THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

THE RITZ CARLTON

NAPLES, FLORIDA

1992

THE 54TH ANNUAL MEETING OF

THE

AMERICAN ACADEMY OF

NEUROLOGICAL SURGERY

RITZ CARLTON

NAPLES, FLORIDA

OCTOBER 21 - 25, 1992

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THE AMERICAN ACADEMY OF
NEUROLOGICAL SURGERY

October 21 - 25, 1992
Ritz Carlton
Naples, Florida

Wednesday, October 21, 1992

1:00 - 5:00 PM	Registration Plaza Foyer
	Executive Committee Meeting Doctor Henry D. Garretson's Suite
6:00 - 8:30 PM	Welcoming Reception Center Court Alternate: Salon III & IV
	"WELCOME TO FLORIDA"

Thursday, October 22, 1992

7:00 - 8:00 AM	Breakfast Business Meeting (members only) Salon I
8:00 - 5:00 PM	Registration Plaza Foyer
8:00 - 10:20 AM	Scientific Meeting Plaza Ballroom
10:20 - 10:40 AM	Coffee Break Plaza Foyer

10:40 - 1:00 PM	Scientific Meeting Plaza Ballroom
1:30 P M	Golf Tournament Audubon Golf Club (transportation provided) John Van Gilder-Coordinator
7:00 - 9:30 PM	Beach Party North Beach Alternate: Salon III & IV

Friday, October 23, 1992

7:00 - 8:00 AM	Breakfast Business Meeting (members only) Salon I
8:00 - 5:00 PM	Registration
8:00 - 10:10 AM	Scientific Meeting Plaza Ballroom
10:10 -10:30 AM	Coffee Break Plaza Foyer
10:30 - 1:00 PM	Scientific Meeting Plaza Ballroom
4:00 - 6:00 PM	Tennis Tournament Tennis Center
6:30 - 7:30 PM	Reception (invitation only) Doctor Garretson's Suite

7:00 - 8:00 PM	Reception Ballroom Foyer
8:00 - 11:00 PM	Dinner Dance Salon III & IV

Saturday, October 24, 1992

7:00 - 8:00 AM	Breakfast Business Meeting (members only) Salon I
8:00 - 10:20 AM	Scientific Meeting Plaza Ballroom
10;20 - 10:40 AM	Coffee Break Plaza Foyer
10:40 - 1:00 PM	Scientific Meeting Plaza Ballroom

Sunday, October 25. 1992

Travel Day

CONTINUING MEDICAL EDUCATION

THE STATEMENT ON CERTIFICATION:

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) credit hours in Category 1 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.

GUEST ACTIVITIES

Wednesday, October 21, 1992

6:00 - 8:00 PM	Welcoming Reception Center Court Alternate: Salon III & IV "WELCOME TO FLORIDA"

Thursday, October 22, 1992

8:00 - 9:30 AM	Guest's Continental Breakfast The Grill
9:30 AM	Walking Tour of the Ritz Carlton Art Collection
1:30 PM	Golf Tournament John Van Gilder - Coordinator Audobon Golf Club Transportation Provided
7:00 - 9:30 PM	Beach Party North Beach Alternate: Salon III & IV

Friday, October 23, 1992

8:00 - 9:30 AM	Guest's Continental Breakfast The Grill
9:30 - 10:00AM	Naples Area Presentation The Grill
4:00 - 6:00 PM	Tennis Tournament Tennis Center Susan and John Tew Coordinators
7:00 - 8:00 PM	Reception Ballroom Foyer
8:00 - 11:00 PM	Dinner Dance Salon III & IV

Saturday, October 24, 1992

8:00 - 9:30 AM	Guest's Continental Breakfast The Grill

SCIENTIFIC PROGRAM

Thursday, October 22, 1992

8:00 AM WELCOME: Henry D. Garretson, President

MODERATOR: CHARLES J. HODGE, JR.

8:10 AM NEW INSIGHTS INTO THE VASCULATURE OF THE NORMAL AND TRAUMATIZED SPINAL CORD
Charles H. Tator

8:30 AM EXPERIMENTAL COMPRESSIVE CERVICAL MYELOPATHY
Ossama Al-Mefty

8:50-10:20 AM SYMPOSIUM: DEGENERATIVE LUMBAR DISEASE

8:50 - 9:08 AM LUMBAR DEGENERATIVE DISEASE: PATHOLOGY IMAGING
Frederick Simeone

9:08 - 9:26 AM LUMBAR STENOSIS AND AVOIDANCE OF HEAVY METAL POISONING
John Jane

9:26 - 9:43 AM INDICATIONS AND TECHNIQUES FOR LUMBAR INSTRUMENTATION
Sanford Larson

9:43 -10:01 AM THE FAILED BACK: ARE WE DOING TOO MANY LUMBAR PROCEDURES?
Donlin Long

Thursday, October 22, 1992

10:01-10:19AM WHAT TO DO WHEN NOTHING WORKS
Burton M. Onofrio

10:20 AM COFFEE BREAK

MODERATOR: BURTON M. ONOFRIO

10:40 AM SURGICAL EXPERIENCE IN THE MANAGEMENT OF HIGH CERVICAL INTRAMEDULLARY SPINAL CORD TUMORS
Bennett M. Stein

11:00 AM NEUROLOGICAL OUTCOME OF ZINC SUPPLEMENTED HEAD INJURY PATIENTS
Byron Young

11:20 AM PALLIDOTOMY FOR PARKINSON'S DISEASE
Lauri Laitinen

11:40 AM PARTIAL SENSORY RHIZOTOMY FOR TRIGEMINAL NEURALGIA
Jacob Young

12:00 PM LESSONS IN HUMAN PAIN PERCEPTION FROM THALAMIC EXPLORATION FOR DBS IN STROKE-INDUCED PAIN
Ronald R. Tasker

12:20 PM PRESIDENTIAL ADDRESS
HENRY D. GARRETSON
INTRODUCTION: BURTON M. ONOFRIO

Friday, October 23, 1992

MODERATOR: PETER BLACK

8:00 AM - 1:00 PM SYMPOSIUM:
NEUROSURGERY AND THE NEW BIOLOGY

8:00 - 8:15 AM INTRODUCTORY REMARKS
PETER BLACK

8:15 - 8:45 AM MOLECULAR BIOLOGICAL MECHANICS IN NEURAL REGENERATION
Larry Benowitz

8:45 - 9:00 AM THE EFFECT OF CILIARY NEUTROTROPHIC FACTOR (CNTF) ON NEURITE OUTGROWTH FROM SPINAL CORD NEURONS: IMPLICATIONS FOR AXONAL REGENERATION
Nelson M. Oyesiku *ACADEMY AWARD - HONORABLE MENTION*

