THE AMERICAN ACADEMY

OF

NEUROLOGICAL SURGERY

SALISHAN LODGE

GLENEDEN BEACH, OREGON

1991

THE 53RD ANNUAL MEETING OF

THE

AMERICAN ACADEMY OF

NEUROLOGICAL SURGERY

SALISHAN LODGE

GLENEDEN BEACH, OREGON

SEPTEMBER 22 - 26, 1991

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THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

September 22- 26, 1991
Salishan Lodge
Gleneden Beach, Oregon

Sunday, September 22, 1991

1:00 p.m - 5:00 p.m. Registration and Activities Sign-up
Terrace/Long House Conference Center

1:30 p.m. - 4:30 p.m. Executive Committee Meeting
Gallery

6:00 p.m. - 8:30 p.m. Welcome Reception
Council House

Monday, September 23, 1991

7:00 a.m. - 8:00 a.m. Breakfast Business Meeting (members only)
Cedar Tree

8:00 a.m. - 5:00 p.m. Registration
Terrace/Long House Conference Center

8:00 a.m. - 1:00 p.m. Scientific Session
Terrace/Long House Conference Center

1:30 p.m. - 5:30 p.m. North Coast Tour
Bus Departs Main Lobby

6:00 p.m. Beach Party Buffet Dinner
Gleneden Beach State Park
Buses depart from Main Lobby
(casual attire; jackets recommended)

Tuesday, September 24, 1991

7:00 a.m. - 8:00 a.m. Breakfast Business Meeting (members only)
Cedar Tree

Tuesday, September 24, 1991

8:00 a.m. - 5:00 p.m. Registration
Terrace/Long House Conference Center

8:00 a.m. - 1:00 p.m. Scientific Session
Long House Conference Center

9:20 a.m. Presidential Address
Nicholas T. Zervas

1:00 p.m. Golf Tournament
Sign-up Registration

1:15 p.m. Tennis Tournament
Sign-up Registration

5:30 p.m. - 10:00 p.m. Dinner
Salishan Lodge Dining Room
Sign-up for dining times at Registration

Wednesday, September 25, 1991

7:00 a.m.- 8:00 a.m. Breakfast Business Meeting (members only)
Cedar Tree

8:00 a.m. - 5:00 p.m. Registration
Terrace/Long House Conference Center

8:00 a.m. - 1:00 p.m. Scientific Session
Long House Conference Center

1:30 p.m. 6:30 p.m. Tour of Oregon Wine Country
Bus departs from Main Lobby

7:00 p.m. - 8:00 p.m. Reception
Terrace Patio/Long House Conference Center

Wednesday, September 25, 1991

8:00 p.m. Annual Banquet and Dance
Long House Conference Center

Thursday, September 26, 1991

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 1 toward the Continuing Education 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

Sunday, September 22, 1991

6:00 p.m - 8:30 p.m.	WELCOME RECEPTION Council House

Monday, September 23, 1991

8:00 a.m. - 9:30 a.m.	Guest Hospitality Continental Breakfast Dining Room
10:00 a.m. - 11:30 a.m.	Salishan Lodge Chef Demonstration Cedar Tree
1:30 p.m. - 5:30 p.m.	North Oregon Coast Tour Bus Departs from Main Lobby
6:00 p.m.	Beach Party Buffet Dinner Buses depart from Main Lobby (casual attire; jackets recommended)

Tuesday, September 24, 1991

8:00 a.m. - 9:00 a.m.	Guest Hospitality Continental Breakfast Dining Room
10:30 a.m. -12:30 p.m.	Tour to Mossy Creek Pottery and Alder House Glass Blowing Studio Bus departs from Main Lobby
1:00 p.m.	Golf Tournament Sign-up at Registration
1:15 p.m. - 5:00 p.m.	Tennis Tournament Sign-up at Registration
5:30 p.m. - 10:00 p.m.	Dinner Salishan Lodge Dining Room Sign-up for dining times at Registration

Wednesday, September 25, 1991

8:00 a.m. - 9:00 a.m.	Guest Hospitality Continental Breakfast Dining Room
9:30 a.m. - 12:30 p.m.	Shopping Excursion to Quality Factory Village Outlet Shopping Mall Bus departs from Main Lobby
1:30 p.m. - 6:30 p.m.	Tour of Oregon Wine Country Bus departs from Main Lobby
7:00 p.m. - 8:00 p.m.	Reception Terrace Patio/Long House Convention Center
8:00 p.m.	Annual Banquet and Dance Long House Conference Center

Thursday, September 26, 1991

Travel Day

SCIENTIFIC PROGRAM

Monday,September 23, 1991

8:00 AM **Welcome:** Nicholas T. Zervas, President

Scientific Session I: MODERATOR: Nicholas T. Zervas,M.D.

8:00 AM THE PREVENTION OF POSTLAMINECTOMY EPIDURAL FIBROSIS BY A NOVEL GLYCOSAMINOGLYCAN
J.T. ROBERTSON, A.L. MERIC

8:20 AM BRAIN STEM CAVERNOMAS - INDICATION FOR SURGERY; TECHNIQUES, INTRAOPERATIVE MONITORING
R. FAHLBUSCH, C. STRAUSS, J. ROMSTOCK, W. HUK

8:40 AM INTRAOSSSEOUS PRESSURES IN THE LUMBAR SPINE UNDER AXIAL LOADING
S.J. LARSON, N. YOGANANDAN, F.A. PINTAR

9:00 AM EXTENDED SUBTOTAL MAXILLOTOMY FOR SKULL BASE AND CRANIOCERVICAL JUNCTION ABNORMALITIES
J.H. ROBERTSON, E.W. COCKE,JR.

9:20 AM MICROSURGICAL MANAGEMENT OF CRANIOPHARYNGIOMA
E.L. SELJESKOG, D.Y. WEN, S.J. HAINES

ACADEMY AWARD PRESENTATION MODERATOR: GEORGE TINDALL

9:40 AM MOLECULAR STRUCTURE AND FUNCTIONAL TESTING OF HUMAN L1 CELL ADHESION MOLECULE: AN INTERSPECIES COMPARISON
MARY LOUISE HLAVIN

10:00 AM EFFECTS OF EXTERNAL PH ON IONIC CURRENTS IN SMOOTH MUSCLE CELLS FROM THE BASILAR ARTERY OF THE GUINEA PIG
G. ALEXANDER WEST

10:15 AM COFFEE BREAK

Tuesday, September 24, 1991

Scientific Session II: SYMPOSIUM ON BRAIN TUMORS
MODERATOR: ROBERTO C. HEROS

10:40 AM LOW GRADE SUPRATENTORIAL GLIOMAS. CURE OR PALLIATION?
S. MULLAN, D. JOHNSON

11:00 A M IMMUNOTOXIN TREATMENT OF CENTRAL NERVOUS SYSTEM NEOPLASIA
W.A. HALL, O. FODSTAD, A. MYKLEBUST, A. GODAL

11:20 AM AN UPDATE IN THE TREATMENT OF PRIMARY AND METASTATIC MALIGNANT BRAIN TUMORS USING COMBINATION CHEMOTHERAPY WITH OSMOTIC OPENING OF THE BBB
E.A. NEUWELT

11:40 AM INTRACEREBRAL AND SYSTEMIC EFFECTS OF TUMOR NECROSIS FACTOR (TNF) SELECTED BY BRAIN TUMORS
K.J. TRACEY

12:00 PM LONG TERM FOLLOWUP AFTER SURGERY FOR THIRD VENTRICULAR TUMORS
J. AUSMAN, C. COCCIA, H. HAMIYET-CAMUSCU, G. MALIK

12:20 PM ANGIOGENIC MODULATION OF TUMORS IN NEUROFIBROMATOSIS
R.L. MARTUZA, Y. TAKAMIYA, R.M. FRIEDLANDER, H. BREM, A. MALIK

12:40 PM SPECIAL LECTURE:
INTERVENTIONAL NEURORADIOLOGY AND THE NEUROSURGEON
L. NICK HOPKINS

1:00 PM ADJOURNMENT

TUESDAY,SEPTEMBER 24, 1991

SCIENTIFIC SESSION III: SYMPOSIUM ON APPLIED TECHNOLOGY
MODERATOR: MARTIN WEISS

8:00 AM STEREOTACTIC VOP/VIM THALAMOTOMY FOR THE TREATMENT OF TREMOR
P.J. KELLY

8:20 AM NEURAL NETWORKS-A UNIQUE METHOD OF DATA ANALYSIS
A.R. WYLER

8:40 AM STEREOTACTIC TECHNIQUES IN VASCULAR NEUROSURGERY
M.B. SISTI, B.M. STEIN

9:00 AM SPATIAL REGISTRATION OF IMAGE COORDINATE SYSTEMS AND STEREOTACTIC COORDINATE SYSTEMS USING A MACHINE VISION TECHNIQUE
M. HEILBRUN, S. KOEHLER, P. McDONALD, W. PETERS, C. WIKER, V. SIEMENOV

9:20 AM PRESIDENTIAL ADDRESS: NICHOLAS T. ZERVAS
INTRODUCTION: STEWART B. DUNSKER

10:10 AM COFFEE BREAK

SCIENTIFIC SESSION IV: MODERATOR: GEORGE OJEMANN

10:40 AM INTRACRANIAL HYPERTENSION AND GLIAL CELL PRODUCTION OF LEUKOTRIENE C4 INDUCED BY ENDOTOXIN
J.B.G. GHAJAR, R.J. HARIRI, R.A.R. FRASER

11:00 AM MONOCLONAL ANTIBODY TO AMINOMALONIC ACID (AMA) EPITOPE REACTIVE TO ARTERIAL AND GRANULOMA FOAM CELLS: A PROBE FOR THE RADIOIMAGING OF ATHEROMAS
W.M. KIRSCH, K. CHENG, S. FOWLER, T. KOCH, R. WELLERSON, S. NEHLSEN-CANNARELLA, J. VAN BUSKIRK, W. KELLN, S. GRINDE, P. WHELLAN

Tuesday, September 24, 1991

11:20 AM INTERMITTENT OR CONTINUOUS TEMPORARY VESSEL OCCLUSION: THE EFFECT OF PRE-EXISTING HYPERTENSION ON PATHOLOGICAL OUTCOME
W.R. SELMAN, C.C. ROSENSTEIN, R.A. RATCHESON

11:40 AM VENTRICULAR SHUNTS FOR HYDROCEPHALUS: A CLINICAL STUDY OF THE ZERO INTRACRANIAL PRESSURE SHUNT SYSTEM
E.L. FOLTZ, R. MEYER, J. BLANKS

