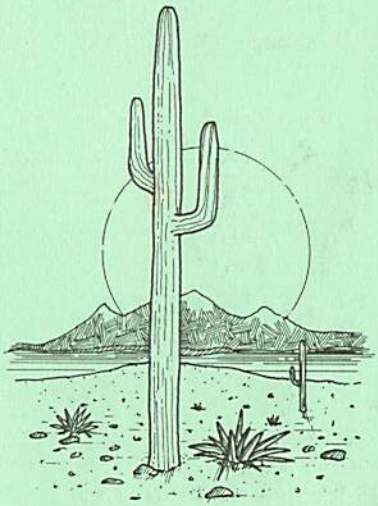
THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY



Loews
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RESORT
TUCSON · ARIZONA
1989

THE 51ST ANNUAL MEETING OF

The
American Academy of Neurological Surgery

Loew's Ventana Canyon Resort
Tucson, Arizona

September 27-October 1, 1989

1989 Officers and Committees

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The American Academy of Neurological Surgery

September 27 – October 1, 1989

Loew's Ventana Canyon Resort
Tucson, Arizona

Wednesday, September 27, 1989

1:00 PM-5:30 PM	Registration *Ballroom Foyer*
1:30 PM-4:30 PM	Executive Committee Meeting *President's Suite*
7:00 PM-9:00 PM	Welcoming Reception *Catalina Ballroom*

Thursday, September 28, 1989

7:00 AM-8:00 AM	Breakfast Business Meeting (Members only) *Catalina Ballroom*
8:00 AM-5:00 PM	Registration *Catalina Ballroom*
8:00 AM-1:00 PM	Scientific Meeting *Salon B (Grand Ballroom)*
Afternoon Free	
6:15 PM	Buses depart for Galleries of the Foothills and Hidden Valley Inn *Grand Ballroom Foyer Entrance*

Friday, September 29, 1989

7:00 AM-8:00 AM	Breakfast Business Meeting (Members only) *Salon C (Grand Ballroom)*

8:00 AM-5:00 PM	Registration *Ballroom Foyer*
8:00 AM-1:00 PM	Scientific Meeting *Salon B (Grand Ballroom)*
12:45 PM	Bus departs for Biosphere II Luncheon at site *Grand Ballroom Foyer Entrance*
1:00 PM	Bus departs for Desert Museum Lunch Provided *Grand Ballroom Foyer Entrance*
6:30 PM	Margaritas, etc. on the Patio Poolside at Bill's Grill
7:30 PM	Mexican Fiesta on the Patio at Bill's Grill

Saturday, September 30, 1989

7:00 AM-8:00 AM	Breakfast meeting (Members and guests) *Salon C (Grand Ballroom)*
8:00 AM-1:00 PM	Scientific Meeting *Salon B (Grand Ballroom)*
9:40 AM	Presidential Address Thoralf M. Sundt, Jr. *Grand Ballroom*
1:00 PM	Golf and Tennis Tournaments *Lakeside Golf Shop and Tennis Courts area*
7:00 PM-8:00 PM	Annual Reception *Ballroom Foyer*
8:00 PM	Crystal Banquet Guest speaker: Colonel Frank Borman Dancing *Grand Ballroom (Black Tie)*

Sunday, October 1, 1989 — Travel Day

Spouses Activities

Wednesday, September 27, 1989

7:00 PM-9:00 PM	Welcoming Cocktail Reception *Catalina Ballroom*

Thursday, September 28, 1989

8:00 AM-4:00 PM	Spouses Hospitality *Suite 2205*
8:00 AM-9:00 AM	Coffee, etc.
9:00 AM	Introduction to Indian Arts by Paul and Sondra Buck *Suite 2205*
9:45 AM	Buses to Kaibab Shop, Desert House Complex *Grand Ballroom Foyer Entrance*
6:15 PM	Depart by bus for Galleries on Campbell Cocktails followed by bus to Hidden Valley Inn for dinner *Grand Ballroom Foyer Entrance*

Friday, September 29, 1989

8:00 AM-4:00 PM	Spouses Hospitality *Suite 2205*
AM	Round Robin Tennis arranged by Molly King *Tennis Courts*
12:45 PM-5:30 PM	Depart by bus for Biosphere II* Luncheon at the site *Grand Ballroom Foyer Entrance*
1:00 PM-4:30 PM	Depart by bus for Desert Museum* Box lunches provided *Grand Ballroom Foyer Entrance*
6:30 PM	Margaritas, etc. *Bill's Grill*
7:30 PM	Mexican Fiesta *Bill's Grill*

Saturday, September 30, 1989

8:00 AM-4:00 PM	Spouses Hospitality *Suite 2205*
1:00 PM	Golf tourney — John Van Gilder,* Coordinator *Lakeside Golf Shop*
2:00 PM-5:00 PM	Tennis — Susan and John Tew, Coordinators *Tennis Courts*
7:00 PM	Cocktails *Ballroom Foyer*
8:00 PM	Crystal Banquet Guest Speaker: Colonel Frank Borman Dancing to the Ventana Trio *Grand Ballroom (Black Tie)*

Sunday, October 1, 1989 — Travel Day

*** The members' scientific sessions will conclude early to allow time to report for afternoon activities.**

Thursday, September 28, 1989

SCIENTIFIC SESSION I

Julian T. Hoff—Moderator
Chairman, Program Committee

8:00 a.m. **WELCOME**

Thoralf Sundt, Jr.
President

8:05 a.m. **Surgery and Follow-up of Intramedullary Ependymomas.**
Bennett Stein, Paul McCormick; New York City, NY

8:25 a.m. **Characteristics and Biological Role of Steroid Hormone Receptors in Human Glial Tumors.**
Pietro Paoletti, G. Butti, N. Gibelli, L. Magrassi, G. Robustelli della Cuna, C. Zibera; Pavia, Italy

8:45 a.m. **Colloid Cysts—Experience with Management of 74 Cases in the Post-CT Era.**
Edward R. Laws, Jr., A. Camacho, P. Kelly; Rochester, MN

9:05 a.m. **Combined Intralesional Immunotherapy Against CNS Neoplasia in Mice.**
Frances Conley, J.A. Duncan, J.R. Adler, J.N. Kennedy, R.C. Sutton; Palo Alto, CA

9:25 a.m. **Infratentorial Ependymomas in Childhood.**
Harold Hoffman, G.B. Nazar, L.E. Becker, D. Jenkins, R.P. Humphreys, E.B. Hendrick; Toronto, Ontario, Canada

Thursday, September 28, 1989 (cont'd).

9:45 a.m. **Autocrine Growth in Astrocytomas: The Role of PDGF.**
Peter McL. Black, H. Antoniades, M. Maxwell; Boston, MA

10:05 a.m. **Efficacy of the Anti-Progestational Agent RV486 in the Treatment of Meningiomas.**
S.M. Grunberg, I. Spitz, L.L. Stevenson, M.H. Weiss; Los Angeles, CA

10:25 a.m. **Coffee**

SCIENTIFIC SESSION II
Robert Grossman—Moderator
Chairman, Award Committee

10:50 a.m. **ACADEMY AWARD PRESENTATION**

11:20 a.m. **Age-Related Changes in Final Feeding Arteries of Arteriovenous Malformations.**
Henry Garretson; Louisville, KY

11:40 a.m. **Dural Sinus AV Fistulae, Congenital and Acquired.**
Sean Mullan; Chicago, IL

12:00 noon **The Spectrum of Dural Arteriovenous Fistulas Excluding Those That Involve the Cavernous Sinus.**
Charles B. Wilson, Grant Hieshima; San Francisco, CA

12:20 p.m. **Seizure Disorder Relative to Surgical Treatment of Arteriovenous Malformations.**
David G. Piepgras, Thoralf M. Sundt, Jr., Lorna P Stevens; Rochester, MN

Thursday, September 28, 1989 (cont'd).

12:40 p.m. **Results of Surgical Treatment of Cluster Headache; Initial Relief Followed by Recurrence.**
Robert H. Wilkins, Joel C. Morgenlander; Durham, NC

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

Friday, September 29, 1989

SCIENTIFIC SESSION III
Stewart Dunsker—Moderator
Program Committee

8:00 a.m. **A New Stereotactic Alignment System for Proton Beam Therapy.**
Paul Chapman, C. Ogilvy, L.J. Verhey, N.T. Zervas; Boston, MA

8:20 a.m. **Functional Stereotactic Neurosurgery with MRI and Neurophysiological Guidance.**
Ronald F. Young, P. Rinaldi, S. Bloomfield, D. Albe-Fessard; Irvine, CA

8:40 a.m. **Stereotactic Radiosurgery: The University of Florida System.**
William A. Friedman, F.J. Bove, A.L. Rhoton, Jr.; Gainesville, FL

9:00 a.m. **Utility of MRI Compatible Subdural Electrode Arrays in the Evaluation and Surgical Management of Patients with Epilepsy and Intracranial Tumors.**
Robert E. Maxwell, M.E. Fiol, J.R. Gates; Minneapolis, MN

9:20 a.m. **The Significance of Limbic Structure Removal in the Surgery of Temporal Lobe Epilepsy, Based on Reoperations.**
Andre Olivier, T. Tanaka, F. Andermann; Montreal, Quebec, Canada

9:40 a.m. **Intraoperative Motor Pathway Monitoring with tcMMEP (Transcranial Magnetic Motor Evoked Potentials).**
Christopher Shields, R.D. Linden, H.L. Edmonds, Jr., J.R. Johnson, H.D. Garretson; Louisville, KY

Friday, September, 29, 1989 (cont'd).

