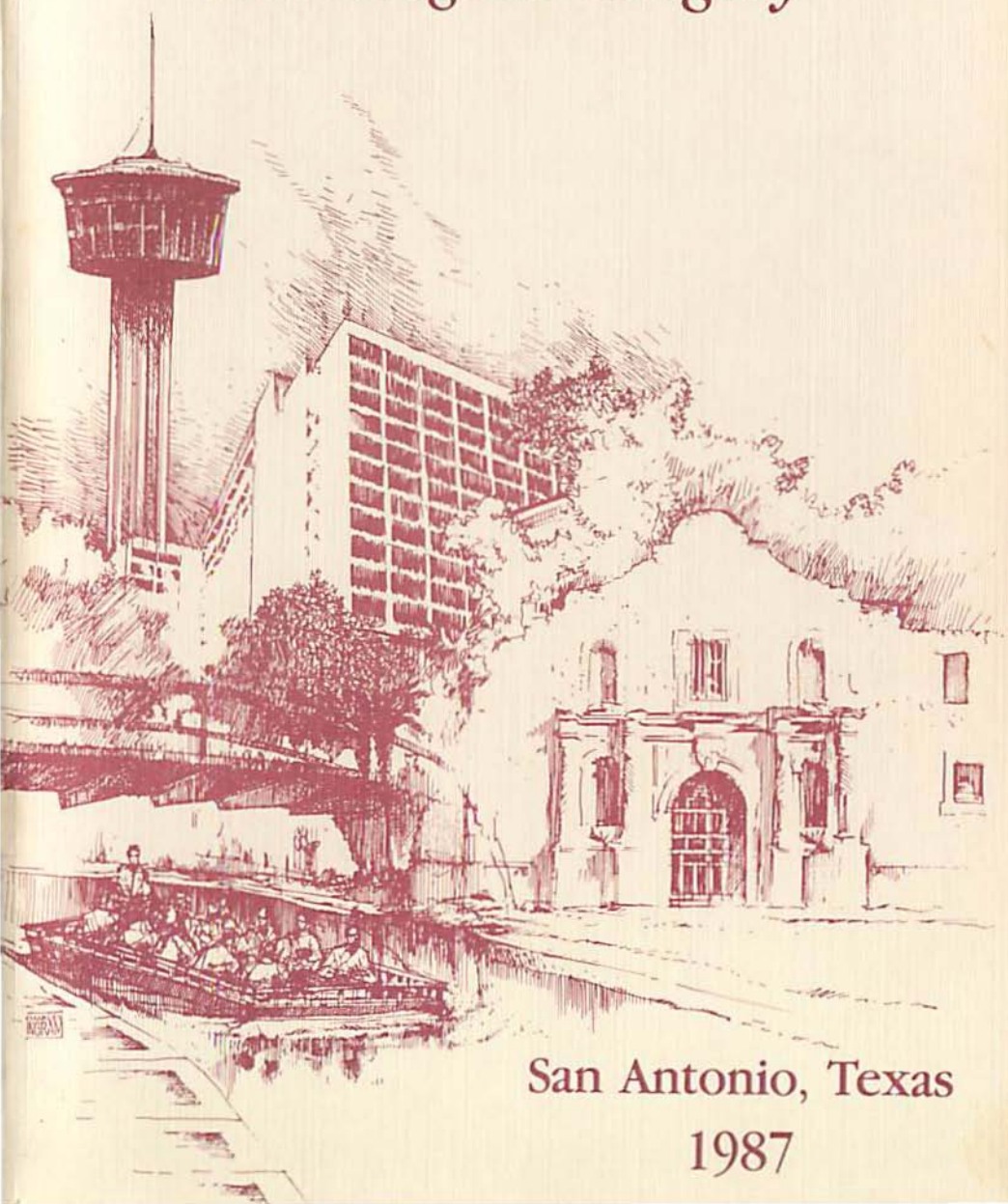


*The American
Academy of
Neurological Surgery*



San Antonio, Texas
1987



ANNUAL MEETING OF

The
*American Academy of
Neurological Surgery*

The Hyatt Regency Hotel
San Antonio, Texas

October 7-10, 1987

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The American Academy of Neurological Surgery

October 7-10, 1987

The Hyatt Regency Hotel
San Antonio, Texas

Wednesday, October 7

- 1:00 PM-5:00 PM Registration-*Los Rios Foyer*
2:00 PM-5:00 PM Executive Committee Meeting
President's Suite
6:30 PM-8:30 PM Welcoming Cocktail Reception
The Garden Terrace

Thursday, October 8

- 8:00 AM-5:00 PM Registration-*Los Rios Foyer*
6:45 AM-8:00 AM Breakfast Business Meeting
(Members Only)
Rio Grande Ballroom West & Center
8:00 AM-11:15 AM Scientific Meeting
Regency Ballroom West
11:15 AM-12:00 Presidential Address
(Noon) Shelley N. Chou, M.D., Ph.D.
Regency Ballroom West
12:00-1:30 PM Presidential Luncheon Buffet
(Noon) *The Garden Terrace*
(Members, Guests, Spouses)
1:30 PM-5:00 PM Scientific Meeting
Regency Ballroom West
6:30 PM Depart by River Boats at
Hyatt Regency River Level for
Cocktails and Dinner at
Institute of Texan Cultures

Friday, October 9

- 8:00 AM-5:00 PM Registration-*Los Rios Foyer*
6:45 AM-8:00 AM Breakfast Business Meeting
(Members Only)
Rio Grande Ballroom West & Center
- 8:00 AM-12:30 PM Scientific Meeting
Regency Ballroom West
- 12:30 PM Group Photo-*The Alamo*
- 1:30 PM-5:00 PM Golf and Tennis Tournaments
Oak Hills Country Club
Optional Tours
- 7:00 PM-8:00 PM Annual Reception-*Regency Foyer*
8:00 PM-Midnight Dinner Dance-*Regency Ballroom* (Black Tie)

Saturday, October 10

- No Breakfast Meeting
- 8:00 AM-12:30 PM Scientific Meeting
Regency Ballroom West

Spouses Activities

Wednesday, October 7

- 6:30 PM-8:30 PM Welcoming Cocktail Reception
The Garden Terrace

Thursday, October 8

- 8:00 AM-4:00 PM Spouses Hospitality
Crescendo Room
- 9:00 AM-11:00 AM "Bienvenidos"-Modeling and Fashions by
Mexican Boutique
Crescendo Room
- 11:15 AM-12:00 Presidential Address
(Noon) Shelley N. Chou, M.D., Ph.D.
Regency Ballroom West
- 12:00-1:30 PM Presidential Luncheon Buffet
(Noon) *The Garden Terrace*
(Members, Guests, Spouses)

1:30 PM-5:00 PM OPTIONAL: SHOPPING TOURS

- North Star Mall*-
250 stores (via shuttle)
- El Mercado Mexican Market*
(via trolley)
- Guided Antique Tour*

6:30 PM Depart by River Boats at Hyatt Regency River
Level for Cocktails and Dinner at *Institute of
Texan Cultures*

Friday, October 9

8:00 AM-4:00 PM Spouses Hospitality
Crescendo Room

8:30 AM-9:15 AM Cooking Demonstration
Eduard Peyer, Executive Chef
Crescendo Room

9:30 AM-12:00
(Noon) Buses depart Crockett Street entrance of Hyatt
for walking tour of *Botanical Gardens*

1:30 PM-5:00 PM Golf and Tennis Tournaments
Oak Hills Country Club

1:30 PM-5:00 PM OPTIONAL: GUIDED TOURS
-*Mission San Jose, Historical King William,*
McNay Art Museum
-*Japanese Tea Garden, San Antonio Zoo,*
Brackenridge Park

ACTIVITIES TO DO ON YOUR OWN:

- La Villita* walking distance from hotel
- El Mercado* (via trolley)
- Outdoor swimming pool and exercise room for
hotel guests

7:00 PM-8:00 PM Annual Reception-*Regency Foyer*

8:00 PM-Midnight Dinner Dance-*Regency Ballroom* (Black Tie)

Saturday, October 10

8:00 AM-12:00
(Noon) Spouses Hospitality
Crescendo Room

Thursday, October 8

8:00 **Welcome:** Shelley N. Chou, M.D., Ph.D. - President

SCIENTIFIC SESSION I

MODERATOR: Henry Garretson, M.D., Ph.D.

8:15 **Symposium on Arteriovenous Malformations**

Roberto Heros, M.D.

Raymond Kjellberg, M.D.

Russel Patterson, M.D.

Bennett Stein, M.D.

10:00 **Coffee/Tea**

SCIENTIFIC SESSION II

MODERATOR: Jim Robertson, M.D.