12:00 PM ADENOSINE IN THE REGULATION OF NEONATAL CEREBRAL BLOOD FLOW
T.S. PARK

12:20 PM SPECIAL BASIC SCIENCE LECTURE:
MOTOR SYSTEM CONTROL MECHANISMS
TIMOTHY J.EBNER

1:00 PM ADJOURNMENT

WEDNESDAY,SEPTEMBER 25, 1991

SCIENTIFIC SESSION V: MODERATOR: CHARLES HODGE

8:00 AM THE ANATOMY OF INCOMPLETELY CLIPPED ANTERIOR COMMUNICATING ARTERY ANEURYSMS
W. SHUCART

8:20 AM STUDIES OF L-NNA AND L-ARGININE ON CEREBRAL COLLATERAL BLOOD FLOW AND ENDOTHELIUM-DEPENDENT RELAXATION
M.G. MUHONEN, C.M. LOFTUS

8:40 AM SPONTANEOUS SEIZURES IN BABOONS, (PAPIO CYNOCEPHALUS SP): A NEW PRIMATE MODEL OF EPILEPSY
S. MURK, J.L. STORY, J. LUTHER, K. KAGAN-HALLET, J. ODOM, E. EIDELBERG

9:00 AM THE MIGRATION OF FETAL ASTROCYTES AND ASTROCYTOMA CELLS THROUGH NORMAL BRAIN
E.R. LAWS, J.J. BERNSTEIN, W. GOLDBERG

Wednesday, September 25, 1991

9:20 AM TUMORS OF THE LATERAL WALL OF THE CAVERNOUS SINUS
M. EL-KALLINY, H.R. vanLOVEREN, J.M. TEW, JR., J.T. KELLER

9:40 AM THE EARLY NEUROSURGERY OF AUSTRALIA
N.G.DAN

10:00 AM COFFEE BREAK

SCIENTIFIC SESSION VI: MODERATOR: STEWART B. DUNSKER

10:30 AM SPECIAL LECTURE:

STEREOTACTIC "RADIOSURGERY" AND THE NEUROSURGEON
CHARLES WILSON

10:50 AM SPECIAL SYMPOSIUM:

"CONGENITAL CRANIAL BASE & SPINAL PROBLEMS

MODERATOR: STEWART B. DUNSKER

PANELISTS:

JOHN C. VANGILDER:	CRANIOVERTEBRAL ANOMALIES
JOHN A. JANE:	CRANIOFACIAL ANOMALIES
CHARLES J. HODGE, JR.	SYRINGOMYELIA AND THE CHIARI MALFORMATION; MECHANISMS
ALBERT L. RHOTON,JR.:	SYRINGOMYELIA AND THE CHIARI MALFORMATION: TREATMENT
HAROLD J. HOFFMAN:	TETHERED CORD
PAUL H. CHAPMAN:	SPINAL LIPOMAS

1:00 PM ADJOURNMENT

Monday,September 23, 1991
8:00 a.m.

The Prevention of Postlaminectomy Epidural Fibrosis by a Novel Glyosaminoglycan

James T. Robertson and Albert L. Meric: Memphis, TN

The formation of epidural fibrosis following total laminectomy in New Zealand White Rabbits was significantly decreased by the intraoperative application of GL402, a synthetic glycosaminoglycan (Gliatech, Inc.,Beachwood, OH) with fibroblast inhibiting activity. Laminectomies at L2 and L4 were performed in 24 rabbits. Absorbable gelatin sponge matrix soaked with Gl402 or vehicle was applied in a blinded fashion to the operative sites. Sham operated sites (no treatment) served as controls. Animals were sacrificed after two or four weeks. The extent of epidural fibrosis was evaluated by videotaped microdissection, histological analysis and magnetic resonance imaging. Dense scar formation waS evident at both time intervals in the sham and vehicle-treated sites. The sites with GL402 treatment showed practically no evidence of epidural scar formation or dural adhesions. The healing of skin and lumbodorsal fascia were not affected by treatment of the laminectomy site with GL402. These results suggested that GL402 may prove beneficial in preventing postlaminectomy pain syndromes associated with dural adhesions and epidural fibrosis.

8:20 a.m. Brain Stem Cavernomas - Indication for Surgery; Techniques, Intraoperative Monitoring

R.Fahlbusch, C.Strauss, J.Romstock, W.Huk
Erlangen, Germany

21 patients with brain stem cavernomas were admitted to our department: 11 underwent surgery, 10 are under observation. Diagnosis was performed by MRI with gadolinium, which revealed an additional venous malformation in 3 cases, furthermore by histological examinations.

Surgery: Selective radical removal was performed in the subacute phase of a recurrent bleeding and resulted in an improvement of symptoms in all but one case. This patient developed a complete VI. nerve paresis, following late surgery 3 months after a recurrent bleeding. Mesencephalic cavernomas were operated via a subtemporal or supracerebellar/velum medullare approach, pontine and pontomedullary cavernomas via a suboccipital midline approach. In the two cases with additional venous malformations the cavernoma was removed selectively.

Intraoperative neurophysiological monitoring: Monitoring of AEP and SEP, performed in all cases, covers only 20% of the brain stem pathways, including motor pathways. The surgical risk was minimized and the approach to the cavernoma improved by introducing direct stimulation of the nuclei of cranial nerves VII, XII f.i.

Observation: Since the biology and natural course of brain stem cavernomas is still under discussion, incidental cavernomas and patients, in whom hemorrhages occurred more than 2 months ago, no operation was performed (n = 10). Late surgery increases the risk of additional neurological deficits.

8:40 a.m. Intraosseous Pressures in the Lumbar Spine Under Axial Loading
Sanford J. Larson, Narayan Yoganandan, Frank A Pintar Milwaukee, WI

Recent investigations conducted in our laboratory studying the mechanics of injury of the human lumbar spine have indicted that the initiation of trauma can occur under physiologic, sub-failure loads. Micro-level injury in the form of endplate fractures was demonstrated using cryomicotomy and correlated with the first onset of decrease in the stiffness of the lumbar spinal unit. The present study was undertaken to quantify and correlate the alterations in the intraosseous pressures prior to, during, and after endplate failure under single cycle loading.

Lumbar functional spinal units from fresh human cadavers were prepared by embedding the superior and inferior portions of the vertebral bodies in polymethyl methacrylate. Under fluoroscopy, 1-2 ml of radio-opaque medium was injected into the nucleus of the disc through a 22 gauge needle. A pressure gauge was introduced into the vertebral body to measure intraosseous pressure. The specimens were mounted in a custom design testing apparatus and placed under the fluroscopy unit. Axial compressive displacements were applied quasistatically. The resulting force, compression, and the vertebral pressures were recorded digitally as a function of time using a modular data acquisition system.

The specimens were loaded initially to 50% of the undeformed disc height. Following this, an acute cycle of loading was performed until endplate fracture occurred as demonstrated by passage of the contrast medium from the intervertebral disc into the vertebral body. While the overall stiffness of the structure gradually decreased, the intraosseous pressure suddenly increased indicating an accentuated load in this area. Repeated loading of the specimen simulating normal physiologic conditions also exhibited an increase in the intraosseous pressure compared to the pressures recorded from the intact specimen. These observations suggest that micro-level injury of the lumbar spine, (usually not demonstrable by conventional radiography), can be followed by raised pressure within the vertebral body. Increased pressure within bone has been implicated as a source of pain. Consequently, miniature endplate fractures may be responsible for acute low back pain in patients whose spine films appear normal.

Monday, September 23, 1991

9:00 a.m. Extended Subtotal Maxillotomy for Skull Base and Craniocervical Junction Abnormalities

Jon H.Robertson and Edwin W. Cocke,Jr. Memphis TN

The extended subtotal maxillotomy is accomplished through a degloving approach to the mid face. The maxilla is mobilized below the level of the infraorbital foramen with its blood supply preserved via the mucoperiosteum of the hard palate and contralateral tonsillar pillar. The exposure is accomplished by removal or mobilization of a part of the malar bone, coronoid process of the mandible, superior and middle turbinates, ethmoid and sphenoid sinuses, posterior nasal septum, and pterygoid plates. Extension of a retromolar incision through the anterior tonsillar pillar and lateral pharyngeal wall following mobilization of the maxilla exposes the craniocervical region from the roof of the sphenoid sinus to the fifth cervical vertebra and the skull base between each eustachian tube and carotid canal. The superior exposure allows for transposition of the temporalis muscle, bone, and skin graft to reconstruct the surgical defect if needed. The morbidity of the procedure is less than that associated with the numerous approaches described in the literature for removal of lesions involving the clivus. Cranial nerve function is preserved, and vascular injury avoided. Dysphagia and aspiration are not encountered, nor is an oroantral fistula created.

It is the purpose of this communication to describe the authors' three year experience with this direct, wide-field approach to the clivus and the upper cervical spine in the management of clivus tumors and craniocervical junction abnormalities. A total of fourteen cases were operated upon during this period. Clivus lesions included three chordomas, four meningiomas, two low grade chondrosarcomas, one fibromyxoid tumor, one prolactin-secreting adenoma and one cystic adenocarcinoma. Two craniovertebral junction abnormalities involved the odontoid process compressing the lower brain stem. A presentation of the surgical technique in detail of this new approach to the clivus and upper cervical spine will be described. A discussion of selected cases and surgical outcome will follow.

9:20 a.m. Microsurgical Management of Craniopharyngioma

Edward L. Seljeskog, Dennis Y. Wen and Stephen J. Haines Minneapolis, MN

Over the past several decades, optimal management for craniopharyngioma has and continues to remain controversial. Advances in surgical techniques, postoperative supportive care and radiologic imaging have permitted contemporary neurosurgeons to often remove these tumors with a minimum of mortality and morbidity. Although histologically a benign lesion, craniopharyngiomas have historically exhibited a major propensity for recurrence, tempting the surgeon to always consider an aggressive surgical excision has all too often resulted in major endocrine and hypothalamic dysfunction, along with significant neuropsychological alteration.

In an attempt to determine our best course for the future in the management of these lesions, we have carried out a retrospective analysis of our experience involving 34 patients, who underwent craniotomy and microsurgical excision of their craniopharyngioma from 1975 through 1989. This was a period of time when CT/MRI imaging and microsurgical approaches were routinely used. The mean followup of these 34 cases was 6.4 years and no patient was lost to followup.

Among those 25 patients treated with surgery alone, there were ten in whom a "total resection" was felt to have been achieved. Within this group of ten patients, eight remained tumor free at followup. Two patients had recurrences, a rate of 20%.

In contrast were those 15 patients in whom residual tumor was known to have not been removed. This patient group had a recurrence rate of some 60%. In comparison were eight individuals who underwent subtotal excision, but who also had adjuvant radiation. Here there was a recurrence rate of only 12% and a significantly better recurrence free interval, compared to those patients who underwent apparent total surgical excision or subtotal excision without radiation ($P<.05$) and $P<0.25$).

