



ANNUAL MEETING 1976



The Mills Hyatt House Charleston, South Carolina November 10 - 13, 1976

THE AMERICAN ACADEMY OF **NEUROLOGICAL SURGERY**

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DRAWINGS
BY GILL GUERRY



PROGRAM 1976

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Wednesday, November 10
4:00 - 7:30 p.m
Pre-Assembly Area, Mills Hyatt House
6:30 - 8:30 p.m Opening Cocktail Party
Nathaniel Russell House
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Thursday, November 11
7:30 a.m Breakfast and Business Meeting (Members)
Planter's Suite
7:30 a.m Breakfast for Academy Guests (Men)
Middleton Room
8:00 a.m 3:00 p.m Registration
Pre-Assembly Area, Mills Hyatt House
8:20 a.m 10:30 a.m
Lynch/Heyward
10:30 a.m 10:45 a.m
Pre-Assembly Area, Mills Hyatt House
10:50 a.m 12:00 noon
Lynch/Heyward
12:00 noon - 1:00 p.m Luncheon (Members and Guests)
Planter's Suite
1:00 p.m 3:00 p.m
Lynch/Heyward
4:00 p.m Buses leave for Plantation Evening
(Queen Street entrance of Mills Hyatt House)
Middleton Gardens - Dinner and Entertainment
Friday, November 12
7:30 a.m Breakfast and Business Meeting (Members)
Rutledge Room
7:30 a.m Breakfast for Academy Guests
Buffet - Barbados Dining Room
8:00 a.m 3:00 p.m
Pre-Assembly Area, Mills Hyatt House
8:30 a.m 10:25 a.m
Lynch/Heyward
10:25 a.m 10:40 a.m
Pre-Assembly Area, Mills Hyatt House
10:40 a.m 12:30 p.m
Lynch/Heyward
12:00 noon - 12:30 p.m

1:00 p.m Buses leave for Seabrook Island
(Queen Street entrance, Mills Hyatt House)
Tennis and Golf - Lunch available
Return at 5:30 p.m.
7:00 p.m 8:00 p.m
Poolside, Mills Hyatt House
8:15 p.m until
Hibernian Hall (Upstuirs)
Dinner and Entertainment
Saturday, November 13
8:00 a.m Breakfast and Business Meeting (Members)
Rutledge Room
8:00 a.m Breakfast for Academy Guests
Buffet - Barbados Dining Room
9:00 a.m 10:30 a.m Scientific Session
Lynch/Heyward
10:30 a.m 10:45 a.m
10:45 a.m 1:00 p.m
Lynch/Heyward



LADIES PROGRAM 1976

Wednesday, November 10	
4:00 p.m 7:30 p.m	
Pre-Assembly Area, Mills Hyatt House	
6:30 p.m 8:30 p.m Opening Cocktail Party	
Nathaniel Russell House	
Thursday, November 11	
9:00 a.m Buses leave 9:00 and 9:15 for	
coffee and doughnuts at Edmondston-Alston	
House followed by Walking Tour of	
Charleston and Lunch	
4:00 p.m Buses leave for Plantation Evening	
(Queen Street Entrance of Mills Hyatt House)	
Middleton Gardens - Dinner and Entertainment	
N 1 10	
Friday, November 12	
9:00 a.m 10:30 a.m Ladies Hospitality Middleton Room	
10:00 a.m Ansonborough Walking Tour	
On your own for lunch	1
1:00 p.m	
(Queen Street entrance, Mills Hyatt House)	
Tennis and Golf - Lunch available	
Return at 5:30 p.m	
2:30 p.m Gray Line Harbor Tour	•
7:00 p.m 8:00 p.m	š
Poolside, Mills Hyatt House	
8:15 p.m until	,
Hibernian Hall (Upstairs	
Dinner and Entertainmen	t
Outsides Name 12	
Saturday, November 13 9:00 a.m. • 10:30 a.m	J
9:00 a.m. • 10:30 a.m	<i>!</i>
noon normalist	

Scientific Program

THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY Charleston, South Carolina November 10 - 13, 1976 MODERATOR, William H. Feindel

Thursday, November 11

8:20 a.m.

Welcoming Remarks and Announcements

8:30 a.m.

1. Neurological Deterioration Following Subarachnoid Hemorrhage: Prevention, Diagnosis and Treatment

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

Progressive central nervous system deficit is common in patients with aneurysms and subarachnoid hemorrhage and occurs with distressing frequency both pre and postoperatively. When deterioration occurs a diagnosis of "spasm" is often made and a fatalistic attitude adopted since no specific therapeutic measures are available. However, the cause of deterioration in patients with ruptured aneurysms is most often multifactorial. While vasospasm is usually a contributing if not the dominant factor, other more readily treatable factors are usually operant. Reversal of these factors may result in dramatic improvement, even in the presence of severe spasm.

Factors responsible for progressive neurological dysfunction in patients with subarachnoid henorrhage include, among others: cerebral arterial vasospasm, arterial thromboembolism, iatrogentic partial or complete arterial occlusion, venous thrombosis, aneurysmal swelling or shrinkage, intracranial hematoma, hydrocephalus, brain edema, ischemic encephalopathy secondary to intraoperative hypotension, fluid and electrolyte disorders, steriod psychosis, respiratory, renal or hepatic dysfunction, meningitis, epileptic seizures, and "floppy brain" syndrome.

The nature of most of these factors is such that they combine in a compound manner accelerating both the rate of development and the severity of the neurological deficit. Often the deficit is reversible, if

definitive treatment is begun before permanent changes in the brain have occurred. Understanding of the underlying pathophysiology and treatment of each individual factor and meticulous monitoring of appropriate parameters is the foundation for effective prevention and early detection.

This report relates the experiences with neurological deterioration, both pre and postoperatively, in a series of 60 patients with aneurysms and subarachnoid hemorrhage. The individual factors responsible for the progressive deficit and the underlying pathophysiology and management of each will be discussed.

(Discussion)

8:50 a.m.

2. Indication and Timing of Surgery for Ruptured Intracranial Aneursym with Vasospasm

Keiji Sano, and Isamu Saito Tokyo, Japan

It is generally accepted that cases of ruptured aneurysms with vasospasm are poor candidates for surgical treatment regardless of their condition. In the author's experience, however, the subsiding vasospasm (subsiding usually about one week after its onset) has never been aggravated, whereas the progressing vasospasm will be aggravated by surgical intervention. Moreover, rebleeding may quite often occur when the vasospasm becomes relieved. In order to prevent rebleeding of ruptured aneurysms which have begun to show signs of remission, the best timing of surgery is the second week after the onset of vasospasm (not of rupture). The rationale will be discussed in detail.

(Discussion)

9:10 a.m.