10:15

1. **When is the Outlook Hopeless after Aneurysm Rupture?**

Bryce Weir, Lew Disney, Michael Grace

University of Alberta

A study of 188 poor grade aneurysm cases were carried out in a prospective, multicenter trial of the calcium antagonist Nimodipine. Patients had to be admitted within 3 days of their SAH. Admission work-up included angiography of anterior and posterior circulations as well as CT scans. The angiograms were repeated as close to day 7 post-SAH as possible and the CT scans were repeated at 3 months, at the time of follow-up neurological assessment. Radiological assessment was performed independently of knowledge of drug treatment or patient outcome. A discriminate function analysis was performed which indicated that the relative importance of factors prognostic for outcome (good, mild deficits versus severe deficits, vegetative, dead) to be, in order of importance: whether the patient was operated, neurologic grade on admission, age, initial systolic BP and aneurysm size.

The mean age of good outcome cases was 46 years, for fatal cases, 58 years. The oldest patient admitted as a grade 3 to have a good outcome was 77 years and the oldest patient admitted as grade 4 with a good outcome was 66 years. The percentage of cases with a bad outcome, for a given feature were: large ICH (90%), large IVH (87%), acute severe hydrocephalus (84%), operated (50%), not-operated (100%), thick layer SAH

(73%), grade 5 (94%), grade 4 (72%), grade 3 (43%), more than 21 mm (72%), 4-6 mm (56%), severe diffuse VSP (61%), no VSP (43%), rebleed (90%), no rebleed (51%), history of hypertension (76%), no history hypertension (62%). The mean systolic admission BP for cases with a good outcome was 137 mmHg and for those who died it was 168 mmHg.

The discriminant function analysis correctly classified 80% of cases. It seems reasonable to avoid any active intervention in such cases as a grade 4 or 5 octagenarian who has massive ICH, IVH, and SAH, with systolic blood pressure above 180 mmHg and a history of hypertension. Decisions in less extreme examples will still depend partly on "clinical judgement", which remains partly intuitive and individually based.

10:35

2. Ophthalmic Artery Aneurysms

Eugene S. Flamm, M.D.

New York University School of Medicine

Although aneurysms arising from the proximal supraclinoid carotid artery represent only 10% of most series, they can often be the most challenging of the anterior circulation because of their size, inaccessibility and relation to the cavernous sinus. They often reach giant sizes and present with ocular symptoms rather than subarachnoid hemorrhage. We have reviewed our experience with 75 carotid ophthalmic aneurysms from a total of 800 aneurysms.

In the present series 42% of the aneurysms in the region of the ophthalmic artery were 2 cm or greater. This is reflected in the finding that 32 of these patients did not have SAH as the first presentation. Multiplicity was noted in 17.8%, 11.8% had another aneurysm and 6% had bilateral ophthalmic artery aneurysms. Outcome was related to the preoperative clinical grade, but there were 2 deaths in the Grade 0-2 group, (3%), both in patients with aneurysms of 3 cm. Overall there were 5 deaths, a mortality rate of 7%. Increased visual symptoms occurred in 3 patients (4%) and increased neurological deficit in 4 (5%), a combined morbidity and mortality rate of 16%.

Particular issues that have been examined regarding complicated ophthalmic artery aneurysms include the prediction of clipability, the potential for balloon occlusion, temporary occlusion of the ICA, suction decompression of the aneurysm prior to clipping, and appropriate clip selection. The increased use of CT and MRI scanning has led to more frequent diagnosis of

these aneurysms before hemorrhage had occurred. These issues and the general surgical management of ophthalmic artery aneurysms that has evolved with this experience will be presented.

10:55

3. Surgical Considerations in the Treatment of Massive Vertebral Artery Aneurysms: A Report of Three Cases

Wolff M. Kirsch, M.D., Crister Lindquist, M.D., Ph.D., Yong-Hua Zhu, M.D., Wei-Ming Cheng, M.D., William Orrison, M.D., Mario Kornfeld, M.D.

University of New Mexico School of Medicine and Karolinska Sjukhuset, Sweden

Three critically ill patients were surgically treated by debulking of large, clot-filled, ventrally situated vertebral artery aneurysms; two cases by an oblique suboccipital approach and one by a transoral-clival approach. All three cases had tenuous collateral circulation mandating conservation of the PICA on the parent vertebral artery. Only one case benefited from surgical intervention.

The transoral-clival approach gave excellent visualization of the aneurysm but poor access to the parent right vertebral artery and sessile neck. Opening of the aneurysm resulted in profuse hemorrhage. The right vertebral artery was taken with the knowledge that the opposite vertebral artery was hypoplastic. The patient died 48 hours after surgery of brain stem infarction.

The two cases approached suboccipitally provided visualization of the parent left vertebral arteries from the right side. In both cases the brain stem and upper cervical cord were so displaced that clot removal from the aneurysm permitted visualization and preservation of the critical PICA's. One case with slow but progressive improvement in breathing, swallowing, and balance after surgery was not associated with significant hemorrhage from the opened aneurysm. This patient survived for three years after surgery.

The third case was operated with provision for intraoperative angiography, balloon occlusion, and FDA approval for the use of a new macroflange approximator clip. Significant backbleeding from the opened proximal left vertebral artery could not be controlled by balloon occlusion. The macroflange enabled closure and complete hemostasis. Despite this effort the patient awoke quadriplegic and died two months later. Post-mortem examination revealed remarkable thinning of the medulla and

upper cervical cord, the vertebral artery repair was noted, yet another 1.5 cm. aneurysm on the right vertebral artery that had escaped prior detection. Despite debulking the calcified arterial wall retained its deforming posture.

11:15 Presidential Address

Shelley N. Chou, M.D., Ph.D.

Introduction by Ellis Keener, M.D.

12:00 Presidential Luncheon Buffet

SCIENTIFIC SESSION III

MODERATOR: William Buchheit, M.D.

1:30 Special Presentation - Ethical and Legal Challenges for Neurosurgery

Professor Alexander Capron

The Norman Topping Professor of Law, Medicine and Public Policy, University of Southern California, *and* former Executive Secretary of The Presidential Commission on Biomedical Ethics

Introduction by Shelley N. Chou, M.D., Ph.D.

2:15 Discussion Questions

2:30 Academy Award Presentation

To be announced by Frederick A. Simeone, M.D.

2:55 Coffee/Soft Drinks

SCIENTIFIC SESSION IV

MODERATOR: Nicholas Zervas, M.D.

3:20

4. New Techniques for Studying the Pathophysiology of Normal and Neoplastic Human Pituitary Tissue

William E. Chandler, M.D.

University of Michigan Medical Center

In recent years a variety of scientific and technical advances have provided several new techniques for tissue analysis which may be applied to the study of the pituitary and its associated tumors. In conjunction with Dr. Ricardo Lloyd, an endocrine pathologist, I have worked with several of these techniques and would propose to discuss these techniques along with our preliminary results.

Tissue culture systems have been used to maintain growth of normal and neoplastic pituitary cells for up to four weeks. The cultured cells are separated from the normal influences of the hypothalamus and end organ feedback and therefore can be studied and manipulated in this isolated environment. Cells have been manipulated with dopamine, diethylstilbesterol (DES), and TRH with serial sampling of the media for prolactin. Results include a decrease in prolactin to dopamine, an increase to TRH and a variable response to DES.

The *reverse hemolytic plaque assay* technique isolates individual pituitary cells and identifies the specific hormone being actively secreted by those cells. Cultured pituitary cells are mixed with sheep red blood cells coated with protein-A, as well as complement and a specific antibody, and the presence of hormone production results in red cell lysis. We have compared the accuracy of this assay to immunocytochemistry.

Dopamine receptors can be labeled and quantified in both normal and neoplastic pituitary tissue. We have used ³H labeled spiperone which binds to dopamine receptor sites. We have found normals and null cell adenomas to be 1+ for receptors, prolactinomas to be 3+ and acromegalic tumors to be negative.

In situ hybridization is a technique in which radiolabeled oligonucleotide DNA probes are utilized to label and identify messenger RNA for specific hormones. This technique allows one to study the biosynthesis and localization of specific mRNAs. We have studied the effect of estrogen on the production of prolactin mRNA.

3:40

5. Long Term Evaluation of Large Pituitary Tumors After Transphenoidal Surgery

John C. VanGilder, M.D.

University of Iowa Hospital

One hundred two patients underwent transsphenoidal resection of pituitary tumors greater than 3 cm in diameter between October, 1976 and September, 1982 by the author. There were 56 males and 46 females between the ages of 7-82 years. The tumors were classified as pituitary adenomas 63, prolactin secreting 20, growth hormone secreting 17, ACTH secreting 1 and TSH secreting 1. Thirteen of the 102 patients presented with recurrent pituitary tumors following craniotomy 1-13 years previously.

Each patient underwent endocrine evaluation pre-operatively as well as evaluation every 1-2 years after operation. In addition

to elevated pituitary hormone levels in the secretory tumors, 30% of the patients were hypothyroid and 23% had an insufficient pituitary-adrenal axis prior to surgery. Radiologic evaluation demonstrated 89 patients to have suprasellar extension of the tumor and 13 did not. Seventy-eight patients had a visual field deficit pre-operatively.

