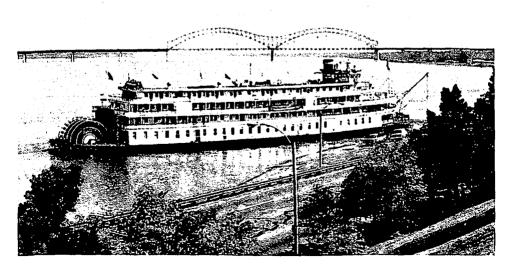
American Academy of Neurological Surgery

ANNUAL MEETING

Memphis, Tennessee November 7-10, 1979



ANNUAL MEETING 1979



Hyatt Regency Memphis Memphis, Tennessee November 7-10, 1979

THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

Officers 1979

President	Robert B. King
President-Elect	Eben Alexander, Jr.
Vice-President	H. Thomas Ballantine, Jr.
Secretary	Phanor L. Perot, Jr.
Treasurer	
Historian	Edwin B. Boldrey

Committees

Program Committee Chairman - Leonard Malis Julian Hoff S. J. Peerless Academy Award Committee Chairman · Thomas W. Langfitt Joseph Ransohoff Sidney Goldring

Membership Advisory Committee Chairman - William H. Sweet Arthur A. Ward, Jr. Robert B. King Phanor L. Perot, Jr. John T. Garner Robert G. Fisher Byron C. Pevehouse

Representative to Board of Directors AANS Raeburn C. Llewellyn

Subcommittee on Corresponding Membership Chairman - Charles Drake William Sweet Delegates to World Federation of Neurosurgical Societies Gilles Bertrand Russell H. Patterson

Round Robin Committee

John R. Green

Chairman - C. Hunter Shelden Geore Ehni Robert Wilkins John T. Garner Representative to Council of the National Society for Medical Research John Mullan

Representative to the ABNS Byron C. Pevehouse Representative to the International Committee on Neurosurgical Implants David G. Kline

Representative to the Inter-Agency Committee on Irreversible Coma and Brain Death A. Earl Walker

Local Hosts

Dr. and Mrs. James T. Robertson Dr. and Mrs. James C. H. Simmons Dr. and Mrs. Richard L. DeSaussure

PROGRAM 1979

REGISTRATION: GALLERY	
Wednesday, November 7	4:00-6:30 P.M.
Thursday, November 8	8:00-10:00 A.M.
That bady, 110 to this cro	2:00-4:00 P.M.
Friday, November 9	8:00-10:00 A.M.
i ilday, itovember 5	0.00 10.00 M.M.
WEDNESDAY, NOVEMBER 7	
6:30-9:30 P.M Welcomi	ng Cocktail-Buffet-Ridgeway B
THURSDAY, NOVEMBER 8	
7:00 A.M Breakfast and	Business Meeting-Ridgeway B
	(Members Only)
	Breakfast - Director's Room
	(Guests)
8:20-10:30 A.M.	Scientific Session-Ridgeway A
10:30-10:50 A.M.	Coffee Break-Foyer
10:50-12:00 noon	Scientific Session-Ridgeway A
12:00-1:00 P.M	
1:00-3:00 P.M.	Scientific Session-Ridgeway A
3:00-3:20 P.M.	Coffee Break-Foyer
3:20-4:00 P.M.	Scientific Session-Ridgeway A
5:30 P.M	Board Buses for Riverboat Trip
6:00-10:00 P.M River	boat Trip-Cocktails and Dinner
	-
FRIDAY, NOVEMBER 9	
7:00 A.M Breakfast and	Rusiness Mosting-Ridgeway R
1.00 A.M Dreaklast allu	(Members Only)
	Breakfast-Parlor 108 and 109
	(Guests)
8:30-9:50 A.M.	
9:50-10:10 A.M.	
10:10-12:00 noon	Scientific Session-Ridgeway A
12:30 P.M Lui	ochoon-Mamphis Country CLub
1:30 P.M Golf,	Tannis-Mamphis Country Club
Too I in	ur of Chucalissa Indian Village
7:00 P.M	
8:00 P.MMidnight	
Sing Firm Minning III Control of Control	Ridgeway B.
After Ding	ner Speaker: John J. Thomason
Arter Dilli	ici opcanci, uvim o, ritulliasun

SATURDAY, NOVEMBER 10	
8:00-9:00 A.M Breakfast and Business Meeting-Hugos	
(Members Only)	
Breakfast-Parlor 104 and 105	
(Guests)	
9:00-10:20 A.M Scientific Session-Ridgeway A	
10:20-10:50 A.M Coffee Break-Foyer	
10:50-12:00 noon Scientific Session-Ridgeway A	
LADIES PROGRAM	
WEDNESDAY, NOVEMBER 7	
6:30-9:30 P.M Welcoming Cocktail-Buffet-Ridgeway B	
0.50-5.50 1 .M Welcoming Cocktan-Dunet-Ittageway D	
THURSDAY, NOVEMBER 8	
8:00 A.M Hospitality Room, Continental Breakfast	
Parlor 112 and 113	
9:00 A.M Depart for Breakfast at Carriage House.	
Tour of Surrounding Homes in Victorian	
Village-The Fontaine House and Mallory	
House. Brief Bus Tour of Memphis including	
the Medical Center.	
2:00-4:00 P.M Tour of Dixon Gallery and Gardens	
5:30 P.M Board Buses for Riverboat Trip	
6:00-10:00 P.M Riverboat Trip-Cocktails and Dinner	
FRIDAY, NOVEMBER 9	
8:00 A.M Hospitality Room, Continental Breakfast	
Parlor 112 and 113	
9:00 A.M Depart for Coffee and Doghnuts Served	
at Woman's Exchange. Shopping to Follow.	
12:30 P.M Luncheon-Memphis Country Club	
1:30 P.M. Golf, Tennis-Memphis Country Club	
Tour of Chucalissa Indian Village	
7:00 P.M Cocktails, Shiloh Room	
8:00 P.MMidnight Banquet and Dancing-Ridgeway B	
After Dinner Speaker: John J. Thomason	
•	
SATURDAY, NOVEMBER 10	
8:00 A.M Hospitality Room, Continental Breakfast	
Parlor 112 and 113	
9:00 A.M Workshop and Demonstration of Silk Flowers	
Facials by Mary Kay Beauty Consultant	

SCIENTIFIC PROGRAM THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY Memphis, Tennessee November 7-10, 1979

MODERATOR: Leonard I. Malis

THURSDAY, November 8 8:20 a.m. Welcoming Remarks and Announcements

8:30 a.m.

1. A New Non-suture Technique for Rapid Vascular Anastomosis

W. M. Lougheed and F. Gentili Toronto, Ontario, Canada

Despite advances in microvascular techniques, problems in cerebrovascular reconstructive surgery remain, including that of providing an adequate blood volume to the brain and maintaining blood flow while the microscopic anastomosis is being created. We have developed a technique based on the use of a specially devised anastomotic clip and ring prosthesis which allows for rapid anastomosis of vessels without the need for placement of sutures. Twenty-five vascular anastomoses have been carried out in the dog using cartoid, femoral and brachial vessels ranging from 1 to 3 mm in diameter.

Time taken for actual anastomosis has averaged 10 to 12 minutes. The patency rate in animals followed from six weeks to six months with serial angiography has been 92%. Post-mortem histological and scanning electronmicroscopic studies have revealed excellent intima to intima junctional anastomosis with no evidence of thrombosis or stenosis. The high long-term patency rates appear to be related to the precise alignment and intima to intima relationship at the anastomotic site without the presence of foreign suture material.

These results demonstrate the technical feasibility of this technique which could easily be adapted for clinical use in man for the treatment of inaccessible intracranial regional stenotic lesions of the internal carotid, middle cerebral and vertebral arteries, as well as in dealing with large complex aneurysms.

Part of the presentation will include a short (5 to 6 minute) film to demonstrate this technique.

(Discussion)

2. Elective Extracranial Intracranial Arterial Bypass in the Treatment of Giant Aneurysms

R. F. Speltzler, H. Schuster and F. E. Nulsen Cleveland, Ohio

We are presenting a series of nine patients with large internal carotid artery aneurysms, treated by internal carotid artery ligation following the establishment of a patent extracranial intracranial arterial bypass. All aneurysms were deemed unsuitable for direct clipping by angiographic appearance or through direct exposure. Two patients presented with subarachnoid hemorrhage, and the remainder with evidence of intracranial mass lesion.

Operative Technique: The parietal or frontal branch of the superficial temporal artery is used to create a superficial temporal artery middle cerebral artery bypass. The internal carotid artery is then exposed in the neck and a Selverstone clamp placed around it and partially closed. Criteria for achieving 50% reduction in blood flow will be discussed.

On the second or third postoperative day, selective cerebral angiography is performed. The Selverstone clamp is closed completely when the bypass is proved patent.

Results: The bypass was patent in all patients. No patient experienced ischemic complications. The pre-op third nerve palsies disappeared over time and visual acuity improved in the visually compromised patients. No patient has had a subarachnoid hemorrhage in this relatively brief follow-up. Late angiography is planned for all patients - its present status will be presented.

Discussion: The rationale behind performing a bypass prior to internal carotid artery ligation is the obvious attempt to minimize ischemic complications. A more theoretical but seemingly plausible advantage is that the bypass would decrease the amount of collateral blood flow adjacent to the aneurysm with less turbulence at that site. Presumably, this could afford some protection to the aneurysm and enhance the chances for complete thrombosis.

We feel that the results of this admittedly small series suggest that whenever a carotid artery is to be ligated electively, an extracranial intracranial arterial bypass should be considered.

3. Strokes in Children - Their Relationship to Intrinsic Pathology of the Carotid Artery

Robert G. Fisher Plainfield, New Jersey

Vascular insufficiency of the carotid artery may cause inadequate perfusion to a child's brain of such extent that not only temporary but permanent neurologic deficit may ensue.

