AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

Thirty-Seventh
Annual Meeting

The Wigwam
Litchfield Park (Phoenix) Arizona

November 5-8, 1975

ANNUAL MEETING 1975

THE WIGWAM

LITCHFIELD PARK (PHOENIX)

ARIZONA

NOVEMBER 5-8, 1975

THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

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PROGRAM 1975

Wednesday, November 5

12:00 - 6:00 p.m. ..... Registration
The Lobby
3:00 p.m. ..... Executive Committee Meeting
John Green's Suite
6:30 - 8:00 p.m. ..... Reception
Sachem Hall West
8:00 p.m. ..... Dinner (American Plan)
Terrace Dining Room
(Section reserved for Academy members.)

Thursday, November 6

7:30 - 12:30 ..... Scientific Session — Arizona Room
9:55 ..... Coffee Break
12:30 ..... Business meeting
8:00 - 12:00 ..... Registration — The Lobby
8:00 - 12:00 ..... Ladies Hospitality — Sun Lounge
9:00 - 12:00 ..... Ladies Heard Museum Tour
8:00 - 12:00 ..... Ladies Alternate Activities
(round robin tennis, golf, horseback riding)
1:00 ..... Luncheon — Pool-side Buffet
1:30 - 5:30 ..... Golf (Ernest Mack)
Tennis (Georgia Green)
5:30 ..... Western Steak Cookout
(Buses leave from entrance.)

Friday, November 7

7:30 - 12:30 ..... Scientific Session — Arizona Room
10:05 ..... Coffee Break
12:30 ..... Business Meeting
8:00 - 12:00 ..... Registration — The Lobby
8:00 - 12:00 ..... Ladies Hospitality — Sun Lounge
9:00 ..... Ladies Scenic Grand Canyon Flight
8:00 - 12:00 ..... Ladies Alternate Activities
(round robin tennis, golf, horseback riding)
1:00 p.m. ..... Luncheon — Pool-side Buffet
2:00 p.m. ..... Scenic Grand Canyon Flight
(Members, guests, wives)
1:30 - 5:30 ..... Golf (Ernest Mack)
Tennis (Georgia Green)
6:30 - 7:30 ..... Cocktails — Sachem Hall
7:30 ..... Official Academy group photograph,
members and guests
7:30 - 12:00 ..... Academy dinner-dance (black tie optional)
Sachem Hall

Saturday, November 8

7:30 - 12:30 ..... Scientific Session — Arizona Room
9:50 ..... Coffee break
8:00 - 12:00 ..... Ladies Hospitality — Sun Lounge

Scientific Program

THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
Litchfield Park, Arizona

November 5 - 8, 1975

THURSDAY, NOVEMBER 6

7:30 a.m.

Welcoming Remarks and Announcements

CEREBRAL VASOSPASM

7:35 a.m.

1. Experimental Cerebral Vasospasm: An Angiographic and Pathologic Comparison

Richard A. R. Fraser and Russel H. Patterson, Jr.
New York, N. Y.

Cerebrovascular spasm in association with subarachnoid hemorrhage provides the major source of morbidity and mortality in aneurysm patients surviving their first subarachnoid hemorrhage. At present, our clinical management of such patients is confused. This stems from our inability to recognize when angiographically identifiable vasospasm is associated with significantly reduced cerebral blood flow (CBF) or a potential for CBF reduction. Prior clinical and experimental CBF studies of vascular spasm have not revealed consistent blood flow changes in either direction despite severe spasm demonstrated by angiography. As part of an investigation into cerebrovascular spasm, we have attempted to confirm or deny a CBF perfusion deficit in experimental cerebrovascular spasm, and further to correlate this with the degree of spasm.

Twenty Rhesus monkeys underwent bi-brachial angiography, and subsequent subarachnoid hemorrhage. These animals were rearteriogrammed at daily intervals and the presence or absence of arterial narrowing documented. Animals were sacrificed at varying intervals after subarachnoid hemorrhage (immediate to five days). Each animal was perfused with carbon black at arterial pressure. The brains were then removed and placed in formalin. Each brain was later photographed, serially sectioned and photographs made of each section. The circle of Willis in each animal was submitted for pathological studies. The above data was compared with angiograms.

In this small series no correlation was found between the severity of the spasm measured by angiography and the existence of non-carbon black perfused areas of the brain In several instances, gross perfusion deficits were apparent in animals with apparently adequate subarachnoid hemorrhage but only minor angiographic spasm. Conversely, severe spasm was occasionally associated with normal perfusion. No correlation existed between the degree of spasm perfusion deficits and neurological function. These data suggest that:

(1) Angiography is an inadequate means of evaluating cerebrovasospasm.

(2) That vasospasm in association with subarachnoid hemorrhage involves both large caliber ("conductive") arteries and the smaller arteries and arterioles ("resistance vessels").

(3) Large caliber vessels provide a negligible contribution to total CBF under normal circumstances.

(4) We suggest that spasm may adversely contribute to the patient's clinical status only when cerebrovascular resistance has significantly increased; either by involvement of a significant number of "resistance" pial arteries and arterioles, or by conversion of the large caliber "conductive" arteries to a significant "resistance" function.

7:50 a.m.

2. The Role of Cyclic Nucleotides in Cerebral Vasospasm

Eugene S. Flamm and Joseph Ransohoff
New York, N. Y.

Agents known to alter the levels of cyclic AMP have been shown effective in altering experimental cerebral vasospasm. Levels of cyclic AMP in circulating blood and in the basilar artery of cats were determined following the production of spasm and following treatment with agents known to alter cyclic AMP. Drugs known to stimulate adenylate cyclase, as well as inhibit phosphodiesterases, thus impairing the degradation of cyclic AMP, were studied alone and in combination. Cyclic AMP of the basilar artery fell from a mean control value of 50 picomoles/mg protein to a mean of 25 picomoles/mg protein following the production of spasm. Those drugs that produced a dilatation of the basilar artery following the application of blood to the vessel were found to elevate the vascular cyclic AMP as high as three-fold to a mean of 120 picograms/mg protein. The clinical implications of this work have been explored and will be presented.

8:05 a.m.

Discussion of 1 and 2

NEUROSURGICAL CONSIDERATIONS IN SOME NEUROLOGICAL AND MEDICAL DISEASES

8:15 a.m.

3. Neurologic Problems Seen in Patients Receiving Chronic Renal Dialysis

Stewart B. Dunsker, Tom Saul, Ken Neumarck, and Frank H. Mayfield
Cincinnati, Ohio

Chronic renal dialysis is being used in many hospitals and home programs throughout the country. As more experience has accumulated, various neurologic problems and syndromes have been encountered. Their incidence is unknown, but they should be recognized.

This paper will present a discussion of the following neurologic problems seen in patients receiving chronic renal dialysis.

