The American Academy of Neurological Surgery 50th Anniversary

Cincinnati, Ohio

1988

THE 50th ANNUAL MEETING OF

The *American Academy of Neurological Surgery*

The Omni Netherland Plaza Hotel
Cincinnati, Ohio

September 13-17, 1988

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The American Academy of Neurological Surgery

September 13-17, 1988

The Omni Netherland Plaza Hotel
Cincinnati, Ohio

Tuesday, September 13, 1988

Senior Members Program

6:15 PM	Buses depart (5th St. Exit) for Queen City Club
6:30 PM-9:30 PM	Senior Members Dinner *Queen City Club*
9:30 PM	Buses return to hotel

Wednesday, September 14, 1988

1:00 PM-5:30 PM	Registration *Mezzanine*
1:30 PM-4:30 PM	Executive Committee Meeting *President's Suite*
6:00 PM-7:00 PM	Convocation (Members, Guests, Spouses) *Caprice 1-4*
7:00 PM-9:00 PM	Welcoming Reception *Continental Room*

Thursday, September 15, 1988

7:00 AM-8:00 AM	Breakfast Business Meeting (Members Only) *Caprice 1-2*
8:00 AM-5:00 PM	Registration *Mezzanine*
8:00 AM-12:45 PM	Scientific Meeting *Continental Room*

12:45 PM-2:00 PM	Lunch (Members, Guests) *Hall of Mirrors*
2:00 PM-5:00 PM	Scientific Meeting *Continental Room*
6:30 PM	Depart by buses (5th St. Exit) for cocktails and dinner at Contemporary Arts Center

Friday, September 16, 1988

7:00 AM-8:00 AM	Breakfast Business Meeting (Members Only) *Continental Room*
8:00 AM-5:00 PM	Registration *Mezzanine*
8:00 AM-10:00 AM	Scientific Meeting *Hall of Mirrors*
10:00 AM-11:30 AM	50th Anniversary Program (Members, Guests, Spouses) *Hall of Mirrors*
11:30 AM-12:00 Noon	Presidential Address James T. Robertson, M.D. (Members, Guests, Spouses) *Hall of Mirrors*
12:30 PM	Depart by buses (5th St. Exit) for golf and tennis tournaments at Cincinnati Country Club
7:00 PM-8:00 PM	Annual Reception *Third Floor Foyer*
8:00 PM-Midnight	Dinner Dance (Black tie) *Hall of Mirrors*

Saturday, September 17, 1988

7:00 AM-8:00 AM	Breakfast (Members, Guests) *Hall of Mirrors*
8:00 AM-12:00 Noon	Scientific Meeting *Hall of Mirrors*

Spouses Activities

Wednesday, September 14, 1988

6:00 PM-7:00 PM	Convocation (Members, Guests, Spouses) *Caprice 1-4*
7:00 PM-9:00 PM	Welcoming Reception *Continental Room*

Thursday, September 15, 1988

8:00 AM-4:00 PM	Spouse Hospitality *Hospitality Center*
9:30 AM-3:30 PM	Depart by bus (5th St. Exit) for all day tour of Krohn Conservatory, Taft Museum, lunch and sightseeing on the Ohio River
6:30 PM	Depart by bus (5th St. Exit) for cocktails and dinner at Contemporary Arts Center

Friday, September 16, 1988

8:00 AM-4:00 PM	Spouses Hospitality *Caprice 1-2*
8:45 AM-10:00 AM	Gadgets. . . Unique and Unusual *Caprice 1-2*
10:00 AM-11:30 AM	50th Anniversary Program (Members, Guests, Spouses) *Hall of Mirrors*
11:30 AM	Presidential Address James T. Robertson, M.D. (Members, Guests, Spouses) *Hall of Mirrors*
12:30 PM	Depart by bus (5th St. Exit) for golf and tennis tournaments at Cincinnati Country Club *(Lunch on own for those not participating)*

7:00 PM-8:00 PM	Annual Reception *Third Floor Foyer*
8:00 PM-Midnight	Dinner Dance (Black tie) *Hall of Mirrors*

Saturday, September 17, 1988

8:00 AM-11:00 AM	Spouse Hospitality *Hospitality Center*

CONVOCATION

American Academy of Neurological Surgery

50th Anniversary Meeting

Wednesday

September 14, 1988

6:00 p.m. - 7:00 p.m.

(CAPRICE 1-4)

The American Academy of Neurological Surgery
(Its Founding and Early Years)

by
Frank H. Mayfield, M.D.

An institution may for a time reflect the personalities of its founders, but if successful, it acquires a life and a substance of its own. By this process group ethos is fostered and individual character is enhanced. This has been so true with the "Academy."

In response to the Historian, I now place on the record my recollections of the circumstances that led to its founding and the philosophy and events that determined its course during its early years.

The place was the Peabody Hotel in Memphis, Tennessee; the time was April, 1938. The occasion was the Seventh Annual Meeting of the Harvey Cushing Society; Dr. Eustace Semmes of Memphis was host.

Seven young neurosurgeons were in attendance as guests. Each of them had been assured by his sponsor that he had been approved by the Executive Committee and recommended for membership. It seemed probable that all would be elected, but something happened. The "Cushing" changed the rules; none of the seven were elected.

Bill Keith later wrote: "It soon became clear that a lot of backroom politicking was going on. Before the meeting was over, I was swept into a small group with six others, founders of the Academy: Spencer Braden, Dean Echols, Joseph Evans, Frank Mayfield, Francis Murphey and John Raaf."

While waiting in line to register on arrival at the hotel I had met Bill Keith. At the request of the clerk, we agreed to share a room and another life-long friendship began.

The seven of us convened in the room which Bill and I shared and we organized The American Academy of Neurological Surgery. Dean Echols was elected president; Francis Murphey was made the secretary-treasurer. Since Joseph Evans and I lived in the same city, we agreed to accept the responsibilities and the privileges of being hosts and invited the group to meet in Cincinnati in October.

We chose the name of the organization at that first meeting. One or two thought the name a bit ostentatious. But the majority agreed that we could grow to fit it, and that in the interim our glaring lack of prestige might be somewhat obscured by the title.

What was the mood of the founders? We found ourselves bound by a common bond, forged by the insult of being excluded. We shared a feeling of frustration and anger but we relished the challenge and the opportunity.

We, the founders, determined to keep ethical and scientific standards high, but to keep the door open to those who would come after us if they qualified for the brotherhood which we considered an elite tribe. We hoped that fraternal exchanges with each other would better us all; that has been more than fulfilled. We hoped also that our spouses would find comraderie with each other and would look forward to renewing those friendships with each succeeding year.