10:00 a.m. **INVITED PRESENTATION**

"Molecular Genetics, Cell Biology, and the Neurosurgeon."
Robert Martuza, Assoc. Professor of Surgery (Neurosurgery), Harvard Medical School; Boston, MA

10:30 a.m. **Coffee**

SCIENTIFIC SESSION IV
Jim Story—Moderator
Vice President

10:50 a.m. **Posterior Fusions at the Craniovertebral Junction.**
John C. VanGilder; Iowa City, IA

11:10 a.m. **Experimental and Clinical Results of Low Dose Rate Iodine-125 Permanent Implants for the Treatment of Malignant Gliomas.**
J. A. Winfield, G. King, T.J. Watt; Syracuse, NY

11:30 a.m. **Chemotherapy with Osmotic Blood Brain Barrier Disruption for the Treatment of High Grade Gliomas.**
Clark Watts, M.K. Gumerlock; Columbia, MO

11:50 a.m. **Determination of Brain Tumor Patient Response to Therapy Using Volumetric Microcomputer CT Scan Analysis.**
M.S. Mahaley, Jr.; Birmingham, AL

12:10 a.m. **Radiographic and Pathological Assessment of Gangliogliomas with Surgical Outcome.**
M. Khayata, R.A.R. Fraser, S. Erde, L. Heier; New York City, NY

12:30 p.m. **Complications of Posterior Fossa Surgery.**
William Buchheit; Philadelphia, PA

12:50 p.m. **Adjourn**

SCIENTIFIC SESSION V
Roberto Heros—Moderator
Program Committee

8:00 a.m. **An Interfascicular Approach to 110 Neurofibromas.**
David G. Kline, Carlos Garcia, Rand Voorhies; New Orleans, LA

8:20 a.m. **Arginine Vasopressin (AVP) in Triphasic Diabetes Insipidus.**
Paul B. Nelson; Pittsburgh, PA

8:40 a.m. **The DREZ Operation: An Update on Current Technique.**
Blaine Nashold; Durham, NC

9:00 a.m. **Memory Deficits in Patients with Aneurysms.**
William Shucart; Boston, MA

9:20 a.m. **Stroke: A History**
Donald Quest; New York City, NY

9:40 a.m. **PRESIDENTIAL ADDRESS**
(Introduction by Jim L. Story)
The Roots of the Mayo Clinic
Thoralf Sundt, Jr.

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

SCIENTIFIC SESSION VI
Thoralf Sundt, Jr.—Moderator

10:40 a.m. **FUTURE DIRECTIONS IN CEREBROVASCULAR SURGERY: A SYMPOSIUM**

Extracranial Vascular Disease.
James Robertson

Aneurysms
Charles Drake

Focal Ischemia and Flow Augmentation
Robert Grubb

Arteriovenous Malformations
Roberto Heros

Discussion

12:40 p.m. **Adjourn**

Thursday, September 28

8:05 a.m.

1

Surgery and Follow-up of Intramedullary Ependymomas

Bennett M. Stein, Paul McCormick; New York City, NY

We have reviewed our experience with 23 intramedullary ependymomas all totally removed. The average follow-up is over five years and 7 patients have been followed over ten years. In no case, where the tumor has been totally removed, which involves all 23 cases, has there been any evidence of clinical recurrence. With the increased contemporary use of MRI scanning, MRI scan follow-up has been carried out on a number of long-term follow-ups and there has been no evidence of recurrent tumor on these scans.

Briefly mentioned will be the techniques of removal of intramedullary ependymomas, the preoperative clinical syndromes, the complications of operative procedures, postoperative deficits and the follow-up basis which now includes MRI and gadolinium MRI.

It is felt that this long-term follow-up of cases treated purely by surgery is important in the context of a number of articles in the literature suggesting biopsy and radiotherapy as the treatment of ependymoma. The follow-up of those particular papers will be compared with this surgical series.

8:25 a.m.

2

Characteristics and Biological Role of Steroid Hormone Receptors in Human Glial Tumors

P. Paoletti, G. Butti, N. Gibelli, L. Magrassi, G. Robustelli della Cuna, C. Zibera; Pavia, Italy

The role of steroid hormone receptors (SR) has become extremely important in the choice of the treatment modality of some tumors. Recently, the presence of SR has been demonstrated particularly in meningiomas. Glial tumors have been thought to contain few SR but data reported in literature are scanty and controversial. Until this moment, no data on the possible biological role of these receptors in glial tumors have been available.

We determined the content on glucocorticoid (GR), estrogen (ER) progesterone (PR) and androgen (AR) receptors in 25 glioblastomas, 18 anaplastic astrocytomas, and 14 astrocytomas. GR and AR were present in 38.6% and 21.6% of the cases respectively (positively) > 10 fmol/mg. cystosol protein). ER and AR were present in less than 10% of the cases. GR percentage was

higher in astrocytomas while the AR percentage was higher in glioblastomas.

In order to evaluate the possible effects of GR and AR on tumor growth, we studied the influence of scaler doses (from 50 to 0.016 ug/ml) of dexamethasone (for GR) and testerone (for AR) on glial tumor cell growth in 10 cultures.

Dexamethasone induced a cell growth inhibition at the higher doses both in GR positive or negative cultures. Low doses caused a significant stimulation of the cell growth in 4 out of 5 GR positive cultures, while no effect was seen in the negative ones.

Testerone induced a marked significant inhibition of cell growth at the higher diseases in all cultures (all AR negative) while no effects were noted at lower doses.

This study is supported by the grant n. 88.00552.44 from the "Consiglio Nazionale delle Ricerche", Italy.

8:45 a.m.
3
Colloid Cysts-Experience wih Management of 74 Cases in the Post CT Era.

Edward R. Laws, Jr., Arturo Camacho, Patrick Kelly; Rochester, MN

Colloid cysts of the third ventricle often go undetected for prolonged periods of time because of their nonspecific early symptoms. This is a retrospective review of cases diagnosed during the years 1974-1986. In this 12 year period, 84 patients (45 men, 39 women) had a colloid cyst diagnosed. The mean age was 46 (7-82); all patients had CT scans and 11 had MRI.

Surgery was performed in 55 patients, 7 of whom had undergone prior surgery elsewhere. Surgical approaches utilized were: transfrontal-transventricular—43 (7 computer-assisted); transcallosal—2; shunt procedures—10. There was no operative mortality, but some complication occurred in 15. These included malfunctions and infections in patients shunted or operated elsewhere, 5 patients with seizures, 2 with hydrocephalus and one with CSF rhinorrhea. At last follow-up (1-140 mos.) all but one who (died in an auto accident) are alive, and only two are severely disabled as a result of the lesion and attempts at treatment.

In the surgically treated group, the most common presenting symptoms were: headache (42), change in mental status (18), ataxia (16), nausea and vomiting (13), visual disturbance (7), emotional lability/inappropriate affect (5), depersonalization (3),

hypersomnolence (3). Colloid cysts were discovered at autopsy in 5 patients.

No surgery was recommended for 24 of the 84 patients and they are being followed closely. Most of these patients (71%) had normal ventricles.

Direct removal of colloid cysts can be accomplished with low mortality and morbidity, avoiding the frequent revisions and complications related to shunt procedures. Noteworthy in patients with normal ventricles is the association of colloid cysts with anxiety and panic attacks.

9:05 a.m.
4
Combined Intralesional Immunotherapy Against CNS Neoplasia in Mice.

F.K. Conley, J.A. Duncan, J.R. Adler, J.N. Kennedy, R.C. Sutton; Palo Alto, CA

Recent research in our laboratory has utilized a non-replication agent to create an antitumor inflammatory stimulus in the CNS. Report here a series of recent experiments with two known immunostimulants, Corynecbacterium parvum (C.P.) and interleukin 2 (rIL-2) which were used in sequential intralesional therapy directed against implanted brain tumors in mice.

The KHT sarcoma is syngeneic for the C3H mouse, is highly malignant and nonimmunogenic. An intracerebral inoculum of 10,000 cells reliably kills untreated mice in a reproducible, predictable manner. We have previously determined that tumor-bearing mice which receive a systemic priming dose followed by an intracranial injection of C.P. into the tumor site develop a marked inflammatory response in the brain and have significantly prolonged survival compared to control mice. 10-20% of mice are cured of their brain tumors. We also know that rIL-2 is neurotoxic in its free form when injected intracerebrally and that mixing rIL-2 with 3% collagen eliminates neurotoxicity. rIL-2 in either form provides no protection against tumor growth when used as single agent therapy.

All mice were inoculated with tumor on day 0 and randomly divided into experimental groups of 12-18 animals. On day 1 three groups received 350 ugm C.P. i.p. and on day 4, 70 ugm C.P. intracranially (i.c.); the fourth group was treated with saline. On day 6, the first group received 60,000 units of rIL-2 in 3% collagen, and the second group 60,000 units of free rIL-2 i.c. in the same site as the tumor and C.P. inoculations. The third and fourth groups received i.c. saline. Mice were followed to death

or sacrificed at 75 days, and all brains were studied histologically.

The survival curves from 6 different experiments have been identical. All treated mice lived significantly longer than control mice, the addition of rIL-2 to C.P. treatment significantly increased protection against growth of tumor above that provided by C.P. alone. More importantly the cure rate increased from 10-20% to 60-75% in mice treated sequentially with both immunostimulating agents.

These results will be discussed relative to studies from our laboratory demonstrating active division of inflammatory cells throughout the brain, as well as therapeutic implications in current human trials against glioblastoma.

