The American Academy of Neurological Surgery Program

Houston, Texas
1985

ANNUAL MEETING OF THE
AMERICAN ACADEMY OF
NEUROLOGICAL SURGERY
1985

The Lincoln Hotel Post Oak
Houston, Texas
October 27-30, 1985

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THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
SUNDAY, OCTOBER 27 - WEDNESDAY, OCTOBER 30, 1985
THE LINCOLN HOTEL POST OAK
HOUSTON, TEXAS

Sunday, October 27th:

1:00-6:00 p.m.	Registration - Foyer
1:00-4:00 p.m.	Ladies Hospitality Room - Concorde A B
2:00-5:00 p.m.	Historian's slide show - Forum
6:00-8:00 p.m.	Cocktail buffet - Ballroom B

Monday, October 28th:

7:00-10:00 a.m.	Registration - Foyer
7:00-8:00 a.m.	Member's business breakfast - Ballroom A
8:30 a.m.-Noon	Scientific meeting - Ballrooms B & C
	Coffee break - Atrium
10:30-4:30 p.m.	Ladies Hospitality Room - Concorde A B
Noon-1:00 p.m.	Lunch - members and guests - Ballroom A
1:00 p.m.	Group photograph
1:30-5:00 p.m.	Scientific meeting - Ballroom B & C
2:00-4:00 p.m.	Registration - Foyer
6:30 p.m.	Buses depart for Museum of Fine Arts
7:00-10:00 p.m.	Cocktails, jazz combo, buffet - Museum of Fine Arts
9:30 p.m.	Buses depart museum for hotel
10:00 p.m.	
10:30 p.m.	

Tuesday, October 29th:

7:00-10:00 a.m.	Registration - Foyer
7:00-8:00 a.m.	Member's business breakfast - Ballroom A
8:30 a.m.-Noon	Scientific meeting - Ballrooms B & C
	Coffee break - Atrium
9:00 a.m.-4:00 p.m.	Ladies Hospitality Room - Concorde A B
Noon	Lunch with wives - Ballroom A
1:00-1:30 p.m. to	NASA, San Jacinto, golf, tennis, riding, shooting, swimming at hotel, shopping Galleria, slide show repeat
7:00-8:00 p.m.	Cocktails - Black tie - Foyer
8:00 p.m.-Midnight	Dinner-dance - Ballrooms A & B

Wednesday, October 30th:

7:30 a.m.	Breakfast buffet (all) - Ballroom A
8:30 a.m.-Noon	Scientific meeting - Ballrooms B & C
	Coffee Break. Atrium
9:30 a.m.-Noon	Ladies Hospitality Room - Concorde A B
Noon	ADJOURN

LADIES ACTIVITIES

Sunday, October 27th:

1:00-4:00 p.m.	Hospitality Room - Concorde A B
2:00-5:00 p.m.	Historian's Slide Show - Forum

Monday October 28th:

9:00 a.m.-4:00 p.m.	Hospitality Room - Concorde A B
9:00 a.m.-Noon	City Tour by bus
12:30 p.m.	Lunch with President's wife - Poolside
2:00 p.m.	Wearable Art Show - Poolside (San Felipe Room in case of rain)

Tuesday, October 29th:

9:00 a.m.-4:00 p.m.	Hospitality Room - Concorde A B
9:15 a.m.	Buses depart for Bayou Bend
9:45-11:30 a.m.	Guided tour of Bayou Bend (Low-Heeled soft-soled shoes mandatory)
Noon-1:30 a.m.	Lunch with husbands - Ballroom A
Afternoon	NASA, San Jacinto, golf, tennis, riding, shooting, swimming at hotel, shopping Galleria, repeat slide show if demanded

Wednesday, October 30th:

7:30-8:30 a.m. Breakfast buffet (all) - Ballroom A

9:00 a.m.-Noon Hospitality Room - Concorde A B

Across Post Oak from the Hotel: Saks, Abercrombie & Fitch, Cartier and more

At Post Oak and Westheimer: Galleria - Neiman-Marcus, Lord & Taylor, Marshall Field, Tiffany, Gumps and more

At Post Oak and Alabama: The tallest building outside a downtown area - Transco Tower, 64 floors - Adjacent water wall and garden

SCIENTIFIC PROGRAM

Monday, October 28

SCIENTIFIC SESSION I
MODERATOR - M.S. MAHALEY, JR., M.D.

8:30 WELCOME

8:35 **SPECIAL LECTURE - S.J. PEERLESS, M.D.**
"REPORT OF THE EC-IC COOPERATIVE STUDY"

9:30

1. INTELLECTUAL FUNCTIONING FOLLOWING NEAR DROWNING

J. Gordon McComb, M.D., Terece Stovall Bell, Ph.D.,
and Leah Ellenberg, Ph.D.

Between April, 1979, and August, 1983, 49 severe near-drowned children were admitted to Childrens Hospital of Los Angeles (CHLA) with a Glasgow Coma Score of 3, 4 or 5 and underwent intracranial pressure (ICP) monitoring and brain resuscitative therapy. Of the patients in this group, 29 (59%) died in the hospital 1 day to 3 months after admission, 13 (27%) were discharged in a vegetative state, and 7 (14%) made a good recovery. There were no patients who made only a partial neurologic recovery. Sustained mean highest ICP was significantly higher and the sustained lowest cerebral perfusion pressure (CPP) was significantly lower for fatalities than for survivors ($p<0.05$) but it did not significantly distinguish between intact and vegetative survivors. Pupillary reactivity noted on arrival at CHLA also significantlydiscriminated between survivors and fatalities ($p<0.05$) but not between intact and vegetative survivors. The presence of any motor activity after arrival at CHLA, even just posturing or twitching, indicated a significant chance for intact survival ($p<0.05$) although such activity did not discriminate between death or vegetative survival. Extensive neuropsychologic testing indicated that the apparent intact recovered patients generally showed near average levels of cognitive funtioning with mild residual gross motor and coordination deficits.

9:50 COFFEE BREAK

10:05

2. MESIAL TEMPORAL ACTIVATION OF THE HIPPOCAMPUS IN TEMPORAL LOBE EPILEPSY

Robert G. Grossman, M.D.

The neural circuits generating the hippocampal spike, one of the characteristic signs of temporal lobe epilepsy, are still incompletely understood. Since afferent discharge can evoke paroxysmal depolariza-

tion in excitable neurons, the ability of the entorhinal projections from areas 28 and 29, which comprise the major afferent pathway to the hippocampus, to evoke hippocampal discharge was investigated in 20 patients with mesial temporal sclerosis and complex partial seizures. Control of seizures was obtained in 18 of the 20 patients who underwent anterior temporal lobectomy, indicating that the tissue studied contained neurons mediating the patient's seizures.

The entorhinal-hippocampal pathways were found to be highly excitable, with little topographic specificity. A single small electrical stimulus of 1-2 mA at threshold, delivered through 1 mm diameter electrodes with a 1 mm separation, when applied to either the anterior or posterior portions of areas 28-29 evoked a characteristic interictal spike discharge from the hippocampus. The spikes were recorded along the length of the pes and the anterior body of the hippocampus. The evoked spikes were identical to the spontaneous spikes recorded at surgery, and with chronic depth electrodes in the same patients.

The lack of physiological topographical specificity that was found stands in contrast to the specificity of the normal anatomical organization of the entorhinal-hippocampal projections, and the intrinstic circuitry of the hippocampus, which is organized in transverse arrays, with recurrent surround inhibition.

The present electrophysiological data have been correlated with patterns of neuronal loss and preservation in the resected tissue. CA_2 pyramidal cells, which tend to be preserved in mesial temporal sclerosis, and which give rise to the axial association pathway of the hippocampus, may mediate and synchronize the spread of spike activity in the longitudinal axis of the hippocampus.

10:25

3. NON-INVASIVE CEREBRAL ANGIOGRAPHY

Nicholas T. Zervas, M.D. and Allan Nelson, M.D.

Non-invasive visualization of the cerebral arteries would be a major benefit to patients with potential or real occlusive or aneurysmal lesions. The resolution of contemporary MRI scans is such that vessels with diameters greater than one millimeter may be detected in two dimensional brain without contrast enhancement. Proper alignment of sequential images of one millimeter separation permits the reconstruction of vessel segments whose axis is perpendicular to the plane of reference. The Neurosurgical Service in conjunction with the Artificial Intelligence Division and Computer Science Department of the Massachusetts Institute of Technology is now exploring the feasibilty of reconstructing adjacent vessel segments that can then be displayed in a three dimensional form. The preliminary studies of vessels with a diameter of four

millimeters or greater, indicates that such three dimensional representation can be achieved. The three dimensional image can be observed directly simply by viewing the screen of a newly designed optical synthesizer that is directed by a blood vessel computer algorithm. This enables the observer to study directly the anatomy of any desired vessel and to observe any dilatation or construction of its inner surface. This report describes the technical basis for this form of angiography. The three dimensional display will be projected during the presentation.

10:45

4. MULTILOBAR GIANT CEREBRAL ARTERIOVENOUS MALFORMATIONS-EXPERIENCE WITH DIRECT SURGICAL RESECTION

Ghaus M. Malik, M.D., James I. Ausman, M.D.,
Robert S. Knighton, M.D., and Robert Mann, M.D.

The treatment of arteriovenous malformation has gone through significant changes in the recent years but the giant malformations (greater than 5.0 cm) are still considered inoperable by many. These malformations have been primarily treated by embolization or partial ligation. Recently multi-stage resection has also been advocated.

Our report consists of twenty-five patients with giant arteriovenous malformations without extensive basal ganglia involvement out of more than 100 cases treated surgically since 1975. Seizure was the presenting feature in 60% of the patients while 16% had intracranial hemorrhage. Headaches or progressive neurological deficit led to the diagnosis in the others. All of the patients were treated by direct surgical resection without adjunctive therapy such as embolization. The malformation was supplied by two major cerebral vessels in fifteen patients and by all three vessels in the other ten. In addition, several malformations had supply from the meningeal vessels or anterior choroidal artery.

Most of the malformations involved two adjacent lobes with predominant central location. Four patients had aneurysms on the feeding vessels. In three patients these aneurysms were treated before surgery for AVM while in the fourth one, clipping of the aneurysm was done at the same time.

