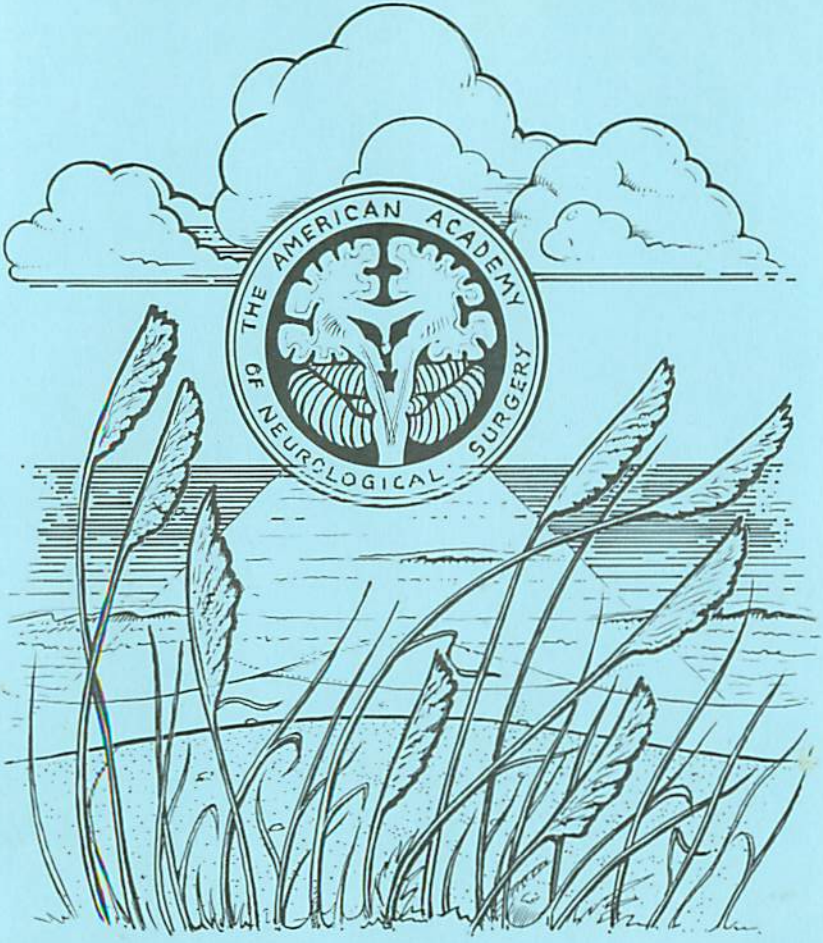

AMELIA ISLAND PLANTATION

AMELIA ISLAND, FLORIDA

1990

THE 52nd ANNUAL MEETING OF

THE

AMERICAN ACADEMY OF

NEUROLOGICAL SURGERY

AMELIA ISLAND PLANTATION
AMELIA ISLAND, FLORIDA

OCTOBER 3 - 7, 1990

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THE AMERICAN ACADEMY
OF NEUROLOGICAL SURGERY

October 3-7, 1990
Amelia Island Plantation
Amelia Island, Florida

Wednesday, October 3, 1990

1:00 PM-5:30 PM — Registration
Executive Conference Center Lobby

1:30 PM-4:30 PM — Executive Committee meeting
President's Suite

6:30 PM-9:00 PM — Welcoming Reception and Buffet
Oceanside at Beach Club

Thursday, October 4, 1990

7:00 AM-8:00 AM — Breakfast Business Meeting
(Members Only)
Ballrooms B, C
Executive Conference Center

8:00 AM-5:00 PM — Registration
Executive Conference Center Foyer

8:00 AM-1:00 PM — Scientific Meeting
Ballroom A
Executive Conference Center

AFTERNOON FREE

6:30 PM — Florida Style Reception and Buffet Dinner
Ballrooms A, B, C
Executive Conference Center

Friday, October 5, 1990

7:00 AM-8:00 AM	Breakfast Business Meeting (Members Only) Ballrooms B, C Executive Conference Center
8:00 AM-5:00 PM	Registration Executive Conference Center
8:00 AM-1:00 PM	Scientific Meeting Ballroom A Executive Conference Center
AFTERNOON FREE	
1:00 PM	Tennis Tournament-Susan and John Tew Coordinators Tennis Center
6:00 PM	Gourmet Dinner (Reservation time to be assigned) Duneside Club Amelia Island Inn

Saturday, October 6, 1990

7:00 AM-8:00 AM	Breakfast Meeting (Members and Guests) Ballrooms B, C Executive Conference Center
8:00 AM-1:00 PM	Scientific Meeting Ballroom A Executive Conference Center
9:40 AM	Presidential Address Robert G. Ojemann, M. D.

AFTERNOON FREE

1:00 PM	Golf Tournament-John VanGilder Coordinator Golf Pro Shop
7:00 PM	Annual Reception Ballrooms A, B, C Executive Conference Center
8:00 PM-1:00 AM	Annual Banquet and Dance Ballrooms A, B, C Executive Conference Center (black tie)

Sunday, October 7, 1990 Travel Day

CONTINUING MEDICAL EDUCATION

The Joint Committee on Education of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons designates this continuing medical education activity for (13.5) credit hours in Category I toward the Continuing Education Award in Neurosurgery and the Physician's Recognition Award of the American Medical Association. The Joint committee on Education of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

Spouses Activities

Wednesday, October 3, 1990

6:30 PM-9:00 PM — Welcoming Reception and Buffet
Oceanside at Beach Club

Thursday, October 4, 1990

8:00 AM-9:30 AM — Spouses Hospitality
Continental Breakfast
Ibis Room, Racquet Park Center

9:45 AM-12:30 PM — Amelia Plantation Tour of Homes
Depart from Racquet Park Center

6:30 PM — Florida Style Reception and Buffet Dinner
Ballrooms A, B, C
Executive Conference Center

Friday, October 5, 1990

8:15 AM-9:45 AM — Spouses Hospitality
Continental Breakfast
Verandah Restaurant

10:00AM-11:15AM — Presentation by:
Mrs. J. Parker Mickle, Author of
THE QUEEN OF OCTOBER
Verandah Restaurant

1:00 PM — Tennis Tournament-
Susan and John Tew, coordinators

6:00 PM — Gourmet Dinner (Reservation times to be assigned)
Duneside Club
Amelia Island Inn

Saturday, October 6, 1990	
8:15 AM-9:20 AM	**Spouses Hospitality** **Continental Breakfast** **Verandah Restaurant**
9:40 AM	**Presidential Address** **Ballroom A** **Executive Conference Center**
11:00 AM-1:00 PM	**Round Robin Tennis** **Tennis Center**
1:00 PM	**Golf Tournament-John VanGilder, coordinator** **Golf Pro Shop**
7:00 PM	**Annual Reception** **Ballrooms A, B, C** **Executive Conference Center**
8:00 PM	**Annual Banquet and Dance** **Ballrooms A, B, C** **Executive Conference Center** **(black tie)**
Sunday, October 7, 1990	**TRAVEL DAY**

SCIENTIFIC PROGRAM

Thursday, October 4, 1990

SCIENTIFIC SESSION I

Stewart B. Dunsker-Moderator
Chairman, Program Committee

8:15AM **WELCOME**

Robert G. Ojemann
President

8:20AM **FOLLOW UP AND EXPERIENCE WITH 114 PINEAL TUMORS OPERATED**
Jeffrey Bruce, Bennett Stein; New York City, New York

8:40AM **RADIOSENSITIZATION WITH CAROTID ARTERIAL INFUSION OF BROMODEOXYURIDINE + 5 FLUOROURACIL WITH EXTERNAL BEAM RADIATION FOR MALIGNANT GLIOMAS**
William F. Chandler, Harry S. Greenberg, Larry Junck; Ann Arbor, Michigan

9:00AM **INTEGRATED MOLECULAR GENETIC MODEL FOR GLIAL TUMOR EVOLUTION**
C.David James, Ju He, James I. Ausman; Detroit, Michigan

9:20AM **MANAGEMENT OF AXIAL LOW GRADE ASTROCYTOMAS IN CHILDHOOD**
H.J.Hoffman, D. Solonluk, B. de Lima, L. Becker, J.M. Drake, R.P. Humphreys, E.B. Hendrick; Toronto,Canada

9:40AM **RESULTS OF RADIOSURGERY 'UPFRONT' FOR MALIGNANT GLIOMAS**
Eben Alexander, III, Jay S. Looffler, Peter McL. Black; Boston, Massachusetts

10:00AM **CORRELATION BETWEEN INVASION AND CLINICAL EVALUATION OF MENINGIOMAS**
Luc Caillauw, Leo de Ridder; Ghent-Belgium