9:00 - 9:25 AM CONCEPTS OF MOLECULAR GENETICS AND BIOLOGICAL FACTORS IN THE COMPREHENSION AND TREATMENT OF C.N.S. NEOPLASIA
Robert L. Martuza

9:25 - 9:40 AM CHROMOSOME LOSS DURING HUMAN ASTROCYTOMA PROGRESSION
Dan Fults

9:40 - 9:55 AM EXPANDED SPECTRUM OF VIRAL THERAPY IN TREATMENT OF NERVOUS SYSTEM TUMORS
James Markert *ACADEMY AWARD-HONORABLE MENTION*

Friday, October 23, 1992

9:55 -10:10 AM DEVELOPMENT OF MONOCLONAL ANTIBODIES FOR GLIOMA THERAPY
David Eng *ACADEMY AWARD - RUNNER UP*

10:10 AM COFFEE BREAK

10:30-10:55 AM MOLECULAR ANALYSIS AND PRINCIPLES OF NEURAL PROTECTION
Julian T. Hoff

10:55-11:20 AM MOLECULAR BIOLOGY OF THE CEREBROVASCULAR WALL
Ralph G. Dacey

11:20-11:35 AM NITRIC OXIDE MEDIATES CHEMOREGULATION AND AUTOREGULATION OF CEREBRAL BLOOD FLOW IN PRIMATES
B.Gregory Thompson *ACADEMY AWARD - HONORABLE MENTION*

11:35-12:10 PM SYNAPTIC PLASTICITY: PHOTOBLEACHING RECOVERY IN LIVING ANIMALS DEMONSTRATES POSTSYNAPTIC RECEPTOR MOVEMENT
Adam P. Brown *ACADEMY AWARD - WINNER*

Friday, October 23, 1992

12:10-12:30 PM FUTURE PROSPECTUS OF INFLUENCE OF MOLECULAR BIOLOGY ON NEUROSURGICAL PRACTICE
Charles J. Hodge, Jr.

12:30- 1:00 PM PANEL
Larry Benowitz
Robert Martuza
Julian T. Hoff
Ralph G. Dacey
Charles J. Hodge

Saturday, October 24, 1992

MODERATOR: HENRY D. GARRETSON

8:00 AM FACTORS THAT PREDICT SUCCESS OR COMPLICATIONS AFTER STEREOTACTIC RADIOSURGERY FOR BRAIN ARTERIOVENOUS MALFORMATIONS
L. Dade Lunsford

8:20 AM PETROSAL DURAL AVM's
Roberto C. Heros

8:40 AM THE SCOPE AND LIMITATIONS OF SURGERY THROUGH THE MOUTH
H. Alan Crockard

9:00 AM MECHANISM OF CEREBRAL VASCULAR 'STEAL' PHENOMENON DURING HYPERCARBIA
C.M. Loftus

Saturday, October 24, 1992

9:20	AM	HYPOTHERMIA: MECHANISM OF ITS PROTECTION IN CNS ISCHEMIA Joung Lee
9:40	AM	VESTIBULAR NEUROTOMY FOR MENIERE'S DISEASE: OUTCOME PREDICTORS Richard Foltz
10:00	AM	SPONTANEOUS TEMPORAL ENCEPHALOCELE: A REVIEW OF FIVE TYPES Robert Wilkins
10:20	AM	COFFEE BREAK
		MODERATOR: GEORGE A. OJEMANN
10:40	AM	NORMAL CONUS POSITION TETHERED SPINAL CORD J.A. Winfield
11:00	AM	CYTOKINE PRODUCTION IN THE PITUITARY GLAND Stanley Martin
11:20	AM	IMPROVEMENT IN PITUITARY FUNCTION AFTER TRANS-SPHENOIDAL REMOVAL OF LARGE ADENOMAS Warren Selman

Saturday, October 24, 1992

11:40 AM TRANSIENT HYPONATREMIA AFTER PITUITARY SURGERY: POSSIBLE SECOND PHASE
Paul Nelson

12:00-1:00 PM SYMPOSIUM: NEUROSURGICAL ASPECTS OF CORTICAL LANGUAGE LOCALIZATION

12:00-12:15 PM CORTICAL LANGUAGE LOCALIZATION BY STIMULATION MAPPING, OPTICAL IMAGING AND NEURONAL RECORDING
George A. Ojemann

12:15-12:30 PM CORTICAL LANGUAGE LOCALIZATION IN GLIOMA RESECTIONS
Mitchel Berger

12:30-12:45 PM CORTICAL LANGUAGE LOCALIZATION IN AVM RESECTIONS
Henry D. Garretson

12:45- 1:00 PM DISCUSSION

PROGRAM OBJECTIVES FOR THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

1. Discuss the indications for surgical therapy, particularly instrumentation in degenerative lumbar spine disease.

2. Describe findings from molecular biology that are germane to aspects of neurosurgery including neurogeneration, growth of brain tumors, neuroprotection and structure of cerebral blood vessels.

3. Indicate the value of cerebral cortical language localization techniques in neurosurgical management, particularly of gliomas and arteriovenous malformations.

Thursday, October 22, 1992
8:10 AM

New Insights Into the Vasculature of the Normal and Traumatized Spinal Cord

Charles H.Tator, Izumi Koyanagi,David Anthes,Elizabeth Theriault
Toronto Canada

Spinal cord trauma profoundly effects the vasculature of the spinal cord. Intramedullary hemorrhage and ischemia are major components, and have been implicated in the etiology of the secondary effects of trauma. Our group has used two new methods as well as transmission electron microscopy for examining the vasculature of the normal and traumatized rat spinal cord. The first method is silicone angiography produced by injecting colored silicone rubber into the vasculature which is then viewed three-dimensionally in tissues cleared by methylsalicylate. The second method is corrosion cast angiography produced by injecting a polyester resin into the vasculature followed by corrosion of the tissues with sodium hypochlorite and viewing by scanning electron microscopy.