Except in two cases when the posterior cerebral artery was clipped prior to actual excision of the malformation, the AVM was excised in one stage. One other patient needed a second operation for a small residual malformation. Total excision of the malformation was achieved in all cases, verified by angiography.

There was one operative death (4%) and morbidity included variable visual field defects in six patients not interfering with their work and significant paresis or dysphasia in four patients (16%). The "Normal

Pressure Breakthrough Phenomenon" was not observed in these patients or the others undergoing surgery during this period of time. Generally, the operating microscope was a hindrance in these cases. A new type of bipolar coagulation was found highly valuable. The technical aspects will be illustrated and discussed.

Contrary to general opinion, this report indicates that giant multilobar AVM's are resectable with the potential for excellent results without the need for adjunctive therapy.

11:05

5. STUDIES ON PERI-AVM VASCULAR CHANGES

Bennett M. Stein, M.D., Robert Solomon, M.D.,
and Karin Muraszko, M.D.

In a review of 200 operative AVM cases, certain of these cases have been selected for particular study of the AVM vessels. The AVMs selected for the most part are those with large shunts and markedly dilated feeding arteries. The nutrient arteries demonstrate abnormal vessel reactivity following the occlusion or resection of the AVM.

Methods of study include angiographic analysis, cerebral blood flow analysis and direct analysis of the segment of the feeding artery. In the latter study at the time of operation, a short proximal segment of the feeding artery is removed for physiological (dynamic study), electron-microscopy and catecholamine analysis.

The studies to date suggest a marked derangement in vessel reactivity of large arteries going to major AVMs. It appears that vessels respond in abnormal fashion to the usual provocative pharmacological agents and furthermore may exhibit anatomical abnormalities of their walls. This correlates with angiographic data which indicate that these arteries are slow to regain normal size following resection of AVMs and with blood flow studies that suggest increased pressure, but decreased flow in the surrounding arterial bed following the removal of large AVMs. These phenomena will be demonstrated by appropriate cases.

6. DIRECT SPINAL ARTERIOVENOUS FISTULA INVOLVING THE ANTERIOR SPINAL ARTERY, AN UNUSUAL TYPE OF SPINAL ARTERIOVENOUS MALFORMATION

Roberto C. Heros, M.D., Gerard Debrun, M.D.,
and Robert Ojemann, M.D.

A 31-year-old man had suffered from progressive paraparesis for 2 years. At the time of referral he was wheelchair-bound. Selective arteriography demonstrated a direct arteriovenous fistula at the T3-4 level. The fistula was formed by large descending and ascending anterior spinal arteries that communicated directly with a distended vein draining up to the posterior fossa. The fistula was obliterated by a direct transthoracic anterior surgical approach. Clinical, radiographic, and operative details will be presented.

There are three distinct types of spinal AVMs. Type I ("long dorsal AVM", "single coiled vessel malformation", "angioma racemosum venosum"), the most common, occurs in middle-aged men, usually in the mid and lower thoracic and lumbar regions. It consists of a long coiled vessel containing arterial blood under low pressure. It appears that in most of these cases the true AVM is extradural and it drains intradurally by the one or two efferent "feeding" vessels which enter the dura in close proximity to a dorsal root. These arterialized draining veins are the "feeders" which connect with the dorsal coronal venous plexus of the cord which then becomes arterialized and distended. These patients usually present with progressive paraparesis, probably from venous hypertension.

Type II spinal AVM ("glomus types", "arteriovenous angioma") occurs as frequently in males and females throughout adulthood. The lesion is usually intramedullary, compact, and under high pressure. They occur both in the cervical and the thoracic regions and usually have multiple feeders mostly from the anterior spinal artery. These patients present either with hemorrhage or with a progressive neurologic syndrome.

The third and least common type is the "juvenile" (Type III) spinal AVM, which appears usually in children and young adults. These lesions are extensive with intra- and extramedullary as well as spinal and sometimes paraspinal extensions. They present with hemorrhage or progressive neurologic dysfunction.

Our case does not fit into any of the above categories. There was no true compact angiomatous mass in our case and the fistula involved the intrinsic arterial supply of the cord. An unusual surgical approach had to be devised to treat this patient. We found no case like ours in our review of the literature, but we suspect that other cases exist and have probably

been classified as unusual types under one of the formerly described categories. We propose a new category (Type IV) of spinal AVM to denote a direct arteriovenous fistula involving the intrinsic arterial supply of the cord.

11:45

7. ELECTRICAL STIMULATION IN QUANTITATIVE ASSESSEMENT OF CUTANEOUS SENSIBILITY IN TRIGEMINAL NEURALGIA

Lauri Laitinen, M.D., Ph.D. and Marwan Hartz, M.D.

Electrical stimulation was used for quantitative assessment of facial sensibility before, during and after percutaneous electrocoagulation of the Gasserian ganglion in 19 patients with tic douloureux. A portable stimulator was of a constant current type, which generated rectangular monophasic pulses of 0.2 ms in length and 100 Hz in frequency. The bipolar electrode consisted of saline-soaked felt discs with a surface of 1 cm^2 and an interpolar distance of 1 cm. The thresholds for perception and pain were measured over six regions of each side of the face. Additionally, maximal pain tolerance was measured in the painful area and its corresponding healthy area. Shortlasting intravenous anesthesia with Brevital was given before each electrocoagulation. As soon as the patient began to react to speech, the threshold for pain was measured in the painful and the corresponding healthy area.

Preoperatively, the average threshold for perception was 2.5 mA and for pain 3.5 mA. The average maximal pain tolerance was 10.0 mA. There were no differences between the painful and the healthy sides.

Electrocoagulations were stopped when the threshold for pain in the trigger area had become twice as high as that on the contralateral side.

The postoperative measurements showed that the average thresholds for perception and pain had doubled, measuring 4.7 and 8.0 mA, respectively. The average pain tolerance had risen from 10.0 to 22.5 mA. A marked rise of both thresholds was also seen in the ipsilateral areas adjacent to the trigger zone. There was good correlation between a heavy sensory deficit and a favourable clinical result.

We conclude that electrical stimulation is an excellent method for quantitative assessment of facial sensibility in tic douloureux. There is no preoperative sensory deficit. Electrocoagulation affects tactile and nociceptive sensibility equally. The sensory deficit is not restricted to the painful area. A heavy sensory loss predicts a good clinical outcome.

12:00 Lunch

1:00 Group Photograph

SCIENTIFIC SESSION II
MODERATOR - THOMAS W. LANGFITT, M.D.

1:30

SPECIAL LECTURE - THOMAS GENNARELLI, M.D. "NEUROBIOLOGY OF TRAUMATIC AXONAL DAMAGE"

2:25

8. EVALUATION OF CEREBRAL HEMODYNAMICS IN PATIENTS WITH CAROTID ARTERY DISEASE USING POSITRON EMISSION TOMOGRAPHY

Robert L. Grubb, Jr., M.D. and William J. Powers, M.D.

The following series of events appears to take place as local cerebral perfusion pressure (CPP) falls. Local cerebral blood flow (CBF) is initially maintained by dilation of pre-capillary resistance vessels manifested as an increase in local cerebral blood volume (CBV). When compensatory vasodilation is maximal, cerebral autoregulation fails and CBF begins to fall. Local cerebral oxygen metabolism ($CMRO_2$) is then maintained by a progressive increase in the local oxygen extraction (OEF) by the brain. Once local OEF becomes maximal, a further decrease in CBF will result in disruption of normal cellular metabolism and function. Whether this disruption is reversible or progresses to irreversible infarction depends on a complicated interplay of a variety of poorly understood factors.

CBF, $CMRO_2$, and CBV were measured with positron emission tomography (PET) in twenty two patients with a "flow-reducing" carotid artery lesion (90% stenosis or occlusion of the internal carotid artery (ICA). Nine patients had 90-99% stenosis of an ICA. Six of these patients had normal cerebral hemodynamics and metabolism, while two patients had no reduction of CBF, but evidence of vasodilation (increased CBV/CBF ratio) in the cerebral hemisphere ipsilateral to the carotid artery lesion. One of these patients had reduced CBF with increased OEF in the ipsilateral cerebral hemisphere. In thirteen patients with an occluded ICA, two patients had normal cerebral hemodynamics and metabolism. Seven of these thirteen patients had no reduction of CBF, but had an increase in the CBV/CBF ratio in the ipsilateral cerebral hemisphere. Four patients had reduced CBF and increased OEF in the ipsilateral cerebral hemisphere.

The effect of carotid artery lesions on the cerebral circulation cannot be determined from the degree of angiographic stenosis or the presence of ICA occlusion. The degree of carotid stenosis or the finding of ICA occlusion are not reliable indicators of a hemodynamic cause of cerebral symptoms or the need for surgical revascularization to improve

CBF. Further studies are needed to determine if patients with truly hemodynamically significant carotid artery lesions are at an increased risk for stroke.

2:45

9. METABOLIC EVALUATION OF FOCAL STROKE REGIONS DEFINED BY NEUTRAL RED DISTRIBUTION

Robert A. Ratcheson, M.D., Craig A. VanDerVeer, M.D., Warren R. Selman, M.D., and W. David Lust, Ph.D.