Thursday, October 4, 1990

10:20AM SELECTIVE DESTRUCTION OF HUMAN GLIOMA CELLS BY A THYMIDLINE KINASE DELETION MUTANT OF HERPES SIMPLEX VIRUS-1
Robert L. Martuza, Amy Malick, Donald Coen; Boston, Massachusetts

10:40AM COFFEE

Thursday, October 4, 1990

SCIENTIFIC SESSION II
Albert Rhoton-Moderator
Chairman, Award Committee

11:05AM ACADEMY AWARD PRESENTATION

ANATOMIC EVIDENCE OF NOCICEPTIVE INPUTS TO PRIMARY SOMATOSENSORY CORTEX: RELATIONSHIP BETWEEN SPINOTHALAMIC TERMINALS AND THALAMOCORTICAL CELLS IN SQUIRREL MONKEYS
Scott I. Gingold,M.D.; State University of N.Y. College of Medicine, Syracuse, NY

11:20AM THE ROLE OF THE EXTERNAL CAROTID ARTERY IN THE TREATMENT OF CEREBRAL AND RETINAL ISCHEMIA
Arthur Day, Robert L. Masson,Jr.; Gainesville, Florida

11:40AM CEREBRAL ISCHEMIA DURING CAROTID ENDARTERECTOMY
Richard Morawetz;
Birmingham, Alabama

Thursday, October 4, 1990

12:00PM LATEX BALLOON TREATMENT OF 28 INTRACRANIAL ANEURYSMS
Robert Crowell, John Pile-Spellman, Robert Heros, John K. Chin, Lofti Haclen-Bey, Robert M. Crowe; Boston, Massachusetts

12:20PM MANAGEMENT OF INTRAOPERATIVE ANEURYSM RUPTURE WITHOUT HYPOTENSION
Steven Giannotta, Jeffrey H. Oppenheimer, Michael Levy; Los Angeles, California

12:40PM SPECIAL CHARACTER OF MACROCIRCULATION
Ralph Dacey; St. Louis, Missouri

1:00PM ADJOURN

Friday, October 5, 1990

SCIENTIFIC SESSION III
Roberto Heros-Moderator
Program Committee

8:00AM ANEURYSM SURGERY: OUTCOME COMPARISON (EARLY VS. DELAYED)
Richard Fraser, Dirk Brunner, Michael Deck; New York, New York

8:20AM TRANSTORCULAR EMOBLIZATION OF VEIN OF GALEN ANEURYSMS: AN UPDATE OF THE USE OF THIS TECHNIQUE IN 24 PATIENTS
J. Parker Mickle; Gainesville, Florida

8:40AM ARTERIAL RECONSTRUCTION UTILIZING BOVINE PERICARDIAL PROSTHESIS AND NON-PENETRATING CLIPS DURING THE COURSE OF ANEURYSM SURGERY
Wolff M. Kirsch, Zh Zhu, R. Cushman, R.A.Hardesty; Loma Linda, California

Friday, October 5, 1990

9:00AM PULSED DYE LASER TREATMENT OF EXPERIMENTAL VASOSPASM
Robert Macfarlane, Nicholas T. Zervas
Boston, Massachusetts

9:20AM INTRAOPERATIVE ANGIOGRAPHY DURING ANEURYSM REPAIR
Charles Hodge, Jeffery Winfield, Gerald Rodziewics, Mark Jones, Edwin Cacayorin, Catherine Chu; Syracuse, New York

9:40AM PRINCIPAL OF DYNAMIC NEOCORTICAL FUNCTION: A NEUROLOGICAL BASIS OF FUNCTIONAL RECOVERY FOLLOWING BRAIN INJURY
Michael Merzenick; San Francisco, CA

10:10AM HUMAN MOTOR AND LANGUAGE LOCALIZATION AFTER CORTICAL INJURY
George Ojemann; Mercer Island, Washington

10:30 AM COFFEE

Friday, October 5, 1990

SCIENTIFIC SESSION IV
George Ojemann-Moderator
Program Committee

10:50AM PAIN MANAGEMENT IN HERPES ZOSTER
Robert King; Syracuse, New York

11:10AM IMPLANTATION OF NERVE GROWTH FACTOR PRODUCING FIBROBLASTS INTO THE BRAIN PROTECTS AGAINST EXCITOTOXIC STRIATAL LESIONS
James Schumacher; Boston, Massachusetts

11:30AM THE ROLE OF THALAMOTOMY IN THE PROGRESS OF PARKINSON'S DISEASE
Ronald Tasker, G.T. de Carvalho, C.S. Li; Toronto, Ontario, Canada

Friday, October 5, 1990

11:50AM SECTION OF THE CORPUS CALLOSUM IN CHILDREN
Robert Maxwell, Frank Ritter; Minneapolis, Minnesota

12:10PM MOTOR EVOKED RESPONSES AND H-REFLEXES ARE SENSITIVE INDICATORS OF SPINAL CORD ISCHEMIA
Lawrence Borges, Nicholas T. Zervas
Boston, Massachusetts

12:30PM VASCULAR MECHANISMS OF SECONDARY SPINAL CORD INJURY
Charles Tator; Toronto,Ontario,Canada

12:50PM ADJOURN

Saturday, October 6, 1990

SCIENTIFIC SESSION V
John Jane-Moderator
Vice-President

8:00AM SPONTANEOUS CEREBROSPINAL FLUID LEAKS FROM THE MIDDLE FOSSA
Suzie Tindall, Atlanta, Georgia

8:20AM HYDROCEPHALUS; OVERDRAINAGE BY VENTRICULAR SHUNTS-A REVIEW WITH RECOMMENDATIONS
Eldon Foltz, Robert H. Pudenz; Orange, California

8:40AM SURGICAL MANAGEMENT OF CHRONIC THORACIC HERNIATED DISCS
Martin Weiss; Los Angeles, California

Saturday, October 6, 1990

9:00AM LATERAL PARASCAPULAR EXTRAPLEURAL APPROACH TO THE THORACIC SPINE
Richard Fessler, Albert L. Rhoton Jr.; Gainesville, Florida

9:20AM PROGRESSION AND RECURRENCE OF LUMBAR STENOSIS FOLLOWING SURGICAL DECOMPRESSION
Anthony Caputy, Alfred J. Lussenhop; Washington, D.C.

9:40AM PRESIDENTIAL ADDRESS-ROBERT G. OJEMANN
INTRODUCTION BY JOHN JANE

10:20AM COFFEE

Saturday, October 6, 1990

SCIENTIFIC SESSION VI

Robert G. Ojemann-Moderator
President

10:40AM SKULL BASE SURGERY: A SYMPOSIUM

10:45AM SURGERY OF CAVERNOUS SINUS
Vinko V. Dolenc; Ljub.jana, Yugoslavia

11:15AM APPROACHES TO THE CLIVUS
John Tew, Jr.; Cincinnati, Ohio

11:45AM SKULL BASE SURGERY IN CHILDREN
Derek Bruce; Dallas, Texas

12:15PM QUESTIONS

12:30PM ADJOURN

Thursday, October 4

8:20 a.m.

Follow Up and Experience With 114 Pineal Tumors Operated

Jeffrey Bruce and Bennett Stein; New York City, NY

114 cases of pineal tumor surgery done over the past decade have been reviewed in terms of pathology, appropriate treatment and long-term results.

The principle of our treatment is to identify histologically all tumors in the pineal region before treatment. Treatment can include combinations of the following: surgery, radiation and chemotherapy.

Overall 30% of the tumors are benign, resectable and require no additional treatment. The story is not glum however, for the malignant tumors that cannot be totally resected. The results in the treatment of germinoma for example have been excellent, with 85% success in terms of tumor control and minimal morbidity.

The indications for chemotherapy are few and will be discussed in some detail. The role of stereotactic biopsy will also be discussed in light of improved preoperative diagnostic techniques, including MRI with gadolinium.

The overall results of treatment of any pineal lesion are in the range of 80% good to excellent result. Mortality rate has been 5%.

8:40 a.m.