3. Clinical Usefulness of Haptoglobin in the Treatment of Vasospasm following SAH

Shozo Ishii, Hiroo Chigasaki, Toshifusa Nonake and Makoto

Miyaoka Tokyo, Japan

In a previous study, a biochemical isolation of the vasoactive substances from fresh or aged serum, whole blood and blood-CSF mixture was performed. Through in vitro and in vivo experiments, it has been confirmed that oxyhemoglobin in either aged blood or blood-CSF mixture was most essential causative factor of prolonged vasospasm. It was also found that haptoglobin, a normal constituent of serum and known to bind with hemoglobin easily, demonstrated spasmolytic action upon the vasospasm. In this report, the clinical results of haptoglobin upon the vasospasm will be discussed. Haptoglobin was applied topically at the time cases, who all had the vasospasm verified by 13 of surgery angiography done immediately before operation. Period between last SAH and surgery ranged 8-23 days. Vasospasm was diffuse in 6 cases and segmental in 7. After a clip was applied at the neck of the aneurysm, haptoglobin was introduced and its immediate effects were checked with the operating microscope. Only in 3 cases marked dilation of the contracted arteries was seen. On the other hand, angiography done 24-48 hours following surgery demonstrated improvement of vasospasm in 8 cases out of 13. Correlation between the changes in vasospasm and neurological conditions will be discussed. Experience of prophylactic use of haptoglobin in the early stage surgery will also be discussed.

(Discussion)

9:30 a.m.

4. Thrombosed Aneurysms of the Cerebral Arteries

Jim L. Story, John P. Wissinger, Willis E. Brown and Edward L. Seljeskog San Antonio, Texas

The authors have encountered three large totally and spontaneously thrombosed aneurysms of the intracranial vessels. None filled angiographically, but all demonstrated mass effect on various contrast studies.

Two patients presented as seizure disorders. Recent subarachnoid hemorrhage was suspected in one and remote subarachnoid and

intracranial hemorrhage was verified in another. Only one patient presented with clinical mass effect which was characterized by visual loss secondary to optic nerve and chiasmal compression.

This paper will deal with a discussion of our patients and a review of the literature pursuant to the clinical presentation, preoperative evaluation and management of unverified spontaneously thrombosed aneurysms of the intracranial circulation.

(Discussion)

9:50 a.m.

5. Induced Thrombosis in the Treatment of Twenty Carotid Cavernous Fistulae

Sean Mullan Chicago, Illinois

In the course of this experience we have investigated a variety of thrombogenic materials which include electrical needles, stereotactic copper needles, thrombogenic wire, gelfoam and balloon. There has been no mortality and no morbidity. The object in all instances has been to obliterate the fistula while preserving an intact carotid. All of these technics are effective. The problem is to find the simplest technic, or combination of technics for each individual fistula. At the moment the anterior approach (extradural insertion of gelfoam through the inferior orbital fissure) and the lateral approach (intradural insertion of wire or needles) have been fairly well worked out. The posterior approach is still difficult but one solution may be the trans jugular, trans inferior petrosal sinus, intra cavernous sinus occlusive balloon.

(Discussion)

10:10 a.m.

6. Increased Secretion of Antidiuretic Hormone in Patients with Intracranial Aneurysms

Paul B. Nelson, Said M. Seif, Alan G. Robinson and Robert H.

Wilkins Pittsburgh, Pennsylvania

Abnormalities in the secretion of antidiuretic hormone (ADH) are known to occur in intracranial disorders. We used a highly specific radioimmunoassay for plasma ADH (sensitive to 0.2 بالس ml plasma) to study normal individuals and neurosurgical patients. The normal plasma ADH was 0.46 uU/ml ± 0.09 SEM. Thirteen consecutive patients with intracranial aneurysms were studied serially with a total of 59 plasma samples. Twelve of thirteen patients underwent surgery. Plasma ADH in patients with intracranialaneurysms was 2.80 µU/ml, significantly higher than normal (p < 0.01). The aneurysm patients were divided into one group of ten patients with ruptured aneurysms and a second group of three patients without subarachnoid hemorrhage. ADH levels were significantly higher in the patients with hemorrhage, 3.11 μ U/ml as compared with unruptured aneurysms, 1.32 _uU/ml (p< 0.05). Hyponatremia with inappropriate concentration of the urine occurred at some time in eight of the ten patients with ruptured aneurysms. Elevated ADH has therapeutic implication in fluid balance because the syndrome of inappropriate ADH may lead to cytoxic cerebral edema. The association of ruptured aneurysms with higher levels of ADH suggests that hypothalamic damage may be an important cause of the ADH release.

(Discussion)

10:30 a.m.

Coffee Break

10:50 a.m.

7. Experimental Strokes and Reperfusion: The Golden Period.

R.A.R. Fraser, S. Yoshida, R.H. Patterson, Jr. New York, New York

Attempts at re-establishing CBF in patients with acute cerebral ischemia are becoming increasingly popular despite poor results reported

in the past. Fifty-four rhesus monkeys were used in a study of reversible primate cerebral ischemia. Each animal underwent transorbital microsurgical exposure of the carotid bifurcation. An aneurysm clip was placed upon the (proximal) middle cerebral artery (MCA) for varying periods - 30 minutes to six hours in 38 animals. The remainder included four controls and 12 subjected to a permanent clip. One half of the entire group were treated with a vasopressor (metaraminor) raising the systemic arterial pressure (SAP) an average of 25 torr for the duration of clip application. Animals with permanent clips were kept hypertensive for six hours. Three weeks after the initial experiment each animal was sacrificed (via perfusion) with a formalin-carbon black suspension at arterial pressure. The brains were removed, photographed then serially sectioned. H & E sections were performed. Comparisons of the clinical course, perfusion. Restoration of flow after this period resulted in a significant animals tolerated up to 3 hours of MCA occlusion with minimal to moderate clinical defects and minor perfusion defects and modest or no infarctions. These defects were significantly less in the hypertensive group. Six hours MCA occlusion produced a major defect in clinical grade and prefusion. Restoration of flow after this period resulted in a significant number of macroscopic hemorrhagic infarctions. Hypertension did not improve this group.

These data suggest that restoration of cerebral blood flow is completely ineffective after six hours of cerebral anemia but may be useful prior to this period. Hypertension may provide a useful clinical adjunct if used early after the onset of cerebral ischemia. The "no-reflow" phenomenon is not synonymous with infarction but "no-reflow" does occur after long periods of large artery occlusion.

(Discussion)

11:10 a.m.

8. Intravascular Navigation - Applications in Neurosurgery

John M. Tew, Jr., M.D. Cincinnati, Ohio

Since the Fogarty catheter was developed at the Good Samaritan Hospital in Cincinnati in 1961, the surgical treatment of peripheral arterial emboli has been greatly changed. The inflatable balloon for extraction of emboli gained ready acceptance and has replaced open arteriotomy.

Applications of intravascular balloon techniques in neurosurgery have been largely limited to removal of carotid thrombi from the acutely occluded artery. More recently heightened interest in balloon catheters has led to their use in the control of a variety of other arterial and venous disorders. Clinical application has taken several directions such as control of traumatic carotid laceration, carotid cavernous fistulae, vertebral artery fistulae, occlusion of cavernous carotid aneurysms and dilatation of stenotic areas in the arteries involved by fibromuscular dysplasic arteries.