Previous studies on the metabolic status of tissue following focal ischemia have been compromised due to an inability to define the areas affected by the occlusion. Sampling of cerebral tissue for metabolite analyses was based on regional landmarks of the brain rather than on the spatial characteristics of the insult, which vary widely between animals. Radiographic blood flow measurements can identify the areas of interest, but preclude assessing tissue viability by metabolite determinations. The intravenous injection of the diffusible dye, neutral red, after middle cerebral artery occlusion (MCAO) in the rat permits the visualization of zones of altered perfusion, not only during sectioning but also after lyophilization. The staining pattern delineated by neutral red has been demonstrated to correspond to areas of altered flow as determined after intravenous administration of [^{14}C]iodo-antipyrine 30 sec prior to fixation in animals subjected to MCAO and neutral red injection. Thus, sampling of tissues for metabolite measurements including glucose, lactate, ATP, P-creatine and adenylates can be made reliably with reference to the visually detectible stain intensity. When 2 ml of 4% neutral red solution was infused 30 min prior to *in situ* fixation, three major regions of neutral red intensity were evident after 1, 2.5 and 6.5 hours of occlusion. A blanched region in both the ipsilateral cerebral cortex and striatum was low in energy reserves and high in lactate, which undoubtedly reflects the ischemic focus. In contrast, most of the contralateral hemisphere, as well as that portion on the ipsilateral side served by the anterior cerebral circulation, exhibited relatively intense neutral red staining and a metabolite profile typical of a brain from a control rat. In general, the neutral red distribution and the metabolite levels in these 2 regions did not change between 1 and 6.5 hours of occlusion. There was, however, a third region of patchy neutral red staining which encapsulated the ischemic core. While the size of this intermediate zone was quite small in the striatum, it was substantial in the cerebral cortex. Discrete metabolic analysis of this region showed that: 1) an energy imbalance in the tissue had occurred, as indicated by a significant

decrease in total adenylates, 2) the metabolic condition of this region was not uniform throughout the region, as indicated by increasing high-energy phosphate levels progressing from the medial to the lateral boundary in the cortex, and 3) the viability of the tissue tended to improve with time following the occlusion. The use of neutral red permitted for the first time the direct sampling of tissue on the basis of perfusion. This technique in combination with other biochemical and physiological methods should provide a greater understanding of the complexities of focal ischmeia.

3:05 COFFE BREAK

3:20 THE ACADEMY AWARD PRESENTATION

3:50

10. MICROSURGICAL ENDARTERECTOMY UNDER BARBITURATE PROTECTON: A PROSPECTIVE STUDY

Robert F. Spetzler, M.D., Neil A. Martin, M.D.,
Richard A. Thompson, M.D., Peter A. Randzens, M.D.
and Lisa Wilkinson, M.D.

Several studies have demonstrated that patients with appropriate clinical symptoms who have significant ipsilateral angiographic disease of the carotid bifuracton benefit by a decreased risk of stroke following a "successful" carotid endarterectomy. Success in this case is defined by the absence of perioperative mortality or permanent neurological morbidity. The combined rates of perioperative stroke and death have ranged in various series from 1.5% to more than 20%. The benefit of carotid endarterectomy in stroke prevention is negated when complication rates fall into the upper range. It is therefore incumbent on surgeons to make every effort to achieve and maintain complication rates that are at an irreducible minimum.

It is not clear as to how one can consistently achieve a "successful" endarterectomy. The literature is replete with articles on surgical technique, intraoperative shunting, monitoring, anesthesia, and antithrombic agents and their use in order to enhance the safety of the operation.

We are reporting a series of 200 consecutive endarterectomies performed in 180 patients where the following protocol was followed:

1. no use of an internal shunt in order to avoid trans-shunt embolization and shunt-related intimal injury;
2. barbiturate administration in every case to protect the brain during the period of ischemia accompanying carotid clamping;
3. use of the operating microscope to aid in the precise removal of atherosclerotic plaque and placement of fine sutures to close the arteriotomy without stenosis;

4. delayed or no heparin reversal and perioperative aspirin therapy to minimize thrombus formation at the endarterectomy site;
5. rigid control of postoperative hypertension to avoid intracerebral hemorrhage.

No single study can definitively identify the specific elements of management that will reduce the perioperative complication rate for any procedure to its absolute minimum. We will present the theoretical advantages of our management protocol. We have documented the safety of this protocol in practice. When carotid endarterectomy can be performed with the degree of success, and minimal incidence of serious complications, that we have demonstrated with this protocol, the procedure can be expected to have a significant positive impact on stroke incidence in appropriately selected patients.

4:00

11. ENDARTERECTOMY FOR RECURRENT CAROTID STENOSIS

David G. Piepgras, M.D. and Thoralf M. Sundt, Jr., M.D.

Among 1992 patients undergoing carotid endarterectomy between January 1972 through December 1984, 57 operations were performed in 51 patients for recurrent carotid stenosis. Thirty-four of these cases had undergone initial surgery at this institution while 23 had endarterectomy elsewhere. Fifty-two of the 57 operations were for symptomatic disease while 5 were for evidence of a progressing lesion. All operative procedures were monitored with intracerebral blood flow measurements and continuous electroencephalograms. Twenty-three patients required intraoperative shunting. There were no complications related to shunt usage or to the period of temporary occlusion in patients who did not require shunting. Recurrent stenosis was related to intimal hyperplasia in 14 cases, recurrent atherosclerosis with interluminal thrombi or degenerated plaque in 27, unexplained soft thrombus in 8, proximal scarring in 6, and to aneurysms in 2.

The operative complication rate was 10.5 percent or 4 times the risk of surgery for primary atherosclerosis at this institution. Complications were attributed primarily to intra-operative and postoperative thromboembolic events related to apparent increased thrombogenicity of these vessels. The highest complication rate occurred in the group of patients undergoing surgery for thrombotic material in the internal carotid artery without underlying atherosclerosis. There were no neurological complications in the group with myointimal hyperplasia.

Our experience suggests that on-lay patch grafting without endarterectomy should be used in patients with myointimal hyperplasia.

Patients with complicated recurrent atherosclerosis can be treated with endarterectomy and patch grafting, but interposition vein grafts should be considered in cases in which the vessels are extensively damaged by the recurrent plaque. Interposition vein grafts are recommended for cases with an unexplained thrombus at the site of the previous endarterectomy.

4:30

12. COMPARISON OF SUTURE AND CLIP FOR MICROVASCULAR ANASTOMOSIS

W.M. Kirsch, M.D., Y.H. Zhu, M.D., R. Cushman, K. Becker, M.D., C. Kirsch, G. Brion, W. McCabe, M. Kornfeld, M.D., L. Saland, Ph.D., and V.R. Cooper, M.D.

Two fundamentally different surgical techniques for end-to-end microvascular anastomoses have been compared repairing adult rat femoral arteries (O.D. 0.8 to 1.0 mm) and veins (1.5 mm). Conventional anastomosis with 10-0 nylon (atraumatic needle) has been compared to microclip closure everting endothelium without transgression of the intimal surface. Silver microclips 700μ long, 150μ thick, cinch the vessel margins at a standard 25μ aperture. The following parameters have been compared: long and short term patency rate; procedure duration; light, scanning and transmission electron microscopic appearance of the anastomosis; and incidence of false aneurysm at the anastomotic site. Results are given below. (n refers to number of vessels in each group.)

	Veins		Arteries	
	Sutures n=36	**Clip n=37**	**Sutures n=36**	**Clip n=37**
Patency rate				
0-7 days	84%	84%	97%	100%
7-60 days	75%	92%	97%	100%
False aneurysms	0%	0%	25%	47%

Suture anastomosis (10-12 sutures) takes 30 minutes, whereas the same number of clips requires 5-8 minutes. Venous anastomosis by clip is remarkably facile. The intimal coaption provided by the clip results in rapid endothelial coverage without a foreign body within the vascular lumen. The microclip technique obviates adventitial stripping, but is associated with a significantly higher incidence of false aneurysms. False aneurysm incidence appears related to the dimensions of clip closure. The surgical technique of microclip application is illustrated by a videotape.

4:50

13. **DERMAL SINUS TRACT**

William R. Cheek, M.D. and John P. Laurent, M.D.

Dermal sinuses are tubular tracts that extend internally from a defect in the integument. They may extend all the way to the spinal cord or brain. They are congential in origin and occur in the midline at various locations. They have been known to occur in the occipital region, the dorsum of the nose and all levels of the spine with the exception of the lower sacral or sacrococcygeal areas. They are considered a form of dysraphism. Because tracts in all these locations probably have a similar embryologic origin, the authors felt it would be helpful to review a series of patients with all types of dermal sinuses rather than those confined to one location, as is frequently done with reviews in the literature. Thirty patients with sinuses of nasal, occipital and spinal origin, treated by the authors between 1972 and 1984 are presented. There were 15 sinuses in a spinal location, 4 in an occipital location and 11 in a nasal location. Physical and roentgenographic findings including polytomography are presented. The termination of each lesion is detailed, as they vary from superficial to dermoid lesions within the central nervous system. The outcome of these patients as well as complications will be presented. The authors will make recommendations relative to diagnostic procedures for workup of sinuses in each location, as well as appropriate surgical therapy.

Tuesday, October 29

SCIENTIFIC SESSION III
MODERATOR-BENNETT M. STEIN, M.D.

8:30 **SPECIAL LECTURE - STANLEY APPEL, M.D.**
"ALZHEIMER'S DISEASE: THE POTENTIAL ROLE OF NEUROTROPIC FACTORS"

9:30

14. **CLINICAL APPICATION OF MORPHINE PUMPS FOR THE RELIEF OF PAIN ASSOCIATED WITH ADVANCED MALIGNANCY**

James T. Robertson, M.D.

Since 1982, the author has had experience with the placement of over 50 implantable pumps for the intrathecal administration of morphine in an attempt to relieve pain associated with advanced malignant disease. Patient selection employs test doses of morphine sulfate into the lumbar subarachnoid space and, if immediate relief occurs and lasts for

six hours or more on one or two occasions, the patient becomes a candidate for this form of pain relief therapy. There have been no side effects associated with the test dose application and apnea monitoring has been infrequent. Standard morphine sulfate has been used for this test dose procedure.

Subsequent to positive test dose results, two types of implantable pumps have been utilized. The first is the Infusaid pump which has a constant rate of flow based on a bellows principle. A second type of pump was recently introduced by Medtronics which is programmable and allows higher concentrations which will flow over greater lengths of time. Both pumps have been satisfactory for the application of morphine by the intrathecal route. The advantages and disadvantages of the pumps will be discussed.

Our best results have occurred with pain below the diaphragm, particularly with carcinoma of the cervix and colon carcinoma. In selected patients, the results have been very satisfactory and followup outpatient filling of the pumps have created no untoward difficulty.

There has been an extremely low morbidity and no mortality of the procedure.

9:50

15. THE EFFICACY OF CINGULOTOMY FOR THE TREATMENT OF CHRONIC PAIN

H. Thomas Ballantine, Jr., M.D., Elizabeth K. Thomas, Ed.D., and Karl W. Swann, M.D.

Bilateral stereotactic anterior cingulotomy has been employed at the Massachusetts General Hospital since 1962 for the treatment of intractable psychiatric illnesses and chronic pain. As of July 1, 1985, 683 procedures had been performed on 458 patients. No deaths have resulted from the operations. There have been two major complications in the psychiatric patients, acute subural hematomas with right hemiparesis; one was transient, the other persistent. Except in these two patients, intellectual function has not been impaired.