Concerning endocrine and visual field defects, both of these perioperative problems were commonplace before, as well as following surgery. Our review, along with numerous other reports, clearly show that aggressive surgical excision can be both safe and effective. Still evident, however, was the rough correlation between postoperative endocrine dysfunction, and the aggressiveness of surgical excision.

While any retrospective review of this sort, cannot definitively resolve issues relating to the ideal and most effective surgical approach, it would appear from our data that when total surgical excision is not easily achieved, leaving a small residual element of tumor, coupled with postoperative radiation will not adversely effect long term patient outcome or rate of tumor recurrence.

ACADEMY AWARD **Moderator:** George T. Tindall
Winner: Mary Louise Hlavin
Runner up: G. Alexander West

9:40 a.m. Molecular Structure and Functional Testing of the Human L1 Cell Adhesion Molecule: An Interspecies Comparison
Mary Louise Hlavin

Monday, September 23, 1991

10:00 am. Effects of External pH on Ionic Currents in Smooth Muscle Cells From the Basilar Artery of the Guinea Pig
G. Alexander West

10:15 a.m. Coffee Break

Scientific Session II: Symposium on Brain Tumors.
Moderator: Roberto C. Heros

10:40 a.m. Low Grade Supratentorial Gliomas. Cure or Palliation?
S. Mullan and D. Johnson

There is a group of intermediate grade gliomas, mostly but not exclusively, situated in the frontal lobe, which appear to be discrete on MRI with gadolinium. They do not invade an eloquent area, the internal capsule nor the basal ganglia. At surgery, by biopsy they do not infiltrate beyond their apparent bed. By a combination of MRI gadolinium (preferably 3D) for localization, mapping by evoked sensory potentials, direct electrical stimulation and local anesthesia (if necessary), for preservation of eloquent cortex and by biopsy for confirmation of margins it is possible to effect an apparently total removal. Ultra sonic suction is a definite technical help. Laser and ultra sonic localization are of some but lesser value. In a small series variously diagnosed as anaplastic, intermediate,grade II, grade III and astrooligo followed for five years, there has been no sign of recurrence on the MRI gadolinium scan. It is recognized, and will be demonstrated, that such tumor free intervals do not denote a "cure". Nevertheless it is felt that this attempted "cure" is preferable to some of the alternative methods currently in use, which are biopsy or subtotal removal with irradiation, or implanted radio active isotopes.

11:00 a.m. Immunotoxin Treatment of Central Nervous System Neoplasia
W.A.Hall, O.Fodstad, A. Myklebust, A. Godal Minneapolis, MN

The poor prognosis associated with primary central nervous system cancer and carcinomatous meningitis has led investigators to seek new, innovative treatment modalities. Immunotoxins are cell-type selective carrier ligands linked to extremely potent toxic proteins that have excellent in vitro and invivo efficacy against many malignant cell types. We have developed three different immunotoxins using the human growth factor, transferrin (Tfn), as the carrier ligand and an abrin variant, Pseudomonas exotoxin A (PE), and a diphtheria toxin mutant, CRM 107, as the toxin moieties. Transferrin-abrin variant and Tfn-PE inhibited protein synthesis better than Tfn-CRM 107 in LOX melanoma, SNB19 and SF295 glioma cell lines.

An animal model of leptomeningeal neoplasia was established in the nude rat using melanoma, small cell lung carcinoma (SCLC), and lymphoma cell lines. After placing an indwelling intrathecal catheter, animals were inoculated with $5x10^5$ cells growing as a monolayer or in suspension. Lower extremity paraplegia resulted at 9.24 days for melanoma (n=20) and 25 days for SCLC (n=3). Because transferrin receptors (TR) were detected on LOX cells, one microgram of Tfn-PE was instilled intrathecally 24 hours after tumor cell inoculation. Control animals (n=10) treated with phosphate-buffered saline became paraplegic at 10.7 ± 2.75 days, compared to treatment animals (n=10) where paralysis was seen at 15.5 ± 4.58 days, representing a delay in onset of paraplegia of 31% ($p=0.015$).

The in vitro cytotoxicity of Tfn-toxin conjugates against glioma and melanoma cells supports the use of the TR as an appropriate target for these immunotoxins, until more specific cell-surface antigens are identified. The observed delay in the onset of paraplegia in treated animals with neoplastic meningitis suggests that immunotoxins could be clinically tested in phase I/II clinical trials in patients with carcinomatous meningitis.

11:20 a.m. An Update in the Treatment of Primary and Metastatic Malignant Brain Tumors Using Combination Chemotherapy With Osmotic Opening of the BBB

E.A.Neuwelt Portland, Oregon

Currently, 200 patients have undergone blood-brain barrier (BBB) disruption about 2,000 times. Major tumor responses have been observed in patients with malignant gliomas particularly when treated prior to radiation. The most dramatic results are in primary CNS lymphoma patients who have been included prior to radiation therapy. We have achieved a nearly 50% relapse-free survival rate (median survival 45 months) with long-term survival up to 100 months. Improved or stable cognitive function has been documented in the nonirradiated patients followed at least 1 year with serial medical psychological testing (n=8).

The choice of chemotherapeutic agents has been expanded recently in 22 patients, from methotrexate and cytoxan to include etoposide and carboplatinum. Germ cell tumors have been particularly responsive to these latter drugs and objective responses also have been seen with lymphoma and breast cancer. We are interested in treating patients with metastatic breast cancer and lung cancer with carboplatinum and etoposide in view of the sensitivity of these carcinomas to these agents. The addition of carboplatinum and etoposide, as well as the recent introduction of bone marrow growth factors (i.e., G-CSF), has significantly expanded our ability to deliver effective solid tumor agents in both primary CNS disease and metastatic disease prior to radiotherapy with less myelosuppression and with maintenance of cognitive function.

Monday, September 23, 1991

11:40 a.m. Intracerebral and Systemic Effects of Tumor Necrosis Factor (TNF) Secreted by Brain Tumors
K.J.Tracey Nre York NY

TNF, a cytokine produced by astrocytes, hypothalamic neurons, and inflammatory cells, is involved in the host response to meningitis, AIDS, head injury, edema formation, and multiple sclerosis. Because it is also cytotoxic against certain tumors, there is some interest in its role as a potential anti-neoplastic agent. Very little is known however, about the local or systemic effects of TNF produced in the region of a brain tumor. We have studied these effects using a genetically engineered cell line (CHO-TNF) that secretes h-TNF. When stereotactically injected into the brains of nude mice, these cells form TNF-producing brain tumors; control mice received genetically similar cells (CHO-NEO) that differed only by the absence of the h-TNF gene. RESULTS: h-TNF produced by CHO-TNF brain tumors crossed the blood-brain barrier, resulting in marked elevations of serum h-TNF (0.66 $\pm$ 0.22 ng/ml); no h-TNF was detectable in controls. The local production of h-TNF in brain induced several striking histological changes, most notably peritumoral neovascularity, dilated blood vessels with focal thrombosis in adjacent microvasculature, and gliosis. These changes were not observed in controls with similar-sized CHO-NEO tumors, which grew at similar rates. Mice bearing TNF-secreting tumors succumbed within days however, because they developed profound anorexia and weight loss. Interestingly, an entirely different metabolic response developed when the TNF-secreting tumor was implanted in the hindlimb: although serum TNF levels were similarly elevated, anorexia and weight loss did not develop for eight weeks. CONCLUSION: TNF secretion in the region of a brain tumor causes prominent neovascularity and local inflammatory responses. Moreover, TNF-induced alterations of CNS neuroendocrine outflow caused profound derangements of metabolic homeostasis independent of circulating peripheral TNF. These studies describe a useful model for investigating the biology of TNF in intracerebral tumor growth. It is hoped that additional studies may advance our understanding of the role of cytokines in the pathogenesis of brain tumors.

12:00 p.m. Long Term Followup After Surgery for Third Ventricular Tumors
J.Ausman, C.Coccia, H. Hamiyet-Camuscu, G. Malik Detroit MI

Many series of open microsurgical resection of third ventricular lesions have been published. In this series we have had the opportunity to review treatment of tumors from all regions of the third ventricle at long-term follow-up with a special emphasis on the patient's ability to return to and maintain over several years his premorbid level of function. From 1979 to 1990, 49 patients underwent surgical procedures for third ventricular region neoplasms excluding craniopharyngioma and pituitary adenomas. For analysis the lesions were divided into colloid cysts (16), anterior and mid-third ventricular tumors (13), and pineal region tumors

(20). For all groups long-term survival was primarily dependent on the natural history of the histopathology. At follow-up (clinic interview and exam or phone interview if not available for clinic exam) function was assessed as excellent (back to work and/or pre-morbid level of function), good (able to work, some at original job but have some deficit which impairs performances), fair (significant deficits but able to care for self, may be able to hold menial job), and poor/dead.

In all groups morbidity in long-term survivors were primarily related to cognitive and memory deficits related to surgery. At a mean follow-up of 4.7 years, 23% of colloid cyst patients were so impaired that they were no longer capable of reasonable work and another 15% had deficits preventing them from attaining their pre-morbid level of function. Of the 11 of 13 patients with anterior or mid-third ventricular lesions assessed at a mean of 7.6 years, 27% had excellent function, 27% good, 18% fair, and 27% were dear or poor. At a mean follow-up of 5.4 years, over half of the pineal tumor patients ere functioning well but the significance of this is small since the histopatholgy is so variable in this region and survival is very dependent on patholgy. More importantly, 10 of 11 long-term survivors are functioning well (5-excellent, 5-good) at follow-up.

Open resection of third ventricular lesions is known to be technically possible. The standard open and perhaps even the limited approach resection of colloid cysts can leave patients with very subtle but profoundly disabling deficits. Because of the varied pathology and frequency of relative radioresistance of pineal region tumors coupled with 90% of long-term survivors functioning well, open resection remains the procedure of choice for initial treatment of these lesions. Anterior and mid-third ventricular tumors can also be treated acceptably with standard open resection; however, new image guided stereotaxic resection may allow for more extensive resection with a lower morbidity.