9:25 a.m.
5
Infratentorial Ependymomas in Childhood

H.J. Hoffman, G.B. Nazar, L.E. Becker, D. Jenkins, R.P. Humphreys, E.B. Hendrick; Toronto, Ontario, Canada

The prognostic factors and survival data for 35 children with surgically treated childhood infratentorial (IT) ependymomas at the Hospital for Sick Children in Toronto treated during the years 1970-1987, were analysed. Tumor histology was reviewed individually and grouped into three categories (I-III) for survival analysis. An overall 5 year survival of 44.6% was obtained after the exclusion of peri-operative mortality. Factors which were associated with an improved five year survival were total tumor removal, non-invasive tumors, category I histology, age greater than 6 years, and absent physical signs of parenchymal invasion or lower cranial nerve involvement. Five year survivals were worse when associated with category III histology, brainstem or focal cerebellar signs, age less than 2 years, tumor invasion and/or cranial nerve involvement, and subtotal tumor removal. Clinical evidence of spinal metastases was found to be uncommon (2.9%). Surgical excision followed by radiation therapy was the primary mode of treatment for these tumors. Controversies regarding the volume of radiotherapy to be delivered and the use of adjuvant chemotherapy are discussed.

Thursday, September 28

9:45 a.m.

6

Autocrine Growth in Astrocytomas: The Role of PDGF

P. McL Black, H. Antoniades, M. Maxwell; Boston, MA

Self-stimulation by secreted growth factors has been suggested as one mechanism for the formation of human astrocytomas. We present evidence that the conditions for such growth exist through PDGF-2 and its congeners. PDGF-2 is an important mitogen for human cells; a major advance in tumor biology was the realization that the sequence coding PDGF-2 and the sison-cogene were homologous.

Normal astrocytes appear to express PDGF-R. We studied the expression of PDGF related gene sequences in 29 anaplastic astrocytomas or glioblastoma multiforme taken from the OR. All tumor specimens were frozen in liquid nitrogen in the operating room. Northern blot analysis was used to establish the expression of PDGF-l, PDGF-2 and PDGF-R. Three specimens of brain tissue from patients without neoplasm were used as controls.

All tumor tissue expressed the c-sis/PDGF-2 oncogene; none of the non-neoplastic tissue did so. Twenty-seven out of twenty-nine astrocytomas also expressed PDGF-1. The PDGF receptor was expressed both by neoplastic tissue and by non-neoplastic glial cells. In situ hybridization techniques with GFAP counter-staining established that expression was, in fact, in astrocytomas.

Expression of the PDGF-2/sis sequence in neoplasia may be accompanied by activation of this receptor and initiation of mitogenesis by an autocrine mechanism. This mechanism is present in astrocyte tissue and is not an artifact of cell culture.

10:05 a.m.

7

Efficacy of the Anti-Progestational Agent RU486 in the Treatment of Meningioma

S.M. Grunberg, I. Spitz, L.L. Stevenson, M.H. Weiss, Los Angeles, CA

Epidemiologic observations first suggested that sex hormones can affect meningioma growth. The finding that a high percentage of meningiomas are progesterone receptor positive even in the absence of estrogen receptors provides a mechanism for this effect. We therefore initiated a study of the antiprogestational agent RU486 200 mg po qd for the treatment of unresectable meningioma. Fourteen patients have not been treated for a median of 9 months (range 2-19+). The study group includes 6 males, 2 pre-menopausal females, and 6 post-menopausal

females. Performance Status is 90% (range 60-100). Histologies include 9 meningothelial, 2 fibrous, 1 malignant, and 2 unbiopsied. Toxicity has been mild and has included gynecomastia (3/6 male) , hot flashes (2/6 male), cessation of menstrual periods (2/2 pre-menstrual female), and fatigue (5/14 patients). Three of the 10 patients presently considered evaluable response have responded. One male had minor shrinkage of meningioma on CT scan and improvement in extraocular muscle function, a second male had improvement in extraocular muscle function and visual fields, and one pre-menopausal female had minor shrinkage of meningioma on MRI scan and disappearance of occipital headache. The first male responder experienced gynecomastia and hot flashes, the second male responder experienced gynecomastia, and the female responder noted cessation of menstrual periods. Long-term treatment with careful endocrinologic monitoring continues. RU486 is a practical agent for long-term treatment of unresectable meningioma and appears to have efficacy against this disease.

11:20 a.m.
8
Age Related Changes in Final Feeding Arteries of Arteriovenous Malformations
Henry Garretson; Louisville, KY

Postoperative brain swelling and attendant morbidity after excision of arteriovenous malformations (AVMs) has been ascribed to a loss of "autoregulation" in the proximal feeding arteries. In our surgical series, "autoregulation" appears to be directly related to the age of the patient. Proximal feeding arterial calibre returns to normal within a few days in young patients. The rate of return to normal calibre of these proximal feeding arteries and return of the intraluminal pressure toward normal decreases with each decade of life. The pathophysiology of this phenomenon has not been forthcoming. Modern techniques for excision of AVMs rarely present the opportunity for study of the proximal feeding arteries. These lesions are normally excised along a tissue plane established on the perimeter of the AVM with feeding arteries occluded at the margin of the malformation. This technique has precluded histological study of these final feeding vessels as well as the ability to correlate these studies with the postoperative behavior of the proximal feeding arteries. We have histologically studied significant segments of a major final feeding artery in 3 patients with major AVMs. Although our experience is limited, the histological findings are striking and

appear to be related to the patient's age. All 3 patients showed significant delay in the return to normal calibre of the proximal feeding arteries compared to patients less than 30 years old. These specimens showed progressive intimal thickening, disruption of the internal elastic lamina and strikingly progressive replacement of the muscularis media with collagen. These changes progress to the point of the proximal feeding artery which becomes essentially a collagenous tube with attendant loss of contractility. It is postulated that these histological changes are the principle reason for the postoperative hyperperfusion state and attendant cerebral swelling in older patients undergoing excision of AVMs. Pre- and postoperative angiographic studies with accompanying histopathology will illustrate these phenomena. Preoperative management designed to reduce the postoperative sequelae as a result of these histopathological changes will be reviewed.

11:40
9
Dural Sinus AV Fistulae, Congenital and Acquired
Sean Mullan; Chicago, IL

A study of fistulae of the dural sinuses and their related veins indicates:
1) These arise in areas of previous thrombosis.
2) The fistula is in the wall of the sinus.
3) Those that drain into a cortical vein are dangerous with a high risk of intracerebral hemorrhage.
4) The fistulous site or nidus is frequently quite small.
5) When the actual fistulous site is occluded by induced thrombosis, *all* of the multitudinous feeding vessels from multitudinous sources spontaneously disappear. In other words, the fistula is best managed from the venous end.
6) Excision of the sinus is dangerous and not needed.
7) Embolization of feeding vessels is sometimes useful but generally redundant.
8) Percutaneous occlusion of the lateral sinus is sometimes possible. Otherwise the approach is by a limited craniotomy.
9) Examples of sagittal, lateral, sigmoid, superior petrosal, inferior petrosal, sphenoidal, and cavernous sinus fistulae will be presented.
10) Congenital fistulae of the vein of Galen are best tackled from the venous end *after* percutaneous occlusion of all possible arterial feeders.

11) The same principle of venous occlusion, without excision, but subsequent to percutaneous arterial embolization, and carried out under operative and postoperative hypotension may be applicable to some standard congenital cerebral arteriovenous malformations.
12) Examples of 10 and 11 will be presented.

12:00 noon
10
The Spectrum of Dural Arteriovenous Fistulas Excluding Those That Involve the Cavernous Sinus
Charles B. Wilson, Grant Hieshima; San Francisco, CA

Among intracranial dural arteriovenous (AV) fistulas, those that involve the cavernous sinus constitute the majority. Next in frequency are fistulas involving the transverse and sigmoid sinuses, almost always accompanied by some degree of venous occlusive disease. Other intracranial dural AV fistulas are rare, but in the aggregate, they constitute a significant number of cases, the majority of which become symptomatic secondary to venous overload and intracranial hemorrhage.

Dural AV fistulas have been encountered along the vein of Galen; a fistula in this location is particularly difficult to treat. More comon and more readily approached are those fistulas involving the superior sagittal and straight sinuses, among these the AV fistulas in the posterior fossa being more common. Another group of fistulas occurs at the base along the petrous bone and often draining into veins of the posterior fossa. A final site of a typical AV fistula is the ethmoid plate.

This spectrum of fistulas will be presented, classified and defined according to the preferred methods of management.

12:20 p.m.
11
Seizure Disorder Relative to Surgical Treatment of Arteriovenous Malformations
David G. Piepgras, Thoralf M. Sundt, Jr., Lorna P. Stevens; Rochester, MN

A series of 281 patients ranging in age from 4 months to 81 years were treated surgically for angiographically identifiable arteriovenous malformations of the brain betwen 1959 and 1989 and reviewed with respect to their preoperative and postoperative seizure occurrence. One-hundred sixty patients had no seizures preoperatively while 121 had had at least one seizure.

Follow-up information was available in 277 patients ranging from 3 days to 30 years with a mean of 36 years.

Of the 160 patients with no seizures preoperatively, only four had a new ongoing seizure disorder following arteriovenous malformation surgery. Eleven had experienced new seizures but not an ongoing seizure disorder. Of the 121 patients with preoperative seizures, 83 were seizure free after their surgery, 7 had seizures considered to be the same or worse than preoperatively, and in the remainder seizure frequency and severity was improved.

This review will relate risks for new seizure disorder and improvement or resolution of seizures relative to arteriovenous malformation size and location.