All would agree, I am sure, that this too has been "a cup that runneth over." The grace and charm of each of them would have assured success anyway, but the fortuitous event of having John Raaf bring lovely Lorene to the first meeting at the beginning of their honeymoon, proved the catalyst that guaranteed success.

As our spouses "circled the wagon," so to speak, to welcome the new bride with love and tenderness, they found themselves tied together by the same bond that they had fashioned to protect her. This tradition continues like the "trailing arbutus" of antiquity.

Our first mission was to look for worthy candidates for membership. We reviewed the list while we were in Memphis. By mail and telephone we continued the process through the summer.

At the first meeting here in Cincinnati at the Netherland Plaza the following were invited and elected: Keith Bradford, Harvey Chenault, Donald Coburn, Wallace Hamby, Earl Walker, Exum Walker and Stewart Rowe. Dorothy and Howard Brown were also here as guests, and Howard should have been elected but a technical defect in his papers delayed the vote until the following year.

The meeting in Cincinnati was delightful. Headquarters was the Netherland Plaza Hotel. Scientific sessions were at the Good Samaritan Hospital and the Cincinnati General. Recreation and the banquet were held at the Maketewah Country Club.

The next year we met in New Orleans with Dean Echols as host. Howard Brown, Rupert Raney, Arthur Elvidge, David Reeves and Samuel Snodgrass were added to the roster.

The third meeting was in Cleveland with Spencer Braden as host. This meeting took place immediately after the first examination given by The American Board of Neurological Surgery and that event is a tale unto itself that must be dealt with elsewhere.

The fourth meeting of the society was held in part in Los Angeles with Rupert Raney as host, and part in San Francisco with Howard Brown as host.

The bombs at Pearl Harbor changed things, however. A few of us, many in uniform, met for one day the following year at the Palmer House in Chicago.

The next meeting was at Battle Creek, Michigan. I had the high privilege of holding the meeting at the Percy Jones General Hospital which I was serving.

The next year Barnes Woodhall entertained us at the Greenbriar at White Sulphur Springs, West Virginia, then the Ashford General Hospital of the U.S. Medical Department.

The next few meetings took place during the confusing post-war period. Somehow the policy of open-door for all who qualified was lost. This was occasioned in part by the fact that the Cushing Society had been persuaded to permit all who had passed the "Board" to apply for membership rather than waiting for nomination.

At the meeting in Colorado Springs the first indication that the Academy might change course was noted. The following year in Montreal the compass was reset.

Efforts to establish one major organization by merging the existing neurosurgical societies was not successful and other vigorous organizations were formed. The results of the effort to bring about a single organization are on the record elsewhere.

So let me close by referring to the early extracurricular and post-meeting activities of Academy members. It is best, of course, for most of that to remain as verbal history; recollections of individuals do seem to vary about what occurred.

Welcome to Cincinnati on the Fiftieth Anniversary!

*SCIENTIFIC PROGRAM**

Thursday, September 15, 1988

8:00 AM **Welcome,** James T. Robertson, M.D., President

SCIENTIFIC SESSION I

MODERATOR: James T. Robertson, M.D.

8:15 AM **Symposium on Neural Restoration**
Basal Ganglion Neurotransmitters and Plasticity
Steven Kitai, Ph.D.

8:45 AM **Trophic Factors and Axonal Growth in the Mature Brain**
Keith Crutcher, Ph.D.

9:00 AM **Engineering the Regeneration of the Sensory Fibers of Spinal Cord of the Adult Mammal**
Jerry Silver, Ph.D.

9:20 AM **Neural Transplantation: Toward a Therapy for Parkinsonism**
John Sladek, Ph.D.

9:50 AM **Discussion**

10:00 AM **Recess**

10:15 AM

1. **Behavioral and Neuronal Circuitry after Fetal Substantia Nigra Grafts and Dopamine Infusions in a Rat Model of Unilateral Substantia Nigra Injury**
   Howard M. Eisenberg, M.D., Massako Kadekaro, Ph.D., Mary Lee Terrell and Helena A. Lekan

*As an organization for Continuing Medical Education, The Christ Hospital certifies that this C.M.E. activity meets the criteria for 15 credit hours in Category I.

Unilateral substantia nigra (SN) injury results in characteristic behavioral abnormalities. The abnormality most commonly studied is rotation of the animal away from the side of the lesion after injection of apomorphine, a postsynaptic dopamine (DA) agonist. This effect is believed to be due to deafferentation hypersensitivity in the striatum after loss of projecting DA neurons from the injured SN. Implantation of cells capable of producing DA, fetal adrenal medulla or SN, into the ipsilateral striatum have been shown to attenuate this behavior. This finding has been an impetus for development of the use of these grafts for treatment of patients with Parkinson's disease. However, despite the adaptation of these techniques to patients, several important issues remain unresolved. Among these are: does the graft affect behavior by normalization of altered circuitry or by activation of secondary or novel pathways; and is the effect due to elaboration and diffusion of DA from the graft neurons or due to some other mechanism? We are attempting to address these issues studying rats with unilateral SN lesions (made by injection of 6-hydroxydopamine in the SN), after implantation with fetal SN or chronic infusion of DA. The behavioral effect of these manipulations is studied by counting rotations after injection of apomorphine; changes in the neuronal circuitry are studied using 2-deoxyglucose (2-DG) autoradiography. Thus far data from the 2-DG experiments in animals with SN lesions ($N = 23$) and animals with SN lesions given SN implants ($N = 15$) indicate that mitigation of abnormal behavior is the result of normalization of the fundamental circuit. This effect can be traced through the globus pallidus and entopeduncular nucleus to the ventrobasal thalamus. However, infusion of DA ($N = 15$) does not appear to mitigate the abnormal behavior, indicating that the effect on neuronal circuitry may not be due to diffusion of DA, but may depend on some other mechanism.

10:30 AM

2. Patterns of Vascularization in CNS Allographs

Michael Salcman, M.D., Richard Broadwell, Ph.D., R.J. Schlegel, M.D.

Survival of any grafted tissue depends on the development of its vascular supply. We investigated the angiogenesis of central nervous system grafts versus that of grafts of peripheral tissue and identified the types of blood vessels supplying the grafted tissues. Grafts from fetal/neonatal brain or adult pituitary gland were grafted into the third ventricle or caudate/putamen of adult host mice. Post transplantation survival times ranged from 1 day to 3 months. Host brains fixed by immersion and developed for endogenous peroxidase activity in red blood cells demonstrated that grafted central nervous system tissue was not perfused with host blood until after 7 days while grafted pituitary tissue was vascularized by the third day. Intravenous administration of HRP into host mice indicated that blood vessels supplying central nervous system grafts at 10 days or longer manifested intact BBB to circulating protein whereas blood vessels in pituitary grafts did not. Ultrastructural inspection of the central nervous system grafts revealed that the grafted vessels had interendothelial tight junctional complexes. Blood vessels of the pituitary grafts were fenestrated and did not possess BBB characteristics. Immunocytochemical staining for γ-interferon induced MHC of host versus graft vessels demonstrated that blood vessels in the grafted tissue were derived primarily from the donor and less so from the host brain.