During this 23 year period, 133 patients have undergone cingulotomy for the treatment of chronic pain; 35 suffered from terminal cancer and 98 from variety of non-malignant conditions. One hundred and twenty-three patients are the subject of this report.

Severe, constant disabling pain, refractory to all commonly accepted treatment methods, constituted the primary indication for cingulotomy. The presence, however, of a clear cut depression was also a factor favoring cingulotomy as the operation of choice.

The standard operative approach has been as follows: bilateral burr

holes are placed 9.5 cm. posterior to the nasion, followed by air ventriculography. Using the lateral ventricles as landmarks, bilateral cingulate heat lesions designed to be 1 cm. in diameter and 2 cm. in vertical length are placed from 0 to 4 cm. posterior to the tips of the anterior horns.

Of the 35 patients with cancer pain, 25 lived three months or less. During that time, 20 of the 35 (57%) were felt to have obtained moderate to complete relief of intractable pain. Of the ten who survived more than three months, pain relief was sustained in two.

The 98 patients with chronic, disabling pain of non-malignant origin had had multiple operations. Ninety-one of the patients were 1 to 20 years postoperative, 3 could not be traced, but information on the remaining 88 has been updated to April, 1985.

The following categories describing the locus or "cause" of the chronic pain have been employed:

Locus or "Cause" of Pain		Patients
Low Back		58 (1 lost to follow-up)
Abdomen and Flank		7 (1 lost to follow-up)
Unknown Etiology		6
Miscellaneous:		20
Herpetic	4	
Headache	3	
Thalamic	3	
Facial Neuralgia	1	
Phantom Limb	5	
Tabetic & "Spinal"	2	
Upper Extremity (Trauma)	2 (1 lost to follow-up)	

Our postoperative evaluation placed each of the patients in one of five categories related to the degree of pain relief and return to "normal function". Patients in the top three categories had moderate to complete relief of pain, were functioning from 40% to 100% of "normal" and medication intake varied from non-narcotic analgesic and psychotropic drugs to abstinence. Patients in these three categories were thought to have obtained "worthwhile improvement"

The 58 patients with back pain suffered from what is commonly termed the "failed back syndrome". Worthwhile improvement was documented postoperatively in 37 (65%) of them.

We categorized the seven patients with abdomen and flank pain as "failures of abdominal surgery". Of the six patients followed in this study, five sustained worthwhile postoperative improvement in their symptoms.

Of the six patients with pain of undetermined etiology, two had marked to moderate pain relief and improvement in their symptoms.

The 19 patients in the "miscellaneous" category showed worthwhile improvement in only 32% of the cases, but the numbers in each of the subcategories are too small to suggest more than trends.

The results of this study indicate that cingulotomy for relief of the chronic pain of "the failed back syndrome", secondary to multiple operative interventions and/or arachnoiditis, carries a very favorable risk/benefit ratio. For this reason it is felt to be superior to such operative interventions as spinal nerve root transection and cordotomy.

10:10 COFFEE BREAK

10:40

16. MANAGEMENT OF PINEAL REGION TUMORS

Derek A. Bruce, M.D.

Pineal region tumors continue to trigger controversy among neurosurgeons. There are still strong feelings on the needs for sugical biopsy, the possibilities for sugical excision and the appropriate follow-up therapy. In childhood where such post-surgical therapies as radiation and chemotherapy may be life-saving and cure tumors, but also may be detrimental to neurocognitive development, it is extremely important to use only as much therapy as is needed to cure the particular tumor. Over the last eight years we have operated upon 38 pineal region tumors of childhood. This constitutes approximately ten percent (10%) of the pediatric tumor population, a figure considerably higher than has previously been reported. These children have all been operated upon using a modification of Dandy's interhemispheric approach, which we feel is more appropriate for children than the suboccipital supracerebellar approach. Pros and cons of this approach as it applies to children will be discussed, particularly the benefits of blood pressure control, preventing heat loss and comfort for the surgeon. One of the useful offshoots of interhemispheric surgery has been the ability to open the back of the IIIrd ventricle and, thus, in only approximately fifteen percent of patients has it been necessary to insert a shunt.

The pathology of the tumors is quite different from what has previously been reported in the pathology literature. While there were fourteen germ-cell line tumors out of 38, only 6 of these tumors were true germinomas, the other 8 being embryonal cell or choriocarcinomas. There was an almost equal number of primary pineal tumors (pineocytomas and pineoblastoma), accounting for 12 of the 38 tumors. The next largest group were of exophytic glial tumors followed by ganglioneuroblastoma, teratoma, primitive neuroectodermal tumors, etc. Mortality was clearly related to tumor type and now with improved chemotherapy

for the embryonal cell tumors and with many chemotherapeutic possibilities available for the treatment of brain tumors in childhood, we feel an appropriate diagnosis must be made in the child prior to commencement of therapy. The old adage that 2,000 rad of radiation produces marked shrinking of only the germinoma cell tumor is, in fact, untrue in our experience. We have seen embryonal cell tumors also shrink with equal rapidity. We have also seen PNETs in this area shrink rapidly following radiation therapy.

This paper will argue that current best therapy for the child with a pineal region tumor is tissue diagnosis, radical debulking if possible followed by appropriate local or axis radiation therapy with or without chemotherapy based on the pathology. At the present time, sixty-six percent (66%) of the children are alive and functioning well.

11:00

PRESIDENTAL ADDRESS - THOMAS W. LANGFITT, M.D.
"THE PRACTICE OF NEUROSURGERY IN A MANAGED HEALTH CARE SYSTEM"

12:00 LUNCH

Wednesday, October 30

SCIENTIFIC SESSION IV
MODERATOR-MARTIN H. WEISS, M.D.

8:30

17. INTERSTITIAL BRAIN TUMOR TREATMENT WITH RADIATION ENHANCEMENT

Joseph H. Goodman, M.D., Reinhard Gahbauer, M.D.,
Ralph Fairchild, Ph.D., Nancy Clendenon, Ph.D.
Christos Kannelitsas, Ph.D., and William E. Hunt, M.D.

Halogenated pyrimidines are effective as radiosensitizing agents. The mechanism of action involves interference with utilization of thymidilic acid and results in incorporation of a thymidine analogue into DNA. Intravenous infusion of iododeoxyuridine (IUDR) can achieve a 5% thymidine replacement at which levels radiosensitization is obseved. An additional dose enhancement can be obtained by generating K and L shell vacancies in the stable iodine nucleus through a photoelectric process using appropriate low kilovoltage irradiation. Furthermore, Auger electron cascades are initiated in the process. These are biologically very effective due to the dense ionization produced. Samarium 145 is a low kilovoltage gamma emmitter capable of providing photoelectric

energies just above the K-absorption edge of iodine. Interstitial implantation of samarium sources confines the destructive effects to sensitized tumor tissue. Since CNS cells do not take up IUDR appreciably, there is relative sparing of normal cells within brain.

This concept, proposed by Fairchild, is ideally suited to the treatment of brain tumor for several reasons. Up to 20% thymidine replacement can be achieved experimentally in proliferating tissue with acceptable toxicities. Experimental brain tumors localize IUDR with negligible iodine detectable in adjacent normal brain. IUDR is a more potent radiation sensitizer than bromodeoxuridine, is less toxic and can be administered intravenously rather than intraarterially. Stereotactic implantation of samarium sources prevents the attendant morbidity associated with conventional external irradiation by confining the high dose field to the tumor thereby sparing normal DNA containing tissues of the head and neck.

Cellular and animal studies are in progress to determine the potential effectiveness of IUDR as an enhancing agent. Calibration of prepared samarium 145 seeds indicates suitable energy sources are available for implantation. Experimental data and techniques for initiation of clinical trials are presented.

8:50

18. MANY APPARENTLY NON-FUNCTIONING PITUITARY ADENOMAS MAY SECRETE SUBUNITS OF LH, FSH, OR TRH

Peter McL. Black, Dora Hsu, E. Chester Ridgway, Jr.,
Anne Klibanski, Larry Jameson, and Nicholas T. Zervas

In the past, most attention in pituitary adenomas has been paid to prolactin (PRL), growth hormone (GH), and adrenocorticotropic hormone (ACTH). Our data suggest that many pituitary tumors thought to have no hormone products may in fact be secreting portions of the glycoprotein hormones LH, FSH, and TSH. These hormones are composed of an alpha subunit which is common to all three hormones and a beta subunit which distinguishes them.

We stained thirty-five apparently non-functioning pituitary adenomas with antibody to the beta subunits of FSH, LH, and TSH as well as for prolactin, growth hormone, and the alpha subunit of these hormones. Twenty-three out of thirty-five (65%) had positive staining for at least one of these moeities. One out of 35 stained positively for only the alpha subunit with no beta or other hormones; seven had both alpha and beta subunits; ten out of thirty-five had beta subunits without alpha. Five of the thirty-five had prolactin and growth hormone staining as well as glycoprotein hormones.

These data suggest that some apparently non-functioning pituitary

tumors may produce fragments or subunits of glycoprotein hormones. This may not be clinically evident because the beta subunits are biologically inactive when they are not combined with the alpha subunits; however, it may render these tumors amenable to pharmacological manipulation. Further studies on the biosynthesis and secretion of the free fragments of the glycoprotein hormones in these tumors are necessary to extend these observations.

9:10

19. V NERVE NEURINOMAS: PRESENTATION, TREATMENT, AND DEFICITS

Kalmon D. Post, M.D.

Five cases of V nerve neurinomas will be presented. Their symptoms and signs will be reviewed, particularly with regard to the implications for sensory and motor V nerve function.

A review of the literature will be done.

Surgical approaches including subtemporal, transtentorial, suboccipital, or combined procedures will be discussed and evaluated.

An analysis of the clinical deficit caused by a motor V dysfunction will be reviewed in depth.

9:30

20. TUMORS OF THE TRIGEMINAL COMPLEX: AN ANALYSIS OF THE DIFFERENT PRESENTATIONS ASSOCIATED WITH PRIMARY AND METASTATIC LESIONS

Willis E. Brown, Jr., M.D., Jim L. Story, M.D., Robert E. Abraham, M.D., G. Richard Holt, M.D., and Douglas E. Mattox, M.D.