In the normal rat the anterior sulcal arteries supply the anterior two-thirds of the spinal cord including both white and gray matter. Contrary to the classical view, there is no so-called "pial plexus" for supplying the peripheral white matter. The venous drainage of the dorsal cord was partly through longitudinal veins coursing at the grey-white junction at the dorsal median septum. In addition to major loss of the capillary microcirculation, trauma caused marked changes in the sulcal arterial system and longitudinal veins. The sulcal system was the origin of some of the post-traumatic hemorrhages, but more importantly showed progressive obliteration and narrowing, and played a major role in producing post-traumatic ischemia. The longitudinal veins were the origin of many of the hemorrhages at the site of injury, and for the majority of the remote hemorrhages rostrally and caudally. The microvasculature showed evidence of stasis, and occlusion.

These studies revealed several new anatomical features of the arterial supply and venous drainage of the normal rat spinal cord, and showed the anatomical substrate for previously recognized post-traumatic vascular changes, especially hemorrhage and ischemia.

8:30 AM
Experimental Compressive Cervical Myelopathy
Ossama Al-Mefty Maywood IL

A wealth of clinical and laboratory studies have led to some understanding of the pathophysiology of cervical spondylotic myelopathy but they have also raised a host of questions. No satisfactory animal model of chronic spinal cord compression without intentional vascular compromise outside of the spinal canal has been previously devised which can adequately address these questions. The authors present a dog model which simulates chronic spinal cord injury seen in cervical spondylosis and results in a delayed progressive myelopathy. This model allows for controlled compression, ongoing neurological assessment, diagnostic imaging, frequent electrophysiologic testing, local blood flow measurement, and post mortem histologic examination.

Fourteen dogs were treated with subclinical cervical cord compression utilizing a posterior teflon washer and anterior teflon screw to produce a 35% canal stenosis while four dogs underwent sham operations. The animals were followed for 18 months and then sacrificed. Evidence of myelopathy was produced in 12 dogs and tended to appear, on average, 7 months after initial compression. Clinical, radiologic, and histopathologic correlates will be presented.

Thursday, October 22, 1992

Spinal cord blood flow studies using the hydrogen clearance method showed significant transient increase in spinal blood flow immediately after compression. Somatosensory evoked potential studies paralleled clinical deterioration. MRI studies revealed intramedullary cavitation depicted in 6 dogs. Histological studies showed cavitation of cord at compression level, loss of anterior horn cells and vessel enlargement at and immediately adjacent to the compressed area, and lack of significant glial infiltration or scarring. Staining for degenerated fibers revealed sparse amounts of axonal debris in animals with long term compression.

8:50-10:20 AM	**SYMPOSIUM: DEGENERATIVE LUMBAR DISEASE**
8:50 - 9:08 AM	**LUMBAR DEGENERATIVE DISEASE: PATHOLOGY IMAGING** Frederick Simeone Philadelphia PA
9:08 - 9:26 AM	**LUMBAR STENOSIS AND AVOIDANCE OF HEAVY METAL POISONING** John Jane Charlottesville VA
9:26 - 9:43 AM	**INDICATIONS AND TECHNIQUES FOR LUMBAR INSTRUMENTATION** Sanford Larson Milwaukee WI
9:43 -10:01 AM	**THE FAILED BACK: ARE WE DOING TOO MANY LUMBAR PROCEDURES?** Donlin Long Baltimore MD
10:01-10:19 AM	**WHAT TO DO WHEN NOTHING WORKS** Burton M. Onofrio Rochester MN
10:20 AM	**COFFEE BREAK**

Thursday, October 22,1992
10:40 AM
Surgical Experience in the Management of High Cervical Intramedullary Spinal Cord Tumors
Bennett M. Stein, Paul C. McCormick New York NY

Ten patients with mixed pathology (mostly ependymoma) tumors located in the high cervical and cervical medullary junction are used for an evaluation of the MRI features and the surgical techniques as well as results.

These tumors represent special cases since they involve respiratory centers and obviously all of the spinal cord function below this level. Many of the tumors were extremely large when evaluated and operated.

Special considerations were given to respiratory control both during the operation and afterwards and evoked potential studies were used but not found to be overly useful in many of the cases.

The surgical results appear to justify an aggressive approach to these tumors even though large and located in treacherous area of the nervous system.

11:00 AM
Neurological Outcome of Zinc Supplemented Head Injury Patients
Byron Young Lexington KY

Patients with severe head injury have hypozincemia and increased urinary zinc loss. We have suggested these patients have an increased requirement for zinc. Zinc may be important in neural reorganization and may decrease secondary neural damage. Zinc supplementation or standard therapy (placebo) was given from hospitalization until 3 months post injury to 68 patients with severe head injury in a prospective randomized controlled trial. The groups were not significantly different in admission Glasgow

Coma Scale Score (GCS), the Dementia Rating Scale (DRS), and a neurological battery. At 15 days post injury, the zinc patients (Z) had improved GCS scores compared to placebo control patients (P) ($F(1,27) = 3.9$; $p < 0.001$). At 3 months post-hospitalization, those patients who were testable, yet too impaired to complete a full neuropsychological battery, were administered the DRS. (Z) patients ($n = 6$) were compared to (P) controls ($n = 4$) on the DRS. The (Z) patients exhibited higher scores on the DRS (mean Attention score: (Z) = 34.5±3.02 (P) = 26.75±9.03, $p=0.04$; mean Concept score: (Z) = 30.83±2.48 (P) = 22.25±.18, $p=0.03$) even though patients were comparable at study entry (GCS: (Z) = 7.170.90; (P) = 6.75±0.43). The GOS scores tended to be more favorable (Good Recovery, Moderate Disability) in the (Z) group at 3 months and 1 year post injury. (3 months (Z) = 51% (P) = 35%; 1 year (Z) = 70% (P) = 51%). At one year post injury mortality rate was 15% in the (Z) group and 26% in the (P) group. No significant differences were observed at 1 year between groups of patients given the neuropsychological battery at 1 year post injury and twelve percent were lost to follow-up. We conclude that zinc supplementation appears to improve short term neurological outcome. Further studies are required to assess its possible beneficial effects on mortality and cost of hospital stay/rehabilitation.

