THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

58th Annual Meeting

The Greenbrier
WHITE SULPHUR SPRINGS
WEST VIRGINIA 24986
A CSX Resort

September 18 - 22, 1996

Jointly Sponsored by The American Association of Neurological Surgeons

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Julian T. Hoff

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LOCAL HOSTS

L. Nelson (Nick) and Bonnie Hopkins

THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

Activities Program

Tuesday, September 17

4:00 PM - 7:30 PM	ABNS Exam Committee Review Pierce Room
7:30 PM - 9:30 PM	ABNS Dinner Main Dining Room

Wednesday, September 18

8:00 AM - 3:00 PM	ABNS Primary Exam Committee Pierce Room
2:00 PM - 5:00 PM	Registration Lower Lobby, Area B
2:00 PM - 5:00 PM	Speaker Ready Room Editing Room (across from Chesapeake Room)
2:00 PM - 3:00 PM	Executive Committee Buchanan Room
3:00 PM - 5:00 PM	Membership Committee Buchanan Room
6:30 PM - 8:00 PM	Welcome Reception* Cameo Ballroom
7:30 PM - 10:00 PM	Dinner* Crystal Room
7:30 PM - 10:00 PM	Executive Committee Dinner* Taft Room

* Evening Wear:
Gentlemen - Business suits, jackets and slacks, neckties or black tie
Ladies - Dresses (short or long), evening suits

Thursday, September 19

6:00 AM - 2:00 PM	Speaker Ready Room Editing Room
7:00 AM - 8:00 AM	Breakfast/Business Meeting (Members only)* Crystal Room

* **Guest breakfast** available in Draper's Cafe at 7:00 AM or the Main Dining Room at 7:30 AM

8:00 AM - 1:00 PM	Registration Chesapeake Room Foyer
8:00 AM - 1:00 PM	Symposium & Scientific Session I Chesapeake Room
10:05 AM - 10:30 AM	Coffee Break Colonial Lounge & Terrace
1:00 PM - 2:30 PM	ABNS Advisory Council Wilson Room
1:04 PM - 6:00 PM	Golf Sign up at Registration Desk
2:45 PM - 5:00 PM	Journal of Neurosurgery Editorial Board Pierce Room
3:00 PM - 5:00 PM	Tennis Sign up at Registration Desk
6:30 PM - 8:00 PM	Reception Colonial Lounge & Terrace
7:30 PM - 10:00 PM	Dinner* Main Dining Room

* Sign up for 7:30 or 8:00 PM seating at Registration Desk

Friday, September 20

6:00 AM - 2:00 PM	Speaker Ready Room Editing Room
7:00 AM - 8:00 AM	Breakfast/Business Meeting (Members only)* Crystal Room

* Guest breakfast available in Draper's Cafe at 7:00 AM or the Main Dining Room at 7:30 AM

8:00 AM - 12:30 PM	Registration Chesapeake Room Foyer
8:00 AM - 12:30 PM	Symposium & Scientific Session II Chesapeake Room
10:05 AM - 10:35 AM	Coffee Break Colonial Lounge & Terrace
11:45 PM - 12:30 PM	Presidential Address: John M. Tew, Jr., MD Chesapeake Room
1:12 PM - 6:00 PM	Golf Tournament Sign up at Registration Desk
3:00 PM - 5:00 PM	Tennis Sign up at Registration Desk
6:30 PM - 8:00 PM	Reception and Putting Contest* Chesapeake Bay & Terrace

* Sign up for contest at Registration Desk

7:30 PM - 10:00 PM	Dinner* Main Dining Room

* Sign up for 7:30 or 8:00 PM seating at Registration Desk

Saturday, September 21

6:00 AM - 12:45 PM	Speaker Ready Room Editing Room
7:00 AM - 8:25 AM	Breakfast for Members and Guests Individual arrangements*

* Draper's Cafe at 7:00 AM or the Main Dining Room at 7:30 AM

7:30 AM - 8:25 AM	The Society of Neurological Surgeons Lee Room
8:25 AM - 12:45 PM	Registration Chesapeake Room Foyer
8:25 AM - 12:45 PM	Scientific Session III & IV Chesapeake Room
9:45 AM - 10:30 AM	Keynote Address: Leadership Without Easy Answers Ronald Heifetz, Kennedy School of Government, Harvard University Chesapeake Room
10:30 AM - 11:00 AM	Coffee Break* Colonial Lounge & Terrace

*Group Photograph to be taken during break
Sign up at Registration Desk for a copy of photo

1:04 PM - 6:00 PM	Golf Sign up at Registration Desk
3:00 PM - 5:00 PM	Tennis Tournament Sign up at Registration Desk
6:30 PM - 7:30 PM	Reception Colonial Lounge & Terrace
7:30 PM - 12:00 AM	Banquet - Dance Black tie Chesapeake Room

Sunday, September 22 — Departures

SCHEDULE OF ACTIVITIES FOR SPOUSES

Wednesday, September 18

6:30 PM - 8:00 PM	Welcome Reception* Cameo Ballroom
7:30 PM - 10:00 PM	Dinner* Crystal Room
7:30 PM - 10:00 PM	Executive Committee Dinner* Taft Room

* Evening Wear:
Gentlemen - Business suits, jackets and slacks, neckties or black tie
Ladies - Dresses (short or long), evening suits

Thursday, September 19

Walking Course on your own: maps available at the Meeting Registration Desk

7:00 AM - 10:00 AM	Breakfast – Individual Arrangements*

* Draper's Cafe from 7:00 - 10:00 AM or Main Dining Room from 7:30 - 10:00 AM

9:00 AM - 11:00 AM	Hospitality Suite Spring Room
10:00 AM - 11:00 AM	Book Review Fair and Tender Ladies by Lee Lewis Spring Room
1:04 PM - 6:00 PM	Golf Sign up at Registration Desk
3:00 PM - 5:00 PM	Tennis Sign up at Registration Desk
3:00 PM - 5:00 PM	Hospitality Suite* Spring Room

* Card tables available; bridge and backgammon tables are in the nearby Green and Trellis Lobbies

4:15 PM - 5:00 PM	Readings of Essays, Poetry, and Short Stories Spring Room
4:15 PM - 5:00 PM	Tea and Concert Main Lobby
6:30 PM - 8:00 PM	Reception Colonial Lounge & Terrace
7:30 PM - 10:00 PM	Dinner* Main Dining Room

* Sign up for 7:30 or 8:00 PM seating at Registration Desk

Friday, September 20

Walking Course on your own

7:00 AM - 10:00 AM	Breakfast - Individual Arrangements*

* Draper's Cafe from 7:00 - 10:00 AM or Main Dining Room from 7:30 - 10:00 AM

9:00 AM - 11:00 AM	Hospitality Suite Spring Room
10:30 AM - 11:30 AM	Interior Tour of The Greenbrier* Meet guide at the Spring Room

* Sign up at Registration Desk

1:12 PM - 6:00 PM	Golf Tournament Sign up at Registration Desk
3:00 PM - 5:00 PM	Tennis Sign up at Registration Desk
3:00 PM - 5:00 PM	Hospitality Suite Spring Room
4:15 PM - 5:00 PM	Tea and Concert Main Lobby
6:30 PM - 8:00 PM	Reception and Putting Contest* Chesapeake Bay & Terrace