12:40 p.m.
12
Results of Surgical Treatment of Cluster Headache; Initial Relief Followed by Recurrence

Robert H. Wilkins, Joel C. Morgenlander; Durham, NC

Chronic cluster headache is ordinarily managed medically, but may become refractory to such medical management. In this setting, surgical treatment has occasionally been performed, based on evidence that pertinent pain pathways and parasympathetic pathways may be interrupted at the main sensory root (MSR) of the trigeminal nerve and at the nervus intermedius (NI).

Between 1976 and 1987, the senior author operated upon 12 patients with chronic cluster headache that was refractory to medical therapy (14 procedures). Nine patients had partial sectioning of the MSR and sectioning of the NI; one patient had only sectioning of the NI; and two patients had NI sectioning plus vascular decompression of the MSR.

Postoperatively, all patients had relief initially but then experienced return of their headaches, except one patient who had relief after a repeat procedure. Average follow-up was 35 months, with a range of 1 to 135 months. Headache began to return on an average of 2 months postoperatively, with a range from 2 days to 14 months. Three patients are currently free of headache; two of these three patients had NI sectioning plus vascular decompression. Together with recurrence of headache, cluster-associated autonomic disturbances recurred after 13 of 14 operations but are currently absent in the three headache-free patients. We conclude that partial sectioning of the MSR and sectioning of the NI are of limited value in the treatment of cluster headaches.

Friday, September 29

8:00 a.m.

13

A New Stereotactic Alignment System for Proton Beam Therapy

Paul H. Chapman, Christopher Ogilvy, Lynn J. Verhey, Nicholas T. Zervas; Boston, MA

The Neurosurgical Service at Massachusetts General Hospital in collaboration with the Departments of Radiation Medicine, Biomedical Engineering, and the Harvard Cyclotron Laboratory, is presently constructing a system of stereotaxic alignment for radiosurgery (STAR). This will be used to deliver proton beam radiation to lesions of the craniocervical axis. STAR can be adapted to either single dose or fractionated therapy. Target coordinates are stereotaxically defined from CT, MRI, and angiographic images using the BRW system. Once the stereotaxic head ring has been fitted, the patient is supported in a reclining position. The patient can now be precisely moved within the x,y,z coordinate system in order to translate the target volume to beam isocenter. Once this adjustment is made, the couch can be rotated about horizontal and vertical axes, with each rotational axis centered on the target volume. This allows one to select any desired beam path to target, minimizing radiation to normal tissue. Three dimensional treatment planning will be used to determine optimum beam angles and to design individually shaped collimators and compensators for each field. The ability to shape lateral and distal edges of the dose distribution for each field, combined with excellent lateral and axial dose sharpness, will result in high dose volumes which conform almost exactly to the target volume. The radiation dosage within the high dose volume will be uniform to within +/- 5% of the prescribed value. Beams of arbitrary shape up to 10 cm can be designed. The versatile design features of STAR will allow us to incorporate anticipated advances in stereotaxis and three dimensional treatment planning technology as these materialize in the future.

8:20 a.m.

14

Functional Stereotactic Neurosurgery with MRI and Neurophysiologic Guidance.

Ronald F. Young, Patricia Rinaldi, Stephen Bloomfield, Denise Albe-Fessard; Irvine, CA

The authors describe a series of functional stereotactic neurosurgical procedures utilizing anatomic target location by magnetic resonance imaging and functional target localization by single

unit and evoked potential micro electrode recording and micro stimulation. Procedures include placement of stimulating electrodes for treatment of chronic pain and lesioning procedures for movement disorders and chronic pain. MRI provides excellent anatomic target identification without the need for the injection of intraventricular contrast material and with anatomic resolution far superior to computerized axial tomographic scanning. Functional target localization with neurophysiologic recording and stimulation is critical to success in functional procedures. Anatomic target localization alone is insufficient. The authors describe the characteristic neurophysiologic responses to recording and stimulation in thalamic nucleus, ventralis posterior lateralis, ventralis posterior medialis, centralis lateralis and central medianum as well as ventralis intermedius and ventralis lateralis. We believe that the combination of MRI and functional neurophysiologic localization provides the best anatomic and physiologic target identification criteria for functional stereotactic neurosurgical procedures.

8:40 a.m.
15
Stereotactic Radiosurgery: The University of Florida System.
William Friedman, Frank Bove, Albert Rhoton Jr.; Gainesville, FL

In 1986, after carefully reviewing the available options for implementing a radiosurgical capability, the Departments of Neurosurgery and Radiation Therapy at the University of Florida decided to design and develop a new, linear-accelerator based radiosurgical system. The system includes unique mechanical and software features which we believe to be improvements on those previously described. In May of 1988, after nearly two years of development and physical testing, the first patient was treated. As of 6/10/89, a total of 33 patients including 25 AVMs, 5 acoustic neuromas, 1 meningioma, 1 chordoma, and 1 glioblastoma had been treated.

Standard Brown-Roberts-Wells equipment is used for angiographic or computerized tomography localization of the lesion. A SUN 4/280 computer, with array processor, is used for dose planning. The CT is reconstructed at the axial level of target center. The user then selects start and stop angles for any patient position. The CT slices through the plane are automatically contoured and the ray paths along each degree of arc computed and displayed. Up to 16 individual arcs of radiation, all focused on the same target, may be used. Multiple isocenters may be

treated if the lesion is nonspherical. Isodose profiles are computed and can be rapidly displayed (10 seconds) in any CT plane (for example, every axial CT slice may be examined to determine if any vital structures are irradiated). The system allows near real time adjustments in collimator size, arcing planes and angles, arc weights, number of arcs, or isocenter location(s).

One of the primary objects of our approach was to provide a mechanical system which would function independently of the accuracy and precision inherently provided by the LINAC gantry and patient support systems. A system of precision bearings with a sliding collimator mount was designed to prevent any torque transfer from the LINAC head to the radiosurgical mechanical system. The measured radiation beam accuracy of this system is .2mm +/- .1mm (n+150).

The physical accuracy of the system has been examined as follows: Angiographic localization was found to be accurate to .5mm +/- .2mm. CT localization had an accuracy of 1 mm +/- .3 mm. Dosimetry accuracy was tested with electronic diodes, thermal luminescent dosimeters, and film dosimetry for single arcs of radiation. A special, water-filled, spherical phantom was constructed to test the accuracy of multiple arc treatments. Measured and computed values were always within the +/- 5% tolerance of the measuring devices.

The University of Florida system was designed to build upon the advances of the Winston-Lutz system. Extensive physical testing has verified that the radiation beam accuracy and dose gradient produced by this system compares favorably to any other published radiosurgical method.

Preliminary patient results will be discussed in detail.

9:00 a.m.
16
Utility of MRI Compatible Subdural Electrode Arrays in the Evaluation and Surgical Management of Patients with Epilepsy and Intracranial Tumors.

R.E. Maxwell, M.E. Fiol, J.R. Gates; Minneapolis, MN

A subdural array of 64-80 electrodes was used to map functional cortical anatomy and to record the sites of spontaneous partial seizure onset in 40 patients. Patients were selected because of widespread interictal and ictal activity by surface EEG; a seizure pattern or interictal-ictal discharge suggesting involvement of speech or motor cortex; or the demonstration by MRI or CT scanning of well defined structural lesions suspected of

being in, adjacent, or immediately subjacent to eloquent functional cortex.

Electrocorticographic (ECOG) data obtained by subdural arrays over a mean duration of 21 days correlated poorly with surface and sphenoidal EEG localization in patients with partial complex seizures of temporal lobe origin not judged to be straightforward candidates for a standard temporal lobectomy. Only 25 percent of these cases showed agreement with respect to the site of ictal activity and 30 percent with regard to the location of interictal fields. Accounting for the lack of agreement between surface and ECOG data was the frequent finding of previously unsuspected interictal and ictal foci in mesio-basal temporal lobe cortex. After delineating the epileptogenic areas and mapping the cortex by stimulation of all electrodes, the patients had aggressive temporal lobe resections up to 9 cm. back from the temporal tip. Nineteen of the 20 patients were improved: 10 were seizure free; 3 had over 90 percent reduction in seizure frequency, and 6 had over 50 percent improvement.

Adaptation of previously used stainless steel electrodes embedded in a silicone matrix to a new 90 percent platinum, 10 percent iridium alloy electrode now permits artifact-free MRI imaging with the electrodes in situ.

Cases are presented to demonstrate how this array has provided improved localization and encouraged more aggressive management of medically intractable partial epilepsy and given the surgeon confidence to excise intracranial neoplasms in close proximity to important functional cortex.

9:20 a.m.
17
The Significance of Limbic Structure Removal in the Surgery of Temporal Lobe Epilepsy, Based on Reoperations
A. Olivier, T. Tanaka, and F. Andermann; Montreal, Quebec, Canada

Out of a series of over 500 temporal resections carried out by the same surgeon (A.O.) 20 patients were reoperated on the ipsilateral temporal lobe with further removal of temporal lobe tissue, usually temporo limbic (amygdala and hippocampus), and in a few cases additional cortical removal at the margin of the previous removal. Out of 20 patients, 16 (80%) have had a successful result, 6 patients (30%) have become seizure free, 3 patients (15%) have had a marked improvement while 7 patients (35%) have had a worthwhile reduction of their seizures. 4 patients (20%) were not helped by the reoperation, 13 (65%) of

the 20 patients reoperated had had no hippocampal removal at the first surgery. The present findings suggest an important role for the hippocampus in the persistence of seizure following earlier temporal resection.