11:20 AM

Pallidotomy for Parkinson's Disease

Lauri V. Laitinen, Stockholm, Sweden

Laboratory evidence in the early 1980's suggested that dopamine-deficiency in the putamen leads to an increased inhibitory action on the medial pallidum where a release of all parkinsonian symptoms were thought to take place. Therefore, it was tempting to interrupt some striopallidal pathways in the ventroposterolateral (VPL) pallidum between the putamen and the medial pallidum.

Patients and Methods: From 1985 to June 1992, I performed VPL pallidotomies on 117 parkinsonian patients who suffered from drug-resistant bradykinesia, rigidity, tremor, and L-dopa induced dyskinesias. The pallidotomy was unilateral in 110 and bilateral in 7 patients. Several patients had previously had thalamotomy on one side and pallidotomy was done on the other side.

Results: All patients tolerated the surgery well and left the hospital within 1-2 days postoperatively. The optimal pallidal target lay 2 mm anterior to the midcommissural point, 6 mm below the intercommissural line, and 21 (female) to 22 mm (male) lateral to the midline. At the clinical follow-up study 1-88 months later, a good or fair result was recorded in 95% of the patients. Pallidotomy had a good effect on all parkinsonian symptoms and on the L-dopa induced dyskinesias. The gait, the balance and the speech volume also showed good improvement. The effect seemed to be long lasting. Pallidotomy increased the psychomotor speed and precision. Stroop's Color Word Test also indicated that some cognitive functions had improved Complications were observed in 12 patients (10%). Ten had a permanent partial homonymous scotoma in the central lower field. One of them also had a transient dysphasia and facial weakness. One patient had a minor stroke with transitory hemiparesis one week after surgery.

Conclusions: This study shows that all parkinsonian symptoms (tremor, rigor, bradykinesia and L-dopa induced chorcoathetosis) can be effectively abolished by VPL pallidotomy. Left thalamotomy may still have place in the treatment of resting tremor without marked hypokinesia. A combination of thalamotomy on one side and pallidotomy on the other side may be a good solution for some patients.

11 40 AM
Thursday, October 22, 1992

Partial Sensory Rhizotomy For Trigeminal Neuralgia

Jacob N. Young, Robert H. Wilkins Durham, NC

In the treatment of trigeminal neuralgia, our first choice of open procedures is microvascular decompression (MVD). However, in approximately 25% of cases, MVD cannot be done, either because no vascular compression is found at surgery, or because a patient's vascular anatomy prohibits the safe performance of MVD. Partial sensory rhizotomy (PSR) has been our alternative in these instances. The outcome after PSR was reviewed retrospectively in 83 patients with an average follow-up of 72 months. Outcome was considered excellent if there was no pain recurrence, transient or otherwise. Outcome was considered good if pain recurred, but was less severe than the preoperative pain, either with or without medications. A poor outcome was defined as pain recurrence equal in severity to the preoperative pain and refractory to medical therapy, or any recurrence severe enough to require subsequent surgery. Forty (48%) patients had excellent outcomes with no pain recurrence. The outcome was good in 17 (21%) and poor in 26 (31%) patients. Follow-up durations were similar for the three outcome categories. The first year failure rate was 17% with a yearly failure rate averaging 2.6% thereafter. Using survival analysis, two variables were predictive of a poor outcome: prior surgery and lack of preoperative involvement of the third trigeminal division. Postoperative sensory deficits in the trigeminal distribution were absent or mild in 82% of patients. Twenty-eight (34%) patients had no sensory deficits after PSR, forty (48%) patients had slight decreases in facial sensation and 15 (18%) were densely numb in one or more trigeminal distributions. The rate of major complications was 3% and that of minor complication was 11%. We conclude that PSR is a safe and effective alternative to MVD when neurovascular compression is not identified at operation, or when MVD cannot be performed for technical reasons.

Thursday, October 22,1992

12 00 PM

Lessons In Human Pain Perception From Thalamic Exploration For DBS In Stroke-Induced Pain

Ronald R. Tasker, Jo Dostrovsky, A. Parrent Toronto, Canada

Some patients who have had strokes develop intractable pain on the affected side of the body for which the most appropriate surgical treatment appears to be chronic brain stimulation (DBS). Originally called "thalamic pain", and many workers still consider the pathophysiology thalamic, such pain occurs after large or small lesions located at any level within the central nervous system which usually alter somatosensory (particularly spinothalamic) function. Be that as it may the pathophysiology remains obscure.

In patients with stroke-induced central pain we have used single cell recordings and microstimulation for physiological localization of DBS electrode implant sites. Recording examines the somatosensory pathways from receptor to thalamus, stimulation from thalamus to cortex. We have recognized a variety of abnormalities:· absence of all or part of either limb of the system, reorganization, neurons with a bursting firing pattern, abnormal responses to stimulation and changes in threshold for sensory stimulation. Yet the clinical picture is similar in all

Case histories will be presented to illustrate these points and to suggest that in some patients at least contralateral thalamocortical function is not responsible for the pain; rather ipsilateral thalamocortical function may be responsible.

12:20 PM PRESIDENTIAL ADDRESS
HENRY D. GARRETSON
Introduction: Burton M. Onofrio

Friday, October 23, 1992

MODERATOR: PETER BLACK

8:15 AM - 1:00 PM SYMPOSIUM:

NEUROSURGERY AND THE NEW BIOLOGY

8:00 - 8:15 AM INTRODUCTORY REMARKS
Peter Black Boston MA

8:15 - 8:45 AM MOLECULAR BIOLOGICAL MECHANICS IN NEURAL REGENERATION
Larry Benowitz

8:45 - 9:00 AM THE EFFECT OF CILIARY NEUTROTROPHIC FACTOR (CNTF) ON NEURITE OUTGROWTH FROM SPINAL CORD NEURONS: IMPLICATIONS FOR AXONAL REGENERATION
Nelson M. Oyesiku *Academy Award - Honorable Mention*