12:20 p.m. Angiogenic Modulation of Tumors in Neurofibromatosis
R.L.Martuza, Y.Takamiya, R.M.Friedlander, H.Brem,A.Malik Boston MA

There has been neither a useful animal model for neurofibromatosis (NF) nor a beneficial non-surgical therapy for NF and NF-associated tumors. We have used a human tumor/nude mouse xenograft model to study growth and neovascularization of benign and malignant human schwann cell tumors from both NF1 and NF2 as well as tumors of similar histologic type from non-NF patients. In prior reports, we demonstrated that tumor "take" and growth for schwannomas (acoustic neuromas) correlated with the extent of induction of neovascularization. We also demonstrated that inhibition of neovascularization with heparin and cortisone also inhibited growth of a human malignant schwann cell tumor (neurofibrosarcoma). However, this regimen

is not applicable to human use. Using human acoustic neuromas and neurofibromas, we now present data demonstrating that the angiogenic modulator, AGM-1470, will inhibit neovascularization in this model. Our initial studies suggest that AGM-1470 is effective both in neurofibromas from NF1 and acoustic neuromas from NF2 patients as well as from histologically similar tumors not occurring in NF. Further, in one human neurofibrosarcoma from a patient with NF1, we have demonstrated growth inhibition by AGM-1470. Administration of AGM-1470 is well-tolerated and is compatible with human use. Angiogenic tumor growth modulation provides a novel means of therapy for both benign and malignant tumors of the central and peripheral nervous system in people with NF1 or NF2 and for histologically similar tumors in the general population.

12:40 a.m. SPECIAL LECTURE
Interventional Neuroradiology and the Neurosurgeon
L.Nick Hopkins Buffalo NY

1:00 p.m. Adjournment

Tuesday, September 24, 1991

SCIENTIFIC SESSION III SYMPOSIUM ON APPLIED TECHNOLOGY
MODERATOR: MARTIN WEISS

8:00 a.m. Stereotactic VOP/VIM Thalamotomy for the Treatment of Tremor in the Post-L-Dopa Era
P.J. Kelly Rochester MN

Following the general availability of L-dopa in 1968, ventralis lateralis (VL) thalamotomy has been only rarely offered to patients with medically refractory tremor. However, new stereotactic instrumentation and improved stereotactic localization methods which employ computer-based imaging, neurophysiologic techniques and intraoperative speech monitoring render these procedures safer now than in the past.

Seventy three patients underwent stereotactic ventralis lateralis thalamotomy (24 right, 44 left, 5 bilateral) at Mayo Clinic between September 1984 and March 1991 for the treatment of medically refractory movement disorders, including 42 patients with Parkinson's disease, 8 patients with essential tremor, 17 patients with intention tremor, and 6 patients with complex dystonias. Long term postoperative follow-up examinations (mean follow-up 28 months) in each of these groups revealed significant improvement (complete ablation) in 39 (36) of 42

Parkinson's patients, 8 (4) of 8 essential tremor patients, 15 (13) of the 17 patients with intention tremor and in 4 (3) of the 6 patients with dystonia. There was no mortality. Morbidity in this series includes 2 patients with contralateral limb dyspraxia (mild/moderate), and 4 patients with mild to moderate hypokinetic dysarthria following left thalamotomy. Modern VL thalamotomy provides an excellent surgical alternative to patients with parkinsonian tremor, familial or essential tremor, and intention tremor.

8:20 a.m. Neural Networks--A Unique Method of Data Analysis
A.R.Wyler Memphis TN

A neural network is a form of computer simulated artificial intelligence that looks for complex relationships between numerous variables. Once given a set of variables, the network begins to learn relationships between them and can be trained to generalize from them. This approach is especially good for pattern recognition and completing incomplete data sets. Neural networks have gained increasing interest from a variety of scientific disciplines and have been developed to solve such complex problems as predicting protein structure from amino acid sequences. We have begun developing neural network to help select candidates for epilepsy surgery. Preliminary results show that neural networking can predict with a high degree of accuracy which patients will or will not do well with resective surgery. The theoretical background to neural networks and how they may be applied to other clinical problems will be discussed.

8:40 a.m. Stereotactic Techniques in Vascular Neurosurgery
M.B.Sisti and Bennett M. Stein New York, NY

Improvements in stereotactic techniques have expanded the indication of these methods in the management of arteriovenous malformations (AVMs) and cavernous malformations (CMFs). Primarily, the precision of stereotactic localization can be utilized for either the microsurgical resection of small or inaccessible vascular lesions or for use with radiosurgical devices for similar purposes. Although much attention has recently been devoted to the stereotactic radiosurgery of AVMs and CMFs, less attention has been directed to the stereotactically guided resection of these lesions. Both radiosurgery and stereotactically guided microneurosurgery have been available at our institution for the last 2 years. During this period 25 CMFs and 10 AVMs were resected by stereotactically guided craniotomy and 1 CMF and 8 AVMs were treated with a linac radiosurgery unit.

The primary indications for the use of stereotactic localization in this group of 44 patients were lesion size and location. There is a substantial overlap in the use of either radiosurgery or

stereotactically guided craniotomy in the treatment of AVMs or CMFs less than 2.5 cm in diameter. Stereotactically guided craniotomy for the complete resection of these lesions was used whenever possible and radiosurgery was reserved for lesions which were larger, more irregular, and those lesions that involved the internal capsule, thalamus, or brain stem. In general, patients treated with radiosurgery did not present with intracerebral hemorrhage as their primary presenting symptom, whereas the group treated with craniotomy presented with symptomatic hemorrhages from their vascular malformations.

The operative results, indications and follow-up of this group of patients will be presented. The indications for stereotactically guided craniotomy vs. radiosurgery will be presented with regards to the management of small AVMs and CMFs.

9:00 a.m. Spatial Registration of Image Coordinate Systems and Stereotactic Coordinate Systems Using a Machine Vision Technique
M.Heilbrun, S.Koehler, P.McDonald, W.Peters, C.Wiker, V. Slemonov Salt Lake City, UT

CT and nondistorted MRI image data sets can be gathered as a volume array that can be defined as a Cartesian image coordinate system. Through computer graphic techniques external fiduciary marks such as natural facial structures or markers noninvasively fixed to scalp can be assigned a coordinate in the image coordinate system which can be related to the position of intracranial structure. A machine vision technique used in the industrial workplace as a three dimensional digitizer can be used to define a stereotactic coordinate system. This technique uses a perspective transformation to determine the three dimensional position of objects seen on digitized video images. Such objects can be the identical external fiduciary marks defined in the image coordinate system. Using computers for spatial registration of image coordinate systems with stereotactic coordinate systems has been termed frameless stereotaxy.

We have described initial accuracy tests of stereotactic localization with video cameras tested with a standard BRW simulator and a phantom using a specially constructed video localizer similar to the Gybels-Suetens and Siddon stereotactic angiographic localizers. Localization accuracy was within two millimeters.

Initial experience with this technique will be described. With PC and Macintosh computers these techniques can be used for monitoring the position of orientation of instruments within the cranial vault without the constraint of a stereotactic arc.

With sufficient computer speed these techniques can be used for continuous registration on images of the position of a surgical instrument.

Tuesday, September 24, 1991

9:20 a.m. PRESIDENTIAL ADDRESS: NICHOLAS T. ZERVAS

10:10 a.m. Coffee Break

SCIENTIFIC SESSION IV: MODERATOR: GEORGE OJEMANN, M.D.

10:40 a.m. Intracranial Hypertension and Glial Cell Production of Leukotriene C4 Induced by Endotoxin
J.B.G.Ghajar, R.J.Hariri, R.A.R.Fraser New York, NY

Marked deterioration of neurologic function accompanies organ dysfunction in systemic infection. Although previous hypotheses have suggested that either cerebral hypoperfusion, anoxia, or progressive brain edema may be causative, the pathogenesis remains unknown. Septic patients with stable or supported hemodynamics and adequate oxygenation may manifest confusion, stupor, or coma. Many mediators of inflammation have been implicated in derangements of host response which precede organ dysfunction in systemic sepsis. Leukotrienes (LTs) have been identified as mediators of the cerebrovascular dysfunction in models of central nervous system malignancy and traumatic brain injury, which may be characterized by alternations in blood brain barrier function leading to increased brain free water content. We previously demonstrated that cultured human glial cells generate LTC4 de novo in response to mechanical injury and calcium ionophore A23187. The acid (AA) from membrane phospholipid pools. Endotoxin (LPS) is a specific phospholipase A2 stimulant, the enzyme responsible for release of AA from membrane phospholipids. These studies were performed to test the hypothesis that neurologic dysfunction associated with sepsis is mediated by similar alterations in glial cell metabolism stimulated by LPS exposure resulting in the generation of these lipoxygenase products in vitro and whether endotoxemia in miniature swine was associated with intracranial hypertension associated with the appearance of these eicosanoids in cerebrospinal fluid. Confluent cultures of human glial cells obtained from normal brain surgical specimens were employed in this study. Baseline supernatants were collected and cultures were exposed to varying concentrations of endotoxin in serum free media. Supernatants were collected at 5, 15, 30 and 60 minutes following exposure and evaluated using a competitive displacement enzyme immunoassay. LPS stimulated glial cell LTC4 production at doses>100pg/ml supernatant. Sexually mature miniature yucatan swine were surgically prepared as previously described (Hariri,et al, Surg. Forum, Vol 38, 1989). Animals were then exposed to endotoxin (LPS W E. Coli 0127:B8) 0.1 mg/kg I.V. with hemodynamic and intracranial pressure monitoring. Lactated Ringer's solution was infused to maintain a mean arterial blood pressure of 55 mm Hg. Intracranial pressure rose dramatically within 3 hours of endotoxemia despite significant hypotension. In addition, CSF levels of LTC4

were significantly elevated above baseline 4 hours after endotoxin exposure compared to baseline values. These data indicated that: 1-concentrations of LPS greater than 100 pg/ml can stimulate the production of LTC4 from glial cells in similar amounts to that seen following mechanical injury and ionophore exposure. 2-endotoxemia results in intracranial hypertension associated with the appearance of LTC4 in CSF. This strongly suggests a role for LPS induced leukotriene production by human glial in the pathophysiology of cerebrovascular dysfunction and neurologic derangements associated with sepsis.

11:00 a.m. Monoclonal Antibody to Aminomalonic Acid (AMA) Epitope Reactive to Arterial and Granuloma Foam Cells: A Probe for the Radioimaging of Atheromas

W.M.Kirsch, K.Cheng, S. Fowler, T.Koch, R. Wellerson, S.Nehlsen-Cannarella, J.VanBuskirk, W. Kelln, S.Grinde, P.Wheelan. Albuquerque, NM

Aminomalonic acid (AMA) is an acid labile amino recently identified in protein hydrolysates of calcified tissues including atherosclerotic plaque. We have prepared hybridomas producing monoclonal antibodies (MoAbs) to the AMA epitope (anti-Ama). An Ama-containing peptide (-NH-CS-NH-C_6-NH-CS-NH-$(CH_2)_5$-CO-NH-CH(CO_2H)-CO-NHEt) has been attached to lysine residues of bovine serum albumin and keyhole limpet hemocyanin at high epitope densities (molar ratios of Ama to protein: 40 - 50). A panel of monoclonal antibodies has been produced that exhibit high affinity to peptide-bound Ama (on the order 10^{-9} M) with some cross reactivity to synthetic peptide bound aspartyl or glutanyl residues. Immunocytochemical studies using anti-Ama MoAb reveal striking reactivity with arterial foam cells in four species (human,subhuman primate, rabbit, and rat), in addition to experimentally induced foam cell granulomas.