* Sign up for contest at Registration Desk

7:30 PM - 10:00 PM	Dinner* Main Dining Room

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Saturday, September 21

Walking Course on your own

7:00 AM - 10:00 AM	Breakfast - Individual Arrangements*

* Draper's Cafe from 7:00 - 10:00 AM or Main Dining Room from 7:30 - 10:00 AM

9:00 AM - 11:00 AM	Hospitality Suite Spring Room
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6:30 PM - 7:30 PM	Reception Colonial Lounge & Terrace
7:30 PM - 12:00 AM	Banquet - Dance Black tie Chesapeake Room

Sunday, September 22 — Departures

SCIENTIFIC PROGRAM
AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
1996 LEARNING OBJECTIVES
September 18-22, 1996

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

September 19-22, 1996

Jointly Sponsored by The American Association of Neurological Surgeons

Thursday, September 19

8:00-8:05	**WELCOME** – John Tew, President
8:05-10:05	**SYMPOSIUM – Evaluating and Improving Neurosurgical Results** Moderators: **Julian Hoff, Martin Camins**
8:05-8:25	Critical Pathways – Charles Rich
8:25-8:45	Assessing and Reporting Complications – Roberto Heros
8:45-9:05	Outcome Analysis – Stephen Haines
9:05-9:25	Institutional Guidelines – Joseph Hahn
9:25-9:45	Federal Regulations – Paul Nelson
9:45-10:05	Discussion
10:05-10:30	**Coffee Break**

SCIENTIFIC SESSION I

Moderators: **Edward Connolly, Harry van Loveren**

10:30-10:55 *Pathobiology of Cerebral Cavernous Malformations: An Integrative Paradigm*
Issam A. Awad, Murat Gunel, KM Desai, John Pizzonia, Richard P. Lifton

10:55-11:20 *Posteroventral Pallidotomy: Indications, Surgical Technique and Clinical Outcome*
Kim Burchiel, Jacques Favre, Jamal Taha

11:20-11:45 *Pathophysiology of Syringomyelia Associated with Chiari I Malformation*
JD Heiss, H DeVroom, A Eidsath, T Talbot, N Patronas, EH Oldfield

11:45-12:10 *Differentiation between Cortical Atrophy and Hydrocephalus using ^{1}H MRS*
Stefan Blum, J. Gordon McComb, Brian D. Ross

12:10-12:35 *The Risk of Major Morbidity after Stereotactic Radiosurgery*
Douglas Kondziolka, John C. Flickinger, L. Dade Lunsford, David J. Bissonette

12:35-12:50 **Academy Award Presentations, Bryce Weir, Chair**
Drug-Regulated Apoptotic Death in a Rat Glioma Model Mediated by a Novel Retroviral Vector
Academy Award Paper
John S. Yu, Massachusetts General Hospital, Boston, Massachusetts

12:50-1:00 **Academy Award Honorable Mention**
In Vivo Angioplasty Prevents the Development of Vasospasm in Canine Carotid Arteries: Pharmacological and Morphological Analyses
Joseph F. Megyesi, McKenzie Health Sciences Center, Edmonton, Alberta

Friday, September 20

8:00 AM - 10:00 AM **SYMPOSIUM - New Directions in Image-Guided Minimally Invasive Neurosurgery**
Moderators: **John Tew, Kalmon Post**

8:00-8:20 Frameless Sterotaxis – Peter Heilbrun

8:20-8:40 Surgery in the MRI – Peter Black

8:40-9:00 Intravascular Therapies – L. N. Hopkins

9:00-9:20 Spinal Endoscopy – Volker Sonntag

9:20-9:40 Video-based Systems – David Thomas

9:40-10:05 Discussion

10:05-10:35 **Coffee Break**

10:35-12:30pm
SCIENTIFIC SESSION II
Moderators: **Roberto Heros, Volker Sonntag**

10:35-11:00 *Extra-foraminal Lumbar Disc Herniations*
V. Rajaraman, N. Swami, W. S. Tobler, S. B. Dunsker

11:00-11:20 *The Miami Project – Scientific Achievements*
Barth A. Green, Richard P. Bunge

11:20-11:45 *Clinical and Experimental Studies of the Value of Surgical Decompression in Spinal Cord Injury*
Charles H. Tator

11:45-12:30 **Presidential Address: John Tew**
Introduced by Edward Connolly

Saturday, September 21

SCIENTIFIC SESSION III
Moderators: **David Piepgras, Fredric Meyer**

8:25-8:45 *AVM Radiosurgery: Truths and Consequences*
L.D. Lunsford, B. E. Pollock, D. Kondziolka, A. Maitz, J.C. Flickinger

8:45-9:05 *Mechanism of Radiation Effect On Arterial Smooth Muscle Cells and Endothelium*
Marc M. Mayberg

9:05-9:25 *The Role of Neuronal NO in Collateral and Normal rCBF*
Christopher M. Loftus, Scott C. Robertson

9:25-9:45 *Repair of Carotid Endarterectomy with Collagen Dacron Graft*
Fredric B. Meyer, Wanda L. Windschitl

9:45-10:30 **Keynote Address:**
Leadership Without Easy Answers
Ronald Heifetz

10:30-11:00 **Coffee Break**

Saturday, September 21 Cont'd

SCIENTIFIC SESSION IV
Moderators: **L.N. Hopkins, Keith Black**

11:00-11:20 *Drug Delivery by Intracerebral Clysis in a Rat Glioma Model*
Jeffrey Bruce, B. Birch, J. Johnson, J. Yoon, C. Lycette, A. Falavigna

11:20-11:40 *Comprehensive Image-Guided Surgery of Sellar Tumors*
Michael A. Schulder, Peter Carmel

11:40-12:00 *Dorsal Cervical-Medullary Bulge in Chiari I Malformations*
Peter W. Carmel, Jacqueline A. Bello

12:00-12:20 *Brain Tumor Invasion – Biological Rationale and Preclinical Studies*
Mark L. Rosenblum, Tom Mikkelsen, Klaus Edvardsen, Kevin Nelson, Elise Kohn, Robert Smith, Donna Spencer, David Rasnick, Bonnie Sloane

12:20-12:40 *Immunobiology of Primary Brain Tumors*
WH Brooks, RL Roszman, LE Elliott

12:45 *Adjourn*

Thursday, September 19
10:30 AM

Pathobiology of Cerebral Cavernous Malformations: An Integrative Paradigm

Awad I, Gunel M, Desai KM, Pizzonia J, Lifton R

The cerebral cavernous malformation (CCM) is a common vascular lesion of the brain, affecting 0.5% of the population, and manifesting myriad neurologic sequelae including headache, epilepsy, focal neurologic deficits and hemorrhagic stroke. We review epidemiologic data from a multi-institutional data base of cases evaluated during one decade. There is marked difference in probability of lesion behavior among the genders, and in different age groups. Lesions which have hemorrhaged previously are more likely to hemorrhage again and cause clinical disability. Probability of untoward clinical events is driven by host and lesion factors, and does not follow a simple lesion-year hemorrhagic risk model. In collaboration with twelve centers across the United States, we have examined the genetics of the lesion, and mapped a gene causing CCM to 7q11.2-q21. Linkage dysequilibrium studies in Hispanic Americans have demonstrated a preserved haplotype in the region of the CCM gene in familial and apparently sporadic cases, indicating that all CCM cases in this population are likely genetic and inherited from a common ancestor. We have uncovered evidence of genetic locus heterogeneity among non-Hispanic patients, with one additional gene likely causing the disease. Vascular biology studies have included immunohistochemical staining for markers of vessel wall maturity, and expression of angiogenesis growth factors. Studies on lesion epileptogenicity have focused on iron assays and impact of heme breakdown products on glial and neuronal excitability. Data from these studies is integrated into a disease paradigm with potential clinical applications including genetic screening, prediction of penetrance and clinical sequelae, and novel strategies for molecular modification of lesion behavior.

