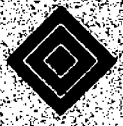
THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

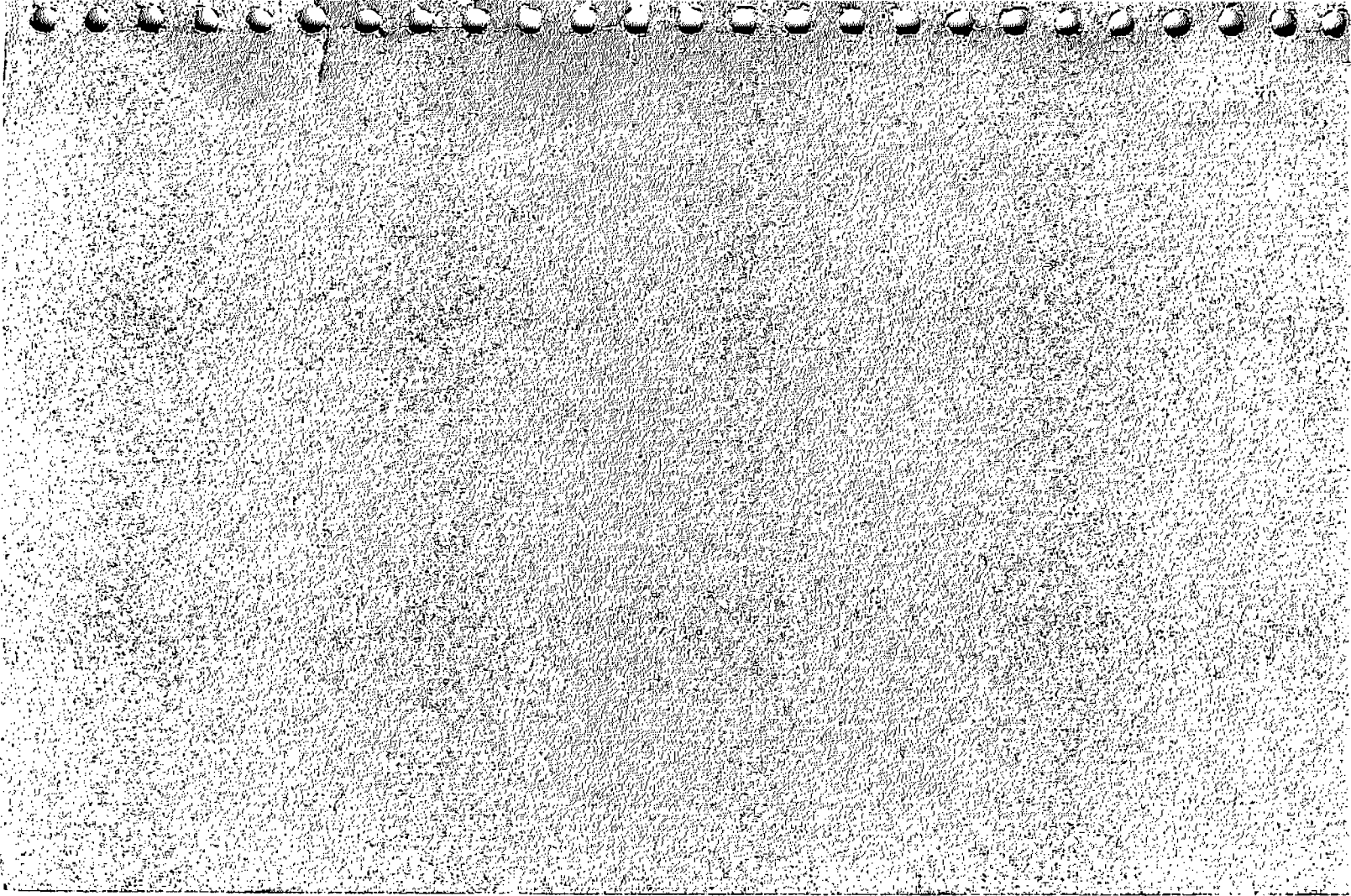
October 27-30, 1993



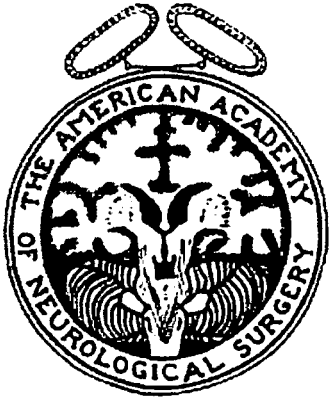
THE WIGWAM

Authentic Arizona

Litchfield Park, Arizona



The American Academy of Neurological Surgery



55th Annual Meeting

Wigwam Resort

Litchfield Park, Arizona

October 27 -30, 1993

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THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
ACTIVITIES PROGRAM
OCTOBER 27 - 31, 1993

WEDNESDAY,OCTOBER 27:

12:00PM - 4:00PM	Registration Wigwam Foyer
6:00PM - 9:00PM	Reception Wigwam Terrace (B/U Sachem Hall)

THURSDAY, OCTOBER 28:

7:00AM - 8:00AM	Breakfast/Business Mtg. **(Members only)** Sachem West
8:00AM - 1:00PM	Registration Wigwam Foyer
8:00AM - 10:10AM	General Scientific Session Hopi/Pima
10:10AM - 10:40AM	Coffee Break
10:40AM - 1:00PM	General Scientific Session Hopi/Pima
1:00PM	Golf and Tennis; Free Time
5:45PM	Transportation to Sunset Pointe, Porte Cocher
6:00PM - 7:00PM	Reception Sunset Pointe (B/U Aztec/Hopi)
7:00PM - 10:00PM	Western Cookout Sunset Pointe (B/U Aztec/Hopi)

FRIDAY, OCTOBER 29:

7:00AM - 8:00AM	Breakfast/Business Mtg. **(Members only)** Wigwam Terrace (B/U Wigwam Foyer)
8:00AM - 1:00PM	Registration Wigwam Foyer
8:00AM - 10:00AM	General Scientific Session Hopi/Pima
10:00AM - 10:20AM	Coffee Break
10:20AM - 1:00PM	General Scientific Session Hopi/Pima
1:00PM	Golf and Tennis; Free Time
6:00PM - 6:30PM	President's Reception Suite 633 **(By invitation)**
6:30PM - 7:30PM	Reception Sachem Terrace (B/U Aztec/Hopi)
7:30PM - 10:00PM	Dinner Aztec/Hopi

SATURDAY, OCTOBER 30:

7:00AM - 8:00AM	Breakfast **(Members and Guests)** Wigwam Terrace (B/U Wigwam Foyer)
8:00AM - 10:20AM	General Scientific Session Hopi/Pima
10:20AM - 10:40AM	Coffee Break

SATURDAY, OCTOBER 30:

10:20AM - 1:00PM — General Scientific Session
Hopi/Pima

SUNDAY, OCTOBER 31:

Departures

SIGNIFICANT OTHER ACTIVITIES.

WEDNESDAY, OCTOBER 27:

6:00PM - 9:00PM	Reception Wigwam Terrace (Backup is Sachem Hall)

THURSDAY, OCTOBER 28:

8:00AM - 9:30AM	Continental Breakfast East Pool Patio (Backup is Aztec A/B)
9:30AM - 11:00AM	Jewelry Exhibit/ Sampling East Pool Patio (Backup is Aztec A/B)
1:00PM	Golf and Tennis; Free Time
5:45PM	Transportation to Sunset Pointe, Porte Cochere
6:00PM - 7:00PM	Reception Sunset Pointe (Backup is Aztec/Hopi)
7:00PM - 10:00PM	Western Cookout Sunset Pointe (Backup is Aztec/Hopi)

FRIDAY, OCTOBER 29:

8:00AM - 9:30AM	Continental Breakfast Sachem Terrace (Backup is Aztec A/B)
10:00AM - 11:00AM	Spouse's Aerobic Exercise
1:00PM	Golf and Tennis; Free Time
7:30PM - 10:00PM	Dinner Aztec/Hopi

SATURDAY, OCTOBER 30:

8:00AM - 9:30AM	Continental Breakfast East Pool Patio

PROGRAM
AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
OCTOBER 27-30, 1993

Thursday, October 28

8:00 AM	**Welcome** Dr. George Tindall
8:00 AM	**Scientific Session I** Moderator: George Tindall
8:00 AM	G. Heishima, R. Higashida, V. Halbach, C. Dowd, C. Wilson, T. Terada *Dural Arteriovenous Fistulas: Pathogenesis and Progression*
8:20 AM	D. Barrow, R. Dawson *Surgical Management of Arteriovenous Malformations of the Ventricular Trigone*
8:40 AM	F. Meyer *Brain pH_i, Acidic Foci, and the Ischemic Penumbra*
9:00 AM	J. Story, K. Story-Held, W. Brown, Jr., J. Harrison *The Ocular Ischemic Syndrome-Neurosurgical Implications of Ophthalmic Artery Color Doppler Blood Flow and Electroretinography*
9:20 AM	J. Lustgarten, R. Solomon, D. Quest, A. Khanjdi, J. Mohr *Carotid Endarterectomy After Non-Invasive Evaluation*
9:40 AM	**Academy Award Presentation** Dr. Howard Eisenberg

Thursday, October 28

9:40 AM — **Academy Award Paper**
Michael Tymianski,
M. Wallace, M. Charlton
Toronto Hospital Research Institute
Discovery and Characterization of a New Treatment for Cerebral Ischemia by Cell-Permanent Ca^{2+} Chelators

10:10 AM — **Coffee Break**

10:40 AM — **Scientific Session II**
Moderator: Dr. Charles Hodge

10:40 AM — W. Butler, N. Zervas, R. Cosgrove
A New Device for Internal Stereotactic Radiosurgery

11:00 AM — A. Olivier, D. Lacerte, I. Germano, A. Cukiert
Frameless Stereotactic Craniotomies in the Surgical Treatment of Epilepsy: Preliminary Experience in 70 Patients

11:20 AM — P. Gleason, P. Black, R. Kikinis, F. Jolesz
Virtual Reality for Localizing Central Nervous System Masses