I-131 anti-Ama labelled MoAb (IgG_2) has been administered to both homozygous EHL-WW atherosclerotic rabbits and normal NZW rabbits. Sequential dynamic studies with the Picker Gamma Camera provided imaging until the animals were sacrificed and the major organs harvested. Though higher specific binding to the atheromatous arteries was detected, background imaging reduced the target/non-target ratio to approximately 1.2 to 1.3. This immunopharmacokinetic problem is being currently studied by utilizing both smaller immunoreactive fragments and preloading with cold MoAb. This study attempts to image the atheromatous lesion at early stages of its evolution when reversibility or halting of the disease may be accomplished.

11:20 a.m. Intermittent or Continuous Temporary Vessel Occlusion? The Effect of Preexisting Hypertension on Pathological Outcome

W.R.Selman, C.Rosenstein, R.A.Ratcheson Cleveland, Ohio

INTRODUCTION:
Temporary occlusion has become a standard procedure in the treatment of difficult aneurysms. The optimal method of temporary vessel occlusion has not been determined since the tolerance of the brain to focal ischemia is not well defined, the role of reperfusion injury may be significant, and differences in the baseline cerebrovascular status may influence the response to temporary occlusion. In order to elucidate the role of these factors, we developed a small animal model of temporary, reversible, focal ischemia.

METHODS:
Four groups of male, wistar rats, underwent middle cerebral artery occlusion. Two strains of rats were used; spontaneously hypertensive (SH) and normotensive (NT). Two paradigms of vessel occlusion were used; one episode of 60 minutes duration or 3 episodes of 20 minutes of occlusion with 10 minutes of reflow between each episode. Cerebral blood flow was monitored with a laser doppler flow probe over the affected hemisphere. Pathological analysis for volume of infarction was determined 72 hours after the ischemic insult.

RESULTS:
Blood flow was decreased in all animals in the affected territory, and reperfusion was confirmed in all multiple occlusion animals. The volumes of infarction were as follows: SH-multiple (n=6) 220 ± 11 mm^3; SH-single (n=5) 195 ± 26 mm^3; NT-multiple (n=5) 98 ± 19 mm^3 NT-single (n=5) 164 ± 24 mm^3. Five of six multiple SH animals demonstrated severe hemorrhagic infarction in the affected hemisphere.

CONCLUSIONS:
Intermittent, temporary occlusion is associated with a smaller volume of infarction in normotensive animals. In hypertensive animals, although the volumes of infarction were similar, intermittent temporary occlusion may actually be more deleterious in light of the hemorrhagic nature of the infarction.

11:40 a.m. Ventricular Shunts for Hydrocephalus: A Clinical Study of the Zero Intracranial Pressure Shunt System

E.L.Foltz, R.Meyer, J.Blanks Orange CA

Tuesday, September 24, 1991

Overdrainage of CSF by ventricular shunts is increasingly recognized, though the incidence is still somewhat imprecise. Subdural hematoma, acquired craniostenosis, slit-ventricle syndrome and low ICP syndrome may result. The overall incidence is 10-15% occurring at 6 1/2 to 7 years after initial shunt. Acquired disability can be severe.

Our report in 1987 identified the low ICP syndrome in 14 patients. A Flo-Control valve was initially effective in this group, but the low ICP syndrome returned in 9 of the 14 patients within two years, apparently due to the valve still functioning on a CSF pressure gradient.

Therefore, ICP in normal patients as compared to those with ventricular shunts for hydrocephalus was reexamined. Emphasis appeared concerning the difference in ICP in supine and upright body positions. Gravity induced CSF shunt flow clearly played a major role in hydrocephalus shunt overdrainage, resulting in an unusual degree of negative ICP.

The concept of a "zero intracranial pressure level" which could be varied and controlled by use of a shunt which included a device already on the market (Siphon Control Device) resulted in the Zero Pressure Shunt System. This system should control ICP by closing shunt function at a selected negative ICP in the body upright position (determined ONLY by device position on the skull relative to gravity). The opening pressure of the shunt functions in the supine body position controlled only by a standard reservoir - low pressure Flo-Control valve. This system has a selected opening pressure (upright body) and a closing pressure (supine body; Siphon Control Device).

We report the use of this system in 58 patients with obstructive hydrocephalus. This includes indications for use, accuracy of estimated and achieved post-operative ICP, effect on ventricle size (CT), complications and clinical effectiveness over the past four years. Comparisons are made with 27 "control patients" where possible.

12:00 p.m. Adenosine in the Regulation of Neonatal Cerebral Blood Flow
T.S.Park St. Louis MO

Our laboratory has demonstrated evidence that adenosine, a purine nucleoside, is involved in mediation of vasodilatory adjustments during hypotension, hypoxia and seizures in the neonate. In newborn piglets subjected to hemorrhagic hypotension, we measured interstitial fluid adenosine concentrations of the cerebral cortex using the microdialysis technique and pial anteriolar diameters using the cranial window technique. Hemorrhagic hypotension caused progressive increases in cortical interstitial adenosine concentrations in parallel with progressive dilation of the pial arterioles. Furthermore, the pial arteriolar dilation in

response to hypotension was enhanced by topical application of dipyridamole, an adenosine transport inhibitor. Detailed experimental data supporting adenosine as a metabolic mediator for neonatal cerebral blood flow will be presented.

12:20 p.m. Intraoperative Angiography as an Adjunct to Vascular Neurosurgery Mechanisms
D.L. Barrow Atlanta GA

Intraoperative angiography is useful in verifying the results of neurovascular operations during the procedure and prior to wound closure. In the past, the popularity of intraoperative angiography has been hindered by technical limitations such as bulky, expensive equipment, inefficient film cassettes, lengthy development time, the need for large volumes of contrast, poor resolution and obscuration of angiographic detail by radiopaque surgical implements.

We report our technique and experience with 141 intraoperative angiographic procedures performed with a portable digital subtraction unit that has eliminated many of the earlier detractors to the routine use of the technique. Of these 141 studies, intraoperative angiography provided information that altered the operative procedure on 25 occasions, thus reducing the need for reoperations and potentially reducing operative complications. In seventy cases, postoperative angiography was performed and compared to intraoperative studies.

We have found intraoperative angiography to be valuable in documenting the complete obliteration of intracranial aneurysms and both intracranial and spinal AVM's, assuring the patency of normal vessels in the vicinity of vascular anomalies, verifying the patency of endarterectomies and bypass grafts, for intraoperative localization of small vascular lesions that are not readily apparent on the surface of the brain or spinal cord, and for visualization during intraoperative endovascular procedures.

The quality of images provided by digital subtraction angiography makes the technique a useful adjunct to neurovascular surgery.

12:40 p.m. <u>Special Basic Science Lecture:</u> Motor System Control Mechanisms
T.J. Ebner Minneapolis MN

1:00 p.m. Adjournment

WEDNESDAY, SEPTEMBER 25, 1991

SCIENTIFIC SESSION V MODERATOR: CHARLES HODGE

8:00 a.m. The Anatomy of Incompletely Clipped Anterior Communicating Artery Aneurysms
W.Shucart Boston, MA

In a series of 132 patients with anterior communicating artery aneurysms the author has had an incidence of incompletely clipped aneurysms of approximately 6%. Seven of those eight cases had similar anatomic set ups which were related to the incomplete clipping. This situation was one in which the aneurysm pointed superiorly and posteriorly. This anatomy makes it easy for the surgeon to only visualize part of the dome of the aneurysm while thinking he is visualizing all the dome. Case illustrations and ways to avoid the problem will be presented.

8:20 a.m. Studies of L-NNA and L-arginine on Cerebral Collateral Blood Flow and Endothelium-Dependent Relaxation
M.G.Muhonen, C.M.Loftus Iowa City, Iowa

Nitric oxide (NO) is postulated to act as an endothelium-derived relaxing factor (EDRF). This study tested the hypothesis that inhibition of NO formation with N^G-nitro-L-arginine (L-NNA, which blocks synthesis of NO) during hypotension would decrease collateral cerebral blood flow, and that this effect would be reversed by administration of L-arginine (a precursor in NO synthesis). In 25 anesthetized dogs, a branch of the middle cerebral artery was cannulated, and the collateral dependent tissues (CDZ) were identified with a shadow flow technique. Regional cerebral blood flow (rCBF) was determined with radiolabeled microspheres. In five animals with phlebotomy induced hypotension (MAP 50 mmHg), CDZ rCBF decreased from 70 ± 22 ml min^{-1} $100g^{-1}$. (mean$\pm$SE) to 55 ± 18 and 46 ± 17 at 20 and 40 minutes after injection of L-NNA 5 mg/kg ($p<0.05$). Vessel diameter decreased insignificantly and microvascular pressure did not change. When both L-NNA and L-arginine (60 mg/kg) were infused in five separate hypotensive dogs rCBF increased from 63 ± 20 to 83 ± 18 and 91 ± 17 at 20 and 40 minutes ($P<0.05$ for both), with an insignificant increase in vessel diameter.

Fifteen animals were then studies under normotensive conditions. Although vessel diameter decreased 13% after L-NNA infusion, and increased 18% after L-arginine infusion, there was no significant change in CDZ rCBF.

There were two major findings from these studie.First, inhibition of NO significantly decreased rCBF under demand (hypotensive) but not normal conditions. Second, L-arginine produced

significant increases in rCBF during hypotension. The hypothesis was proved, and these finding support the role of L-arginine derived nitric oxide in mediating vasorelaxation under demand conditions, in this case hypotension.

8:40 a.m. Spontaneous Seizures in Baboons (Papio Cynocephalus SP): A New Primate Model of Epilepsy

S.Murk, J.L.Story, J.Luther, K.Kagan-Hallet, J.Odom, E. Eidelberg San Antonio, Texas

A unique group of baboons (Papio cynocephalus sp.) in a large colony of the Southwest Foundation for Biomedical Research, display typical grand mal seizures without any obvious provocation. Most of the animals studied so far seem to also have frequent episodes suggestive of complex partial seizures. Several animals have been monitored with scalp and/or stereotactically implanted deep brain electrodes, with simultaneous video and EEG recordings to correlate the electro-physiologic substrate with the observed clinical seizure activity. Anayfis of this data suggests that the origin of these spontaneouly occuring seizure events lie in the mesial temporal lobe structures. Neuropathologic evaluation with serial section studies of the brains of these animals, including two who died in status epilepticus, showed no obvious pathology with usual light microscopic studies including Nissl and Golgi strains. GABA receptor binding and enhancement of specific flunitrazepam binding by GABA have been studied in these animals in an attempt to determine the possible involvement of these receptor systems in the seizures. Preliminary studies suggest lower levels of GABA binding in the cerebral cortex of epileptic baboons relative to controls, and decreased coupling between GABA and benzodiazepine receptor sites in cortex and hippocampus.