2

Radiosensitization with Carotid Arterial Infusion of Bromodeoxyuridine ± 5 Flourouracil with External Beam Radiation for Malignant Gliomas

William Chandler, Harry Greenberg and Larry Junck; Ann Arbor, MI

A permanently implantable Infusaid pump system has been developed for safe continuous intra-arterial (IA) carotid BUdR ± 5FU infusion. BUdR ± 5FU is delivered IA in the carotid system because of its regional advantage. Two clinical studies were initiated for the treatment of malignant glioma of the brain with IA BUdR ± 5FU with concurrent partial brain radiation to 5,940 cGy. Twenty-three patients have been treated on the initial protocol with IA BUdR alone in doses of

400 to 600 mg/m²/day. The maximum tolerated dose was 400 mg/m²/day of continuous infusion for 8 1/2 weeks. The median survival is 20 months with a median followup of 20 months. In the second trial, 25 patients were entered on IA BUdR ± 5FU. Two patients died before treatment assessment due to pulmonary embolus and bee sting anaplaxis. The median survival of 23 patients completing therapy is 17 (± 3.5 S.E.) months with a median followup of 9 months. The Kaplan Meier estimated median survival of all adequately treated patients in trials 1 and 2 (total = 46) is 20.0 (± 3.5 S.E.) months. No vascular complications have occurred in either trial. Continuous IA infusion of BUdR ± 5FU is feasible, safe, and represents a potential means enhancing the effectiveness of radiotherapy in the treatment of malignant gliomas.

9:00 a.m.
3
Integrated Molecular Genetic Model for Glial Tumor Evolution

C. David James, Ju He, James I. Ausman; Detroit, MI

We have applied molecular genetic analysis to a panel of primary glial tumors to examine these tumors for alterations of two types: loss of genetic information, as revealed by RFLP analysis, and gene amplification. Our data imply a minimum of six specific genetic alterations which frequently occur and accumulate during the malignant evolution of gliomas.

For low malignancy grade gliomas, deletion of sequences from chromosomes 13, 17p, or 22 are the most frequently detected alterations. Amongst tumors of intermediate and high histologic malignancy, loss of genetic information from chromosome 10 and amplification of the epidermal growth factor receptor (EGFR) gene are associated with the glioblastomas, and are therefore indicative of advanced tumor malignancy.

Of the frequently occurring alterations, one clearly involves a specific gene, EGFR. Three additional alterations imply the targeting of specific genes (chromosome 17p deletions suggest the p53 gene, 13 deletions suggest the Rb gene, and 9p deletions suggest the interferon alpha and beta

loci). Loss of genetic information from chromosomes 10 and 22 involve tumor suppressor genes yet to be identified.

In total, these data form an outline of genetic events in glioma progression which is consistent with those developed for other human solid tumors, by suggesting a subset of genes whose alteration are of fundamental importance to glial tumor development and evolution. Furthermore, these data suggest that there is a preferred sequence of accumulation of such alterations and, therefore, provide a framework for the molecular genetic staging of these tumors. A presentation of this data and discussion of its implications in brain tumors will be made.

9:20 a.m.
4
Management of Axial Low Grade Astrocytomas In Childhood
H.J. Hoffman, D. Soloniuk, B. de Lima, L. Becker, J.M. Drake, R.P. Humphreys, E.B. Hendrick; Toronto, Canada

Patients with axial low grade astrocytomas involving the midline structures of the brain, unlike patients with astrocytomas in the cerebral hemisphere and cerebellum, can rarely have their tumor totally excised by virtue of the location of the tumor. For many decades extreme conservatism dominated the neurosurgical attitude towards these axial tumors in childhood. CSF diversion followed by radiotherapy with or without biopsy was the standard of care.

We have reviewed our experience with these low grade astrocytomas during an era when modern technology has allowed us to safely operate on these tumors, namely the period between January,1976 and December, 1989. A total of 88 patients were operated upon during this period of time. Forty-one patients had tumors within optic pathways and hypothalamus, 12 within the thalamus, 5 within the pineal region, 17 within the midbrain and 13 within the medulla.

Thirty-six of these 88 patients received radiotherapy in addition to their surgery. The remainder were treated by resection alone or in combination with chemotherapy.

Four of the 36 patients who received radiotherapy developed radiation induced tumors. Twelve of the 88 patients have died. The survival probability at 10 years of a patient with a midline low grade astrocytoma is 80% at 10 years.

Direct surgery can now be carried out on low grade midline astrocytomas without significant morbidity or mortality. Many of these patients with such tumors do well with resection as the only form of therapy. The residual tumor will frequently involute after resection with no other therapy being necessary. The response to radiotherapy is variable and since radiotherapy can produce serious sequelae, we now only use this modality of therapy for those low grade tumors which recur quickly or which are endangering vital function.

Axial low grade astrocytomas can no longer be regarded as inoperable neoplasms to be treated with radiotherapy and diversionary shunting.

9:40 a.m.
5
Results of Radiosurgery "Upfront" for Malignant Gliomas

Eben Alexander, III, Jay S. Loefler, Peter Black; Boston, MA

Malignant gliomas have been difficult lesions to control with surgery and fractionated external beam irradiation alone. In those patients with small (35 mm maximum diameter), deep high grade gliomas, or in those who were not deemed candidates for placement of afterloading catheters for stereotactic brachytherapy, stereotactic radiosurgery with a modified linear accelerator offers an alternative. Most of the patients were treated as outpatients with a single fraction. Treatment planning, which is fully three-dimensional, is performed on a Stardent GS-1000 computer based on stereotactic MRI or CT information. Multiple non-coplanar arcs are directed from a Clinac 6/100 linear accelerator with special collimators and BRW headstand. Treatments have required 3-10 non-coplanar arc rotations. Collimators between 22.5 mm and 40 mm (median 30mm) have been used to

treat lesions with volumes between 6 cc and 38 cc (median 14 cc), utilizing multiple isocenters in two cases. Doses prescribed varied inversely with the size of the collimator, and ranged from 700 cGy to 2000 cGy (median 1500 cGy). Doses were prescribed to the periphery of the lesion and were normalized to the 45-90% isodose line (median 80%). Ten of twelve patients treated had GBMs, the others anaplastic lesions. Nine of twelve patients are alive with clinical and radiographic stabilization, with follow-up ranging from 2 to 18 months (4 are greater than nine months). Six received 6000 cGy of standard radiotherapy, and three elderly patients were treated with radiosurgery alone. Two patients underwent craniotomies for removal of necrotic tumor at 10 and 14 months after radiosurgery because of neurologic deterioration. Pathologic examination resembled that seen for reoperation after i-125 brachytherapy: widespread necrosis with occasional scattered tumor cells at the boundary, which were very difficult to grow in culture. Three patients have died: at 3 months (rapidly progressive ALS undiagnosed at treatment), 4 months (seizure/hypoxia leading to herniation; post mortem revealed little remaining tumor), and 10 months (marginal failure,tumor regrowth). Radiosurgery, used as a "boost" upfront at diagnosis, with appropriate margins around the enhancing volume, is a powerful tool for gaining local control of these lesions.

10:00 a.m.
6
Correlation Between Invasion and Clinical Evaluation of Meningiomas
Luc Calliauw, Leo de Ridder; Ghent, Belgium

Thirty two freshly isolated meningiomas are cut into small fragments and explanted in Falcon plastics. During incubation in vitro, the fragments adhere to the artificial substrate and did grow out and formed monolayers. At saturation density the cells are scraped off as cellular flaps and confronted with embryonic host tissue. As host tissue 9 days old embryonic chick heart fragments are used. During confrontation in vitro the evolution of both compartments, the chick heart and the meningioma derived cell flaps, are followed. After 1, 2, 4 and 7 days in vitro the confrontations

are fixed and stained.

Three different histological patterns can be observed; Type I, includes necrotized meningial cells; type II presents a survival of meningial cells encircled by heart cells or heart cells lying next to the meningial cells. Type III includes meningial cells proliferating and invading the host tissue. In this last type, the heart tissue is progressively replaced by the meningioma derived cells.

These growth data are confronted with clinical parameters and, in some cases, with DNA flow cytometry of the culture. The culture system presented provides valuable information about the intrinsic characteristics of meningiomas and especially their invasive capacity.

10:20 a.m.

7

Selective Destruction of Human Glioma Cells by a Thymidine Kinase Deletion Mutant of Herpes Simplex Virus-1

Robert L. Martuza, Amy Malick, Donald Coen; Boston, MA

Glioblastomas are the most common malignant brain tumors and are almost universally fatal due to local growth. We are exploring a novel form of local treatment using a thymidine kinase negative mutant of herpes simplex virus-1 (HSV-1). A glioblastoma is a dividing cell population expressing DNA replication enzymes, but normal brain is mostly composed of non-dividing cells with such enzymes minimally expressed. We postulated that a HSV-1 negative for thymidine kinase activity might replicate within the malignant glioma cells but not within normal brain.

To explore this, we used the HSV-1 deletion mutant, HSV1-dlsptk. A human glioma cell line (U-87) was grown in monolayer culture using Vero cells as a positive control. To each were applied either wild type (HSV1-KOS) or mutant(HSV1-dlsptk) virus at multiplicities of infection ranging from 10^{-4} to 10. Cytopathic effect was noted in each cell type within 24 hours and was proportional to the multiplicity of infection. By 9 days following infection at the lowest multiplicity, both cell types were completely destroyed demonstrating that even at a low multiplicity of

infection, **HSV-dlsptk** was able to sustain a spreading infection to destroy the entire monolayer of **U-87** cells. These studies have been repeated with similar results in another human glioma cell line (**T98G**) and in 3 primary human gliomas in cell culture.