11:40 AM — C. Tator, D. Anthes, E. Therian
Evidence for Vasospasm from Arteriolar Electron Microscopic Morphometry Following Traumatic Spinal Cord Injury

12:00 PM — S. Papadopoulos, J. Hoff
Results of the University of Michigan Acute Spinal Cord Injury Surgical Protocol

Thursday, October 28

12:20 PM — F. Wirth
Analysis of Three Different Surgical Approaches for Herniated Cervical Discs

12:40 PM — **Special Lecture**
Dr. Alan Hudson
The Canadian Health Care System: Priorities and Their Relationship to Academic Centers

1:00 PM — Adjourn

Friday, October 29

8:00 AM — **Scientific Session III**
Moderator: Dr. Michael Apuzzo

8:00 AM — S. Peerless, J. Hernesniemi, F. Gutman, C. Drake
Early Surgery for Ruptured Posterior Circulation Aneurysms

8:20 AM — J. Morcos, R. Heros
Intracranial Aneurysms: Surgical Complications and Technical Pitfalls: A 12 Year Experience With 611 Cases

8:40 AM — E. Flamm
Intraoperative Endovascular Surgery of Aneurysms

9:00 AM — B. Weir
Intracranial Aneurysms: North and South of the 49th Parellel

Friday, October 29

9:20 AM — C. Loftus, J. Gerdes, M. Muhonen
Effects of Serotonin (5-HT) and 5-HT_1 and 5-HT_2 Antagonists on Blood Flow to Normal Brain and Collateral Dependent Tissue

9:40 AM — L. D. Lunsford, D. Kondziolka
Percutaneous Retrogasserian Glycerol Rhizotomy for Trigeminal Neuralgia: Long Term Assessment

10:00 AM — **Coffee Break**

10:20 AM — **Scientific Session IV**
Moderator: Dr. Martin Weiss

10:20 AM — R. Spetzler, M. Hamilton, J. Herman, S. Beals, E. Jorganic
Transfacial Approach to the Skull Base with Emphasis on Preservation of Olfaction

10:40 AM — D. Bruce, I. Munro
Fibrous Dysplasia of the Optic Foramen and Ethmoid Complex in Children

11:00 AM — J. Hahn
Is Quality Medical Care Affected Negatively by Cost Containment?

11:20 AM — W. Couldwell, D. Hinton, M. Weiss
Signal Transduction and Growth Regulation of Pituitary Adenomas

11:40 AM — L. Calliauw, L. de Ridder
A Comparative Study of Invasion Tests <u>in vitro</u> for Brain Tumor-Derived Cells

Friday, October 29

12:00 PM — **Academy Award Runner Up**
P. Le Roux
T. Reh
University of Washington
Regional Differences in Glial Derived Factors That Promote Dendritic Outgrowth From Mouse Cortical Neurons in vitro

12:20 PM — **Presidential Address**
Dr. George Tindall
Introduced by Dr. Martin Weiss

1:00 PM — Adjourn

Saturday, October 30

8:00 AM — **Scientific Session V**
Moderator: Dr. Suzie Tindall

8:00 AM — K. Lillehei, B. Kleinschmidt-DeMasters, E. Ridgway
Radiation Therapy As An Adjunct to the Treatment of the Pituitary Macroadenoma. Is It Always Necessary?

8:20 AM — H. Brem
Polymers As An Intracranial Implantable Controlled Drug Delivery System

8:40 AM — W. Selman, R. Wasserman, R. Tarr, R. Ratcheson
Two-Dimensional Gated Phase Contrast MRI: Flow Quantitation of Arteriovenous Malformations

Saturday, October 30

9:00 AM — D. Peterson, M.Tullous
Cine-Mode Magnetic Resonance Imaging in the Evaluation and Treatment of the Chiari I Malformation

9:20 AM — D. Wen, W. Hall, O. Fodstad
Transferrin Receptor Expression and Efficacy of a Transferrin Toxin Conjugate Against Human Medulloblastoma in vitro and in vivo

9:40 AM — J. Robertson, C. Hamm
Acoustic Tumor Surgery: Quality Assessment by Cost Analysis

10:00 AM — **Special Lecture**
Dr. Clark Watts
Legal Aspects of Neurosurgical Practice

10:20 AM — **Coffee Break**

10:40 AM — **Symposium**
Stereotaxis: Its Future Role
Moderator: Dr. Michael Apuzzo

M. Apuzzo
Concepts and Trends

P. Kelly
The Evolution of the Computer as a Neurosurgical Tool

P. Heilbrun
Frameless Systems

L. D. Lunsford
Future Applications of Focused Energy Sources for Structural Brain Lesions

Saturday, October 30

P. McL. Black
Molecular Neurosurgery: Dream or Reality

R. Young
Radiosurgery of Pain and Functional Syndromes

K. Burchiel
Modulation Devices for Movement Disorders

R. Maxwell
A Renaissance for Behavioral Modification

Panel Discussion
M. Apuzzo

1:00 PM Adjourn

American Academy of Neurological Surgery
Annual Meeting Educational Goals
October 27-30, 1993

The goals of attending the scientific sessions of the American Academy of Neurological Surgery Meeting in Phoenix, October 27-30, 1993, are as follows:

1. At the end of the meeting, the participants will be able to demonstrate an understanding of the basic principles of the technical proficiency in the treatment of intracranial aneurysm, other neurovascular disorders, pain problems and CNS neoplasia.

2. The participants will be able to demonstrate an understanding of new techniques for the treatment of skull base tumors and epilepsy.

3. The participants will be able to demonstrate an understanding of the means of identification and treatment of congenital lesions of the central nervous system with emphasis on Chiari malformations and hydrocephalus.

4. The participants will be able to demonstrate an understanding of recent regulations dealing with managed care issues and will demonstrate an understanding of the implications of recent legal decisions regarding the practice of neurosurgery.

The Joint Committee on Education of the American Association of Neurological Surgeons and the Congress of the Neurological Surgeons is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

The Joint Committee on Education of the American Association of Neurological Surgeons designates this continuing medical education activity for 13.5 credit hours in category 1 towards the Continuing Education Award in Neurosurgery and the Physician's Recognition Award of the American Medical Association.

Thursday, October 28
8:00 a.m.

Dural Arteriovenous Fistulas: Pathogenesis and Progression

G. Hieshima, R. Higashida, V. Halbach, C. Dowd, C. Wilson, T. Terada

Dural arteriovenous fistulas (DAF's) are acquired A-V shunts, in many cases known to be preceded by thrombosis of a dural venous sinus. We suspected that venous hypertension might constitute another mechanism in the genesis of AVF's, and a rat model provided confirmatory evidence. Many AVF's progress from innocent but annoying symptoms to conditions accompanied by major morbidity. This evolution from a small and simple fistula to complex and life-threatening arteriovenous communications will be described.

Notes

Thursday, October 28
8:20 a.m.

Surgical Management of Arteriovenous Malformations of the Ventricular Trigone

D. Barrow, R. Dawson

Arteriovenous malformation (AVMs) of the ventricular trigone represent a distinct subset of vascular malformation associated with unique challenges for the neurosurgeon. Factors that contribute to the difficulties of these lesions include; (1) invariable location of the AVM within functionally important or eloquent brain tissue; (2) lack of cortical representation of the AVM thus requiring retraction or traversion of important brain tissue; (3) deep and often obscure arterial supply; (4) deep venous drainage; (5) juxtaposition to choroid plexus with which arterial supply and venous drainage are shared, adding to the bulk of the lesion; and (6) tangential surgical approaches to the AVM rather than safer and more standard perpendicular approaches.

We report our experience over the last 3 years with 24 AVMs of the ventricular trigone, all of which underwent complete surgical removal of the post MRI era. In this report we emphasize those adjuncts that are instrumental in the management of these difficult cases, including preoperative embolization to assist in obliterating a portion of the deep arterial supply; use of a variety of operative approaches to these AVMs, chosen on the basis of MRI and angiographic criteria; intraoperative ultrasound and angiography to aid in intraoperative localization and to document complete excision prior to closure. Caveats gleaned from our management complications will also be detailed.

Notes

Thursday, October 28
8:40 a.m.

Brain pH_I, Acidic Foci, and the Ischemic Penumbra
F. Meyer

An *in vivo* panoramic imaging system was used to study cortical pH_I by using a pH sensitive fluorescent indicator in the anesthetized New Zealand rabbit. In the nonpathologic state, overall cortical pH_I measured 7.05 $\pm$ 0.02. A detailed analysis of pH_I across the brain's surface revealed minimal variation ranging from 0.005 to 0.04 pH units with a slight acidosis in parenchyma adjacent to veins. Alternatively, there was marked heterogeneity of CBF with flow being greatest in parenchyma adjacent to cortical veins. With a progressive increase in $P_. CO_2$ to 120 mmHg, brain pH_I remained stable despite a significant extracellular acidosis. This data indicates that cortical pH_I is homogeneous and tightly regulated with the ability to upregulate pH_I homeostatic mechanisms in response to an acidic challenge. During focal ischemia, an ischemic penumbra can be identified which has an overall cortical pH_I of 6.61 $\pm$ 0.02. Within the ischemic penumbra, there is the development of acidic foci which have an initial pH_I of 6.4 $\pm$ 0.10. These acidic foci do not occur in a vascular distribution. Despite improvements in pH_I of the majority of ischemic penumbra, these foci remain acidic and have evidence of neuronal injury on light microscopy. Associated with these acidic foci is elevated NADH fluorescence indicating mitochondrial failure. This supports the hypothesis that these acidic foci may lead to recruitment of ischemic penumbra into infarction. Furthermore, acidic foci have been identified in both global ischemia and hypoxia. This suggests that there is a cortical selective vulnerability in regard to pH_I regulatory mechanisms.

Notes

Thursday, October 28
9:00 a.m.

The Ocular Ischemic Syndrome-Neurosurgical Implications of Ophthalmic Artery Color Doppler Blood Flow and Electroretinography

J. Story, K. Story-Held, W. Brown, Jr., J. Harrison

The ocular ischemic syndrome occurs in about 10-15% of patients with occlusive disease of the carotid artery. The syndrome is characterized by 1) rapid loss of vision (finger counting only in 50% of patients one year following the onset of symptoms); 2) marked intolerance to bright light; and 3) ocular pain. Glaucoma is a common complication and results from rubeosis iridis (neovascularization of the iris), which occurs as the syndrome progresses. Although the syndrome may be associated with transient focal hemispheral symptoms or stroke, it may also be the sole manifestation of carotid occlusive disease, or it may be associated with generalized, vague manifestations of cerebral hypoperfusion.