The results suggest that the vascularization of grafted CNS tissue is not rapid compared to peripheral tissue, that vascularization may depend on the integration of the tissue within the host brain and that the blood vessels developing within the grafted tissue present morphological characteristics determined by the graft.

10:45 AM

3. Utilization of Unilateral and Bilateral Stereotactically Placed Adrenomedullary-Striatal Autographs in Parkinsonian Humans: Rationale, Techniques and Observations

Michael L.J. Apuzzo, M.D., John H. Neal, M.D., Leslie P. Weiner, M.D., Paul T. Moore, M.D., Vicki L. Wheelock, M.D., Stuart M. Boyd, M.D., Para T. Chandrasoma, M.D.

The prospect for achieving restoration of neurological function through tissue grafting has recently initiated a combination of excitement, intense scientific focus and controversy. The complexity of the concept and its comprehension have been elucidated further as the initial fragmentary data concerning clinical trials in Parkinsonian humans have been reported. At this time, few if any of the key issues attendant to clinical employment of the concept have been satisfactorily answered.

Our approach was to initiate a limited clinical study involving an amalgam of specialized disciplines including neurology, neuropsychology, neurosurgery, neuroradiology, surgical pathology and urological surgery to develop clarification of issues related to patient selection, optimization of grafting materials, surgical technique, and modification of clinical status.

Following initial assessment of 82 Parkinsonian patients for periods of two months to six months, ten (aged 38-68) were elected for unilateral (5) or bilateral (5) adrenomedullary autografts to the caudate nucleus employing image directed stereotactic methods. Glands were harvested by a retroperitoneal approach. Reproducible precise targeting and transit trajectory have allowed for placement of tissues within the brain parenchyma with access to the ventricular fluid of the frontal horn. One patient did not undergo cerebral grafting as the gland material was hemorrhagic on delivery. In the remaining nine patients no surgical complications were noted and all patients were ready for discharge at five days postoperatively.

Postoperative observations have been made for periods from 3-15 months including parameters of clinical observation, strict maintenance of medication schedules and records, patient and family commentaries, imaging studies and spinal fluid assessments. These observations will be presented with implications related to both clinical observations and technical experiences as a guide for the development of future directions related to the grafting concept.

11:00 AM

4. Fetal Cerebellar Grafts for Cerebellar Atrophy in Humans: A Preliminary Report

Wu Cheng-Yuan, M.D.

We have shown in a rat model that surgical injury to the right cerebellum with resultant ataxia can be corrected by implantations of embryonic cerebellar tissue into the injured cerebellum. Histological examinations of the cerebellar tissue revealed

that mitoses in Purkinje cells of the implanted group were increased substantially over the control groups. The biologic surgically induced ataxia resolved more rapidly in the cerebellar implant group than the control groups.

Based on this experimental data, we applied a similar technique in six patients with severe hereditary cerebellar degenerative ataxia. The preliminary results in the six surgically implanted patients with heredity degenerative cerebellar disease show two patients with excellent improvement, three patients with moderate improvement and one patient with improvement for two months followed by mild deterioration, however, his condition remains much better than prior to tissue implantation. Rapid improvement of symptoms suggests that the mechanism is likely related to stimulation of trophic nerve growth factors.

We also studied immunological markers in the blood and CSF in an attempt to determine whether rejection of implanted tissue occurs.

11:15 AM

5. Neural Reorganization and Sensory Recovery after Anterolateral Cordotomy

Elizabeth Bullitt, M.D., W.S. Stofer, M.D., C.J. Vierck, M.D., E.R. Perl, M.D.

Following anterolateral cordotomy, many patients develop the gradual recovery of sensory function over a period of months to years. We have used sub-human primates to investigate the possibility that this delayed recovery might be related to sprouting of primary afferent neurons within the spinal cord.

Pig-tail macaques (macaca nemestrina) were trained to pull a lever in order to limit the duration of an electrical stimulus, and the vigor and frequency of these escape responses were used to differentiate painful from non-painful levels of stimulation. Following cordotomy, behavioral testing revealed contralateral hypalgesia in all animals, with sensory recovery in half of the group by 12 months.

At the terminal experiment, dorsal rootlets caudal to the spinal lesion were labeled with HRP, and the distribution of labeled synaptic complexes was determined within the dorsal horn. When compared to controls, animals undergoing cordotomy showed a loss of terminals in the superficial dorsal horn and an increase of synaptic enlargements in deeper layers. These effects were bilateral, but most pronounced on the side contralateral to cordotomy. Animals with diffuse spinal

lesions showed a completely different change in the distribution of primary afferent terminals. Animals with sensory recovery demonstrated a more normal terminal distribution pattern than persistently hypalgesic monkeys, but there was considerable variability in the data and analysis by different statistical methods yielded varying results.

Analysis of lesion sites demonstrated that monkeys with lesions confined to the classically defined location of the spinothalamic tract failed to show sensory recovery. Monkeys with lesion sites that extended more medially sometimes did and sometimes did not recover sensory function.

11:30 AM

6. Clinical Correlation of Specific Neuronal Loss and Neurotransmitter Alterations in Human Temporal Lobe Epilepsy

Dennis D. Spencer, M.D.

45 patients undergoing enblock anteromedial temporal lobectomy and radical hippocampectomy had the hippocampal, subicular, and entorhinal cortex analyzed for the neurotransmitters glutamate and GABA and the neuropeptides somatostatin, cholecystokinen, vasoactive intestinal polypeptide, and dynorphin via immunohistochemistry (IHC) and quantitative radioimmunoassay (RIA) of the neuropeptides. Neuronal cell counts were performed in all CA fields plus the dendate and entorhinal cortex. 32 of these patients were selected by medial temporal lobe ictal events during chronic depth electrode recordings (non mass temporal lobe epilepsy (NMTLE). The remaining 13 patients had temporal lobe mass lesions (TLML) identified by either CT or MRI scanning techniques. No specific demographic correlations were noted save for febrile seizures which appeared in 60% of non mass patients. All neuronal counts and biochemical analyses were compared to autopsy control brains procured within 12 hours of death. A statistically significant loss of neurons was noted in the CA^1, CA^3, CA^4, and dendate granule layer of all patients with well localized medial temporal lobe seizures. This was most profound in patients with febrile seizures and much less in TLML patients compared to autopsy controls. Of the neuropeptides analyzed, only somatostatin (SS) showed a change. There was a significant decrease of SS, particularly in CA^4 and the dendate granule cells, paralleling the neuronal cell loss and most profound in patients with febrile seizures with less depression

noted in TLML patients. There was a significant correlation between neuronal cell loss and decreased SS with the duration of the silent period between the patient's first childhood seizure and the first intractable seizure. This is the first human data to corroborate the animal experiments demonstrating precisely the same neuronal loss and SS decrease after excitotoxic injury from perforant pathway stimulation which results in decreased hippocampal inhibition. This data will be used to explore the hypothesis of excitotoxic injury as the initiating event in disinhibition of specific structures in patients suffering temporal lobe epilepsy.