Alterations to the carotid artery in this unique group of cases have included the followina:

- 1. Tear in the innominate artery with secondary inadequate carotid flow.
- Extracranial carotid aneurysm with secondary cerebral embolus.
- 3. Thrombosis of the carotid artery antegrade to an aneurysm.
- 4. Kinking of the carotid artery.
- 5. Bilateral carotid artery thrombosis.
- Thrombosis of the carotid artery at the "siphon."

The etiology, therapy and results will be discussed. The recovery was poor in three cases.

(Discussion)

9:30 a.m.

4. Intracerebral Hemorrhage Following Carotid Endarterectomy

James W. Correll and Jack Stern New York, New York

The risk of intracerebral hemorrhage following carotid endarterectomy, in the presence of acute infarction, is well recognized. However, this complication may occur even though intensive investigation has apparently failed to demonstrate evidence of preoperative infarction.

Of more than 1,000 endarterectomies performed by the senior author, we have had experience with intracerebral hemorrhage in 10 patients in whom there had been no evidence of acute infarction. Preoperative evaluation (including angiography, computerized

tomography and EEG), the clinical course and possible etiological factors will be discussed. In addition, the indication for craniotomy and evacuation of the hematoma will be reviewed.

(Discussion)

9:50 a.m.

5. Angiography in Vertebral Basilar Insufficiency and Brainstem Infarction

James I. Ausman, Suresh Patel and Roushdy S. Boulos Minneapolis, Minnesota

Because of the supposed risks of angiography in vertebral basilar insufficiency (VBI) and brainstem infarction (BSI) and the lack of any surgical alternative in these diseases, there has been little angiographic evaluation of vascular patients with VBI and BSI.

In this presentation, examples selected from our experience with VBI and BSI will demonstrate lesions in the vertebral artery from its origin to its junction at the basilar, in the basilar itself and in combination which produce VBI and BSI. The treatment approaches to these lesions, medical and surgical, will be outlined to indicate that a variety and combination of vascular lesions may produce VBI and BSI and should be investigated and appropriately managed.

(Discussion)

10:10 a.m.

6. Status Report - The Cooperative EC/IC Bypass Study

S. J. Peerless London, Ontario, Canada

10:30 a.m. Coffee Break

10:50 a.m.

7. The Use of Steroids in Laminectomies A Clinical and Laboratory Evaluation

David L. Cunningham, J. T. Robertson John Dusseau, Stanley Patterson, Peter Boehm and John Wilson Memphis, Tennessee For the past several decades, laminectomy for the purpose of disc removal has been performed without significant change from the original technique. It is the opinion of the authors that topical application of a long-acting steroid to the nerve root at the time of surgery offers definite short-term advantages over the conventional methods with little or no risk of serious side effects. A clinical review of 400 consecutive laminectomy patients substantiates the reduced length of hospital stay and marked decrease in initial post-op pain. The additional advantage of early ambulation may help prevent post-op complication of pulmonary and urinary origin.

In addition, laboratory studies on rabbits, utilizing Gelfoam, Gelfoam soaked in vehicle only, Gelfoam soaked in DepoMedrol (Methyl prednisolone, 21 Acetate) and no Gelfoam or other agent have been carried out. The laboratory studies revealed a decrease in the nerve root edema and adjacent tissue reaction (both soft and osseous) when using DepoMedrol. Photomicrographs will be shown to demonstrate these findings.

(Discussion)

11:10 a.m.

8. Foreign Medical Graduate Legislation and its Effect on the United States' Role in International Medical Education

Joseph P. Evans Chicago, Illinois

It is important that Program Directors concerned over the role of the United States in international postgraduate education have a clear understanding of the present restrictive legislation affecting foreign medical graduates.

It is important also to realize the impact of the legislation on our international relations.

A brief summary of the current situation and a report of the activity of the Task Force on Foreign Medical Graduate Legislation and International Relations will be presented. Discussion will be welcomed in the effort to achieve consensus.

(Discussion)

11:30 a.m.

9. ACADEMY AWARD

Relation Between Intrafusal and Extrafusal Activity in Triceps Surae Muscles of the Decerebrate Cat; Evidence for Beta Action

Elisabeth M. Post Syracuse, New York

12:00 noon Lunch

1:00 p.m. MODERATOR: John T. Garner

10. Initial Experience with Dimethyl Sulfoxide for Postoperative Vasospasm

Sean Mullan, Jafar Jafar and Frederick Brown

Dimethyl sulfoxide, a water soluble organic solvent (in a class with glycerol, n prophyl alcohol, ethanol, methanol, and others), has in common a strong affinity for water and unique ability to penetrate membranes. It belongs also to the lists of cryoprotective and radiation protective drugs. It reduces or dissipates swelling, as in a swollen ankle. These properties have led us to consider a possible role in the management of a swollen brain. In the laboratory, it has been shown to be effective in reducing mortality and morbidity in experimental head injury (gun shot and balloon), spinal cord injury and in middle cerebral artery occlusion. This paper will summarize these results.

We have additionally embarked upon the earliest of an F.D.A. controlled study in which the primary objectives are (a) safety of the drug, (b) effective dose, (c) possible therapeutic indications. This antedates the standard type of clinical trial. We have now treated two patients with head injury, 11 with subarachnoid hemorrhage and one with spinal paralysis. At the moment, it would appear that the safety of the drug has been established. In exploring its range of effectiveness, its clinical appraisal might best be summarized as one of cautious optimism. These observations will be presented.

(Discussion)

1:20 p.m.

11. Preoperative Evaluation of Cerebral Autoregulation and Systemic Tolerance of Induced Hypotension

Duke S. Samson, Chester Beyer Richard M. Hodosh and Kemp Clark

Dallas, Texas

The use of interoperative induced hypotension has markedly facilitated the development of modern microvascular surgical technique in the treatment of intracranial vascular lesions, such as arterial aneurysms and arteriovenous malformations. Clinical experience indicates that the majority of patients undergoing intracranial procedures tolerate the moderate reduction of mean arterial pressure (MABP = 70 mm Hg) with a low instance of neurological and systemic side effects. Unfortunately, more profound levels of hypotension for protracted time intervals, especially in patients with impaired or altered autoregulatory capacity, may be expected to produce a higher incidence of intolerance as manifested by interoperative and postoperative neurologic and systemic dysfunction.

Learning of Mullan's initial work, we began in 1978 to evaluate, in the immediate preoperative period, the response to induced hypotension in patients planned to undergo surgery for ruptured intracranial aneurysms and arteriovenous malformations. This evaluation is done in the operating suite immediately prior to the induction of anesthesia, the mean arterial pressure being lowered into the 50-60 mm Hg range while the patient's cardiac, neurologic, and systemic responses are closely observed. Hypotension is induced by intravenous infusion of graduated doses of sodium nitroprusside or nitroalycerine. Arterial pressure is continuously monitored via an indwelling radial artery catheter. Ninety-eight (98) patients have been evaluated using this procedure to date, and 16 have been shown to be intolerant to these levels of hypotension; in 11 neurologic symptoms appeared or worsened; in 4 patients cardiac dysfunction became manifest, and one patient developed cyanide toxicity. Fifteen (15) of these 16 patients ultimately underwent a successful surgical procedure following modification of the timing of operation and the degree of interoperative induced hypotension; one patient who on two occasions demonstrated marked cardiac sensitivity to even the slightest degrees of hypotension was treated non-surgically. Of greater interest is that the incidence of symptomatic cerebral vasospasm was found to be significantly higher in those patients found intolerant to preoperative induced hypotension. We believe that the preoperative testing of tolerance to induced hypotension offers important information in the interoperative and postoperative management of this group of patients. It may well serve as a predictor for the identification of those patients prone to develop postoperative symptomatic cerebral ischemia.

(Discussion)

1:40 p.m.

12. Unilateral Occlusion of the Circle of Willis for the Management of Certain Inoperable Cerebral Arteriovenous Malformations

Alfred J. Luessenhop and Patricio Mujica Washington, D.C.

Approximately fifty percent of all cerebral arteriovenous malformations may be resected or obliterated by surgery. Another twenty-five percent can be managed by surgical embolization. This leaves approximately twenty-five percent of all the lesions untreatable at present. The majority of these reside in the territories of the penetrating arteries arising from the circle of Willis and the proximal branches of the major cerebral arteries. When these lesions are large, the distal circulations are adequately supplied by collateral circulation. It is possible to occlude all the major arteries around the circle of Willis without inducing a deficit. The authors will present a series of patients and in whom this was accomplished by a technique of controlled embolization and direct clipping. A follow-up period of over one year shows no incidence of recurrent hemorrhage or further progression of neurological deficits.

(Discussion)

2:00 p.m.

13. Proximal Arterial Alterations in Pressure and Flow Characteristics After Removal of Hemispheral Arteriovenous Malformations

H. D. Garretson Louisville, Kentucky

Improvements in surgical technique now permit dissection and removal of arteriovenous malformations of the cerebral hemispheres without surgical trauma to immediately adjacent areas of critical function. These techniques, however, increase the liklihood of cerebrovascular congestion occurring in the immediately adjacent areas attendant on the occlusion of enlarged, high flow, low pressure arteriovenous shunts. Occlusion of these final feeding arteries at the margin of the AVM convert the physiological situation of a high flow. low pressure intraluminal state to a slow flow, high pressure situation, with the adjacent arteries leaving the feeding arteries suddenly being subjected to a major increase in intraluminal pressure at the time of the distal occlusion. Particularly in the immediate vicinity of the AVM, these normal side collaterals have been functioning with a lower-than-normal intraluminal pressure with frequent clinical evidence of relative ischemia in their perfusion territory. The ability of these normal proximal branches and their parent vessel to compensate for the sudden increase in intraluminal pressure appears to be dependent on two factors. The first and most important appears to be the "age" of the artery, and secondly, the degree of dilitation or enlargement the artery has undergone. It is now well accepted and documented that the enlargement of these vessels is a passive phenomenon, concommitant to the low pressure sump with attendant high flow created by the arteriovenous malformation. Earlier surgical techniques which frequently included a fairly large block ressection of the lobe containing the AVM tended to minimize the problem of proximal vascular congestion through the removal of a greater portion of the proximal feeding artery, together with a fairly large quantity of cerebral tissue providing a significant internal decompression.