We present two patients with the ocular ischemic syndrome. The first patient had occlusion of the right common carotid artery and was treated early in the syndrome with a bypass graft from the subclavian artery to the distal common carotid artery. The patient's mild visual loss, extreme light intolerance, and intolerable ocular pain were totally relieved. The second patient had a high grade stenosis of the internal carotid artery and far-advanced symptoms including severe visual loss and glaucoma. Following carotid endarterectomy, the vision improved notably, the neovascularization of the iris regressed. Both patients were strikingly relieved of their non-focal symptoms of cerebral hypoperfusion. These two patients, one treated early in the syndrome and one treated late, also illustrate the advantages of early surgical intervention.

Ophthalmic artery blood flow determination by the color Doppler method was used in both patients. In the first patient, there was an equalization of blood flow in the two eyes with a 150-180% increase in ipsilateral

ophthalmic artery blood flow following revascularization. In the second patient, ophthalmic flow was markedly retrograde preoperatively, indicating a "carotid steal." Postoperative flow was restored to normal. Electroretinography in both patients also showed a striking improvement in the b wave of the electroretinogram. We shall discuss the neurosurgical implications of the color Doppler flow studies and electroretinographic changes before and after surgery. These studies provide objective criteria for neurosurgical intervention when patients present with ocular symptoms alone (or ocular symptoms associated with non-focal neurological symptoms) and compromised blood flow in the carotid system.

Notes

Thursday, October 28
9:20 a.m.

Carotid Endarterectomy after Non-invasive Evaluation by Doppler and Magnetic Resonance Angiography*

J. Lustgarten, R. Solomon, D. Quest, A. Khanjdi, J. Mohr

Recent studies documenting the efficacy of carotid endarterectomy in selected patients provide further impetus for developing noninvasive techniques to evaluate carotid occlusive disease. Eliminating the morbidity due to preoperative angiography would further refine the treatment of this condition. Recent improvements and greater experience with magnetic resonance angiography (MRA) of extracranial vessels have increased the accuracy of this technique. We present our experience using MRA in combination with duplex ultrasonography as the primary mode of preoperative evaluation for carotid endarterectomy (CEA). Fifty-two patients referred for CEA underwent these two studies. In 47 patients (90%) significant stenosis (>70%) was unambiguously identified on both ultrasound and MRA. Forty-one of these patients underwent CEA on the basis of these studies alone, without conventional angiography. In all of these cases significant stenosis was identified at surgery (100%), and CEA was performed without difficulty or complications. In 5 cases (9.6%) the MRA and ultrasound findings did not concur exactly. In 3 of these cases the interpretation of the two studies differed with respect to the severity of stenosis; in the others one of the studies was indeterminate. These patients underwent conventional angiography prior to surgery.

Our experience suggests that the combined use of MRA and ultrasonography affords an accurate noninvasive evaluation of carotid occlusive disease sufficient for surgical planning in most cases.

Notes

Thursday, October 28
10:40 a.m.

A New Device for Internal Stereotactic Radiosurgery

W. Butler, N. Zervas, R. Cosgrove

Two years ago, the theoretical and laboratory investigations to design, build and evaluate an internal radiation source to treat malignant brain tumors were presented. The device was designed to be used in conjunction with stereotaxic biopsy. It is compatible with standard stereotaxic frames and gives the surgeon the option of radiating a lesion at the time of biopsy rather than waiting for a later radiosurgical or radiotherapeutical procedure. The power supply is a 9 volt NiCd battery. Microtransformers raise power to 40kVp. A thermionic emitter produces electrons that are then accelerated to the anode to produce low energy photons (40kVp-2.0mA) at the tip of a cannula. The cannula is 10 cm in length and 3.2 mm in outer diameter. The device can produce a spherical or oval lesion. The photons produced at the tip fall in tissue at 1/r3. The photons at the tip are 6000 Gy and 20 Gy at a diameter of 3 cm. At the skull surface, radiation is less than 4 rad, and personnel receive no background radiation, hence the procedure can be carried out in a standard operating room without need for shielding. A 3 cm lesion can be treated with 2000 rad at the edge in less than an hour. Heat production is negligible. In the past five months, we have completed a ten treatment FDA trial. The pathological diagnoses were: eight metastatic tumors, one lymphoma and one infarction. No patient suffered an adverse neurological event. Post treatment scanning revealed a small (1x0.5 cm) asymptomatic hemorrhage at the operative site in Pt 3, prior to radiation.

Follow up: The first patient recurred 5 months later and had total removal of a mixture of necrosis and recurrent tumor. One patient with lymphoma recurred outside the site of radiation. All the other lesions appear to be the same size or smaller. (Follow-up = 3-7 months).

Conclusion: This device gives stereotaxic surgeons the ability to deliver a dose of photons of 15-20 Gy to an

edge at 3-1/2 cm within 25-60 minutes. Thus it may have significant application in tumors deemed too difficult to remove surgically, as an alternative to open surgery or to radiosurgery. Finally, it may be useful in open surgical procedures to radiate residual lesions that would otherwise require postoperative fractionated radiotherapy.

Notes

Thursday, October 28
11:00 a.m.

Frameless Stereotactic Craniotomies in the Surgical Treatment of Epilepsy: Preliminary Experience in 70 Patients.

A. Olivier, D. Lacerte, I. Germano, A. Cukiert

Frameless stereotaxy is a method which can improve the precision of several procedures used in the surgical treatment of epilepsy.

Since March 1992, we have used the Allegro-viewing Wand system (ISG) in 70 craniotomies for epilepsy. 3-D reconstruction of the brain was achieved with 62, 2mm thick, T-1 weighted images. The Allegro software was used for the presurgical planning to localize and colour code volumes or structures of interest. Most registration procedures were based on natural landmarks and on surface fitting of the head or on skin fiducial markers. The topographic accuracy was in the order of 1-4mm.

A variety of useful applications were developed such as 1. optimal centering of the craniotomy and delineation of principal cranioencephalic landmarks, 2. localization of small epileptogenic lesions, 3. localization of cortical dysplasias, 4. evaluation of the extent of callosotomy, 5. identification of the central area and central sulcus, 6. performance of selective amygdalo-hippocampectomy with colour coding insertion of acute depth electrodes, 8. data-base storage of ECOG and stimulation responses, 9. display of the resection zone.

No adverse reactions were encountered. The disadvantages are the lengthening of some procedures and the difficulty in compensating for movement and distortion of the brain during surgery. The procedure has been found useful and safe in a variety of applications for epilepsy surgery.

Notes

Thursday, October 28
11:20 a.m.

Virtual Reality for Localizing Central Nervous System Masses

P. Gleason, P. Black, R. Kikinis, F. Jolesz

We have developed a technique for merging live video images with three-dimensional computer reconstructions of diagnostic neuroimaging. The process involves three-dimensional reconstructions of MR and CT images which can be manipulated in real time on a computer workstation. A video camera photographs the patient from the surgeon's intra-operative perspective. The 3-D reconstruction is then simultaneously displayed in the same perspective. The images from the video camera and the 3-D computer reconstruction are combined using a video mixer. This permits the two images to be superimposed, similar to a double-exposure in photography. The patient's position and the 3-D rendering are adjusted until the two images are identical in terms of scale, position and rotation using surface landmarks. Once the video and 3-D computer images of the patient's skin have been aligned the computer image of the skin is removed leaving the 3-D image of the underlying cranial or spinal contents superimposed on the video image of the patient's skin. The surgeon then outlines the borders of the tumor along with important cortical sulci on the patient's skin using indelible markers. These markings allow the surgeon to plan an adequate opening with minimal exposure of adjacent structures. Further use of this technique intraoperatively permits definition of tumor margins and localization of subcortical tumors using sulci as registration landmarks.

We have used this procedure in twelve patients with good success; this group includes patients with parasagittal, temporal and spinal meningioma, as well as several parenchymal masses. Good correlation was obtained in these cases between video imaging and reconstruction. This technique is an important step in the development of frameless approaches to accurate cortical and spinal localization.

Notes

Thursday, October 28
11:40 a.m.

Evidence for Vasospasm from Arteriolar Electron Microscopic Morphometry following Traumatic Spinal Cord Injury

C. Tator, D. Anthes, E. Theriault

While several mechanisms of ischemia following spinal cord trauma have been hypothesized (vessel rupture, shearing, compression, intravascular thrombosis), vasospasm has not been convincingly characterized. Nine adult female Wistar rats underwent a 51 g clip compression injury at C8-T1. Three animals were sacrificed at each postinjury time: 15 min, 2 hrs and 24 hrs. Three additional sham control rats were sacrificed 24 hrs postoperatively. Following transcardial aldehyde perfusion, sulcal arterioles within the ventral median fissure were sectioned coronally midway along the fissure at the injury site and prepared for electron microscopy. Medial smooth muscle cells from control arterioles were very long and thin accompanied by flat endothelial cells lining the generally large round lumina. In contrast, at 15 minutes and 2 hours postinjury, there was a substantial decrease in smooth muscle cell length and an increase in width. Examples of extreme vasospasm observed at 24 hours postinjury were characterized by further decreased length and increased width of smooth muscle cells with large endothelial cells squeezed centripetally, forming an acinar pattern about a virtually obliterated lumen. Smooth muscle cells and luminal area were quantitatively analyzed in a blinded manner on an IBAS image analysis system. A decreasing trend was observed for luminal cross-sectional area achieving statistical significance by 24 hours ($p=0.02$). Smooth muscle cell length was dramatically reduced ($p=0.0001$) and width dramatically increased ($p=0.0001$) postinjury. The reductions in luminal cross-sectional area correlate directly with the constrictive changes measured in the smooth muscle cells. The results of this study support the concept of enhanced vascular tone ("vasospasm") following acute spinal cord injury.