C-fos levels have been obtained from several regions of epileptic and control baboon brain as well and results indicate a several fold increase in the hippocampus of seizure animals over controls. Analysis of the breeding colony database has yielded preliminary evidence that the seizure phenomena may be genetically determined.

Data from on-going studies is presented, with examples of scalp and depth electrode EEG, light microscopic pathologic analysis of serial brain sections as well as receptor systems studies and C-fos measurements from multiple cerebral locations. Pedigree data tracing the appearance of seizure affected animals through several generations of the breeding colony at Southwest Foundation for Biomedical Research is also presented.

Wednesday, September 25, 1991

9:00 a.m. The Migration of Fetal Astrocytes and Astrocytoma Cells through Normal Brain

E.R.Laws, J.J.Bernstein, W.Goldberg Washington DC

We performed transplantation studies of labeled fetal rat astrocytes in normal rat brain which revealed impressive migration of transplanted graft-derived cells moving in stereotypical pathways over basal lamina and along white matter tracts. Similar studies were performed using C6 astrocytoma cells, and their migratory ability was even more impressive, with tumor cells migrating long distances by creating tunnels and tubes through matrix tissues of the brain, including basement membrane. We now report studies of C6 cells migrating through hydrated collagen I gel and through artificial basement membrane, with further insights as to the mechanisms involved and the tumor cell-specific nature of such migratory activity. It is of great interest that artificial basement membrane is an effective barrier for fetal rat astrocytes but not for astrocytoma cells.

The studies of tumor cells migrating through brain matrix may be a model for the phenomenon of multifocal glioma and also for recurrence of malignant glioma after effective local therapy. In the experimental situation, individual cells migrate through matrix tissues, and secondarily form masses by coalescence of adjacent micropockets of tumor cells. The collagenases, proteases, cell adhesion molecules and other basic mechanisms involved are currently under study.

9:20 a.m. Tumors of the Lateral Wall of the Cavernous Sinus

M.El-Kalliny, H.R.vanLoveren, J.M.Tew,Jr., J.T.Keller Cincinnati, Ohio

The lateral dural wall of the cavernous sinus (CS) is composed of two layers: the outer dural layer (dura propria) and the inner membranous layer. The membranous layer is formed by the sheathes of cranial nerves III, IV, V_1 and occasionally V_2 and a reticular membrane extending between these nerve sheaths. These two layers are loosely attached and easily separable. Tumors arising from the contents of the lateral dural wall are located between these two layers and are classified as interdural. They are in essence extradural/extracavernous. The inner membranous layer separates these tumors from the venous channels of the CS.

Preoperative recognition of tumors in this location is critical to selection of an appropriate microsurgical approach. The MRI characteristics of interdural tumors include an oval shaped, smooth bordered mass with medial displacement but not encasement of the cavernous internal carotid artery. Tumors in this location can be safely resected without entering the CS proper by techniques which permit reflection of the dural propria of the lateral wall.

Wednesday, September 25, 1991

During the last four years we have identified and treated five patients with interdural cavernous sinus tumors: two trigeminal neurinomas arising from V_1, two epidermoid tumors, and one malignant melanoma presumed primary. The authors will discuss the pathoanatomic characteristics, the clinical/radiologic findings, and the microsurgical approach of this unique group of tumors. Comparison of the results of treatment of interdural versus other CS tumors documents a more favorable prognosis for tumor resection and cranial nerve preservation for those which are interdural.

9:40 a.m. The Early Neurosurgery of Australia
N.G. Dan Sydney, Australia

10:00 a.m. Coffee Break

SCIENTIFIC SESSION VI: MODERATOR: STEWART B. DUNSKER

10:30 a.m. SPECIAL LECTURE:

Stereotactic "Radiosurgery" and the Neurosurgeon
Charles Wilson San Francisco, CA

10:50 a.m. SPECIAL SYMPOSIUM: MODERATOR: STEWART B. DUNSKER
Congenital Cranial Base & Spinal Problems
Panelists:

John C. VanGilder: Iowa City, IA	**Craniovertebral Anomalies**
John A. Jane: Charlottesville VA	**Craniofacial Anomalies**
Charles J. Hodge, Jr.: Syracuse,NY	**Syringomyelia and the Chiari Malformation; Mechanisms**
Albert L. Rhoton: Gainesville, FL	**Syringomyelia and the Chiari Malformation; Treatment**
Harold J. Hoffman: Toronto, CA	**Tethered Cord**
Paul H. Chapman: Boston, MA	**Spinal Lipomas**

1:00 p.m. ADJOURNMENT

NOTES

RESIDENTS PAPER AWARD WINNERS

WINNER

MARY LOUISE HLAVIN, M.D

DEPARTMENT OF NEUROSURGERY
CASE WESTERN RESERVE UNIVERSITY SCHOOL OF MEDICINE
CLEVELAND, OHIO

MOLECULAR STRUCTURE AND FUNCTIONAL TESTING OF THE HUMAN L1 CELL ADHESION MOLECULE: AN INTERSPECIES COMPARISON

RUNNER UP

G. ALEXANDER WEST, M.D.

DEPARTMENT OF NEUROSURGERY
HARBORVIEW MEDICAL CENTER
SEATTLE, WASHINGTON

EFFECTS OF EXTERNAL pH ON IONIC CURRENTS IN SMOOTH MUSCLE CELLS FROM THE BASILAR ARTERY OF THE GUINEA PIG

ACADEMY AWARD WINNERS

Paul M. Lin	1955
Hubert L. Rosomoff	1956
Byron C. Pevehouse	1957
Norman Hill	1958
Jack Stern	1959
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James T. Rutka	1988
Christopher D. Heffner	1989
Scott I. Gingold	1990
Mary Louise Hlavin	1991

GUEST	GUEST OF
DANIEL L. BARROW Atlanta GA	GEORGE T. TINDALL
KIM J. BURCHIEL Portland, OR	THE ACADEMY
MAURICE COLLADA Salem OR	DAVID G. KLINE
JAMES C. COLLIAS Hartford CT	NICHOLAS T. ZERVAS
ROBERT C. DAUSER Ann Arbor MI	JULIAN T. HOFF
TIMOTHY J. EBNER Minneapolis MN	ROBERTO HEROS
EDUARDO EIDELBERG San Antonio TX	JIM L. STORY
RUDOLPH FAHLBUSCH Erlangan, Germany	NICHOLAS T. ZERVAS
ROBERT C.A. FREDERICKSON Beachwood OH	JAMES T. ROBERTSON
JAMSHID GHAJAR New York NY	RICHARD A.R. FRASER
JOSEPH H. HAHN Cleveland OH	RALPH G. DACEY
WALTER A. HALL Minneapolis MN	LYLE A. FRENCH
NICHOLAS HOPKINS Buffalo NY	RUSSELL H. PATTERSON,JR

GUEST	GUEST OF
MARK JONES Syracuse NY	CHARLES HODGE,JR.
PATRICK J. KELLY Rochester MN	THORALF SUNDT
CHRISTOPHER LOFTUS Iowa City IA	JOHN VAN GILDER
JOHN C. MULLEN Minneapolis MN	SHELLEY N. CHOU
STEVEN E. MURK San Antonio TX	JIM L. STORY
EDWARD NEUWELT Portland OR	CLARK WATTS
THOMAS O. OESTERLING Beachwood OH	JAMES T. ROBERTSON
JON ROBERTSON Memphis TN	JAMES T. ROBERTSON
ROBERT H. ROSENWASSER Philadelphia PA	WILLIAM A. BUCHHEIT
WARREN R. SELMAN Cleveland OH	ROBERT A. RATCHESON
EDWARD SELJESKOG Minneapolis MN	ROBERTO HEROS
GORDON B. THOMPSON Vancouver CA	ELLIS B. KEENER
CHARLES H. TATOR Toronto CA	HAROLD HOFFMAN

PAST PRESIDENTS

DEAN H. ECHOLS	1938-39
SPENCER BRADEN	1940
JOSEPH P. EVANS	1941
FRANCIS MURPHEY	1942
FRANK H. MAYFIELD	1943
A. EARL WALKER	1944
BARNES WOODHALL	1946
WILLIAM S. KEITH	1947
HOWARD A. BROWN	1948
JOHN RAAF	1949
E. HARRY BOTTERELL	1950
WALLACE B. HAMBY	1951
HENRY G. SCHWARTZ	1952
J.LAWRENCE POOL	1953
RUPERT B. RANEY	1954
DAVID L. REEVES	1955
STUART N. ROWE	1956
ARTHUR R. ELVIDGE	1957
JESS D. HERRMANN	1958
EDWIN B. BOLDREY	1959
GEORGE S. BAKER	1960
C.HUNTER SHELDEN	1961-62
SAMUEL R. SNODGRASS	1963
THEODORE B. RASMUSSEN	1964
EDMUND J. MORRISSEY	1965
GEORGE MALTBY	1966
GUY L. ODOM	1967
JAMES G. GALBRAITH	1968
ROBERT H. PUDENZ	1969-70
WILLIAM B. SCOVILLE	1971
ROBERT L. McLAURIN	1972
LYLE A. FRENCH	1973
BENJAMIN B. WHITCOMB	1974
JOHN R. GREEN	1975
WILLIAM H. FEINDEL	1976
WILLIAM H. SWEET	1977
ARTHUR A. WARD	1978
ROBERT B. KING	1979
EBEN ALEXANDER, JR.	1980
JOSEPH RANSOHOFF II	1981
BYRON C. PEVEHOUSE	1982
SIDNEY GOLDRING	1983
RUSSEL H. PATTERSON,JR.	1984
THOMAS LANGFITT	1985
PHANOR L. PEROT, JR.	1986
SHELLEY N. CHOU	1987
JAMES T. ROBERTSON	1988
THORALF M. SUNDT	1989
ROBERT G. OJEMANN	1990