To explore the effects of **HSV-dlsptk** on normal brain, we inoculated male Fischer rats (180g) in the right frontal lobe with $2X10^5$ plaque forming (**PFU**) **HSV-dlsptk.** Four weeks following infection, 11/11 were alive and healthy. This is consistent with prior studies of a **TK**-deficient herpes virus and contrasts with prior studies using wild type virus (**HSV1-KOS**) wherein a similar dose caused death in 7/8 rats within 1 week.

We conclude that genetically altered viruses are worthy of further investigation as a novel means of tumor therapy.

Friday, October 5

8:00 a.m.

1

Aneurysm Surgery: Outcome Comparison (Early vs. Delayed

Richard A. R. Fraser, Dirk Brunner, Michael Deck
New York, NY

The risks and benefits of early aneurysm surgery (i.e.: within 3 days of subarachnoid hemorrhage) are a source of continued dispute. Some recent reports would suggest there is no significant difference in outcome, both in terms of mortality and morbidity and advocate therefore early surgery in order to eliminate the risk of rebleeding while awaiting the optimal surgical opportunity.

A retrospective review of the last 100 patients admitted to The New York Hospital with a diagnosis of subarachnoid hemorrhage (SAH) yielded 89 patients' with a documented aneurysm. 85 of these were located in the anterior circulation. These patients hospital charts were retrospectively reviewed to determine their outcome. The latter was allocated into the various levels of the Glasgow outcome scale or a mortality category. Approximately one third of aneurysms operated on at NYH/CUMC underwent

surgery within 3 days of an SAH, two thirds of our SAH/aneurysm population received a delayed procedure. A good recovery was achieved in 74% of those receiving a late operation and 65% in those patients operated on within 3 days of an SAH. Morbidity and mortality were similarly higher in those patients receiving an early operation. None of these outcome categories reached a statistically significant difference.

The contributions of vasospasm (documented in 32 patients) intraoperative aneurysm rupture and postoperative documented infarctions to these outcome categories have been reviewed.

These data do not reveal an outcome that clearly documents a statistically significant advantage to early or to late operation.

8:20 a.m.
2
Transtorcular Embollzation of Vein of Galen Aneurysms: An Update of the Use of This Technique in 24 Patients
J. Parker Mickle; Gainesville, FL

The vein of Galen malformations remain a frustrating and significant challenge to the pediatric neurosurgeon and his medical colleagues. It has become clear that the therapeutic goal in this central shunt has to be individualized, especially as relates to the presentation and the age related symptomology. We have utilized the transtorcular approach in the treatment of 24 vein of Galen aneurysm patients at the University of Florida between the years 1982-1989. Nine neonates with severe high output cardiac failure have been treated with 4 survivors in this group. Fifteen infants and older children are included in this series and as expected have faired better with one death occurring in this group. There are nine persistent fistulae in this group. Follow-up ranges from 6-60 months. Detailed presentation of the surgical technique of transtorcular embolization and critical analysis of the outcome in these 24 patients will be presented. The endovascular treatment of vein of Galen aneurysms is becoming the treatment of choice in this complex disease process and the results are improving dramatically.

Friday, October 5
8:40 a.m.
3

Arterial Reconstruction Utilizing Bovine Pericardial Prosthesis and Non-Penetrating Clips During the Course of Aneurysm Surgery

Wolff M. Kirsch, Zh Zhu, R. Cushman, R.A. Hardesty; Loma Linda CA

Five technically problematic intracranial aneurysms had parient arterial reconstructions and preservation during the course of surgical intervention when sacrifice was imminent. The lesions included: Case I, a giant left vertebral artery aneurysm with medullary compression; Case 2, a right vertebral dissecting aneurysm with multiple subarachnoid bleeds; Case 3, a traumatic, medially presenting, infraclinoid right internal carotid aneurysm; Case 4, a wide based basilar bifurcation aneurysm that fractured at its neck following conventional clip placement. Intraoperative angiography was utilized to monitor arterial patency. Case 5, a megagiant left middle cerebral artery aneurysm that enlarged despite previous investiture. Reconstructions were successful in Cases 2,3,4, and 5. Case 1 required eventual ligation of the left vertebral artery. Case 5 had an acute occlusion of an interposed saphenous vein graft.

The non-penetrating clip, its unique detachable tang and slender applier enables facile and unambiguous tissue approximation and fixation in awkward anatomical sites. Glutaraldehyde treated and deantigenized bovine pericardium provides and excellent patch for either arterial wall substitution or plication when attached with clips. The non-penetrating feature prevents intimal damage.

The combination of clips and pericardial prosthesis have other applications in neurosurgical procedures to include dural and sinus closure.

Friday, October 5
9:00 a.m.
4

Pulsed Dye Laser Treatment of Experimental Vasospasm

Robert Macfarlane, Nicholas T. Zervas; Boston, MA

The ability of laser energy at a wavelength of 480um to treat vasospasm was investigated in 2 animal models. Rabbit common carotid arteries (CCA) were constricted by the applicaton of human blood within a silicone sleeve, impregnated with 70% isopropanol to induce slow lysis of erythrocytes. Maximum constriction was achieved 24-48 hours later, and persisted for 5-6 days. Vessels showed histological characteristics of vasculopathy. Endovascular laser treatment was delivered from a lus pulsed dye laser via a 200um quartz fiber introduced from the femoral artery. In 30 vessels, CCA diameter 24 hours after the induction of spasm was increased from 60% of control to a minimum post laser diameter of 81% (P 0.01). There were no cases of laser-induced perforation or of arterial thrombosis. Aneurysmal dilatation was not observed for up to 60 days after treatment, even after ligation of the contralateral CCA, which induced a uniform 28% increase in arterial caliber. Prophylactic irradiation of normal CCAs (n=5) was able to prevent the development of immediate vasospasm. Histological examination demonstrated focal loss of endothelial cells immediately after treatment, but there was no disruption of the medial or adventitial layers.

In a second model, 4 dog basilar arteries were constricted by 2 intracisternal injections of autologous blood, 3 days apart. Two dogs received endovascular laser treatment 10 days later. In each case, the basilar artery was restored from 69% to 104% of control diameter. No neurological sequelae developed after treatment. In both groups basilar artery diameter was less than control at 30 days (78% and 79% respectively), but the vasodilatory response to hypercapnia was preserved.

These findings suggest a possible role for endovascular laser therapy in both the treatment of established cerebral vasospasm, and as prophylaxis in patients at high risk of developing this complication after SAH.

Friday, October 5
9:20 a.m.
5

Intraoperative Angiography During Aneurysm Repair

Charles Hodge, Jeffery Winfield, Gerald Rodzieics, Mark Jones, Edwin Cacayorin, Catherine Chu; Syracuse, NY

Technical errors that occur during repair of intracranial aneurysms include incomplete obliteration of the aneurysm neck and occlusion of the major arterial branch or the parent vessel of the lesion. This report describes the use of intraoperative angiography to attempt to identify and correct these errors.

Eighty-five patients undergoing 89 intracranial procedures for repair of 120 aneurysms were studied with intraoperative angiography. Immediately prior to surgery the patient was taken to the angiography suite where a heparinized catheter was placed transfemorally in the appropriate external carotid artery. Patency of the catheter was maintained with a slow heparin drip. After what the surgeon judged adequate obliteration of the lesion, the catheter was pulled down into the common carotid artery and an angiographic run done using portable digital subtraction technique.

No patients were found in whom the aneurysm(s) was not obliterated. A single patient with an anterior communicating aneurysm was found on follow up angiography, done for spasm, to have residual aneurysm requiring a second procedure. Six (6.7%) of the patients were found to have major branch occlusions. Four of these were corrected. Four (4.5%) patients were found to have occlusion or severe stenosis of the parent vessel. Two of these were repaired. Another patient undergoing simultaneous endarterectomy and repair of an ophthalmic artery aneurysm was found to have residual carotid stenosis requiring revision of the arteriotomy. The outcomes of the patients without successful repair of the intraoperatively identified abnormality was poor in 1 case, excellent in 2 cases and fair in the remainder. One patient in whom repair was successful had a fair recovery, while the other 6 had excellent recoveries. This technique was least useful in internal carotid-posterior communicating aneurysms and most useful in lesions of the anterior communicating artery, the middle cerebral artery and the carotid-ophthalmic artery junction. No patient had identifiable ischemic deficit related to prolonged catheterization of the carotid artery.