Our experimental studies have taken two directions: (1) the development of a flow directed catheter for intracranial navigation, and (2) the application of detachable balloons for obliteration of arterial lesions. The results of these studies and future applications will be discussed.

(Discussion)

11:30 a.m.

Tribute to Egas Moniz - 50th Anniversary of Cerebral Arteriography

J. Lobo Antunes by invitation

12:00 noon

Lunch

MODERATOR - Frank E. Nulsen

1:00 p.m.

9. Screening of Human Brain Tumors for SV40-Related T Antigen

Wolff M, Kirsch and Kazuo Tabuchi Denver, Colorado

Since 1971 several new papovaviruses have been found and isolated from humans, especially from the brains of patients suffering from progressive multifocal leucoencephalopathy (PML). These human

papovaviruses (such as JC, BK, SV40-PML viruses), though antigenically dissimilar to papilloma or polyoma viruses bear certain physicochemical similarities to simian virus 40(SV40). The oncogenic potential in hamsters for these human papovaviruses (JC, BK) has been reported, raising a suspicion that some human brain tumors may have a viral etiology. As virus-induced animal tumors display virus-specific common antigen(s) irrespective of tumor histology or site, our attention has been directed to the immunohistochemical screening of human brain tumors for the presence or absence of SV40-related T(tumor) antigen, because this protein is known to share common antigenic activity with T antigen(s) induced by human papovaviruses.

At the time of writing of this abstract, 39 human brain tumors have been screened for SV40-related T antigen in vivo by immunoperoxidase technique using hamster anti-SV40 T antibody as well as normal hamster serum as a control. Two (one ependymoma, one plexus papolloma) out of 39 cases examined displayed marked positive staining in the nuclei of tumor cells with anti-SV40 Tserum, but neither with normal hamster serum nor with anti-SV40 V(nucleo capsid antigen) antibody. An independent complement fixation assay of the nuclear extracts from one of the two positive tumors also confirmed the presence of the antigen which specifically reacted with hamster anti-SV40 T serum.

There have been few systematic searches for papovavirus-related antigens in human neoplasms, however, in 1975 SV40-related T and U antigens have been observed in three cases of human meningioma cells by an immunoflourescence test. Since human cells are semipermissive for papovaviruses, these and our findings strongly suggest a viral etiology for some human brain tumors.

(Discussion)

1:20 p.m.

10. Observations on the Synergistic Effect of Heat and BCNU: Possible Application as an Adjuvant Therapy

Robert G. Selker Pittsburgh, Pennsylvania

The effect of elevated temperatures on tumor tissue is currently being investigated as a form of adjuvant tumor therapy. A combination of

warm tumor tissue (40 C) and cold normal tissue (28 C) forms a so called "thermal or metabolic gradient" which serves to effect differential uptake of chemotherapeutic agents.

In order to determine that level of drug administration which is destructive to warm tissue but harmless to normal cooled tissue, 90 non-tumor bearing Sprague-Dawley rats were grouped into 3 BCNU dose levels at 3 different total body temperatures (7 mg/kg IP, 14 mg/kg IP, 31 mg/kg IP at 28°, 37° and 40°). Results were judged by death rates, CBCs and liver toxicity.

Results indicate heat to profoundly enhance the effect of BCNU and its toxic side effects. Hypothermia, on the other hand, is capable of conferring protection from the ravages of toxicity on body organ systems and can be likened to the now well known "citrovorum rescue."

Because of the rapid breakdown of BCNU into several thought-to-be toxic products, and the uncertainty of their activity, a second set of 10 control and 20 experimental rats were divided into two groups, given 14 mg/kg IP BCNU and heated at 5 days (Group I) and 10 days (Group II) after injection. Results of these studies paralleled the experience with the group heated immediately, indicating retained toxic effect of the breakdown products of BCNU up to 10 days after injection.

The following can be concluded from these experiments:

- 1. Heat does indeed have a synergistic effect when used with a chemotherapeutic agent such as BCNU.
- Hypothermia will protect normal body systems from the effect of BCNU and permit the infusion of greater dose levels of chemotherapy.
- The results substantiate the concept of the creation of a thermal gradient in the treatment of solid tumor systems.
- 4. Those who are using BCNU clinically should be alerted to the fact that a febrile episode from whatever cause during the first 10 days of BCNU infusion will create untoward toxicity. This finding is of immediate clinical importance.

(Discussion)

1:40 p.m.

11. Method of Visualizing and Removing Minute (5mm) Brain Tumors.

C. Hunter Shelden Pasadena, California

A method has been developed to allow visualization and afford a possible means of removing very small intracerebral brain tumors.

We are rapidly approaching a surgical dilemma. We can now localize small asymptomatic intracerebral tumors but have no safe method of removal by any of the conventional methods.

Stereotactic placement of a specially designed end-opening dissector tube allows an accurate approach to the lesion. Removal is accomplished by means of a <u>first-of-its-kind</u> binocular stereoscopic endoscope. This allows tumor removal under direct 3-D vision.

Tumors of 5 mm and less can barely be detected even under most ideal conditions at the present time but should be a routine finding within a year. One can be certain that neurosurgeons soon shall have seen the last of large tumors.

(Discussion)

2:00 p.m.

12. Management and Results in 114 Cases of Optic Nerve Glioma

Edgar M. Housepian New York, New York

One hundred fourteen cases of optic nerve glioma seen and treated at the New York Neurological Institute over a forty—year period (1935-1975) have been reviewed. Some of the controversial aspects of the natural history, response to radiation therapy and rationale for surgery will be discussed in light of this experience.

(Discussion)

2:20 p.m.

13. Experience with Intramedullary Spinal Cord Tumors.

E. Garrido and Bennett M. Stein Boston, Massachusetts

Nine cases of removal of intramedullary spinal cord tumors are presented. The majority of these tumors occurred in adults in the cervical region. Total removal was carried out in 8 cases, and a 90 percent removal in one other case. The ependymomas and astrocytomas predominated in adults while a lesser number of teratomatous type tumors were encountered in children in the lumbo-sacral regions of the spinal cord.

All patients were improved or those with severe deficit remained static following operation.

Many of the cases had been previously explored and subjected to radiation prior to the removal of the spinal cord tumor. Experiences derived from this facet of the disease and surgical experience in general will be reviewed in this paper. A movie and 35 mm, slides are part of the presentation.

(Discussion)

2:40 p.m.

14. Computerized Monitoring in a Neurosurgical Intensive Care Unit.

G.W. Kindt and R.C. Schneider Ann Arbor, Michigan

Standard patient data systems for monitoring intensive care patients have been used in cardiac units for a few years. The goal of these systems is to obtain and record data directly from the patient as well as data entered into the system by the nurse or desk clerk. Desired calculations can then be made and the data can be displayed using the computer. Hard copies of the data in the desired form can then be obtained for the patient's record.