Primary and metastatic tumors of the trigeminal complex are rare lesions. Patients usually present with trigeminal symptoms and signs; however, some patients do not present in the expected manner. We have collected seven patients (four with trigeminal schwannomas and three with metastatic carcinoma that metastasized intracranially along the mandibular division) who illustrate the variable presentation of these tumors: three of the four patients with trigeminal schwannoma presented in an atypical manner; the three patients with metastatic carcinoma had characteristic trigeminal dysfunction. Our analysis includes our own four cases of trigeminal schwannoma and 79 cases drawn from the literature and reveals that only 60% of the patients with trigeminal schwannoma present with trigeminal dysfunction. On the other hand, an

analysis of our three cases of metastatic carcinoma and 80 additional cases reported elsewhere confirms that trigeminal involvement with carcinoma extending from the head and neck can be expected to present with typical trigeminal symptoms and signs.

9:50 COFFEE BREAK

10:10

SPECIAL LECTURE—R. NICK BRYAN, M.D., Ph.D.
"NMR - THEORETICAL AND PRACTICAL"

11:10

21. **THE CLINICAL BASIS FOR POSTERIOR SPINAL INSTRUMENTATION**

Stewart B. Dunsker, M.D.

Over the past decade the ability to correct the unstable spine has advanced. However, with different rods and with various classifications of injury, it has become difficult to decide on the appropriate approach. After reviewing the literature we believe that the 3 column classification of spinal structure as proposed by Denis lends itself to the best understanding of the various types of trauma. We will review the advantages and limitations of segmental spinal instrumentation (SSI), such as that proposed by Luque, and the distraction and compression systems of Harrington.

The key to selecting the appropriate method of stabilization lies in evaluating the integrity of the middle column of the spine which is the posterior half of the vertebral body, its attached disc and ligaments. We will classify the various types of injury and present case histories to illustrate the preferred method of treatment.

11:30

22. **LONG TERM FOLLOW-UP OF PATIENTS TREATED WITH CHEMONUCLEOLYSIS**

Robert J. Maciunas, M.D. and Burton M. Onofrio, M.D.

The long term clinical outcome is evaluated for 268 patients after chymopapain chemonucleolysis for radicular complaints referrable to documented intervertebral disk disease. Ninety-two percent were available for followup at ten years' time. No complications due to chymopapain toxicity were observed. 80.1 percent of patients were relieved of their presenting radicular leg pain and 75.1 percent were employed at a capacity equal to or more strenuous than before injection. Chemonu-

cleolysis is demonstrated to be a safe and effective treatment modality, with long term results which compare favorably with those of similarly selected patients undergoing open surgical procedure. In those patients who fail chymopapain therapy, the outcome of subsequent open surgical procedures is not necessarily compromised by prior chemonucleolysis. A higher rate of failure and subsequent surgical intervention is seen in those patients with injections performed soon after an unsuccessful open procedure on the same side and at the same interspace; with compensation or litigation pending; with a history of work-related injury; with employment involving heavy manual labor or extensive driving; and with preinjection spine x-rays indicating retrograde spondylolisthesis.

11:50	FINAL ANNOUNCEMENTS
12:00	ADJOURN

Paul M. Lin 1955
Hubert L. Rosomoff 1956
Byron C. Pevehouse 1957
Norman Hill 1958
Jack Stern 1959
Robert Ojemann 1960
Lowell E. Ford 1962
Charles H. Tator 1963
Earle E. Crandall 1964
Stephen Mahaley, Jr. 1965
Chun Ching Kao 1966
John P. Kapp 1967
Yoshio Hosobuchi 1968
Gary G. Ferguson 1970
Richard L. Pressley 1971
David G. McLeone 1972
Arden F. Reynolds, Jr. 1973
Richard L. Rapport 1974
Andrew G. Shetter 1975
John F. Howe 1976
Howard W. Blume 1977
Howard J. Senter 1978
Elisabeth M. Post 1979
David Dubuisson 1980
Dennis A. Turner 1981
Marc R. Mayberg 1982
David S. Baskin 1983
Kevin J. Kiwak 1984

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Francis Murphey	1942	William S. Keith	1942
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A. Earl Walker	1944	Rupert B. Raney	1944
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William S. Keith	1947	John Raaf	1947
Howard A Brown	1948	Arthur R. Elvidge	1948
John Raaf	1949	F. Keith Bradford	1949
E. Harry Botterell	1950	David L. Reeves	1950
Wallace B. Hamby	1951	Henry G. Schwartz	1951
Henry G. Schwartz	1952	J. Lawrence Pool	1952
J. Lawrence Pool	1953	Rupert B. Raney	1953
Rupert B. Raney	1954	David L. Reeves	1954
David L. Reeves	1955	Stuart N. Rowe	1955
Stuart N. Rowe	1956	Jess D. Herrmann	1956
Arthur R. Elvidge	1957	George S. Baker	1957
Jess D. Herrmann	1958	Samuel R. Snodgrass	1958
Edwin B. Boldrey	1959	C. Hunter Shelden	1959
George S. Baker	1960	Edmund Morrissey	1960
C. Hunter Sheldon	1961-62	Donald F. Coburn	1961-62
Samuel R. Snodgrass	1963	Eben Alexander, Jr.	1963
Theodore B. Rasmussen	1964	George L. Maltby	1964
Edmund J. Morrissey	1965	Robert Pudenz	1965
George Maltby	1966	Francis A. Echlin	1966
Guy L. Odom	1967	Benjamin Whitcomb	1967
James G. Galbraith	1968	Homer S. Swanson	1968
Robert H. Pudenz	1969-70	Augustus McCravey	1969-70
William B. Scoville	1971	Edward W. Davis	1971
Robert L. McLaurin	1972	John R. Green	1972
Lyle A. French	1973	George J. Hayes	1973
Benjamin B. Whitcomb	1974	Richard L. DeSaussure	1974
John R. Green	1975	Ernest W. Mack	1975
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Arthur A. Ward	1978	Robert G. Fisher	1978
Robert B. King	1979	H.T. Ballentine, Jr.	1979
Eben Alexander, Jr.	1980	George Ehni	1980
Joseph Ransohoff II	1981	Courtland H. Davis, Jr.	1981
Byron C. Pevehouse	1982	John F. Mullan	1982
Sidney Goldring	1983	Hugo Rizzoli	1983
Russel H. Patterson, Jr.	1984	Thomas W. Langfitt	1984
Thomas Langfitt	1985	E B Hendrick	1985
Phanor Perot	1986	Griff Harsh III	1986
Shelley Chou	1987		1987

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A. Earl Walker	1941-43	Robert L. McLaurin	1958-62
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Wallace B. Hamby	1948-50	Robert G. Fisher	1966-68
Theodore B. Rasmussen	1951-53	Byron C. Pevehouse	1969-72

PAST SECRETARIES

Byron C. Pevehouse	1973
Russel H. Patterson, Jr.	1974-76
Phanor L. Perot, Jr.	1977-80
John T. Garner	1981-83
James T Robertson	1984-

PAST TREASURERS

Russel H. Patterson, Jr.	1973
Phanor L. Perot, Jr.	1974-76
John T. Garner	1977-80
James T. Robertson	1981-83
Nicholas Zervas	1984-

Hotel Netherlands Plaza, Cincinnati, Ohio .... October 28-29, 1938
Roosevelt Hotel, New Orleans, Lousisana .... October 27-29, 1939
Tudor Arms Hotel, Cleveland, Ohio ..... October 21-22, 1940
Mark Hopkins Hotel, San Francisco, and Ambassador Hotel, Los Angeles, California ..... November 11-15, 1941
The Palmer House, Chicago, Illinois ..... October 16-17, 1942
Hart Hotel, Battle Creek, Michigan ..... September 17-18, 1943
Ashford General Hospital, White Sulphur Springs, West Virginia ..... September 7-9, 1944
The Homestead, Hot Springs, Virginia ..... September 9-11, 1946
Broadmoor Hotel, Colorado Springs, Colorado ... October 9-11, 1947
Windsor Hotel, Montreal, Canada ..... September 20-28, 1948
Benson Hotel, Portland, Oregon ..... October 25-27, 1949
Mayo Clinic, Rochester, Minnesota ..... September 28-30, 1950
Shamrock Hotel, Houston, Texas ..... October 4-6, 1951
Waldorf-Astoria Hotel, New York City September 29-October 1, 1952
Biltmore Hotel, Santa Barbara, California ..... October 12-14, 1953
Broadmoor Hotel, Colorado Springs, Colorado ... October 12-14, 1954
The Homestead, Hot Springs, Virginia ..... October 27-29, 1955
Camelback Inn, Phoenix, Arizona ..... November 8-10, 1956
✓The Cloister, Sea Island, Georgia ..... November 11-13, 1957
The Royal York Hotel, Toronto, Canada ..... November 6-8, 1958
Del Monte Lodge, Pebble Beach, California ... October 18-21, 1959
Copley Sheraton Plaza, Boston Massachusetts ... October 5-8, 1960
✓Royal Orleans, New Orleans, Louisiana ..... November 7-10, 1962
✓El Mirador, Palm Springs, California ..... October 23-26, 1963
Elected The Key Biscayne, Miami, Florida ..... November 11-14, 1964
✓Terrace Hilton Hotel, Cinncinnati, Ohio ..... October 14-16, 1965
✓Fairmont Hotel & Tower, San Fransicso, California ..... October 17-19, 1966
✓The Key Biscayne, Miami, Florida ..... November 8-11, 1967
✓Broadmoor Hotel, Colorado Springs, Colorado ... October 6-8, 1968
S-T ✓St. Regis Hotel, New York City ..... September 21, 1969
S-T ✓Camino Real Hotel, Mexico City ..... November 18-21, 1970
S-T ✓Sahara-Tahoe Hotel, Stateline, Nevada ..... September 26-29, 1971
S-T ✓New College, Oxford, England ..... September 4-7, 1972
S ✓Huntington-Sheraton Hotel, Pasadena, California ..... November 14-17, 1973
✓Southhampton Princess Hotel, Southhampton, Bermuda ..... November 6-9, 1974
✓The Wigwam (Litchfield Park), Phoenix, Arizona November 5-8, 1975
✓The Mills Hyatt House, Charleston, South Carolina ..... November 10-13, 1976
✓Mauna Kea Beach Hotel, Kamuela, Hawaii .... November 2-5, 1977

Hotel Bayerischer Hof, Munich, Germany .... October 22-25, 1978
Hyatt Regency, Memphis, Tennessee ..... November 7-10, 1979
Waldorf Astoria, New York, New York ..... October 1-4, 1980
Sheraton Plaza, Palm Spring, California ..... November 1-4, 1981
Ritz-Carlton Hotel, Boston, Massachusetts .... October 10-13, 1982
The Lodge at Pebble Beach, California ..... October 23-26, 1983
The Homestead, Hot Springs, Virginia ..... October 17-20, 1984
Houston, Texas oct. 29-30 1985
Sea Island, Ga. Nov. 5-8 1986
San Antonio Oct 7-10 1987
Cincinnati Sept 14-17 1988
WFNS oct 8-13, 1989 New Dehli -

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26 Rue D Auriol
31 Toulouse, France

VALENTINE LOGUE — 1974
16 Rowan Road
Hammersmith
London W6 7DU
U.K.