9:40 a.m.
18
Intraoperative Motor Pathway Monitoring with tcMMEP (Transcranial Magnetic Motor-Evoked Potentials)
Christopher B. Shields, R. Dean Linden, Harvey L. Edmonds, Jr., John R. Johnson, Henry D. Garretson; Louisville, KY

Current techniques for the intraoperative assessment of motor function include the Stagnara "wake-up" test and transcranial electrical stimulation of the motor cortex. Disadvantages of the former include its discontinuous nature and the potential hazard of accidental extubation, while the pain accompanying the latter often precludes vital pre- and postoperative evaluation. Therefore, for the past two years we have investigated a painless alternative method of cortical stimulation that utilizes a pulsed magnetic field to evoke individual motor potentials (tcMMEP) in skeletal muscles.

Precise orientation of the magnetic coil near the vertex results in bilaterally symmetrical upper and lower limb responses to tcMMEP. Because of the enormous amplitude variation of individual potentials, only the stimulus-to-peak-onset latency is used for quantification. Reliable production and accurate interpretation to tcMMEP require careful control and monitoring of anesthesia. The adequacy of cerebral perfusion/oxygenation is documented by continuous respiratory and anesthetic gas measurement as well as quantitative EEG, while upper facial EMG monitors anesthetic adequacy and guides delivery of nitrous oxide and narcotic infusion. Evoked limb EMG responses to electrical train-of-four stimulation define the degree of neuromuscular blockade and determine relaxation fusion rate. Reproducible tcMMEP can be elicited with up to 90% neuromuscular block.

Thus far, intraoperative recordings have been made on 40 patients. Their age ranged from 8-88 (38 mean) and 50% were male. The preoperative success rate (91%) in obtaining reproducible tc MMEP fell to 70% intraoperatively. Clinically useful tcMMEP were obtained in 15/17 scoliosis surgeries, but only in 12/23 cases involving compression of the spinal cord or nerve roots. In these later cases, the normal 31 ± 4 ms latency obtained from anterior tibilalis was often dramatically prolonged. These

preliminary findings illustrate that intraoperative tcMMEP recording is technically feasible and may provide a valuable addition to current monitoring modalities.

10:50 a.m.
19
Posterior Fusions at the Craniovertebral Junction
John C. VanGilder; Iowa City, IA

Posterior cervical fusions at the craniovertebral junction result in immediate stability; however, osseous integration of the complex is necessary to maintain long term stability. Review of the literature has demonstrated approximately 20% failure with craniovertebral junction fusions. The author has reviewed 230 patients who underwent posterior fusion at the craniovertebral junction between 1977-1987 at the University of Iowa to allow for a minimum of 2-12 years follow-up.

The pathologic entities include a wide variety of congenital and acquired lesions at the craniovertebral junction, trauma, rheumatoid arthritis, and instability following transoral ventral decompression. One hundred sixty occipitocervical fusions were done and 70 atlantoaxial fusions. One hundred thirty seven patients were reducible and underwent fusion with the posterior elements intact. Ninety three patients required posterior decompression combined with lateral fusion for stability.

Bone and wire fixation followed by postoperative halo immobilization was done in the majority of cases. Acrylic was added to the wire bone fixation in patients with rheumatoid arthritis with subsequent maintenance in a SOMI brace or cervical collar. The complication rate was < 2% in this series. Stratagems concerned with selection of patients, operative technique, and immobilization will be discussed.

11:10 a.m.
20
Experimental and Clinical Results of Low Dose Rate Iodine-125 Permanent Implants for the Treatment of Malignant Gliomas.
J.A. Winfield, G. King, and T.J. Watt; Syracuse, NY

Considerable theoretical controversy exists as to whether accumulated radiation per cell cycle or a minimum dose rate of radiation is critical for tumoricidal effects. Currently, the majority of adjuvant brachytherapy protocols use removal after-loading catheters delivering radiation from high dose rate radionuclides. Although significant improvement in survival has been

documented, radiation necrosis with hemispheric swelling has been reported in up to 50% of treated patients.

In order to evaluate the tumoricidal potential of permanent implant low dose rate Iodine-125 (I-125) for the adjuvant radiation treatment of malignant gliomas, laboratory studies have been initiated in conjunction with a clinical trial. In this report, our experimental results using the feline forelimb motor cortex model and treatment of heterotransplanted human malignant gliomas in nude mice with I-125 will be presented. The dose rate resulting in preservation of normal brain function and anatomical integrity, and tumor cell kill/tumor control, defines a qualitative Therapeutic Index for the I-125 radionuclide.

This qualitative Therapeutic Index can be used in the dosimetry planning for adjuvant radiation therapy of malignant gliomas in humans using permanent I-125 implants. The clinical results of 30 patients harboring newly diagnosed and recurrent glioblastoma multiforme treated with radical resection and same operation permanent implant with I-125 seeds will be presented. In addition to significantly improved survival, using this technique, post-operative hospitalization remains 5 to 7 days, no post-operative external beam radiation is required, and patients are spared the social and physical morbidity of external beam radiation therapy.

11:30 a.m.
21
Chemotherapy with Osmotic Blood Brain Barrier Disruption for Treatment of High Grade Gliomas
Clark Watts, Mary K. Gumerlock; Columbia, MO

Current chemotherapeutic treatment of patients with high grade malignant gliomas following surgery and radiation has not added significantly to the 12-14 months median survival rate. Over four years 37 patients with high grade malignant gliomas underwent 246 treatment procedures with a combination of methyltrexate, cyclophosphamide, and procarbazine given in association with hyperosmolar mannitol transient breakdown of the blood brain barrier. This study group had a mean age of 43 years. Sixteen percent of the patients remained in complete remission at 22 months while 24 patients (65%) had partial or temporary remission. Significant neurological complications included one post-procedural mortality and 5 patients who suffered a stroke-like increase in neurological deficit at the time of BBBD/chemotherapy. Nineteen patients suffered periprocedural temporary seizures. Chemotherapy in conjunction

with osmotic disruption of the blood brain barrier may provide a pharmaco-kinetic advantage sufficient to significantly improve survival in patients with high grade gliomas.

11:50 a.m.
22
Determination of Brain Tumor Patient Reponse to Therapy Using Volumetric Microcomputed CT Scan Analysis.
M.S. Mahaley, Jr.; Birmingham, AL

In an effort to define more precisely and objectively CT brain scan evidence of glioma patient response to treatment, planimetric measurements of serial CT images of enhancing tumor areas were made, using a Numonics 2200 digitizing tablet interfaced to a Zenith 248 microcomputer, running PC3D software for computing 3 dimensional tumor volumes. In order to determine what change in tumor volume was above the standard error of measurement ("significant change") the investigator's coefficient of variation (COV) was first determined by performing triplicate volume measurements (1701) on 155 scans on 27 patients with malignant gliomas. A significant tumor volume change (COV ± 2 standard errors of measurement) was determined to be 20% for all scans. These planimetric generated volume data were compared with mathematical computation of volumes based upon the product of the maximum diameter of enhancing tumor times the diameter perpendicular to the maximum for each image (same number of scan measurements, in triplicate). Since the COV for this observer was less using the planimetric method than with the mathematical computation, for determination of response to therapy by volume change analysis in these same patients, the planimetric method was used. This study has further shown that the smaller the tumor volume being measured, the greater the % change required to be significant. Thus, individual % significant volume changes were defined for large (>14 cc), medium (>8, <14cc), and small (<8 cc) tumor volumes. Next, baseline (prior to investigational therapy) and subsequent serial CT scans were compared, with response defined as the extent of significant change, if any, in the computed tumor volumes. Response to therapy using these criteria for volume computation was compared in each instance to the conventional visual viewbox comparison ("gestalt") which we have generally used in the past when comparing two scans. This study showed that there were no instances when the gestalt technique declared progression of tumor and the planimetric volume technique declared otherwise. However, the sensitivity of the planimetric technique

permitted determination of significant enlargement or reduction in tumor size, while gestalt comparison suggested no change, in 28% of scan comparisons. The use of quantitative tumor volume analysis of planimetric determinations of changes in tumor size during investigational therapy appears to permit recognition of either progression or regression of tumor size earlier than by gestalt comparison in one fourth of instances. This volumetric method is easily adaptable to office microcomputers, and, once the observer's COV has been established, planimetric measurement of all images on a patient's scan can be accomplished in less than 5 minutes and the resulting volume determination compared with previous baseline for definition of response and advice to the patient regarding further therapy during a single clinic visit.

12:10
23
Radiographic and Pathological Assessment of Gangliogliomas with Surgical Outcome.
M. Khayata, R.A.R. Fraser, S. Erde, and L. Heier; New York City, NY

Gangliogliomas are tumors of mature ganglion cells, typically found in children and young adults and account for 1.7% of all brain tumors. We present twelve cases of gangliogliomas that have been surgically treated in the past five years at our institution. The patients ranged in age from 2 to 27. The most common site of these tumors was the temporal lobe followed by the cerebellum.

CT scan typically revealed non-enhancing low density lesions but completely missed three cases of this tumor. Calcification was present in one case. In all cases MRI demonstrated a hypointense lesion on the T1 image with an extremely hyperintense T2 image without associated edema. Angiography was not useful.

Microscopically, the tumors consisted of two neoplastic elements: neurons and glial cells.

Patients typically had a normal neurological examination and usually presented with a history of seizures (up to 20 years.) Seizures were complex partial if the tumor was in the temporal lobe, grand mal if it was elsewhere. Gait ataxia was seen in the cerebellar lesions. Follow up was available in eight patients over a mean period of 5 years (range 2 to 14 years). Gross total excision was accomplished in 63% of these cases. All seizure patients were better controlled post-operatively, with decreased

seizure frequency or reduced anti-convulsant medication. One third of patients remain seizure free off all medication. There was no morbidity or mortality.