1. Subdural Hematoma: a treatable cause of dementia and headache, probably resulting from use of anticoagulants during dialysis treatments.
2. Dialysis Dementia: idiopathic confusion, myoclonus and seizures progressing to death. The duration ranges from several weeks to several years.
3. Neuropathy: progressive loss of vibratory and position sense. Its progression may serve as a guide to the need for dialysis.
4. Myopathy: a progressive proximal muscle weakness. It is not clear if this is separate from neuropathy.
5. Autonomic neuropathy: drop in blood pressure without a corresponding tachycardia. Responds to small doses of epinephrine.
6. Disequilibrium syndrome: occurs in early course of dialysis treatments. Characterized by severe headache and sometimes seizures. Probably caused by disequilibrium of fluid and elecrolytes as the uremic picture is corrected.
7. Seizures: seen with disequilibrium syndrome, subdural hematoma and dialysis dementia.
8. Vascular disease: diffuse atherosclerosis progresses at an accelerated rate. Patients are prone to all the complications of diffuse atherosclerosis including stroke, myocardial infarction.

Discussion

8:35 a.m.

4. Peri-Operative Mini-Dose Heparin Administration to the Neurosurgical Patient

Raeburn C. Llewellyn and Hugh Glen Barnett
New Orleans, Louisiana

Thrombophlebitis and pulmonary embolus in the postoperative and spinal cord injured neurosurgical patient has long been a source of increased morbidity and mortality. Recent work using I^{125} fibrinogen screening points out the magnitude of the problem of deep venous thrombosis. A recent report has shown a 43% incidence of deep venous thrombosis in neurosurgical patients screened with I^{125} fibrinogen. There are many other reports showing high incidences of deep venous thrombosis in orthopedic, general, and urological surgery patients.

Mini-dose heparin administration to general and orthopedic surgery patients has shown that this treatment is both feasible from an operative standpoint and helpful in reducing the incidence of postoperative thrombophlebitis and pulmonary embolus. There has been no report of the use of peri-operative mini-dose heparin in the neurosurgical patient. This study reports on the preoperative and postoperative administrations of heparin to 150 elective adult neurosurgical patients. These patients were evaluated for bleeding disorders preoperatively by hematological tests and clinical history. If this evaluation was within normal limits, the patient received 5,000 units aqueous heparin subcutaneously on call to surgery and every 12 hours postoperatively. The length of heparin administration was varied according to the length of bed confinement.

Our study has shown that it is feasible to do a wide range of neurosurgical procedures on patients receiving preoperative heparin. There was no increase in blood loss at surgery or difficulty with hemostasis.

Discussion

8:55 a.m.

5. Reye's Syndrome: Observations on Surgical Treatment and Ultrastructural Pathology

Robert L. McLaurin
Cincinnati, Ohio

During the past year three patients with Reye's syndrome have undergone extensive craniodecompression as a life-saving procedure.

All three patients have survived and two have made full neurologic recovery while the third has residual behavioral deficit. The criteria for selection of these patients from a larger number who have been treated by exchange transfusion have consisted primarily of failure to respond to exchange transfusion and clinical signs consistent with impending death from intracranial decompensation.

The technical features of craniodecompression including replacement of preserved bone flaps will be reviewed. Cortical biopsies have been made at the time of decompression and again at the time of cranioplasty in each case. The typical ultrastructural pathology has been noted and described. The cardinal ultrastructural changes consist of astrocyte swelling, myelin bleb formation and injury to neuronal mitochondria. These changes are reversible as indicated by later biopsies.

Discussion

9:15 a.m.

6. Significance of Febrile Infantile Convulsions in Focal Epilepsy

Theodore Rasmussen
Montreal, Canada

While it is well know that prolonged febrile convulsions in infancy can produce significant brain damage, the relative importance of isolated infantile convulsions, in general, febrile or non-febrile, as a cause of focal epilepsy and in particular, temporal lobe epilepsy, is not agreed upon. About 15% of the 1409 cases of medically refractory focal epilepsy who have undergone cortical resection at the Montreal Neurological Institute have a history of one or more isolated infantile convulsions, usually associated with fever well before the onset of the recurring seizures. In nearly a third of these patients the infantile convulsion was associated with an identifiable febrile illness (measles, encephalities, meningitis, etc.) which probably caused brain injury and the convulsion. In another fourth of these patients there was definite brain injury at birth and the fever triggered off a seizure tendency which was developing presumably due to a pre-existing birth brain injury. In additional 7% some other pre-existing cause for brain injury was present. In the remaining 42% of the patients with a history of isolated infantile convulsions there was no other obvious cause for brain injury and epileptogenicity. These patients represent 7% of the total patients in this surgical seizure series. These data suggest that isolated febrile infantile convulsions, not associated with an identifiable brain disease, are a potential cause for a small but definite percentage of cases of focal epilepsy. Somewhat more frequently a febrile episode in childhood triggers off a maturing seizure tendency due to some pre-existing brain injury or lesion.

Discussion

9:35 a.m.

7. The Cranial Nerve Dysfunction Syndrome. Etiology and Definitive Microsurgical Treatment

Peter J. Jannetta
Pittsburgh, Pennsylvania

By serendipitous observation, the author noted arterial cross-compression of the trigeminal nerve in a patient with tic douloureux who was operated upon using the surgical binocular microscope in 1966. By extrapolation from a sensory to a motor nerve, vascular cross-compression at the brainstem was first noted and successfully relieved in a patient with hemifacial spasm in 1966. Further extrapolation was subsequently carried to cases of glossopharyngeal neuralgia and dysfunction of the acoustic nerve (Meniere's disease (?) and variants). All of these entities appear to be caused by mechanical compression-distortion of the appropriate cranial nerve in the cerebellopontine angle, almost always vascular and usually arterial, and precisely at the nerve root entry zone in all cases except those in whom symptoms are purely cochlear. Isolated cochlear dysfunction may be due to more peripheral neural cross-compression in the cerebellopontine angle, presumably because central nervous system myelin travels all the way to the porus acusticus. Analysis of the first 117 of over 300 operative cases with 100% follow up will be presented. It is of interest that the symptoms of both hyperactive dysfunction (i. e. pain, muscle, spasms, tinnitus, vertigo, nystagmus) and hypoactive dysfunction (i. e. facial numbness, weakness, decreased hearing, decreased vestibular function) are relieved by neurovascular decompression using microsurgical techniques through a small retromastoid exposure. These patients were operated upon from February, 1966 through December, 1974. Morbidity and mortality have been minimal. There has been no mortality in over 175 recent cases. Operative findings will be demonstrated with the use of 16 mm. color motion picture film and 35 mm. color slides.

Discussion

9:55 a.m.

Coffee Break

10:30 a.m.