Notes

Thursday, October 28
12:00 p.m.

Results of the University of Michigan Acute Spinal Cord Injury Surgical Protocol

S. Papadopoulos, J. Hoff

The surgical management of acute spinal cord injury (ASCI) remains controversial. We have developed an ASCI protocol that employs immediate stabilization and reduction with cranio-spinal traction, in-traction MRI, followed by emergent surgical spinal cord decompression (if persistent cord compression is demonstrated on MRI), and fusion. Routine medical management includes treatment with methylprednisolone. The results of the initial thirty patients treated according to this protocol are presented. The average time from injury to initial presentation was 3.5hr. Average time from admission to alignment with skeletal traction was 2.3hr., to completion of MRI was 4.0hr., and to operative decompression was 14.6hr. Mean follow up is 18 months. Of the patients who initially presented as a Frankel grade A, 7 of 13 remained a grade A (54%), 3 improved to B (23%), 1 to C (8%), and 2 to D (15%). Of the patients who presented as grade B, only one of 10 remained grade B (10%), 4 improved to grade C (40%), and 5 to D (50%). Of the four Frankel grade C patients, one remained a C (25%), and 3 improved to D (75%). Three Frankel D patients remained a D (100%). The persistent compressive lesions documented on MRI were incompletely reduced bone fragments(11), associated herniated disc(7), and persistent malalignment(2). Total length of hospital care, including rehabilitation, decreased from 105 days to 84.8 days for those patients treated by this protocol, compared to a matched group of "non-protocol treated" patients.

Although this represents a preliminary report, we believe it emphasizes the value of early MRI in the treatment of ASCI and may suggest improved neurologic recovery with early operative intervention.

Notes

Thursday, October 28
12:20 p.m.

Analysis of Three Different Surgical Approaches for Herniated Cervical Discs

F. Wirth

74 patients with acute unilateral herniated cervical discs, unresponsive to conservative therapy, were prospectively randomized to three surgical treatment groups. One-third were operated upon via a partial laminectomy/foraminotomy approach, one-third underwent anterior cervical discectomy and one-third underwent anterior cervical discectomy and fusion. Patients with cervical spondylosis, central disc, and/or myelopathy were excluded. The results of treatment were analyzed with respect to length of stay, cost of treatment, and complications encountered as well as for pain relief. The average follow-up was 2 years. The findings will be discussed.

Notes

Friday, October 29, 1993
8:00 a.m.

Early Surgery for Ruptured Posterior Circulation Aneurysms

S. Peerless, J. Hernesniemi, F. Gutman, C. Drake

The majority of the 1767 patients operated upon for treatment of their vertebrobasilar aneurysms (VBAA) had their surgery 14 days or more following their last subarachnoid hemorrhage (SAH). Since 1970, 206 patients with VBAA have been operated on within 7 days following their last SAH (day of SAH counted as 0). In Grade 1 and 2 patients, a good or excellent outcome was obtained in 80% irrespective of timing of surgery. Curiously, the outcome was worse for patients operated upon Day 2. All except 1 of the Grade 5 patients died and 70% of the Grade 4 patients were ultimately significantly disabled or dead. Grade 3 patients operated on early resulted in one-third of the cases with poor outcome. The operative mortality was the same whether operated on in the first week or delayed. The frequency of intraoperative rupture of the aneurysm was not higher than in delayed surgery. Thirteen percent developed a delayed ischemic neurologic deficit as a consequence of reactive arterial narrowing (vasospasm). We recommend to operate early in those patients who are good-grade (Botterell Grade 1-2, Hunt-Hess 1-3), whose aneurysm does not present a particular technical difficulty because of size, configuration or location, and occasionally in those patients whose lives appear to be in jeopardy because of recurrent hemorrhage.

Notes

Friday, October 29, 1993
8:20 a.m.

Intracranial aneurysms: Surgical complications and technical pitfalls--A 12 year experience with 611 cases

J. Morcos, R. Heros

The purpose of this study is to evaluate the role of surgical technique in the overall management outcome, through a retrospective analysis of a 12 year series of 611 operated aneurysms. In particular, we address the issue of surgical pitfalls as pertains to specific aneurysmal locations.

Causes of surgical morbidity were classified as intraoperative (aneurysmal rupture, arterial occlusion, perforator injury, distal embolization, direct neural injury) and postoperative (intracranial hemorrhage, delayed ischemic deficits, incomplete clipping, systemic complications).

The series was broken down into cavernous (4), paraclinoid (83), supraclinoid (143), anterior cerebral complex (132), middle cerebral (136), basilar tip (63), posterior cerebral (13), superior cerebellar (8), basilar trunk (7) and vertebral-PICA (22) aneurysms. Complications related to surgical technique are discussed and recommendations made regarding their avoidance.

In a disease where surgical intervention has the potential of magnifying the morbidity of the initial insult, scrupulous attention to technical detail remains a major determinant of overall management outcome.

Notes

Friday, October 29, 1993
8:40 a.m.

Intraoperative Endovascular Surgery of Aneurysms
E. Flamm

Endovascular approaches to cerebral aneurysms are now considered to be neuroradiologic procedures performed away from the operating room. This paper will review the application of endovascular techniques utilized during the course of 1100 neurosurgical procedures for intracranial aneurysms.

The standard neurosurgical approach to cerebral aneurysms is from the abluminal surface. This is enhanced by the application of procedures that can be considered intravascular intraoperative neurosurgery such as suction decompression, endaneurysmectomy and aneurysmorrhaphy. A review of the last 1100 aneurysms operated upon for direct clipping disclosed that these methods were utilized in 47 cases. Carotid artery aneurysms accounted for 28, 10 were located on the middle cerebral, 5 occurred in the vertebrobasilar distribution and 4 arose from the anterior cerebral artery. The indications for these methods include large size, intraluminal thrombus, broad neck, plaque at the neck, the potential compromise of branches at base of aneurysm or a combination of these problems.

The methods used included suction decompression, direct removal of plaque and thrombus utilizing suction, dissection and ultrasonic aspiration. All cases in which the aneurysm was opened prior to definitive clipping required the application of temporary clips. The occlusion time ranged from 1.5 to 30 minutes. No special pharmacologic cerebral protective regimen was employed although moderate hypothermia is utilized. In those cases in which greater occlusion times were anticipated, cardiopulmonary bypass with profound hypothermia was employed.

A favorable outcome was achieved in 80% of these difficult cases. These methods should be considered and anticipated before surgery for unusual aneurysms. With

attention to details such as the need for opening the aneurysm, many cases now being considered for embolization may be suitable for definitive surgical obliteration.

Notes

Friday, October 29, 1993
9:00 a.m.

Intracranial Aneurysms: North and South of the 49th Parallel

B. Weir

The existence of a universal Medicare insurance plan and a rigid pass/fail system of qualification to practice results in the relatively small number of Canadian neurosurgical centers and neurosurgeons each seeing the full spectrum of aneurysmal subarachnoid hemorrhages relatively quickly. The high ratio of neurosurgeons to population in the United States, the variability of insurance coverage and the competition for cases between university and other centers results in a highly variable and skewed experience at some university centers. A series of anecdotal cases will demonstrate the author's recent experience in Canada and the United States.

Notes

Friday, October 29, 1993
9:20 a.m.

Effects of Serotonin (5-HT) and 5HT and $5HT_2$ Antagonists on Blood Flow to Normal Brain and Collateral Dependent Tissue.

C. Loftus, J. Gerdes, M. Muhonen

This study examined the effects of 5-HT on rCBF to normal and collateral-dependent cerebrum, and of pretreatment with methiothepin (a $5\text{-}HT_1$-like antagonist) or ketanserin (a $5\text{-}HT_2$ antagonist). In dogs an MCA branch was cannulated following left frontal craniotomy. Collateral-dependent zone (CDZ) was identified by the "shadow flow" technique, rCBF was measured using radioactive microspheres and MCA pressure was measured using a micropipet. 5-HT was then infused intravenously at 10 μg/kg/min for 30 minutes, following which rCBF to CDZ and normal cerebrum, mean arterial pressure, and pial artery pressure were measured. The dose of 5-HT was then increased to 40 μg/kg/min for 30 minutes and the rCBF and measurements repeated.

Normal brain rCBF following MCA occlusion was 110.7 ±7.6 cc/100 gm/min(mean ± SEM) and 72.5 ±.7 in the CDZ ($P<.05$). Following 10 mg/kg/min 5-HT, flow to normal cerebrum remained constant (114 ±7), while, rCBF in the CDZ declined 36% to 46.3 ±3.8 ($P<.05$). With 40 μg/kg/min of 5-HT, normal brain rCBF again did not decline significantly (99.2 ±2.8 $P<.05$), but in the CDZ, rCBF dropped to 23.8 ±2.0. ($P<.05$)

The serotonin receptor antagonists methiothepin ($5\ HT_1$) and ketanserin ($5\ HT_2$) were then studied. Group 1 dogs received methiothepin (1 mg/kg); group 2 received ketanserin (1 mg/kg), after which 5-HT was infused at 10 μg/kg/min for 30 minutes. The dose of 5-HT was then increased to 40 μg/kg/min for 30 minutes. rCBF and arterial pressures were measured throughout.

Both of the antagonists alone exhibited vasoactive properties: in group 1 normal brain rCBF declined 26%

(187.2 ±32.7 to 138.8 ±10.5) and CDZ flow declined 28% (145.2 ±15.2 to 89.2 ±8.5). In group 2 normal brain flow declined 21% (188.4 ±30.7 to 149.8 ±17.3) and CDZ declined 14% (113.2 ±18 to 96.9 ±13.1). When the two doses of 5-HT were given, there was no significant change from levels following the antagonists, with an actual increase in rCBF to the CDZ of group 1 following 40 µg/kg/min compared to the level following methiothepin. In group 1 CDZ flow was 89.5 ±8.5 following methiothepin compared to 98.7 ±48.5 following 40 µg/kg/min of 5-HT, while in normal brain it was 138.77 ±10.5 versus 130.36 ±12.2. In group 2 flow to the CDZ following ketanserin was 96.97 ±13.1 versus 78.4±15.4 following 40 µg/kg/min of 5-HT, and in normal cerebrum it was 149.8 ±17.3 versus 130.6 ±16.2. (P=NS)

This study shows that 5-HT has a profound vasoconstrictive effect on cerebral collateral vessels, and that this effect can be attenuated by antagonists acting at both 5-HT_1, and 5-HT_2 receptors.