In conclusion, these benign tumors occur mainly in childhood and typically present with seizures. Characteristic MRI features make this the best imaging modality. Gross total excision is recommended where this can be achieved with little or no neurological cost. It is important to report that in this series, where subtotal removal or biopsy (3 cases) was undertaken, no growth of the documented residual lesion has been observed in any patient. This fact brings into question the neoplastic nature of these lesions.

12:30 p.m.
24
Complications of Posterior Fossa Surgery.
William Buchheit; Philadelphia, PA

This is a retrospective review of 500 adult posterior fossa operations done at Temple in the last fourteen years. The review focuses on complications related directly to the nervous system. Medical complications, such as pneumonia, urinary tract infections, and pulmonary emboli, are not included.

The complication rate was 28%, including problems such as brainstem contusions, hydrocephalus, transient cranial nerve palsies, septic and aseptic meningitis, spinal fluid fistula and air emboli.

In spite of the high complication rate, the overall mortality was 1.5%, which compares to an uncomplicated cholecystectomy.

Saturday, September 30

8:00 a.m.

25

An Interfascicular Approach to 110 Neurofibromas

David G. Kline, Carlos Garcia, Rand M. Voorhies; New Orleans, LA

The literature concerning the two most frequent benign tumors of neural origin suggests that schwannomas are resectable with little or no added deficit but that neurofibromas are frequently not. This is presumably because schwannomas, due to their origin, tend to displace fascicular structure rather than arising from it like neurofibromas. It has also been recognized that there are histological differences between these two lesions but that in many peripheral nerve tumors, especially those unassociated with Von Recklinghausen's Disease, such differences can be difficult to discern.

Because of pain onset, size, and/or location 185 presumed benign tumors required surgical removal. As a result, a technique for the complete removal of the majority of such lesions has evolved. After recording across the tumor to establish a baseline tracing of function, surrounding fascicles or elements were dissected free taking care to dissect down to the tumor itself and working between that and the more peripheral fascicular structure. Dissection was developed in a circumferential fashion both to the proximal entry and distal exit points of the fascicles. Thus, the fascicular structure at either end of the tumor was partially exposed. In most neurofibromas and even some schwannomas one to three fascicles were found entering the core of the tumor proximally and leaving it distally. The key finding has been that stimulating and recording across such fascicles has almost always *not* produced a nerve action potential (NAP) while recording across those more peripheral has. These entry and exit points can then be sectioned, the bulk of the tumor removed, and remaining fibrous or capsular tissue trimmed from the peripheral fascicles. Of 185 lesions 110 were felt histologically to be neurofibromas.

Such an approach has permitted gross total resection of 68% of solitary neurofibromas as well as slightly over 50% of those associated with VRD without additional significant deficit. There has been one recurrence of a plexus neurofibroma in a 15 year experience. There remains of course a smaller group of "up and down" or contiguous lesions which are either not resectable or require repair, — partial or complete after resection.

8:20 a.m.

26

Arginine Vasopressin (AVP) in Triphasic Diabetes Insipidus.

Paul B. Nelson, Pittsburgh, PA

We tested the hypothesis, previously unsupported by actual vasopressin determinations, that unregulated release of arginine vasopressin (AVP) is responsible for the antidiuretic second (or inter-) phase of triphasic diabetes insipidus (DI). Thirteen rhesus monkeys underwent pituitary stalk transection at the level of the median eminence using microneurosurgical technique. Plasma and urine samples were obtained twice daily and assayed for sodium (NA+) and osmolality (mOsm/kg). In addition, plasma AVP (pAVP) was determined by RIA. Water and solute intake and output were closely monitored. Hydrocortisone was administered at 10 mg/kg/day to prevent stress-induced hypocortisolism. Six (6) monkeys developed triphasic DI, which included an interphase characterized by hyponatremia and hypotonicity coupled with inappropriate urinary concentration. Five (5) monkeys had DI only; of these two had a brief (<4 days) period of polyuria while the remainder were polyuric throughout the study period. Two monkeys remained normal, and were subsequently proved to have an incomplete stalk-section. In five of the six monkeys which manifested the antidiuretic interphase, pAVP concentrations were clearly elevated during the period of hyponatremia: mean pAVP was 3.78 +/- 1.04 pg/ml at the nadir of pNA+ (126.8 ± 3.0 meq/L). This was significantly different from preoperative concentrations of pNA+ and pAVP ($p < 0.01$, Mann-Whitney U test). These results support the hypothesis that the antidiuresis obtained during the interphase after stalk/median eminence trauma has its genesis in an inappropriate release of AVP from the damaged neurons of the supraopticohypophyseal tract.

8:40 a.m.

27

The DREZ Operation: An Update on Current Technique

Blaine S. Nashold, Jr.; Durham, NC

The DREZ operation described as a focal destruction of the substantia gelatinosa of Rolando was first done in 1975 on a patient with arm pain following a brachial plexus avulsion. Since then over 500 patients have undergone the DREZ procedure for treatment of various pain syndromes. The DREZ lesion was designed to destroy the first five layers of Rexed in the dorsal

root entry zone of the lower brain stem and spinal cord thereby disrupting hyperactive neuronal discharges responsible for pain. The DREZ procedure was first used in patients with deafferentation pain syndromes including those with brachial and sacral plexus avulsion injuries, paraplegia and post-herpetic neuralgia.

Currently we used two types of Radionics electrodes for lesion production. The first is the standard .25 mm diameter, thermocouple, temperature monitoring electrode which has a 2 mm long tip for introduction into the spinal cord. A second type, recently modified from the original, is used only for lesioning the trigeminal nucleus caudalis in patients with trigeminal post-herpetic neuralgia. Its tip is 3 mm long with insulation along the first 1 mm. This allows lesioning of the caudalis nucleus while sparing the more superficial spinocerebellar tracts.

As for technique modification, we no longer lesion only the dorsal root entry zones at each root level, but include all the contiguous substantia gelatinosa between roots with lesions 1 mm apart. In patients undergoing caudalis lesioning, we make two rows of lesions, one above the other, from C2 to slightly above the obex. This prevents sparing of the facial midline with resultant residual pain. Finally, lesions are made by heating the electrode tip to 75° for exactly 15 seconds, thus allowing for a more uniform lesion. Following these modifications, we have improved the results of the DREZ procedure and have a decreased incidence of incomplete postoperative pain relief as well as a decreased incidence of complications, especially in patients undergoing caudalis lesioning.

9:00 a.m.
28
Memory Deficits in Patients with Aneurysms
William Shucart; Boston, MA

A series of 90 patients with basilar artery aneurysms is presented in which the incidence of memory deficit is similar to that reported in anterior communicating artery aneurysms. Clinical characteristics of the deficit, the results of formal testing, and the possible causes are discussed. The similarities and differences between these deficits and those seen with anterior communicating artery aneurysms are reviewed.

Saturday, September 30
9:20 a.m.
29
Stroke: A History
Donald Quest; New York City, NY

The clinical description of stroke and speculation about its etiology occupy a prominent position in man's early writings about disease. This presentation traces the development of understanding of the pathogenesis of stroke as it evolves through the course of history.

The Greek word Apoplexia means "struck with violence" and the word "stroke" is thus related. The historical description of stroke begins with Hippocrates and Galen with emphasis on the humoral theories invoked at that time. A thousand years later Leonardo Da Vinci and Andreas Vesalius laid the foundation of scientific anatomy. Thomas Willis and Johan Wepfer expanded these studies with observations of cerebral vessels and pathology. Morgagni made further advances with the development of pathological anatomy and its correlation with clinical syndromes.

Physiology became better elucidated in the 19th century and experimental evidence began to accumulate regarding the pathogenesis of stroke. By the turn of the century work by Chiari began to emphasize the role of the extracranial vasculature. The development by Moniz of cerebral angiography and careful clinico-pathological descriptions of stroke by C. Miller Fisher continued to advance the understanding of the mechanisms of stroke. The roles of stenosis of the carotid artery with resultant cerebrovascular hemodynamic insufficiency and embolism from ulcerated plaque at the carotid bifurcation with occlusion of intracranial vessels now occupy center stage as major etiologies of stroke. Carotid artery surgery in the 1950s opened a new path for prevention of this common disease. Many members of the Academy have contributed to this area of study including our Past-President, current President, and President-Elect.

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WINNER

Christopher D. Heffner, M.D. and Dennis D.M. O'Leary, M.D.

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and
The McDonnell Center for Studies of Higher Brain Function
Washington University School of Medicine

"Target Contol of Collateral Initiation and Directional Axon Growth in the Mammalian Brain"

RUNNERS UP

Michael G. Fehlings, M.D., Ph.D.

Playfair Neuroscience Unit
Toronto Western Hospital

"The Effect of Direct Current Field Polarity on Recovery after Acute Experimental Spinal Cord Injury: A Behavioral, Electrophysiological and Anatomical Analysis"

Richard H. Tippets, M.D.