8. Hypophysectomy in Diabetic Retinopathy

Nicholas T. Zervas, Richard Field, and
William Sweet — Boston, Massachusetts

During the period of 1958-1975, 248 patients underwent either pituitary stalk section or radiofrequency hypophysectomy for angiopathic hemorrhagic diabetic retinophaphy.

Three operative deaths ocurred prior to 1964 — these were due to: 1) myocardial infarction; 2) pulmonary embolism; and 3) hypoglycemia. One patient died of meningitis six months following operation in 1965.

Complications: meningitis ocurred in two patients but responded to proper antibiotic therapy; visual complications failed to occur. Four patients were not panhypopituitary and required secondary operations.

The average overall survival for all patients was 6.9 years. 20 patients have survived more than ten years with continuing arrest and/or regression of diabetic retinophaphy. Overall visual stabilization for the entire group was 79% as compared to less than 20% for untreated patients.

With advances in laser therapy, the indications for pituitary ablation are quite specific. Hypophysectomy should be reserved for patients under age 40 who have received maximal Argon Laser therapy and whose visual loss continue to progress or who cannot receive laser therapy because of the proximity of the lesions to the macula. The authors stress the point that young patients who continue to lose vision despite laser therapy should be evaluated for pituitary ablation before vision is lost since visual function can be preserved during the lifetime of the majority of patients.

Discussion

ACADEMY AWARD

10:50 a.m.

9. Dorsal Column Stimulation: Its Effect on Medial Bulboreticular Unit Activity Evoked by Noxious Stimuli.

Andrew Shetter
Phoenix, Arizona

SPINAL CORD TRAUMA

11:20 a.m.

10. The Role of Vesicular Transport in the Pathogenesis of Edema Following Compression Injury to the Spinal Cord

John L. Beggs and John D. Waggener
Phoenix, Arizona

Routes of vascular leakage resulting in trauma-induced edema have not been clarified. To explore the problem we followed the fate of intra-

vascular horseradish peroxidase (HRP) (100 mg/kg wt) after compression injury (200 gm/cm^2-5 min) of the thoracic cord (cats). At 90 sec and up to 1 hr HRP was confined to the gray matter, occupying perivascular spaces, unexpanded extracellular channels, and cytoplasmic compartments of injured cells. Adjoining segments contained similar but lesser deposits. At 4 hr tracer occupied the expanded extracellular space of the lesion white matter; gray matter deposits were present up to 3 cm distal. Vessels revealed no evidence of rupture. Open interendothelial junctions were not found. At 1 hr gray matter vessels (less than 10 microns) in the lesion exhibited a 10 fold increase in the number of HRP-filled vesicles (over controls). A 5 fold increase was found in adjoining segments. Caudally, vesicle counts diminished as a function of distance from the lesion; enchanced activity persisted up to 9 cm. HRP (quantitative assay) in segmental cord extracts correlated closely with vesicle frequency. Where basement membrane was not yet inundated with HRP, vesicles open to the abluminal surface appeared to be releasing tracer. Serial sections revealed that vesicles could be adjoined contiguously from luminal to abluminal surfaces, thus providing facilitated pathways. The data suggest that vesicular transport plays a significant role in the genesis of trauma-induced edema.

Discussion

11:40 a.m.

11. Experimental Spinal Cord Injury Models: Their Evaluation and Use

John C. VanGilder and W. F. Collins, Jr.
New Haven, Connecticut

Experimentally produced spinal cord trauma in animal models has proven to be a significant technique in the study of spinal cord injury. In order to use animal injury models for assessing the efficacy of a spinal cord injury treatment regime, it is necessary to establish the predictability of the untreated response to the experimental injury. By standardizing the entire experimental procedure with respect to the traumatizing device and the major physiological variables of the animal at the time of trauma, a predictable response to thoracic spinal cord injury in cats was demonstrated in 150 animals. The responses have been analyzed as a trauma dose-response curve (TDR) with the trauma dose being calculated as the potential gm-cm of drop force used for injury and the response being changes in cortical evoked responses, neurological function, and tissue pathology of the traumatized segment. The control trauma dose-response curve from 100-700 potential gm-cm force was compared with the trauma dose-response curve in experiments in which the animals received steroids, mannitol aminocaproic acid, ethyl alcohol, mylotomy or hypothermia as a therapy of spinal cord injury. The ef-

fects of the aforementioned drugs or surgical manipulation were found to be limited to the approximate mid portion of the TDR curve with no significant variations seen at the extremes of trauma.

Our conclusion from these studies is that inconsistencies in reports from various laboratories concerning response or lack of response in the therapy of experimental spinal cord injuries may relate to lack of standardization of experimental parameters and/or failure to study the significant portions of the trauma dose-response curve of these experimental injury models.

Discussion

NEUROSURGICAL MANPOWER

12:00 p.m.

12. Report and Discussion — By Invitation

Joseph Ransohoff
New York, N. Y.

12:30 p.m.

Business Meeting

FRIDAY, NOVEMBER 7

BRAIN TUMORS

7:30 a.m.

13. Preservation of Hearing with the Malis Procedure for Medium-Sized Acoustic Neurinomas

Wolff M. Kirsch and Gerald English
Denver, Colorado

Risks associated with acoustic tumor surgery bear a direct relationship to the size of the neoplasm. The hazards of medium size tumor removal include eighth nerve dysfunction to facilitate conservation of the seventh cranial nerve. The oblique approach to the cerebello pontine angle conceived by Dr. Leonard Malis has not received wide publicity. This report deals with the use of the Malis Procedure in the treatment of three successive medium-size acoustic neurinomas with preservation of useful hearing in addition to facial nerve function. The technical aspects of the procedure will be discussed. It would appear that this approach would render the translybyrinthine dissection with the attendent loss of hearing obsolete for medium-size acoustic neurinomas.

7:45 a.m.

14. Microsurgical Preservation of Hearing in Acoustic Neuroma

Leonard Malis
New York, N. Y.

Serviceable hearing was present preoperatively in 7 of the last 100 patients with acoustic neuroma. With total microsurgical removal by the retrolabyrinthine posterior fossa lateral approach, useful hearing was preserved in 5 of these 7 patients. The tumors ranged in size from 1.2 cm to 3.0 cm.

The techniques and the complications will be discussed, as will the need to abandon the translabyrinthine approach.

8:00 a.m.

Discussion of 13 and 14

8:10 a.m.

15. Unilateral Proptosis and Intracranial Schwannoma

E. Bruce Hendrick and Anthony D. Hockley
Toronto, Canada

Two cases of unilateral proptosis associated with intracranial schwannomas in children are described. In both cases, the tumour occupied both the orbit and middle cranial fossa, where in one case the intracranial component was extradural and in the other intracerebral. The possible sites of tumour origin are discussed.

Discussion

8:30 a.m.