11:45 AM **Discussion**

SCIENTIFIC SESSION II

12:00 PM **Shining Moments: 35 Years of Academy Awards**
Eben Alexander, M.D.

12:15 PM **Academy Award Paper**
To be announced by George Ojemann, M.D.

12:45 PM **Lunch**

SCIENTIFIC SESSION III
MODERATOR: John M. Tew Jr., M.D.

2:00 PM **Symposium on Arteriovenous Malformations**

7. Three Dimensional Magnetic Resonance Angiography of the Carotid Artery and Intracranial Vessels
Thomas Masaryk, M.D., Robert Ratcheson, M.D.

Blood flow effects in magnetic resonance imaging were initially appreciated by the absence of signal in vascular structures as well as the presence of distracting motion related artifacts. Understanding of these effects led to the recognition of their dependence upon: 1) character of flow, 2) MR pulse sequence parameters and, 3) spatial relationship of the vessel to the imaging plane. The types of signal intensity changes produced are typically categorized as "time-of-flight effects" and "spin-phase phenomena." Since 1985, serious efforts have been directed towards modifying MR images (with special magnetic gradients and pulse sequences) such that angiogram like images are obtained non-invasively and without contrast

material. Recent work in the development of magnetic resonance angiography has indicated that gradient-echo, volume imaging may provide images with the greatest fidelity. Rather than acquiring standard, two dimensional matrix scans, these techniques render three dimensional (hence the name volume) data sets. In conjunction with special computer processing utilizing ray tracing algorithms and thresholding techniques, three dimensional cerebrovascular images have been obtained of intracranial aneurysms, arteriovenous malformations, arterial occlusions, and carotid stenoses. Experience to date indicates that these techniques may provide an entire new dimension to standard MR neuro-diagnostic imaging.

2:15 PM

8. Intracranial Arteriovenous Malformation: Factors Influencing Hyperemic Complications and Outcome

H. Hunt Batjer, M.D., Michael D. Devous, Ph.D., G. Burton Seibert, Ph.D., Duke S. Samson, M.D.

Serious morbidity and hyperemic states continue to complicate the treatment of certain intracranial AVM's. Clinical, radiographic, and cerebral blood flow characteristics of 62 patients treated over three years were analyzed to determine if hyperemic complications (HC) and outcome could be predicted. Single-photon emission computed tomography following Xe-133 inhalation was used to calculate pre-operative total brain blood flow (TBF) defined as the average of bilateral hemispheric rCBF. Regions of ipsilateral and contralateral hypoperfusion were noted in all patients. An ipsilateral and contralateral Steal Index was calculated to express severity:

$$\text{I Steal (i), I Steal (c)} = \frac{\text{rCBF (Steal area)}}{\text{TBF}}$$

25 (40%) of patients were less than 30 years, 28 (45%) were 30-50 years, and 9 (15%) were over 50. 48% had a history of hemorrhage and 34% presented with progressive deficits. 13 patients (21%) developed HC. 51 (82%) ultimately had a good outcome, 4 (6%) had poor outcome and 7 (11%) died.

Analysis of clinical and rCBF data allowed a profile to be constructed of risk factors for the development of HC and unfavorable outcome:

INCREASED RISK OF HC

1. Perforating vessels ($p < .001$)
2. Angiographic "Steal" ($p < .05$)
3. Large size ($p = .082$)
4. Larger sum of diameter of feeding vessels ($p < .05$)
5. Intermediate contralateral steal severity ($p = .086$)
6. Dramatic increase in hemispheric rCBF after therapeutic embolization
7. Vasoreactivity: enhanced vasodilation to Diamox may be a marker.

UNFAVORABLE OUTCOME

1. Older patients ($p < .05$)
2. Perforating vessels ($p = .07$)
3. Right hemispheric AVM's ($p < .05$)
4. Large size ($p = .13$)
5. Depressed TBF ($p = .16$)
6. Less severe ipsilateral steal ($p = .16$)
7. Less severe contralateral steal ($p = .12$)
8. Hyperemic complications ($p < .001$)

2:30 PM

9. Intraoperative Angiography as an Adjunct for the Treatment of Arteriovenous Malformations and Aneurysms

Wolff M. Kirsch, M.D., William W. Orrison, M.D., Viraf R. Cooper, M.D., Jerry King, M.D.

Three patients with complex arteriovenous malformations were operated on with the assistance of intraoperative angiography in order to facilitate surgical ablation. Two of the malformations were deeply situated with intraventricular and callosal extension and deep venous drainage into the Galenic system. The third malformation was temporal-parietal with feeding from A1, M1, M2, and P1 segments of respective cerebral arteries on the right. Aneurysm surgery is more precise with this adjunct. This fact will be illustrated with a videotape of surgical management of a dissecting vertebral artery aneurysm.

Intraoperative angiography is performed with a mobile DSA C-arm system (OEC Diosonic series, 9000 mobile C-arm, Salt Lake City) consisting of a microprocessor controlled x-ray main frame and dual screen monitor with integrated real time digital image processing. The system has the capabilities of image storage with video cassette recorder and disc sys-

tem. The unit is powered by a 115 volt, 20 amp electrical outlet and allows the independent selection of real time subtraction of 30 frames per second with the availability of catheter roadmapping. The additional feature of peak opacification enables rapid hand injection screening angiographic capability. Catheter roadmapping permits a significant reduction, in contrast requirements with selective catheter placement.

The two patients with deep intraventricular and callosal malformations were approached by inferior parietal occipital cortical incisions, through the trigone in order to gain access to the malformations. Both of these malformations were totally ablated and the patients have made uneventful recoveries from disabling intraventricular hemorrhage. The third patient with a right parietal temporal lesion has made a good recovery and intraoperative control enabled a more sensitive surgical approach for elimination of the lesion.