GOSTA NORLEN — 1973
Neurokirurgiska Kliniken
Sahlgrenska Sjukhus
Goetborg, SV Sweden

KEIJI SANO — 1975
Dept. of Neurosurgery
School of Medicine
University of Tokyo
Tokyo, Japan

SENIOR MEMBERS — ELECTED

EBEN ALEXANDER, JR. (BETTY) — 1913 57 / 1950
Bowman-Gray
School of Medicine
of Wake Forest University
Winston-Salem, North Carolina 27103

GEORGE S. BAKER (ENID) — 1905 35 / 1940
607 North Litchfield Road
P.O. Box 1234
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H. THOMAS BALLANTINE, JR. (ELIZABETH) — 1912 39 / 1951
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Boston, Massachusetts 02114

EDWIN B. BOLDREY (HELEN) — 1906 35 / 1941
University of California Hospital
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San Francisco, California 94143

E. HARRY BOTTERELL (MARGARET) — 1906 32 / 1938
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Lexington, Kentucky 40503

DONALD F. COBURN (ELLIE) — 1907 31 / 1938
The Plaza 812
1303 Delaware Avenue
Wilmington, Delaware 19806

EDWARD W. DAVIS (BARBARA) — 1913 36 / 1949
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Portland, Oregon 97213

RICHARD DE SAUSSURE (PHYLLIS) — 1917 45 / 1962
920 Madison Avenue
Memphis, Tennessee 38103

R.M. PEARDON DONAGHY (FRANCES) — 1910 60 / 1970
P.O. Box 5035
RDI-Horn of the Moon Road
Montpelier, Vermont 05602

CHARLES G.DRAKE (RUTH) — 1958
University Hospital
339 Windermere Road
London, Ontario, Canada N6G 2K3

FRANCIS A. ECHLIN (LETITIA) — 1944
P.O. Box 342
New Paltz, New York 12561

DEAN H. ECHOLS (FRAN) — 1904 Founder 34
Ochsner Clinic
1514 Jefferson Highway
New Orleans, Lousisana 70121

GEORGE EHNI (LARI) — 1914 50 / 1964
6560 Fannin St., ñ1250
Scurlock Tower
Houston, Texas 77030
Deceased 9/2/96

THEODORE C. ERICKSON (MARTHA)
425 North Livington St.
Madison, Wisconsin 53703

1906 34
1940

ROBERT FISHER (CONSTANCE)
909 Park Avenue
Plainfield, New Jersey 07060

1917 39
1956

JOHN D. FRENCH (DOROTHY)
The Center for the Health Sciences
University of California
Los Angeles, California 90024

1911 40
1951
Disabled

LYLE A. FRENCH (GENE)
University of Minnesota
Medical Center
Minnieapolis, Minnesota 55455

1915 39
1954

JAMES G. GALBRAITH (PEGGY)
2515 Crest Road
Birmingham, Alabama 35223

1914 33
1947

PHILIP D. GORDY (SILVIA)
1727 East Second Street
Casper, Wyoming 82601

1918 SC
1968

EVERETT G. GRANTHAM (MARY CARMEL)
234 East Gray Street
Louisville, Kentucky 40202

1912 30
1942

JOHN R. GREEN (GEORGIA)
Barrow Neurological Institute
2910 W. 3rd Avenue
Phoenix, Arizona 85013

1915 35
1953

JAMES GREENWOOD, JR. (MARY)
1839 Kirby Drive
Houton, Texas 77019

1907 45
1952
Deceased ?

WALLACE B. HAMBY (ELEANOR)
2001 N.E. 47th Court
Fort Lauderdale, Florida 33308

1938

JESS D. HERRMANN (MARY JO)
Post Office Box 135
Mountain Pine, Arkansas 71956

1907 41
1948

WILLIAM S. KEITH (ELEANOR) — Founder
55 St. Leonards Crescent
Toronto, Ontario, Canada M4N 3A7

ROBERT S. KNIGHTON (LOUISE) — 1914 52 / 1966
9388 Avenida San Tinetto
Cherry Valley, California 92223

WILLIAM M. LOUGHEED (GRACE) — 1962
Room 219, 7th Floor
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101 College Street
Toronto, Canada M5G 1L7

JOHN J. LOWREY (CATHERINE "Katy") — 1913 52 / 1965
P.O. Box 4302
Kawaihae, Hawaii 96743

GEORGE L. MALTBY (ISABELLA "Sim") — 1909 33 / 1942
470 Black Point Road
Scarsborough, Maine 04074

FRANK MAYFIELD, M.D. — Founder 1908
506 Oak Street
Cincinnati, Ohio 45219

AUGUSTUS McCRAVEY (HELEN) — 1910 34 / 1944
1010 East Third Street
Chattanooga, Tennessee 37403

WILLIAM F. MEACHAM (ALICE) — 1913 34 / 1952
Vanderbilt University Hospital
Division of Neurosurgery
Nashville, Tennessee 37232

EDMUND J. MORRISSEY (KATE) — 1941
909 Hyde Street, Suite 608
San Francisco, California 94109
Deceased 2/8/86

FRANCIS MURPHEY (MARGE) — Founder 1906
3951 Gulf Shores Road
Apt. 1102
Naples, Florida 33940

GUY L. ODOM (MATALINE) — 1911 1946 35
2812 Chelsea Circle
Durham, North Carolina 27707

J. LAWRENCE POOL (ANGELINE) — 1940
Box 40
West Cornwell, Connecticut 06796

ROBERT H. PUDENZ (RITA) — 1911 1943 32
574 Garfield Avenue
South Pasadena, California

JOHN RAAF (LORENE) — 1905 Founder
1120 N.W. 20th Avenue, ñ100
Portland, Oregon 97209

AIDEN A. RANEY (MARY) — 1911 1946 35
2010 Wilshire Blvd.
Suite 203
Los Angeles, California 90057

THEODORE B. RASMUSSEN (CATHERINE) — 1947
29 Surrey Drive
Montreal, Quebec, Canada H3P 1B2

RICHARD C.SCHNEIDER (MADELEINE) — 1913 1970 57 Deceased
Room 3605
Kresge Medical Research Bldg.
University of Michigan Medical Center
Ann Arbor, Michigan 48109

HENRY G. SCHWARTZ (REEDIE) — 1909 1942 33
Barnes Hospital Plaza
Division of Neurological Surgery
St. Louis, Missouri 63110

C. HUNTER SHELDEN (ELIZABETH) — 1907 1941 34
734 Fairmont Avenue
Pasadena, California 91105

HOMER S. SWANSON (LaMYRA) — 1911 1949 38 Disabled
3649 Peachtree Road, N.E.
Unit 205
Atlanta, Georgia 30319

1910 40
1950

WILLIAM H. SWEET (ELIZABETH)
309 Goddard Avenue
Brookline, Massachusett 02146

1921 46
1967

JOHN TYTUS (VIRGINIA "Gina")
Mason Clinic
Seattle, Washington 98107

1950

ALFRED UIHLEIN (IONE)
200 First Street S.W.
Rochester, Minnesota 55901

1907 31
1938

A. EARL WALKER (AGNES)
1477 Wagontrain Drive, S.E.
Albuquerque, New Mexico 87123

1907 31
1938

EXUM WALKER (NELLE)
490 Peachtree Street, N.E.
Atlanta, Georgia 30308

1912 31
1943

THOMAS A. WEAVER, JR. (MARY)
146 Wyoming Street
Dayton, Ohio 45409

deceased

1908 39
1947

BENJAMIN B. WHITCOMB (MARGARET)
50 Union Street
Ellsworth, Maine 04605

ACTIVE MEMBERS	ELECTED
JAMES I. AUSMAN (CAROLYN) Henry Ford Hospital 2799 West Grand Blvd. Detroit, Michigan 48202	1937 41 1978
GILLES BERTRAND (LOUISE) Montreal Neurological Institute 3801 University Street Montreal Quebec, Canada H3A 1B4	1967
ROBERT S. BOURKE (MARLENE) ~~Division of Neurosurgery~~ ~~Albany Medical College~~ ~~Albany, New York 12208~~	1935 48 1983
JERALD S. BRODKEY (ARIELLE) 24755 Chagrin Boulevard Suite 205 Beachwood, Ohio 44122	1934 43 1977
Willis ~~WILLIAM~~ E. BROWN, JR. Division of Neurosurgery 7703 Floyd Curl Drive San Antoino, Texas 78284	1938 46 1984
DEREK BRUCE 34th-Civic Ctr. Blvd Division of Neurosugery Philadelphia, Pennsylvania 19014	1984
WILLIAM A BUCHHEIT, M.D. 3401 North Broad Street Philadelphia, Pennsylvania 19140	1933 47 1980
PAUL H. CHAPMAN (TANSY) Department of Neurosurgery Massachusetts General Hospital Boston, Massachusetts 02114	1938 45 1983
SHELLY CHOU (JOLENE) University of Minnesota Medical Center Minneapolis, Minnesota 55455	1924 50 1974

GALE G. CLARK (MARION) 1916 54
University of California Medical Center 1970
San Francisco, California 94143