All patients have been followed 5-10 years subsequent to surgery. There were 14 deaths between 6 months-8 years after operation from etiologies unrelated to pituitary surgery. Twenty-six patients underwent irradiation therapy to the pituitary after surgery (6 previously after craniotomy) and 76 had no irradiation. Three patients in the non-irradiated group have undergone re-operation for recurrent tumor and none in the irradiated patients.

Post-operative visual fields were normal in 34 (47%), greater than 50% improvement 19 (26%), less than 50% improvement 9 (12%), no change 10 (14%) and increased deficits in 1 (1%). At the end of follow up, 50% of the cases were hypothyroid and 43% had pituitary-adrenal axis insufficiency.

The significance of these results will be discussed including selective criteria for post-operative irradiation.

4:00

6. The Usefulness of Magnetic Resonance Imaging in Selecting the Operative Approach to Large Pituitary Tumors

Robert B. Snow, M.D., Ph.D., Michael H. Lavynne, M.D., Susan Morgello, M.D., Russel H. Patterson, Jr., M.D.
Cornell University

The transsphenoidal approach has been used for removing large pituitary tumors when the tumor has not extended into the anterior, middle, or posterior fossae. The transcranial route has been employed for these cases. An additional indication for craniotomy is a fibrous tumor because it does not collapse into the sella with surgical reduction of its size, making radical removal difficult. CT cannot define those cases in which the tumor is of firm consistency.

In an earlier report, we presented fifteen patients with large pituitary tumors who had both MRI and CT. Firm vs. soft tumors were found to be differentiable on MRI but not CT in all cases. We now report our experience with MRI on a larger group of patients.

In the past four years we have operated on 145 pituitary tumors. Approximately 50% of these were large tumors with suprasellar extension. Based on the operative findings, the

tumors were divided into two groups: 1) were described by the surgeon as soft and easily removed by suction and curettage, and 2) were of firm consistency and required sharp dissection or the laser for removal. The specimens were examined by two pathologists (without knowledge of the operative consistency or MRI studies) for evidence of fibrosis. A neuroradiologist independently divided the tumors into two groups based on MRI signal: 1) isointense, or 2) hyperintense with surrounding brain on T₂-weighted images. Results were as follows: 1) all firm tumors were isointense, while all soft tumors were hyperintense on T₂-weighted images; 2) tumor consistency was not differentiable on CT; and 3) firm tumors as compared to soft tumors more commonly evidenced marked perivascular fibrosis or dense collagen formation when examined pathologically. The implications of these findings with regard to preoperatively choosing an approach to large pituitary tumors is discussed.

4:20

7. Adrenal Autotransplant in Hemiparkinson Monkeys

Barbara Brooks-Eidelberg, Ph.D., Eduardo Eidelberg, M.D., Jim Story, M.D., Rebecca Barrett-Tuck, M.D., Frederick Boop, M.D.
University of Texas Health Science Center-San Antonio

Unilateral injection of MPTP (1-methyl-4-phenyl-1,2,3,6, tetrahydropyridine) into one internal carotid artery was used to produce contralateral akinesia, bradykinesia, and tremor in young adult cynomolgus monkeys. As a behavioral monitor of the drug effects, animals were trained, prior to injection to press a bar rapidly and repetitively with either hand for food reward. Following MPTP-injection the ipsilateral limb continued to press normally, while bar pressing on the side contralateral to the injection was essentially eliminated. The deficit could be alleviated temporarily by oral levodopa-carbidopa. One subject was treated by grafting 3 small pieces (total 10-20 mg) of adrenal medulla into the head of the caudate nucleus on the same side as the MPTP injection. Six days later bar pressing began to appear in the incapacitated hand, and stabilized at a significantly improved, but lower than normal, rate. The improvement persisted until sacrifice 4 weeks post-operatively. We confirmed histologically that the grafts were properly placed in the head of the caudate, that they contained typical chromaffin cells with dense core granules, and that the pars compacta of the SN was nearly totally destroyed in the injected side while the SN of the opposite hemisphere appeared normal. This suggests that the success of the graft was due to the continuing synthesis and

secretion of catecholamines by the adrenal chromaffin cells (probably most importantly dopamine). This model of Parkinson's disease seems excellently suited for studying the physiological mechanisms underlying the adrenal autotransplant as well as other therapeutic procedures.

4:40

8. Is Autologous Transplant of Adrenal Medulla Into the Striatum a New and Effective Therapy for Parkinson Disease?

Eduardo Garcia Flores, M.D.

Osler Centro Medico, Monterrey, Mexico

Recently contradicting reports from two different groups of physicians one in Sweden (Backlund et al) and the other in Mexico (Madrazo et al) have described the effects of transplanted (autologous) adrenal tissue into the neostriatum of patients who suffered parkinson disease. In an effort to clarify this apparently different results we have performed one of these operations following all the guide lines provided by Dr. Madrazo and coworkers, in a young 37 year old female with rapidly progressing parkinson disease of 5 years duration and with symptoms of unwanted side effects to Levodopa. The results of such an operation and the possible mode of action of this new therapy is the purpose of this report.

Friday, October 9

SCIENTIFIC SESSION V

MODERATOR: George Ojemann, M.D.

8:00 Symposium on the Management of Epilepsy

James Ferrendelli, M.D.

Sidney Goldring, M.D.

Robert Maxwell, M.D.

Theodore Rasmussen, M.D.

10:00 Coffee/Tea

10:15

9. A Chemical and Histochemical Architecture of the
Human Epileptic Focus

Allen R. Wyler, M.D., Suzanne Nadi, Ph.D.
University of Tennessee

The chemical parameters in the spiking and non-spiking portions of the human brain are largely unknown. Recently we have analyzed spiking and non-spiking regions from the human temporal lobe (n=25) as well as the hippocampus for catecholamines, amino acids, neuropeptides, enzymes and receptors. The cortical samples studied were surgically removed under general anesthesia or frozen and fixed within one minute after resection. In each case the non-spiking region was compared to the spiking region from the same patient. The catecholamines were elevated in the spiking region when compared to the non-spiking region: norepinephrine +47.4%, dopamine +58.4% and DOPA +23.5%. Several putative neurotransmitter amino acids were also elevated in the spiking region: glutamate +96.7%, aspartate +209.1%, and glycine +75.6%. GABA, alanine, taurine, and leucine were unchanged in the spiking vs non spiking regions. Of the neurotransmitter enzymes investigated the spiking cortex elevated tyrosine hydroxylase +60.2%, and choline acetyltransferase +57.1%. Glutamate decarboxylase was unchanged. The epileptic cortex had elevated somatostatin +313%, neuropeptide Y +128%, and atrial natriuretic factor +42.7%. The levels of β -endorphin, metenkephalin, cholecystokinin, substance P and neurotensin were not different in the spiking vs non-spiking region of the cortex. Vasoactive intestinal polypeptide was decreased in the epileptic cortex by 25.7%. Of the receptors measured the spiking region had elevated NMDA receptors +145% but decreased muscarinic receptors -32.5%; b receptors -51.4%, a receptors -38.4%. GABA and benzodiazepine receptors were unchanged. The K_d measurements of the receptors showed no change. The hippocampus had glutamate, aspartate, glycine, somatostatin, and neuropeptide Y comparable to the spiking cortex. The increase in somatostatin in the spiking region may have contributed to the excitability of the focus, since this molecule regulates the action of acetylcholine. Glycine by virtue of increasing the potency of glutamate at the NMDA sites may also have contributed to local excitability. The increases in catecholamines may be interpreted as a secondary phenomenon in response to the increased excitability of the region. The histochemical studies in human brain showed a diffuse distribution of tyrosine hydroxylase and a localization of

somatostatin to layers II, III, and V. The distribution of neuropeptide Y was similar to that of somatostatin indicating possible colocalization of the peptides.

10:35

10. Diencephalic Seizures in Posttraumatic and Hydrocephalic Patients

Eugene Rossitch, Jr., M.D., Dennis E. Bullard, M.D.
Duke University Medical Center

In 1929, Penfield described a patient with acute episodes of autonomic dysfunction. He termed these episodes diencephalic seizures (DS). Subsequently, eight additional cases have been reported by various authors. Nine new cases of diencephalic seizures characterized by autonomic dysfunction and extensor posturing are presented. Seven of the patients presented following trauma. In two others, and in one of the posttraumatic patients, the diencephalic seizures appeared to be closely correlated with episodes of hydrocephalus.

Despite the different precipitating factors, the diencephalic seizures were similar in many respects. Hyperthermia, hypertension, tachypnea, pupillary dilation, and tachycardia were seen in all our patients. Increased extensor posturing was a component of the DS in eight of nine cases. Diaphoresis was seen in seven of nine patients. ICP was measured in five posttraumatic patients and was a component of the DS in two. The rise in ICP in these two cases always occurred after the DS were in progress. The episodes lasted from minutes to hours with onset of the DS correlated with the development of hydrocephalus in three patients. In the remaining six patients, the episodes began within 24 hours of the traumatic event.