16. Screening of Immunotherapy and Chemotherapy Protocols Utilizing the Avian Sarcoma Virus-Induced Anaplastic Glioma as a Model

M. S. Mahaley, Darell Bigner, and Bob Gentry
Durham, North Carolina

Several chemotherapy and immunotherapy protocols of treatment, separately and in combination, have been evaluated in an experimental brain tumor model system. The model consists of anaplastic gliomas

induced in rats by inoculation with avian sarcoma virus. The therapy protocols have included immunotherapy with BCG and sarcoma cell inoculation. Chemotherapy has included intravenous administration of BCNU, an agent which is currently under study and the treatment of human gliomas. The results of the studies so far have suggested some mild toxicity with BCG alone and what appears to be a significant prolongation of survival time of animals treated with the combination of immunotherapy and chemotherapy. It is anticipated that screening studies with this model system will permit selection of adjunctive modes of therapy which can be applied to the eventual treatment of human anaplastic gliomas.

Discussion

8:50 a.m.

17. ACTH-Secreting Pituitary Tumors

Charles B. Wilson and Larry Pitts
San Francisco, California

During the past three years the authors have treated twenty-five patients harboring ACTH-secreting pituitary adenomas. The majority of adenomas became evident following adrenalectomy, but an increasing proportion of patients with active Cushings disease are being referred for surgery. These tumors present a spectrum ranging from 4 mm. adenomas presenting as Cushings Disease to the extremely aggressive adenomas producing Nelson's syndrome.

Present evidence favors a pituitary origin in a significant proportion of patients with Cushings disease. The aggressive behavior of many adenomas discovered after adrenalectomy argues for selective microscopic removal of small adenomas as an alternative to bilateral adrenalectomy. We have documented Hardy's experience of removing microadenomas with restoration of normal pituitary and adrenal function. The selection of patients is a matter requiring additional experience and perhaps diagnostic methods that are not presently available. Our criteria for recommending transsphenoidal exploration in the presence of a suspected ACTH-secreting adenoma will be presented.

Discussion

INTERVERTEBRAL DISC PROBLEMS

9:10 a.m.

18. Anterior Removal of Cervical Disc Without Fusion

James T. Robertson
Memphis, Tennessee

A long term followup of a significant series of cervical discs removed anteriorly without fusion will be presented. The incidence of fusion and the indications for the procedure will be discussed in detail. An effort will be made to try to firmly establish the criteria for anterior removal of cervical discs without fusion and contrast these results against the posterior approach as well as the approach with fusion.

Discussion

9:30 a.m.

19. The Clinical Use of Chymopapain

Burton M. Onofrio
Rochester, Minnesota

With four years experience using chymapapaise I am ready to evaluate the results to date; its efficacy, complicated late recurrences, disc surgery later at other levels. With the results, the proper place in my practice of neurosurgery including patient selection will be discussed.

9:45 a.m.

20. Curent Status of Double Blind Studies on Chymopapain

(Special Report and Discussion — By Invitation)

Robert S. Knighton
Detroit, Michigan

10:05 a.m.

Coffee Break

NEUROSURGERY AND NEURORADIOLOGY

10:30 a.m.

21. A New Technique for Evaluation of Data from Computed Brain Tomography

George Ehni and **Robert A. Evans**
Houston, Texas

Computerized axial tomography produces digital information used to compute absorption coefficients of the material being examined. Display of this data is normally accomplished by converting the digital in-

formation into an analog picture where a usually small number of discrete values (window) are used to generate the pictures. By sliding this window through the data range, slight changes in absorption values are detected. Archival and diagnostic hard copy is then achieved by taking pictures of the information on the CRT and by printing out the absorption values on a tabulated sheet.

A new method for the analysis of data generated by computerized tomography has been developed and clinically tested. Using a unique technique of combining digital and analog information into one display, anatomic information is precisely defined by multi-level gray shade picture elements. Each picture element is generated as a directly readable number representing the absorption coefficient value as computed by the scanner system. Subtle changes in soft tissue are readily detected and can be quickly evaluated. Material composition of more pronounced changes can be accurately determined from the data since the absorption coefficients are presented as numbers. The numbers used to generate a display may range from as few as 10 values to as many as 70 values. This increased range of unambiguous gray shades enables the radiologist to examine a wider spectrum of material while maintaining the ability to detect slight changes in density.

Results from clinical tests indicate that certain pathology is more easily detected, the involved area is more accurately defined and the composition of the material more quickly determined.

10:45 a.m.

22. Recent Advances In Computerized Tomographic Scanning

(Special Report and Discussion — By Invitation)

Juan Taveras
Boston, Massachusetts

11:20 a.m.

23. Further Experiences in Angiographic Dynamics Following Embolization of Large Cerebral Hemisphere Arteriovenous Malformations

Bennett M. Stein and Samuel Wolpert
Boston, Massachusetts

Fifteen cases of large cerebral arteriovenous malformations in a variety of sensitive locations have now been successfully embolized and resected. Circulatory changes following single or repeated embolizations will be demonstrated by serial angiography. Complications due to the embolization procedure will be discussed in some detail.

The surgical removal was greatly facilitated by preoperative embolization. In no case was there evidence of untoward vascular dynamics occurring within the malformation such as hemorrhage or swelling due to the embolization. Histological evaluation of the resected specimens indicate that the emboli lodge primarily in the arterial side of the malformation although this is not inevitable.

Embolization is a safe and adjuvant means of treating patients with large hemispheral arteriovenous malformations. Embolization may be carried out with up to 3 mm. sized spheres in either the anterior or posterior circulations of the cerebral hemisphere.

Discussion

BRACHIAL PLEXUS

11:40 a.m.

24. Posterior Approach for Selected Brachial Plexus Lesions

David G. Kline, Lester Bryant, and Raymond Dahl
New Orleans, Louisiana

Claggett's (1962) posterior approach for resection of the first rib was based on earlier operative approaches for pulmonary tuberculosis and thoracoplasty. While resecting the first rib by posterior approach in a patient who also had an old brachial plexus injury, it was noted that the approach afforded exceptional exposure of roots as well as trunks of the plexus and permitted early exposure and thus protection of the great vessels. Subsequently, five more posterior approaches have been done on patients with proximal plexus lesions due to tumor, gunshot wound, contusion, and laceration. The procedure has particular merit in cases where there has been prior anterior operation and thus heavy scar. Technique includes elevation of the scapula as well as rib resection and thus was only used in selected instances, the remaining 30 operative patients in our brachial plexus series having more classical approached used.

Details of the posterior operative approach will be shown as well as a brief historical review of its place in Surgery.

Discussion

THE PRESIDENTIAL ADDRESS

12:00 p.m.

25. Is Voluntary Regionalization of Neurological Surgery Feasible?

John R. Green
Phoenix, Arizona

12:30 p.m.