We have accumulated experience with 32 arteriovenous malformations of the cerebral hemispheres treated principally by direct excision under magnification along the immediate margin of the AVM with postoperative angiographic assessment of the surgical endeavor in each patient. This has given some insight into the changes in pressure/flow characteristics of th proximal feeding arteries to the excised AVM.

In spite of major enlargement of feeding arteries, prompt return to normal calibre with a short period of clinically significant cerebral swelling is seen in patients up through the second decade of life. Each subsequent decade shows a slower return to normal calibre of the large feeding vessels, with correspondingly longer clinical signs of cerebral vasotongestion and swelling around the area of removal. Several patients have been encountered in the sixth decade of life who have shown no significant reduction in size of the enlarged proximal vessels in question over an extended period of time postoperatively.

It is suggested that hyalinization and fibrotic changes in the walls of the feeding arteries with advancing age interferes with the prompt elastic return to normal calibre noted in young individuals. Allowance for this factor in the pre- and intraoperative management of the older patients may assist in reducing morbidity in this older age group. Early and late postoperative changes in intraluminal pressure in the proximal vessels have been suggested by angiographically documented changes in a proximally situated basilar bifurcation aneurysm in films taken pre-, early, and late postoperatively in a patient with a large posterior cerebral AVM. The changes noted appear consistent with the concept of a transient conversion of a low pressure, high flow state to a high pressure, low flow state in the early postoperative period. The inter-relationship of this concept with postoperative clinical course will be illustrated by appropriate angiographic studies and clinical synopses.

(Discussion)

2:20 p.m.

14. Blood Volume Expansion and Induced Hypotension in the Management of Progressive Neurological Deficit Secondary to Ischemia

S. J. Peerless, C. G. Drake Q. J. Durward and N. F. Kassell London, Ontario, Canada

Progressive neurological deterioration secondary to cerebral ischemia has been aggressively treated in patients with vasospasm following subarachnoid hemorrhage; in patients undergoing intentional ligation of intracranial vessels in the treatment of giant intracranial aneurysms and in patients following spontaneous thromboembolic occlusion of major cerebral arteries or secondary to angiography.

Thirty patients with ischemic complications secondary to these causes have been treated with a vigorous regimen to increase the cerebral perfusion by expanding the circulation blood volume, elevating the blood pressure and lowering the intracranial pressure. As well, steps to decrease blood viscosity and improve rheology and to a lesser extent, increase cerebral arterial calibre has been attempted.

These complex and aggressive management techniques have

resulted in remarkable, immediate and sustained clinical improvement in 23 patients. Five patients showed only a transient or minimal improvement and two deteriorated.

The technique of monitoring blood pressure, intracranial pressure, pulmonary-capillary wedge pressure and blood volume, as well as the treatment protocol and the pulmonary, cardiac and cerebral complications, will be described.

(Discussion)

2:40 p.m.

15. Second Intranational Workshop on Cerebral Vasospasm

Robert H. Wilkins Durham, North Carolina

3:00 p.m. Coffee Break

3:20 p.m.

16. High Dose BCNU with Autologous Bone Marrow Rescue

Leroy Parker, Frederick Hochberg Nicholas T. Zervas and George Cannelos Boston, Massachusetts

The lipid soluble nitrosoureas represent the single, most widely accepted chemotherapeutic agents for the treatment of glioblastoma multiforme. In animal and human systems, there is a linear relationship between dose and response. In clinical trials, the major dose limiting toxicities are referable to delayed myelosuppression. Given every six weeks, BCNU treatment can be provided for approximately five courses to a median dose of 1200 mg/m2. We have investigated the use of autologous bone marrow reinfusion as a means of providing single doses of BCNU in excess of this cumulative dose level. Patients with recurrent glioblastoma (after surgery and irradiation) are harvested of iliac crest marrow under general anesthesia. Marrow is stored at 4°C until used. Split doses of BCNU are given four and eighteen hours after harvest. Two patients each have received BCNU

600, 800, 1000 mg/m2. The previously harvested autologous bone marrow (2.8 x 1010 nucleates cells) is reinfused 48 hours after harvest (24 hours after BCNU). A colony forming (CFU-c assay) has been used to evaluate the repopulation of bone marrow. High dose BCNU toxicity has been limited to nausea and vomiting. To date, there have been no associated pulmonary, liver or renal toxicity. Characteristically, patients show a white blood cell and platelet nadir 10-15 days after BCNU therapy. Following this, white blood cells. platelets and CFU-c rise until repopulation of both systemic and marrow sites is complete. This repletion of marrow occurs with a greater rapidity in higher dose patients. The clinical response to BCNU has been acceptable. At constant steroid doses all patients have shown clinical improvement. Four of six patients have shown amelioration of CT scan. High dose BCNU with autologous marrow rescue would appear to be an acceptable easily performed approach to the treatment of aliablastoma.

(Discussion)

3:40 p.m.

17. The Role of the Ribosome in Malignant Glial Tumors

Wolff M. Kirsch, Kazuo Tabuchi John Van Buskirk and Margaret Low Denver, Colorado

In terms of potential therapeutic control points for malignant aliomas, the ribosome appears to be a critical subcellular target. Steadily accumulating evidence indicates that one of the earliest detectable biochemical changes in the transition from a resting to a proliferating cell is an increase in the rate of ribosome synthesis. There is a clear relationship between the growth rate of a cell and its RNA content. Our laboratory has been interested in ribosomal RNA maturation, with specific attention to certain drugs that specifically inhibit maturation of ribosomal RNA. Our interest has been prompted by a study of the drug, racemic sodium warfarin, and its effect upon the cell cycle of malignant glial tumors. Both in vitro and in vivo studies indicate that this drug, which has a structural resemblance to camplothecin, does alter ribosomal maturation. Current research is directed toward elucidating the effects of warfarin upon specific ribosomal proteins. The effect is inhibition of the vitamin K mediated gammacarboxylation of alutamic acid residues in selected ribosomal proteins. Our laboratory has demonstrated the ubiquity of an unusual amino acid, gammacarboxyglutamic acid, in the ribosomes of a variety of tissues. The posttranslation modification of ribosomal proteins may provide important clues for further understanding of the biology and function of the ribosome, particularly for the necessary role of magnesium ion in protein biosynthesis. The central role of the ribosome in such critical events, such as viral transformation, will also be discussed.

(Discussion)

FRIDAY, November 9

MODERATOR: S. J. Peerless

8:30 a.m.

18. Controversies in the Management of Prolactin-Secreting Pituitary Adenomas

Charles B. Wilson and Robert Jaffe San Francisco, California

Within the past five years, a new entity, the prolactin-secreting micro-adenoma, has evolved with a frequency that was unpredictable before sella polytomography and plasma prolactin determination were applied to the large population of young women with amenorrhea and galactorrhea. Based upon experience with 200 prolactinomas treated by transsphenoidal removal and a smaller number either treated by other means or not treated at all, my medical colleagues and I have reached certain conclusions concerning the management of micro- and macro-prolactinomas. Transsphenoidal surgery is recommended for patients with microadenomas who (1) desire pregnancy, or (2) have primary amenorrhea, or (3) male. Our views on the roles of bromocriptine, irradiation and no treatment are changing, and a current summary will be presented when the paper is delivered.

(Discussion)

8:50 a.m.

Transsphenoidal Microsurgery for Prolactinomas Causing the A/G Syndrome

George T. Tindall Atlanta, Georgia This report is based upon a total of 58 women with the amenorrhea-galactorrhea syndrome due to a prolacin secreting pituitary tumor (prolactinoma) who underwent transsphenoidal microsurgical removal of the tumor. The study extends over a period of six years (July 1973-June 1979).

Endocrinologic, neuroradiologic and clinical criteria for selection of patients with prolactinomas for transsphenoidal microsurgery will be reviewed and the results of treatment presented. Criteria for successful treatment will be reviewed.

As expected, successful therapy was more frequent in women with the enclosed microadenoma (Hardy-Vezina, grade I) and whose preoperative fasting prolactin value was < 200 ng/ml. Seventy percent (70%) of 40 women with preoperative prolactin < 200 either resumed normal menses or had return of prolactin to normal values (< 25 ng) postoperatively. Unfortunately, only 40% of patients with preoperative prolactin > 200 ng/ml had successful results in terms of restoring normal menses or reducing prolactin values to normal.

The possible causes for persistent hyperprolactinemia postoperatively will be examined and followup study (up to six years) will be presented in 15 women with this laboratory finding. A common cause of postoperative hyperprolactinemia is persistent tumor but another mechanism to consider is stalk damage with interference with PIF transport.

(Discussion)

9:10 a.m.

20. CT Localization and Immunoperoxidase Staining of Small Pituitary Adenomas

Wolff M. Kirsch, John Stears Bruce Jafek and Doris Gaskin Denver Colorado

As our experience with transsphenoidal surgery for pituitary neoplasia has enlarged, we have found two diagnostic tests to be helpful in the diagnosis of small pituitary adenomas. These tests are refined CT scanning of the sella turcica and the application of immunoperoxidase staining for identification of hormones on frozen section. Angiography has never demonstrated a discrete or convincing "stain" and in the past we have relied on subtle pneumotomographic findings for indirect tumor localization ("stalk shift" eccentric diaphragm elevation). We now have 12 patients with CT demonstration of microadenomas, of which 3 have been confirmed in detail by transsphenoidal surgery.