In summary, intraoperative angiography has proven to be a valuable adjunct for the surgical management of complex arteriovenous malformations.

2:45 PM

10. Surgery of Epilepsy Associated with Cerebral Arteriovenous Malformations

Hwa-shain Yeh, M.D., John M. Tew, M.D.,
Shiro Kashiwaga, M.D., Thomas S. Berger, M.D.

Twenty-seven patients with seizure disorder due to cerebral arteriovenous malformation (AVM) have been surgically treated in the period between 1982 and 1986. There was no past history of clinical manifestation of intracranial hemorrhage in these patients. The age of the patients ranged from 13 to 61 years. There were 13 males and 14 females. The size of the AVMs was larger than 4 cm in 18 patients. The most frequent location of the AVM was in the temporal lobe, followed by the frontal, parietal and occipital lobes. All patients had preoperative electroencephalographic study, intraoperative electrocorticography and acute depth electrode recording of the amygdala and the hippocampus, if the AVM was located in the temporal lobe. Superficial or posterior temporal lobe AVMs often have remote seizure foci which involve the amygdala and hippocampus. All patients underwent craniotomy and total excision of their AVM; surgery was carried out under local anesthesia to

allow localization by electrical stimulation if the AVM involved the speech area or the sensorimotor cortex. Based on the electroencephalographic findings, excision of the epileptogenic lesion, in addition to the AVM was performed in 18 patients.

There were no operative deaths. Two patients developed hemiparesis and 3 had temporary dysphasia after surgery. Two patients had visual field deficit. Results of postoperative seizure control during the average follow-up period of 3 years were excellent in 21 patients, good in 3, fair in 2 and poor in 1. The latter, whose epileptic lesion was not completely excised because of its location in the motor cortex, had poor seizure control postoperatively. Another patient required a second operation to remove a remote seizure focus.

In our series, mechanisms of seizure associated with cerebral AVM include focal cerebral ischemia secondary to arteriovenous shunting, gliosis of the surrounding brain and a secondary epileptogensis in the temporal lobe. Successful seizure control can be obtained with wide excision of the epileptogenic foci surrounding or remote from the AVM.

3:00 PM

11. Photon Beam Treatment of Arteriovenous Malformations Using Dynamic Radiosurgery

A. Olivier, M.D., E. Podgorsak, M.D., J.J. Hazel, M.D., A. DeLotbiniere, M.D., L. Souhami, M.D., B. Pike, M.D., G. Bertrand, M.D.

Dynamic radiosurgery using a linear accelerator was developed at McGill University. Its principle resides in the continuous and simultaneous rotation of both the head and the accelerator photon beam around a common isocenter which provides sharp fall off of the radiation outside the target volume.

Target determination and dose-structures correlations are established with specially designed stereotactic software.

Our experience with the first 20 cases of arteriovenous malformations treated by this modality at the Montreal Neurological Institute and McGill University is presented. Results for 6 patients having had angiography at or around 1 year post-treatment is available. 3 patients have had total occlusion of the arteriovenous malformation, 2 patients have had occlusion in the order of 85-90% and 1 patient in the order of 50%.

The combined use of stereotactic DSA and MRI permits the use of small target volume in relation to lesion volume, thus decreasing the risk of necrosis to neighboring structures.

3:15 PM Discussion

3:30 PM Recess

SCIENTIFIC SESSION IV
MODERATOR: Ellis B. Keener, M.D.

3:45 PM

12. Pressure Gradiments in Ischemic Brain Edema
Julian T. Hoff, M.D., S. Hatashita, M.D.

After acute occlusion of a cerebral artery edema formation develops predictably, accompanied by a progressively rising hydrostatic gradient between the ischemic zone and surrounding tissue. Flow interruption triggers the formation of both hydrostatic and osmotic pressure gradients which play important roles in the formation of ischemic edema. The hydrostatic pressure gradient is important in the first six hours after occlusion while the osmotic gradient effect comes earlier then disappears by the time tissue hydrostatic pressure in the ischemic zone has reached its peak. Experimental evidence for the above contentions will be presented.

4:00 PM

13. Oh Minus Free Radicals in the Genesis of Brain Edema: A New Therapeutic Hope
Don M. Long, M.D., Ph.D., Yukio Ikeda, M.D.

There has been no new therapy for brain edema for nearly thirty years. Steroids remain the only proven treatment. New studies suggest that free radicals may be important in the genesis of all forms of brain edema. The current study was undertaken to investigate the effects of a hydrozyl free radical scavenger Desferoximine, on the development of edema secondary to brain injury.

Standard cold injury, traumatic vasogenic brain edema, was produced in 24 adult cats. In one group, no therapy was given. A second group was pre-treated with Desferoximine and then the drug continued to sacrifice at 24 hours. In the third group, the delivery of drug was delayed until 15 minutes after injury and then continued for 24 hours.

Brain edema was measured at 3, 6, 12, and 24 hours. Competency of the blood brain barrier was assessed by planimetry of the area of spread of Evans Blue dye injected intravenously at the time of injury.

Animals were sacrificed at each time point and the brain serially sectioned. Brain samples from eight consistent areas were obtained from each animal. The area of spread of Evans Blue was obtained from the matching section by means of mechanical planimetry. The specific gravity method was utilized for the measurement of brain edema.

4:15 PM

14. MRI Correlates of Normal Pressure Hydrocephalus

E.R. Laws, M.D., C.R. Jack, B. Mokri, O.W. Hauser, M.D., R.C. Peterson, M.D.

Normal pressure hydrocephalus (NPH) remains a difficult diagnosis, and advice with regard to shunting is often based on circumstantial evidence. MRI studies of patients suspected of having NPH allow for evaluation of several aspects of functional neuroanatomy previously unavailable or poorly demonstrated by CT. Based on preliminary studies, we systematically evaluated four MRI findings: increased periventricular signal (PVS); increased white matter signal (WMS); CSF flow void sign in the aqueduct (CFVS); and atrophy or thinning of the corpus callosum (CCA).

The study group consisted of 54 patients: 17 with NPH; 8 with obstructive hydrocephalus; 8 with Alzheimer's disease; and 21 with non-Alzheimer dementia. PVA was most commonly seen in obstructive hydrocephalus and NPH, but was occasionally seen in the other groups. WMS was correlated with PVS, and most frequently seen in NPH and non-Alzheimer's dementia, and was also related to age. CFVS was most frequent and most pronounced in the NPH group, but some degree was present in patients in other groups. CCA was present in all cases of NPH, but also overlapped into other groups.

Improvement following shunting occured in all 11 NPH patients with adequate followup after surgery. Improvement correlated with thinning of the corpus callosum and a marked or moderate CFVS. Results were best in those patients without WMS. MRI evaluation adds significantly to the data used to diagnose and to treat NPH.