Business meeting

SATURDAY, NOVEMBER 8

CEREBRAL TRAUMA

7:30 a.m.

26. Cerebral Blood Flow and Metabolism Following Experimental Missile Injury to the Brain

H. Alan Crockard, Frederick D. Brown,
and Sean Mullan
Chicago, Illinois

Twenty-six Rhesus monkeys were used in a project to measure CSF, CMRO2, CMR Lactate as well as ICP etc. The stereotaxically placed injury to the right cerebral hemiaphere was adjusted to give a known energy to the tissues. Following injury there was an immediate rise in ICP and a bradycardia; CSF became dependent on perfusion pressure. CMRO2 fell to less than 50% of control at one minute and remained low for the following hour, while the CMR lactate rose to 200% of control at 10 minutes and then subsided. A nine variable regression analysis of the data was compared to survival time after injury; this produced a good mathematical fit (reg. coeff 0.96). The most significant data to predict survival time was that obtained within the first 10 minutes after injury and in order of importance was 1) CMR lactate at one minute and 10 minutes. 2) CMRO2 at 10 minutes and 3) Perfusion pressure at 10 minutes after trauma.

Using this type of physiological preparation and data analysis it is hoped to construct mathematical models which will be used in the evaluation of therapy.

Discussion

7:50 a.m.

27. The Clinical and Laboratory Significance of Intracranial Pressure-Reserve Testing

H. A. Wilkinson, S. Weems, D. Kaner,
and D. Arredondo
Boston, Massachusetts

The clinical importance of continuous monitoring of intracranial pressure (ICP) has gained widespread acceptance, limited primarily by technical problems. Less well recognized but perhaps equally important

is the possibility of quantitating ICP reserve or "cerebral elastance." This dynamic parameter is a measurement of the remaining capacity of the brain's natural compensatory mechanisms for counteracting increases in ICP. With increasing intracranial mass, ICP rises at a drastically sharper rate once the ICP reserve mechanisms have been exhausted, leading to brain herniations and rapid cerebral deterioration. Measurements of ICP reserve should be of predictive value in impending cerebral decompensation.

The reliability of ICP reserve testing was proven in dogs with known volumes of extracerebral intracranial mass introduced in normal and edematous brains and at various intracranial pressures. Initial human studies in postcraniotomy patients have confirmed the impression of extreme usefulness and safety of the method.

Discussion

8:10 a.m.

28. Cerebral Concussion: A New Theory

Robert G. Fisher, Joseph W. Young,
G. Townly Price, and A. Reozin
Plainfield, New Jersey

The accepted theories of cerebral concussion are reviewed. We wish to present a new theory of paralytic coma resulting from head impact.

Concussion, we believe, is a transient or permanent temporal lobe herniation due to violent cerebral mass changes at the time of impact and this is labelled reversible or irreversible. Concussion is the earliest event occuring in all cases of coma resulting from head injury.

We cite clinical and laboratory events to justify this theory.

Discussion

8:30 a.m.

29. Traumatic Aneurysms of the Petrous Carotid Artery with Eustachean Tube Hemorrhage

D. Wayne Laster, George A. Gates,
and **Jim L. Story**
San Antonio, Texas

Traumatic petrous carotid aneurysms are rare. The presence of such an aneurysm may result in delayed, intermittent and fatal epistaxis by way of the eustachean tube.

This report deals with a review of the literature and our management of a patient with such an aneurysm by the direct surgical approach.

8:50 a.m.

30. Phantom Pain. Relief by Focal Destruction of Substantia Gelatinosa

Blaine S. Nashold, Jr., David S. Zorub,
and Bruno Urban
Durham, North Carolina

The origin of phantom limb pain is still unknown. Although central mechanisms have been implicated, there is little direct evidence for either the theory of partial deafferantation hypersensitivity or breakdown of the control of the spinal gate due to local pathophysiology in the spinal cord. The present observations were made in three patients with phantom limb pain due to total avulsion of the brachial plexus. Although the C5 to T1 roots were avulsed, the pain was localized primarily to the distribution of the C5-C6 nerve root. We believe the pain from this disorder originates from the secondary neuron which originates in the dorsal root entry zone, forming the spinothalamic tract. The cervical spinal cord was exposed over the area of the avulsion. A fine stainless steel electrode was then introduced into the depths of the cord and a series of multiple coagulations (16) were made in the region of the substantia gelatinosa. All patients experienced initial complete relief of phantom pain with the average follow-up now six months. These observations strengthen our idea of the importance of the dorsal root entry zone as a possible origin of central pain.

Discussion

9:10 a.m.

31. Reflex Syncope Associated with Glossopharyngeal Neuralgia

Marc A. Levin and Donald F. Dohn
Cleveland, Ohio

Glossopharyngeal neuralgia is a relatively rare condition. Thirty-one cases have been seen at the Cleveland Clinic during the past 30 years. Four of these patients had syncope associated with the pain paroxysms as a result of a cordiovascular reflex phenomenon.

The clinical manifestations of these four patients will be presented. Our most recent patient had dramatic and disabling episodes of cardiac slowing at times with syncope, with each paroxysm. His problem was managed with a transvenous demand cardiac pacemaker followed by

surgery. Surgery revealed an abnormal loop of PICA compressing the 9th and 10th cranial nerves. Neurolysis of the nerves and separation of the vessel successfully relieved the problem.

Compressive lesions as a cause of glossopharyngeal neuralgia has been reported by others. These etiologic factors, including vascular anomalies, will be discussed. Theoretical possibilities concerning the mechanism of the associated cardiovascular reflex syncope will also be presented.

Discussion

9:30 a.m.

32. Neurological Surgery and the Discipline of Neuro-science

Lowell E. White, Jr.
Mobile, Alabama

Neuroscience is spoken of as an emerging discipline in many academic circles. The knowledge base upon which the discipline is founded is broad and multi-disciplinary. This fragmentation of the subject between Colleges of the University as well as within the usual College of Medicine leads to decreased quality and relevance of the curriculum on the nervous system. The present state of Neuroscience in Medical Schools will be discussed in terms of the developing program at the University of South Alabama. This program encompassed by the Division of Neuroscience includes the following Departments: Neurobiology, Psychiatry, Neurological Surgery, Neurology, Ophthalmology and Otolaryngology. The one discipline which interfaces with all of the others is Neurological Surgery. This observation indicates a potential leadership role for the academically inclined Neurological Surgeon.

9:50 a.m.

Coffee Break

CEREBRAL VASCULAR OCCLUSIVE DISEASE

10:20 a.m.