Friday, October 5
9:40 a.m.
6
Basic Science Lecture
Principal of Dynamic Neocortical Function: A Neurological Basis of Functional Recovery Following Brain Injury
Michael Merzenich; San Francisco, CA

10:10 a.m.
7
Human Motor and Language Localization After Cortical Injury
George A. Ojemann; Boston, MA

Functional localization has been investigated with intraoperative electrical stimulation mapping in patients with chronic lesions in motor and language areas. In a patient with a tumor in hand motor cortex and minimal functional deficit, the entire upper extremity motor representation had shrunk to a fraction of its usual extent, demonstrating in human motor cortex the type of reorganization described by Merzenich following lesions of monkey somatosensory cortex. In patients with lesions of language cortex (previous frontotemporal stroke and trauma, frontal tumor) and mild to moderate expressive aphasia, consistent residual language representation was confined to one posterior language area, on the margin of the area of injury. Remapping of patients with progressive extension of tumors through language cortex demonstrated that the appearance of a mild aphasia was associated with the replacement of one site previously consistently related to language by a wider area less consistently essential for language, with little change in other essential language areas. Thus with injury to language association cortex there does not seem to be displacement of language representation to new areas, but rather residual language function is maintained by the remaining essential areas.

10:30 a.m. Coffee

Friday, October 5
10:50 a.m.
8
Pain Management In Herpes Zoster
Robert King; Syracuse, NY

A simple method of achieving substantial pain control in patients with documented herpes zoster, or postherpetic neuralgia, has been effective in the vast majority of patients in whom it has been implemented. It is far more effective than the usual analgesics, or any operative procedure that we have used in the past. Although its origins are pragmatic, there is now at least a reasonable rationale on the basis of which to consider its effectiveness.

The technique involves topical application of aspirin dissolved in chloroform, applied directly over the areas of scarring and the surrounding area of skin. Dramatic relief is usually evident within less than five minutes, and by thirty minutes, most patients indicate they have minimal or no discomfort at all. The relief lasts for a variable period of time, from two to ten hours. Side effects (mild skin rash on three cases) have not been a problem. Application of the suspension/solution is difficult in the posterior portion of a dermatome and some patients need help in that regard. The details of the effectiveness and characteristics of the response to this simple procedure will be presented. Virtually all of the patients managed in this fashion have been dramatically relieved of their immediate pain, and over a period of weeks or months, even those with postherpetic neuralgia clear and remain comfortable.

11:10 a.m.
9
Implantation of Nerve Growth Factor Producing Fibroblasts Into the Brain Protects Against Excitotoxic Striatal Lesion
James B. Schumacher; Boston, MA

Except for L-dopa pharmacological replacement therapy in Parkinson's disease, neurodegenerative diseases lack effective treatment. Previous studies suggest that symptoms arise secondary to defects in local neuronal circuitry and cannot be

treated effectively with systemic drug delivery. Therefore, stereotaxic application of fetal or genetically engineered cells which replace or protect deficient regions is promising. Engineered cells can be derived from cell-lines or grown from recipient fibroblasts, then modified to produce and secrete substances at the target. Previous studies using pharmacologic nerve growth factor (NGF) infusions in parallel with excitotoxic lesions of rat striatum have indicated NGF protective effects (Aloe, 1987). In order to further test this hypothesis we have utilized a biological delivery system of NGF by implanting fibroblasts genetically engineered to secrete high levels of NGF into the rat brain, prior to infusing an excitotoxin into the striatum.

A rat derived immortalized fibroblast cell-line (208F) was infected by a NGF retroviral vector (N.8) and selected for neomycin resistance. A NGF+ cell-line and a non-NGF variant (NGF-) of the 208F fibroblast cell line was injected into the lateral ventricle and mid-line structures. After eight days, the excitotoxin quinolinic acid was infused into the ipsilateral striatum. Histological evaluation showed surviving grafts both in the NGF- and the NGF+ group. In Nissl and GFAP stained sections, the NGF+ group had smaller lesions than the NGF- group.

These results indicate that implantation of genetically engineered cells can be used to protect and modify brain function. This technique also challenges the use of systemic drug therapy in neurodegenerative disorders and provides an alternative to using fetal tissue in neurotransplantation.

11:30 a.m.
10
The Role of Thalamotomy in the Progress of Parkinson's Disease

R. Tasker, G.T. de Carvalho and C.S. Li; Toronto, Ontario

The possible role of deprenyl in arresting the progress of Parkinson's disease recalls earlier impressions expressed by Irving Cooper that bilateral surgery effective for the control of tremor and rigidity could arrest the progress of the disease. The interesting work of Matsumoto in Tokushima, Japan, a former fellow of Cooper's, seemed to suggest he was right.

Friday, October 5

Since we have records of 55 patients with Parkinson's disease who underwent bilateral thalamotomy and who were followed long-term, many over 15-20 years after their last procedure, all assessed by one of us (RRT) at each followup visit according to a fixed protocol, a review was carried out in 1988-89, visiting elderly or disabled patients in their place of residence wherever necessary and feasible.

Techniques of assessment, results and complications of bilateral surgery, and long-term progress of the disease will be presented.

The bottom line is that patients with post-encephalitic disease or that of onset below age 40 may progress so slowly so as to appear arrested, regardless of bilateral successful surgery. The progress of disease in such patients is an order of magnitude slower than that in some akinetic-rigid patients treated with deprenyl. There is an occasional non-youthful, non-post-encephalitic patient whose disease progresses similarly. There is only slight evidence that "successful" bilateral surgery is more likely to result in arrest of disease progress than unilateral surgery or than bilateral "unsuccessful" surgery.

11:50 a.m.

11

Section of the Corpus Callosum In Children

Robert Maxwell and Frank Ritter; Minneapolis, MN

The published experience with corpus callosotomy for intractable epilepsy in children has been limited to two small series and case reports included within series of predominantly older patients. Cases in the literature to date were selected mainly as an alternative to hemispherectomy and on the basis of secondary generalization of seizure discharges.

The purpose of this paper is to report the Minnesota experience with corpus callosotomy on 19 children, 12 years of age or younger, and to compare our experience with that in the combined literature. Children were initially selected for surgery because: 1. seizures were refractory to medications; 2. seizures were associated with frequent falls and injuries; 3. no localized area for focal resection was identified; 4. seizures were secondarily generalized; 5. the seizures were

harming the quality of the child's life and making care of the child difficult. Later in the series, intractable secondary generalized tonic-clonic seizures were included in the selection criteria as an alternative seizure type.

In the Minnesota series, 9 patients had recurrent status epilepticus prior to surgery and only one patient has had an episode of status epilepticus following callosotomy. Sixteen of 19 children had Lennox-Gastaut Syndrome and 12 of the 16 had a positive outcome from surgery. Tonic (drop) seizures associated with an ictal electrodecremental change on EEG have been the most responsive. Atonic seizures and secondarily generalized tonic-clonic seizures also improve following corpus callosotomy.

Corpus callosotomy is tolerated better in children than adults with faster recovery. In addition to reduced severity and frequency of generalized seizures, there is often a dramatic impact of surgery on the quality of life. Behavior, self-care skills, attention span, language, social skills, peer interactions, alertness, and sleep patterns are improved following section of the corpus callosum in this selected population.

12:10 a.m.
1 2
Motor Evoked Responses and H-reflexes are Sensitive Indicators of Spinal Cord Ischemia
Lawrence F. Borges and Nicholas T. Zervas; Boston, MA

The prompt and efficient recognition of spinal cord ischemia remains an unfulfilled goal. We hypothesized that electrical monitoring of spinal cord function that included transmission of spinal cord impulses across a synapse should be a sensitive method of early detection of spinal cord ischemia. To test this hypothesis, we studied a model of spinal cord ischemia in 100 adult Sprague Dawley rats. The spinal cords were rendered ischemic by inflating a Fogarty balloon catheter in the aorta, just distal to the take-off of the left subclavian artery. Before, during and after ischemia we measured motor evoked potentials from the motor cortex to the left gastrocnemius muscle. We observed that both the motor evoked potentials and the H-reflex disappeared within 30 to 60 seconds after balloon inflation, indicating that these

measures are very sensitive to spinal cord blood flow. Ischemia times of 5 minutes or less resulted in the return of these responses and was often accompanied by significant spinal cord reflex hyperexcitability. These observations demonstrate that the monitoring of synaptic function in the spinal cord is a sensitive measure of spinal cord ischemia and deserves further investigation.