W. KEMP CLARK (FERN) 1925 45
5323 Harry Hines Blvd. 1970
Dallas, Texas 75235

WILLIAM F. COLLINS, JR. (GWEN) 1924 39
Yale University School of Medicine 1963
333 Cedar Street
Nw Haven, Connecticut 06510

EDWARDS S. CONNOLLY (ELISE) 1934 39
Ochsner Clinic 1973
1514 Jefferson Highway
New Orleans, Louisiana 70018

JAMES W., CORRELL (CYNTHIA) 1919 47
710 West 168th Street 1966
New York, New York 10034

COURTLAND H. DAVIS, JR. 1921 46
Bowman-Gray School of Medicine 1967
Winston-Salem, North Carolina 27103

DONALD F. DOHN (CAROLYN) 1925 43
Singing River Neurosurgical Associates 1968
3003 Short Cut Road
Pascagoula, Mississippi 39567

STEWART B. DUNSKER (ELLEN) 1934 41
Mayfield Neurological Institute 1975
506 Oak Street
Cincinnati, Ohio 45219

Howard Eisenberg 1939 1885

WILLIAM H. FEINDEL (FAITH) 1918 41
Montreal Neurological Institute 1959
3801 University Street
Montreal, Quebec, Canada
H3A 2B4

EUGENE FLAMM (SUSAN) 1937 42
N.Y.U. Medical Center 1979
550 First Avenue
New York, New York 10016

ELDON L. FOLTZ (CATHERINE) 1919 41 1960
UCI Medical Center, Division of Neurosurgery
101 City Drive, S.
Orange, California 92668

RICHARD A.R. FRASER (SARAH ANNE) 1937 39 1976
525 East 68th Street
New York, New York 10021

JOHN T. GARNER 1931 40 1971
50 Alessandre Place
Suite 400
Pasdena, California 91105

HENRY GARRETSON (MARIANNA) 1929 44 1973
Health Sciences Center
316 MDR Bldg.
University of Louisville
Louisville, Kentucky 40292

SIDNEY GOLDRING (LOIS) 1923 41 1964
Barnes Hospital Plaza
Division of Neurosurgery
St. Louis, Missouri 63110

ROBERT G. GROSSMAN (ELLIN) 1933 54 1984
Baylor College of Medicine
6501 Fannin, A404
Houston, Texas 77030

Robert Grubb 1940 45 1985

JOHN W. HANBERY (SHIRLEY) 1919 46 1959
Division of Neurosurgery
Stanford University Medical Center
300 Pasteur Drive
Stanford, California 94305

GRIFFITH R. HARSH III, M.D. (CRAIG) 1924 56 1980
University of Alabama Medical Center
Birmingham, Alabama 94305

MAJ. GEN. GEORGE S. HAYES (CATHERINE) 1918 44 1962
MC USA
303 Skyhill Road
Alexandria, Virginia 22314

? Senior

1937 47
MARK PETER HEILBRUN 1984
Division of Neurosugery, 3B320
University of Utah Medical Center
Salt Lake City, Utah 84132

E. BRUCE HENDRICK (GLORIA) 1968
Hospital for Sick Children
555 University Avenue, Room 1502
Toronto, Ontario, Canada M5G 1X8

Senior

1942 43
Roberto Heros 1985

1941 41
CHARLES HODGE, M.D. 1982
Department of Neurosurgery
Upstate Medical Center
Syracuse, New York 13210

1936 41
JULIAN HOFF (DIANNE) 1975
Department of Neurosurgery
University of Michigan
Ann Arbor, Michigan 48104

HAROLD HOFFMAN (JO ANN) 1982
The Hospital for Sick Children
Suite 1502, 555 University Avenue
Toronto, Ontario M5G 1X8

1928 48
EDGAR M. HOUSEPIAN (MARION) 1976
710 West 168th Street
New York, New York 10032

ALAN R. HUDSON (SUSAN) 1978
St. Michaels Hospital
38 Shutter Street
Toronto, Ontario Canada
M5B 1A6

1921 51
WILLIAM E. HUNT (CHARLOTTE) 1970
Division of Neurological Surgery
University Hospital
410 West 10th Avenue
Columbus, Ohio 43210

Senior

1931 51
JOHN A. JANE, M.D. (NOELLA) 1982
Department of Neurosurgery
University Virginia
Charlottesville, Virginia 22901

John Kapp 1938 47
1985

ELLIS B. KEENER (ANN) 1926 52
915 East Lake Drive, NW 1978
Gainesville, Georgia 30506 Senior

DAVID KELLY (SALLY) 1935 40
Bowman-Gray School of Medicine 1975
Winston-Salem, North Carolina 27103

WILLIAM A KELLY (JOAN) 1927 50
Department of Neurological Surgery 1977
RI-20
University of Washington
Seattle, Washington 98195

GLENN W. KINDT (CHARLOTTE) 1930 47
Division of Neurosurgery 1977
Box C-307
University of Colorado Medical Center
4200 East 9th Avenue
Denver, Colorado 80262

ROBERT B. KING (MOLLY) 1922 36
University Hospital 1958
Upstate Medical Center
750 East Adams Street
Syracuse, New York 13210

WOLFF M. KIRSCH (MARIE—CLAIRE) 1931 40
531 Chamiso Lane, NW 1971
Albuquerque, New Mexico 87107

DAVID G. KLINE 1934 35
Louisiana State University Medical Center 1972
1542 Tulane Avenue
New Orleans, Louisiana 70012

RICHARD S. KRAMER (ROBIN) 1936 42
Duke Hospital 1978
Durham, North Carolina 27710

THEODORE KURZE 1922 45
10 Congress Street 1967
Suite 340
Pasadena, California 91105

1927 44
THOMAS W. LANGFITT (CAROLYN)
1971
Hospital of the University of Pennsylvania
34th and Spruce Streets
Philadelphia, Pennsylvania 19104

1938 45
EDWARD R. LAWS, JR. (PEGGY)
1983
Mayo Clinic
Rochester, Minnesota 55905

1920 43
RAEBURN C. LLEWELLYN (CARMEN)
1963
5640 Read Blvd.,
Suite 840
New Orleans, Louisiana 70127

1934 49
DONLIN M. LONG
1983
Department of Neurological Surgery
John Hopkins Medical School
Baltimore, Maryland 21205

1929 36
HERBERT LOURIE (BETTY)
1965
725 Irving Avenue, Suite 504
Syracuse, New York 13210

1926 50
ALFRED J. LUESSENHOP
1976
Georgetown University Hospital
Washington, D.C. 20007

1913 43
ERNEST W. MACK (BOBBIE)
1956
505 South Arlington Avenue
Suite 212
Reno, Nevada 89509

Senior ~~Senior~~

1932 40
M. STEPHEN MAHALEY, JR. (JANE)
1972
Division of Neurological Surgery
148 Clinical Sciences Bldg., U.N.C.
Chapel Hill, North Carolina 27514

1919 54
LEONARD MALIS (RUTH)
1973
1176 Fith Avenue
New York, New York 10029

1922 33
ROBERT L. McLAURIN
1955
Holmes Hospital
Eden & Bethesda Avenue
Cincinnati, Ohio 45219

JOHN F. MULLAN, (VIVIAN)
University of Chicago Clinics
Department of Neurosurgery
950 East 59th Street
Chicago, Illinois 60634

1925 38
1963

BLAINE S. NASHOLD, JR. (IRENE)
Duke University Medical Center
Durham, North Carolina 27710

1923 44
1967

FRANK E. NULSEN (GINNEY)
University Hospital of Cleveland
2074 Abington Road
Cleveland, Ohio 44106

Senior

1916 40
1956

GEORGE OJEMANN (LINDA)
6424 E. Mercer Way
Mercer Island, Washington 98040

1935 40
1975

ROBERT G. OJEMANN (JEAN)
Neurosurgical Service
Massachusetts General Hospital
Boston, Massachusetts 02114

1931 37
1968

BURTON ONOFRIO (JUDITH)
Mayo Clinic
Rochester, Minnesota 55901

1933
1975 42

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

1929 42
1971

S.J. PEERLESS (ANN)
P.O. Box 5339
Terminal A
University Hospital
London, Ontario, Canada N6A 5A5

1977

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

1928 42
1970

BYRON C. PEVEHOUSE (LUCY)
815 Eucalyptus Avenue
Hillsborough, California 94010

1927 37
1964

ROBERT W. PORTER (AUBREY DEAN)
6461 Bixby Hill Road
Long Beach, California 90815

1926 36
1962

JOSEPH RANSOHOFF II (LORI ELLEN)
New York University Medical Center
550 First Avenue
New York, New York 10016

1915 50
1965
? Senior

ALBERT L. RHOTON, JR.
University of Florida, Box J265
Department of Neurosurgery
Gainesville, Florida 32610

1932 52
1984

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

1916 57
1973

THEODORE S. ROBERTS (JOAN)
4375 Zarahemia Drive
Salt Lake City, Utah 84117

1926 50
1976

JAMES T. ROBERTSON (VALERIA)
Department of Neurosurgery
UTCHS, 956 Court Avenue
Memphis, Tennessee 38163

1931 40
1971

FREDERICK A. SIMEONE (KATE)
800 Spruce Street
Philadelphia, Pennsylvania 19107

1936 45
1981

JAMES C. SIMMONS (VANITA)
920 Madison Avenue
Memphis Tennessee 38103

1926 49
1975

BENNETT M. STEIN
710 West 168th Street
New York, New York 10034

1931 39
1970

JIM L. STORY, M.D. (JOANNE)
7703 Floyd Curl Drive
San Antonio, Texas 78284

1931 41
1972

THORALF M. SUNDT, JR. (LOIS)
200 1st Street, S.W.
Rochester, Minnesota 55901

1930 41
1971

ANTHONY F. SUSEN (PHYLLIS)
3600 Forbes Avenue
Pittsburgh, Pennsylvania 15213

1921 44
1965

RONALD R. TASKER (MARY)
Toronto General Hospital
Room 7-221E
101 College Street
Toronto, Ontario, Canada
M5G 1L7

1971

JOHN TEW JR. (SUSAN)
506 Oak Street
Cincinnati, Ohio 45219

1936 37
1973

GEORGE TINDALL (SUZIE)
Emory University School of Medicine
Division of Neurosurgery
1365 Clifton Road, N.E.
Atlanta, Georgia 30322