This report concerns our experience using such a standardized

patient data system in an eight bed neurosurgical intensive care unit during the past year. Some modifications in the system were made, such as for recording intracranial pressure and EEG. Overall, our experience with this system during the past year has been good. The nurses are burdened with less writing and have more time for bedside care. The house staff have been conditioned to obtain the desired data for decisions on patient care by the use of the displays or printed copies. Trends, such as intracranial compliance changes can be computed. Electronic problems have occurred but these result in returning to the previous system of recording patient functions. Patient care has probably improved because of the generalized stress of the importance of accuracy and close monitoring of patient functions.

(Discussion)

Friday, November 12

MODERATOR, James W. Correll

8:30 a.m.

15. Fenestrations in the Endothelial Cells of Pituitary Portal Vessels

Richard M. Bergland, Robert B. Page and Alphonse Leure-DuPree Hershey, Pennsylvania

The capillary bed of the median eminence (the neuro-hemal contact zone) is separated from the capillary bed of the adenohypophysis by vessels which are commonly referred to as "portal venules." In rat and man, electron microscopic studies demonstrate endothelial fenestrations in these large (300-500 micra) vessels. Thus, the "primary plexus" of median eminence is not separated from the "secondary plexus" of pars distalis by veins. The functional implications will be discussed.

(Discussion)

8:50 a.m.

16. Surgical Experiences with the Forbes-Albright Syndrome

J.S. Brodkey, J. Trujillo and O. Pearson Cleveland, Ohio

Twenty women, ages 18 to 41, were operated trans-sphenoidally since 1970 for presumed prolactin secreting pituitary adenomas. All but one patient presented with galactorrhea and all but one manifested amenorrhea.

Three patients had visual field defects on formal testing. Borderline hypoadrenalism was found in only one patient. There was no case of pituitary hypothyroidism. Urinary gonadotropins were in the low range of normal in all but three patients, one of which had normal menses.

Basal serum prolactins were elevated in all patients ranging between 38-1200 ng/ml. Prolactin response to a chlorpromazine provocative test was abnormal in all patients; however, normal prolactin fall was seen in 8 patients given L-Dopa.

Radiological workup including PEG with fine section polytomography in all patients showed minimal to marked abnormalities in all but 3 patients.

The surgical results depend on the size of the tumor and its degree of extrasellar extension as follows:

	Number	Resolve Galactorrhea	Resolve Amenorrhea	Pregnancies
Microadenoma	3	3	3	3
Large adenoma	6	5	3	1
Large adenoma with extension	7	2	2	1
Normal exploration	4	2	2	0
	20	12	10	5

9:05 a.m.

17. The Surgical Treatment of Hyperpituitarism

Gilles Bertrand Montreal, Quebec, Canada

A review will be made of 28 patients who presented with clinical and laboratory evidence of pituitary hyperfunction: Elevated growth hormone levels and acromegaly; elevated prolactin and amenorrhea-galactorrhea in females; impotence in males; elevated T.S.H. and hyperthyroidism; increased skin pigmentation following adrenalectomy for Cushing's disease.

All had radiological evidence, sometimes very subtle, of an intrasellar expanding lesion and were proven to harbor pituitary adenomas of various sizes and degree of extension.

All were treated surgically by a transphenoidal rhinoseptal microsurgical approach,

The preoperative physical findings and endocrine status will be reviewed together with the effects of surgical ablation of the tumor on the clinical symptoms and the hormonal serum levels.

Some points of surgical technique will be discussed and the after effects and complications of this surgical approach will be analyzed.

9:20 a.m.

18. Pituitary Microadenoma Associated with the "Empty Sella" Syndrome - A Report of 3 Cases

George T. Tindall Atlanta, Georgia

While the empty sella syndrome with certain exceptions (e.g., visual loss due to chiasmal traction) is generally considered a nonsurgical entity, nevertheless, there are a few scattered reports of pituitary microadenoma associated with this entity. The purposes of this presentation are: (1) to present 3 cases of prolactin-secreting microadenoma that occurred in

association with an empty sella and which were documented by pneumoencephalography (PEG) and trans-sphenoidal microsrugical exploration of the sella, and (2) to illustrate the value of the prolactin determinations in separating cases of asymptomatic empty sella from those associated with certain microadenoma.

The important features of each case will be presented. Each patient was a young woman who had had amenorrhea for several years or months prior to surgery. One patient also had spontaneous galactorrhea. In each case, fasting serum prolactin values were elevated significantly above normal level (approximately 40 ng/ml). The important finding in each case was the failure of chlorpromazine, or thorazine, to induce a further significant rise in prolactin values. PEG demonstrated air in the sella and transsphenoidal exploration revealed a microadenoma (less than 1 cm in diameter) in each patient. Pathologically, each tumor was a chromophobe adenoma. Post-operative values (at 10 days) for prolactin were normal and the response to chlorpromazine stimulation was appropriate in each case.

Despite the fact that all 3 of our cases had an empty sella, the results of the prolactin determinations were the major deciding reason for surgery. Not only were the fasting levels elevated, but there was no significant increase with chlorpromazine. In normal patients and in cases with empty sella without microadenoma, there is a 2 to 3-fold increase in prolactin 2 to 3 hours following the administration of chlorpromazine. In our 3 cases, the failure to respond normally suggested the presence of a tumor within the sella producing some degree of stalk compression and thus interferring with the normal flow of prolactin inhibiting factor (PIF) down the portal system into the adenohypophysis.

The author believes that the association between empty sella and microadenoma may be more common than previously suspected and that the use of prolactin determinations with chlorpromazine stimulation will prove helpful in studying all cases of empty sella.

9:35 a.m.

Discussion of Papers # 16, 17 and 18.

9:45 a.m.

19. The Role of Trans-sphenoidal Pituitary Adenectomy in Infertility

Theodore Roberts and Pedro Arlant Salt Lake City, Utah

Certain states of infertility, as in the Forber-Albright Syndrome, appear to be due solely to the effects of prolactin oversecretion. In patients with hyperprolactinemia (as seen in small pituitary adenomas), inhibition of normal hormone function at hypothalamic, pituitary and gonadal sites occurs. Data on the endocrinological evidence for prolactin's inhibitory effects will be presented. It appears fertility cannot be restored until the elevated prolactin levels are reduced. Four patients hyperprolactinemia that became pregnant within a year post adenectomy will be discussed. Thirty percent of patients with pituitary hypersecretion states had a normal sella per plain skull films. L-DOPA suppression and phenothiazine stimulation tests were found to be ineffective measures for determining presence of an adenoma.

Early adenectomy offers the best opportunity for return of the patient to an eupituitary state.

(Discussion)

10:05 a.m.

20. Serial Studies of Regional Cerebral Blood Flow in Acute Head Injury

Walter D. Obrist and Thomas W. Langfitt Philadelphia, Pennsylvania

Atraumatic measurements of regional cerebral blood flow (rCBF) were obtained on 20 acute head injuries, using the Xe-133 intravenous method. An average of four serial studies were performed during the acute phase and on later follow-up in each patient; 16 regional detectors were employed.

rCBF was found to be markedly depressed during coma (30 to 55

ml/100 g/min grey matter blood flow), but progressively increased as consciousness and neurological status improved (55 to 80 ml/100 g/min). Significant regional variations were observed in 15 patients, corresponding to the site of the lesion demonstrated by EMI scan. Transient hyperemia (luxury perfusion) was observed in 8 instances.