9:00 - 9:25 AM CONCEPTS OF MOLECULAR GENETICS AND BIOLOGICAL FACTORS IN THE COMPREHENSION AND TREATMENT OF C.N.S. NEOPLASIA
Robert Martuza
Washington DC

9:25 - 9:40 AM CHROMOSOME LOSS DURING HUMAN ASTROCYTOMA PROGRESSION
Daniel Fults
Salt Lake City, UT

Friday, October 23, 1992

9:40 - 9:55 AM	EXPANDED SPECTRUM OF VIRAL THERAPY IN THE TREATMENT OF NERVOUS SYSTEM TUMORS James Markert Academy Award - Honorable Mention
9:55 -10:10 AM	DEVELOPMENT OF MONOCLONAL ANTIBODIES FOR GLIOMA THERAPY David Eng **Academy Award Runner Up**
10:10-10:30 AM	COFFEE BREAK
10:30-10:55 AM	MOLECULAR ANALYSIS AND PRINCIPLES OF NEURAL PROTECTION Julian T. Hoff Ann Arbor MI
10:55-11:20 AM	MOLECULAR BIOLOGY OF THE CEREBROVASCULAR WALL Ralph Dacey St. Louis MO
11:20-11:35 AM	NITRIC OXIDE MEDIATES CHEMOREGULATION AND AUTOREGULATION OF CEREBRAL BLOOD FLOW IN PRIMATES B. Gregory Thompson - Academy Award - Honorable Mention

Friday,October 23, 1992

11:35-12:10 PM — **SYNAPTIC PLASTICITY: PHOTOBLEACHING RECOVERY IN LIVING ANIMALS DEMONSTRATES POSTSYNAPTIC RECEPTOR MOVEMENT**
Adam P. Brown
ACADEMY AWARD-WINNER

12:10-12:30 PM — **FUTURE PROSPECTUS OF INFLUENCE OF MOLECULAR BIOLOGY ON NEUROSURGICAL PRACTICE**
Charles J. Hodge, Jr.
Syracuse NY

12:30- 1:00 PM — **PANEL:**
Larry Benowitz
Robert Martuza
Julian T. Hoff
Ralph G. Dacey
Charles J. Hodge,Jr.

Saturday, October 24, 1992

MODERATOR HENRY D. GARRETSON

8:00 AM
Factors That Predict Success or Complications After Stereotactic Radiosurgery for Brain Arteriovenous Malformations
L.Dade Lunsford Pittsburgh PA

In order to assess factors that predict successful obliteration or complications and to improve selection of patients with arteriovenous malformations (AVM) for stereotactic radiosurgery, we reviewed our five year experience.

Methods and Materials: During a five year interval, 348 patients (50% female, 50% male) with angiographically identified AVMs underwent stereotactic radiosurgery using a 201 source Cobalt-60 gamma knife. Prior cerebral hemorrhage occurred in 59% of patients, 47% had headaches and 32% had a seizure disorder. At least one attempt at surgical removal had been performed in 47 patients (13.5%). Intravascular embolization was performed in 56 patients (16%) in order to reduce the size of the AVM to one suitable for radiosurgery. AVM volumes ranged from .06 to 46 cm^3.

Results: Radiosurgical obliteration rates (n=111) were dependent upon the initial AVM volume: $\leq 1 cm^3$.. 88% total obliteration; $<4 cm.^3$ 81% complete obliteration. For AVM volumes above 7 cm^3 five of 16 had complete obliteration. Failure to obtain complete obliteration was related to increasing initial AVM volume, time of angiography, and recognition of an additional AVM nidus not identified at the time of initial stereotactic radiosurgery. No statistically significant conclusion regarding efficacy of a specific dose was possible. Magnetic resonance imaging had a 50% predictive accuracy for total obliteration subsequently confirmed by angiography. Seizures improved in more than 50% of patients and headaches improved in more than 70%. Re-hemorrhage after radiosurgery was observed in 18 patients compared with 16 patients projected by a statistical model to re-hemorrhage during the latency interval after undergoing stereotactic radiosurgery ($p=.65$).

Discussion: Stereotactic radiosurgery using the multi-source Cobalt-60 gamma unit was a valuable treatment option for carefully selected AVM patients. Larger volume AVMs responded completely in fewer patients and required multi-modality approaches, including intravascular embolization and staged radiosurgical procedures performed at different intervals (dose staging) or for different regions of the AVM (volume staging). Conservative management of most patients with brain AVMs can no longer be recommended.

Saturday, October 24, 1992

8:20 AM

Petrosal Dural AVMs

Roberto C. Heros, Daniel Ruefenacht Minneapolis MI

Dural AVMs most commonly develop in the region of the cavernous and transverse-sigmoid sinuses. In our experience the third most common site is the region of the tentorial incisura. The latter lesions have several peculiarities that make them unique enough to be considered as a separate type of dural AVM; herein referred to as "petrosal dural AVM". They are fed primarily by tentorial branches of the internal carotid artery; it is difficult and risky to embolize these vessels. They drain primarily into the petrosal sinus and the petrosal vein. At the time of clinical presentation, the petrosal sinus is most frequently occluded so that the drainage is primarily in a retrograde fashion into the petrosal vein and the pial venous system which then becomes diffusely arterialized, particularly throughout the posterior fossa. Because of the latter, patients present primarily with hemorrhage, or with signs of increased intracranial pressure and cerebellar or brain stem dysfunction.

The treatment of these petrosal dural AVMs is also different. Mullan has emphasized the safety and effectiveness of treating dural fistulas by simple obliteration of the venous drainage. This is particularly applicable to petrosal AVMs because at the time of presentation they almost always drain through a single dilated vein, the petrosal vein. Therefore the treatment of these AVMs is primarily surgical rather than endovascular. We approach them subtemporally, transtentorially with obliteration of the obvious arterial feeders in the tentorium to prevent recurrence and then obliteration and transection of the draining vein.

In our series of five patients, three presented with hemorrhage and two with signs of cerebellar and brain stem dysfunction. All were treated successfully by surgical obliteration of draining vein, preceded by embolization in two cases.