Morphine was given to three patients and in all cases stopped the episodes. Dantrolene was given to one patient and reduced the severity of the extensor posturing without affecting the other components of the DS. Bromocriptine was given to three patients and appeared to have both short term and long term effects. Acutely, the drug partially corrected the hyperthermia and diaphoresis associated with these episodes. Two patients were given bromocriptine chronically. In one patient, the DS were completely controlled and in the other, the frequency of the episodes decreased.

In our posttraumatic cases, DS may represent a cortical release phenomenon. However, the mechanism appears to be different in our hydrocephalic patients. Pharmacologically, the

responses to bromocriptine and morphine appear to indicate a role for both dopaminergic and opiate systems in diencephalic seizures.

10:55

11. Severe Head Trauma and Direct Hospital Costs in Predictable Non-Survivors—A Sample Survey

Jose Rodriques, M.D., Eldon Foltz, M.D.

University of California Irvine Medical Center

Experienced neurosurgeons have a sense of futility when patients with a severe brain injury are admitted to the hospital after an emergency room evaluation demonstrates that the patient: (1) has suffered severe brain trauma with presisting apnea within the preceding 60 minutes, (2) has a persisting Glasgow Coma Scale of 3 or 4, (3) has been intubated and is on mechanical ventilation, and (4) has normal blood gases with negative drug screen.

A review of 42 such cases brought to the emergency room at UCI Medical Center during 1986 has been done, using only selection criteria as above. The goal was two-fold: (1) to assess survival under very aggressive Neurosurgical and Trauma Service management, and (2) to calculate direct costs of these efforts

RESULTS

1. Clinical Population (Sex/Age): 7 females/9 to 38 yrs
35 females/3 to 71 yrs
2. Three groups of patients (Groups I, II, III) were identified with specific characteristics and results:

	Initial GCS	Predictive Trauma Score	5-Day Mortality	# Patients
Group I	3	<2	100%	30
Group II	3	3 to 4	100%	7
Group III	4	5	0%	5

(Groups I and II had multi-system injuries; Group III had only brain injury.)

3. Treatment:

	# Patients	Non-Survivors	Survivors
Operative	17	13	4
Non-Operative	25	24	1
	42	37	5

(brain injury only)

4. Costs

Direct costs billed to health provider for the 37 non-survivors was \$990,000 total, or \$27,000 per patient.

These 37 patients could have been identified as non-survivors in the E.R. and not admitted. Direct costs then would have been \$7,400 total, or \$200 per patient.

Are we really using our considerable professional skills at the appropriate ethical level?

11:15

12. Effects of Experimental Cerebral Revascularization on rCBF and Hypercapnic Reactivity

Christopher M. Loftus, M.D., Julius A. Silvini, M.D., Daniel D. Bernstein, B.S.

Iowa City Veterans Administration Hospital and The University of Iowa Hospitals

rCBF was measured with radiolabeled microspheres in a canine model simulating prophylactic and delayed cerebral revascularization (standardized STA-MCA bypass followed by occlusion of ipsilateral A2, ophthalmic, ethmoidal, MCA, PCoA, anterior cerebellar arteries). Our hypothesis was that prophylactic bypass

would be superior to delayed revascularization in supporting rCBF and preserving hypercapnic reactivity following acute ischemia.

rCBF measurements in seven dogs with the bypass first closed ($68.9 \pm 10\text{SEMcc}/100\text{gm}/\text{min}$) and then opened (76.8 ± 8.9) showed no significant contribution of bypass flow in the normal brain. Following vascular occlusion, rCBF was preserved by bypass flow (60.18 ± 8.95). Bypass clipping produced a significant flow decrease (7.95 ± 2.55 , ANOVA $p < 0.05$). Reopening of the bypass following 15' ischemia restored 76% of previous flow (46.61 ± 6.29). This was a significant increase from global ischemia values ($p < 0.05$), and not statistically different from pre-occlusive values.

In the CO_2 experiments, (six animals) flow was preserved and some hypercapnic response (not statistically significant) remained following proximal occlusion with patent bypass ($42.55 \pm 3.13\text{SEM cc}/100/\text{gm}/\text{min}$ @ $p\text{CO}_2$ 40, 62.52 ± 4.70 @ $p\text{CO}_2$ 70). In the opposite hemisphere hypercapnia produced much greater increases (217%) in rCBF (ANOVA $p < 0.05$). During complete ischemia (bypass occluded) hypercapnia produced no rCBF increase (4.23 ± 1.77 @ $p\text{CO}_2$ 40, 3.94 ± 2.34 @ $p\text{CO}_2$ 70). Significant flow was restored to the ischemic area ($p < 0.05$) following bypass reopening, but hypercapnia produced an rCBF decrease consistent with post-ischemic vasomotor paralysis and vasodilatory steal by the contralateral normal hemisphere (66.98 ± 9.71 @ 40torr, 34.47 ± 7.0 torr).

CONCLUSIONS: 1) Prophylactic bypass flow did not increase native hemispheric rCBF, but was sufficient to protect against acute vascular occlusion and was superior to delayed graft reopening. 2) Some hypercapnic reactivity, certainly less than normal, was preserved by bypass flow even following acute ischemia, while delayed revascularization was ineffective in restoring hypercapnic reactivity despite return of significant flow to the previously ischemic region. 3) Our data supports the usefulness of prophylactic STA-MCA bypass as a protective measure prior to elective carotid sacrifice or in surgery where the possibility of vascular injury is high.

11:35

13. Long Term Clinical and Radiographic Follow-Up in Patients With Vertebral to Carotid Transpositions

Willis E. Brown, Jr., M.D., David A. Cavanaugh, M.D. Jim L. Story, M.D.

University of Texas Health Science Center-San Antonio

The authors report their long term clinical and radiographic follow-up after end-to-side vertebral artery to common carotid artery transposition for the treatment of posterior circulation ischemia. This series began in 1978 and complete clinical follow-up has been obtained on all 20 patients in the series. Ten patients are living, 4-1/2 - 7 years following operation (73 months average). Radiographic follow-up has been 95%. Over the last three years, intravenous digital subtraction angiography has been used for routine follow-up studies. Three postoperative fatalities have occurred: 2 from acute myocardial infarction and 1 from acute occlusion of the transposed vertebral artery with a propagating thrombus to the basilar artery. The 7 late deaths were all from non-neurologic causes. Total relief of symptoms was achieved in 75% of the patients, and improvement was noted in an additional 10% of patients. The results with interposition grafts of PTFE (8 patients) and with saphenous vein segments (3 patients) were also reviewed. There have been no progressive stenoses, and only one late occlusion (in a vein) has occurred. Vertebral artery to common carotid artery transposition is a valuable procedure: it can relieve ischemic symptoms in carefully selected patients with vertebral basilar insufficiency and it can produce long term patency rates.

11:55

14. Surgical Management of Spinal AVMs

Robert F. Spetzler, M.D., Joseph M. Zabramski, M.D.
Barrow Neurological Institute

We present our experience in the surgical management of 22 consecutive patients with spinal AVMs. Fourteen patients had Type I (dorsal) lesions, seven had Type II (glomus) lesions, and one had a Type III (juvenile) spinal AVM.

In the literature the Type I (dorsal) spinal AVM is usually described as a localized extramedullary malformation, commonly related to the dural root sleeve, that is fed by a single spinal root artery, with drainage via the coronal spinal venous plexus. Based on our experience, however, we believe that there is a second type of dorsal spinal AVM that is fed by multiple arterial branches: We have termed this Type I (B). This

type of malformation may show multiple arterial feeders or be angiographically occult. At surgery, this type of lesion has the typical dorsal venous-arterial plexus but has multiple small feeders arising from more than one spinal root artery. These lesions may explain the occasional recurrence of dorsal spinal AVMs after single vessel obliteration. In our series of 14 patients with dorsal spinal AVMs, three cases fit the criteria described above for classification as Type I (B).

We are also presenting the first reported case of complete obliteration of a Type III (juvenile) spinal AVM.

Overall the results of surgical management in this series is as follows:

<u>Outcome</u>	<u>Type I</u>	<u>Type II</u>	<u>Type III</u>
Improved	10	5	1
Unchanged	3	2	
Worse	1		

The evaluation, classification, and surgical management of these patients will be discussed.

12:30 Group Photo at the Alamo

Saturday, October 10

SCIENTIFIC SESSION VII

MODERATOR: Theodore Roberts, M.D.

8:00 Symposium on Stereotaxis

Edward Ganz, M.D.

M. Peter Heilbrun M.D.

9:30 Coffee/Tea

SCIENTIFIC SESSION VIII

MODERATOR: M. Stephen Mahaley, M.D.