33. Collateral Flow in the Cerebral Microcirculation

William Feindel, Henry Garretson, Patrick Murray,
L. Yamamoto, and C. P. Hodge
Montreal, Canada

Neurosurgical techniques dealing with the arterial supply to the brain rely heavily on the principle of collateral flow first elucidated by Thomas Willis (1621-75). He demonstrated the functional anatomy of

the arterial circle or polygon which bears his name and also noted the absence of neurological deficit in a dog after carotid ligation and in a patient with carotid occlusion later proven postmortem. Using fluorescein angiography during craniotomy in cerebral vascular occlusive disorders, we have demonstrated now that the Willisian Principle of collateral flow applies to the smaller epicerebral arteries and veins and to the microcirculation of the cortex, which unfortunately are not visible on x-ray angiography.

Three surgical cases are shown in which fluorescein angiography demonstrated the collateral flow and microcirculatory perfusion in varying degrees. In one patient with middle cerebral occlusion near the takeoff from the carotid artery, almost the entire cortical circulation and the middle cerebral distribution displayed perfusion flow. In a 2nd patient, the filling of the cortical microcirculation with a similar occlusion was less complete. In the 3rd patient in which a scalp artery was anastomosed to a branch of the middle artery, the extent of the cortical microcirculatory flow was clearly displayed by fluorescein angiography. The implication of these findings for microvascular surgery will be briefly discussed.

Discussion

10:40 a.m.

34. Experience with Superficial Temporal Artery Bypass Surgery

T. M. Sundt, F. W. Sharbrough, and O. W. Houser
Rochester, Minnesota

Experiences with 35 cases of extracranial to intracranial bypass procedures will be presented. These surgical procedures have been performed primarily for chronically occluded internal carotid arteries that have continued to be symptomatic after the occlusion. Other indications have included: middle cerebral artery stenosis, bypass surgery for giant aneurysms prior to internal carotid ligation, and fibromuscular hyperplasia of the internal carotid artery. Complications have included intracerebral hemorrhage distal from the site of anastomosis, graft occlusion, and emboli from the site of anastomosis. Two cases, one of hemorrhage, and one of emboli, will be presented in detail. Current estimations relative to the amount of blood flow obtained through these grafts will be presented.

Discussion

11:00 a.m.

35. Dissecting Aneurysms of the Carotid Artery

John M. Tew
Cincinnati, Ohio

Trauma is a well recognized cause of dissecting aneurysms of the internal carotid artery. Recent experience indicates that non traumatic dissecting lesions are a frequent cause of cerebral infarction particularly in young individuals. A clinical syndrome of unilateral headache, facial and neck pain associated with high carotid bruit, and Horners syndrome, transient or progressive cerebral ischemia has been documented. An etiological classification based on angiographic and pathologic findings is posed.

Classification

Dissectings Aneurysms

1) Traumatic
   a) Direct
      1. penetrating
      2. nonpenetrating
   b) Indirect

2) Spontaneous

   a) Cystic medical Necrons
   b) Fibromuscular Dysplasia
   c) Atherosclerosis

Treatment may be medical or surgical.

Medical

1) Anticoagulation
2) Reduction of blood pressure

Surgical

1) Arterial ligation
   a) Partial
   b) Complete
2) Thrombectomy and suture of intima
3) Excision and grafting (end to end)
4) Bypass grafting

The literature is reviewed and nine additional cases of spontaneous or nontraumatic dissecting aneurysms of the carotid artery will be reported.

Discussion

11:20 a.m.

36. Detection of Intracranial Aneurysms by a Non-Invasive Acoustic Method

Charles P. Olinger and Jack F. Wasserman
Cincinnati, Ohio

In our search for a non-invasive acoustic method for the detection of intracranial aneurysms, we found that cerebral aneurysms normally generate an aneurysm-characteristic sound wave and that these sound waves are detectable not only at the surface of the aneurysm but externally of the head via transmission through the eye and the closed eyelid. By monitoring sound waves emanating from the eye, and by analyzing the waves to distinguish the aneurysm-characteristic sound from other sounds, the probable existence of an aneurysm can be diagnosed with a specially designed electronic stethoscope and an advanced acoustic analyzer computer system.

The method is non-invasive, does not require hospitalization, and is repeatedly used to diagnose probable aneurysm presence and the onset and continuation of spasm. Information from repeated samplings aids in following and diagnosing patient status pre-bleed, during bleed, post-bleed and pre-and post-operative.

The system has identified angiographically proven intracranial aneurysms. Computer analysis of recordings taken over patients' eyes showed distinct acoustic evidence of the presence of aneurysms before surgery and their absence after surgery.

The early acoustic detection of many intracranial aneurysms before rupture should now be possible.

Discussion

11:40 a.m.

37. Plastic Coating of Aneurysms — A Follow-Up Study

Robert S. Knighton and J. Speed Rogers
Detroit, Michigan

We are presenting thirty patients whose intracranial aneurysms were treated by plastic coating since 1955. Twenty-six of these patients were treated by EDH (Biobond). Twenty-one of these patients are still living and all but one is "well". Of the nine deceased patients seven

died from aneurysm related causes and two from incidental causes. There were five "early rebleeders" and one patient who probably rebled after seven and a half years. Eighteen of these patients have been followed for a median time of eight years. We have been able to study only one late case, a patient dying of carcinoma of the lung eleven and a half years after his aneurysm was coated. At autopsy we were surprised to find that almost all of the coating material had disappeared and that none was adjacent to the unruptured aneurysm lying on the optic chiasm. We raise the question as to the long term effects of this particular plastic substance as a coating material.

Discussion

12:00 a.m.

38. Embolism from Intracranial Aneurysms — Report of Two Cases

J. L. Antunes and J. W. Correll
New York, New York

Increasing attention has been paid to the importance of cerebral ischemia and infarction following the rupture of intracranial aneurysms. Cerebral vasospasm has been implicated as the major pathophysiologic mechanism responsible for these changes. However it will be shown that ischemic changes also result from thromboembolic occlusion of arteries distal to the aneurysm. In the present report, an aneurysm, in each of 2 patients, arising from the internal carotid artery at the origin of the posterior communicating was the source of emboli resulting in infarction. This led to death with documentation at autopsy in one patient, and in the other clipping of the aneurysm preventing further embolization was followed by recovery. It is concluded that thromboembolic phenomena should be considered as a possible important cause of the morbidity associated with intracranial aneurysm and may be quite frequent.

Discussion

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1975

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302 Iroquois Building
3600 Forbes Street
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HENRY G. SCHWARTZ — 1942
Barnes Hospital Plaza
Division of Neurological Surgery
St. Louis, Missouri 63110

C. HUNTER SHELDEN — 1941
734 Fairmount Avenue
Pasadena, California 91105

HOMER S. SWANSON 1949
1938 Peachtree Road, N. W..
Atlanta, Georgia 30309

A. EARL WALKER 1938
Johns Hopkins Hospital
Division of Neurological Surgery
601 N. Broadway
Baltimore, Maryland 21205

EXUM WALKER 1938
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Apartado 5254
Lima, Peru

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Mexico, D. F.