Notes

Friday, October 29, 1993
9:40 a.m.

Percutaneous Retrogasserian Glycerol Rhizotomy for Trigeminal Neuralgia Long Term Assessment

L. D. Lunsford, C. Duma, D. Kondziolka

In order to assess the long term success of percutaneous retrogasserian glycerol rhizotomy (PRGR) for management of medically refractory trigeminal neuralgia, we retrospectively evaluated our 11 year experience. During this interval 517 patients (75% were older than 60 years, eldest was 103) underwent 707 rhizotomies. All patients were intolerant or refractory to medication and 39% had failed prior surgery. Follow-up extended from 1 to 11 years (173 had greater than 5 years follow-up).

Overall 72.2% of patients had satisfactory results (56.5% pain free off medication and 15.7% pain free on medication). Post rhizotomy sensation data was recorded on 366 patients; 179 (48.9%) had no sensory loss. Deafferentation sequelae were unusual, and often related to the development of postoperative herpes simplex perioralis. No patients developed anesthesia dolorosa. Depending on the length of follow-up and number of rhizotomies performed, between 87% (24-36 month follow-up) and 48% (>60 month follow-up) remained asymptomatic without medication.

PRGR is an anatomical operation defined by intraoperative contrast cisternography; a normal cisternogram correlated with both short and long term success. Extended pain control (with usually absent or mild facial deafferentation) was possible in 72% of patients, including those refractory to prior surgical procedures. Successful pain management most often accompanied by preservation of trigeminal sensation characterized PRGR. Permanent marking of the cistern with a radiodense agent facilitates repeated procedures if necessary. PRGR is an effective and low risk management strategy that should be offered to trigeminal neuralgia patients unresponsive to or unsuitable for microvascular decompression.

Notes

Friday, October 29, 1993
10:20 a.m.

Transfacial Approach to the Skull Base with Emphasis on Preservation of Olfaction

R. Spetzler, M. Hamilton, J. Herman, S. Beals, E. Joganic

Resection of extensive deep-seated neoplasms involving the anterior skull base and clivus is surgically challenging. The anatomic site of these lesions can be used as a guide to classify a logical approach for transfacial exposure. We have defined six levels at which facial osteotomies can be performed to provide excellent exposure for tumor resection. These include exposures by a transfrontal (Level I), transnasal (Level II), transfrontal nasal-orbital (Level III), transnasomaxillary (Level IV), transmaxillary (Level V), or transpalatal (Level VI) route. This classification system can be used to guide surgical planning and if required, can be used alone or combined with other approaches to allow for simultaneous, combined intracranial and extracranial tumor resection. These approaches provide direct access to lesions of the anterior skull base and clivus, thereby minimizing brain retraction. Except for Level IV, all approaches can be accomplished without facial incision. A technique for preserving the cribriform plate through circumferential osteotomies has been developed and used successfully in four patients with preservation of olfaction.

We present with 14 patients who underwent transfacial exposure for resection of extensive anterior skull base or clival neoplasms. There was no significant surgical morbidity and no surgical mortality. Ten of the 14 patients survived long term. Four patients died due to tumor progression: 2 patients with chordoma 19 and 15 months postsurgery; 1 patient with malignant fibrous histiocytomas 10 months postsurgery; and 1 patient with melanoma 12 months after surgery. We conclude that the transfacial approaches are important in treating deep-seated lesions of the anterior skull base and clivus.

Notes

Friday, October 29, 1993
10:40 a.m.

Fibrous Dysplasia of the Optic Foramen and Ethmoid Complex in Children

D. Bruce, I. Munro

In the last five years, we have encountered 14 cases of fibrous dysplasia involving one or both optic foramen and producing optic nerve compression. These lesions have all involved the anterior skull base and/or the ethmoid sinuses and maxillae. These lesions represent a special challenge since the ideal therapy involves resection of the involved bone, decompression of the optic nerves and reconstruction of the cranium and facial skeleton. The use of the transdural route makes extensive resection of the ethmoid mass extremely hazardous because of the risk of a CSF leak and often a two stage operation is performed.

Using craniofacial techniques, it is possible to decompress the optic nerve, one or both, starting in the orbit where the nerve is easily identified and decompressing the nerve circumferentially back to the intracranial dura; thus, leaving the dural intact. This permits resection of the skull, ethmoid and maxillary tumor in one setting without concern for CSF leakage and with olfaction spared. Using split cranial bone, the skull and facial skeleton can be rebuilt at the time of the initial surgery.

In these 14 patients, improvement in optic nerve function was obtained in 30%; in 60%, the vision stabilized. In one patient with 20/200 vision preoperatively, the acuity stabilized for three years then gradually dropped to light perception only. There were no postoperative infections and no early CSF leakage. In none of the patients has it been necessary to repeat the optic nerve decompression, although several of the patients have had further cosmetic facial surgery.

This technique will be described in detail with five year follow-up of the patients.

Notes

Friday, October 29, 1993
11:00 a.m.

Is Quality Care Affected Negatively By Cost Containment?

J. Hahn

The healthcare industry is going through a transition at the present time. Initiatives are coming from many directions including the government, payors, providers and consumers. These have taken many shapes and forms and no one at this time is quite sure what the healthcare industry will look like in the year 2000.

At the time this abstract is being written, no information has been divulged by the healthcare task force under Hillary Rodham Clinton and/or President Clinton. Hopefully, this issue will be resolved in the not-too-distant future.

As an example of an initiative that had a direct impact on our institution, the business leaders of Cleveland developed a program entitled "Cleveland Quality Health Choice." This was an attempt by the CEOs and other business leaders to determine which ones provided "quality care", they would then try and direct their employees to these institutions. Their concern was not only the rising cost of healthcare but also the fact that their consumers (employees) were not informed shoppers. They brought together the business community as well as the hospital community to evaluate institutions in three categories. The first was patient satisfaction, the second was outcomes in each of the intensive care units within the city, and the third was to evaluate the outcomes in several surgical DRGs that included coronary artery bypass surgery, lung resection, lower bowel resection, spine surgery, repair of fracture and hip replacement, prostatectomy, and hysterectomy. The concerns raised by this study will be discussed in more detail at the meeting.

As a result of initiatives like this, there is always a concern raised about the quality of health care as it relates to cost. In virtually every other industry, it has been shown that there is an inverse relationship of

quality to cost. As the quality of the product goes up, the costs related go down.

Quality is defined as conformance to requirements. It follows a simple formula of COQ (cost of quality) = POC (price of conformance + PONC (price of nonconformance). The question that needs to be answered to determine whether quality care has been delivered is who chooses what outcomes require conformance. Is it the physician, is it the patient, or is it the payor ? As these questions continue to be asked, institutions will be driven towards eliminating excess cost and trying to become as efficient and productive as possible.

Hughes, in an article entitled "Reducing Healthcare Costs: A Case for Quality," believes that the cost of "waste, rework, complexity and variations, (PONC)" approach 40-50% of the healthcare bill. This would lead one to believe that there is a great amount of revenue to be captured by eliminating or reducing this factor.

The Cleveland Clinic Foundation has undertaken several initiatives to reduce the price of nonconformance. In those areas pertaining to physicians a detailed analysis is generated by physician by DRG by patient for each code or procedure that is done. This information is provided on a monthly basis to the Chairmen of the Departments to allow him or her to assist in managing the department. The institution has been able to reduce the cost in virtually every DRG as far as eliminating excesses and inefficiencies. Two examples will be provided: DRG005 (carotid endarterectomy) and DRG106 and DRG107 (coronary bypass surgery with and without catheterization). By providing this information to the Chairmen, significant advances have been made in reducing the length of stay as well as reducing pharmacy costs, anesthesia costs, recovery room costs and intensive care costs. These parameters will be discussed in greater detail.

In summary cost containment does not signify a reduction in quality and in fact it is the reverse.

Notes

Friday, October 29, 1993
11:20 a.m.

Signal Transduction and Growth Regulation of Pituitary Adenomas

W. Couldwell, D. Hinton, M. Weiss

Previous work has demonstrated an important role of the Protein Kinase C (PKC) signal transduction system in regulating glioma growth; malignant gliomas express very high PKC activity which correlates strongly with their proliferation rates in vitro. These observations have led to clinical trials utilizing PKC inhibitors as adjuncts in the therapy of patients harboring malignant gliomas. To explore the role of the PKC system in growth regulation of pituitary adenomas, primary tumor cultures were plated from fresh surgical tumor specimens. The following day, the PKC inhibitors Staurosporine and Tamoxifen were added to the cultures; measurements of cell proliferation were performed by (^{3}H)-thymidine uptake and the MTT assay. After a 48 hour period, cells were harvested for the proliferation assays. Both (^{3}H)-thymidine uptake and absorbance on the MTT assay decreased in a dose-related manner in both the staurosporine and tamoxifen treated cultures (IC_{50} of 10 nM and 30 μM respectively). Direct measurement of PKC activity using an in vitro assay revealed very high activity (range of 1465-5708 pmol/min/mg protein; within the range recorded for malignant glioma specimens) in 12 frozen specimens of pituitary adenomas (9 nonfunctional adenomas, 3 prolactinomas and 1 corticotroph-secreting adenoma). These preliminary data indicate that pituitary adenoma cells display high PKC activity and are sensitive to growth inhibition of PKC inhibitors. These data suggest a role for the PKC system in regulating pituitary tumor growth, which may have implications for future therapy of these tumors.

Notes

Friday, October 29, 1993
11:40 a.m.

A Comparative Study of Invasive Tests In Vitro for Brain Tumour - Derived Cells

L. Calliauw, L. de Ridder

Proliferation and invasion in the surrounding brain tissue are characteristics of malignant brain tumours. At a meeting of the American Academy (Amalia Island) we proposed a study on the invasiveness, using organ cultures. These cultures served as invasive substrate when confronted with cells derived from brain tumour specimens. At another meeting of the Academy (Shalishan Lodge) Ed. Laws proposed a test in which artificial matrices composed of collagen type I were used as a model for evaluation of the migrating capacity of brain tumour-derived cells.