(Discussion)

9:30 a.m.

21. The Pseudo-Diabetes Insipidus of Transsphenoidal Pituitary Gland Surgery

Clarence B. Watridge and James T. Robertson Memphis, Tennessee

The transsphenoidal approach to pituitary tumors is a widely used technique with low morbidity and mortality. One sequela that occurs in approximately one-third of these patients is a postoperative diuresis. This has been referred to as a diabetes insipidus by some authors. We have analyzed the antidiuretic hormone levels of twelve consecutive patients undergoing transsphenoidal removal of pituitary tumors and have found that there is no deficit of antidiuretic hormone in these patients. The postoperative diuresis following transsphenoidal pituitary surgery is not a true diabetes insipidus. The syndrome of Pseudo-Diabetes Insipidus following transsphenoidal surgery for pituitary tumors is described.

(Discussion)

9:50 a.m. Coffee Break

10:00 a.m.

22. Updated Experience with Surgery for Pineal Tumors

Bennett M. Stein Boston, Massachusetts

An up-to-date experience with twenty-four operations on pineal tumors using the posterior fossa route will be presented. Nine of these patients presented with benign tumors which were encapsulated and could be removed through this exposure. A wide variety of tumor types existed in all cases, and it was my opinion that histological diagnosis was useful in all. Current therapy dictates specific forms of therapy tailored for the type of tumor that exists in this region, e.g., the germinoma type of tumor known as an atypical teratoma is probably best treated by radiation to the entire nervous system as well as chemotherapeutic agents that have been used in the treatment of germinal tumors existing elsewhere in the body. My experience with this type of tumor in which conventional radiation

has been used solely to the tumor site has been discouraging.

The techniques of the operative procedure as well as operative morbidity and mortality will be discussed in detail.

(Discussion)

10:30 a.m.

23. A Parapetrosal Approach to Clival Tumors

Leonard I. Malis New York, New York

Tumors of the petrous apex, clivus, and anterior tentorial margin have been a particularly difficult and dangerous group for surgical removal, even with microsurgical technique. Adequate exposure has been difficult to achieve, and ligation of the vein of Labbe has often been needed to permit sufficient elevation of the temporal lobe posteriorly. The lack of anastomosis of the "great anastomotic vein" has increased temporal lobe damage.

A combined posterior fossa and subtemporal approach with division of the lateral sinus and tentorial petrous attachment has been devised so that the vein of Labbe and the temporal venous drainage is spared. This procedure has now been used since 1972. Twenty tumors (thirteen clival meningiomas, three fifth nerve neuromas, and one each of cholesteatoma, craniopharyngioma, chordoma, and collicular astrocytoma) have been resected with total removal in eighteen. There was one death and one severe, but fortunately temporary, deficit to occur. This approach has greatly improved the results as compared to this author's prior experience with lesions of this region.

(Discussion)

10:50 a.m.

24. Surgical Management of Massive Glomus Jugulare

Theodore Kurze, James House and Fong Tsai Pasadena, California

Extension of non-chromaffin paragangliomas to involve intracranial structures is a well-recognized clinical problem, but seldom experienced in neurosurgical practice. The value of radiation therapy remains controversial and highly questionable. Surgical mortality and morbidity in these massive lesions is formidable.

The authors present the surgical anatomical details of a highly satisfactory technique of embloc total removal with modest blood loss and no permanent morbidity.

The peri-surgical care including embolization will be included. (Discussion)

11:10 a.m.

25. Microsurgical Removal of Primary Intra-Orbital Optic Nerve Meningiomas

Joseph C. Maroon and John Kennerdell Pittsburgh, Pennsylvania

Recent advances in computerized tomography now permit the diagnosis and clear delineation of primary tumors of the optic nerve. Heretofore, the treatment of primary intra-orbital optic nerve meningiomas with standard operating techniques has almost uniformly resulted in increased visual impairment or blindness.

Over the last three years, we have refined a lateral microsurgical approach to intra-orbital tumors, and we have removed three consecutive optic nerve meningiomas with uniformly improved postoperative visual function. Besides an understanding of the pathology of such lesions, special instrumentation, including a new self-retaining intra-orbital retractor, are considered essential for the removal of such tumors.

Based on our preliminary experiences, we believe that primary optic nerve meningiomas no longer should be considered harbingers of inevitable progressive blindness. Exploration should be considered in the patient with progressive unilateral visual loss, proptosis, afferent pupillary light defect, and optic-ocililary shunting if there is radiographic and ultrasonic evidence of a primary optic nerve tumor. A surgical cure may now be possible.

(Discussion)

11:30 a.m.

PRESIDENTIAL ADDRESS "PAIN AND TRYPTOPHAN"

Robert B. King

SATURDAY, November 10

MODERATOR: Phanor Perot

26. Electrically Evoked Responses Recorded from the Dorsal Surface of the Human Spinal Cord During Spinal Cord Surgery

Blaine S. Nashold, Jr., Robert H. Wilkins Bob Pearlstein and John B. Mullen Durham, North Carolina

The cortical and spinal evoked somatosensory responses have been extensively used to evaluate certain cerebrospinal disorders. In the monkey, the CEP (cortical evoked potential) response may be mediated via the dorsal columns, a sensory pathway increasing in size and importance in the higher primates.

This is a preliminary clinical report of a technique for intraoperative recording of electrically evoked responses produced by direct electrical stimulation of the spinal cord and peripheral nerves. The direct spinal evoked responses (DSER) were recorded above and below spinal cord pathology during surgical intervention. The concept under study is the use of direct intraoperative direct spinal evoked recordings (DSER) to monitor spinal cord function. Direct stimulation and recording on the dorsal surface of the human spinal cord may give a more sensitive indicator of spinal transmission, since both conduction velocity via the dorsal columns, and the production of a complex wave form are easily recorded and analyzed in the operating room.

Three patients have been studied using this DSER technique. One man was suffering from progressive weakness of his legs plus intractable burning pain which had developed years after an open bilateral thoracic cordotomy. A second man had DSER recordings made before and after a thoracic commissurotomy for relief of pain due to generalized pelvic cancer. A third man who was experiencing weakness of the legs and bladder and sexual dysfunction caused by a spinal angioma at the level of the conus had direct spinal recordings carried out before and after the successful removal of the angioma. The results of direct spinal recording of evoked responses in the human will be discussed in the light of our current knowledge of spinal pathophysiology.

9:20 a.m.

27. Effect of Trauma Dose on Spinal Cord Edema

Franklin C. Wagner, Jr., William B. Stewart and William F. Collins, Jr. New Haven, Connecticut

Post-traumatic edema has been suggested as a complicating factor in spinal cord injury. Before the possible pathological effects of edema can be assessed, it would be helpful to know more regarding the factors responsible for its formation and distribution within the spinal cord. In the current investigation, forty cats were subjected to impact trauma of different magnitudes. The presence of edema was determined by either of two methods. The distribution of various flourescent tracers which were injected either prior to trauma or at different intervals after trauma was studied microscopically. In other animals, the difference between the wet and dry weight of spinal cord samples was measured. Attention was directed to ascertaining the portions of spinal cord maximally involved by edema and the duration of time after trauma that it may form. Longitudinal extension of edema as determined by both measures occurred in animals subjected to severe impact injury. Little spread rostral and caudal to the impact site was noted in animals subjected to moderate or mild trauma doses. When extension was observed in fluorescent studies, it was noted primarily in the mid-portion of the lateral, anterior, and posterior white matter where fibers are predominantly longitudinally directed. Fluorescent tracers injected at intervals greater than three hours after trauma were not readily apparent in tissue samples. The findings of the present study indicate that the spread of posttraumatic edema is directly related to the amount of the initial trauma, that the structural features of the spinal cord influence its spread, and that the extravasation of large molecules does not continue indefinitely.

(Discussion)

9:40 a.m.

28. Noradrenalin, Serotonin, and the Dorsal Horn

Charles J. Hodge, Jr., Charles I. Woods and Jonathan Delatizky Syracuse, New York

There are abundant anatomic, physiologic, and drug interaction studies indicating that noradrenalin (NA) and serotonin (5-HT) modify sensory processing at the segmental level. While the role of 5-HT as an inhibitory transmitter, used by the raphe-spinal system and linked to morphine - and stimulation produced - analgesia is quite firm, the function of the descending NA bulbo-spinal system is unclear. These systems were investigated by studying the effects of L-DOPA triagered release of 5-HT and NA on the responses of dorsal horn neurons to both innocuous mechanical and noxious thermal cutaneous stimulation. Using pretreatment with a variety of drugs, the availability of NA or 5-HT in axonal terminals was altered, thus allowing separation of the effects of these two systems. Dorsal horn cells were divided into two groups: Group 1 responded only to innocuous stimuli and Group 2 responded to either only noxious or innocuous and noxious stimuli. We found that 1) 5-HT release inhibits the response of Group 2 cells to both noxious and innocuous stimuli and that 5-HT has no apparent effect on Group 1 cells, 2) NA release increases the responsiveness of Group 1 and Group 2 cells to innocuous stimuli while mildly inhibiting the response of Group 2 cells to noxious stimuli.

The conclusion was reached that these two systems have different, and at times opposing, roles, depending on type of afferent input and dorsal horn cell being effected. While these two aminergic systems have been assigned the function of a "pain control mechanism", this seems too limited a concept of their action in view of the ubiquity of their connections and other physiologic processes that they can modify. Nonetheless, further understanding of their spinal actions and suprasegmental interactions may lead to non-operative and non-narcotic methods of controlling altered sensory states such as denervation syndromes and chronic pain.

(Discussion)

10:00 a.m.