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Dipartimento di Neurochirurgia
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Neurochir. Abt. d. Landersnervenklink
Salzberg, 5020, Austria

JOHN GILLINGHAM 1962
Boraston House, Ravelson
Edinburg 4, Scotland

JOHN HANKINSON 1973
Department of Neurosurgery
Newcastle General Hospital
Newcastle-upon-Tyne 4
England

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Australia 4000

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Department of Neurological Surgery
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Tagensvej 18, 2200
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BETTY 1950
1941 Georgia Ave.
Winston-Salem, N. C. 37104

JAMES R. ATKINSON
Barrow Neurological Institute
302 W. Thomas Road
Phoenix, Ariz. 85013

LONA 1970
5806 East Lewis Ave.
Scottsdale, Ariz. 85257

H. THOMAS BALLANTINE, JR.
Massachusetts General Hosp.
Boston, Massachusetts 02114

ELIZABETH 1951
30 Embankment Road
Boston, Mass. 02114

GILES BERTRAND
Montreal Neurological Inst.
3801 University St.
Montreal, Quebec, Canada

LOUISE 1967
385 Lethbridge
Montreal 16, Quebec, Canada

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University of Calif. Hosp.
3rd Avenue & Parnassus
San Francisco, Calif. 94122

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MARTHA 1968
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JOLENE 1974
2 Otter Lane
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12621 Brookpark Road
Oakland, California 94619

MARIAN 1970
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Oakland, Calif 94619

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Yale Univ. School of Med.
333 Cedar Street
New Haven, Conn. 06510

GWEN 1963
403 St. Ronan Street
New Haven, Conn. 06511

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Ochsner Clinic
New Orleans, Louisiana 70118

JAMES W. CORRELL
Neurological Institute
710 West 168th St.
New York, New York 10032

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Bowman-Gray School of Med.
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20 S. Dudley Street
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2020 East 93rd Street
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111 Waterloo Street, Suite 211
London, Ontario, Canada

DEAN H. ECHOLS
Ochsner Clinic
1514 Jefferson Highway
New Orleans, Louisiana 70121

GEORGE EHNI
1531 Hermann Professional Bldg.
6410 Fannin Street
Houston, Texas 77025

WILLIAM H. FEINDEL
Montreal Neurological Institute
3801 University Street
Montreal, Quebec, Canada

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800 N. E. 13th Street
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Chairman, Div. of Neurosurgery
Univ. of Calif. School of Med.
Irvine, California 92664

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Algonquin Trail
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MARILYN 1967
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Winston-Salem, NC 27106

PHYLLIS 1962
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RUTH 1958
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New Orleans, Louisiana 70130

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16 Sunset
Houston, Texas 77025

FAITH 1959
A-31 Cote des Neiges
Montreal, H3H1W2

1957
107 Lake Aluma Drive
Oklahoma City, Okla. 73121

CATHERINE 1960
2480 Monaco Drive
Laguna Beach, Calif. 92651

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LYLE A. FRENCH University of Minn. Med. Center Minneapolis, Minn. 55455	GENE 85 Otis Lane St. Paul, Minn. 55104	1954
JOHN T. GARNER 744 Fairmount Avenue Pasadena, Calif. 91105	BARBARA 3075 Monterey Rd. San Marino, Calif. 91108	1971
HENRY GARRETSON Dept. of Neurosurgery University of Louisville Louisville, Kentucky	MARIANNA 517 Tiffany Lane Louisville, Kentucky 40207	1973
SIDNEY GOLDRING Barnes Hospital Plaza Division of Neurosurgery St. Louis, Misouri 63110	LOIS 11430 Conway Road St. Louis, Missouri 63131	1964
PHILIP D. GORDY 1025 Walnut Street Philadelphia, Pennsylvania 19107	420 N. Rose Lane Haverford, Pennsylvania 19041	1968
JOHN R. GREEN Barrow Neurological Institute 302 West Thomas Street Phoenix, Arizona 85013	GEORGIA 2524 E. Crittendon Ln., Sutton Place Phoenix, Ariz. 85016	1953
JOHN W. HANBERY Division of Neurosurgery Stanford Medical Center Palo Alto, California 94304	SHIRLEY 70 Mercedes Lane Atherton, Calif. 94025	1959
MAJ. GEN. GEORGE S. HAYES, MC USA Principal Deputy Office of the Assistant Sec. of Defense (Health & Environment) Washington, D. C. 20301	CATHERINE 303 Skyhill Road Alexandria, Va. 22314	1962
E. BRUCE HENDRICK Hospital for Sick Children 555 University Avenue Toronto, Ontario, Canada	GLORIA 63 Leggett Avenue Weston, Ontario, Canada	1968
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RAEBURN C. LLEWELLYN Tulane University 1430 Tulane Avenue New Orleans, La. 70012	CARMEN 32 Versailles Blvd. New Orleans, La. 70124	1963
WILLIAM M. LOUGHEED Medical Arts Building, Suite 430 170 St. George St. Toronto 5, Ontario, Canada	GRACE ELEANOR 67 Ridge Drive Toronto, Ontario Canada	1962
HERBERT LOURIE 713 East Genesee Street Syracuse, New York 13210	BETTY 101 Thomas Road DeWitt, New York 13214	1965
JOHN J. LOWREY Straub Clinic 888 S. King Street Honolulu, Hawaii 96813	CATHERINE (KAY) 2299-B Round Top Dr. Honolulu, Hawaii 96822	1965

ERNEST W. MACK 505 S. Arlington Avenue, Suite 212 Reno, Nevada 89502	ROBERTA 235 Juniper Hill Road Reno, Nevada 89502	1956
M. STEPHEN MAHALEY, JR. Duke University Med. Cent. Durham, North Carolina 27706	JANET 3940 Nottaway Road Durham, North Carolina 27707	1972
LEONARD MALIS 1176 Fifth Avenue New York, New York 10029	RUTH 219-44 Peck Avenue Hollis Hills, N.Y. 11427	1973
FRANK MAYFIELD 506 Oak Street Cincinnati, Ohio 45219	QUEENEE 1220 Roodwood Drive Cincinnati, Ohio 45208	Founder
ROBERT L. McLAURIN Division of Neurosurgery Cincinnati General Hosp. Cincinnati, Ohio 45229	KATHLEEN 2461 Grandin Road Cincinnati, Ohio 45208	1955
WILLIAM F. MEACHAM Vanderbilt University Hosp. Division of Neurosurgery Nashville, Tenn. 37203	ALICE 3513 Woodmont Boulevard Nashville, Tenn. 37215	1952
JOHN F. MULLAN Univ. of Chicago Clinics Department of Neurosurgery 950 E. 59th St. Chicago, Ill. 60637	VIVIAN 6911 S. Bennett Ave. Chicago, Ill. 60649	1963
BLAINE S. NASHOLD, JR. Duke University Medical Cent. Durham, North Carolina 27706	IRENE 410 E. Forest Hills Blvd. Durham, North Carolina	1967
FRANK E. NULSEN Div. of Neurosurgery University Hospital 2065 Adelbert Road Cleveland, Ohio 44106	GINNEY 31200 Fox Hollow Dr. Pepper Pike, Ohio 44124	1956
GUY L. ODOM Duke University Med. Center Durham, North Carolina 27706	MATALAINE 2812 Chelsea Circle Durham, North Carolina	1946
ROBERT G. OJEMANN Massachusetts General Hospital Division of Neurological Surg. Boston, Mass. 02114	JEAN 85 Nobscot Road Weston, Mass. 02193	1968
RUSSEL H. PATTERSON, JR. 525 East 68th Street New York, New York 10021	JULIET 535 East 86th St. New York, N.Y. 10028	1971