1928 40
1968

JOHN C. VAN GILDER, M.D. (KERSTIN)
University of Iowa Hospital
Iowa City, Iowa 55242

1935 45
1980

ARTHUR A . WARD, JR. (JANET)
Department of Neurological Surgery
RI-20
University of Washington
Seattle, Washington 98195

1916
1953 37

Senior

CLARK WATTS (PATTY)
One Hospital Drive
Ste. N522
Columbia, Missouri 65212

1938 37
1975

1936 48
1984

BRUCE K.A. WEIR
University of Alberta
Clinical Bldg.
Alberta, Canada
T6G 2S3

1939 42
1981

MARTIN H. WEISS (DEBBY)
USC Medical Center
1200 N. State Street
Los Angeles, California 90033

1920 37
1957

W. KEASLEY WELCH (ELIZABETH)
Childrens Hospital Medical Center
300 Longwood Avenue
Boston, Massachusetts 02115

1928 43
1971

LOWELL E. WHITE, JR. (MARGIE)
University of Southern Alabama
Division of Neuroscience
Mobile, Alabama 36688

1934 39
1973

ROBERT WILKINS (GLORIA)
Duke University Medical Center
Box 3807
Durham, North Carolina 27710

1929 37
1966

CHARLES B. WILSON
Department of Neurological Surgery
University of California Medical Center
Third and Parnassus
San Francisco, California 94143

1922 51
1973

FRANK WRENN (BETTY)
27 Memorial Medical Drive
Greenville, South Carolina 29605

? Senior

1935 37
1972

DAVID YASHON (MYRNA)
50 McNaughton Road
Columbus, Ohio 43213

1929 43
1972

NICHOLAS T. ZERVAS (THALIA)
Massachuetts General Hospital
Boston, Massachusetts 02114

SENIOR CORRESPONDING MEMBERS	ELECTED
KARL AUGUST BUSHE Neurochirurgischen Klinik D-8700 Wurzburg Josef-Schneider-Strasse 11 West Germany	1972
SHOZO ISHII Department of Neurosurgery Juntendo Medical College Tokyo, Japan	1975
KRISTIAN KRISTIANSEN (KARI) Oslo Kommune Uleval Sykehus Oslo, Norway	1962
WILLIAM LUYENDIJK Pr Bernhardlaan 60 Oegstgeest, The Netherlands	1973
KURT SHURMANN Director Neurochirurg Univ-Klinik Mainz Langebeskstr 1 6500 Mainz, West Germany	1978

CORRESPONDING MEMBERS	ELECTED
JEAN BRIHAYE 1 Rue Heger-Bordet B-1000 Brussels, Belgium	1975
FERNANDO CABIESES Inst. Peruano De Formento Educativo Av. Arenales 371, of. 501 Apartado 5254 Lima, Peru	1966
JUAN CARDENAS, C. Neurologo 4 Neurocirujano Av. Insurgentes Sur 594, Desp. 402 Mexico 12 D.F.	1966

JUAN C. CHRISTENSEN 1970
Ayacucho 2151 4 P
Buenos Aires, Argentina

GUISEPPE DALLE ORE 1970
Dipartimento Di Neurochirugia
Ospedale Maggiore 371000
Verona, Italy

HANS ERICH DIEMATH 1970
Hofrat Univ. Prof. Dr. Med.
TraunstraBe 31
A5026
Salzburg, Austria

HERMANN DIETZ 1980
Neurosurgical Clinic
Hannover School of Medicine
Hannover 3000-61
West Germany

JOHN GILLINGHAM 1962
Edinburg, Scotland EH43 PB

JAIME G. GOMEZ 1975
Transversal 4 No. 42-00
Commutador 2-32 4070
Bogota 8, Columbia, South America

SALVADOR GONZALES-CORNEJO (ROSALIE) 1982
Av. Chapultepec Sur 130
Guadalajara, Mexico 44100

ERNEST H. GROTE (JULIAN) 1984
Neurosurgery Department
University Clinic
7400 Tubigen
Fed. Republic of Germany H. Handa 1985

JOHN HANKINSON 1973
Department of Neurological Surgery
Newcastle General Hospital
Newcastle-Upon-Tyne 4
England

HANS-PETER JENSEN (RETA) 1980
Neurochirurgische Universitatsklinik Kiel
Weimarer StraBe 8
D-2300 Kiel/West Germany

RICHARD JOHNSON 1974
Department of Neurological Surgery
Royal Infirmary
Manchester, England

KATSUTOSHI KITAMURA 1970
University Kyushu Hospital Senior
Faculty of Medicine
Maidashi, Fukuoka 812, Japan

LAURI LAITINEN 1971
Department of Neurosurgery
University Hospital
S-901 85 Umea Sweden

WILLIAM MARGUTH 1978
Director, Department of Neurochirurgischen
Universitat Muchen
Marchioninistrasse 15
8000 Munchen 70, West Germany

RAUL MARINO, JR. 1977
Rua Maestro Cardim, 808
S. Paulo-SP
Brazil 01323

HELMUT PENZHOLZ 1978
Michael Gerber Ln. 55
6903 Neckargemund
West Germany

HANS-WERNER PIA 1978
Director
Zentrums fur Neurochirurgie Deceased 7/9/86
Universitat Giessen
Klinisktr. 37
6300 Giessen, West Germany

B. RAMAMURTHI 1966
2nd Main Road G.I.T. Coloney
Madras 4, India 600 004

CHARAS SUWANWELA 1972
Chulalongkorn Hospital
Medical School
Bangkok, Thailand

LINDSAY SYMON (PAULINE) 1982
The National Hospital, Queen Square
London, WC1E 3BG, England

KJELD VAERNET (ANN) 1970
Department of Neurosurgery
Rigshospitalet
9 Blegdamsvej
2100 Copenhagen, Demmark

SIDNEY WATKINS 1975
The London Hospital
Whitechapel, London E 1
England

GAZI YASARGIL 1975
Neurochirurgische
Universitatsklinik
Kantonsspital
8000 Zurich, Switzerland

DECEASED MEMBERS		DATE ELECTED
DR. SIXTO OBRADOR ALCALDE (Honorary) Madrid, Spain	4/27/67	1973
DR. JAMES R. ATKINSON (Active) Phoenix, Arizona	2/78	1970
DR. PERCIVAL BAILEY (Honorary) Evantson, Illinois	8/10/73	1960
DR. WILLIAM F. BESWICK (Active) Buffalo, New York	5/12/71	1959
DR. SPENCER BRADEN (Active) Cleveland, Ohio	7/20/69	Founder
DR. F. KEITH BRADFORD (Active) Houston, Texas	4/15/71	1938
DR. WINCHELL McK. CRAIG (Honorary) Rochester, Minnesota	2/12/60	1942
DR. ARTHUR ELVIDGE (Senior) Quebec, Canada	1/17/85	1934
JOSEPH P. EVANS Kensington, Maryland	5/8/85	Founder
DR. WESLEY A. GUSTAFSON (Senior) Jensen Beach, Florida	7/16/75	1942
DR. HANNIBAL HAMLIN (Senior)	6/28/82	1941
DR. HENRY L. HEYL (Senior)	3/01/75	1951
DR. OLAN R. HYNDMAN (Senior) Iowa City, Iowa	6/23/66	1942
MR. KENNETH G. JAMIESON (Corresponding) Brisbane, Australia	1/28/76	1970

SIR GEOFFREY JEFFERSON (Honorary) Manchester, England	3/22/61	1951
HUGO KRAYENBUHL (Honorary) Zurich, Switzerland	1985	1974
DR. WALPOLE S. LEWIN (Corresponding) Cambridge, England	1/23/80	1973
DR. DONALD D. MATSON (Active) Boston, Massachusetts	5/10/69	1950
DR. KENNETH G. McKENZIE (Honorary) Toronto, Ontario, Canada	2/11/64	1960
DR. JAMES M. MEREDITH (Active) Richmond, Virginia	12/19/62	1946
DR. W. JASON MIXTER (Honorary) Woods Hole, Massachusetts	3/16/58	1951
DR. WILDER PENFIELD (Honorary) Montreal, Canada	4/05/76	1960
DR. RUPERT B. RANEY (Active) Los Angeles, California	11/28/59	1939
DR. DAVID L. REEVES (Senior) Santa Barbara, California	8/14/70	1939
DR. DAVID REYNOLDS (Active) Tampa, Florida	4/03/78	1964
DR. R.C.L. ROBERTSON (Senior) Houston, Texas	2/85	1946
DR. STUART N. ROWE (Senior) Pittsburgh, Pennsylvania	10/11/84	1938
DR. WILLIAM B. SCOVILLE (Senior) Hartford, Connecticut	2/25/84	1944

DR. R. EUSTACE SEMMES (Honorary) Memphis, Tennessee	3/2/82	1955
DR. SAMUEL R. SNODGRASS (Senior) Nashville, Indiana	8/08/75	1939
DR. C. WILLIAM STEWART (Corresponding) Montreal, Quebec, Canada	1948	1948
DR. GLEN SPURLING (Honorary) LaJolla, California	2/07/68	1942
DR. HENDRIK SVIEN (Active) Rochester, Minnesota	6/29/72	1957
DR. BARNES WOODHALL (Senior) Durham, North Carolina	1985	1941

AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
1985 ANNUAL MEETING

EVALUATION

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

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

_____ Excellent
_____ Good
_____ Poor

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

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

(3) Did the speakers aim their talks:

_____ Too low?
_____ Too high?
_____ Just about right?

SCIENTIFIC PROGRAM

Monday's Sessions Excellent _____ Good _____ Poor _____
Comments _____
_____

Tuesday's Sessions Excellent _____ Good _____ Poor _____
Comments _____
_____

Wednesday's Sessions Excellent _____ Good _____ Poor _____
Comments _____
_____

SOCIAL PROGRAM

Comments _____

_____

_____

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

_____

Changes of address and/or telephone (indicate office or home address): _____

_____

_____

Please Print Name:

Return to: James T. Robertson
UTCHS, 956 Court Avenue
Memphis, Tennessee 38163

Keiji Sano Honorary
Shozo Ishii Senior Corres.
Katsutoshi Kitamura Corres.