29. Observations on the organization of human language cortex

George A. Ojemann and Catherine A. Mateer Seattle, Washington

With the electrical stimulation mapping technique the localization of changes in object naming, reading, short-term verbal memory, the ability to mimic single and sequential oral facial movements and to identify phonemes was identified at each of 9-15 sites in periSylvian cortex of the dominant hemisphere in 5 patients,

and in nondominant hemisphere of 2 patients undergoing craniotomies under local anesthesia for resection of epileptic foci. The only one of these functions altered from any nondominant hemisphere periSylvian site was the ability to mimic single oral-facial movements from sites at the foot of rolandic cortex. On the other hand, the patterns of changes in these different functions in dominant hemisphere periSylvian cortex (with dominance having been established by preoperative intracarotid amytal testing) identify the sites of several different language subdivisions. At the foot of rolandic cortex and extending into inferior premotor cortex is an area where the ability to mimic single oral movements is disrupted along with an arrest of all types of speech output, regardless of how evoked. This seems to represent a final motor pathway for speech. Surrounding this in periSylvian cortex of both frontal, parietal and superior-temporal lobe is an area where the ability to mimic sequential, but not single oral-facial movements, was disrupted, and at these same sites and only these same sites the ability to identify phonemes was also altered. This seems to identify a motor discrimination system for speech, the system which provides the anatomic basis for the common features that have been identified in both expressive and receptive aphasias, and also the anatomic substrate behind the motor theory of speech perception. Surrounding the motor discrimination system frontally, parietally and temporally are sites where only short-term verbal memory is disrupted, with parietal and temporal sites showing alterations in memory when the current is applied during the input or storage phases of the task while frontal and occasional parietal sites when the current is applied during output phases of the task. Between the motor discrimination and short-term verbal memory systems are individual sites where the only disruption is in grammatical aspects of reading, identifying sites that seem to be related specifically to syntax. Although the exact location of the sites of each of these subsystems varied from patient to patient. the general relationships are the same in the dominant hemispheres of each of the 5 patients tested. Thus this study suggests that the maior subdivisions of language cortex are not an anterior expressive, posterior receptive, but rather a periSylvian system common to sequential motor movements and phonemic discrimination surrounding a final motor pathway for speech and being surrounded by a cortical short-term verbal memory system.

(Discussion)

10:20 a.m. Coffee Break 10:50 a.m.

30. Simultaneous Subacute Measurement of ICP and Brain 02 Availability

R. B. Morawetz, H. G. Mitchem E. R. Strong, J. G. Galbraith and J. H. Halsey Birmingham, Alabama

Simultaneous intracranial pressure (ICP) monitoring and brain oxygen availability (02a) measurements were carried out on a group of patients whose intracranial pressure was being monitored. A ventricular catheter was modified by addition of an array of bare platinum electrodes along its shaft so that while the tip of the catheter lay in the ventricle, the electrodes were in contact with brain tissue. A group of hydrocephalic children had catheters inserted when medically indicated for monitoring ICP and/or for ventricular drainage.

Relationship of ICP to brain 02a, and relationship of 02a to cerebral blood flow will be discussed. Response to changes in ICP, inspired 02 and inspired CO2 is documented. The increased intracranial compliance seen with ventricular drainage is significant. Inhalation of 100% 02 results in a significant rise in 02a at ICP in both normal and elevated levels. There is a marked drop in 02a with plateau waves, and no rise in 02a is seen before appearance of plateau waves despite the postulated increase in CBF as the initial event in production of A waves. The observations suggest that high concentration of 02 in inspired air may be of help in management of high ICP. (Discussion)

11:10 a.m.

31. Hydrocephalus Diagnosis: CSF Pulse Pressure Index and Wave Form Analysis

Eldon L. Foltz and Cheryl Aines Orange, California

In 1976, initial data on the "CSF pulse pressure index" characteristic of hydrocephalus (child and adult) was presented (AANS). Subsequent refinement of the concept of intracranial factors acting on the choroid plexus-generated CSF pulse pressure to produce a "damped" wave under normal conditions has been based on

the force, or power, formula applied to CSF as a moving fluid mass (m) traveling at a certain velocity (v) reduced to no movement during a certain time epoch ($\Delta \uparrow$): $P = mv2/2 \Delta \uparrow$.

It has been postulated that the energy involved in this intracranial CSF phenomenon will be greatly enhanced when the normal pulsatile outflow of intracranial venous blood is lacking, converting a lower power system (volume vented with each CSF pulse wave) to a higher power system (no pulsatile volume venting). In the formula, this is accomplished by the very small Δ t value. This equates to loss of intracranial compliance. This should be associated with progressing hydrocephalus if the power generated is critical in producing the ventriculomegaly of hydrocephalus.

The relationship of CSF pulsatility to mean CSF pressure has not been thoroughly investigated under these assumptions.

Therefore, as part of a broad and continuing investigation of hydrocephalus in CSF dynamics, our ongoing clinical study is reported. Measurements of CSF pressures in 110 patients with suspected hydrocephalus have included:

- Mean CSF and CSF pulse pressures under homestatic (baseline) and jugular compression;
- CSF ascending wave slopes and wave shapes at homeostatic (baseline) and during inspirationexpiration cycling.

The results give statistically significant correlation of CSF pulse amplitude with hydrocephalus as proved by clinical studies. The CSF wave form, as measured by ascending slope rate, correlates even better with no hydrocephalus, aqueduct stenosis hydrocephalus, communicating hydrocephalus, and to a lesser degree with arrested hydrocephalus.

Effective analysis of this simple recording can diagnose difficult hydrocephalus problems with reliability, including adult occult hydrocephalus.

(Discussion)

11:30 a.m.

32. Arnold-Chiari Malformation: Observations on Teratogenesis

Robert L. McLaurin, Josef Warkany and Harold Kalter Cincinnati, Ohio The mechanism of development of the Arnold-Chiari malformation has been the subject of considerable investigation and speculation in the past. Theories have included: 1) traction from caudal tethering, 2) pressure from hydrocephalus, and 3) a primary dysgenesis of the brain stem.

The availability of a strain of mice in which spina bifida is carried as an autosomal recessive characteristic has permitted observations on the teratogenesis of the malformation. Mice with spina bifida have been serially sectioned and studied from the 11th gestational day to the 18th (full-term) day. Sagittal and frontal sections of 20 micra thickness have been studied.

Based on these observations, it is concluded that one of the primary events is stenosis or occlusion of the aqueduct. This results in hypoplasia of the posterior fossa, presumably as a result of pressure differential between the supra- and infratentorial compartments. The consequence is then crowding within the posterior fossa and subsequent herniation as the neural tissues undergo normal enlargement with maturation. There was evidence negating the theories of traction from below or pressure from above.

In addition to herniation of the medulla and cerebellum, other characteristics of the Arnold-Chiari deformity were studied. It appears that tectal beaking is the result of arrest of development or a primary dysgenesis rather than due to mechanical factors. The medullo-cervical fold was not observed, as it probably occurs in the post-natal period in mice. There was no evidence to support the theory of aqueduct occlusion resulting from hydrocephalus.

(Discussion)

ACADEMY AWARD

1979

ELISABETH M. POST, M.D. State University of New York Upstate Medical Center Syracuse, New York

"Relation Between Intrafusal and Extrafusal Activity in Triceps Surae Muscles of the Decerebrate Cat; Evidence for Beta Action"

ACADEMY AWARD WINNERS

Paul M. Lin
Hubert L. Rosomoff
Byron C. Pevehouse
Norman Hill
Jack Stern
Robert Ojemann
Lowell E. Ford
Charles H. Tator
Earle E. Crandall
Stephen Mahaley, Jr
Chung Ching Kao
John P. Kapp
Yoshio Hosobuchi
Gary C. Ferguson
Richard L. Pressley
David G. McLone
Arden F. Reynolds, Jr
Richard L. Rapport
Andrew G. Shetter
John F. Howe
Howard W. Blume
Howard J. Senter

1979 GUESTS

GUEST	SPONSOR
Jesse B. Barber, Jr. Washington, D.C.	William H. Sweet
William S. Buchheit Philadelphia, Pennsylvania	James T. Robertson
Peter Carmel New York, New York	Richard A. R. Fraser
David H. Chepovsky Pueblo, Colorado	Wolff M. Kirsch
David Cunningham Memphis, Tennessee	The Academy
William Dawson Reno, Nevada	Ernest W. Mack
Rembrandt H. Dunsmore Hartford, Connecticut	Benjamin B. Whitcomb
Fred Gentili Toronto, Canada	William M. Lougheed
Joseph H. Goodman Columbus, Ohio	William E. Hunt
Jens Haase Odense, Denmark	Phanor L. Perot, Jr.
Griffith R. Harsh, III Birmingham, Alabama	James G. Galbraith
Charles J. Hodge Syracuse, New York	Robert B. King
Joseph C. Maroon Pittsburgh, Pennsylvania	Robert H. Wilkins

Lawrence Pitts San Francisco, California	Charles B. Wilson
Duke Samson Dallas, Texas	W. Kemp Clark
Richard Saunders Hanover, New Hampshire	Robert G. Fisher
A. B. Sisco Pensacola, Florida	James C. Simmons
Robert F. Spetzler Cleveland, Ohio	Frank E. Nulsen
Charles A. Sterbergh Chattanooga, Tennessee	Augustus McCravey
Mrs. George Tindall (Suzie) Atlanta, Georgia	George Tindall
Paul T. Turner Albuquerque, New Mexico	A. Earl Walker
Frank Wagner New Haven, Connecticut	William F. Collins