4:30 PM

15. Hydrocephalus: Zero Pressure Adjustable CSF Shunt
Eldon L. Foltz, M.D.

Evidence has increased that a percentage of patients shunted for hydrocephalus will show disabling symptoms and signs of low intracranial pressure several years after shunt placement. Such patients show a dramatic fall in ICP when in the upright position whereas normal ICP is present in the supine position. The fall to as much as -350 mm CSF when upright correlates with disabling symptoms. To date, shunt valves have been relative pressure valves which operate on a pressure gradient basis for opening pressure. This design favors "overshunting" with the resulting "low ICP syndrome".

This disabling sequence can be avoided by the zero pressure shunt system designed to function on the basis of a closing CSF pressure device which allows ICP to fall only to zero on the upstream side. It is an absolute pressure device when inserted into the shunt system, but must have a one-way valve in the system as well.

The system design is based on the concept that the siphoning process in ventricular shunts is the primary ICP process in need of absolute control. Such hydraulic parameters vary related to body position of supine and upright, and therefore placement of this device in the subgaleal space has two vertical coordinates - 1) the distance from vertex when upright; and 2) the distance from frontal tip when supine. At those specific points, the intra-luminal shunt pressure cannot go below zero. At reference points of vertex (when upright) and frontal tip (supine) the ICP can fall only to a negative value equal to the vertical distance the siphon control device (or zero pressure device) is below that point for that body position.

This system has been effectively used in 22 patients suffering from disabling low ICP syndrome. All patients had precise ICP records pre- and post-op. 12 have had brain map EEG's to assess electro-physiologic status. 20 patients were markedly improved clinically, all showed markedly improved ICP status, but 2 patients demonstrated impaired ability to function at "normal" ICP levels, and required a graduated return to such levels.

The basic characteristics of the new shunt will be reviewed. The need of such a system in the initial treatment

of hydrocephalus will be obvious in the specific review of the results in 22 cases in which the system is now functioning.

4:45 PM

16. Intercostal Nerve Transfers for Restoration of Elbow Flexion

A.H. Friedman, M.D., J.A. Nunley, II, M.D., R.D. Goldner, M.D., W.J. Oakes, M.D., J.R. Urbaniak, M.D.

Avulsion injuries of the brachial plexus most frequently occur in healthy young males who then are left with a severe permanent deficit. Despite technical advances, our ability to restore upper extremity motor function following a brachial plexus avulsion injury is limited. In the past ten years, we have attempted to restore elbow flexion following a brachial plexus avulsion injury in 14 patients by anastomosing intercostal nerves to the musculocutaneous nerve and in four patients by anastomosing intercostal nerves to a transposed free gracilis muscle graft. Four patients had undergone prior dorsal root entry zone lesions for persistent pain at which time nerve root avulsions were confirmed by direct inspection. The remainder of the patients underwent an exploration of the proximal plexus to confirm the presence of avulsion.

Six (63%) of patients who underwent an intercostal to musculocutaneous nerve anastomosis and two (50%) of the patients who underwent an intercostal to gracilis graft anastomosis achieved good elbow flexion. Two additional patients were noted to have some return of biceps function but not enough to flex the elbow against gravity. Patients destined to have a return of biceps function first noted elbow flexion to occur with coughing or laughing six to eight months after the anastomosis. As the anastomosis matured, patients developed the ability to contract the biceps brachialis voluntarily. Electromyography in four patients revealed simultaneous activity of the biceps brachialis and intercostal muscles with deep breathing. A permanent interference pattern developed in the biceps brachialis but no intercostal motor unit activity was recorded when the patient attempted biceps brachialis contraction. It appears that neurotization of the biceps brachialis is a practical alternative in the treatment of patients with brachial plexus avulsion injury.

5:00 PM Discussion

5:15 PM Recess

Friday, September 16, 1988

SCIENTIFIC SESSION V
MODERATOR: Byron Pevehouse, M.D.

8:00 AM Historical Papers

17. The Role of the Academy in the Evolution of Psychiatric Surgery

H. Thomas Ballantine, Jr., M.D.

Two years before the founding of this Academy Egas Moniz published his first paper on frontal leucotomy for psychiatric illness. The initial enthusiasm for this innovative treatment of intractable psychiatric disorders and chronic pain was soon tempered by reports of undesirable side-effects, and neurosurgeons began a search for modifications of leucotomy which would increase safety without reducing efficacy.

As a result of these clinical investigations, the original imprecise, radical frontal leucotomy has been superseded by precise, small stereotactically placed lesions in the limbic system.

Members of the Academy have played a prominent role in the evolution of psychiatric surgery. This presentation will document some of their more important contributions to a currently under-utilized, often criticized approach to the treatment of suffering individuals chronically disabled by psychiatric illness and pain.

8:15 AM

18. The First Scalp - Cortical Arterial Shunt: 1951

J. Lawrence Pool, M.D.

At the 1986 Stonewin Conference, sponsored by Dr. Bennett M. Stein, Dr. Gazi Yasargil publicly stated that he was inspired to perform extracranial to intracranial arterial shunts because of my report of such a procedure—with a plastic tube for the shunt—in 1951. A brief description of this 1951 operation, illustrated with one slide, is offered for the Academy's 1988 historical program. While the procedure may not have done the least bit of good, three things are certain: 1. It did no harm. 2. It was the first of its kind in history. 3. It saved the patient's life—for a reason to be explained.

8:30 AM

19. Dandy's Contributions to the Foundation of Pediatric Neurological Surgery

Hugo V. Rizzoli, M.D.

WALTER DANDY (1886-1946) made many monumental contributions to Neurosurgery. His contributions to PEDIATRIC NEUROLOGICAL SURGERY include his introduction of ventriculography and his numerous contributions to the understanding and treatment of hydrocephalus. Hydrocephalus had been recognized as a clinical entity since Hippocrates. The pathophysiology remained obscure and no rational treatment had been proposed. Dandy and Blackfan were the first to produce hydrocephalus in an experimental animal. A preliminary report appeared in 1913. They dealt with the experimental, clinical and pathologic aspects of hydrocephalus. This work resulted in a logical and rather precise understanding of the formation, circulation and absorption of CSF in normal and hydrocephalic animals and patients. Dandy's interest in hydrocephalus continued until his death. He was able to experimentally produce in animals the various types of hydrocephalus which he encountered clinically in humans. He then designed and performed corrective operative procedures which were often successful.

I would like to show an eleven minute video of a movie recoding the post operative course of a patient who had surgery for a pituitary tumor in 1937. In this film we see Dandy in the operating theater and several scenes of Dandy and Sir Geoffrey Jefferson who was visiting Dandy at the time.