Serial rCBF studies appear to have prognostic value in acute head injury, and offer a potential means of assessing the effects of surgical and medical therapy not heretofore available. When combined with ICP and blood pressure measurements, this new information can facilitate patient management.

(Discussion)

10:25 a.m.

Coffee Break

10:40 a.m.

21. Management of Intracranial Pressure in Severe Cerebral Edema

Joan L. Venes, Dennis Spencer and William F. Collins, Jr. New Haven, Connecticut

Five consecutive cases of Reye's syndrome have been monitored for assessment of intracranial pressure, arterial blood pressure and end tidal PC02. Analysis of our data has allowed us to make certain observations concerning the management of intracranial pressure in patients with cerebral edema and low compliance unaccompanied by the complicating factors of trauma or mass effect. 1) Noxious stimulation produces a rapid and at times sustained rise in intracranial pressure even in the curarized, sedated patient 2) Non-noxious stimulation (e.g. pupil checks) may produce an increase in intracranial pressure in the recovering patient with low compliance even though no response is discernible 3) focal and subclinical (electrical activity only) seizures produce a sustained increase in intracranial pressure.

These responses occur in the sedated patient with stable arterial blood pressure and controlled ventilation and may indicate a response to an increase in cerebral blood flow secondary to activation of the reticular

formation. Such observations have led to a treatment protocol designed to minimize the effect of exogenous stimuli.

(Discussion)

11:00 a.m.

22. Subdural Hematoma in Infancy - A 20 year Review

E. Bruce Hendrick Toronto, Ontario, Canada

At the Hospital for Sick Children, we have had 315 cases of subdural hematoma in infancy admitted and treated between the years 1954 and 1973. During the first 10-year period, there were 253 cases and during the subsequent period of 8 years, we had 62 cases. An attempt will be made to correlate not only treatment with result but etiology with results, and to show that in many cases, the cause is the major factor in the prognosis. The different treatments such as burr hole drainage, subdural taps, shunting, craniotomy and excision of membranes will be discussed and compared.

(Discussion)

11:20 a.m.

23. The Chiari Type I Malformation, Our Experience with Ten Cases

David C. Fryer, John S. Tytus, M. Theodore Margolis and Edward Reifel

Seattle, Washington

Our experience with ten patients with the Chiari Type I malformation will be described. Seven have been decompressed with improvement in five. Three of the improved patients presented with signs of syringomyelia.

We believe that decompression of the displaced cerebellar tonsils may be helpful in selected patients with this condition.

(Discussion)

11:40 a.m.

24. The Internal Shunt: A Study in the Physiology of the Distal Subarachnoid Space in Hydrocephalus

Don DeFeo, Eldon L. Foltz and Scott Lederhaus Irvine, California

In an attempt to evaluate the function of the distal subarachnoid spaces and their potential availability in the treatment of hydrocephalus, thirty dogs were made hydrocephalic by the introduction of Kaolin into the cisterna magna. Ten hydrocephalic animals remained untreated as controls. Experiments were designed to discern the efficacy of microsurgically produced ventricular subarachnoid space fistulae. These fistulae create an intracranial path for cerebrospinal fluid (CSF) bulk flow to an absorptive site which bypasses the CSF flow obstruction at either the aqueduct of Sylvius or the basal cisterns and thus re-establishes a balance between production and absorption of intracranial CSF and thereby correction of the hydrocephalic state. The ventricle to subarachnoid fistulae were performed with the aid of the operating microscope connecting the convexity subarachnoid space of the right frontal region to the ventricle.

An experimental protocol was established utilizing a four step plan in which intraventricular pressures and RISA isotope studies were implemented initially on dogs in a normal state, later after hydrocephalus had been produced by Kaolin in the cisterna magna, and finally, four weeks after the transcerebral shunt.

At the conclusion of the study the animals were sacrificed and brain photographs were made along with histological examination of the ventricles, the fistulae and the distal subarachnoid space in the vicinity of the internal shunt.

(Discussion)

12:00 noon

Presidential Address: Wilder Penfield: the Man and His Work

William H. Feindel

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Saturday, November 13

MODERATOR - Phanor L. Perot, Jr.

9:00 a.m.

25. Primary Wire Mesh Cranioplasty in Flap Infections

Maxim Koslow and Joseph Ransohoff New York, New York

The occurrence of craniotomy flap infections where removal of the involved bone flap will result in a disfiguring appearance and leave unprotected a significant portion of the brain presents a difficult problem in management.

Recently, we have been performing primary wire mesh cranioplasties in this group of patients at the time of flap removal. A series of patients with large frontal or fronto-temporal flap infections is presented. One patient had an infected acrylic-mesh cranioplasty. All patients had active infections with staphlococcus aureus. Four patients with craniotomy infections had pathologic confirmation of osteomyelitis of the removed bone flaps. The patients were treated pre and postoperatively with antibiotics. Preoperatively, daily irrigation of the flap was carried out with Betadine solution. In each case, a wire mesh cranioplasty was carried out at the time of flap removal with thorough debridement of the wound.

There have been no recurrent infections in this group of patients. The average follow-up has been 10 months, with a range of 2 to 22 months. The pre and postoperative management and rationale for primary wire mesh cranioplasty in the presence of infection will be discussed.

(Discussion)

9:20 a.m.

26. Acute Cervical Spinal Cord Injury. Early Management and Long Term Results.

F. John Gillingham and James Steers Edinburgh, Scotland

Immediate and early management of patients over the past 15 years with acute cervical spinal cord injury in the Head and Spinal Injuries Research Unit of the University of Edinburgh is surveyed. Long term results are considered in the light of the evolution of methods of investigation and management.

(Discussion)

ACADEMY AWARD

9:40 a.m.

Mechano-Sensitivity of Dorsal Root Ganglia in Chronically Injured Axons: A Physiological Basis for Radicular Pain of Nerve Root Compression

John F. Howe, M.D. Seattle, Washington

10:10 a.m.

27. The Anatomy, Development and Distribution of the Recurrent Artery of Heubner in Normal and Pathologic Conditions.

R.H. Licata and E.W. Mack Reno, Nevada

The detailed anatomy and distribution of the recurrent artery of Heubner is presented. Its ultimate termination in the anterior perforated space where it supplies the basal ganglia and striatal branches are descirbed. The origin of the recurrent artery is variable and relates anatomically to the position of the anterior communicating artery. The development and phylogeny of this trunk is portrayed in association with vessels along the orbital surface of the frontal lobe. The intricate neuroregulatory mechanism of the recurrent artery is also demonstrated by means of cholinesterase staining and catecholamine autofluorescence.

The role of this vessel in stroke syndromes and arterial aneurysms is also noted.

(Discussion)

10:30 a.m.

Coffee Break

10:45 a.m.