Past Presidents

Past Vice-Presidents

Dean H. Echols 1938-39	Francis Murphey 1941
Spencer Braden 1940	William S. Keith 1942
Joseph P. Evans 1941	John Raaf
Francis Murphey 1942	Rupert B. Raney 1944
Frank H. Mayfield 1943	Arthur R. Elvidge 1946
A. Earl Walker 1944	John Raaf 1947
Barnes Woodhall 1946	Arthur R. Elvidge 1948
William S. Keith 1947	F. Keith Bradford 1949
Howard A. Brown 1948	David L. Reeves 1950
John Raaf 1949	Henry G. Schwartz 1951
E. Harry Botterell 1950	J. Lawrence Pool 1952
Wallace B. Hamby 1951	Rupert B. Raney 1953
Henry G. Schwartz 1952	David L. Reeves 1954
J. Lawrence Pool 1953	Stuart N. Rowe 1955
Rupert B. Raney 1954	Jess D. Herrmann 1956
David L. Reeves 1955	George S. Baker 1957
Stuart N. Rowe 1956	Samuel R. Snodgrass 1958
Arthur R. Elvidge 1957	C. Hunter Shelden 1959
Jess D. Herrmann 1958	Edmund Morrissey 1960
Edwin B. Boldrey 1959	Donald F. Coburn 1961-62
George S. Baker 1960	Eben Alexander, Jr 1963
C. Hunter Shelden 1961-62	George L. Maltby 1964
Samuel R. Snodgrass 1963	Robert Pudenz 1965
Theodore B. Rasmussen . 1964	Francis A. Echlin 1966
Edmund J. Morissey 1965	Benjamin Whitcomb 1967
George Maltby 1966	Homer S. Swanson 1968
Guy L. Odom 1967	Augustus McCravey 1969-70
James G. Galbraith 1968	Edward W. Davis 1971
Robert H. Pudenz 1969-70	John R. Green 1972
William B. Scoville 1971	George J. Hayes 1973
Robert L. McLaurin 1972	Richard L. DeSaussure 1974
Lyle A. French 1973	Ernest W. Mack 1975
Benjamin B. Whitcomb 1974	Frank E. Nulsen 1976
John R. Green 1975	Robert S. Knighton 1977
William H. Feindel 1976	Robert G. Fisher 1978
William H. Sweet 1977	
Arthur A. Ward 1978	

Past Secretary-Treasurers

Francis Murphey 1938-40 A. Earl Walker 1941-43 Theodore C. Erickson 1944-47 Wallace B. Hamby 1948-50 Theodore B. Rasmussen 1951-53	Eben Alexander, Jr. 1954-57 Robert L. McLaurin 1958-62 Edward W. Davis 1963-65 Robert G. Fisher 1966-68 Byron C. Pevehouse 1969-72
Secretary	Treasurer
Russel H. Patterson, Jr. 1974-76 Phanor L. Perot, Jr. 1977-78	Russel H. Patterson, Jr. 1972-74 Phanor L. Perot, Jr. 1974-76
	John T. Garner 1977-78

Past Meetings of the Academy

	-
Hotel Netherlands Plaza, Cincinnati, Ohio	October 28-29, 1938
Hotel Netherlands Plaza, Cincinnati, Ohio Roosevelt Hotel, New Orleans, Louisiana	October 27-29 1939
m 1. A TI 4 1 Oliveriand Objection	O-4-101-00-1040
Tudor Arms Hotel, Cleveland, Ohio	October 21-22, 1940
Mark Hopkins Hotel, San Francisco and Amba	assador Hotel,
Los Angeles, California	
The Palmer House, Chicago, Illinois	Oatobor 16 17 1049
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 20-28, 1948
Benson Hotel, Portland, Oregon	
Mayo Clinic, Rochester, Minnesota	
Shamrock Hotel, Houston, Texas	
Waldorf-Astoria Hotel, New York City Septe	ember 29-October 1, 1952
Biltmore Hotel, Santa Barbara, California	
Diffinore Hotel, Salita Darbara, Camorina	O-1-1 01 00 1054
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	
The Royal York Hotel, Toronto, Canada	
Del Monte Lodge, Pebble Beach, California	October 18-21, 1959
Capley Sheraton Plaza, Boston, Massachusetts	
Royal Orleans, New Orleans, Louisiana	
El Mirador, Palm Springs, California	
The Key Biscayne, Miami, Florida	November 11-14, 1964
Terrace Hilton Hotel, Cincinnati, Ohio	October 14-16, 1965
Fairmont Hotel & Tower,	
Talliffold Hotel & Tower,	0 . 1 . 17 10 1000
San Francisco, California	
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
One in Deal Hatal Maning Older	N 1000
Camino Real Hotel, Mexico City	
Sahara-Tahoe Hotel, Stateline, Nevada	
New College, Oxford, England	September 4-7, 1972
Huntington-Sheraton Hotel,	
Pasadena, California	Naucushau 14 17 1070
Southampton Princess Hotel,	
Southampton Princess Hotel, Southampton, Bermuda	November 6-9, 1974
The Wigwam (Litchfield Park), Pheonix, Arizona	November 5-8 1075
The Mills Usest House Charleston	2140 vehiber 0.0, 1970
The Mills Hyatt House, Charleston, South Carolina	
South Carolina	November 10-13, 1976
Mauna Kea Beach Hotel, Kamuela, Hawaii	November 2-5, 1977
Hotel Bayerischer Hof, Munich Germany	
(Joint Meeting with Deutsche Gesellschaft for	ır
Neurochirurgie)	

1979 MEMBERSHIP LIST

AMERICAN ACADEMY OF NEUROLOGICAL SURGERY Founded October, 1938

Honorary Members	Elected
HUGO KRAYENBUHL Neurochirurgische University Kantonsspital, Ramistrasse 100 8000 Zurich, Switzerland	1974
GUY LAZORTHES 26 Rue D Auriol 31 Toulouse, France	1973
VALENTINE LOGUE Naida Vale Hospital London, W. 9, England	1974
GOSTA NORLEN Neurokirurgiska Kliniken Sahlgrenska Sjukhus Goteborg, SV Sweden	1973
KEIJI SANO Department of Neurosurgery School of Medicine University of Tokyo Tokyo, Japan	1975
R. EUSTACE SEMMES 920 Madison Avenue Memphis, Tennessee 38103	1955

Senior Members		Elected
GEORGE S. BAKER 200 First Street, S.W. Rochester, Minnesota 55901	(ENID)	1940
E. HARRY BOTTERELL Faculty of Medicine Queens University Kingston, Ontario, Canada	(MARGARET)	1938
HOWARD A. BROWN 2001 Union Street San Francisco, California 94123	(DOROTHY)	1939
HARVEY CHENAULT 2370 Nicholasville Road Lexington, Kentucky 40503	(MARGARET)	1938
DONALD F. COBURN Suite 112 2401 Pennsylvania Avenue Wilmington, Delaware 19806	(ELLIE)	1938
EDWARD W. DAVIS Providence Medical Office Bldg. 545 N. E. 47th Avenue Portland, Oregon 97213		1949
FRANCIS A. ECHLIN 100 East 77th Street New York, New York 10021	(LETITIA)	1944
DEAN H. ECHOLS 1550 Second Street New Orleans, Louisiana 70130	(FRAN)	Founder
ARTHUR ELVIDGE 275 Brittany Avenue Montreal, Quebec, Canada HQR	2B3	1939
THEODORE C. ERICKSON 425 North Livingston Street Madison, Wisconsin 53703	(MARTHA)	1940

JOSEPH P. EVANS American College of Surge 55 East Erie Street Chicago, Illinois 60611	(HERMENE) eons	Founder
JOHN D. FRENCH The Center for the Health University of California Los Angeles, California 90		1951
JAMES G. GALBRAITH 2515 Crest Road Birmingham, Alabama 355	(PEGGY) 223	1947
EVERETT G. GRANTHA 234 East Gray Street Louisville, Kentucky 4020	, ,	1942
JAMES GREENWOOD, J 1117 Hermann Professions 6410 Fannin Street Houston, Texas 77025		1952
WALLACE B. HAMBY 3001 N. E. 47th Court Ft. Lauderdale, Florida 33	(ELEANOR) 308	1938
HANNIBAL HAMLIN 270 Benefit Street Providence, Rhode Island	(MARGARET) 02903	1941
JESS D. HERRMANN Post Office Box 135 Mountain Pine, Arkansas	(MARY JO) 71956	1948
WILLIAM S. KEITH 55 St. Leonards Crescent Toronto, Ontario, Canada	(ELEANOR) M4N 3 A 7	Founder
JOHN J. LOWERY P.O. Box 4302 Kawaihae, Hawaii 96743	(CATHERINE "Katy")	1965

GEORGE L. MALTBY 470 Black Point Road Scarsborough, Maine 04074	(ISABELLA "Sim")	1942
AUGUSTUS McCRAVEY 1010 East Third Street Chattanooga, Tennessee	(HELEN)	1944
EDMUND J. MORRISSEY 450 Sutter Street, Suite 1504 San Francisco, California 9410	(KATE)	1941
FRANCIS MURPHEY 3951 Gulf Shores Road Apt. 1102 Naples, Florida 33940	(MARGE)	Founder
LAWRENCE J. POOL Box 31 West Cornwall, Connecticut 0	(ANGELINE) 6796	1940
ROBERT H. PUDENZ Box 79, Rt. 1 Vineyard Drive Paso Robles, California 93446	(RITA)	1943
R.C.L. ROBERTSON 2210 Maroneal Boulevard Shamrock Professional Bldg. Suite 404 Houston, Texas 77025	(MARJORIE)	1946
STUART N. ROWE 302 Iroquois Building 3600 Forbes Street Pittsburgh, Pennsylvania 152	(ELVA)	1938
WILLIAM B. SCOVILLE 85 Jefferson Street Hartford, Connecticut 06106	(HELEN)	1944
HENRY G. SCHWARTZ Barnes Hospital Division of Neurological Surg St. Louis, Missouri 63110	(REEDIE) ery	1942