9:50

15. The Value of Image Guided Stereotactic Localization in the Management of Vascular Lesions of the Brain

M. Peter Heilbrun, M.D., Mark V. Reichman, M.D., Brian K. Willis, M.D.

University of Utah Health Sciences Center

From 1980 to 1987, our group has performed over 300 operative procedures utilizing the Brown-Roberts-Wells (BRW) image guided stereotactic system for localization and guidance of intra-axial brain lesions. The first 100 cases we reported were predominantly for biopsy alone¹. We now use the system more often as an operative platform for a wide range of operative procedures once localization with either CT, MRI, or angiography and guidance has been accomplished.

During the period of the BRW system evolution, there has been a concomitant advancement in brain imaging techniques, resulting in a more refined classification of vascular lesions into typical high flow arteriovenous malformation and low to no flow lesions including capillary telangiectasia, cavernous angiomas, and venous angiomas. We described the evolution of our stereotactic technique in the management of both high flow and low flow vascular lesions over the past seven years.

¹Heilbrun, MP, Roberts, TS, Apuzzo, MJ, Wells, TH, Sagshin, JK. Preliminary experience with Brown-Roberts-Wells (BRW) computerized tomography stereotaxic guidance system. *J. Neurosurg* 59:217-222, 1983.

10:10

16. Genetic Mechanisms of Tumorigenesis in Neurofibromatosis

Robert L. Martuza, Bernd R. Seizinger, Raymond A. Sobel, Guy Rouleau, Andrew H. Lane, James F. Gusella
Massachusetts General Hospital

Two autosomal dominant forms of neurofibromatosis (NF) are well characterized. NF-1 (von Recklinghausen NF; peripheral NF) is associated with pigmentary abnormalities (cafe-au-lait spots), iris hamartomas, optic gliomas, and multiple neurofibromas. NF-2 (bilateral acoustic NF; central NF) is characterized by the development of bilateral acoustic neuromas in association with meningiomas, astrocytomas and neurofibromas. The gene for NF-1 is on chromosome 17 and the gene for NF-2 is on chromosome 22. Because tumors histologically similar to

those in NF also occur sporadically in the normal population, mechanisms of tumor formation determined for NF may also apply to tumors in patients without NF.

We used recombinant DNA techniques to study tumors of NF-1 and NF-2. Genomic DNA was isolated from tumor tissue and lymphocytes and typed with four polymorphic markers on chromosome 22 (SIS; D22S1; D22S9; IGLC), four markers on chromosome 17 (NGF-R; GH; TK; PTHH59), and multiple markers on other chromosomes. In NF-2, four acoustic neuromas, one meningioma, and three spinal neurofibromas have been informative and consistently show loss of genes only on chromosome 22. In patients with multiple tumors, the gene loss occurred on the same chromosomal copy in each tumor. This suggests the possibility that tumor formation in NF-2 occurs by loss or inactivation of a recessive "tumor suppressor" gene analogous to that previously described for retinoblastoma. In contrast, studies of multiple tumor types in NF-1 (33 cutaneous neurofibromas, 3 optic gliomas, 1 cerebellar astrocytoma, 2 other gliomas, 1 spinal neurofibroma, 1 neurofibrosarcoma) have not shown any loss of genes on chromosome 17, 22, or other chromosomes studied. To minimize the possibility that these negative findings were caused by contamination of tumors with stromal elements, the tumors were immunohistochemically stained with antibody to S100 protein and the percentage of S-100 positive cells in each specimen was assessed. While some neurofibromas contained substantial non-staining portions, others contained >90% S-100 positive cells of presumed Schwann cell origin. This suggests the possibility that the genes for NF-1 and NF-2 are not only on different chromosomes but also may induce tumorigenesis by different mechanisms.

10:30

17. Auto-Immune Mechanism of Vasospasm

J. Peterson, Ph.D., Takao Bun, M.D., Nicholas Zervas, M.D.
Massachusetts General Hospital

In the hemorrhaged canine model of cerebrovasospasm, the first experimental subarachnoid hemorrhage produces little or no reaction in the basilar artery within three days. A second subarachnoid hemorrhage, 72 hours after the first, produces, however, fully developed vasospasm in the next 48 hours. Experiments in our laboratories suggest that this process involves an auto-immune reaction between the agent blood clot in the peradventitial space and the freshly injected blood. In experiments in which foreign bodies (dextran or latex beads)

or immunological incompatible whole blood (human) are injected into the basal cistern, cerebral vasospasm develops within 6-8 hours and persists more than 72 hours. In these circumstances, the periadventitial space as a basilar artery is massively invaded by macrophages and other immuno-reactive lymphocytes. Critical studies of subarachnoid hemorrhage patients have shown that the development of cerebral-vasospasm correlates strongly with higher than control levels of activated serum immuno-complexes.

In 10 animals, we attempted to intervene against this hypothesized immuno-reactive process by administration of Cyclosporine A initiated prior to the second subarachnoid hemorrhage and continued daily until sacrificed at 72 hours after the second subarachnoid hemorrhage. This regimen was moderately effective in blocking the development of cerebrovasospasm. Compared to controlled results in untreated animals, Cyclosporine-A reduced the severity of the angiographic constriction by 40-50%. These results are encouraging since Cyclosporine-A treatment should block only one of two possible mechanisms of complement protein activation. It is possible that complete blockade of complement protein activation will completely block cerebrovasospasm after subarachnoid hemorrhage.

10:50

18. Clinical Physiological Investigation of Deafferentation and Central Pain

R. Tasker, J. Gorecki, F. Lenz, T. Hirayama, J. Dostrovsky
Toronto General Hospital

The physiological localization necessary for functional neurosurgery operations offers an almost serendipitous opportunity to study brain function in health and disease. If microelectrodes are used to record thalamic single cells and to microstimulate at the same site with the same electrode, a unique strategy becomes available. The afferent path can be studied from receptor to thalamus by recording and the thalamofugal path to consciousness and, presumably, cortex by microstimulation. Abnormalities of location and size of receptive field, of spontaneous and induced firing rate, latency, and response to various manipulations of the thalamic neurons, and abnormalities of stimulation threshold and quality, and location of induced response can all be correlated with one another and with the clinical picture.

We have begun an off-line analysis of our data in 35 patients and 1 "control" and have found the following abnormalities in patients with deafferentation and central pain:-

Somatotopic Reorganization. Portions of thalamus normally devoted to the deafferented body part may be taken over by afferents with apparently normal receptive fields in other parts of the body, or with unusual receptive fields. Ongoing projection consciousness apparently may be rearranged as well.

Firing Patterns of Neurons. In the thalamus of patients with central or deafferentation pain, bursting cells are widely distributed and firing patterns at somatotopically deranged sites may be altered. It is impossible to say whether these two processes are related to pain or merely deafferentation.

Quality of Stimulation Reduced Responses. In parts of the thalamus related to deafferented body parts, microstimulation may induce two abnormal somatosensory responses, burning and pain. The latter appears confined to patients with hyperpathia—a central hyperpathia if you will. Previous observations on the stimulation induced perception of pain suggest that both reticulo- and spino-thalamic pathways are involved in the pathophysiology of hyperpathia and that the reticulothalamic system acquires access to consciousness and somatotopographic organization in the process.

11:10

19. Cranial Pains Relieved by DREZ Lesions of the Trigeminal Nucleus Caudalis

B.S. Nashold, Jr., M.D., Estrada Bernard, M.D., Franco Caputi, M.D., John J. Moosy, M.D.

Duke University Medical Center

Chronic intractable pain of the head always presents a challenge to the neurosurgeon. Eighteen patients with chronic intractable head pain have been treated by localized coagulation of the nucleus caudalis of the trigeminal nerve at the medullary junction of the spinal cord with an overall relief of pain in 58%. It is known that the majority of pain afferents from the cranial structures (5,7,9,10,12) are localized in or near the nucleus caudalis and the secondary neurons in this nucleus send their central fibers across the midline medially to more cephalad levels of the midbrain and thalamus.

The eighteen patients presented with a variety of intractable pains including post-tic trigeminal pain unsuccessfully treated. Several patients had unusual sources for their pain, such as orbital cancer, and one woman with a unique type of bilateral

painful glaucoma. The best initial relief of pain occurred in those patients with post herpetic involvement (5/7) all with good relief. About one in three of the post tic pain patients were relieved and chronic dental pain was relieved in one of three patients with good relief of pain in the one patient with unilateral orbital cancer and in one patient with bilateral glaucoma pain. One patient had intractable pain due to involvement of tumor of the trigeminal nerve in the posterior fossa. Another patient suffering from localized pain around the lower part of the jaw and oral cavity due to multiple surgical procedures to relieve chronic salivary gland calculi. CONCLUSION: Difficult and unusual cranial pains may be relieved by localized lesions of the trigeminal nucleus caudalis.

11:30

20. Delivery of Enzyme and Genes Across the Blood-Brain Barrier

Edward A. Neuwelt, M.D.