28. Microsurgical Treatment of Intracranial Vascular Malformations

Charles B. Wilson and Hoi Sang U San Francisco, California

Experience with vascular malformations during the course of the last five years has modified our view toward operative indications and surgical techniques. In general the operative indications have been broadened with no reason to revert to earlier conservative attitudes. The surgical technique has evolved to utilize fully the enormous advantages of the operating coagulation under constant irrigation microscope, bipolar intraoperative injection of radiographic contrast media and visible dyes. Hypothermia has been a useful adjunct. Two patients have died following removal of massive vascular malformations, the cause in each instance being a sudden diversion of blood from the malformation into the defective capillaries of an ischemic hemisphere. Based upon this experience we recommend staged obliteration of very large malformations, allowing time for circulatory readjustment in the uninvolved brain. Particular attention will be given to malformations in certain locations: corpus callosum, medial trigone, and posterior fossa.

(Discussion)

11:05 a.m.

29. Angiopathy in Peripheral Nerve Pathology

Alan R. Hudson and Juan Bilbao Toronto, Ontario, Canada

The authors have performed sixty human sural nerve biopsies and reviewed the pathology by conventional and electron microscopy. Included in this group are fifteen patients suffering from chronic ischemia, whose biopsies were obtained immediately proir to limb amputation. The purpose of this presentation is to review the role of angiopathy in the pathogenesis of the wide variety of disease states suffered by these sixty patients. Electron photomicrographs of arterioles and capillaries are shown alongside electron photomicrographs of the abnormal nerve fibers. The relevance of these findings to peripheral nerve surgery will be discussed.

(Discussion)

11:25 a.m.

30. Autogenous Fat for the Prevention of the "Laminectomy Membrane"

Frank H. Mayfield Cincinnati, Ohio

Antogenous fat has been used for several years to cover the dura after laminectomy for the purpose of minimizing scar tissue attachment to the dura, to repair dural rents and to reconstruct the dura when postoperative meningoceles develop. Occasional clinical follow-up at reoperation has suggested that it is an effective method.

Studies in the human cannot adequately determine the fate and effectiveness of the fat graft, however, so mongrel dogs have been used and the results of the laboratory experiments will be the principal basis of this report. Our experience with fat grafts in humans will also be described.

Our data suggest that a layer of autogenous fat should be used to cover the dural tube after laminectomy and that it is a satisfactory substance for dural graft.

(Discussion)

11:45 a.m.

31. New Aspects of Pain Related to Spinal Cord Mechanisms

B.S. Nashold, Jr., D. Albe-Fessard and C. Lombard Durham, North Carolina

Since the introduction of the gate control theory of pain, considerable clinical and neurophysiological experimentation has been devoted to spinal cord pain mechanisms. The theory has generated new interest in the role of the spinal cord in pain. Recent neurophysiologic data suggest that mechanisms other than a gate control may be important in pain physiology. There is evidence that regional control in the spinal cord exists with local excitatory and inhibitory loops not requiring a gate control type of mechanism. The present report will outline this new experimental data and add to it recent observations made by the author with Professor Albe-Fessard at the Laboratoire de Physiologie des Centres Nerveux in Paris. The authors have developed a chronic experimental animal model for hyperesthesia related to avulsion of the spinal dorsal roots. In the rat, after surgical avulsion of the dorsal root filaments, a hyperesthetic state develops within 5 to 10 days and lasts for many months. The hyperesthesia appears to be related to alterations in local spinal cord mechanisms and can be influenced by Bradykinin (a pain inducing drug) and Strychnine (a drug known for its localized effect on presynaptic inhibitory mechanisms).

The experimental data will be presented and discussed.

(Discussion)

12:05 p.m.

32. Surgical Management of Epilepsy using Extra-operative Electrocorticography: Experience with 75 Cases.

Sidney Goldring St. Louis, Missouri

It has become increasingly clear that the most reliable evidence for

localizing an epileptogenic focus is that which is obtained in electrical recordings made during a spontaneous convulsion - evidence difficult to obtain during the time of surgery. Futhermore, not all patients can undergo surgery under local anesthesia. Yet the latter is essential for the appearance of the spontaneous spiking, production of electrical after-discharge and localization of sensorimotor region by cortical stimulation - evidence needed to identify and safely excise the focus. For these reasons we have resorted to extra-operative electrocorticography with indwelling electrodes.

Patients had one or more of the following kinds of seizures: partial without impairment of consciousness and those developing into generalized ones; psychomotor; generalized convulsion without focal onset.

Under general anesthesia a craniotomy is performed and the sensorimotor region is quickly identified (few minutes) by recording sensory evoked responses. A silastic template holding 24 electrodes is sewn in place extradurally to bridge the sensorimotor area and the region that is suspect. On the following day with the patient awake and resting, sensory evoked responses are again recorded and transdural stimulation of the cortical surface is carried out through the recording electrodes to further delineate the sensorimotor region. On the dominant side electrical stimulation is used to determine the boundries of the area controlling speech. Thereafter, continuous recording is carried out for the purpose of monitoring spontaneously occurring seizures. Both the on-going EEG and the subject's behavior is video-taped. Replays on the split screen of a television monitor permit determination of the area showing the first sign of abnormal EEG activity in the momnets preceding clinical convulsion. If the studies (24-48 hrs) identify the boundries of a single epileptogenic focus it is excised during a secondary craniotomy; if not the silastic template is simply removed. When depth electrodes are used (temporal lobe epilepsy) the procedure is modified appropriately. Fifty-three patients have had excision of an epileptogenic focus (cortical resections, 20; anterior temporal lobectomy, 24; hemishperectomy, 5; sub-total hemispherectomy, 4). Sixty-two percent have been benefited by surgery. The series includes patients that probably would have been amenable to surgical management by traditional methods and examples of these will be demonstrated.

12:25 p.m.

33. A Quantitative Study of the Effect of Dentatectomy in Animal Models of Spasticity.

F. Gentili and R.R. Tasker Toronto, Ontario, Canada

Spasticity from stroke, trauma, and cerebral palsy is a significant cause of human disability. Although various neurosurgical procedures (thalamotomy, dentatectomy, cerebellar stimulation) have been proposed for its relief, failure to use quantitative measurment has made it difficult to assess the effectiveness of the procedures and the paucity of laboratory studies has hindered the assembly of guidelines for patient treatment. The present study with Dr. Gentili, one of our neurosurgical trainees, was made in an attempt to produce animal models of spasticity, to develop a quantitative means for their evaluation, and to study the effectiveness of stereotactic dentatectomy.

The integrated evoked E.M.G. method, developed for quantitative measurement of spasticity in humans, was modified for use in the experimental animal. Spasticity resulted in an elevation of the y-intercept and/or an increase in slope when integrated evoked E.M.G. was computer plotted against rate of muscle stretch. Virtually no E.M.G. was evoked in normal muscles even by very rapid stretch. Three animal models were prepared: - intercollicular decerebrate and anaemic decerebrate cats, and motor decorticate squirrel monkeys. In keeping with most published experience it was necessary to ablate the primary motor cortex extensively on both sides before muscle tone increased in a striking fashion. The resulting spasticity, chiefly kinetic in type, usually but not always affected the biceps and quadriceps preferentially.