NOTES

Thursday, September 19
10:55 AM

Posteroventral Pallidotomy: Indications, Surgical Technique and Clinical Outcome

Burchiel K. Favre J. Taha J

From January 1993 through May 1996, we performed 113 posteroventral pallidotomies (PVP) on 82 patients. In this series, two patients had probable "Parkinson's plus" syndromes, one had hemiballismus from cerebral palsy, and one had a combination of simultaneous pallidotomy with a thalamotomy performed contralaterally. The remainder (78) were patients with previously Sinemet-responsive Parkinson's disease, now medically intractable despite maximal adjunctive medications. Indications for PVP included in order of decreasing importance: Predominant symptoms of Levodopa-induced dyskinesias, bradykinesia, rigidity, dystonia, severe on-off fluctuations, freezing, gait disturbance, and tremor. Patients underwent Fast Spin Echo Inversion Recovery (FSE IR) MRI imaging for stereotactic localization using the Leksell frame system. Pallidotomy target parameters of 19-22 mm lateral, 2 mm anterior to the midcommissural point and 1-7mm below the intercommissural plane were used. Microelectrode recording in GPe/GPi, and in optic tract was used to confirm target localization. Anatomic (MRI) and physiologic (microelectrode) target localizations differed by <2mm in 98% of cases. Thus, MRI directed targets were rarely altered by microelectrode recordings. Furthermore, macrostimulation was used to rule out target localization within the internal capsule and optic tract. Typically three sequential lesions were created at 840 C for 60 seconds with a 2 X 1.3mm lesion electrode. Outcome measures showed that the majority of patients were satisfied with the procedure, and that excellent to good results were obtained in 65% of patients. Age appeared to be an important determinant of outcome in that patients over 70 years had a good/excellent recovery 37% of the time, while those under 70 had good/excellent results 72% of the time. Predictors of good outcome included age < 70, dyskinesias, rigidity, bradykinesia, and tremor. Dyskinesias, bradykinesia, rigidity, tremor, and walking were all significantly improved. Some deterioration in voice volume and articulation occurred in the majority of patients, and speech deterioration was quantitatively worse with bilateral pallidotomy. Overall, in comparing unilateral and bilateral pallidotomy, improvements (e.g., dyskinesia, rigidity, tremor and walking were better after bilateral pallidotomy, whereas complications with bilateral pallidotomy were more prominent (e.g., decreased voice volume and articulation, increased drooling). In summary, pallidotomy is a valuable surgical treatment for parkinsonian symptoms, but complications, particularly with bilateral procedures, are not negligible.

NOTES

Thursday, September 19
11:20 AM

Pathophysiology of Syringomyelia Associated with Chiari I Malformation

Heiss JD, DeVroom H, Eidsath A, Talbot T, Patronas N, Oldfield, EH

Recent investigations using anatomic and cine MRI combined with intraoperative ultrasound (IOUS) in patients with syringomyelia associated with a Chiari I malformation of the cerebellar tonsils suggest that syringomyelia may result from occlusion of the subarachnoid space (SAS) at the foramen magnum by limiting the free pulsatile movement of CSF between the cranial and spinal SAS, partial entrapment of the CSF in the spinal SAS, and excess pulsatile pressure waves in the spinal SAS. To investigate this further, we performed a clinical protocol in which clinical assessment, anatomic and cine MRI, preoperative pressures measured simultaneously from the syrinx and the cervical and lumbar SAS at rest and with Valsalva and Queckenstedt maneuvers, intraoperative pressure measurements from the same sites (and a cerebral ventricle for the intraoperative component of the study) combined with IOUS monitoring of the spinal cord, syrinx, and tonsils, and postoperative MRI and pressure testing similar to the preoperative testing was performed. The pressure waves in the syrinx and SAS at the various sites were recorded digitally and the cine MRI and IOUS were related to the cardiac cycle (EKG) so that the physiological and anatomic changes that occur over the course of the cardiac cycle could be related and compared. The results, which confirm the hypothesis of the study, and their clinical significance will be presented.

NOTES

Thursday, September 19
11:45 AM

Differentiation between Cortical Atrophy and Hydrocephalus using ^{1}H MRS

Blum S, McComb JG, Ross BD

Quantitative ^{1}H MRS to determine cerebral metabolite patterns, and MRI to determine CSF flow, were applied to 12 patients with ventricular dilatation - Group A, cortical atrophy (N=5), or Group B, hydrocephalus (N=7) - and in 9 normal controls.

While mean brain water (Group A = 80% ± 6; Group B = 86% ± 5; normal = 95% ± 4) did not differ between the 2 groups of patients and controls, ^{1}H MRS distinguished those patients with cortical atrophy (Group A) (*NAA/Cr = 0.69 ± 0.17, vs normal = 1.06 ± 0.16; $P < 0.002$; [NAA] = 5.9 ± 1.3 mmoles/kg. vs normal 8.0 ± 1.4; $P < 0.02$) from those with hydrocephalus (Group B) (NAA/Cr = 1.16 ± 0.11; [NAA] = 9.2 ± 1.2; $P > 0.13$ and $P > 0.07$). Lactate levels were elevated in 3/5 patients with cortical atrophy, but 0/7 of those with hydrocephalus. Mean absolute concentrations (mmoles/kg) of the five major cerebral osmolytes were 41 ± 4 (Group A), 43 ± 6 (Group B), and 42 ± 4 (normal), so that despite massive brain deformation, constant osmolality was maintained.

Thinning of the cortical mantle in hydrocephalus may result from osmotically driven reduction in individual cell-volumes, (shrinkage), rather than brain-compression.

*N-acetylaspartate/creatine

NOTES

Thursday, September 19
12:10 PM

The Risk of Major Morbidity After Stereotactic Radiosurgery

Kondziolka D, Flickinger JC, Lunsford LD, Bissonette DJ,

Are current dose-selection guidelines acceptable for radiosurgery? We performed an analysis of major morbidity after Gamma Knife radiosurgery from a population of 1860 patients, to define the risks for parenchymal or cranial nerve deficits related to lesion type, location, size, and radiation dose. We selected prospectively a dose based on a theoretical risk of < 3% for permanent tissue injury. Morbidity occurred within 3 years.

1600 patients were evaluable at least 12 months after radiosurgery. Diagnosis included AVM (n=531). cavernous malformation (CM) (n=49), acoustic tumor (n=318), meningioma (n=204), pituitary tumor (n=63), malignant tumor (metastasis/glioma) (n=309), other tumor (n=94), and trigeminal neuralgia (n=32). We determined that major morbidity impacted function (e.g., hemiparesis, sensory loss, ataxia, hemianopsia, dysphasia, visual loss, dementia, lower cranial nerve palsy) or death. Acoustic, facial, or trigeminal deficits were excluded. Morbidity was defined by clinical exam and neuroimaging. Major morbidity developed in 30 patients (1.9%): 16 AVM (3.0%), 5 CM (10%), 1 acoustic (0.3%), 4 meningioma (2.0%), 1 pituitary (1.6%), 2 malignant tumors (0.6%), 1 other (1.1%), and no trigeminal neuralgia (0%). 11/16 AVMs were located in the brainstem or diencephalon. Lesion volume was related significantly to risk. 8 of 30 patients (27%) had received prior irradiation. 26 of 30 patients improved (87%). 14 of 1600 patients (0.9%) developed permanent major neurologic deficits after radiosurgery. Death following radiation-related neurologic morbidity occurred in 2 patients (.12%).