Department of Neurosurgery
University of Utah at Salt Lake City

"Loss of Constitutional Heterozygosity on Chromosome 17p in Human Malignant Astrocytoma"

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George S. Baker	1960	Edmund Morrissey	1960
C. Hunter Shelden	1961-62	Donald F. Coburn	1961-62
Samuel R. Snodgrass	1963	Eben Alexander, Jr.	1963
Theodore B. Rasmussen	1964	George L. Maltby	1964
Edmund J. Morrissey	1965	Robert Pudenz	1965
George Maltby	1966	Francis A. Echlin	1966
Guy L. Odom	1967	Benjamin Whitcomb	1967
James G. Galbraith	1968	Homer S. Swanson	1968
Robert H. Pudenz	1969-70	Augustus McCravey	1969-70
William B. Scoville	1971	Edward W. Davis	1971
Robert L. McLaurin	1972	John R. Green	1972
Lyle A. French	1973	George J. Hayes	1973
Benjamin B. Whitcomb	1974	Richard L. DeSaussure	1974
John R. Green	1975	Ernest W. Mack	1975
William H. Feindel	1976	Frank E. Nulsen	1976
William H. Sweet	1977	Robert S. Knighton	1977
Arthur A. Ward	1978	Robert G. Fisher	1978

Robert B. King	1979	H.T. Ballantine, Jr.	1979
Eben Alexander, Jr.	1980	George Ehni	1980
Joseph Ransohoff II	1981	Courtland H. Davis, Jr.	1981
Byron C. Pevehouse	1982	John F. Mullan	1982
Sidney Goldring	1983	Hugo Rizzoli	1983
Russel H. Patterson, Jr.	1984	James W. Correll	1984
Thomas Langfitt	1985	E.B. Hendrick	1985
Phanor L. Perot, Jr.	1986	Griffith R. Harsh III	1986
Shelley N. Chou	1987	Ellis B. Keener	1987
James T. Robertson	1988	Robert Grossman	1988

PAST SECRETARY-TREASURER

Francis Murphey	1938-40	Eben Alexander, Jr.	1954-57
A. Earl Walker	1941-43	Robert L. McLaurin	1958-62
Theodore C. Erickson	1944-47	Edward W. Davis	1963-65
Wallace B. Hamby	1948-50	Robert G. Fisher	1966-68
Theodore B. Rasmussen	1951-53	Byron C. Pevehouse	1969-72

PAST SECRETARY		*PAST TREASURER*	
Byron C. Pevehouse	1973	Russel H. Patterson, Jr.	1973
Russel H. Patterson, Jr.	1974-76	Phanor L. Perot, Jr.	1974-76
Phanor L. Perot, Jr.	1977-80	John T. Garner	1977-80
John T. Garner	1981-83	James T. Robertson	1981-83
James T. Robertson	1984-86	Nicholas T. Zervas	1984-86
Nicholas Zervas	1987---	William Buchheit	1987---

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 22-25, 1978
Hyatt Regency, Memphis, Tennessee .....November 7-10, 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

1989
MEMBERSHIP LIST
AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
FOUNDED OCTOBER, 1938

HONORARY MEMBERS *ELECTED*

GUY LAZORTHES 1973
26 Rue D Auriol
31 Toulouse, France

VALENTINE LOGUE (Anne) 1974
16 Rowan Road
Hammersmith
London W6 7DU
England

GÖSTA NORLÉN (Gunvor) 1973
Linnégaten 35 IV
11447 Stockholm, Sweden

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

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

SENIOR MEMBERS	*ELECTED*
EBEN ALEXANDER, JR. (Betty) Bowman Gray School of Medicine 300 South Hawthorne Winston-Salem, North Carolina 27103	1950
GEORGE S. BAKER (Enid) 607 North 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
HOWARD A. BROWN	1939
HARVEY CHENAULT (Bille) 6340 Briar Hill Road Paris, Kentucky 40361	1949
GALE G. CLARK (Marion) 12621 Brookpark Road Oakland, California 94619	1970
W. KEMP CLARK (Fern) 5323 Harry Hines Boulevard Dallas, Texas 75235	1970
COURTLAND H. DAVIS, JR. (Carrie) Bowman-Gray School of Medicine Winston-Salem, North Carolina 27103	1967
RICHARD DESAUSSURE, JR. (Phyllis) 920 Madison Avenue Memphis, Tennessee 38103	1962
DONALD F. DOHN (Carolyn) Cleveland Clinic Florida 3000 West Cypress Creek Road Ft. Lauderdale, Florida 33309	1968
R.M. PEARDON DONAGHY (Frances) P.O. Box 5035, Road 1 Horn of the Moon Road Montpelier, Vermont 05602	1970

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

DEAN H. ECHOLS (Fran) Founder
1550 Second Street
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ROBERT FISHER (Connie) 1957
Box 846, Star Rt. 1
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ELDON L. FOLTZ (Catherine) 1960
UCI Medical Center
Division of Neurosurgery
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LYLE A. FRENCH (Gene) 1954
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JAMES G. GALBRAITH (Peggy) 1947
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SIDNEY GOLDRING (Lois) 1964
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PHILIP D. GORDY (Silvia) 1968
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Casper, Wyoming 82601

EVERETT G. GRANTHAM (Mary Carmel) 1942
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JOHN R. GREEN (Georgia) 1953
Barrow Neurological Institute
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JAMES GREENWOOD, JR. (Mary) 1952
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WALLACE B. HAMBY (Eleanor) 1941
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JOHN W. HANBERY (Shirley) 1959
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MAJ. GEN. GEORGES S. HAYES (Catherine) 1962
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JESS D. HERRMANN (Mary Jo) 1938
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WILLIAM E. HUNT (Carole) 1970
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ROBERT B. KING (Molly) 1958
University Hospital
Upstate Medical Center
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ROBERT S. KNIGHTON (Louise) 1966
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THOMAS W. LANGFITT (Carolyn) 1971
Glenmede Trust Company
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RAEBURN C. LLEWELLYN (Carmen) 1963
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WILLIAM M. LOUGHEED (Grace) 1962
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JOHN J. LOWREY (Katy) 1965
P.O. Box 4302
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FRANK MAYFIELD (Belle Clay) Founder
506 Oak Street
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AUGUSTUS McCRAVEY (Helen) 1944
1414 Continental Dr. #1005
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ROBERT L. McLAURIN 1955
111 Wellington Place
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WILLIAM F. MEACHAM (Alice) 1952
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FRANCIS MURPHEY (Marge) Founder
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BLAINE S. NASHOLD, JR. (Irene) 1967
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GUY L. ODOM (Mataline) 1946
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J. LAWRENCE POOL (Angeline) 1940
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ROBERT W. PORTER (Aubrey Dean) 1962
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ROBERT H. PUDENZ (Rita) 1943
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JOHN RAAF (Lorene) Founder
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AIDEN A. RANEY (Mary) 1946
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JOSEPH RANSOHOFF II (Lori) 1965
New York University Medical Center
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THEODORE B. RASMUSSEN (Catherine) 1947
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HENRY G. SCHWARTZ (Reedie) 1942
Washington University School of Medicine
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C. HUNTER SHELDEN (Elizabeth) 1941
Huntington Medical Research Institute
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ANTHONY SUSEN (Phyllis) 1965
504 Remora Circle
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WILLIAM H. SWEET (Elizabeth) 1950
309 Goddard Avenue
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JOHN TYTUS (Virginia) 1967
P.O. Box 279
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ALFRED UIHLEIN (Ione) 1949
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A. EARL WALKER (Agnes) 1938
1445 Wagontrain Drive, S.E.
Albuquerque, New Mexico 87123

EXUM WALKER (Nellie) 1938
490 Peachtree Street, N.E.
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W. KEASLEY WELCH (Elizabeth) 1957
25 Gould Road
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BENJAMIN B. WHITCOMB (Margaret) 1947
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ACTIVE MEMBERS	*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
GILLES BERTRAND (Louise) Montreal Neurological Institute 3801 University Street Montreal, Quebec, Canada H3A 1B4	1967
PETER M. BLACK (Katharine) Brigham and Women's Hospital 75 Francis Street Boston, Massachusetts 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 Center 7703 Floyd Curl Drive San Antonio, Texas 78284-7843	1984
DEREK A. BRUCE (Frances) 7777 Forrest Lane, #C703 Dallas, Texas 75230	1984
WILLIAM A. BUCHHEIT (Lyn) 3401 North Broad Street Philadelphia, Pennsylvania 19140	1980
PAUL H. CHAPMAN (Tansy) Department of Neurosurgery Massachusetts General Hospital Boston, Massachusetts 02114	1983
SHELLEY N. CHOU (Jolene) University of Minnesota Medical Center 420 Delaware Street S.E., Box 96 Minneapolis, Minnesota 55455	1974

WILLIAM F. COLLINS, JR. (Gwen) 1963
Yale University School of Medicine
333 Cedar Street
New Haven, Connecticut 06510

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

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
UAB Station
Birmingham, Alabama 35294

MARK PETER HEILBRUN (Robyn) 1984
Division of Neurosurgery, #3B320
University of Utah Medical Center
50 N. 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 Southeast Delaware Street Box 96
Minneapolis, MN 55455

CHARLES HODGE (Linda) 1982
750 E. 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

JOHN P. KAPP (Lureese) 1985
406 North Main Street
Galax, Virginia 24333

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

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

WILLIAM A. KELLY (Joan) 1977
Department of Neurological Surgery
RI-20
University of Washington
Seattle, Washington 98195

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
531 Chamiso Lane, N.W.
Albuquerque, New Mexico 87107

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

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

THEODORE KURZE 1967
521 East 14th Street #11G
New York, New York 10009

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

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

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

ERNEST W. MACK (Bobbie) 1956
505 South Arlington Avenue
Suite 106
Reno, Nevada 89509

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

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

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

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

SYDNEY J. PEERLESS (Ann) 1977
University Hospital
339 Windermere Road
London, Ontario, Canada N6A 5A5