12:30 p.m.
13
Vascular Mechanisms of Secondary Spinal Cord Injury
Charles H. Tator; Toronto, Ontario, Canada

In patients with spinal cord injury, the primary or mechanical injury seldom causes total transection, even though the functional loss may be complete. In addition, the biochemical and pathological changes in the cord may worsen after injury. To explain these facts the concept of the secondary injury has evolved for which numerous pathophysiological mechanisms have been postulated. This paper reviews the concept of the secondary injury with special emphasis on vascular mechanisms. Our laboratory and others have found evidence to support the theory of the secondary injury and that its chief mechanism is pottraumatic ischemia and infarction of the spinal cord. The evidence for the role of vascular mechanisms has been obtained with a variety of models of acute spinal cord injury (ASCI) in several species. Various investigators have used several different angiographic methods for assessing the microcirculation of the cord and for measuring spinal cord blood flow (SCBF) after trauma. With these tehniques major systemic and local vascular effects of ASCI have been identified and implicated in the etiology of the secondary injury.

The systemic effects of ASCI include hypotension and reduced cardiac output. The local effects include loss of autoregulation in the injured segment of the spinal cord, and a marked reduction of the microcirculation in both the grey and white matter, expecially in hemorrhagic regions and in adjacent zones. The microcirculatory loss extends for a considerable distance proximal and distal to the site of injury.

Friday, October 5

Many studies have shown a dose-dependant reduction of SCBF varying with the injury severity, and that the reduction of SCBF worsens with time after injury. The functional deficits due to ASCI have been measured electrophysiologically with techniques such as motor and somatosensory evoked potentials and have been found to be proportional to the degree of posttraumatic ischemia. The histological effects of ASCI include early hemorrhagic necrosis leading to major infarction at the injury site.

These posttraumatic vascular effects can be treated. Systemic normotension can be restored with volume expansion or vasopressors, and SCBF can be improved with dopamine, nimodipine or volume expansion. The combination of nimodipine and volume expansion improves posttraumatic SCBF and spinal cord function measured by evoked potentials. These results provide strong evidence that posttraumatic ischemia is an important secondary mechanism of injury, and that it can be counteracted.

12:50 a.m. Coffee

Saturday, October 6

8:00 a.m.

Spontaneous Cerebrospinal Fluid Leaks From the Middle Fossa

Suzie C. Tindall; Atlanta, GA

Spontaneously occurring cerebrospinal fluid (CSF) leaks, not associated with trauma, previous surgery, or skull base tumors, are uncommon. Five cases of such fistulas arising from congenitally thin regions in the greater wing of the sphenoid wing have resulted in CSF leaks into the sphenoid sinus. Two of these cases had an associated empty sella which was not the source of the leak. In one case a defect in the petrous portion of the temporal bone was the source of a leak into the middle ear. Such lesions are best investigated using thin section computed tomography with bone window imaging techniques, and metrizamide cisternography. Attempts to treat these leaks transsphenoidally have resulted in either failure to stop the leak or in the development of medial temporal lobe abscess. Our experience with these lesions suggests that the leak sites are best obliterated using a direct transcranial repair.

Saturday, October 6
8:20a.m.
2

Hydrocephalus: Overdrainage by Ventricular Shunts-A Review With Recommendations

Eldon L. Foltz, Robert Pudenz; Pasadena, CA

Ventricular shunting for hydrocephalus is essential in neurosurgery but is associated with many complications. This literature review concerns overdrainage by ventricular shunts. Several observations and conclusions are presented:

1) In reference to overdrainage problems, normal ICP in the supine position is 100 to 150 mm of water above reference point; in the upright position, the maximum negative ICP is 120 mm of water. Overdrainage problems may show as low as -480 mm H_2O (upright).

2) Four primary clinical entities occur from ventricular overshunting:

A) Subdural hematoma;
B) Acquired craniostenosis;
C) Slit ventricle syndrome;
D) The low ICP syndrome.

Diagnosis, cause, and treatment are briefly reviewed.

3) Conclusions presented are:

A) Overall incidence of overdrainage is 10-12% of patients shunted by ventricular shunts;

B) Average time of appearance broadly averaged is $6\frac{1}{2}$ years after the initial shunt.

C) Reference points for measuring intracranial pressure are varied and inconsistent, and precise comparisons of literature reports are difficult.

4. Three major recommendations are presented from this review:

A) Common usage of a reference point for intracranial pressure is necessary in view of the increasing recognition of the importance of supine position and upright position pressures; the uppermost portion of the CSF system for whatever body position is used is recommended as a logical reference point for such;

B) An external ventricular shunt should be limited to patients in which no other method will work, and then the system must control the negative ICP when upright;

C) Intracranial operations should be considered as an early option in all cases and a more complex work-up including measurement of residual CSF absorption capacity and CSF absorption deficit can be done and should be further developed. This will allow better long-term prognosis in these difficult cases of hydrocephalus.

8:40 a.m.
3
Surgical Management of Chronic Thoracic Herniated Discs

Martin H. Weiss; Los Angeles, CA

Over the period 1971-1989, the author has operated upon 46 patients with chronic herniated thoracic discs. The patients ranged in age from 19 to 72; 29 were females, 17 males. Most lesions involved the lower thoracic spine, 14 were located at T9-10, 11 at T8-9; the remainder were scattered from T4-5 to T11-12. Thirty-three patients (72%) had demonstrable calcification in the involved interspace; 6 discs (13%) had penetrated the dura. Selective angiography was performed for all lesions at T9-10 or lower; the artery of Adamkiewicz was identified in one case at the T9-10 level resulting in modification of the planned approach.

The earliest symptom was pain, either radiating in a thoracolumbar distribution (12) or focal (9). The most common presenting symptom was a gait disturbance in 31 patients; 21 patients complained of distal paresthesiae upon presentation. Bowel or bladder symptoms were found in only 8 patients (17%). On physical examination, the most common finding was an extensor plantar response in one or both toes (61%); 57% exhibited a spastic paresis. MR is the best screening technique to define the existence and locus of these lesions; CT myelography appears to be better to define the presence of dural penetration.

All patients underwent an antero-lateral trans-thoracic resection of the herniated disc. Thirty-five patients

underwent fusion across the excised interspace; the need for post excisional fusion appears to be diminishing as experience has been gained.

There was no mortality. Of the 26 patients with motor deficit preoperatively, 25 were improved and one patient was unchanged; one patient without preoperative motor deficit was made transiently worse. Preop sensory deficits improved in 13 of 17 patients (76%); pain syndromes improved in 23 of 25 (92%), and extensor plantar responses reverted to normal in 27 of 28 (96%) patients.

It appears that this approach provides a reasonable option to the neurosurgeon to resolve these difficult problems.

9:00 a.m.
4
Lateral Parascapular Extrapleural Approach to the Thoracic Spine
Richard G. Fessler, Albert L. Rhoton; Gainesville, FL

The upper thoracic vertebrae are difficult to approach surgically because of the narrowing of the thoracic inlet, the brachial plexus, and the dorsomedial shoulder musculature. Although pathology in this region is not common, the occurrence of upper thoracic vertebral metastases necessitates the availability of decompressive surgery for neurologic complications.

We have developed a lateral parascapular extrapleural approach to the upper thoracic vertebrae. The dorsomedial shoulder musculature (levator scapulae, rhomboid and trapezius muscles) are reflected off the spinous processes to the scapula as a myocutaneous flap preserving the neurovascular supply. the upper dorsal ribs are removed with caution to avoid injury to the C8 and T1 nerve roots. The vertebrae of T2-T4 can then be approached unobstructed. the T1 nerve root and the stellate ganglion obstruct posterolateral access to the T1 vertebrae, necessitating an inferolateral approach underneath the T1 nerve root axilla.

Four patients with compressive myelopathy from upper thoracic spinal metastasis underwent neural decompression, intervertebral body fixation, and posterior spinal stabilization

with this approach. The postoperative neurologic status was unchanged or improved. Complications included radiographic pleural effusion, and one patient required posterior spinal reinstrumentation for progressive kyphosis. There was no functional disability of the shoulder, Horner's syndrome, or medial arm hypalgesia. One patient developed pneumonia seven days postoperatively which was unresponsive to appropriate treatment.

We feel that the limitations to this region have been overcome, and that excellent exposure for neural decompression and intervertebral body fixation can be performed safely. A major advantage is that posterior spinal instrumentation can be performed at the same time. Multiple level disease can be treated effectively. The limitation to this approach is disease extending into the C7 vertebrae.

Saturday, October 6
9:20 a.m.
Progression and Recurrence of Lumbar Stenosis Following Surgical Decompression
Anthony J. Caputy, Alfred J. Luessenhop; Washington, DC

Seventy-seven patients who underwent lumbar spine surgery for degenerative spinal stenosis from 1980 to 1985 were reviewed. The medial age was 67 years ranging from 43 to 84 years. Thirty-nine percent presented with neurogenic claudication only and 42 percent had various combinations of radicular symptoms only. All patients underwent myelography. The surgery involved facet and wide bony and ligamentous decompression in all cases and 70 percent had wide foraminal decompression as well. Sixteen percent had significant intervertebral disc pathology that necessitated simultaneous disc removal. The number of levels decompressed ranged from one to five.