Oregon Health Sciences University

An autosomal recessive feline model of GM₂ gangliosidosis in the Korat cat that is highly analogous to the human Sandhoff disease has been characterized in our laboratory. In previous studies, normal human hexosaminidase, the enzyme deficient in GM₂ gangliosidosis, has been delivered across the blood-brain barrier in normal rats after osmotic blood-brain barrier modification. After crossing the barrier, the enzyme has been demonstrated to enter cells and then subcellular organelles presumed to be the lysosome, the normal site of the enzyme. Since the normal human gene for hexosaminidase has been recently cloned by Dr. Roy Gravel and provided to us for evaluation in the feline model of GM₂ gangliosidosis current studies are focusing on delivering this gene across the blood-brain barrier. The gene has been packaged in a replication defective retrovirus vector by Drs. Richard Bestwick and David Kabat at our institution. Sandhoff fibroblasts are being infected *in vivo* with this vector in an attempt to restore active enzyme to these cells. Simultaneously, parameters have been established to open the blood-brain barrier in the feline using hypertonic arabinose. In the past, this has been difficult using standard agents such as mannitol because of the absence of a patent internal carotid artery in the feline after gestation. It is planned to give the replication defective vector containing the cDNA clone directly *in vivo* either intrathecally or in association with osmotic blood-brain barrier modification. Because the vector is

replication-defective, it poses little in the way of a biohazard. In summary, the following question is being asked: Can the normal human gene be delivered across the blood-brain barrier and inserted into cells in a functional state in cats with GM₂ gangliosidosis?

11:50

21. Retroperitoneal Hematoma With Femoral Neuralgia

Suzie G. Tindall, M.D.

Emory University School of Medicine

Experience with the treatment of five cases of retroperitoneal hematoma with associated femoral neuralgia forms the basis for this report. Spontaneous retroperitoneal hematoma occurs most often in patients taking anticoagulants. Symptoms generally follow minor trauma to the leg or hip such as stepping into a hole. Patients complain of inability to walk, severe deep aching pain in the hip and thigh, and paresthesias in the distribution of the femoral nerve. Examination discloses inability to extend the leg at the hip, diminished knee jerk, and variable hypesthesia in the thigh and anterior foreleg. Quadriceps function is difficult to assess due to patient discomfort. A bruise may appear in the flank.

CT scanning demonstrates hematoma in a characteristic location beneath the iliacus muscle posteromedial to the hip joint.

Of the treatment options available experience has shown that surgery provides the most effective relief of symptoms and potential for recovery of neurological function. Following reversal of anticoagulation, surgical treatment is indicated for evacuation of the hematoma and decompression of the femoral nerve which may be entrapped beneath the inguinal ligament. The surgical approach and operative findings will be discussed.

12:10

22. The Effect of Dilantin on Cognitive Function in Patients Recovering From Cerebral Trauma

Kenneth R. Smith, Jr., M.D.

St. Louis University

The effects of Dilantin (phenytoin) given prophylactically to patients recovering from head injury or neurosurgical procedures is being studied using a double blind placebo controlled parallel group design. Patients who are ready to discontinue prophylactic monotherapy with Dilantin after being treated for 4-24 months are given a battery of 18 psychometric tests on four different occasions over a 14 week period. Two tests are

given for a baseline four weeks apart. Patients are then given double blind medication and in five weeks they are given a third test at which time half are receiving Dilantin and half are receiving placebo. Then all medication is withdrawn and patients are tested again in four weeks. The tests are designed to assess tension, concentration, attention, visual discrimination, visual scanning, mental flexibility, psychomotor speed, immediate and delayed recall, verbal memory, visual memory, reaction time and coordination, verbal fluency, IQ, and depression/anxiety.

The results of four serial testing sessions in each of the first 10 patients entered into the study will be presented. Previous studies reporting impaired cognition from Dilantin during short term administration to normal volunteers or in chronic epileptics who have been treated for long periods of time may not be applicable to most neurosurgical patients who are receiving prophylactic anticonvulsants. This study will determine the effect of Dilantin given for many months to patients without seizures who have minimal or no neurological deficit.

(Supported by a grant from Warner-Lambert Co.)

12:30 Adjourn

NOTES

RESIDENTS PAPER AWARD WINNERS

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Joseph R. Madsen, M.D., Dora W. Hsu, M.S.,
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*Neurosurgery and Neuropathology,
Massachusetts General Hospital and Harvard Medical School*

"Expression of X-hapten Immunoreactivity
by Human Rat Adenohypophyseal Cells:
Alternation in Tumors and Estrogen-induced Hyperplasia"

RUNNER UP

Donald W. Marion, M.D. and Raymond D. Lund, Ph.D.

*Dept. of Neurosurgery and Neurobiology
University of Pittsburgh School of Medicine*

"Projections from Neocortex Grafts
Transplanted into the Rodent Brain Stem"

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WALLACE B. HAMBY (Eleanor) 3001 N.E. 47th Court Fort Lauderdale, Florida 33308	1938
JESS D. HERRMANN (Mary Jo) P.O. Box 135 Mountain Pine, Arkansas 71956	1948
WILLIAM E. HUNT University Hospital 410 West 10th Avenue Columbus, Ohio 43210	1970
WILLIAM S. KEITH (Eleanor) 55 St. Leonards Crescent Toronto, Ontario, Canada M4N 3A7	Founder
ROBERT S. KNIGHTON (Louise) 9388 Avenida San Tinetto Cherry Valley, California 92223	1966
WILLIAM M. LOUGHEED (Grace) Toronto General Hospital 101 College Street Toronto, Canada M5G 1L7	1962
JOHN J. LOWREY (Catherine "Katy") P.O. Box 4302 Kawaihae, Hawaii 96743	1965
GEORGE L. MALTBY (Isabella "Sim") 470 Black Point Road Scarborough, Maine 04074	1942
FRANK MAYFIELD (Belle Clay) 506 Oak Street Cincinnati, Ohio 45219	Founder
AUGUSTUS McCRAVEY (Helen) 1010 East Third Street Chattanooga, Tennessee 37403	1944
WILLIAM F. MEACHAM (Alice) Vanderbilt University Hospital Division of Neurosurgery Nashville, Tennessee 37232	1952
FRANCIS MURPHEY (Marge) 3951 Gulf Shores Road Naples, Florida 33940	Founder
GUY L. ODOM (Mataline) 2812 Chelsea Circle Durham, North Carolina 27707	1946

J. LAWRENCE POOL (Angeline) P.O. Box 40 West Cornwell, Connecticut 06796	1940
ROBERT W. PORTER (Aubrey Dean) 6461 Bixby Hill Road Long Beach, California 90815	1962
ROBERT H. PUDENZ (Rita) 574 Garfield Avenue South Pasadena, California 91030	1943
JOHN RAAF (Lorene) 1120 N.W. 20th Avenue, #100 Portland, Oregon 97209	Founder
AIDEN A. RANEY (Mary) 2010 Wilshire Boulevard Los Angeles, California 90057	1946
THEODORE B. RASMUSSEN (Catherine) 29 Surrey Drive Montreal, Quebec, Canada H3P 1B2	1947
HENRY G. SCHWARTZ (Reedie) Barnes Hospital Plaza St. Louis, Missouri 63110	1942
C. HUNTER SHELDEN (Elizabeth) 734 Fairmont Avenue Pasadena, California 91105	1941
WILLIAM H. SWEET (Elizabeth) 309 Goddard Avenue Brookline, Massachusetts 02146	1950
JOHN TYTUS (Virginia "Gina") Mason Clinic Seattle, Washington 98107	1967
ALFRED UHLEIN (Ione) 200 First Street, S.W. Rochester, Minnesota 55901	1950
A. EARL WALKER (Agnes) 1477 Wagontrain Drive, S.E. Albuquerque, New Mexico 87123	1938
EXUM WALKER (Nelle) 490 Peachtree Street, N.E. Atlanta, Georgia 30308	1938
BENJAMIN B. WHITCOMB (Margaret) RFD 1 P.O. Box 124 Surrey, Maine 04684	1947

*ACTIVE MEMBERS**ELECTED*

- JAMES I. AUSMAN (Carolyn) 1978
Henry Ford Hospital
2799 West Grand Blvd.
Detroit, Michigan 48202
- GILLES BERTRAND (Louise) 1967
Montreal Neurological Institute
3801 University Street
Montreal, Quebec, Canada H3A 1B4
- ROBERT S. BOURKE (Marlene) 1983
5802 Nicholson Lane
Rockville, Maryland 20852-2967
- JERALD S. BRODKEY (Arielle) 1977
24755 Chagrin Boulevard
Suite #205
Beachwood, Ohio 44122
- WILLIS E. BROWN, JR. (Ann) 1984
Division of Neurosurgery
The University of Texas Health Science Center
7703 Floyd Curl Drive
San Antonio, Texas 78284-7843
- DEREK A. BRUCE (Frances) 1984
34th-Civic Ctr. Blvd.
Division of Neurosurgery
Philadelphia, Pennsylvania 19014
- WILLIAM A BUCHHEIT (Lin) 1980
3401 North Broad Street
Philadelphia, Pennsylvania 19140
- PAUL H. CHAPMAN (Tansy) 1983
Department of Neurosurgery
Massachusetts General Hospital
Boston, Massachusetts 02114
- SHELLEY N. CHOU (Jolene) 1974
University of Minnesota Medical Center
Minneapolis, Minnesota 55455
- W. KEMP CLARK (Fern) 1970
5323 Harry Hines Blvd.
Dallas, Texas 75235
- 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 70018
- JAMES W. CORRELL (Cynthia) 1966
 710 West 168th Street
 New York, New York 10034
- COURTLAND H. DAVIS, JR. (Carrie) 1967
 Bowman Gray School of Medicine
 Winston-Salem, North Carolina 27103
- 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
 N.Y.U. Medical Center
 550 First Avenue
 New York, New York 10016
- ELDON L. FOLTZ (Catherine) 1960
 UCI Medical Center, Division of Neurosurgery
 101 City Drive, S.
 Orange, California 92668
- 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