Saturday, October 24, 1992
8 40 AM

The Scope and Limitations of Surgery Through the Mouth

H. Alan Crockard FRCS London, England

Transoral surgery is not new. Various attempts to reach the skull base and craniovertebral junction have been described since the turn of the century, but, until the widespread availability of non-invasive imaging techniques, the extent and variety of pathology in the area, was not appreciated. A further problem, now resolved, has been the availability of dedicated instrumentation to allow exposure and surgical manipulation at depths of up to 20 cms from the incisors.

Over the last decade, our group has evaluated a large number of craniovertebral and clival pathology and performed a variety of surgical procedures through the mouth on almost 300 patients. All patients have been followed up on a long-term basis and, based on the outcome, disability and complications, we have modified and re-assessed our indications for various procedures.

In general terms, if there is an anterior midline compressive lesion, particularly if it is extradural, of the clivus, craniovertebral junction and the first two cervical vertebra, then transoral surgery is particularly useful; lateral extensions may be inaccessible For lesions below the foramen magnum, elevation of the palate will allow exposure; division of the soft palate will expose the foramen magnum. In extreme basilar invagination, or extensive midline tumors involving the whole of the clivus, a Le Fort I osteotomy in combination with a midline palatal split (the extended maxillotomy or open door maxillotomy), will provide good exposure.

Our main pathological material has been severe basilar invagination and translocation associated with end-stage rheumatoid arthritis, congenital malformations and extradural

tumors. A small number of intradural tumors and mid-basilar aneurysms have also been tackled.

The major drawback to the procedure is the risk of CSF leak and meningitis. These risks are minimized with lumbar drainage for five days immediately post-operatively. Thrombin fibrin glue and a multi-layer closure at the site of surgery and, if the defect is large, a vascularized muscle flap, such as sternomastoid is used.

9:00 AM

Mechanism of Cerebral Vascular "Steal" Phenomenon During Hypercarbia

Christopher M. Loftus Iowa City IA

After occlusion of an artery to the brain, hypercarbia and seizures may produce a paradoxical reduction or "steal" in cerebral blood flow to the ischemic area. We measured pressure in an occluded branch of the middle cerebral artery and used a new method to measure regional cerebral blood flow (rCBF) to collateral-dependent cerebrum in dogs (n=25), to examine hemodynamic mechanisms that account for the steal. During hypercarbia ($P_aCO_2 = 70 \pm 1$), rCBF to collateral-dependent cerebrum, measured with microspheres and identified using the "shadow flow" technique, decreased from 95±6 ml/100 g/min (mean ± SE) to 71± 9 ($p<0.05$), while flow to normal brain increased from 105±9 to 281±15 ($p<0.05$). Pressure in a branch of the middle cerebral artery decreased during hypercarbia from 50±6 mmHg to 25±3 ($p<0.05$), concurrent with a significant increase in resistance of collateral vessels. Small vessel resistance was the same in collateral-dependent cerebrum from 128±16 to 67±11 ($p<0.05$), and flow to normal brain increased from 169±14 to 418±17 ($p<0.05$). Small vessel resistance decreased in both regions, but the decrease was much greater in normal cerebrum.

Changes in cerebral artery pressure and resistance of collateral vessels during seizure were similar to those during hypercarbia. Thus, in collateral-dependent cerebrum, the steal phenomenon during hypercarbia and seizures is the result of a large decrease in perfusion pressure, a greater decrease in resistance in normal cerebrum than in collateral-dependent cerebrum, and an increase in resistance of collateral vessels.

9:20 AM

Hypothermia: Mechanism of its Protection in CNS Ischemia

Joung H. Lee, John Jane Charlottesville VA

Treatment and protection of the CNS cells following ischemia remains as a major challenge today. Hypothermia's protective role in ischemia has been well demonstrated. However, its specific mechanism of neuronal protection is not clearly elucidated to date. In the past, metabolic inhibition was proposed as a primary mechanism of its protection. However, barbiturates, which reduce the CNS metabolism to approximately 40% of the basal level by completely inhibiting synaptic activity, were found to not have the same capability of neuronal protection as hypothermia. This implies that metabolic inhibition alone is not the critical mechanism which accounts for hypothermia's protective effects in ischemia.

In this study, bovine chromaffin cells were used as a model for neuronal cells. The whole-cell recording configuration of the patch-clamp technique was utilized to study the effects of varying degrees of hypothermia on Na, CA and K currents of the neuronal membrane. The results are as follows:

T(°C)	I_{Na}(pA)	I_{Ca}(pA)	I_K(pA)
37-38	1428	629	4361
31-32	904	406	4127
25-26	526	359	3406
19-20	561	125	2375

(n=15-18 recordings in each group)

This study clearly shows that hypothermia nonspecifically reduces transmembrane ionic currents of Na, K and Ca. Although effects on K currents were less dramatic with only 5% reduction, mild hypothermia (31-32°C) caused 37% reduction in Na currents and 35% reduction in Ca currents. This study provides direct electrophysiological evidence for membrane-stabilizing effects of hypothermia. By reducing transmembrane ionic fluxes, hypothermia may 1) reduce cellular energy requirement, 2) reduce Na-mediated cellular swelling, 3) decrease cytotoxic intracellular Ca load, 4) lower membrane excitability, 5) decrease synaptic activity, and 6) reduce cellular secretions, including release of excitotoxic neurotransmitters.

9:40 AM

Vestibular Neurotomy for Meniere's Disease: Outcome Predictor

Richard M. Foltz, J.T. McElveen, Robert Wilkins Durham NC

We treated 50 patients with Meniere's disease by vestibular neurotomy between May 1983 and July 1992. To evaluate the postoperative status, each patient was sent a questionnaire. As a measure of surgical outcome, the questionnaire focused on 4 areas:

1.) The frequency of spells per month
2.) The influence of the operation on vertigo
3.) The patient's disability status (work capacity)
4.) The overall patient satisfaction

There were 43 respondents to the questionnaires

The operative group of 50 patients was composed of 22 males and 28 females. For the 43 respondents, the length of follow-up ranged from 12 to 110 months (median = 31 months). Initial symptoms occurred predominantly in the 4th and 5th decades of life. The duration of symptoms ranged from 7 to 360 months (median = 33 months) before the vestibular neurotomy

Postoperatively the frequency of spells per month declined significantly in both the males and females: 25 of 43 respondents reported no postoperative vertigo (V_0= 58%) and an additional 13 patients reported improvement (V_1=88%). Patients ranked their pre- and postoperative disability as none, mild, moderate, or severe: 6 patients ranked their preoperative disability as none, 10 as mild, 9 as moderate, and 18 as severe. Postoperative disability improved in 28 patients (65%), remained the same in 9 (21%), worsened in 6 (14%). However, when patients rated their overall result, 81% reported that they were better (35/43), 14% the same (6/43), and 5% worse (2/43). When asked whether the operation was worthwhile, 88% (38/43) responded yes while 12% (95/43) reported no.