C. HUNTER SHELDEN 734 Fairmount Avenue Pasadena, California 91105	(ELIZABETH)	1941
HOMER S. SWANSON 1951 Mount Paran Rd., N.W. Atlanta, Georgia 30327	(LaMYRA)	1949
JOHN TYTUS (V Mason Clinic Seattle, Washington 98107	IRGINIA "Gina")	1967
ALFRED UIHLEIN 200 First Street, S.W. Rochester, Minnesota 55901	(IONE)	1950
A. EARL WALKER John Hopkins Hospital Division of Neurological Surg 601 North Broadway Baltimore, Maryland 21205	(TERRYE) gery	1938
EXUM WALKER 49 Peachtree Street, N.E. Atlanta, Georgia 30308	(NELLE)	1938
THOMAS A. WEAVER, JR. 146 Wyoming Street Dayton, Ohio 45409	(MARY)	1943
BARNES WOODHALL Duke University Medical Cer Durham, North Carolina 2770		1941
Corresponding Members		Elected
JEAN BRIHAYE Rue Heger Bordet 1 1000 Brussels, Belguim		1975
KARL AUGUST BUSHE Neurochirurgischen Klinik D-8700 Wurzburg Josef-Schneider-Strasse 11 West Germany		1972

FERNANDO CABIESES Instituto Peruano de Formento Educativo Av. Arenales 371, Of. 501 Apartado 5254 Lima, Peru	1966
JUAN CARDENAS, C. Neurologo 4 Neurocirujano Av. Insurgentes Sur 594, Desp. 402 Mexico 12, D. F.	1966
JUAN C. CHRISTENSEN Ave. Quintana 474 80 A Buenos Aires, Argentina	1970
GIUSEPPE DALLE ORE Dipartimento di Neurochirurgia Ospedale Maggiore 37100 Verona, Italy	1970
HANS ERICH DIEMATH Prim. Univ. Doz Neurochir. Abt. D. Landersnervenklink Salzburg, 5020, Austria	1970
JOHN GILLINGHAM Boraston House 22, Ravelson Dykes Road Edinburgh, Scotland EH43PB	1962
JAIME G. GOMEZ Director, Neurological Institute of Columbia Cra. 13 No. 43-23 Bogota 8, Columbia, South America	1975
JOHN HANKINSON Department of Neurosurgery Newcastle General Hospital Newcastle-upon-Tyne 4, England	1973
SHOZO ISHII Department of Neurosurgery Juntendo Medical College Tokyo, Japan	1975

RICHARD JOHNSON Department of Neurological Surgery Royal Infirmary Manchester, England	1974
KATSUTOSHI KITAMURA University Kyushu Hospital Faculty of Medicine Fukuoka, Japan	1970
KRISTIAN KRISTIANSEN Oslo Kommune Ullval Sykehus Oslo, Norway	1962
LAURI LAITINEN Department of Neurosurgery 5016 Haukeland Sykehus Norway	1971
WALPOLE S. LEWIN Department of Neurosurgery Addenbrookes Hospital Hills Road Cambridge, England	1973
WILLIAM LUYENDIJK Pr Bernhardlaan 60 Oegstgeest, Netherlands	1973
FRANK MARGUTH Director Department of Neurochirurgischen Universitat Munchen Marchioninistrabe 15 8000 Munchen 70, West Germany	1978
RAUL MARINO, JR. Rua Itaoeva 490, 11 Andar 01000 Sao Paula, SP Brazil	1977

HELMUT PENZHOLZ Director Neurochirurgischen Universitat Heidelberg Gebaudes 110 im Neuenheimer Feld 6900 Heidelberg, West Germany	1978
HANS-WERNER PIA Director Zentrums fur Neurochirurgie Universitat Giessen Klinikstr 37 6300 Giessen, West Germany	1978
B. RAMAMURTHI 2nd Main Road G.I.T. Colony Madras 4, India	1966
KURT SCHURMANN Director Neurochirurg Univ-Klinik Mainz Langenbeckstr 1 6500 Mainz, West Germany	1978
CHARAS SUWANWELA Chulalongkorn Hospital Medical School Bangkok, Thailand	1972
KJELD VAERNET Rigshospitalets Neurokirurgis Tagensvfj 18,2200 Copenhagen, Denmark	1970
SIDNEY WATKINS The London Hospital Whitechapel, London El England	1975
GAZI YASARGIL Neurochirurgische Universitatsklinik Kantonsspital 8000 Zurich, Switzerland	1975

Active Members

EBEN ALEXANDER, JR. Bowman-Gray School of Medicin Winston-Salem, North Carolina		1950
JAMES I. AUSMAN Henry Ford Hospital 2799 West Grand Blvd. Detroit, Michigan 48202	(CAROLYN)	1978
H. THOMAS BALLANTINE, J. Massachusetts General Hospital Boston, Massachusetts 02114	•	1951
GILLES BERTRAND Montreal Neurological Institute 3801 University Street Montreal, Quebec, Canada	(LOUISE)	1967
EDWIN B. BOLDREY University of California Hosp. 3rd Avenue and Parnassus San Francisco, California 94122	(HELEN)	1941
JERALD S. BRODKEY 2065 Adelbert Rd. Cleveland, Ohio 44106		1977
BARTON A. BROWN 2001 Union Street San Francisco, California 94123	(MARTHA)	1968
SHELLEY CHOU University of Minnesota Medical Center Minneapolis, Minnesota 55455		1974
GALE G. CLARK University of California Medical Center San Francisco, California 94143	(MARIAN)	1970

W. KEMP CLARK 5323 Harry Hines Boulevard Dallas, Texas 75235	(FERN)	1970
WILLIAM F. COLLINS, JR. Yale University School of Med. 333 Cedar Street New Haven, Connecticut 06510	(GWEN)	1963
EDWARD S. CONNOLLY Ochsner Clinic New Orleans, Louisiana 70018	(ELISE)	1973
JAMES W. CORRELL 710 West 168th Street New York, New York 10032	(CYNTHIA)	1966
COURTLAND H. DAVIS, JR. Bowman-Gray School of Medicin Winston-Salem, North Carolina		1967
RICHARD L. DeSAUSSURE 920 Madison Avenue Memphis, Tennessee 38103	(PHYLLIS)	1962
DONALD F. DOHN 9500 Euclid Avenue Cleveland, Ohio 44106	(CAROLYN)	1968
R. M. PEARDON DONAGHY Mary Fletcher Hospital Burlington, Vermont 05401	(FRANCES)	1970
CHARLES G. DRAKE University Hospital 339 Windermere Road London, Ontario, Canada N6G 2	(RUTH) 2K3	1958
STEWART B. DUNSKER Mayfield Neurological Institute 506 Oak Street Cincinnati, Ohio 45219	(ELLEN)	1975

GEORGE ENHI 6410 Fannin Street Houston, Texas 77025	(VALAIRE "Lari")	1964
WILLIAM H. FEINDEL Montreal Neurological Inst 3801 University Street Montreal, Quebec, Canada	(FAITH) .itute	1959
ROBERT G. FISHER Rutgers Medical School Piscataway, New Jersey 08	(CONSTANCE) 854	1957
ELDON L. FOLTZ Chairman, Division of Neur University of California School of Medicine Irvine, California 92664	(CATHERINE) rosurgery	1960
RICHARD A. R. FRASER 525 East 68th Street New York, New York 1002		1976
LYLE A. FRENCH University of Minnesota Medical Center Minneapolis, Minnesota 55	(GENE) 455	1954
JOHN T. GARNER 744 Fairmount Avenue Pasadena, California 91105	(BARBARA)	1971
HENRY GARRETSON Health Sciences Center University of Louisville Louisville, Kentucky 40201	(MARIANNA)	1973
SIDNEY GOLDRING Barnes Hospital Plaza Division of Neurosurgery St. Louis, Missouri 63110	(LOIS)	1964
PHILIP D. GORDY 1727 East 2nd Street Casper, Wyoming 92601	(SILVIA)	1968

JOHN R. GREEN Barrow Neurological Institute 302 West Thomas Street Phoenix, Arizona 85013	(GEORGIA)	1953
JOHN W. HANBERY Division of Neurosurgery Stanford Medical Center Palo Alto, California 94304	(SHIRLEY)	1959
MAJ. GEN. GEORGE G. HAY MC USA 303 Skyhill Road Alexandria, Virginia 22314	'ES (CATHERINE)	1962
E. BRUCE HENDRICK Hospital for Sick Children 555 University Avenue Room 1502 Toronto, Ontario, Canada 1X8	(GLORIA)	1968
JULIAN HOFF Department of Neurosurgery University of California San Francisco, California 9414	(DIANNE)	1975
EDGAR M. HOUSEPIAN 710 West 168th Street New York, New York 10032		1976
ALAN R. HUDSON St. Michaels Hospital 38 Shutter Street Toronto, Ontario, Canada M5B LA6	(SUSAN)	1978
WILLIAM E. HUNT Division of Neurological Surge University Hospital 410 West 10th Avenue Columbus, Ohio 43210	(CHARLOTTE) ry	1970
ELLIS B. KEENER 370 Winn Way, #201 Decatur, Georgia 30030	(ANN)	1978

DAVID KELLY Bowman Gray School of Me Winston-Salem, North Carol		1975
WILLIAM A. KELLY University of Washington School of Medicine Seattle, Washington 98195		1977
GLENN W. KINDT University of Michigan Medical Center Ann Arbor, Michigan 48104	(CHARLOTTE)	1977
ROBERT B. KING University Hospital Upstate Medical Center 750 East Adams Street Syracuse, New York 13210	(MOLLY)	1958
WOLFF M. KIRSCH University of Colorado Medical Center Denver, Colorado 80220	(MARIE-CLAIRE)	1971
DAVID G. KLINE Louisiana State University Medical Center 1542 Tulane Avenue New Orleans, Louisiana 700	12	1972
ROBERT S. KNIGHTON 9388 Avenida San Timeteo Cherry Valley, California 92	(LOUISE) 223	1966
RICHARD S. KRAMER Duke Hospital Durham, North Carolina 277	(ROBIN)	1978
THEODORE KURZE 111 Congress Street Suite B Pasadena, California 91105	(BRIGITTE-LAAFF)	1967