Dose-selection guidelines proved accurate. Major morbidity was 1.9% (below the 3% predicted risk), but was increased in patients with large lesion volumes, and those who received prior irradiation. The relative risks of radiosurgery should be compared to the risks of other surgical approaches during selection of management options.

NOTES

Thursday, September 19
12:35 PM

Drug-Regulated Apoptotic Death in a Rat Glioma Model Mediated by a Novel Retroviral Vector

John S. Yu, Miguel Sena-Esteves, Werner Paulus, Xandra O. Breakefiled, Steven A. Reeves

IL-1B-converting enzyme (ICE) is a member of a growing family of cysteine proteases shown to be a crucial component in the activation of a genetic program that leads to autonomous cell death in mammalian cells. In this study a murine ICE-*lacZ* fusion gene was introduced into a novel retroviral vector designed to achieve regulated ectopic expression of a foreign gene in mammalian cells. By delivering the ICE-*lacZ* gene within a retroviral vector and under the control of a tetracycline regulated promoter, we were able to utilize the intrinsic cell death program of ICE as a means for tumoricidal therapy in a rat brain tumor model. Both in culture and *In vivo* suppression of ICE-*lacZ* expression was extremely tight in the presence of tetracycline, as determined by the lack of X-gal positive tumor cells and by cell viability. When tetracycline was withdrawn, ICE-*lacZ* gene expression was rapidly turned on and apoptosis-mediated cell death occurred in essentially all tumor cells.

NOTES

Thursday, September 19
12:50 P.M.

***In Vivo* Angioplasty Prevents the Development of Vasospasm in Canine Carotid Arteries: Pharmacological and Morphological Analyses**

Joseph F. Megyesi, J. Max Findlay, Bozena Vollrath, David A. Cook, Ming H. Chen

Background:
In order to study the effects of *in vivo* transluminal balloon angioplasty (TBA) on the structure and function of the arterial wall, a new canine model of hemorrhagic cerebral vasospasm of the high cervical internal carotid artery (ICA) was developed. This model was used to determine if TBA performed prior to clot placement could prevent the development of vasospasm.

Methods:
Twelve dogs underwent surgical exposure of both distal cervical ICAs, followed by baseline angiography. One randomly-selected ICA in each dog was then subjected to *in vivo* TBA and repeat angiography. Both distal ICAs were then surrounded with blood clots held by silastic sheaths. Seven days later angiography was repeated and all animals sacrificed. The ICAs in four animals were perfusion-fixed *in situ* for morphological analysis by electron microscopy, and the arteries in the remaining eight animals were removed, and immediately immersed in oxygenated Krebs solution. Contractile responses of isolated arterial rings from each ICA were recorded after treatment with potassium chloride (KCl), noradrenaline (NA), serotonin (5-HT), and prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$) while relaxations were obtained after pre-constriction with NA, in response to the calcium ionphore A_{23187}, and bradykinin. The morphology and pharmacological responses of ICAs that had been exposed to blood with or without prior TBA, were compared with data obtained from control arterial segments of intact, more proximal regions of the ICAs from each animal.

Results:
TBA resulted in immediate angiographic enlargement of the ICA lumen that was still evident seven days later despire the placement of clotted blood around the artery. Scanning and transmission electron microscopy demonstrated flattening of the intima and internal elastic lamina in these dilated arteries, associated with patchy losses of endothelial cells. In contrast, ICAs which had been exposed to clotted blood but had not undergone prior TBA, developed consistent angiographic and morphological vasospasm. In comparison with control vessels and non-dilated vasospastic vessels, vessels dilated with TBA and then exposed to clotted blood showed significantly diminished responses to all compounds tested, with the exception of $PGF_{2\alpha}$ and bradykinin.

These results indicate that *in vivo* TBA results in a degree of functional impairment of vascular smooth muscle that persists for at least seven days. This result is consistent with previous observations of the acute effects of TBA in isolated arteries. Furthermore, these results support the hypothesis that normal smooth muscle function is required for the development of vasospasm. Finally, these results indicate that TBA performed prior to the onset of vasospasm prevents its development.

NOTES

Friday, September 20, 1996
10:35 AM
Extra-Foraminal Lumbar Disc Herniations
Rajaraman V, Swami N, Tobler WS, and Dunsker SB

Because of the advent of CT and MRI imaging, extra-foraminal or extreme lateral lumbar disc herniations are diagnosed more easily. To approach these lesions, some individuals continue to perform a unilateral laminectomy and facetectomy, but far lateral (extra-spinal) approaches are being performed more frequently. Moreover, recent biomechanical studies indicate there is increased translational movement following facetectomy alone, and the movements are worsened by combining facetectomy and discectomy.

We will present the important anatomy to know when performing these extra-spinal procedures, including the demonstration of an arterial arcade which encircles the main nerve trunk, and which endangers it. We believe that proper attention to this arcade and to the attached posterior primary ramus of the nerve will prevent the painful burning dysesthesias that commonly follow the extra-spinal approach.

We will also present our clinical experience with the procedure.

NOTES

Friday. September 20
11:00 AM
The Miami Project - Scientific Achievements
Green B. Bunge RP

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 faculty are members of the Department of Neurological Surgery who lead 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 spinal cord based central pattern generator for locomotion can be detected in selected cases of human spinal cord injury; 5) established that axons of the long tract of the spinal cord can be induced to regenerate by favorable cellular environment at the site of injury (for example transplantation of Schwann cells to 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 manipulations 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

Friday, September 20
11:20 AM

Clinical and Experimental Studies of the Value of Surgical Decompression in Spinal Cord Injury

Tator, C

There is a need for a randomized control trial of surgical decompression in patients with persisting compression of the cord after spinal cord injury. There is no definite evidence that decompression improves neurological recovery after either complete or incomplete acute spinal cord injury.

Several retrospective clinical studies have been performed including one by the author, which have failed to show improved recovery following surgical decompression. In contrast, several well controlled experimental studies including two from the author's laboratory have shown improved recovery following posttraumatic decompression of the spinal cord in various species. This presentation will review the past and current clinical and experimental studies of posttraumatic decompression of the cord. In addition, the results will be described of a recent retrospective analysis of the current incidence (66%) of surgical treatment of spinal cord injury in 36 U.S. and Canadian centres. Many of these operations were performed for purposes of decompression. The current protocol will be discussed for a proposed prospective control trial of surgical decompression in patients with persisting acute traumatic compression of the spinal cord.

NOTES

Saturday, September 21
8:25 AM

AVM Radiosurgery: Truths and Consequences

Lunsford LD, Pollock BE, Kondziolka D, Maitz A, Flickinger JC

Introduction:

To clarify recent pejorative reports about outcomes of AVM radiosurgery, we reviewed our experience in 315 patients evaluable at least 3 years afterwards. The obliteration and risk rates were assessed.

Methods & Materials:

Follow-up was available 98%; 197 patients (63%) completed 3 year angiography. MRI was used serially to assess AVM flow and to detect complications.

Results:

The overall angiographically confirmed obliteration rate was 72%. Depending on volume, the obliteration rate varied from 41% to 98%. The minimum nidus dose was the only significant predictor of obliteration. The major cause for failure was poor nidus resolution during intraoperative imaging. MRI proved to have a 96% sensitivity and a 100% specificity of detecting residual AVM flow. No difference in outcomes or obliteration rates were detected in patients who had only MRI follow-up. Deep AVMs had a 5x increased risk of neurological sequelae. A recent AVM hemorrhage before radiosurgery increased the risk of a repeat hemorrhage during the latency interval prior to obliteration. No AVM bled after MRI demonstrated obliteration.