In a recent study we evaluated ten freshly resected brain tumours using the two models. The tumour-derived cells were brought in contact with as well the organ fragments as with the artificial collagen substrate.

From the results it is evident that an organ culture confrontation can distinguish between cells derived from malignant tumours and non-malignant tumours. In the matrix cultures, measuring the depth of infiltration in collagen gel, no clear cut difference was possible. From these data, the conclusion is that the collagen matrix gives information about the cell motility of the tumour-derived cells but the organ culture can distinguish between invasive, this means destructive for the host tissue, and noninvasive cells.

Both systems are evaluating different characteristics of the tumour derived cells and can be considered as complementary.

Notes

Saturday, October 30, 1993
8:00 a.m.

Radiation Therapy as an Adjunct to the Treatment of the Pituitary Macroadenoma. Is It Always Necessary?

K. Lillehei, B. Kleinschmidt-DeMasters, E. Ridgway

Radiation as an adjunct to surgery remains the mainstay of treatment for the nonsecretory pituitary macroadenoma. With the advent of MRI and improved surgery, the necessity for routine irradiation is being questioned. At the Univ of Colorado, we have initiated a protocol whereby radiation is not routinely recommended for patients who satisfy the following criteria: 1) A gross total surgical resection was felt to have been obtained 2) A 3 month post-op MRI scan reveals no obvious residual tumor and 3) The patients are reliable and can be followed with serial MRI scans. Retrospectively we analyzed our experience from 1985-1993 to ascertain whether this is a feasible approach. Fifty-two patients with nonsecretory pituitary macroadenomas fell into this category. Thirty received radiation and 22 no radiation. Immunocytochemical analysis revealed: 26 (+) for gonadotrophs (FSH, LH, and/or alpha subunit), 2 (+) TSH, 2 (+) GH, 2 (+) PRL and 1 (+) ACH. Nineteen did not stain for any of the 7 markers being true Null cell Adenomas. In analyzing our rate of recurrence; in the patients treated with radiation 2/30 recurred (7%) with a mean F/U of 6.1 years. Both presented with visual deterioration and required repeat surgery. In the 22 patients treated with no radiation we have had no clinical and two radiographic recurrences (9%) with a mean F/U of 2.7 years. Both were irradiated, with one requiring additional surgery. Although follow-up at this time is relatively short, our experience to date suggests that in the patients who meet the above criteria, withholding radiation may be feasible.

Notes

Saturday, October 30, 1993
8:20 a.m.

Polymers as an Intracranial Implantable Controlled Drug Delivery System

H. Brem

The blood-brain barrier limits the usefulness of many drugs for applications in the central nervous system. One promising method for bypassing the blood-brain barrier is clinically implantable biocompatible polymers. Several polymer-drug devices have been developed for intracranial implantation to release drugs to the brain over extended periods of time. The polymers deliver higher concentrations of drug to the brain than can be achieved with systemic drug administration while minimizing systemic exposure to the drug. This technology has been used to treat patients with malignant brain tumors. A phase III clinical drug trial assessing the effectiveness of BCNU loaded polymers in patients with recurrent malignant gliomas has recently been completed. Novel chemotherapeutic drugs, immunotoxins, and angiogenesis inhibitors have been delivered against gliomas in the laboratory. Dexamethasone delivered from an implantable polymer has also been investigated as a method for treating cerebral edema. We have demonstrated that dexamethasone delivered by the intracranial polymer was as effective as systemic dexamethasone at reducing cerebral edema in a rat model. Furthermore, the polymer produced significantly lower blood drug levels than occurred with systemically administered drug, suggesting that thePre should be fewer side effects relative to systemically administered drug. Thus, biocompatible polymers are a novel way to administer drugs to the central nervous system.

Notes

Saturday, October 30, 1993
8:40 a.m.

Two-Dimensional Gated Phase Contrast MRI: Flow Quantitation of Arteriovenous Malformations

W. Selman, R. Wasserman, R. Tarr, R. Ratcheson

INTRODUCTION: Flow quantitation in AVMs is important for determining the pathophysiology of the cerebral circulation induced by these lesions and their treatment. Phase contrast MR imaging provides a non-invasive method for quantitative assessment of intracranial blood flow. Two-dimensional gated phase contrast magnetic resonance (2DGPC MR) imaging has been used to examine flow in the ICA and BA, but quantitation of flow beyond these vessels has not been reported in patients with AVMs . The purpose of this investigation was to determine if 2DGPC MR imaging could provide information regarding the hemodynamics of AVMs.

MATERIALS AND METHODS: The feeding vessels and the corresponding contralateral vessels of seven patients with intracerebral AVMs were examined and compared to flow in five normal volunteers. Pre and post treatment studies were performed.

RESULTS: The average mean flow in the MCA for the volunteer group was 1.6 ± 0.23 ml/sec. while that for patients with MCA AVMs was 2.6 ml/sec in the contralateral vessel, and 13.2 ml/sec in the feeding vessels. Blood flow in feeding vessels exceeded that in the corresponding contralateral vessel by an average of 4.4 times. After one to two staged embolizations this ratio was reduced to 2.6. Mean flow reduction in the measured feeding vessels after embolization was 47.7% and ranged from 8.5% to 73.2%.

CONCLUSION: 2DGPC MR is capable of providing non-invasive *quantitative* flow determinations of the intracranial vessels in patients with AVMs. This information may provide insights into the disorders in cerebrovascular physiology that occur with these lesions, and ultimately provide guidelines for the safety of flow reduction in feeding vessels.

Notes

Saturday, October 30, 1993
9:00 a.m.

Cine-Mode Magnetic Resonance Imaging in the Evaluation and Treatment of the Chiari I Malformation

D. Peterson, M. Tullous

Cine-mode magnetic resonance imaging provides information regarding pulsatile flow characteristics of cerebrospinal fluid. The normal patterns of cerebrospinal fluid flow in the ventricles, cisterns, and subarachnoid spaces are dependent on the cardiac cycle and have been well established. The normal pulsatile flow patterns, determined by cine-mode magnetic resonance imaging, (Cine-MR), are reviewed. A patient with Chiari I malformation and associated symptomatic holocord hydromyelia who underwent pre and postoperative cine-mode magnetic resonance imaging is reported. Cine-MR demonstrated total obstruction of pulsatile flow of cerebrospinal fluid dorsally at the level of the foramen magnum and upper cervical subarachnoid space. Images obtained following surgical management of the malformation demonstrate resolution of the obstructive process with reduction in the size of the hydromyelia. The value of cine-mode magnetic resonance imaging in the evaluation of pathologic alterations of cerebrospinal fluid flow is discussed.

Notes

Saturday, October 30, 1993
9:20 a.m

Transferrin Receptor Expression and Efficacy of a Transferrin Toxin Conjugate Against Human Medulloblastoma In Vitro and In Vivo

D. Wen, W. Hall, O. Fodstad

A variety of toxin conjugates (immunotoxins) have been developed for the treatment of neoplasms. The central nervous system is ideally suited for the use of such conjugates given the compartmentalized nature of the subarachnoid space. Medulloblastoma with its propensity for CSF spread and the relative contraindication to radiotherapy in young children makes it especially suitable for testing such new therapeutic modalities. While immunotoxins show marked activity against a variety of tumors *in vitro*, efficacy against human tumors *in vivo* has been less well demonstrated.

A nude rat model of leptomeningeal carcinomatosis with a human medulloblastoma derived cell line (Daoy) has been developed. Transferrin-*Pseudomonas exotoxin* A (Tfn-PE) shows very high *in vitro* activity against Daoy (IC_{50} 3.4 x 10^{-11} M) and was tested in this model. Animals given 1μg of Tfn-PE intrathecally showed a significant prolongation of time to paraplegia, $p<0.05$, Mantel Haenszel test (56 +/-27 days, n = 9) when administered 7 days after tumor innoculation compared to controls (38+/-16 days, n = 18). Animals treated at 14 days did not show a significant effect (67+/-52 days, n=15).

This marginal therapeutic effect may be related to a reduced expression of the transferrin receptor (TfR) on tumor cells *in vivo*. Immunocytochemistry, immunobead binding, 125-Iodine direct binding studies and cytotoxicity studies clearly show a high degree of TfR expression *in vitro* which is reduced in mouse flank and rat intrathecal Daoy xenografts. Northern blot analysis further confirmed this down regulation of TfR expression *in vivo*. This altered expression of TfR *in vivo* may have profound implications for the use of immunotoxins clinically.

Notes

Saturday, October 30, 1993
9:40 a.m.

Acoustic Tumor Surgery: Quality Assessment by Cost Analysis

J. Robertson, C. Hamm

Health Care reform is currently focusing on access and cost with very little attention to the quality of care in controlling the expense of our ever growing health care delivery system. It is assumed that cost will be inversely proportional to the quality of care provided for a specific surgical procedure. For neurosurgical procedures that are relatively infrequent and high cost such as acoustic tumor surgery, no statistics are available to support this contention.

Using the technique of multiple logistic regression analysis, factors of significance in the cost of acoustic tumor surgery were evaluated in the authors' acoustic tumor surgical experience (1980-1993). A series of 282 acoustic tumors were analyzed in detail to identify factors that influenced the overall hospital costs of this group of patients. Statistically significant fixed factors (tumor size, patient age, preexisting hypertension) and variable factors (surgeons' experience, patient selection, hospital charges for supplies-medication-etc.) were revealed.

The knowledge gained from analysis of this series of patients would strongly support the importance of focusing our attention on the quality of care as it relates to the cost of selective neurosurgical procedures. It is felt that publication of this data would be useful to neurosurgeons as well as hospitals in their effort to improve the quality of health care delivery while controlling cost safely.