- SIDNEY GOLDRING (Lois) 1964
 Barnes Hospital Plaza
 Division of Neurosurgery
 St. Louis, Missouri 63110
- ROBERT G. GROSSMAN (Ellin) 1984
 Baylor College of Medicine
 6501 Fannin, #A404
 Houston, Texas 77030
- ROBERT GRUBB (Julia) 1985
 Barnes Hospital Plaza
 St. Louis, Missouri 63110
- JOHN W. HANBERY (Shirley) 1959
 Division of Neurosurgery
 Stanford University Medical Center
 300 Pasteur Drive
 Stanford, California 94305
- GRIFFITH R. HARSH, III (Craig) 1980
 University of Alabama Medical Center
 Birmingham, Alabama 35294
- MAJ. GEN. GEORGE S. HAYES (Catherine) 1962
 MC USA
 303 Skyhill Road
 Alexandria, Virginia 22314
- MARK PETER HEILBRUN (Robyn) 1984
 Division of Neurosurgery, #3B320
 University of Utah Medical Center
 Salt Lake City, Utah 84132
- E. BRUCE HENDRICK (Gloria) 1968
 Hospital for Sick Children
 555 University Ave., Room 1502
 Toronto, Ontario, Canada M5G 1X8
- ROBERTO C. HEROS (Deborah) 1985
 Department of Neurosurgery
 Massachusetts General Hospital
 Boston, Massachusetts 02114
- CHARLES HODGE (Linda) 1982
 Department of Neurosurgery
 Upstate Medical Center
 Syracuse, New York 13210
- JULIAN HOFF (Dianne) 1975
 Department of Neurosurgery
 University of Michigan
 Ann Arbor, Michigan 48104

- 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
 710 West 168th Street
 New York, New York 10032
- ALAN R. HUDSON (Susan) 1978
 St. Michael's Hospital
 38 Shutter Street
 Toronto, Ontario, Canada M5B 1A6
- JOHN A. JANE (Noella) 1982
 Department of Neurosurgery
 University of Virginia
 Charlottesville, Virginia 22901
- JOHN P. KAPP (Lucrese) 1985
 Department of Neurosurgery
 University of Buffalo
 50 High Street, #1202
 Buffalo, New York 14203
- ELLIS B. KEENER (Ann) 1978
 915 East Lake Drive, NW
 Gainesville, Georgia 30506
- DAVID KELLY (Sally) 1975
 Bowman Gray School of Medicine
 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
- ROBERT B. KING (Molly) 1958
 University Hospital
 Upstate Medical Center
 750 East Adams Street
 Syracuse, New York 13210

- 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 70012
- RICHARD S. KRAMER (Robin) 1978
Duke Hospital
Durham, North Carolina 27710
- THEODORE KURZE 1967
10 Congress Street
Suite 340
Pasadena, California 91105
- THOMAS W. LANGFITT (Carolyn) 1971
Hospital of University of Pennsylvania
34th and Spruce Streets
Philadelphia, Pennsylvania 19104
- EDWARD R. LAWS, JR. (Peggy) 1983
Mayo Clinic
Rochester, Minnesota 55905
- RAEBURN C. LLEWELLYN (Carmen) 1963
5640 Read Boulevard
Suite 840
New Orleans, Louisiana 70127
- DONLIN M. LONG 1983
Department of Neurological Surgery
John Hopkins Medical School
Baltimore, Maryland 21205
- ALFRED J. LUSSENHOP 1976
Georgetown University Hospital
Washington, D.C. 20007
- ERNEST W. MACK (Bobbie) 1956
505 South Arlington Avenue
Suite 212
Reno, Nevada 89509
- M. STEPHEN MAHALEY, JR. (Jane) 1972
Division of Neurosurgery
University of Alabama Medical Center
Birmingham, Alabama 35294
- LEONARD MALIS (Ruth) 1973
1176 Fifth Avenue
New York, New York 10029

- ROBERT L. MCLAURIN 1955
111 Wellington Place
Cincinnati, Ohio 45219
- JOHN F. MULLAN (Vivian) 1963
5844 Stony Isle Avenue
Chicago, Illinois 60637
- BLAINE S. NASHOLD, JR. (Irene) 1967
Duke University Medical Center
Durham, North Carolina 27710
- FRANK E. NULSEN (Ginny) 1956
University Hospital of Cleveland
2074 Abington Road
Cleveland, Ohio 44106
- 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 55901
- RUSSEL H. PATTERSON, JR. (Julie) 1971
525 East 68th Street
New York, New York 10021
- S. J. PEERLESS (Ann) 1977
P.O. Box 5339
Terminal A
University Hospital
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
815 Eucalyptus Avenue
Hillsborough, California 94010

- DONALD O. QUEST (Hona) 1986
710 West 168th Street
New York, New York 10032
- JOSEPH RANSOHOFF, II (Lori Ellen) 1965
New York University Medical Center
550 First Avenue
New York, New York 10016
- 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
- HUGO RIZZOLI (Helen) 1973
2150 Pennsylvania Avenue, N.W.
Washington, D.C. 20037
- THEODORE S. ROBERTS (Joan) 1976
Madigan Army Medical Center
Neurosurgical Service
P.O. Box 2511
Tacoma, Washington 98431-5439
- JAMES T. ROBERTSON (Valeria) 1971
Department of Neurosurgery
University of Tennessee, Memphis
956 Court Avenue
Memphis, Tennessee 38163
- FREDRICK A. SIMEONE 1981
800 Spruce Street
Philadelphia, Pennsylvania 19107
- JAMES C. SIMMONS (Vanita) 1975
920 Madison Avenue
Memphis, Tennessee 38103
- BENNETT M. STEIN 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
 200 1st Street, S.W.
 Rochester, Minnesota 55901
- ANTHONY E. SUSEN (Phyllis) 1965
 3600 Forbes Avenue
 Pittsburgh, Pennsylvania 15213
- RONALD R. TASKER (Mary) 1971
 Toronto General Hospital
 Room 7-221E
 101 College Street
 Toronto, Ontario, Canada M5G 1L7
- JOHN TEW, JR. (Susan) 1973
 506 Oak Street
 Cincinnati, Ohio 45219
- GEORGE TINDALL (Suzie) 1968
 Emory University School of Medicine
 Division of Neurosurgery
 1365 Clifton Road, N.E.
 Atlanta, Georgia 30322
- JOHN C. VAN GILDER (Kerstin) 1980
 University of Iowa Hospital
 Iowa City, Iowa 55242
- ARTHUR A. WARD, JR. (Janet) 1953
 Department of Neurological Surgery
 RI-20
 University of Washington
 Seattle, Washington 98195
- CLARK WATTS (Patty) 1975
 One Hospital Drive
 Ste. N522
 Columbia, Missouri 65212
- BRYCE K. A. WEIR (Mary Lou) 1984
 University of Alberta
 Clinical Sciences Building
 Alberta, Canada T6G 2G3
- MARTIN H. WEISS (Debby) 1981
 USC Medical Center
 1200 North State Street
 Los Angeles, California 90033
- W. KEASLEY WELCH (Elizabeth) 1957
 Children's Hospital Medical Center
 300 Longwood Avenue
 Boston, Massachusetts 02115

SENIOR CORRESPONDING MEMBERS

KARL AUGUST BUSHE 1972
Neurochirurgischen Klinik
D-8700 Wurzburg
Josef-Schneider-Strasse II
West Germany

SHOZO ISCHII 1975
Department of Neurosurgery
Juntendo Medical College
Tokyo, Japan