PHANOR L. PEROT, JR. Medical Univ. of South Carolina 80 Barre St. Charleston, S. Carolina 29401	ELIZABETH 704 Willowlake Road Charleston, S. Carolina 29407	1970
BYRON C. PEVEHOUSE 2001 Union St. San Francisco, Calif. 94123	MAXINE 135 Mountain Spring Ave. San Francisco, Calif. 94114	1964
ROBERT W. PORTER 5901 E. 7th Street Long Beach, California 90804	AUBREY DEAN 5400 The Toledo Long Beach, California 90803	1962
JOHN RAAF 833 S. W. 11th Avenue Portland, Oregon 97205	LORENE 390 S. W. Edgecliff Road Portland, Oregon 97219	Founder
AIDEN A. RANEY 2010 Wilshire Blvd., Suite 203 Los Angeles, California 90057	MARY 125 N. Las Palmas Los Angeles, California 90004	1946
JOSEPH RANSOHOFF II New York Univ. Medical Center 500 First Avenue New York, New York 10016	RITA 140 Riverside Drive New York, New York	1965
THEODORE B. RASMUSSEN Montreal Neurological Institute 3801 University Street Montreal 2, Quebec, Canada	CATHERINE 29 Surrey Drive Montreal 16, Quebec, Canada	1947
DAVID H. REYNOLDS 1150 N. W. 14th Street, Suite 209 Miami, Florida 33136	MARJORIE 1701 Espanola Drive Miami, Florida	1964
HUGO RIZZOLI 2150 Penn Avenue, NW Washington, D. C. 20037	HELEN 6100 Kennedy Drive Kenwood, Chevy Chase, Maryland 20015	1973
JAMES T. ROBERTSON 20 S. Dudley Memphis, Tennessee 38103	VALERIA 628 N. Trezevant Street Memphis, Tennessee 38112	1971
R. C. L. ROBERTSON Shamrock Professional Building 2210 Maroneal Boulevard, Suite 404 Houston, Texas 77025	MARJORIE 5472 Lynbrook Drive Houston, Texas	1946
RICHARD C. SCHNEIDER C5135, Cut-Pt. Building University Hospital Ann Arbor, Michigan 48104	MADELEINE 2110 Hill Street Ann Arbor, Michigan 48104	1970

WILLIAM B. SCOVILLE 85 Jefferson Street Hartford, Connecticut 06106	HELENE 27 High Street Farmington, Connecticut	1944
BENNETT M. STEIN Department of Neurosurgery 171 Harrison Avenue Boston, Massachusetts 02111	DOREEN 16 Tamarack Road Weston, Massachusetts 02193	1970
JIM L. STORY 7703 Floyd Curl Drive San Antonio, Texas 78229	JOANNE 3211 Stonehaven Road San Antonio, Texas 78230	1972
THORALF M. SUNDT, JR. 200 First Street, S. W. Rochester, Minnesota 55901	LOIS 1406 Weatherhill Court Rochester, Minnesota 55901	1971
ANTHONY F. SUSEN 3600 Forbes Avenue Pittsburg, Pennsylvania 15213	PHYLLIS 3955 Bigelow Boulevard Pittsburgh, Pennsylvania	1965
WILLIAM H. SWEET Massachusetts General Hospital Division of Neurological Surgery Boston, Massachusetts 02114	MARY 35 Chestnut Place Brookline, Massachusetts	1950
RONALD R. TASKER Toronto General Hospital Room 121, U. W. Toronto, Ontario, Canada	MARY 12 Cluny Drive Toronto 5, Ontario, Canada	1971
JOHN TEW, JR. 506 Oak Street Cincinnati, Ohio 45219	SUSAN 2145 East Hill Avenue Cincinnati, Ohio 45208	1973
GEORGE T. TINDALL Emory Univ. School of Med. Division of Neurosurgery Atlanta, Georgia 30322	SUZIE 2938 Dominique Drive Galveston, Texas	1968
JOHN TYTUS Mason Clinic Seattle, Washington 98101	VIRGINIA (GINA) 1000 N. W. Northwood Road Seattle, Washington 98177	1967
ALFRED UIHLEIN 200 First Street, S. W. Rochester, Minnesota 55901	IONE Box 1127 Naples, Florida	1950
ARTHUR A. WARD. JR. Department of Neurological Surgery University of Washington Hospital Seattle, Washington 98105	JANET 3922 Belvoir Place, N. E. Seattle, Washington 98105	1953

W. KEASLEY WELCH Children's Hospital Medical Center 300 Longwood Avenue Boston, Massachusetts 02115	ELIZABETH 25 Gould Road Waban, Massachusetts	1957
BENJAMIN B. WHITCOMB 85 Jefferson Street Hartford, Connecticut 06106	MARGARET 38 High Farms Road West Hartford, Connecticut	1947
LOWELL E. WHITE, JR. Professor & Chairman Division of Neurosciences Mobile, Alabama 36688	MARGIE 912 Regency Drive West Mobile, Alabama 36609	1971
ROBERT WILKENS Scott &White Clinic Temple, Texas 76501	GLORIA 3409 Aspen Trail Temple, Texas 76501	1973

Deceased Members

	Date	Elected
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 Iowa City, Iowa	(Senior) 6-23-66	1942
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. RUPERT B. RANEY Los Angeles, California	(Active) 11-28-59	1939
DR. DAVID L. REEVES Santa Barbara, California	(Senior 8-14-70	1939
DR. SAMUEL R. SNODGRASS Nashville, Indiana	(Senior) 8-8-75	1939
DR. C. WILLIAM STEWART Montreal, Quebec, Canada	(Corresponding)	1948
DR. GLENN SPURLING LaJolla, California	(Honorary) 2-7-68	1942
DR. HENDRIK SVIEN Rochester, Minnesota	(Active) 6-29-72	1957

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