The lower extremity symptoms were improved immediately in all except four patients in whom the pain persisted in the immediate post-operative period leading to a re-operation during the same hospital stay. Twenty-three percent had a recurrence of symptoms of spinal stenosis with cauda equina

or renewed root involvement at a median time of 3.5 years following surgery with the range being two months to eight years, and of these, most had the recurrence at levels above or below the previously decompressed levels. The remaining patients exhibited recurrence at previously operated sites. Re-operation was carried out in most. Three patients had symptoms referable to acquired instability.

These follow-up results of surgery indicate that while the early post-operative results show a very satisfactory symptomatic improvement, the degenerative process continues with high probability that the symptoms may recur from levels adjacent to the initial sites of involvement or a recurrence at the original operative sites.

9:40 a.m. **PRESIDENTIAL ADDRESS**
Introduction:
John Jane: Vice-President

10:20 a.m. **Coffee**

10:40 a.m. **Skull Base Surgery: A Symposium**

10:45 a.m. **Surgery of Cavernous Sinus**
Vinko V. Dolenc; Ljubljana, Yugoslavia

11:15 a.m. **Approaches to the Clivus**
John Tew; Cincinnati, OH

11:45 a.m. **Skull Base Surgery in Children**
Derek Bruce; Dallas, TX

12:15 p.m. **Questions**

1:00 p.m. **Adjourn**

NOTES

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STATE UNIVERSITY OF NEW YORK COLLEGE OF MEDICINE

ANATOMIC EVIDENCE OF NOCICEPTIVE INPUTS TO PRIMARY SOMATOSENSORY CORTEX: RELATIONSHIP BETWEEN SPINOTHALAMIC TERMINALS AND THALAMOCORTICAL CELLS IN SQUIRREL MONKEYS

RUNNER UP

JAMES M. SCHUMACHER, M.D.

DEPARTMENT OF NEUROSURGERY
MASSACHUSETTS GENERAL HOSPITAL

IMPLANTATION OF NERVE GROWTH FACTOR PRODUCING FIBROBLASTS INTO THE BRAIN PROTECTS AGAINST EXCITOTOXIC STRIATAL LESIONS

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JAMES T. ROBERTSON	1984-86
NICHOLAS T. ZERVAS	1987-89
WILLIAM A. BUCHHEIT	1990-

PAST TREASURER

RUSSEL H. PATTERSON,JR.	1973
PHANOR L. PEROT,JR.	1974-76
JOHN T. GARNER	1977-80
NICHOLAS T. ZERVAS	1984-86
WILLIAM A. BUCHHEIT	1987-89
JULIAN T. HOFF	1990-

PAST MEETINGS OF THE ACADEMY

HOTEL NETHERLAND PLAZA,CINCINNATI, OHIO	OCTOBER 28-29,1938
ROOSEVELT HOTE, NEW ORELEANS, 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, CHICHAGO, ILLINOSI	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 HOTE, 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
BILMORE HOTEK,SANTA BARBARA, CALIFORNIA	OCTOBER 12-14,1953
BROADMOOR HOTE, 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 HOTEL,MEXICO CITY	NOVEMBER 18-21,1970
SAHARA-TAHOE HOTEL, STATELINE, NEVADA	SEPTEMBER 26-29,1971
NEW COLLEGE, OXFORD, ENGLAND	SEPTEMBER 4-7,1972
HUNTINGTON-SHERATON HOTEL, PASADENA, CALIFORNIA	NOVEMBER 14-17,1973
SOUTHAMPTON PRINCESS HOTEL, SOUTHAMPTON, 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 2-25,1978
HYATT REGENCY, MEMPHIS, TENNESSEE	NOVEMBER 76-10,1979
WALDORF ASTORIA, NEW YORK, NEW YORK	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 RESORT, TUCSON, ARIZONA	SEPTEMBER 27-OCTOBER 1, 1989

1990
MEMBERSHIP LIST
AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
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GUY LAZORTHES — 1973
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VALENTINE LOGUE (Anne) — 1974
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EBEN ALEXANDER, JR. (Betty) Bowman Gray School of Medicine 300 South Hawthorne Winston-Salem, North Carolina 27103	1950
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PETER M. BLACK (Katherine) Brigham and Women's Hospital 75 Francis Street Boston MA 02115	1988
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JERALD S. BRODKEY (Arielle) 24755 Chagrin Boulevard Suite #205 Beachwood, Ohio 44122	1977
WILLIS E. BROWN, JR. (Ann) Division of Neurosurgery The University of Texas Health Science Ctr. 7703 Floyd Curl Drive San Antonio, Texas 78284-7843	1984
DEREK A. BRUCE (Frances) 7777 Forrest Lane #C703 Dallas, Texas 75230	1984
WILLIAM A. BUCHHEIT (Lyn) 3401 North Broad Street Philadelphia, Pennsylvania 19140	1980

WILLIAM F. CHANDLER (Sue) 1989
2124D/338 Taubman Center
1500 East Medical Ctr. Drive
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PAUL H. CHAPMAN (Tansy) 1983
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WILLIAM F. COLLINS, JR. (Gwendolyn) 1963
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333 Cedar Street
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EDWARD S. CONNOLLY (Elise) 1973
Ochsner Clinic
1514 Jefferson Highway
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JAMES W. CORRELL (Cynthia) 1966
710 West 168th Street
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STEWART B. DUNSKER (Ellen) 1975
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506 Oak Street
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HOWARD M. EISENBERG (Janet) 1985
The University of Texas Medical Branch
Division of Neurosurgery
Galveston, Texas 77550

WILLIAM H. FEINDEL (Faith) 1959
Montreal Neurological Institute
3801 University Street
Montreal, Quebec, Canada H3A 2B4

EUGENE FLAMM (Susan) 1979
Hospital of the University of Pennsylvania
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RICHARD A.R. FRASER (Sarah Anne) 1976
525 East 68th Street
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HENRY GARRETSON (Marianna) 1973
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316 MDR Bldg.
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ROBERT G. GROSSMAN (Ellin) 1984
Baylor College of Medicine
One Baylor Place
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ROBERT GRUBB (Julia) 1985
Washington University School of Medicine
4901 Barnes Hospital Plaza
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GRIFFITH R. HARSH, III (Craig) 1980
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UBA Station
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MARK PETER HEILBRUN (Robyn) 1984
Division of Neurosurgery #3B320
Univesity of Utah Medical Center
50 North Medical Drive
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Hospital for Sick Children
555 University Avenue, Room 1502
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420 Southwest Delaware Street Box 96
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CHARLES HODGE (Linda) 1982
750 East Adams Street
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JULIAN HOFF (Diane) 1975
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HAROLD HOFFMAN (Jo Ann) 1982
The Hospital for Sick Children
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EDGAR M. HOUSEPIAN (Marion) 1976
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ALAN R. HUDSON (Susan) 1978
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JOHN A. JANE (Noella) 1982
Department of Neurosurgery, Box 212
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ELLIS B. KEENER (Ann) 1978
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Box C-307
University of Colorado Medical Center
4200 East 9th Avenue
Denver, Colorado 80262

WOLFF M. KIRSCH (Marie-Claire) 1971
Loma Linda University Medical Ctr.
Loma Linda CA 92354

DAVID G. KLINE 1972
Louisiana State University Medical Center
1542 Tulane Avenue
New Orleans, Louisiana 70112

RICHARD S. KRAMER (Mollie) 1978
Duke Hospital Medical Center
Durham, North Carolina 27710

THEODORE KURZE 1967
University of Pittsburgh
Department of Neurosurgery
9402 Presbyterian University Hospital
230 Lothrop Street
Pittsburgh, PA 15213

SANFORD LARSON (Jackie) 1989
Medical College of Wisconsin
8700 W. Wisconsin/Neurosurgery
Milwaukee WI 53226

EDWARD R. LAWS, JR. (Peggy) 1983
George Washington Medical Center
2150 Pennsylvania Ave. NW
Washington, D. C. 20037

DONLIN M. LONG (Harriet) 1983
Department of Neurological Surgery
Johns Hopkins Medical School
601 N. Wolfe
Baltimore, Maryland 21205

ALFRED J. LUESSENHOP (Frances) 1976
Georgetown University Hospital
3800 Reservoir Road
Washington, D. C. 20007

JOE MAURICE McWHORTER (Barbara) 1989
Bowman Gray School of Medicine
Winston-Salem NC 27103

