founder
F. KEITH BRADFORD Houston, Texas (Active)	1971	1938
HOWARD BROWN San Francisco, California (Senior)	1990	1939
DONALD COBURN Wilmington, Delaware (Senior)	1988	1938

WINCHELL McK. CRAIG Rochester, Minnesota (Honorary)	2/60	1942
EDWARD DAVIS Portland, Oregon (Senior)	1988	1949
PEARDON DONAGHY Burlington, Vermont (Senior)	1991	1970
FRANCIS ECHLIN New Paltz, 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
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
JESSE HERMANN 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
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
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, FL (Senior)	1994	1956
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
RUPERT B. RANEY Los Angels, 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
STEWART N. ROWE Pittsburgh, Pennsylvania (Senior)	1984	1938
RICHARD C. SCHNEIDER Ann Arbor, Michigan (Senior)	1986	1970
WILLIAM B. SCOVILLE Hartford, Connecticut (Senior)	1984	1944
R. EUSTACE SEMMES Memphis, Tennessee (Honorary)	1982	1955
SAMUEL R. SNODGRASS Galveston, Texas (Senior)	1975	1939
GLEN SPURLING LaJolla, California (Honorary)	1968	1942
C. WILLIAM STEWART Montreal, Quebec, Canada (Corresponding)	1948	1948
THORALF SUNDT, JR. Rochester, Minnesota (Active)	1992	1971

KENICHIRO SUGITA Nagoya, Japan (Senior Corresponding)	1994	1988
HENDRIK SVIEN Rochester, Minnesota (Active)	1972	1957
HOMER S. SWANSON Atlanta, Georgia (Senior)	1987	1949
ALFRED UIHLEIN Rochester, Minnesota (Senior)	1990	1950
A. EARL WALKER Albuquerque, New Mexico (Senior)	1995	1938
THOMAS A. WEAVER, JR. Dayton, Ohio (Senior)	1985	1943
BARNES WOODHALL Durham, North Carolina (Senior)	1985	1941
FRANK WRENN Greenville, South Carolina (Senior)	1990	1973