GUEST	GUEST OF
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Department of Neurosurgery
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Regional Differences in Glial Derived Factors That Promote Dendritic Outgrowth From Mouse Cortical Neurons in vitro

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The Neurosurgeon Award Winners

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, Bermuda.....November 6-9, 1974
The Wigwam (Litchfield Park), Phoenix,
Arizona.....November 5-8, 1975
Mills Hyatt House, Charleston,
South Carolina.....November 10-13, 1976
Mauna Kea Beach Hotel, Kamuela, Hawaii.....November 2-5, 1977
Hotel Bayerischer Hof, Munich, Germany.....October 22-25, 1978
Hyatt Regency, Memphis, Tennessee.....November 7-10, 1979
Waldorf Astoria, New York City.....October 1-4, 1980
Sheraton Plaza, Palm Springs, California.....November 1-4, 1981
Ritz-Carlton Hotel, Boston, Massachusetts.....October 10-13, 1982
The Lodge at Pebble Beach, California.....October 23-26, 1983
The Homestead, Hot Springs, Virginia.....October 17-20, 1984
The Lincoln Hotel Post Oak, Houston,
Texas.....October 27-30, 1985
The Cloister, Sea Island, Georgia.....November 5-8, 1986
Hyatt Regency, San Antonio, Texas.....October 7-10, 1987
Omni Netherland Plaza, Cincinnati, Ohio.....September 13-17, 1988
Loews Ventana Canyon, Tucson,
Arizona.....September 27-October 1, 1989
Amelia Island Plantation, Amelia Island,
Florida.....October 2-7, 1990
Salishan Lodge, Gleneden Beach, Oregon.....September 22-26, 1991
Ritz-Carlton Hotel, Naples, Florida.....October 21-25, 1992
The Wigwam, Phoenix, Arizona,.....October 27-30, 1993

Past Presidents

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

Past Vice-Presidents

Francis Murphey.....1941
William S. Keith 1942
John Raaf.....1943
Rupert B. Raney 1944
Arthur R. Elvidge.....1946
John Raaf.....1947
Arthur R. Elvidge.....1948
F. Keith Bradford.....1949
David L. Reeves.....1950
Henry G. Schwartz 1951
J. Lawrence Pool.....1952
Rupert B. Raney 1953
David L. Reeves.....1954
Stuart N. Rowe.....1955
Jess D. Herrmann.....1956
George S. Baker.....1957
Samuel R. Snodgrass.....1958
C. Hunter Shelden.....1959
Edmund Morrissey.....1960
Donald F. Coburn 1961-62
Eben Alexander, Jr.....1963
George L. Maltby.....1964
Robert Pudenz 1965
Francis A. Echlin.....1966
Benjamin Whitcomb.....1967
Homer S. Swanson.....1968
Augustus McCravey ...1969-70
Edward W. Davis.....1971
John R. Green 1972
George J. Hayes.....1973
Richard L. DeSaussure 1974
Ernest W. Mack.....1975
Frank E. Nulsen.....1976
Robert S. Knighton.....1977
Robert G. Fisher.....1978
H.T. Ballantine, Jr.....1979
George Ehni.....1980
Courtland H. Davis, Jr. ...1981
John F. Mullan.....1982
Hugo Rizzoli.....1983
James W. Correll 1984
E. Bruce Hendrick.....1985
Griffith R. Harsh III.....1986
Ellis B. Keener 1987
Robert Grossman 1988
Jim Story 1989
John Jane.....1990
Stewart Dunsker.....1991
Burton Onofrio 1992
Martin Weiss.....1993

Past Secretary-Treasurer

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

Past Secretary

Byron C. Pevehouse 1973
Russel H. Patterson, Jr. 1974-76
Phanor L. Perot, Jr.....1977-80
John T. Garner.....1981-83
James T. Robertson....1884-86
Nicholas T. Zervas 1987-89
William A. Buchheit...1990-92

Past Treasurer

Russel H. Patterson, Jr. 1973
Phanor L. Perot, Jr.....1974-76
John T. Garner.....1977-80
James T. Robertson.....1981-83
Nicholas T. Zervas 1984-86
William A. Buchheit...1987-89
Julian T. Hoff.....1990-92

HONORARY MEMBERS

Elected

GUY LAZORTHES, (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

KEIJI SANO (Yaeko) — 1975
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Teikyo Univ. Hospital
2-11-1 Kaga, Itabashi-ku
Itabasji-ku
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SENIOR MEMBERS

Elected

EBEN ALEXANDER JR. (Betty) 1950
Wake Forest School of Medicine
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GALE CLARK 1970
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W. KEMP CLARK (Fern) 1970
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WILLIAM COLLINS, JR. (Gwendolyn) 1963
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RICHARD DESAUSSURE JR. (Phyllis) 1962
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DONALD DOHN (Carolyn) 1968
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CHARLES DRAKE (Ruth) 1958
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ROBERT FISHER (Constance) 1955
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ELDON FOLTZ (Catherine) 1960
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LYLE FRENCH (Gene F.) 1954
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JAMES GALBRAITH (Marguerite {Peggy}) 1947
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JOHN GARNER (Candace) 1971
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HENRY GARRETSON (Marianna) 1973
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EDGAR HOUSEPIAN (Marion) 1976
The Neurological Institute
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ROBERT KING (Molly) 1958
State Univ. of NY Health Science Ctr.
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ROBERT KNIGHTON (Louise) 1966
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THEODORE KURZE 1967
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THOMAS LANGFITT Carolyn) 1971
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RAEBURN LLEWELLYN (Carmen Rolon) 1963
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WILLIAM LOUGHEED 1962
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JOHN LOWREY (Catherine {Katty}) 1965
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ERNEST W. MACK (Bobbie) 1956
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ROBERT L. MCLAURIN (Sarah) 1955
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WILLIAM MEACHAM (Alice) 1952
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FRANCIS MURPHEY (Margery) 1938
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BLAINE NASHOLD, JR. (Irene) 1967
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Department of Surgery
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Durham, NC 27710

GUY ODOM (Mataline) 1946
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Durham, NC 27707

ROBERT G. OJEMANN (Jean) 1968
Neurosurgery Service
Massachusetts General Hospital
Fruit Street
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BURTON ONOFRIO (Judith) 1975
Mayo Clinic
Department of Neurosurgery
Rochester, MN 55902

RUSSEL H. PATTERSON, JR. (Julie) 1971
New York Hospital
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BYRON CONE PEVEHOUSE (Lucy) 1964
135 Mountain Spring Avenue
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CA: (415) 661-3575 (home)

J. LAWRENCE POOL 1940
41 Cherry Hill Road
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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) 1938
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.
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THEODORE RASMUSSEN (Catherine) 1947
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HUGO V. RIZZOLI (Helen) 1973
2150 Pennsylvania Avenue, N.W.
Washington, D.C. 20037

HENRY G. SCHWARTZ (Edith) 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

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

JIM STORY (Joanne) 1972
Univ. of TX Health Sci. Ctr., 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

JOHN TYTUS (Virginia) 1967
1100 9th Ave.
Seattle, WA 98101

A. EARL WALKER (Agnes M.) 1938
1445 Wagontrain Drive, S.E.
Albuquerque, FNM 87123

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

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
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
JERALD BRODKEY (Arielle) 24755 Chagrin Blvd., Suite 205 Beachwood, OH 44122	1977
WILLIS BROWN, JR. (Ann) Division of Neurosurger Univ. of Texas Health Science Ctr. 7703 Floyd Curl Drive San Antonio, TX 78284-7843	1984
DEREK BRUCE (Frances) 1935 Motor Street Dallas, TX 75235	1984

WILLIAM BUCHHEIT (Christa) 1980
Department of Neurosurgery
Temple University Hospital
3401 North Broad Street
Philadelphia, PA 19140

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 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
Fruit Street
Boston, MA 02114

EDWARD CONNOLLY (Elise) 1972
Ochsner Clinic
Department of Neurosurgery
1514 Jefferson Highway
New Orleans, LA 70121

JAMES CORRELL (Cynthia) 1966
249 Olde Pointe Rd.
Hampstead, NC 28443

ROBERT CROWELL (Mary) 1990
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

WILLIAM FEINDEL (Faith) 1959
Montreal Neurological Institute
3801 University Street
Montreal, Quebec FH3A 2B4
Canada

EUGENE 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

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

PETER HEILBRUN (Robyn) 1984
Division of Neurosurgery #3B409
Univ. of Utah Medical Center
50 North Medical Drive
Salt Lake City, UT 84132

E. BRUCE HENDRICK (Gloria) 1968
63 Leggett Ave.
Weston, Ontario M9P1X3
Canada

ROBERTO C. HEROS (Deborah) 1985
Department of Neurosurgery
Box 297 UMHC
420 Delaware St., S.E.
Minneapolis, MN 55455

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

JOHN A. JANE (Noella) 1982
Dept. of Neurosurgery
University of Virginia
Charlotteville, VA 22908

ELLIS KEENER (Ann) 1978
434 Academy Street, NE
Gainesville, GA 30501

DAVID KELLY, JR. (Sarah {Sally}) 1975
Department of Neurosurgery
Bowman Gray School of Medicine
Medical Center Blvd.
Winston-Salem, NC 27157-1029

PATRICK KELLY (Caitlin) 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

WOLFF KIRSCH (Marie-Claire) 1971
Loma Linda University Med. Ctr.
Division of Neurosurgery
11234 Anderson Street, Rm. 2539
Loma Linda, CA 92354

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

RICHARD S. KRAMER (Mollie) 1978
Duke University Medical Center
Box 3255
Durham, NC 27710

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

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

DONLIN M. LONG (Harriet) 1983
Dept. of Neurological Surgery
Johns Hopkins Medical School
600 N. Wolfe, Meyer 7-109
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ALFRED LUESSENHOP (Frances) 1977
Georgetown University Hospital
3800 Reservoir Road
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CHRISTOPHER LOFTUS (Sara) 1992
Div. of Neurosurgery, Univ of Iowa Hosp.
200 Hawkins Drive
Iowa City, IA 52242