Conclusions:

AVM radiosurgery is an effective strategy for many AVMs. Intraoperative MRI and angiography are critical to define the target volume. MRI alone can be used effectively to detect success, failure, and complications. AVMs with volumes that prevent minimal marginal doses > 16Gy should be considered for other treatment options, including staged radiosurgery.

NOTES

Saturday, September 21
8:45 AM

Mechanism of Radiation Effect on Arterial Smooth Muscle Cells and Endothelium

Mayberg, M

To determine by which radiation modulates injury for vascular smooth muscle cells (SMC) and endothelium, 108 rats were divided into three groups: (A) unilateral irradiation to cervical common carotid artery, (B) unilateral carotid balloon catheter injury, and (C) bilateral balloon injury with unilateral irradiation. Animals received from 100-2000 cGy (rads) and carotid arteries were processed for immunohistochemistry at 0-72 hours after treatment with terminal transferase end labeling (TUNEL - to detect apoptosis) and in situ nick translation (ISNT - to detect necrosis). Cultured rat aortic SMC and endothelium were placed in either serum-free medium (growth-arrested) or whole blood serum (growth-stimulated), radiated with 500-2000 cGy, and processed for TUNEL and ISNT at 0-72 hours.

Balloon catheter injury only (group B) produced immediate necrosis in SMC on the abluminal aspect of the media. The addition of radiation to balloon injury (group C) did not affect either TUNEL or ISNT labeling. Radiation alone (group A) caused dose-dependent apoptosis in endothelium, but not SMC; no necrosis was observed at any dose. Similarly, irradiated cultured SMC in either media showed mild morphologic changes compared to controls, but no evidence of necrosis or apoptosis up to 2000 cGy. Endothelium in vitro, on the other hand, showed considerable sensitivity to radiation with significant apoptosis at doses as low as 500 cGy, in both media. These data suggest that the primary effect of gamma radiation on medium-sized arteries involves apoptosis in endothelial cells. Strategies to either inhibit or augment the effect of radiation on arterial endothelium may be useful in future treatment of intracranial lesions.

NOTES

Saturday, September 21
9:05 AM

The Role of Neuronal NO in Collateral and Normal rCBF

Loftus CM, Robertson SC

N-Methyl-D-aspartate (NMDA) produces cerebral vasodilation in response to receptor activation, probably mediated by the neuronal isoform of nitric oxide (NO) synthase. Our experiments were studied the effects of NMDA and selective inhibition and neuronal NO synthase with 7-nitroindazole (7-NI) on collateral and normal cerebral blood flow (rCBF) following MCA occlusion. In 9 dogs under halothane anesthesia, a left craniotomy was performed. An MCA branch was cannulated for determination of collateral dependent tissue (CDT). CBF was measured using radioactive microspheres and cerebral vascular pressures were measured with a glass micropipette.

NMDA (300 *u* M) increased flow to DCT by 54.6%, while normal ipsilateral and contralateral cerebrum increased by at least 35% from baseline. 7-NI caused a significant drop in rCBF, with the greatest drop of 33.8% occurring in CDT. Normal ipsilateral and contralateral rCBF were reduced by 32.3% and 20.2% respectively. The response to NMDA was significantly attenuated following 7-NI administration as rCBF actually decreased further from baseline. Vascular resistance decreased in response to NMDA; 7-NI caused a significant increase in cerebral vascular resistance.

In our study agonism at NMDA receptors following MCA occlusion increased blood flow significantly to CDT and normal cerebrum, probably due to the production of neuronal NO resulting in vasodilation. Blockade of neuronal NO synthase activity with 7-NI reduced rCBF to all regions of the brain from increase usually seen in response to NMDA administration. Neuronal NO production appears to play an important role in regulating vasculating vascular tone and CBF and may be especially important in maintaining flow to CDT following MCA occlusions.

NOTES

Saturday, September 21
9:25-9:45 AM

Repair of Carotid Endarterectomy with Collagen Dacron Graft

Meyer FB, Windschitl WL

Closure of the arteriotomy following carotid endarterectomy with a saphenous vein patch graft may decrease the risk of acute postoperative occlusion and recurrent stenosis. However, disadvantages of a vein patch include postoperative rupture (0.15%) and pseudoaneurysm formation. This report analyzes preliminary results using a Hemashield microvel knitted double velour graft. From 1/93 through 7/95, 190 consecutive carotid endarterectomies were repaired with the synthetic graft. The surgical management included intraoperative monitoring with selective shunting, infusion of heparin 5000 units prior to cross clamping, pre and postoperative aspirin 325 mg qd, and perioperative antibiotics. All patients had postoperative noninvasive assessment of vessel patency prior to discharge with either carotid ultrasound or oculoplethysmography. There were no wound hematomas, perioperative occlusions, or graft infections. There was 1 major (embolic) and 1 minor (hemodynamic) stroke occurring in Sundt Grades 3 and 4, respectively. There was 1 death from cardiac arrest which occurred 10 days after surgery at home following combined cardiac and carotid revascularization. There were 2 patients involving 3 vessels who developed recurrent stenosis. The 1 patient who developed bilateral recurrent stenosis 9 months after the second surgery appeared to have a dense fibrotic reaction to surgery as evidenced by hypertrophic scar formation of the skin and myointimal hyperplasia of the endarterectomy site. The second patient developed a focal proximal stenosis underneath the graft site 6 months after surgery. These preliminary results suggest that this synthetic graft may be a viable alternative for those surgeons who prefer to repair a carotid endarterectomy with a patch graft.

NOTES

Saturday, September 21
11:00 AM

Drug Delivery by Intracerebral Clysis in a Rat Glioma Model

Bruce J, Birch B, Johnson J, Yoon J, Lycette C, Falavigna A

To circumvent the limitations of a systemic chemotherapy for malignant brain tumors, a novel local drug delivery method known as intracerebral clysis has been developed which involves the use of high flow microinfusion directly into brain tumors by means of a controllable pump. To establish baseline parameters for preclinical, in vivo drug investigations of intracerebral clysis in a rat glioma model, we have studied the effects of varying infusion parameters on intracranial pressure, infusate distribution and clinical therapeutic response.

C6 rat glioma cells were stereotactically implanted into the frontal lobe of male Fischer rats. Between 10 to 25 days later, catheters were stereotactically implanted into the tumors and 100 ul of 25% albumin was infused at flow rates from 0.5 to 4 ul/min while monitoring ICP. Fluoresceinated dextran (MW 20,000 D) was then infused under similar conditions and the distribution volumes were calculated after brain sectioning and photography under UV light.

Flow rates less than 1 ul/min were tolerated well even after 25 days of tumor growth. The increase in ICP with increasing flow volumes was progressively greater as the flow rate was increased. Flow rates greater than 3 ul/min were associated with morbidity and mortality. Volumes of distribution at tolerated flow rates were sufficient to provide adequate drug delivery to tumors. Preliminary results demonstrating efficacy of several anti-tumor compounds delivered by intracerebral clysis will also be presented.

NOTES

Saturday, September 21
11:20-11:40 AM

Comprehensive Image Guided Surgery of Sellar Tumors

Schulder MA, Carmel P

More than 25 years ago, the advent of the operating microscope and fluoroscopic C-arm rehabilitated the transsphenoidal approach to the sella, making it the route of choice for most patients with pituitary tumors. However, significant obstacles still limit the radical removal of these tumors, including; 1) uncertainty of limits of cavernous sinus extension; 2) lack of lateral landmarks, especially in patients with larger, more invasive tumors; 3) "tunnel" vision of the operating microscope does not provide angles needed to visualize portions of tumor.