PAST VICE-PRESIDENTS

FRANCIS MURPHEY	1941
WILLIAM S. KEITH	1942
JOHN RAAF	1943
RUPERT B. RANEY	1944
ARTHUR R. ELVIDGE	1946
JOHN RAAF	1947
ARTHUR R. ELVIDGE	1948
F. KEITH BRADFORD	1949
DAVID L. REEVES	1950
HENRY G. SCHWARTZ	1951
J. LAWRENCE POOL	1952
RUPERT B. RANEY	1953
DAVID L. REEVES	1954
STUART N. ROWE	1955
JESS D. HERRMANN	1956
GEORGE S. BAKER	1957
SAMUEL R. SNODGRASS	1958
C. HUNTER SHELDEN	1959
EDMUND MORRISEY	1960
DONALD F. COBURN	1961-62
EBEN ALEXANDER, JR.	1963
GEORGE L. MALTBY	1964
ROBERT PUDENZ	1965
FRANCIS A. ECHLIN	1966
BENJAMIN WHITCOMB	1967
HOMER S. SWANSON	1968
AUGUSTUS McCRAVEY	1969-70
EDWARD W. DAVIS	1971
JOHN R. GREEN	1972
GEORGE J. HAYES	1973
RICHARD L. DeSAUSSRE	1974
ERNEST W. MACK	1975
FRANK E. NULSEN	1976
ROBERT S. KNIGHTON	1977
ROBERT G. FISHER	1978
H.T. BALLANTINE, JR.	1979
GEORGE EHNI	1980
COURTLAND H. DAVIS, JR.	1981
JOHN F. MULLAN	1982
HUGO RIZZOLI	1983
JAMES W. CORRELL	1984
E.B. HENDRICK	1985
GRIFFITH R. HARSH III	1986
ELLIS B. KEENER	1987
ROBERT GROSSMAN	1988
JIM STORY	1989
JOHN A. JANE	1990

PAST SECRETARY-TREASURER

FRANCIS MURPHEY	1938-40
A.EARL WALKER	1941-43
C.ERICKSON	1944-47
WALLACE B. HAMBY	1948-50
THEODORE B. RASMUSSEN	1951-53
EBEN ALEXANDER, JR.	1954-57
ROBERT L. McLAURIN	1958-62
EDWARD W. DAVIS	1963-65
ROBERT G. FISHER	1966-68
BYRON C. PEVEHOUSE	1969-72

PAST SECRETARY

BYRON C. PEVEHOUSE	1973
RUSSEL H. PATTERSON, JR.	1974-76
PHANOR L. PEROT, JR.	1977-80
JOHN T. GARNER	1981-83
JAMES T. ROBERTSON	1984-86
NICHOLAS T. ZERVAS	1987-89
WILLIAM A. BUCHHEIT	1989-

PAST TREASURER

RUSSEL H. PATTERSON, JR.	1973
PHANOR L. PEROT, JR.	1974-76
JOHN T. GARNER	1977-80
JAMES T. ROBERTSON	1981-83
NICHOLAS T. ZERVAS	1984-86
WILLIAM A. BUCHHEIT	1987-89
JULIAN T. HOFF	1990-

PAST MEETINGS OF THE ACADEMY

HOTEL NETHERLAND PLAZA,CINCINNATI, OHIO	OCTOBER 28-29,1938
ROOSEVELT HOTEL, NEW ORLEANS, LOUISIANA	OCTOBER 27-29,1939
TUDOR ARMS HOTEL,CLEVELAND,OHIO	OCTOBER 21-22,1940
MARK HOPKINS HOTEL,SAN FRANCISCO AND AMBASSADOR HOTEL LOS ANGELES, CALIFORNIA	NOVEMBER 11-15,1941
THE PALMER HOUSE, CHICAGO, ILLINOIS	OCTOBER 16-17,1942
HART HOTEL,BATTLE CREEK, MICHIGAN	SEPTEMBER 17-18,1943
ASHFORD GENERAL HOSPITAL,WHITE SULPHUR SPRINGS, WEST VIRGINIA	SEPTEMBER 7-9, 1944
THE HOMESTEAD, HOT SPRINGS, VIRGINIA	SEPTEMBER 9-11, 1946
BROADMOOR HOTEL, COLORADO SPRINGS, COLORADO	OCTOBER 9-11,1947
WINDSOR HOTEL, MONTREAL, CANADA	SEPTEMBER 20-22,1948
BENSON HOTEL, PORTLAND, OREGON	OCTOBER 25-27,1949
MAYO CLINIC, ROCHESTER, MINNESOTA	SEPTEMBER 28-30,1950
SHAMROCK HOTEL, HOUSTON, TEXAS	OCTOBER 4-6,1951
WALDORF-ASTORIA HOTEL NEW YORK CITY	SEPTEMBER 29-OCTOBER 1,1952
BILTMORE HOTEL,SANTA BARBARA, CALIFORNIA	OCTOBER 12-14,1953
BROADMOOR HOTEL, COLORADO SPRINGS, COLORADO	OCTOBER 21-23,1954
THE HOMESTEAD, HOT SPRINGS, VIRGINIA	OCTOBER 27-29,1955
CAMELBACK INN, PHOENIX, ARIZONA	NOVEMBER 8-10,1956
THE CLOISTER, SEA ISLAND, GEORGIA	NOVEMBER 11-13,1957
THE ROYAL YORK HOTEL, TORONTO, CANADA	NOVEMBER 6-8,1958
DEL MONTE LODGE, PEBBLE BEACH, CALIFORNIA	OCTOBER 18-21,1959
COPLEY SHERATON PLAZA,BOSTON, MASSACHUSETTS	OCTOBER 5-8,1960
ROYAL ORLEANS, NEW ORLEANS,LOUISIANA	NOVEMBER 7-10,1962
EL MIRADOR,PALM SPRINGS, CALIFORNIA	OCTOBER 23-26,1963
THE KEY BISCAYNE, MIAMI, FLORIDA	NOVEMBER 11-14,1964
TERRACE HILTON HOTEL, CINCINNATI, OHIO	OCTOBER 14-16,1965
FAIRMONT HOTEL & TOWERS, SAN FRANCISCO, CALIFORNIA	OCTOBER 17-19,1966
THE KEY BISCAYNE, MIAMI, FLORIDA	NOVEMBER 8-11,1967
BROADMOOR HOTEL, COLORADO SPRINGS, COLORADO	OCTOBER 6-8,1968
ST. REGIS HOTEL, NEW YORK CITY	SEPTEMBER 21, 1969
CAMINO REAL 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
AMELIA ISLAND PLANTATION AMELIA ISLAND,FL	OCTOBER 2-7,1990

MEMBERSHIP LIST 1991
AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

HONORARY MEMBERS (5)	ELECTED
GUY LAZORTHES 26 Rue D Auriol 31 Toulouse, France	1973
VALENTINE LOGUE (Anne) 16 Rowan Road Hammersmith London W6 7DU England	1974

GOSTA NORLEN (Gunvor) 1973
Linnegaten 35 IV
11447 Stockholm, Sweden

BERNARD PERTUISET(Francoise) 1986
Hopital de la Pitie
83 Boulevard de l'Hopital
75651 Paris, Cedex 13
France

KEIJI SANO (Yaeko) 1975
Teikyo University Hospital
2-11-1 Kaga Itabashi-ku
Tokyo 173, Japan

SENIOR MEMBERS (60)	ELECTED
EBEN ALEXANDER, JR. (Betty) Bowman Gray School of Medicine 300 South Hawthorne Winston-Salem, North Carolina 27103	1950
GEORGE S. BAKER (Enid) 607 Litchfield Road Litchfield Park, Arizona 85340	1940
H. THOMAS BALLANTINE, JR. (Elizabeth) Massachusetts General Hospital 15 Parkman Street - ACC 312 Boston, Massachusetts 02114	1951
E. HARRY BOTTERELL (Margaret) 2 Lakeshore Boulevard Kingston, Ontario, Canada K7M 4J6	1938
HARVEY CHENAULT (Billie) 6340 Briar Hill Road Paris, Kentucky 40361	1949
SHELLEY N. CHOU (Jolene) University of Minnesota Medical Center 420 Delaware Street S. E., Box 96 Minneapolis, Minnesota 55455	1974
GALE G. CLARK 12621 Brookpark Road Oakland, California 94619	1970
W. KEMP CLARK (Fern) 5323 Harry Hines Boulevard Dallas, Texas 75235	1970
WILLIAM F. COLLINS, JR. (Gwendolyn) Yale University School of Medicine 333 Cedar Street New Haven, Connecticut 06510	1963

COURTLAND H. DAVIS, JR. (Carolyn) 1967
Bowman-Gray School of Medicine
Winston-Salem, North Carolina 27103

RICHARD DESAUSSURE, JR. (Phyllis) 1962
920 Madison Avenue
Memphis, Tennessee 38103

DONALD F. DOHN (Carolyn) 1968
Cleveland Clinic Florida
3000 West Cypress Creek Road
Ft. Lauderdale, Florida 33309

R.M.PEARDON DONAGHY (Francis) 1970
P.O. BOX 5035, Road 1
Horn of the Moon Road
Montpelier, Vermont 05602

CHARLES G. DRAKE (Ruth) 1958
University Hospital
London, Ontario, Canada N6A 5A5

DEANE H. ECHOLS (Fran) Founder
1550 Second Street
New Orleans, Louisiana 70130

ROBERT FISHER (Connie) 1957
Box 846, Star Rt. 1
Bristol, New Hampshire 03222

ELDON L. FOLTZ (Catherine) 1960
UCI Medical Center
Division of Neurosurgery
101 City Drive, South
Orange, California 92668

LYLE A FRENCH (Gene) 1954
P.O. Box 1007
Pauma Valley, California 92061

WILLIAM F. MEACHAM (Alice) 1952
539 Medical Arts Bldg.
Nashville, Tennessee 37212

FRANCIS MURPHEY (Marge) Founder
3951 Gulf Shore Blvd. North
Naples, Florida 33940

BLAINE S. NASHOLD, JR., (Irene) 1967
Duke University Medical Center
Durham, North Carolina 27710

GUY L. ODOM (Mataline) 1946
2812 Chelsea Circle
Durham, North Carolina 27707

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

J. LAWRENCE POOL (Angeline) 1940
P.O. Box 40
West Cornwall, Connecticut 06796

ROBERT W. PORTER (Aubrey Dean) 1962
6461 Bixby Hill Road
Long Beach, California 90815

ROBERT H. PUDENZ (Rita) 1943
574 Garfield Avenue
South Pasadena, California 91030

JOHN RAAF(Lorene) Founder
1120 N.W. 20th Avenue, #100
Portland, Oregon 97209

AIDEN A. RANEY (Mary) 1946
2010 Wilshire Boulevard
Los Angeles, California 90057

B. RAMAMURTHI (Indira) 1966
2nd Main Road G.I.T. Colony
Madras 4, India 600 004

JOSEPH RANSOHOFF II (Lori) 1965
New York University Medical Center
550 First Avenue
New York, New York 10016