THOMAS W. LANGFITT Hospital of the University of Pennsylvania 34th and Spruce Streets Philadelphia, Pennsylvania 191	(CAROLYN)	1971
RAEBURN C. LLEWELLYN Tulane University 1430 Tulane Avenue New Orleans, Louisiana 70012	(CARMEN)	1963
WILLIAM M. LOUGHEED Medical Arts Building Suite 430 170 St. George Street Toronto 5, Ontario, Canada	(GRACE ELEANOR) 1962
HERBERT LOURIE 713 East Genesee Street Syracuse, New York 13210	(BETTY)	1965
ALFRED J. LUESSENHOP Georgetown University Hospita Washington, D. C. 20007	(BETSY) al	1976
ERNEST W. MACK 505 South Arlington Ave. Suite 212 Reno, Nevada 89502	(ROBERTA)	1956
M. STEPHEN MAHALEY, JR Duke University Medical Cente Durham, North Carolina 27706		1972
LEONARD MALIS 1176 Fifth Avenue New York, New York 10029	(RUTH)	1973
FRANK MAYFIELD 506 Oak Street Cincinnati, Ohio 45219	(QUEENEE)	Founder

ROBERT L. McLAURIN Division of Neurosurgery Cincinnati General Hospital Cincinnati, Ohio 45229	(KATHLEEN)	1955
WILLIAM F. MEACHAM Vanderbilt University Hospital Division of Neurosurgery Nashville, Tennessee 37203	(ALICE)	1952
JOHN F. MULLAN University of Chicago Clinics Department of Neurosurgery 950 East 59th Street Chicago, Illinois 60637	(VIVIAN)	1963
BLAINE S. NASHOLD, JR. Duke University Medical Center Durham, North Carolina 27706	(IRENE)	1967
FRANK E. NULSEN Division of Neurosurgery University Hospital 2065 Adelbert Road Cleveland, Ohio 44106	(GINNEY)	1956
GUY L. ODOM (M Duke University Medical Center Durham, North Carolina 27706	ATALAINE) ·	1946
GEORGE OJEMANN University of Washington Department of Neurosurgery Seattle, Washington 98195	(LINDA)	1975
ROBERT G. OJEMANN Massachusetts General Hospita Division of Neurological Surgery Boston, Massachusetts 02114		1968
BURTON ONOFRIO Mayo Clinic Rochester, Minnesota 55901	(JUDITH)	1975

RUSSEL H. PATTERSON, 525 East 68th Street New York, New York 10021		1971
S. J. PEERLESS P.O. Box 5339 Terminal A University Hospital London, Ontario, Canada No	(ANN)	1977
PHANOR L. PEROT, JR. Medical University of South 171 Ashley Avenue Charleston, South Carolina	n Carolina	1970
BYRON C. PEVEHOUSE 2001 Union Street San Francisco, California 94	101	1964
ROBERT W. PORTER 5901 East 7th Street Long Beach, California 9080	(AUBREY DEAN)	1962
0 - ,	, -	
JOHN RAAF 1120 N. W. 20 #100 Pportland, Oregon 97209	(LORENE)	Founder
JOHN RAAF 1120 N. W. 20 #100	(LORENE) (MARY)	Founder 1946
JOHN RAAF 1120 N. W. 20 #100 Pportland, Oregon 97209 AIDEN A. RANEY 125 North Las Palmas	(LORENE) (MARY) 04 (RITA)	
JOHN RAAF 1120 N. W. 20 #100 Pportland, Oregon 97209 AIDEN A. RANEY 125 North Las Palmas Los Angeles, California 9000 JOSEPH RANSOHOFF II New York University Medical Center First Avenue	(LORENE) (MARY) (RITA) (RITA) (EN (CATHERINE) tute	1946

THEODORE S. ROBERTS Division of Neurosurgery University of Utah Medical Ce Salt Lake City, Utah 84132	(JOAN)	1976
JAMES T. ROBERTSON University of Tennessee 800 Madison Avenue Memphis, Tennessee 38163	(VALERIA)	1971
RICHARD C. SCHNEIDER C5135, Out-Patient Building University Hospital Ann Arbor, Michigan 48104	(MADELEINE)	1970
JAMES C. H. SIMMONS 920 Madison Avenue Memphis, Tennessee 38103	(VANITA)	1975
BENNETT M. STEIN New England Medical Center I 171 Harrison Avenue Boston, Massachusetts 02111	(DOREEN) Hosp.	1970
JIM L. STORY 7703 Floyd Curl Drive San Antonio, Texas 78229	(JOANNE)	1972
THORALF M. SUNDT, JR. 200 1st Street, S.W. Rochester, Minnesota 55901	(LOIS)	1971
ANTHONY F. SUSEN 3600 Forbes Avenue Pittsburgh, Pennsylvania 1521	(PHYLLIS)	1965
WILLIAM H. SWEET 1 Longfellow Place Suite 201 Boston, Massachusetts 02114	(ELIZABETH)	1950
RONALD R. TASKER Toronto General Hospital Room 121, U. W. Toronto, Ontario, Canada	(MARY)	1971

JOHN TEW, JR. 506 Oak Street Cincinnati, Ohio 45219	(SUSAN)	1973
GEORGE T. TINDALL Emory University School of Me Division of Neurosurgery 1365 Clifton Road, N. E. Atlanta, Georgia 30322	(SUZIE) ed.	1968
ARTHUR A. WARD, JR. Department of Neurological Sur University of Washington Hosp Seattle, Washington 98105		1953
CLARK WATTS 807 Stadium Road Suite N521 Columbia, Missouri 65212	(PATTY)	1975
W. KEASLEY WELCH Children's Hospital Medical Cer 300 Longwood Avenue Boston, Massachusetts 02115	(ELIZABETH) nter	1957
BENJAMIN B. WHITCOMB 85 Jefferson Street Hartford, Connecticut 06106	(MARGARET)	1947
LOWELL E. WHITE, JR. Professor & Chairman Division of Neurosciences Mobile, Alabama 36688	(MARGIE)	1971
ROBERT WILKINS Duke University Medical Cente Box 3807 Durham, North Carolina 27710	r	1973
CHARLES B. WILSON Department of Neurological Sur University of California Medica Third and Parnasus San Francisco, California 94143	l Center	1966

FRANK WRENN 123 Mallard Street Greenville, South Carolina 29601	(BETTY)	1973
DAVID YASHON 410 West 10th Avenue, N. #911 Columbus, Ohio 43210	(MYRNA)	1972
NICHOLAS T. ZERVAS 330 Brookline Avenue Boston, Massachusetts 02215	(THALIA)	1972

Deceased Members	Date	1	Elected
DR. SIXTO O'BRADOR ALCALDE Madrid 10, Spain	(Honorary)	4/27/78	1973
DR. JAMES R. ATKINSON Phoenix, Arizona	(Active)	2/78	1970
DR. PERCIVAL BAILEY Evanston, Illinois	(Honorary)	8/10/73	1960
DR. WILLIAM F. BESWICK Buffalo, New York	(Active)	5/12/71	1949
DR. SPENCER BRADEN Cleveland, Ohio	(Active)	7/20/69	Founder
DR. F. KEITH BRADFORD Houston, Texas	(Active)	4/15/71	1938
DR. WINCHELL McK. CRAIG Rochester, Minnesota	(Honorary)	2/12/60	1942
DR. WESLEY A. GUSTAFSON Jensen Beach, Florida	(Senior)	7/16/75	1942
DR. HENRY L. HEYL Hanover, New Hampshire	(Senior)	3/1/75	1951
DR. OLAN R. HYNDMAN lowa City, Iowa	(Senior)	6/23/66	1942
MR. KENNETH G. JAMIESON Brisbane, Australia	(Correspon	ding) 1/28/7	76 1970

SIR GEOFFREY JEFFERSON Manchester, England	(Honorary)	3/22/61	1951
DR. DONALD D. MATSON Boston, Massachusetts	(Active)	5/10/69	1950
DR. KENNETH G. McKENZIE Toronto, Ontario, Canada	(Honorary)	2/11/64	1960
DR. JAMES M. MEREDITH Richmond, Virginia	(Active)	12/19/62	1946
DR. W. JASON MIXTER Woods Hole, Massachusetts	(Honorary)	3/16/58	1951
DR. WILDER PENFIELD Montreal, Canada	(Honorary)	4/5/76	1960
DR. RUPERT B. RANEY Los Angeles, California	(Active)	11/28/59	1939
DR. DAVID L. REEVES Santa Barbara, California	(Senior)	8/14/70	1939
DR. DAVID REYNOLDS Tampa, Florida	(Active)	4/3/78	1964
DR. SAMUEL R. SNODGRASS Nashville, Indiana	(Senior)	8/8/75	1939
DR. C. WILLIAM STEWART Montreal, Quebec, Canada	(Correspond	ing)	1948
DR. GLEN SPURLING La Jolla, California	(Honorary)	2/7/68	1942
DR. HENDRIK SVIEN Rochester, Minnesota	(Active)	6/29/72	1957

AMERICAN ACADEMY OF NEUROLOGICAL SURGERY 1979 ANNUAL MEETING

EVALUATION

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

(1) Was the general content of the scientific program:			
	Excellent Good Poor		
(2) If you found it po	or, 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	aim their talks:		
	Foo low Foo high Tust 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:		
		
• • •	2.4	
	•	tina e e e
What changes would yo	ou like to see in future meetin	ıgs?
		· · · · · · · · · · · · · · · · · · ·
	·····	
	•	
Change of address and	or telephone (indicate office	
	<u> </u>	
	•	
Please print Name:	grand v	
	•	
Return to: Phanor L. I 171 Ashley		
	South Carolina 29403	* • · · · · · · · · · · · · · · · · · ·