KRISTIAN KRISTIANSEN (Kari) 1962
Oslo Kommune
Uleval Sykehus
Oslo, Norway

WILLIAM LUYENDIJK 1973
Pr Bernhardlaan 60
Oegstgeest, The Netherlands

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

- LOWELL E. WHITE, JR. (Margie) 1971
 University of Southern Alabama
 Division of Neuroscience
 Mobile, Alabama 36688
- ROBERT WILKINS (Gloria) 1973
 Duke University Medical Center
 Box 3807
 Durham, North Carolina 27710
- CHARLES B. WILSON 1966
 Department of Neurological Surgery
 University of California Medical Center
 Third and Parnassus
 San Francisco, California 94143
- FRANK WRENN (Betty) 1973
 27 Memorial Medical Drive
 Greenville, South Carolina 29605
- DAVID YASHON (Myrna) 1972
 50 McNaughton Road
 Columbus, Ohio 43213
- RONALD F. YOUNG, M.D. (Sheila) 1986
 University of California at Irvine
 101 City Drive
 Orange, California 92668
- NICHOLAS T. ZERVAS (Thalia) 1972
 Massachusetts General Hospital
 Boston, Massachusetts 02114

CORRESPONDING MEMBERS

- JEAN BRIHAYE (Martine Van Geertruyden) 1975
1 Rue Heger-Bordet
B-1000 Brussels, Belgium
- FERNANDO CABIESES 1966
Inst. Peruano De Formento Educativo
Av. Arenales 371, of. 501
Apartado 5254
Lima, Peru
- JUAN CARDENAS 1966
Neurologo 4 Neurocirujano
Av. Insurgentes Sur 594, Desp. 402
Mexico 12 D.F.
- JUAN C. CHRISTENSEN 1970
Ayacucho 2151 4 P
Buenos Aires, Argentina
- GUISEPPE DALLE ORE (Giushi) 1970
Dipartimento Di Neurochirurgia
Ospedale Maggiore 371000
Verona, Italy
- JACQUES DEVILLIERS 1986
Department of Neurosurgery
Groote Schuur Hospital
Observatory
7925 Cape Town
Union of South Africa
- HANS ERICH DIEMATH (Karin) 1970
Hofrat Univ. Prof. Dr. Med.
Traunstrasse 31
A5026
Salzburg, Austria
- HERMANN DIETZ 1980
Neurosurgical Clinic
Hannover School of Medicine
Hannover 3000-61
West Germany
- JOHN F. GILLINGHAM 1962
Royal Infirmary
Lauriston Place
Edinburgh, Scotland EH43 PB
United Kingdom

- JAMIE G. GOMEZ (Lucy) 1975
 Transversal 4 No. 42-00
 Comnutador 2-32 4070
 Bogota 8, Columbia
- SALVADOR GONZALEZ-COMEJO (Rosalie) 1982
 Av. Chapultepec Sur 130
 Guadalajara, Mexico 44100
- ERNEST H. GROTE (Julia) 1984
 Neurosurgery Department
 University Clinic
 7400 Tubigen
 Federal Republic of Germany
- HAJIME HANDA 1985
 Hamamatsu Rosai Hospital
 25 Shogen-Cho, Hamamatsu
 Japan 430
- JOHN HANKINSON 1973
 Department of Neurological Surgery
 Newcastle General Hospital
 Newcastle-Upon-Tyne 4
 England
- FABIAN ISAMAT 1986
 Clinica Sagrade Familia
 Torras y Pujalt, 1
 Barcelona 22, Spain
- HANS-PETER JENSEN (Reta) 1980
 Neurochirurgische Universitätsklinik Kiel
 Weimarer Strasse 8
 D-2300 Kiel/West Germany
- RICHARD JOHNSON 1974
 Department of Neurological Surgery
 Royal Infirmary
 Manchester, England
- KATSUTOSHI KITAMURA (Yoshiko) 1970
 University Kyushu Hospital
 Faculty of Medicine
 Maidashi, Fukuoka 812, Japan
- LAURI LAITINEN (Kerstin) 1971
 Department of Neurosurgery
 University Hospital
 S-901 85 Umea, Sweden

- WILLIAM MARGUTH 1978
 Director, Department of Neurochirurgischen
 Universitat Munchen
 Marchioninstrasse 15
 8000 Munchen 70, West Germany
- RAUL MARINO, JR. 1977
 Rua Maestro Cardim, 808
 S. Paulo - SP
 Brazil 01323
- B. RAMAMURTHI (Indira) 1966
 2nd Main Road G.I.T. Colony
 Madras 4, India 600 004
- CHARAS SUWANWELA 1972
 Chulalongkorn Hospital
 Medical School
 Bangkok, Thailand
- LINDSAY SYMON (Pauline) 1982
 The National Hospital
 Queen Square
 London, WC1E 3BG
 England
- KJELD VAERNET (Ann) 1970
 Department of Neurosurgery
 Rigshospitalet
 9 Blegdamsvej
 2100 Copenhagen, Denmark
- SIDNEY WATKINS 1975
 The London Hospital
 Whitechapel, London E 1
 England
- GAZI YASARGIL 1975
 Neurochirurgische
 Universitätsklinik
 Kantonsspital
 8000 Zurich, Switzerland

*DECEASED MEMBERS**ELECTED*

SIXTO OBRADOR ALCALDE Madrid, Spain (Honorary)	4/1967	
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
SPENCER BRADEN Cleveland, Ohio (Active)	7/1969	Founder
E. KEITH BRADFORD Houston, Texas (Active)	4/1971	1938
HARVEY CHENAULT Lexington, Kentucky (Senior)	1986	1938
WINCHELL McK. CRAIG Rochester, Minnesota (Honorary)	2/1960	1942
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
WESLEY A. GUFFAFSON Jensen Beach, Florida (Senior)	7/1975	1942

HANNIBAL HAMLIN (Senior)	6/1982	1941
HENRY L. HEYL (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
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
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 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 Nashville, Indiana (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

NOTES

NOTES

*THE AMERICAN ACADEMY OF
NEUROLOGICAL SURGERY
1987 ANNUAL MEETING
EVALUATION*

Please complete this evaluation form (omit those sessions or events you did not attend) and return to the Secretary, Nicholas Zervas, at your earliest convenience.

(1) Was the general context of the scientific program:

- Excellent
- Good
- Poor

(2) If you found it poor, was it because:

- Too much review of old knowledge?
- Too simple or elementary?
- Too complex or abstruse?
- Of little practical value?

(3) Did the speakers air their talks:

- Too high
- Too low
- Just about right

SCIENTIFIC PROGRAM

Thursday's Sessions Excellent Good Poor
Comments _____

Friday's Sessions Excellent Good Poor
Comments _____

Saturday's Sessions Excellent Good Poor
Comments _____

SOCIAL PROGRAM

Comments _____

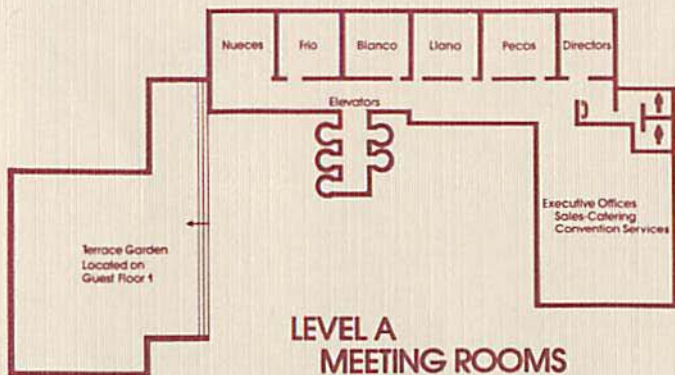
What changes would you like to see in future meetings? _____

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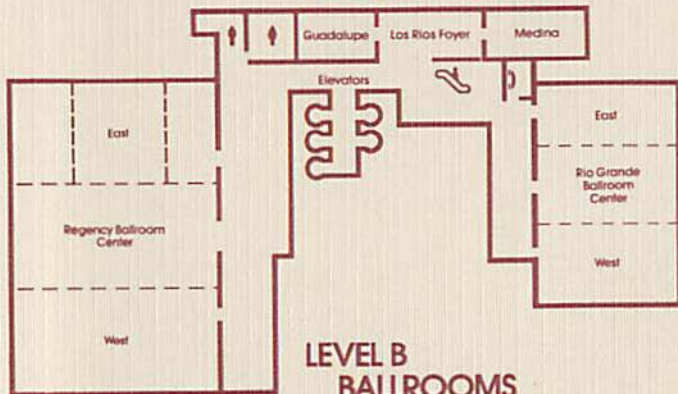
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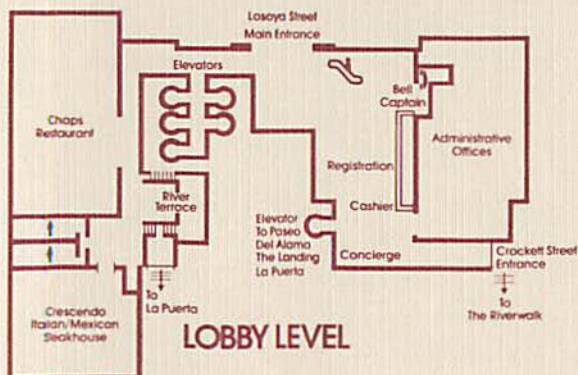
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LEVEL A MEETING ROOMS EXECUTIVE OFFICES



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512 222 1234 TELEX 767249
1-800-228-9000