We have employed new technical advances to optimize surgical planning and removal of sellar tumors. Techniques utilized included: 1) differential hysteresis image processing, which defines cavernous involvement more clearly than standard MR scans; 2) Frameless stereotactic navigation, which allows precise evaluation of lateral resection extent; and 3) working channel endoscopy, which allows visualization and removal of tumor which lies above the planum, upper clivus, and laterally within and above the sella.

These techniques will be illustrated. They have the potential to considerably enhance the scope and safety of transsphenoidal operations. We believe they can be shown to be cost effective in treating patients with these tumors.

NOTES

Saturday, September 21
11:40 AM

Dorsal Cervical-Medullary Bulge in Chiari I Malformations

Carmel PW, Bello JA

A dorsal surface bulge underlying the tonsillar prolapse of Chiari I malformations has been reported by a number of authors. Surprisingly, there has been no systematic study of this associated deformity, correlation with clinical symptomatology, or explanation of its pathogenesis. This report details the pre- and post-operative MR findings, clinical and operative findings and incidence in a series of 137 patients with Chiari I malformations.

In adequately studied Chiari I patients, dorsal deformity was found in 19% (18/99). Patients with dorsal deformities had a higher incidence of lower cranial nerve problems, including a "medullary cough" syndrome. Bulges were infrequent in children and adolescents, and more frequent in adults.

Post-op MRI's showed progressive resolution of this deformity. These data indicate that the bulge is reversible. It is likely to be secondary to progressive tonsillar prolapse and is acquired, rather than congenital, in origin. It is not an anatomically "fixed" malformation.

NOTES

Saturday, September 21
12:00 noon

Brain Tumor Invasion-Biological Rationale and Preclinical Studies

Rosenblum ML, Mikkelsen T, Edvardsen K, Nelson K, Kohn E, Smith R, Spencer D, Rasnick D, Sloane B

The invasiveness of malignant glioma cells is a major cause of therapeutic failure both locally and at distant sites within the brain. We have evaluated the clinically relevant pathways of tumor spread and hypothesized that increased protease activity, cell receptor (integrin) interactions with specific extracellular matrix (ECM) proteins, and second messenger systems are the most logical targets against which to develop antiinvasive strategies. Cathepsin B (CB) is elevated in malignant gliomas, excreted into the extracellular space and prominent in invading cells (Cancer Res 54:6027-6031, 1994). We have utilized two types of specific, irreversible CB inhibitors to demonstrate a 10-70% inhibition of invasion for U251MGn, U87MG and HF66 malignant glioma cells using a Matrigel ECM invasion assay. A clinically relevant spheroid confrontation assay that demonstrates human brain tumor cell invasion into organotypic normal brain aggregates derived from fetal rats was used to show a marked inhibition of invasion using all the CB inhibitors. We have demonstrated the deposition of laminin by normal brain cells at the invading front of tumor cells in both the spheroid confrontation assay as well as an in vivo model for brain tumor invasion. A simultaneous increase in the expression of a laminin-associated integrin in tumor cells was also observed. Furthermore, antiintegrin antibodies were shown to decrease tumor cell invasion in the Matrigel ECM assay. Carboxy-amido-triazole (CAI) is an inhibitor of G-protein-mediated signal transduction and has been developed as an antimetastasis agent. We have shown that 2-20 uM CAI inhibits Matrigel invasion of glioma cell lines by an average of 60% without killing cells. CAI also inhibits tumor cell invasion in the spheroid confrontation model. We conclude that protease inhibitors and agents which block integrin-mediated adhesion and second messenger systems could be developed as novel antiinvasive strategies to treat malignant gliomas.

NOTES

Saturday, September 21
12:20 PM

Immunobiology of Primary Brain Tumors

Brooks WH, Roszman TL, Elliott LE

Individuals harboring primary malignant tumors manifest a broad variety of immunological deficits. These anomalies are confined primarily to the T-helper cell subset and are linked to decreased production of IL-2 and failure to properly assemble the high-affinity receptor for IL-2 in the membrane of stimulated T lymphocytes. In this study, the early transmembrane signaling events on which normal T-cell function is contingent were examined. Concurrently, the mechanism(s) of how these and/or other biochemical and molecular abnormalities are induced in the T-cell was investigated. The results demonstrate that early transmembrane signaling via the T-cell receptor/CD3 complex of lymphocytes obtained from glioma bearing individuals is not normal; these cells are found to have an intrinsic defect which renders them unable to respond to appropriate proliferative stimuli. Concurrently, these investigations reveal that soluble factors secreted by malignant glial cells induce programmed cell death (apoptosis) in T cells. Collectively, the present study indicates that immune suppression observed in patients with malignant gliomas is contingent on and may be correlated with tumor size and the eliberation of glial T cell suppresser factors(s) (GSF). These findings further contribute to elucidating the immunobiological potential of these patients. The relevancy of this immunobiological model to immune modulation as a therapeutic option will be discussed.

NOTES

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Four Seasons Biltmore,
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Division of Neurosurgery
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420 Delaware Street, S.E.
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University Station
Birmingham, AL 35294

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Louisville, KY 40202

SIDNEY GOLDRING (Lois) 1964
#1 Barnes Hospital Plaza
Neurosurgery
St. Louis, MO 63110

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Casper, WY 82604

EVERETT GRANTHAM (Mary) 1942
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210 Gray Street
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Apt. #306/Eastlake
601 S.W. 6th Street
Pompano Beach, FL 30060

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

HAROLD HOFFMAN (Jo Ann) 1982
Hospital for Sick Children
555 University Avenue
Toronto, ONTARIO M5G 1X8
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
Charlotteville, VA 22908

PETER J. JANNETTA (Diana) 1994
Department of Neurological Surgery
Presbyterian University Hospital
Suite B-400
Pittsburgh, PA 15213

ELLIS KEENER (Ann) 1978
915 East Lake Drive, NW
Gainesville, GA 30506

WILLIAM KELLY (Joan) 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 (Joan) 1967
University of California at Irvine Medical Center
101 The City Drive South
Building 3, Room 313
Orange, CA 92868-3298

THOMAS LANGFITT (Carolyn) 1971
Glenmede Corporation
One Liberty Place, Suite 1200
1650 Market Street
Philadelphia, PA 19103-7391

SANFORD LARSON (Jacquelyn) 1989
Department of Neurosurgery
9200 W. Wisconsin Ave.
Milwaukee, WI 53226

RAEBURN C. LLEWELLYN (Carmen Rolon) 1963
#3 Poydras Street
Unit 8-B
New Orleans, LA 70130

DON M. LONG (Harriett) 1983
Dept. of Neurological Surgery
Johns Hopkins Medical School
600 N. Wolfe, Meyer 7-109
Baltimore, MD 21287-7709

WILLIAM LOUGHEED 1962
178 Klempenfeld Drive
Barrie ON L4M 1C3
Canada

JOHN J. LOWREY (Catherine {Katy}) 1965
Box 6989
65-1160 Hokuula Road
Kamuela, 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
415 Bond Place #8A
Cincinnati, OH 45206

WILLIAM MEACHAM (Alice) 1952
709 St. Thomas Medical Plaza East
Nashville, TN 37205

SEAN 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
Durhan, NC 27710

GUY ODOM 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
50 Museum Way
San Francisco, CA 94114

J. LAWRENCE POOL 1940
41 Cherry Hill Road
Westcornwall, CT 06796

ROBERT W. PORTER (Dean) 1962
6461 Bixby Hill Road
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. 2oth 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

ALBERT RHOTON, JR. (Joyce) 1984
Department of Neurological Surgery
College of Medicine, P.O. Box 100265
University of Florida
Gainesville, FL 32610

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

EDWARD L. SELJESKOG (Peg) 1992
2805 Fifth Street South
Suite 110
Rapid City, SD 5770!