L. DADE LUNSFORD (Julianne) 1992
B-400, Presbyterian University Hospital
DeSoto & O'Hara Streets
Pittsburgh, PA 15213

LEONARD MALIS (Ruth) 1973
1148 Fifth Avenue
New York,NY 10128

ROBERT L. MARTUZA (Jill) 1989
Georgetown University Medical Center
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ROBERT E. MAXWELL 1992
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JOE MAURICE MCWHORTER (Barbara) 1989
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RICHARD MORAWETZ (Mary Jean) 1990
University of Alabama
Division of Neurosurgery
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JOHN F. MULLAN (Vivian) 1963
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PAUL B. NELSON 1991
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FRANK NULSEN 1956
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Department of Neurological Surgery RI-20
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Montreal Neurological Hospital
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Department of Neurological Surgery
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Med. Univ. of South Carolina
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The Neurological Institute - Columbia Univ.
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Neurosurgery / Bader 3
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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
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NICHOLAS T. ZERVAS (Thalia) 1972
Massachusetts General Hospital
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JOHN KAPP 1985
P.O. Box 448
Galax, VA 24333
(703) 236-2613

ROBERT BOURKE 1983
5802 Nicholson Lane, Ste. 305
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SENIOR CORRESPONDING — ELECTED

JEAN BRIHAYE (van Geertruyden) — 1975
Belgium 98
avenue Des Franciscains
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KARL AUGUST BUSHE (Eva-Christa) — 1972
Technische Universitat Dresden
Helmholtxstrasse 18
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JOHN HANKINSON (Nicole) — 1973
Westacres
Woolsington Hall
Newcastle-Upon-Tyne, NE13 8DG
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SHOZO ISHII (Akiko) — 1975
Department of Neurosurgery
Juntendo Medical College
Tokyo 113, Japan

HANS-PETER JENSEN — 1980
Neurochirurgische
Universitatsklinik Kiel
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KATSUTOSHI KITAMURA (Yoshiko) — 1970
1-3-1 Kanada
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KRISTIAN KRISTIANSEN 1967
Ulleval Hospital
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WILLIAM LUYENDIJK 1973
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B. RAMAMURTHI (Indira) 1973
Voluntary Health Services
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KURT-FRIEDRICH SCHURMANN 1978
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CORRESPONDING

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LEIGH ATKINSON 1989
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FERNANDO CABIESES 1966
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Av. Arenales 371, of. 501
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LUC CALLIAUW (Dora) 1988
Dept. of Neurosurgery, University Hospital
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JUAN CARDENAS 1966
Insurgentes Sur 594
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JUAN CHRISTENSEN (Diana Poli) 1970
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H. ALAN CROCKARD (Caroline) 1992
Dept. of Surgical Neurology National Hospital
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GUISEPPE DALLE ORE (Guisi) 1970
Clinica Neurochirurgica
Universita di Verona
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NOEL GEORGE DAN (Adrienne) 1989
Specialist Medical Center, Suite 302
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JACQUES DEVILLIERS (Jeanne Marie Erica) 1986
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HANS ERICH DIEMATH (Karin) 1970
Landesnervenklinik, Dept . of Neurosurgery
5020 Salzburg, Ignaz Harrer-StraBe 79
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HERMANN DIETZ (Elfrun) 1970
Department of Neurosurgery
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VINKO DOLENC (Petra) 1988
Univ. of Ljubljana/Neuro.
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RUDOLPH FAHLBUSCH (Hanna) 1991
Neurochirurgische Klinik
Universitat Erlangen-Nurnberg
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JOHN GILLINGHAM 1962
Royal Infirmary
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Scotland, United Kingdom

JAIME G. GOMEZ (Lucy) 1975
5353 N. Federal Highway, #210
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SALVADOR GONZALEZ-CORNEJO (Rosa) 1982
Av. Chapultepec Sur 130-204
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ERNST GROTE (Juliana) 1984
Department of Neurosurgery
University Cliniks Schnarrenberg
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Germany

DAE HEE HAN (Sung Soon Cho) 1991
#28 Yougon-dong
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HAJIME HANDA (Hiroko) 1985
Takeda General Hospital
28-1 Moriminami-cho Ishida
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FABIAN ISAMAT (Maria V. {Marivi}) 1989
Clinica Sagrade Familia
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RICHARD JOHNSON 1974
Dept. of Neurological Surgery
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LAURI LAITINEN (Kerstin) 1972
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FRANK MARGUTH 1978
Clinic in Klinikum Grosshadom
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800 Munich, 70, Germany

RAUL MARINO, JR. (Milu) 1977
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Instituto Neurologico de S. Paulo
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J. DOUGLAS MILLER (Margot) 1988
Department of Clinical Neurosciences
Western General Hospital
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KENICHIRO SUGITA (Yasuko) 1988
Department of Neurosurgery
Nagoya Univ. School of Medicine
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Nagoya, 466, Japan

CHARAS SUWANWELA 1972
Chulalongkorn Hospital Medical School
Bangkok, Thailand

LINDSAY SYMON (Pauline) 1982
Gough-Cooper ept. of Neurological Surgery
Institute of Neurology, The National Hospital
Queen Square London WC1N 3BG
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KINTOMO TAKAKURA 1988
University of Tokyo Hospital
7-2-1 Hongo, Bunkyu-Ku
Tokyo 113, Japan

KJELD VAENET 1970
Department of Neurosurgery
Rigshospitalet 9 Blegdamsvej
Copenhagen 2100
Denmark

SIDNEY WATKINS 1975
The London Hospita
Whitechapel
London E 1, England

M. GAZI YASARGIL (Dianne) 1975
Neurochirurgie FMH
Sonneggstrasse 6
8091 Zurich, Switzerland

DECEASED MEMBERS

	Deceased	Elected
SIXTO O. ALCALDE Madrid, Stain (Honorary)	4/28/78	1973
JAMES R. ATKINSON Phoenix, Arizona (Active)	2/78	1970
PERCIVAL BAILEY Evanston, Illinois (Honorary)	8/73	1960
WILLIAM F. BESWICK Buffalo, New York (Active)	5/71	1959
EDWIN B. BOLDREY San Francisco, California (Senior)	6/6/88	1941
SPENCER BRADEN Cleveland, Ohio (Active)	7/69	Founder
F. KEITH BRADFORD Houston, Texas (Active)	4/71	1938
HOWARD BROWN San Francisco, California (Senior)	2/90	1939
DONALD COBURN Wilmington, Delaware (Senior)	9/88	1938
WINCHELL McK. CRAIG Rochester, Minnesota (Honorary)	2/60	1942

EDWARD DAVIS Portland, Oregon (Senior)	10/88	1949
PEARDON DONAGHY Burlington, Vermont (Senior)	11/26/91	1970
FRANCIS ECHLIN New Paltz, New York (Senior)	4/20/88	1944
DEAN ECHOLS New Orleans, Louisiana (Senior)	11/28/91	Founder
GEORGE EHNI Houston, Texas (Senior)	9/86	1964
ARTHUR ELVIDGE Montreal, Quebec, Canada (Senior)	1/85	1939
THEODORE C. ERICKSON Madison, Wisconsin (Senior)	10/86	1940
JOSEPH P. EVANS Kensington, Maryland (Senior)	5/85	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)	7/75	1942
HANNIBAL HAMLIN Providence, Rhode Island (Senior)	6/82	1949
HENRY L. HEYL Hanover, New Hampshire (Senior)	3/75	1951
OLAN HYNDMAN Iowa City, Iowa (Senior)	6/66	1942
KENNETH G. JAMIESON Brisbane, Australia (Corresponding)	1/76	1970
SIR GEOFFREY JEFFERSON Manchester, England (Honorary)	3/61	1951
WILLIAM S. KEITH Toronto, Canada (Senior)	12/87	Founder
HUGO KRAYENBUHL Zurich, Switzerland (Honorary)	1985	1974
WALPOLE S. LEWIN Cambridge, England (Corresponding)	1/80	1973
HERBERT LOURIE Syracuse, New York (Senior)	3/87	1965
M. STEPHEN MAHALEY Birmingham, Alabama (Active)	1992	1972

GEORGE L. MALTBY Scarsborough, Maine (Senior)	4/88	1942
DONALD D. MATSON Boston, Massachusetts (Active)	5/69	1950
FRANK MAYFIELD Cincinnati, Ohio (Senior)	1991	Founder
AUGUSTUS McCRAVEY Chattanooga, Tennessee (Senior)	1990	1944
KENNETH G. McKENZIE Toronto, Ontario, Canada (Honorary)	2/64	1960
JAMES M. MEREDITH Richmond, Virginia (Active)	12/62	1946
W. JASON MIXTER Woods Hole, Massachusetts (Honorary)	3/68	1951
EDMUND J. MORRISSEY San Francisco, California (Senior)	2/86	1941
GOSTA NORLEN Goteborg, Sweden (Honorary)	1985	1973
PIETRO PAOLETTI Milan, Italy (Corresponding)	1991	1989
HANS-WERNER PIA Geissen, West Germany (Corresponding)	7/86	1978

WILDER PENFIELD Montreal, Canada (Honorary)	4/76	1960
HELMUT PENZHOLZ Heidelberg, West Germany (Corresponding)	1985	1978
RUPERT B. RANEY Los Angels, California (Active)	11/59	1939
BRONSON RAY New York, New York (Honorary)	1993	1992
DAVID L. REEVES Santa Barbara, California (Active)	8/70	1939
DAVID REYNOLDS Tampa, Florida (Active)	4/78	1964
R.C.L. ROBERTSON Houston, Texas (Senior)	2/85	1946
STEWART N. ROWE Pittsburgh, Pennsylvania (Senior)	10/84	1938
RICHARD C. SCHNEIDER Ann Arbor, Michigan (Senior)	6/86	1970
WILLIAM B. SCOVILLE Hartford, Connecticut (Senior)	2/84	1944
R. EUSTACE SEMMES Memphis, Tennessee (Honorary)	3/82	1955

SAMUEL R. SNODGRASS Galveston, Texas (Senior)	8/75	1939
GLEN SPURLING LaJolla, California (Honorary)	2/68	1942
C. WILLIAM STEWART Montreal, Quebec, Canada (Corresponding)	1948	1948
THORALF SUNDT, JR. Rochester, Minnesota (Active)	1992	1971
HENDRIK SVIEN Rochester, Minnesota (Active)	6/72	1957
HOMER S. SWANSON Atlanta, Georgia (Senior)	6/87	1949
ALFRED UIHLEIN Rochester, Minnesota (Senior)	1990	1950
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