Preoperative factors associated with dissatisfaction and a greater postoperative disability were: 1) no preoperative disability (the ability to work was not affected by symptoms), and 2) bilateral symptoms.

Postoperative factors associated with dissatisfaction were: 1) increased imbalance, and 2) progression of disease (especially progressive hearing loss).

10:00 Am

Spontaneous Temporal Encephalocele: A Review of Five Types

Robert H. Wilkins Durham NC

Five main types of spontaneous temporal encephalocele have been described. Each type has typical clinical features which are different from those of the other types. The lateral temporal encephalocele extends through a defect at the pterion or occasionally at the asterion and is apparent in infancy. The anterior temporal encephalocele extends forward into the

ipsilateral orbit and typically produces ipsilateral proptosis. The anteromedial temporal encephalocele extends into the sphenoid sinus and typically presents with CSF rhinorrhea. The posteroinferior temporal encephalocele ordinarily projects through the tegmen tympani into the tympanic antrum or epitympanic recess, causing CSF otorrhea or otorhinorrhea. And the anteroinferior temporal encephalocele, which projects through the floor of the middle fossa into the infratemporal area, is usually associated with medically intractable complex or simple partial seizures. Each of the five types of spontaneous temporal encephalocele can be diagnosed with the aid of modern radiological techniques, and each is usually best managed surgically.

10:40 AM

Normal Conus Position Tethered Spinal Cord

J. A. Winfield, L. Hochhauser, C.J.Hodge,Jr. Syracuse NY

The classical diagnosis of tethered spinal cord (TSC) hinges on 2 radiological features: a low (below L2; Barson 1970) position of the conus medullaris, and an abnormally thick filum terminale (>2mm; Fitz 1976). TSC with a normal conus position (NCP) has infrequently been reported in the Pediatric neurosurgical literature and not well described in adults. We have untethered 4 adults and 1 juvenile patient, all without cutaneous stigmata of spinal dysraphism, who were felt on clinical history to have symptoms best explained by TSC. NCP was observed in these cases, in absence of other spinal pathology such as osteoarthritis or HNP which could account for the clinical presentation.

Case 1: An 80 yr old developed a sudden painless foot drop while moving a large clay pot in his garden. Four days later he was untethered, after which his foot drop resolved within 48 hours.

Case 2: A 41 yr old developed persistent incontinence following a routine D&C procedure. One year later she underwent untethering and regained normal bladder function by hospital discharge.

Case 3: A 38 yr old with back and diffuse bilateral leg pain for 36 months, developed progressive dual sphincter dysfunction and was untethered. Her pain resolved, and sphincter function returned.
Case 4: A 21 yr old male with a left concave scoliosis complained of back pain and an entire left numb leg. He underwent untethering and fusion of a Grade 1 L_5S_1 spondylolisthesis with resolution of his pain and return of sensation in his left leg.
Case 5: A 13 yr old with, severe back and bilateral leg pain, underwent untethering after 3 years of failed conservative physical therapy. His pain and urgency resolved and he has returned to normal activity.

This group of patient's histories and diagnostic studies will be presented to illustrate the radiologic features of the terminal filum and cauda equina in NCP-TSC, and to draw attention to this clinical entity in adults. *A thickened filum, greater in thickness than any adjacent cauda equina nerve root, the presence of fat in the filum, and/or persistent dorsal position of the cord and filum with the double "V" pattern of the cauda equina, are all indicators of the presence of Normal Conus Position Tethered Spinal Cord.* Both pain and dysfunctional sphincters, can resolve in this clinical entity following untethering in adults.

11:00 AM
Cytokine Production in the Pituitary Gland
Stanley B. Martin, Anthony Cerami, Richard Bucala
New York, NY

Recent studies have emphasized the critical role played by cytokines in the systemic response to inflammation. During the course of infection, cells of the immune system produce cytokines such as tumor necrosis factor and interleukin-1. These cytokines have broad biological activities and modulate neuroendocrine activity at the levels of the hypothalamus and pituitary. Because the hypothalamic/pituitary axis plays such a crucial role in maintaining global homeostasis, we hypothesized that the pituitary gland itself might secrete cytokines.

We studied the secretory response of pituitary cells to stimulation by bacterial lipopolysaccharide (LPS). When cultured with LPS for 48 hours, the murine pituitary cell line AtT-20 elaborates a specific 12 kilodalton protein, as detected by gel electrophoresis and silver staining. Under serum-free conditions, peak induction occurs after culture with 50 micrograms/ml of LPS, and is not observed after stimulation with tumor necrosis factor, interleukin-1, interleukin-6, or phorbol myristate acetate. Detailed protein sequence analysis reveals that this 12 kD protein is identical to migration inhibitory factor(MIF).

MIF is a cytokine produced by activated lymphocytes which inhibits macrophage migration and enhances their ability to kill microorganisms. Its expression at a variety of sites of inflammation has suggested a role for MIF in the local regulation of the immune system. Our results indicate that the pituitary gland, in addition to its known indirect effects via ACTH, may directly potentiate the systemic response by producing the cytokine MIF.

11.20 AM

Improvement in Pituitary Function After Transsphenoidal Removal of Large Adenomas

Warren R. Selman, Z.T.Madhoun, Baha M. Arafah Cleveland OH

INTRODUCTION: Patients with pituitary macroadenomas often present with hypopituitarism which is believed to be caused by stalk compression, and as such should be reversible.

METHODS: We examined the function of the pituitary-adrenal axis in 13 patients with hypothyroidism during the first week after selective adenomectomy of non-secreting adenomas.

RESULTS: All patients had a glucocorticoid deficiency, hypogonadotrophism, and 10 of 13 had hypothyroidism. Patients were given glucocorticoids before, during, and after surgery.