C. HUNTER SHELDEN (Betty) 1941
Huntington Medical Research Inst.
10 Pico Street
Pasadena, CA 91105

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

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

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
Route 3
Box 140-J
Burgess, VA 22432

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 (Wendy) 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
735 Peachtree Battle Avenue, NW
Atlanta, GA 30327

ARTHUR A. WARD, JR. (Janet) 1953
Dept. of Neurological Surgery, Univ. of WA
Seattle, WA 98104

BENJAMIN B. WHITCOMB (Peggie) 1947
RDI Box 124
Surrey, ME 04684

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

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

CHARLES B. WILSON (Francie Petrocelli) 1966
Dept. of Neurological Surgery
Univ. of California - San Francisco
533 Parassus Ave., U125 Box 0350
San Francisco, CA 94143-0112

DAVID YASHON 1972
#1201 1492 E. Broad Street
Columbus, OH 43205

NICHOLAS T. ZERVAS (Thalia) 1972
Massachusetts General Hospital
32 Fruit Street
Boston, MA 02114

ACTIVE MEMBERS

Elected

MICHAEL APUZZO (Helene) 1988
1200 N. State Street, Ste. 5046
Los Angeles, CA 90033

JAMES AUSMAN (Carolyn) 1979
Univ. of Illinois-Chicago
Dept. of Neuro/ M/C 799
912 S. Wood St.
Chicago, IL 60612

DANIEL BARROW (Molly) 1993
Dept. of Neurological Surgery
Emory Clinic
1365 Clifton Ave., N.E.
Atlanta, GA 30322

KEITH L. BLACK (Carol Bennett) 1995
UCLA Medical Center, Div. of Neurosurgery
10833 Le Conte Ave., 18-228 NPI
Los Angeles, CA 90095-7039

PETER MCL. BLACK (Katharine) 1988
Brigham and Women's Hospital
75 Francis Street
Boston, MA 02115

LAWRENCE F. BORGES (Susan) 1993
Massachusetts General Hospital
Div. of Neurosurgery, White 1205
32 Fruit Street
Boston, MA 02114

WILLIS BROWN, JR. (Ann) 1984
Division of Neurosurgery
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

KIM J. BURCHIEL (Debra) 1992
Division of Neurosurgery
Oregon Health Sciences University
3181 S.W. Sam Jackson Park Rd.
Portland, OR 97201-3098

MARTIN B. CAMINS (Joan) 1995
205 E. 68th Street
Suite T 1-C
New York, NY 10021

PETER W. CARMEL (Jacqueline Bello) 1991
Suite 7300
90 Bergen Street
Newark, NJ 07103-2499

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

PAUL COOPER (Leslie) 1995
550 First Avenue
New York, NY 10016

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 Euclid
St. Louis, MO 63110

ARTHUR L. DAY (Dana) 1990
University of Florida Health Center
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
California Pacific Medical Center
2100 Webster Street
Suite 420
San Francisco, CA 94115

HOWARD EISENBERG (Janet) 1985
Division of Neurosurgery
University of Maryland
22 S. Greene Street
Baltimore, MD 21201

MEL H. EPSTEIN (Lynn) 1992
Rhode Island Hospital
Department of Neurosurgery
Suite 505
Medical Office Center
2 Dudley Street
Providence, RI 02905

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

WILLIAM A. FRIEDMAN (Ransom) 1995
University of Florida Health Sciences Center
P.O. Box 100265, Neurosurgery
Gainesville, FL 32610-0265

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) 1986
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 Deleware 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., D4-6
Miami, FL 33136

CHARLES HODGE, JR. (Cathy) 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

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 G. KLINE (Helen) 1971
Department of Neurosurgery
Louisiana State University Medical Center
1542 Tulane Avenue
New Orleans, LA 70112

EDWARD R. LAWS, JR. (Margaret {Peggy}) 1983
Department of Neurosurgery
Box 212 HSC
University of Virginia
Charlotteville, VA 22908

CHRISTOPHER LOFTUS (Sara J. Sirna) 1992
Div. of Neurosurgery, Univ of Iowa Hosp.
200 Hawkins Drive, 1844 JPP
Iowa City, IA 52242

L. DADE LUNSFORD (Julie) 1992
B-400, Presbyterian University Hospital
200 Lothrop Streeet
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
Box 142, UMHC
420 Delaware Street, S.E.
Minneapolis, MN 55455

MARC R. MAYBERG (Terry) 1995
Department of Neurosurgery
Box 356470
University of Washington Medical Center
Seattle, WA 98195

J. M. MCWHORTER (Barbara) 1989
2810 N. Maplewood.
Winston-Salem, NC 27103

FREDRIC B. MEYER (Irene)
Department of Neurological Surgery
Mayo Clinic
200 First Street, S.W.
Rochester, MN 55905

RICHARD MORAWETZ (Mary Jean) 1990
University of Alabama
Division of Neurosurgery
MEB 512
Birmingham, AL 35294

PAUL B. NELSON (Tere) 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
1959 N.E. Pacific Street
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., St. 209
Miami, FL 33133

DAVID G. PIEPGRAS (Jane) 1987
Department of Neurological Surgery
Mayo Clinic, 200 First Street, S.W.
Rochester, MN 55905

KALMON D. POST (Linda) 1995
The Mount Sinai Medical Center
1 Gustave L. Levy Place
Box 1136
New York, NY 10029-6574

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
Neurosurgery-University Hospital
11100 Euclid Avenue
M.S. 5042
Cleveland, OH 44106

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

WARREN R. SELMAN (Diana) 1995
Department of Neurological Surgery
University Hospitals of Cleveland
11100 Euclid Avenue, HH-5th Floor
Cleveland, OH 44106

CHRISTOPHER SHIELDS (Deborah) 1993
Department of Neurosurgery
University of Louisville
210 E. Gray St., Suite 1102
Louisville, KY 40202

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

VOLKER K. H. SONNTAG (Lynn) 1995
Barrow Neurological Institute
Division Neurological Surgery
2910 North 3rd Avenue
Phoenix, AZ 85013

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

HARRY VAN LOVEREN (Judy) 1995
3219 Clifton Avenue
Suite 110
Cincinnati, OH 45220

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

H. RICHARD WINN (Debbie) 1993
Univ. of WA, School of Medicine
Dept. of Neurosurgery
325 Ninth ZA86
Seattle, WA 98104

FREMONT P. WIRTH (Penny) 1993
4 Jackson Blvd.
Savannah, GA 31405-5895

ALLEN WYLER (Lily) 1990
Epilepsy Center, Swedish Medical Center
747 Summit
Seattle, WA 98104

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 (Terry) 1994
Medical Col. of Virginia Station
P.O. Box 980631
Richmond, VA 23298

INACTIVE

Elected

ROBERT BOURKE — 1983
5802 Nicholson Lane, St. 305
Rockville, MD 20852
(301) 881-4567

JOHN KAPP — 1985
P.O. Box 448
Galax, VA 24333
(703) 236-2613

RICHARD S. KRAMER — 1978
Duke University Medical Center
Box 3255
Durham, NC 27710

SENIOR CORRESPONDING — ELECTED

JEAN BRIHAYE (van Geertruyden) — 1975
Belgium 98
avenue Des Franciscains
Brussels, BELGIUM