After the animals had been followed for one to two months with serial quantitative determinations of muscle tone, stereotactic lesions were made in one or both dentate nuclei. Their extent, determined from serial sections was plotted and correlated by computer with serial measurements of muscle tone. Dentate lesions diminished muscle tone ipsilaterally by 60% in decorticate monkeys, the effect being proportional to the volume of the lesion. There was no additional benefit from bilateral lesions. Dentatectomy did not alter muscle tone in intercollicular or anaemic decerebrate cats. This study suggests that dentatectomy be considered in

patients spastic from cortical lesions but not in those with lesions of the lower brainstem.

(Discussion)

12:45 p.m.

34. The Organization of Short-Term Verbal Memory in Language Area of Human Cortex

George A. Ojemann Seattle, Washington

Electrical stimulation during a standard test of language and short-term verbal memory was applied to posterior, frontal, parietal, and temporal cortex of the language dominant hemisphere in five patients undergoing craniotomies for the resection of epileptic focci. The standard test has been previously described (Ojemann, et al, Brain 94:225, 1971) and measures short-term verbal memory in a single item paradigm with naming of object pictures as input, a six-second standard distraction (counting backwards by three's) and retrieval by cued recall. Electrical stimulation utilized 60 Hz, 2.5 millisecond biphasic square wave pulses in 4 second trains at current levels varying between 4-10 milliamps. Stimulation is applied during the input, or retrieval, or input and retrieval or only the distraction phases on randomly selected trials of the short-term verbal memory test. Interspersed trials with no stimulation were used as controls. Data was obtained from 25 sites in these patients, each patient contributing 4-6 sites. The short-term program memory test was very easy for these patients, with only one error made by any patient on the non-stimulation control trials. Cortical areas were identified as being concerned with language, if changes in object naming were evoked with stimulation. The cortical areas concerned with language were generally not involved in short-term verbal memory. From nine sites, no changes in either language or short-term verbal memory were evoked. From eight sites only changes in short-term verbal memory were evoked, in five only language changes, and from only three sites were both language and short-term verbal memory changes evoked. "Anomic" disturbances of object naming were evoked from five locations in the temporal-parietal lobe of four patients. The same current levels and the same locations that evoked anomia on no occasion disturbed acute recall from short-term verbal memory. On the other hand, short-term verbal memory changes were evoked from surrounding cortex, with different areas of left frontal, parietal and lateral temporal cortex related to different short-term verbal memory processes. Stimulation during the input or storage phases of the short-term verbal memory tests disrupted short-term verbal memory when the current was applied to the parietal or temporal opercula, immediately anterior to the posterior language area. This effect was noted in all three patients so stimulated in this area. No change in the distracting task occurred during these stimulations. This type of response to stimulation suggests that this area may be the site of the active storage process in short-term verbal memory. Stimulation during retrieval disturbed short-term verbal memory only from the sites above the sylvian fissure, predominantly in posterior frontal lobe.

(Discussion)

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ACADEMY AWARD

1976

JOHN F. HOWE, M.D.

University of Washington

Seattle, Washington

"Mechano-Sensitivity of Dorsal Root

Ganglia in Chronically Injured Axon:

A Physiological Basis for

Radicular Pain of

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1976

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DONALD F. DOHN 2020 East 93rd Street Cleveland, Ohio 44106	(BETTY)	1968
R.M. PEARDON DONAGHY Mary Fletcher Hospital Burlington, Vermont 05401	(FRANCES)	1970
CHARLES G. DRAKE University Hospital 339 Windermere Road London, Ontario, Can. N6G 2K3	(RUTH)	1958
STEWART B. DUNSKER Mayfield Neurological Inst. 506 Oak Street Cincinnati, Ohio 45219	(ELLEN)	1975
GEORGE EHNI 1531 Hermann Prof. Bldg. 6410 Fannin Street Houston, Texas 77025	(VALAIRE "LARRY")	1964
WILLIAM H. FEINDEL Montreal Neurological Inst. 3801 University Street Montreal, Quebec, Canada	(FAITH)	1959
ROBERT G. FISHER Muhlenberg Hospital Plainfield, N.J. 07061	(CONSTANCE)	1957
ELDON L. FOLTZ Division of Neurosurgery Univ. of Cal. School of Med. Irvine, California 92664	(CATHERINE)	1960

JOHN D. FRENCH The Center for the Health Sciences University of California Los Angeles, Cal. 90024	(DOROTHY)	1951
LYLE A. FRENCH University of Minn. Med. Ctr. Minneapolis, Minn. 55455	(GENE)	1954
JOHN T. GARNER 744 Fairmont Avenue Pasadena, Cal. 91105	(BARBARA)	1971
HENRY GARRETSON Dept of Neurosurgery University of Louisville Louisville, Kentucky	(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 82601	(ELIZABETH ANN "LISA")	1968
JOHN R. GREEN Barrow Neurological Inst 302 West Thomas Street Phoenix, Arizona 85013	(GEORGIA)	1953
JOHN W. HANBERY Division of Neurosurgery Palo Alto, California 49304	(SHIRLEY)	1959
MAJ. GEN. GEORGE S. HAYES MC USA, Principal Deputy Office of the Asst. Sec. of Defense Health & Envir Washington, D.C. 20301	(CATHERINE)	1962

E. BRUCE HENDRICK Hospital for Sick Children 555 University Avenue Toronto, Ontario, Canada	(GLORIA)	1968
JULIAN HOFF Dept. of Neurosurgery Univ. of CalSan Fran. San Francisco, Calif. 94143	(DIANE)	1975
WILLIAM E. HUNT 410 West 10th Avenue Columbus, Ohio 43210	(CHARLOTTE)	1970
DAVID KELLY Bowman-Gray School of Medicine Winston-Salem, N.C. 27103	(SALLY)	1975
ROBERT B. KING University Hospital Upstate Medical Center 750 East Adams Street Syracuse, N.Y. 13210	(MOLLY)	1958
WOLFF M. KIRSCH Univ. of Colorado Med. Ctr. Denver, Colorado 80220	(MARIE-CLAIRE)	1971
DAVID G. KLINE Louisiana St. Univ. Med Ctr. 1542 Tulane Avanue New Orleans, Louisiana 70012	(CAROL)	1972
ROBERT S. KNIGHTON Henry Ford Hospital 2799 W. Grand Blvd. Detroit, Michigan 48202	(LOUISE)	1966