Thereafter steroids were abruptly stopped and serum cortisol levels were carefully monitored. Patients could be categorized into two groups based on serum cortisol levels as follows:

	POSTOPERATIVE DAY				
GROUP I, n=9	13.3±1.5	14.3±1.5	13.8±1.1	11.0±1.2	13.3±1.5
GROUP II,n=4	2.2±0.3	2.4±0.3	4.1±0.9		

Serum cortisol levels were normal in group I and were similar to those previously reported by us in patients with normal adrenal function who underwent transsphenoidal surgery. All 9 patients were discharged to home on no replacement therapy and were documented by dynamic testing to have a normal pituitary adrenal axis. The 4 patients in group II, in contrast, had low serum cortisol levels, developed symptoms of glucocorticoid deficiency by the 5th day and were discharged on replacement therapy. Only one of these patients had normal adrenal function when examined 3 months later. In retrospect it was apparent that Group I patients had higher responses to Cortrosyn (increment 13.1±1.2 vs 8.7±2.3p<0.02) and higher serum prolactin levels(40.4±8.5 vs 16.3±4.3p<0.05).

CONCLUSIONS: In the majority of patients with hypopituitarism improved pituitary function can be documented immediately after surgical adenomectomy. A preoperative elevation of serum cortisol ≥9 ug/dl following Cortrosyn administration is associated with a high probability (9 of 10) of immediate recovery of adrenal function. These data support the contention that hypopituitarism in patients with macroadenomas is caused by compression of the portal vessels and/or the infundibulum.

Saturday, October 24, 1992

11:40 AM

Transient Hyponatremia After Pituitary Surgery: Possible Second Phase

Paul B. Nelson Pittsburgh PA

Two recent clinical reports, in addition to two cases of our own have identified patients that developed hyponatremia following pituitary surgery. The onset is generally five to ten days following surgery, and lasts three to four days. The hyponatremia in most instances has been associated with a clinical deterioration in the patient. The onset and resolution of the hyponatremia was similar to a second phase of the triphasic response to diabetes insipidus without seeing the first and third stages. To test this hypothesis, 35 adult male Sprague-Dawley rats underwent stereotactic placed radio-frequency lesions targeted to partially destroy the supraoptico-hypophyseal tract. Sixteen control rats underwent skull incision and drilling, but no radio-frequency lesions were placed.

Twenty-two of the lesioned animals went on to develop either diabetes insipidus or hyponatremia without proceeding diabetes insipidus. The serum sodium nadir was 128.7±1 mmol/L compared to a sodium nadir of 140.0±.39 mmol/L for sham operated rats. The sodium reached its nadir one to three days post lesion and returned to normal by day seven. Antidiuretic hormone levels at the nadir of serum sodium were elevated relative to the serum sodium.

The data supports the concept that diabetes insipidus during pituitary surgery is secondary to partial damage to neurohypophyseal tracts. The remaining intact antidiuretic hormone neurons may protect against the diabetes insipidus of the first and third phase. The leak of antidiuretic hormone from the damaged neuron is sufficient to cause an isolated second phase. Isolated second phase should replace the term SIADH.

Understanding the possibility of an isolated second phase helps anticipate potential morbidity in patients undergoing pituitary surgery.

12:00 AM - 1:00 PM **SYMPOSIUM**

NEUROSURGICAL ASPECTS OF CORTICAL LANGUAGE LOCALIZATION

12:00-12:15 PM CORTICAL LANGUAGE LOCALIZATION BY STIMULATION MAPPING, OPTICAL IMAGING AND NEURONAL RECORDING
George A. Ojemann Seattle WA

12:15-12:30 PM CORTICAL LANGUAGE LOCALIZATION IN GLIOMA RESECTIONS
Mitchel Berger Mercer Is WA

12:30-12:45 PM CORTICAL LANGUAGE LOCALIZATION IN AVM RESECTIONS
Henry D Garretson Louisville KY

12:45-1:00 PM DISCUSSION

1:00 PM ADJOURNMENT

NOTES

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DEPARTMENT OF NEUROSURGERY
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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, CA	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
AMELIA ISLAND PLANTATION AMELIA ISLAND,FL	OCTOBER 2-7,1990
SALISHAN LODGE, GLENEDEN BEACH, OREGON	SEPTEMBER 22-26,1991

MEMBERSHIP LIST 1991
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65 Tsumai-Cho, Showa-Ku
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CHARAS SUWANWELA 1972
Chulalongkorn Hospital
Medical School
Bangkok, Thailand

LINDSAY SYMON (Pauline) 1982
The National Hospital
Queen Square
London, WC1N 3BG England

KINTOMO TAKAKURA 1988
University of Tokyo Hospital
7-3-1 Hongo, Bunkyu-ku
Tokyo 113, Japan

KJELD VAERNET (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

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,Delaware (Senior)	9/1988	1938
WINCHELL McK. CRAIG Rochester, Minnesota (Honorary)	2/1960	1942
EDWARD DAVIS Troutdale, Oregon (Senior)	10/1988	1949

R. M. PEARDON DONAGHY Montpelier, Vermont (Senior)	11/26/91	1970
FRANCIS ECHLIN New Paltz, New York (Senior)	4/1988	1944
DEANE H. ECHOLS New Orleans, Louisiana	11/26/91	Founder
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
JOSEPH 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

M. STEPHEN MAHALEY, JR. Maggie Valley, North Carolina (Inactive)	3/18/92	1972
GEORGE L. MALTBY Boston, Massachusetts (Active)	1988	1942
DONALD D. MATSON Boston, Massachusetts (Active)	5/1969	1950
FRANK H. MAYFIELD Cincinnati, Ohio (Senior)	1/2/91	Founder
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, Massachusetts (Honorary)	3/1968	1951
EDMUND J. MORRISSEY San Francisco, California (Senior)	2/1986	1941

GÖSTA NORLÉN Stockholm, Sweden (Honorary)	1/16/92	1973
PIETRO PAOLETTI Pavia ITALY (Corresponding)	11/18/91	1990
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)	4/1978	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
THORALF M. SUNDT Rochester, Minnesota (Active)	9/9/92	1971
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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