8:45 AM

20. The Evolution of Pituitary Surgery

Edward R. Laws, Jr., M.D.

The development of pituitary surgery is presented, beginning with Lower's recognition of chiasmal compression and the potential for its relief. Experimental approaches were developed in the late 19th century by Horsley, Schloffer and others. In man, craniotomy approaches developed through the efforts of Horsley, Krause, Heuer, Frazier and others. Transsphenoidal approaches were pioneered by von Eiselsberg, Kocher, Hirsch, and Cushing. Cushing's influence dominated pituitary surgery until the important contributions of Olivecrona, Bronson Ray, Guist and Hardy. The

contributions of Academy members (George Ehni, Hannibal Hamlin, Sir Geoffrey Jefferson, W. Jason Mixter, Wilder Penfield, Hendrik Svien, Edwin Boldrey, C. Hunter Shelden, William Collins, Russell Patterson, George Tindall, John Van Gilder, Martin Weiss, Charles Wilson, Nicholas Zervas) to this area of neurosurgery will be acknowledged.

9:00 AM

21. Atlanto-axial Joint Bone Block Arthrodesis and Interlaminar Fusion, the Cone Procedure, for C1-C2 Subluxations

Gilles Bertrand, M.D.

40 cases of anomalies of the C1-C2 junction were treated by a technique consisting of interposing bone blocks in the atlanto-axial joints, through a posterior approach, in association to interlaminar fusion.

The method was devised by the late W.V. Cone, who carried out 17 of these procedures at the Montreal Neurological Institute from 1949 to 1956. The others were done by the author in subsequent years.

The rationale for this procedure, derived from an autopsy study will be reviewed.

The interposition of bone blocks in the atlanto-axial joints does not only provide additional fusion surface to the classical interlaminar grafts, but serves primarily to maintain the separation achieved between C1 and C2 by the preoperative skeletal traction, to flatten the buckling ligaments and to "pull down" upwardly displaced odontoid processes. Thus, a single procedure stabilizes the C1-C2 subluxation and relieves compression of the neuraxis by anterior pathological elements obviating the need for a trans-oral decompression in most instances.

Variations in the technique used will be described. Bone bank tibia was used in 20 of the cases, autologous skull bone in 12, autologous rib and iliac crest in 2, heterologous bovine (Boplant) in 2. (undetermined in 4).

Solid fusion was achieved in 31 of 32 for whom adequate follow-up data is available. 1 required a second operation.

Complications were few and will be discussed.

Expected side-effects are numbness in the C2 dermatomes and limitation of neck rotation.

The clearest indication for this procedure is in chronic atlanto-axial subluxations, congenital or acquired with

overstretched, buckling ligaments compressing the cord anteriorly or with upward migration of the odontoid, as often seen in rheumatoid arthritis.

9:15 AM

22. An H.M.O. Caper

Theodore Kurze, M.D.

Very few if any members of The American Academy of Neurological Surgery have been full time employees of a Health Maintenance Organization (H.M.O.). I would like to report on this 'ex cathedra' experience.

Health Insurance Plan, New York (H.I.P.) is the second largest H.M.O. in the United States and the largest that confines itself to one metropolitan area. Approximately one million subscribers are enrolled in the plan. Recognizing the economic advantages of centralizing an isolated specialty such as Neurological Surgery, the management is attempting to organize one center for both inpatient and outpatient care with a full time staff of neurosurgeons. I was lured into this plan because the opportunity to pursue excellence with such a large number of patients appeared from the outside irresistable.

The view from the inside has been a learning experience without peer. In 1987, there were 500 neurosurgical operations in one hospital by three neurosurgeons. There were 1320 outpatient consultations. The case load continues to rise exponentially and since January, 1988, there are 3½ neurosurgeons, now that I'm on board. There is one Pgy 1 or 2 rotating through each month and three physician assistants. There is an average daily census of 44 patients and this represents 7% of the hospital occupancy.

H.M.O. medical care has been anti-thetical to my traditional professional values, but it is not lilliputian, and currently provides 12% of U.S.A. health care.

Both academic and community neurosurgeons have reason to be concerned with the present volume of operations available for both residents and practicing neurosurgeons. Many neurosurgeons are not able to maintain much less advance their (chi-ergie) handwork skills. Technologic improvement in instruments and anesthesiology have counterbalanced this so that the quality of the product delivered to the patient hasn't changed.

The discussion based on my adventure thus far will focus

on the question "Can H.M.O. be utilized as a future resource for post residency continued training in neurological surgery?".

9:30 AM **Discussion**

9:45 AM **Recess**

10:00 AM **50th ANNIVERSARY PROGRAM**
Historical Contributions of the Academy to Neurosurgery
Byron C. Pevehouse, M.D. — Historian

10:10 AM **Laboratory Research as a part of Residency Training**
Henry Schwartz, M.D.

10:20 AM **Use of the Lucite Calvarium to Study Head Injuries**
Hunter Shelden, M.D.

10:35 AM **Development of Implantable Catheters & Valves for Shunts**
Robert Pudenz, M.D.

10:50 AM **Corticosteroids & Dehydrating Agents in Treatment of Cerebral Edema**
Lyle French, M.D.

11:00 AM **Discussion**

11:15 AM **Ladies of the Academy**
Angeline Pool

11:30 AM **PRESIDENTIAL ADDRESS**
(Introduction by Thoralf Sundt, M.D.)
James T. Robertson, M.D.

SCIENTIFIC SESSION VI
MODERATOR: Julian T. Hoff, M.D.

8:00 AM Tumor Symposium

23. Working Through the Brain Rather Than Against It
Roberto Heros, M.D.

In approaching deep AVM's, aneurysms and tumors the author frequently uses surgical approaches that involve resection on non-eloquent brain tissue. Some of these approaches have been well described in the literature and will not be discussed in any detail (i.e.: gyrus rectus approach for anterior communicating aneurysm, superior temporal gyrus approach for middle cerebral aneurysm, inferior temporal gyrus approach to AVM's of the medial temporal lobe, middle temporal gyrus approach to the atrium and transfrontal approach to the lateral ventricle). Some less well known approaches will be described in detail and illustrated by sketches, photographs and case presentations: These include:

1) Posterior-inferior temporal approach to the incisura. A small amount of brain resection in front or behind the vein of Labbe allows temporal retraction without injury to the vein.

2) Parieto-occipital parasaggital approach to the atrium. An incision centered about 7 cm. above the occipital tip and 2 cm. lateral to the falx allows approach to the atrium without parasaggital retraction or sacrifice of veins.