THEODORE KURZE Los Angles County-U.S.C. Medical Center 1200 North State Street Los Angles, Calif. 90033		1967
THOMAS W. LANGFITT Hospital of the Univ. of Penn. 34th and Spruce Streets Philadelphia, Penn 19104	(CAROLYN)	1971
RAEBURN C. LLEWELLYN Tulane University 1430 Tulane Avenue New Orleans, La. 70012	(CARMEN)	1963
WILLIAM M. LOUGHEED Medical Arts Bldg. 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
JOHN J. LOWREY Straub Clinic 888 S. King Street Honolulu, Hawaii 96813	(CATHERINE "KAY")	1965
ERNEST W. MACK 505 S. Arlington Avenue Suite 212 Reno, Nevada 89502	(ROBERTA)	1956
M. STEPHEN MAHALEY, JR. Duke University Med. Ctr. Durham, N. Carolina 27706	(JANET)	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 fo Neurosurgery Cincinnati General Hosp. Cincinnati, Ohio 45229	(KATHERINE)	1955
WILLIAM F. MEACHAM Vanderbilt University Hosp. Division of Neurosurgery Nashville, Tennessee 37203	(ALICE)	1952
JOHN F. MULLAN, M.D. Univ. of Chicago Clinics Department of Neurosurgery 950 East 59th Street Chicago, Ill. 60637	(VIVIAN)	1963
BLAINE S. NASHOLD, JR. Duke University Med. Center Durham, North Carolina 27706	(IRENE)	1967
FRANK F. NULSEN Div. of Neurosurgery University Hospital 2065 Adelbert Road Cleveland, Ohio 44106	(GINNEY)	1956
GUY L. ODOM Duke University Med. Ctr. Durham, N.C. 27706	(MATALAINE)	1946
GEORGE OJEMANN University of Washington Dept. of Neurosurgery Seattle, Washington 98195	(LINDA)	1975
ROBERT G. OJEMANN Massachusetts Gen. Hosp. Div. of Neurological Surg. Boston, Mass. 02114	(JEAN)	1968

BURTON ONOFRIO Mayo Clinic Rochester, Minn. 55901	(JUDITH)	1975
RUSSEL H. PATTERSON, JR. 525 East 68th Street New York, New York 10021	(JULIE)	1971
PHANOR L. PEROT, JR. Medical Univ. of S.C. 80 Burre Street Charleston, S.C. 29401	(ELIZABETH)	1970
BYRON C. PEVEHOUSE 2001 Union Street San Francisco, Calif. 94101	(MAXINE)	1964
ROBERT W. PORTER 5901 East 7th Street Long Beach, Calif. 90804	(AUBREY DEAN)	1962
JOHN RAAF 833 S. W. 11th Avenue Portland, Oregon 97205	(LORENE)	Founder
AIDEN A. RANEY 2010 Wilshire Blvd. Suite 2 Los Angles, Calif. 90057	(MARY)	1946
JOSEPH RANSOHOFF New York Univ. Med. Center 500 First Avenue New York, New York 10016	(RITA)	1965
THEODORE B. RASMUSSEN Montreal Neurological Inst. 3801 University Street Montreal 2, Quebec, Canada	(CATHERINE)	1947
DAVID H. REYNOLDS Section of Neurosurgery, 112-F Univ. of S. Florida V.A. Hospital, 1300 N. 30th St. Tampa, Florida 33612	(MARJORIE)	1964

HUGO RIZZOLI 2150 Penn Avenue, NW Washington, D.C. 20037	(HELEN)	1973
JAMES T. ROBERTSON 20 South Dudley Street Memphis, Tennessee 38103	(VALERIA)	1971
RICHARD C. SCHNEIDER C5135 Out Pt. Building University Hospital Ann Arbor, Michigan 48104	(MADELEINE)	1970
JAMES C. SIMMONS 20 S. Dudley Street Memphis, Tenn. 38103	(VANITA)	1975
BENNETT M. STEIN Dept. of Neurosurgery 171 Harrison Avenue Boston, Massachusetts 02111	(DOREEN)	1970
JIM L. STORY 7703 Floyd Curl Drive San Antonio, Texas 78229	(JOANNE)	1972
THORALF M. SUNDT, JR. 200 1st Street, S.W. Rochester, Minn. 55901	(LOIS)	1971
ANTHONY F. SUSEN 3600 Forbes Avenue Pittsburg, Pa. 15213	(PHYLLIS)	1965
WILLIAM H. SWEET Massachusetts Gen. Hospital Div of Neurological Surgery Boston, Mass. 02114	(MARY)	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 Univ. School of Med. Division of Neurosurgery 1365 Clifton Road, NE Atlanta, Georgia 30322	(SUZIE)	1968
JOHN TYTUS Mason Clinic Seattle, Washington 98107	(VIRGINIA "GINA")	1967
ARTHUR A. WARD, JR. Department of Neurol. Surg. Univ. of Washington Hosp. Seattle, Washington 98105	(JANET)	1953
CLARK WATTS Univ. of Missouri-Columbia N522 Medical Center Columbia, Missouri 65201	(PATTY)	1975
W. KEASLEY WELCH Childrens Hosp. Med. Ctr. 300 Longwood Avenue Boston, Mass. 02115	(ELIZABETH)	1957
BENJAMIN B. WHITCOMB 85 Jefferson Street Hartford, Conn. 06106	(MARGARET)	1947
LOWELL E. WHITE, JR. Professor & Chairman Division of Neurosciences Mobile, Alabama 36688	(MARGIE)	1971
ROBERT WILKINS Presbyterian-University Hosp. Room 9402 230 Lothrop Street Pittsburgh, Pa. 15213	(GLORIA)	1973

CHARLES B. WILSON Dept. of Neurol. Surgery U. of Cal. Medical Center Third and Parnasus San Francisco, Cal. 94122		1966
FRANK WRENN 123 Mallard Street Greenville, S.C. 29601	(BETTY)	1973
DAVID YASHON 410 W. 10th Avc. N. 911 Columbus, Ohio 43210	(MYRNA)	1972
NICHOLAS T. ZERVAS 330 Brookline Avenue Boston, Mass 02215	(THALIA)	1972

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) 3/01/75	1951
DR. OLAN R. HYNDMAN Iowa City, Iowa	(Senior) 6/23/66	1942
MR. KENNETH G. JAMIESON Brisbane, Australia	(Corresponding) 1/28/7	6 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) 4/05/76	1960
DR. RUPERT B. RANEY Los Angles, 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) 8/08/75	1939
DR. C. WILLIAM STEWART Montreal, Quebec, Canada	(Corresponding)	1948
DR. GLEN SPURLING La Jolla, California	(Honorary) 2/07/68	1942
DR. HENDRIK SVIEN Rochester, Minnesota	(Active) 6/29/72	1957

AMERICAN ACADEMY OF NEUROLOGICAL SURGERY 1976 ANNUAL MEETING

EVALUATION

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

(1)	Was the general content of the scientific program:	
	_	_ Excellent _ Good _ Poor
(2)	If you found it p	oor, 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:
		_ Too low _ Too high _ Just about right
SCIE	NTIFIC PROGRA	AM
Thu	rsday's Sessions	ExcellentGoodPoor
Frid	ay's Sessions	ExcellentGoodPoor
Satu	arday's Sessions	ExcellentGoodPoor Comments

SOCIAL PROGRAM

Comments	
What changes would you like to see in future meeting	
what changes would you like to see in future meeting	35:

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525 East 68th Street

New York, New York 10021