KARL-AUGUST BUSHE (Eva-Christa) — 1972
Lerchenweg 8
D-97074 Wurzburg
GERMANY

FERNANDO CABIESES — 1966
Peruano De Formento Educativo
Av. Arenales 371, of. 501
Apartado 5254
Lima, PERU

JUAN CARDENAS (Delores) — 1966
Insurgentes Sur 594
Av. Insurgentes
Mexico City, 40
MEXICO

JUAN CHRISTENSEN (Diana Poli) — 1970
José C. Paz 234
Acassusi (1641)
Buenos Aires Province
ARGENTINA

HANS ERICH DIEMATH (Karin) — 1970
Landesnervenklinik, Dept. of Neurosurgery
5020 Salzburg, Ignaz Harrer-StraBe 79
AUSTRIA

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
Universitatsklinik 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

GUISEPPE DALLA ORE (Guisi) 1970
Clinica Neurochirurgica
Universita di Verona
Plazzale 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
Bangkog
THAILAND

LINDSAY SYMON (Pauline) 1982
Gough-Cooper Dept. of Neurological Surgery
Institute of Neurology, The National Hospital
Queen Square London WCIN 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 (Sally Anne) 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

NICHOLAS DE TRIBOLET 1995
Service de Neurochirugie
Centre Hospital
Universite Vaudois
Lausanne, CH1011
SUISSE

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 FAHLBUSH (Hanna) 1991
Neurochirurgische Klinik
University; Erlangen-Nurnberg
Schwabachanlage 6
91054 Erlangen
GERMANY

SALVADOR GONZALEZ-CORNEJO (Rosa) 1982
Av. Chapultepec Sur 130-204
Guadalahara, 44140
MEXICO

ERNST GROTE (Juliana) 1984
Department of Neurosurgery
University Kliniks Schnarrenberg
Hoppe Seyler-Str. 3
7400 Tubingen
GERMANY

DAE HEE HAN (Sung Soon Cho) 1991
SNU Hospital
Seoul Nat'l, Univ., School of Medicine
#28 Youngon-dong
Chongno-Gu, Seoul 110-744
KOREA

HAMIME 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

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

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

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

DAVID G.T. THOMAS (Hazel) 1995
Institute of Neurology
Queens Square
London WC1 3BG
ENGLAND

DECEASED MEMBERS

	Deceased	Elected
JAMES R. ATKINSON Phoenix, Arizona (Active)	1978	1970
PERCIVAL BAILEY Evanston, Illinois (Honorary)	1973	1960
GEORGE BAKER Litchfield Park, AZ (Senior)	1993	1940
H. THOMAS BALLANTINE, JR. Boston, Massachusetts (Senior)	1996	1951
WILLIAM F. BESWICK Buffalo, New York (Active)	1971	1959
EDWIN B. BOLDREY San Francisco, California (Senior)	1988	1941
SPENCER BRADEN Cleveland, Ohio (Active)	1969	Founder
F. KEITH BRADFORD Houston, Texas (Active)	1971	1938
HOWARD BROWN San Francisco, California (Senior)	1990	1939

GALE CLARK Oakland, California (Senior)	1996	1970
DONALD COBURN Wilbington, Delaware (Senior)	1988	1938
WINCHELL McK. CRAIG Rochester, Minnesota (Honorary)	1960	1942
EDWARD DAVIS Portland, Oregon (Senior)	1988	1949
PEARDON DONAGHY Burlington, Vermont (Senior)	1991	1970
FRANCIS ECHLIN New POaltz, New York (Senior)	1988	1944
DEAN ECHOLS New Orleans, Louisiana (Senior)	1991	Founder
GEORGE EHNI Houston, Texas (Senior)	1986	1964
ARTHUR ELVIDGE Montreal, Quebec, Canada (Senior)	1985	1939
THEODORE C. ERICKSON Madison, Wisconsin (Senior)	1986	1940
JOSEPH P. EVANS Kensington, Maryland (Senior)	1985	Founder

JOHN FRENCH Los Angeles, California (Senior)	1989	1951
JOHN GREEN Phoenix, Arizona (Senior)	1990	1953
JAMES GREENWOOD, JR. Houston, Texas (Senior)	1992	1952
WESLEY A. GUSTAFSON Jensen Beach, Florida (Senior)	1975	1942
HANNIBAL HAMLIN Providence, Rhode Island (Senior)	1982	1949
JOHN W. HANBERY Palo Alto, CA (Senior)	1996	1959
JESS HERRMANN Oklahoma City, OK (Senior)	1994	1938
HENRY L. HEYL Hanover, New Hampshire (Senior)	1975	1951
OLAN HYNDMAN Iowa City, Iowa (Senior)	1966	1942
KENNETH G. JAMIESON Brisbane, Australia (Corresponding)	1976	1970
SIR GEOFFREY JEFFERSON Manchester, England (Honorary)	1961	1951

WILLIAM S. KEITH Toronto, Canada (Senior)	1987	Founder
HUGO KRAYENBUHL Zurich, Switzerland (Honorary)	1985	1974
KRISTIAN KRISTIANSEN Oslo, Norway (Senior corresponding)	1993	1967
WALPOLE S. LEWIN Cambridge, England (Corresponding)	1980	1973
HERBERT LOURIE Syracuse, New York (Senior)	1987	1965
WILLEM LUYENDIJK Oegstgeest, the Netherlands (Senior Corresponding)	1996	1973
M. STEPHEN MAHALEY Birmingham, Alabama (Active)	1992	1972
GEORGE L. MALTBY Scarsborough, Maine (Senior)	1988	1942
FRANK MARGUTH Munich, Germany (Senior Corresponding)	1991	1978
DONALD D. MATSON Boston, Massachusetts (Active)	1969	1950
FRANK MAYFIELD Cincinnati, Ohio (Senior)	1991	Founder

AUGUSTUS McCRAVEY Chattanooga, Tennessee (Senior)	1990	1944
KENNETH G. McKENZIE Toronto, Ontario, Canada (Honorary)	1964	1960
JAMES M. MEREDITH Richmond, Virginia (Active)	1962	1946
J. DOUGLAS MILLER Edinburgh, Scotland (Corresponding)	1995	1988
W. JASON MIXTER Woods Hole, Massachusetts (Honorary)	1968	1951
EDMUND J. MORRISSEY San Francisco, California (Senior)	1986	1941
FRANCIS MURPHEY Naples, FL (Senior)	1994	Founder
GOSTA NORLEN Goteborg, Sweden (Honorary)	1985	1973
FRANK NULSEN Naples, Florida (Senior)	1994	1956
SIXTO A. OBRADOR Madrid, Spain (Honorary)	1978	1973
PIETRO PAOLETTI Milan, Italy (Corresponding)	1991	1989

HANS-WERNER PIA Giessen, West Germany (Corresponding)	1986	1978
WILDER PENFIELD Montreal, Canada (Honorary)	1976	1960
HELMUT PENZHOLZ Heidelberg, West Germany (Corresponding)	1985	1978
RUPERT R. RANEY Los Angeles, California (Active)	1959	1939
BRONSON RAY New York, New York (Honorary)	1993	1992
DAVID L. REEVES Santa Barbara, California (Active)	1970	1939
DAVID REYNOLDS Tampa, Florida (Active)	1978	1964
R.C.L. ROBERTSON Houston, Texas (Senior)	1985	1946
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

W. KEASLEY WELCH Waban, Massachusetts (Senior)	1996	1957
BARNES WOODHALL Durham, North Carolina (Senior)	1985	1941
FRANK WRENN Greenville, South Carolina (Senior)	1990	1973