LEONARD MALIS (Ruth) 1973
1176 Fifth Avenue
New York, New York 10029

ROBERT L. MARTUZA (Jill) 1989
Massachusetts General Hospital
Fruit Street
Boston MA 02114

JOHN F. MULLAN (Vivian) 1963
5844 Stoney Isle Avenue
Chicago, Illinois 60637

FRANK E. NULSEN (Ginny) 1956
32 10th Avenue, South
Naples, Florida 33940

GEORGE OJEMANN (Linda) 1975
6424 E. Mercer Way
Mercer Island, Washington 98040

ROBERT G. OJEMANN (JEAN) 1968
Neurosurgery Service
Massachusetts General Hospital
Boston, Massachusetts 02114

ANDRE OLIVIER (Nichole) 1989
3801 University Street
Suite #107
Montreal PQ H3A 2B4

BURTON ONOFRIO (Judith) 1975
Mayo Clinic
Rochester, Minnesota 55905

RUSSEL H. PATTERSON, JR. (Julie) 1971
New York Hospital
525 East 68th Street
New York, New York 10021

SYDNEY J. PEERLESS (Ann 1977
University Hospital
339 Windermere Road
London, Ontario, Canada N6a 5A5

PHANOR L. PEROT, JR. 1970
Department of Neurosurgery
Medical Univeristy of South Carolina
171 Ashley Avenue
Charleston, South Carolina 29425

BYRON C. PEVEHOUSE (Lucy) 1964
2351 Clay Street
San Francisco, CA 94115

DAVID G. PIEPGRAS (Jane) 1987
Mayo Clinic
200 First Street, S.W.
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DONALD O. QUEST (Ilona) 1968
The Neurological Institute
710 West 168th Street
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University Hospital
2074 Abington Road
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ALBERT L. RHOTON, JR. (Joyce) 1984
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J. CHARLES RICH, JR. (Jasmine) 1987
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KINTOMO TAKAKURA — 1988
University of Tokyo Hospital
7-3-1 Hongo, Bunkyu-ku
Tokyo 113, Japan

KJELD VAENET (Ann) — 1970
Department of Neurosurgery
Rigshospitalet
9 Blegdamsvej
2100 Copenhagen, Denmark

SIDNEY WATKINS — 1975
The London Hospital
Whitechapel, London E 1 England

GAZI YASARGIL (Dianne) — 1975
Neurosurgical Clinic
University Hospital
Ramistrasse 10
CH-8091 Zurich, Switzerland

SENIOR CORRESPONDING MEMBERS

JEAN BRIHAYE (Martine Van Geertruyden) — 1975
98 Ave. Des Franciscainn
1150 Bruxelles, Belgium

KARL AUGUST BURHE (Eva)
Neurochirurgischen Klinik
Josef-Schneider-Strasse II
D-8700 Wurzburg, West Germany

JOHN HANKINSON (Nicki) 1973
Westacres
Woolsington Hall
Newcastle-Upon-Tyne
England

SHOZO ISHII 1975
Department of Neurosurgery
Juntendo Medical College
Tokyo 113, Japan

HANS-PETER JENSEN (RETA) 1980
Neurochirurgische Universitatsklinik Kiel
Weimarer Strasse 8
D-2300 Kiel/West Germany

KATSUTOSHI KITAMURA (Yoshiko) 1970
Shinkokura Hospital
1-2-1 Kanada
Kokurakita-Ku
Kitakyushu, 803 Japan

KRISTIAN KRISTIANSEN (Brit) 1962
Ullevai Hospital
0407 Oslo, 4 Norway

WILLIAM LUYENDIJK (Tony) 1973
Pr Bernhardlaan 60
Oegstgeest, The Netherlands
B. RAMAMURTHI (Indira) 1966
2nd Main Road G.I.T. Colony
Madras 4, India 600 004

KURT SHURMANN 1978
Director
Neurochirurg
Univ-Klinik Mainz
Langenbeskstr 1
6500 Mainz, West Germany

DECEASED MEMBERS		ELECTED
SIXTO OBRADOR ALCALDE Madrid, Spain (Honorary)	4/1978	1973
JAMES R. ATKINSON Phoenix, Arizona (Active)	2/1978	1970
PERCIVAL BAILEY Evanston, Illinois (Honorary)	8/1973	1963
EDWIN B. BOLDREY San Francisco, California (Senior)	6/1988	1941
SPENCER BRADEN Cleveland, Ohio (Active)	7/1969	Founder
HOWARD A. BROWN Walnut Creek, California (Senior)	2/1990	1939
DONALD COBURN Wilmington,Delware (Senior)	9/1988	1938
WINCHELL McK. CRAIG Rochester, Minnesota (Honorary)	2/1960	1942
EDWARD DAVIS Troutdale, Oregon (Senior)	10/1988	1949
FRANCIS ECHLIN New Paltz, New York (Senior)	4/1988	1944

GEORGE EHNI **Houston, Texas** **(Senior)**	**9/1986**	**1964**
ARTHUR ELVIDGE **Montreal,Quebec, Canada** **(Senior)**	**1/1985**	**1939**
THEODORE C. ERICKSON **Madison, Wisconsin** **(Senior)**	**10/1986**	**1940**
JOSPEH P. EVANS **Kensington, Maryland** **(Senior)**	**5/1985**	**Founder**
JOHN D. FRENCH **Los Angeles, California** **(Senior)**	**1/1989**	**1951**
JOHN R. GREEN **Phoenix, Arizona** **(Senior)**	**1/1990**	**1953**
WESLEY A. GUFTAFSON **Jensen Beach, Florida** **(Senior)**	**7/1975**	**1942**
HANNIBAL HAMLIN **Providence, Rhode Island** **(Senior)**	**6/1982**	**1941**
HENRY L. HEYL **Hanover, New Hampshire** **(Senior)**	**3/1975**	**1951**
OLAN HYNDMAN **Iowa, City, Iowa** **(Senior)**	**6/1966**	**1942**

KENNETH G. JAMIESON Brisbane, Australia (Corresponding)	7/1976	1970
SIR GEOFFREY JEFFERSON Manchester, England (Honorary)	3/1961	1951
WILLIAM KEITH Toronto, Canada (Senior)	12/1987	Founder
HUGO KRAYENBUHL Zurich, Switzerland (Honorary)	1985	1974
WALPOLE S. LEWIN Cambridge, England (Corresponding)	1/1980	1973
HERBERT LOURIE Syracuse, New York (Senior)	3/1987	1965
GEORGE L. MALTBY Boston, Massachusetts (Active)	1988	1942
DONALD D. MATSON Boston, Massachusetts (Active)	5/1969	1950
AUGUSTUS McCRAVEY Chattanooga, Tennessee (Senior)		1944
KENNETH G. McKENZIE Toronto, Ontario, Canada (Honorary)	2/1964	1960
JAMES M. MEREDITH Richmond, Virginia (Active)	12/1962	1946

W. JASON MIXTER Woods Hole, Massachusets (Honorary)	3/1968	1951
EDMUND J. MORRISSEY San Francisco, California (Senior)	2/1986	1941
HANS-WERNER PIA Giessen, West Germany (Corresponding)	7/1986	1978
WILDER PENFIELD Montreal, Canada (Honorary)	4/1976	1960
HELMUT PENZHOLZ West Germany (Corresponding)	1985	1978
RUPERT B. RANEY Los Angeles, California (Active)	11/1959	1939
DAVID L. REEVES Santa Barbara, California (Senior)	8/1970	1939
DAVID REYNOLDS Tampa, Florida (Active)	41978	1964
R.C.L. ROBERTSON Houston, Texas (Senior)	2/1985	1946
STEWART N. ROWE Pittsburgh, Pennsylvania (Senior)	10/1984	1938
RICHARD C. SCHNEIDER Ann Arbor, Michigan (Senior)	6/1986	1970

WILLIAM B. SCOVILLE Hartford, Connecticut (Senior)	2/1984	1944
R. EUSTACE SEMMES Memphis, Tennessee (Honorary)	3/1982	1955
SAMUEL R. SNODGRASS Galveston, Texas (Senior)	8/1975	1939
GLEN SPURLING LaJolla, California (Honorary)	2/1968	1942
C. WILLIAM STEWART Montreal, Quebec, Canada (Corresponding)	1948	1948
HENDRIK SVIEN Rochester, Minnesota (Active)	6/1972	1957
HOMER S. SWANSON Atlanta, Georgia (Senior)	6/1987	1949
THOMAS A. WEAVER, JR. Dayton, Ohio (Senior)	1985	1943
BARNES WOODHALL Durham, North Carolina (Senior)	1985	1941
FRANK WRENN Greenville, South Carolina (Active)	2/1990	1973

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