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

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

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

DONALD O. QUEST (Ilona) 1986
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 Ave. #206
Salt Lake City, Utah 84103

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

THEODORE S. ROBERTS (Joan) 1976
Dept. of Neurological Surgery
University Hospital
1959 Pacific Ave. NE, RI 20
Seattle, Washington 98195

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

FREDRICK 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 Univ. Med. Center/Neurosurgery
1325 S. Grand Blvd.
St. Louis, Missouri 63104

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 Center
7703 Floyd Curl Drive
San Antonio, Texas 78284-7843

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

RONALD R. TASKER (Mary) 1971
Toronto General Hospital
Room 215, 14th Floor
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JOHN TEW, JR. (Susan) 1973
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GEORGE TINDALL (Suzie) 1968
Emory University School of Medicine
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1365 Clifton Road, N.E.
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JOHN C. VAN GILDER (Kerstin) 1980
University of Iowa Hospital
Iowa City, Iowa 55242

ARTHUR A. WARD, JR. (Janet) 1953
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CLARK WATTS (Patty) 1975
One Hospital Drive
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BRYCE K. A. WEIR (Mary Lou) 1984
2D2-24 Mackenzie
Health Sciences Center
8440-112 Street
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MARTIN H. WEISS (Debby) 1981
USC Medical Center
1200 North State Street
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LOWELL E. WHITE, JR. (Margie) 1971
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Mobile, Alabama 36688

ROBERT WILKINS (Gloria) 1973
Duke University Medical Center
Box 3807
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CHARLES B. WILSON (Pamela) 1966
Department of Neurological Surgery
University of Calfornia Medical Center
Third and Parnassus
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FRANK WRENN (Betty) 1973
27 Memorial Medical Drive
Greenville, South Carolina 29605

DAVID YASHON (Myrna) 1972
50 South McNaughton Road
Columbus, Ohio 43213

RONALD F. YOUNG (Sheila) 1986
University of California at Irvine
101 The City Drive South
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NICHOLAS T. ZERVAS (Thalia) 1972
Massachusetts General Hospital
Boston, Massachusetts 02114

INACTIVE MEMBER

M. STEPHEN MAHALEY, JR. (Jane) 1972
P.O. Box 1063
Maggie Valley, North Carolina 28751

CORRESPONDING MEMBERS

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

JUAN CARDENAS 1966
Insurgentes Sur 594
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LUC CALLIAUW 1988
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JUAN C. CHRISTENSEN 1970
Ayacucho 2151 4 P
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Clinica Neurochirurgica
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JACQUES DEVILLIERS (Jeanne Marie) 1986
Department of Neurosurgery
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Neurosurgical Clinic
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Neurosurgery Department
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1150 Bruxelles, Belgium

KARL AUGUST BUSHE (Eva) — 1971
Neurochirurgischen Klinik
Josef-Schneider-Strasse II
D-8700 Wurzburg, West Germany

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

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

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

KATSUTOSHI KITAMURA (Yoshiko) — 1970
Shinkokura Hospital
1-3-1 Kanada
Kokurakita-Ku
Kitakyushu, 803 Japan

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

WILLIAM LUYENDIJK (Tony) — 1973
Pr Bernhardlaan 60
Oegstgeest, The Netherlands

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

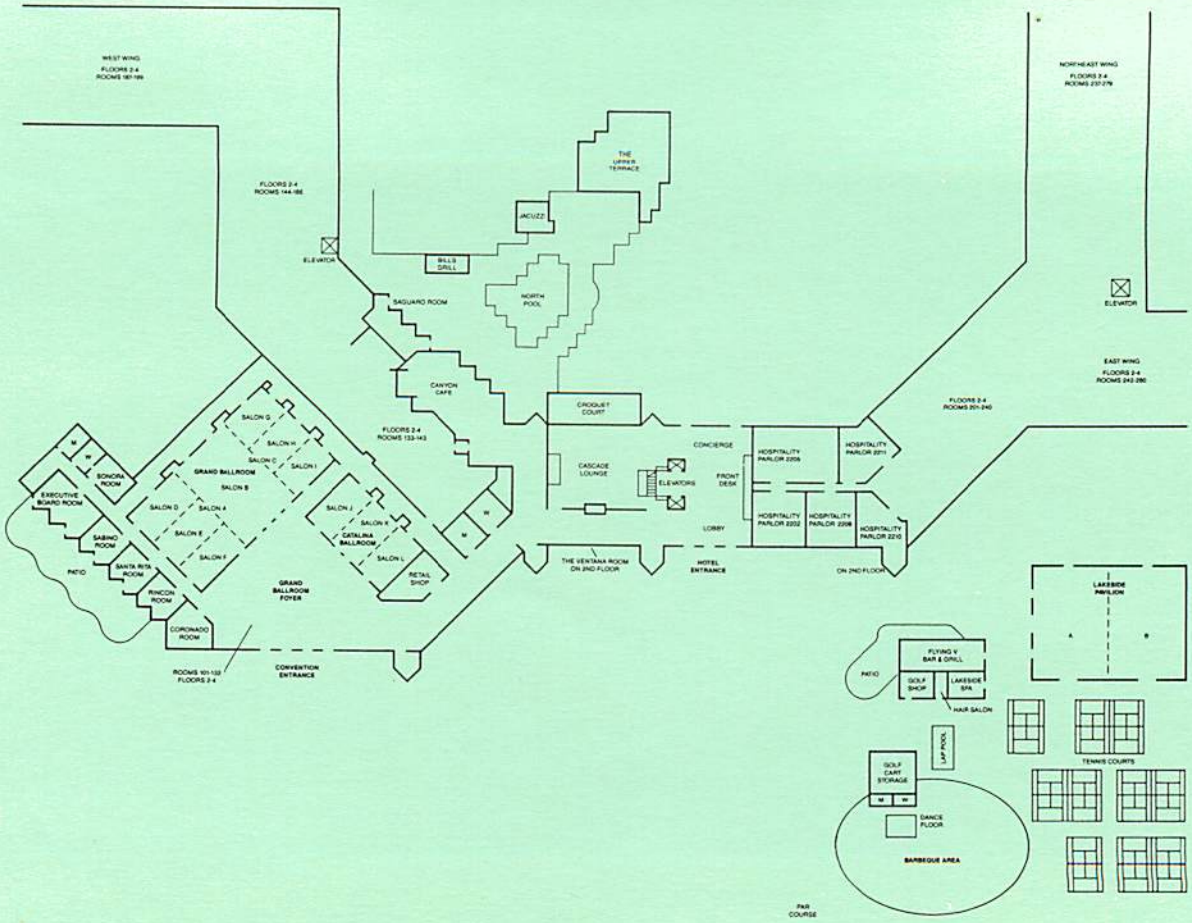
KURT SHURMANN — 1978
Director
Neurochirurg
Univ-Klinik Mainz
Langenbeskstr 1
6500 Mainz, West Germany

DECEASED MEMBERS		*ELECTED*
SIXTO OBRADOR ALCALDE Madrid, Spain (Honorary)	4/1978	1973
JAMES R. ATKINSON Phoenix, Arizona (Active)	2/1978	1970
PERCIVAL BAILEY Evanston, Illinois (Honorary)	8/1973	1960
WILLIAM F. BESWICK Buffalo, New York (Active)	5/1971	1959
EDWIN B. BOLDREY San Francisco, California (Senior)	6/1988	1941
SPENCER BRADEN Cleveland, Ohio (Active)	7/1969	Founder
F. KEITH BRADFORD Houston, Texas (Active)	4/1971	1938
DONALD F. 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
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)	1/1976	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 Scarborough, Maine (Senior)	1988	1942
DONALD D. MATSON Boston, Massachusetts (Active)	5/1969	1950
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


WEST WING
FLOORS 2-4
ROOMS 187-199
FLOORS 2-4
ROOMS 144-186
ELEVATOR
THE
URNER
TERRACE
JACUZZI
BILL'S
GRILL
NORTH
POOL
SAGUARO ROOM
CANYON
CAFE
CROQUET
COURT
FLOORS 2-4
ROOMS 133-143
CASCADE
LOUNGE
ELEVATORS
CONCIERGE
FRONT
DESK
LOBBY
HOSPITALITY
PARLOR 2205
HOSPITALITY
PARLOR 2211
HOSPITALITY
PARLOR 2202
HOSPITALITY
PARLOR 2208
HOSPITALITY
PARLOR 2210
THE VENTANA ROOM
ON 2ND FLOOR
HOTEL
ENTRANCE
ON 2ND FLOOR
NORTHEAST WING
FLOORS 2-4
ROOMS 237-279
ELEVATOR
EAST WING
FLOORS 2-4
ROOMS 243-280
FLOORS 2-4
ROOMS 201-240
SONORA
ROOM
EXECUTIVE
BOARD ROOM
SABINO
ROOM
SANTA RITA
ROOM
PINCON
ROOM
CORONADO
ROOM
PATIO
GRAND BALLROOM
SALON G
SALON H
SALON C
SALON I
SALON B
SALON D
SALON A
SALON E
SALON F
GRAND
BALLROOM
FOYER
CATALINA
BALLROOM
SALON J
SALON K
SALON L
RETAIL
SHOP
ROOMS 101-132
FLOORS 2-4
CONVENTION
ENTRANCE
LAKESIDE
PAVILION
A
B
PATIO
FLYING V
BAR & GRILL
GOLF
SHOP
LAKESIDE
SPA
HAIR SALON
TENNIS COURTS
LAP POOL
GOLF
CART
STORAGE
M
W
DANCE
FLOOR
BARBEQUE AREA
PAR
COURSE