THEODORE B. RASMUSSEN (Catherine) 1947
29 Surrey Drive
Montreal, Quebec, Canada H3P 1B2

HENRY G. SCHWARTZ (Reedie) 1942
Washington University School of Medicine
4901 Barnes Hospital Plaza
St. Louis, Missouri 63110

C. HUNTER SHELDEN (Elizabeth) 1941
Huntington Medical Research Institute
10 Pico Street
Pasadena, California 91105

ANTHONY SUSEN (Phyllis) 1965
504 Remora Circle
Fripp Island, South Carolina 29220

WILLIAM H. SWEET (Elizabeth) 1950
309 Goddard Avenue
Brookline, Massachusetts 02146

JOHN TYTUS (Virginia) 1967
P.O. Box 279
Arlington, Washington 98223

ALFRED UIHLEIN (Ione) 1949
P.O. Box 765
Vail, Colorado 81658

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

ARTHUR A. WARD,JR. (Janet) 1953
4001 N.E. Belvoir Place
Seattle, Washington 98105

EXUM WALKER (Nellie) 1938
490 Peachtree Street, N.E.
Atlanta, Georgia 30308

W. KEASLEY WELCH (Elizabeth) 1957
25 Gould Road
Waban, MA 02168

BENJAMIN B. WHITCOMB (Margaret) 1947
RFD Box 124
Surrey, Maine 04684

LOWELL E. WHITE, JR. (Margie) 1971
University of South Alabama
Division of Neuroscience
Mobile, Alabama 36688

ACTIVE MEMBERS (93)	ELECTED
MICHAEL J. APUZZO (Helene) 1200 N. State Street Los Angeles, California 90030	1988
JAMES I. AUSMAN (Carolyn) Henry Ford Hospital 2799 West Grand Boulevard Detroit, Michigan 48202	1978
DONALD P.BECKER (Marie) UCLA Medical Center Department of Neurosurgery Rm 74-140 Chs 405 Hilgard Los Angeses, California 90024	1990
GILLES BERTRAND (Louise) Montreal Neurological Institute 3801 University Street Montreal, Quebec, Canada H3A 1B4	1967
PETER M. BLACK (Katherine) Brigham and Women's Hospital 75 Francis Street Boston MA 02115	1988
ROBERT S. BOURKE (Marlene) 5802 Nicholson Lane Rockville, Maryland 20852-2967	1983
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) 1984
7777 Forrest Lane #C703
Dallas, Texas 75230

WILLIAM A. BUCHHEIT 1980
3401 North Broad Street
Philadelphia, Pennsylvania 19140

WILLIAM F. CHANDLER (Sue) 1989
2124D/338 Taubman Center
1500 East Medical Ctr. Drive
Ann Arbor MI 48109

PAUL H. CHAPMAN (Tansy) 1983
Department of Neurosurgery
Massachusetts General Hospital
Boston, Massachusetts 02114

EDWARD S. CONNOLLY (Elise) 1973
Ochsner Clinic
1514 Jefferson Highway
New Orleans, Louisiana 70121

JAMES W. CORRELL (Cynthia) 1966
710 West 168th Street
New York, New York 10032

ROBERT .CROWELL(Mary) 1990
Massachusetts General Hospital
Chief, Cerebrovascular Section
Department of Neurosurgery/ACC 312
Boston, Massachusetts 02114

RALPH G. DACEY(Corinne) 1990
Washington School of Medicine
Division of Neurosurgery
Barnes Hospital Plaza
St. Louis, Missouri 63110

ARTHUR L. DAY (Dana) 1990
University of Florida Health Ctr
Department of Neurosurgery
Box J 265
Gainesville, Florida 32610

STEWART B. DUNSKER (Ellen) 1975
Mayfield Neurological Institute
506 Oak Street
Cincinnati, Ohio 45219

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
3400 Spruce Street
Philadelphia,Pennsylvania 19104

RICHARD A.R. FRASER (Sarah Anne) 1976
525 East 68th Street
New York, New York 10021

JOHN T. GARNER (Candace) 1971
50 Allesandro Place
Suite 400
Pasadena, California 91105

HENRY GARRETSON (Marianna) 1973
Health Sciences Center
316 MDR Bldg.
University of Louisville
Louisville, Kentucky 40292

ROBERT G. GROSSMAN (Ellin) 1984
Baylor College of Medicine
One Baylor Place
Houston, Texas 77030

ROBERT GRUBB (Julia) 1985
Washington University School of Medicine
4901 Barnes Hospital Plaza
St. Louis, Missouri 63110

GRIFFITH R. HARSH, III (Craig) 1980
Division of Neurosurgery
UBA Station
Birmingham, Alabama 35294

MARK PETER HEILBRUN (Robyn) 1984
Division of Neurosurgery #3B320
University of Utah Medical Center
50 North Medical Drive
Salt Lake City, Utah 84132

E. BRUCE HENDRICK (Gloria) 1968
Hospital for Sick Children
555 University Avenue, Room 1502
Toronto, Ontario, Canada M5G 1X8

ROBERTO C. HEROS (Deborah) 1985
University of Minnesota Medical Center
420 Southwest Delaware Street Box 96
Minneapolis, MN 55455

CHARLES HODGE (Linda) 1982
750 East Adams Street
Syracuse, New York 13210

JULIAN HOFF (Diane) 1975
Department of Neurosurgery
University of Michigan
Ann Arbor, Michigan 48109

HAROLD HOFFMAN (Jo Ann) 1982
The Hospital for Sick Children
Suite 1502, 555 University Avenue
Toronto, Ontario, Canada M5G 1X8

EDGAR M. HOUSEPIAN (Marion) 1976
The Neurological Institute
710 West 168th Street
New York, New York 10032

ALAN R. HUDSON (Susan) 1978
St. Michael's Hospital
38 Shuter Street
Toronto, Ontario, Canada M5B 1A6

JOHN A. JANE (Noella) 1982
Department of Neurosurgery, Box 212
University of Virginia
Charlottesville, Virginia 22908

ELLIS B. KEENER (Ann) 1978
915 East Lake Drive, N.W.
Gainesville, Georgia 30506

DAVID KELLY, JR. (Sally) 1975
Bowman Gray School of Medicine
300 S. Hawthorne
Winston-Salem, North Carolina 27103

GLENN W. KINDT (Charlotte) 1977
Division of Neurosurgery
Box C-307
University of Colorado Medical Center
4200 East 9th Avenue
Denver, Colorado 80262

WOLFF M. KIRSCH (Marie-Claire) 1971
Chief of Neurosurgery
Univ. of New Mexico Medical School
Albuquerque, New Mexico 87131

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

RICHARDB.MORAWETZ (MaryJean) 1990
Division of Neurosurgery
University Station
Birmingham, Alabama 35294

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
1501 N. W. 9th Avenue
Miami, Florida 33136

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

DAVID G. PIEPGRAS (Jane) 1987
Mayo Clinic
200 First Street, S.W.
Rochester, Minnesota 55905

DONALD O. QUEST (Ilona) 1968
The Neurological Institute
710 West 168th Street
New York, New York 10032

ROBERT A. RATCHESON (Peggy) 1986
University Hospital
2074 Abington Road
Cleveland, Ohio 44106

ALBERT L. RHOTON, JR. (Joyce) 1984
University of Florida, Box J265
Department of Neurosurgery
Gainesville, Florida 32610

J. CHARLES RICH, JR. (Jasmine) 1987
324 10th Avenue #206
Salt Lake City, Utah 84103

HUGO RIZZOLI (Helen) 1973
2150 Pennsylvania Avenue, N.W.
Washington, D.C. 20037

THEODORE S. ROBERTS (Joan) 1976
Department of Neurological Surgery
University Hospital
1959 Pacific Avenue, N.E., RI 20
Seattle, Washington 98195

JAMES T. ROBERTSON (Valeria) 1971
Department of Neurosurgery
University of Tennessee, Memphis
956 Court Avenue
Memphis, Tennessee 38163

WILLIAM SHUCART (Laura) 1989
New England Medical Ctr. #178
750 Washington Street
Boston MA 04401

FREDERICK A. SIMEONE (Kate) 1981
Pennsylvania Hospital
800 Spruce Street
Philadelphia, Pennsylvania 19107

JAMES C. SIMMONS (Vanita) 1975
920 Madison Avenue, 201-N
Memphis, Tennessee 38103

KENNETH R. SMITH, JR. (Marjorie) 1987
St. Louis University Hospital
3635 Vista Avenue
St. Louis, Missouri 63110-2500

ROBERT R. SMITH (Helen) 1989
University of Mississippi Medical Ctr.
Department of Neurosurgery
Jackson MS 39216

DENNIS SPENCER (Susan) 1989
333 Cedar Street
New Haven CT 06510

BENNETT M. STEIN (Bonita) 1970
710 West 168th street
New York, New York 10032

JIM L. STORY (Joanne) 1972
Division of Neurosurgery
The University of Texas Health Science Ctr
7703 Floyd Curl Drive
San Antonio, Texas 78284-7843

THORALF M. SUNDT, JR. (Lois) 1971
Department of Neurosurgery
Mayo Clinic
Rochester, Minnesota 55905

RONALD R. TASKER (Mary) 1971
Toronto General Hospital
Room 215, 14th Floor
200 Elizabeth Street
Toronto, Ontario, Canada M5G 2C4

JOHN TEW, JR. (Susan) 1973
506 Oak Street
Cincinnati, Ohio 45219

GEORGE TINDALL (Suzie) 1968
Emory University School of Medicine
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NICHOLAS T. ZERVAS (Thalia)
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SENIOR CORRESPONDING MEMBERS (10)

JEAN BRIHAYE (Martine Van Geertruyden) 1975
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KARL AUGUST BUSHE (Eva) 1971
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JOHN HANKINSON (Nicki) 1973
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KATSUTOSHI KITAMURA (Yoshiko) 1970
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KRISTIAN KRISTIANSEN (Brit) 1962
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NOEL G. DAN 1989
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JACQUES DEVILLIERS (Jeanne Marie) 1986
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JOHN F. GILLINGHAM (Judy) 1962
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JAMIE G. GOMEZ (Lucy) 1975
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SALVADOR GONZALEZ-CORNEJO (Rosalie) 1982
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ERNEST H. GROTE (Julie) 1984
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FRANK MARGUTH 1978
Director, Department of Neurochirurgischen
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PAUL MARINO, JR. (Milu) 1977
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KJELD VAERNET (Ann) 1970
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SIDNEY WATKINS 1975
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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

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
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**
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
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
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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