3) Resection of parahippocampal gyrus to approach the posterior cerebral artery in the incisura. This avoids deep temporal retraction.

4) Tonsilar resection for distal posterior inferior cerebellar artery aneurysms. This avoids tonsillar retraction and risk of aneurysmal rupture.

5) Cingulate gyrus resection for pericallosal aneurysms. This avoids deep interhemispheric retraction and risk of aneurysmal rupture.

6) Anterior-inferior temporal resection for basilar aneurysms. When the temporal fossa is deep this allows good anterior subtemporal exposure to the pre-peduncular region without excessive temporal retraction.

8:15 AM

24. Total Resection of Benign Tumors and Vascular Lesions of the Cavernous Sinus Without Cardiac Arrest

Harry vanLoveren, M.D., John M. Tew, Jr., M.D., Jeffrey Keller Ph.D.

In spite of dramatic progress in technology, there are frontiers in neurosurgery yet to be explored and conquered by advances in our understanding of fundamental anatomy.

Until recent years, extension of benign tumors into the cavernous sinus was considered synonymous with unresectability and the morbidity or even mortality associated with their growth was accepted. Based upon detailed laboratory cadaveric dissection and the pioneering clinical work of a few neurosurgeons around the world, the cavernous sinus territory has become part of the domain of the general neurosurgeon.

We present a series of twelve cases demonstrating the possibility of total resection of benign tumors and treatment of vascular lesions of the cavernous sinus.

The fundamental anatomy and neurovascular relationships which must be understood to accomplish total resection without morbidity will be presented in cadaveric dissection in conjunction with operative slides and video.

Decision-making in selecting an appropriate operative approach will be analyzed. The approaches to cavernous sinus lesions applied in this series include: anterior extradural-intradural (Dolenc) approach; subtemporal approach with entrance through Parkinson's triangle (optional resection of the posterior clinoid and clivus); petrosal approach; suboccipital approach with entrance through Meckel's cave. Special attention will be given to adjunctive technology utilized in the clinical series including: interventional neuroradiology with navigation of detachable balloons for vascular cavernous sinus lesions; application of the CO_2 laser; application of the Nd:YAG laser.

8:30 AM

25. Application of Craniofacial Techniques to Tumors in Children

D.A. Bruce, M.D., K. Salker, M.D., I. Munro, M.D.

In the first year of full function of the new Craniofacial Institute, 17 of 93 craniofacial operations were for diseases

other than congenital craniofacial syndromes or trauma. The majority of these operations were for tumors. Ages ranged from one month to 19 years. The techniques of split cranial bone graft and sagittal split of the face have permitted reconstruction at the time of definitive surgery, even infants at one month of age.

Three cases were of unusual congenital tumors of the face, mouth, and intracranial cavity. These were all operated on under nine months of age and, despite extensive resection, immediate reconstruction was possible. There were eight cases of optic nerve compression by fibrous dysplasia who underwent optic nerve decompression using a transorbital approach. This was felt to be superior to any other approach. There was no increased visual loss, and both optic nerves could occasionally be decompressed from a unilateral approach. Therefore, because the dura wasn't opened, extensive resection and reconstruction of the cranial base, nasal structures, and orbits could be safely performed. Finally, a transfrontal transethmoidal approach with dissection of the mid-portion of the cranial base was found to be an ideal technique for the repairs of sphenoidal encephaloceles. The techniques and select cases will be presented and discussed in detail.

8:45 AM

26. The Differentiation Potential of a Human Medulloblastoma Cell Line

Henry Schmidek, M.D., Ronald McKay, Ph.D.

The TE 671 cell line is derived from a left cerebellar medulloblastoma prior to the patient receiving radiation therapy or chemotherapy. Because this cell line has been characterized in considerable detail it was selected to explore whether it is possible to induce its differentiation. This neoplasm is presumed to arise from a stem cell whose properties we have characterized and which differentiates in response to retinoic acid and dcAMP. In these cells these agents induce morphologic differentiation and growth arrest. Treatment of the TE 671 cell line with phorbol ester, dibutyrl cyclic AMP, produce clearcut but different morphologic effects within twenty-four hours of their continuous exposure to these agents. In addition, the TE 671 cells treated in this manner show different patterns of growth arrest which is of considerable theoretical importance.

9:00 AM

27. Onogene Activity in Human CNS Tumors

Timothy B. Mapstone, M.D.

Oncogenes are abnormal genes which seemingly play a role in the induction and maintenance of human neoplasia. They are derived from normal human genes known as proto-oncogenes. All oncogenes identified thus far are related to the pathways for cellular proliferation and growth control. Much evidence is available from tumors arising outside the CNS that these genes are central in the neoplastic process. Further work in these systems have begun to elucidate the structure and function of oncogenes and how they differ from their normal counterparts.

Thus far I have been unable to detect an isolated dominant acting oncogene from human CNS using NIH/3T3 cells as receptients which remains the standard assay to identify a dominant acting gene.

Analysis of mRNA expression of 15 CNS tumors provided further insight into oncogene activity. Platelet Derived Growth Factor (PDGF) A&B chain and Transforming Growth Factor beta (TGFB) are present at increased levels in astrocytomas, anaplastic astrocytomas and glioblastoma multiforme but not in meningiomas or ependymonas. When PDGFA is elevated TGFB is also but PDGFB is low. When PDGFB is elevated both PDGFA and TGFB are low. It appears as if higher levels of PDGFB are associated with the more malignant tumors.

Another series of experiments using 10 different tumors was carried out using multiple oncogene probes in an attempt to identify the range of activity. As expected tumors were shown to have elevated levels of TGFB, PDGFB, & A in addition to K-ras, fos and c-myc. There were very elevated levels of N-myc and N-ras detected in all tumors at about 20 times the amount of other oncogenes with N-ras generally twice as high as N-myc.

The possible implications of these findings for tumor behavior and control will also be discussed.

9:15 AM

28. Reoperation in the Management of Childhood Craniopharyngioma

Peter W. Carmel, M.D.

Even some advocates of initial radical removal of cranio-

pharyngiomas have felt re-operation was "precluded because of prohibitive morbidity and mortality". In the period from 1977 to 1986, 46 children and adolescents were operated for craniopharyngioma at the Neurological Institute of New York. This was the initial procedure for 36 children, while 10 had been treated elsewhere. Seven of the initial procedure group were subsequently reoperated. Thus, seventeen patients undergoing secondary operations form the basis of this report. They had previously undergone a total of 22 operations, and eight had been given x-ray therapy. (An additional patient, previously treated with proton-beam irradiation, will be shown).

All patients but one had radical tumor removal. There was no operative mortality. Two patients had decreased vision postoperatively, while vision was improved in eight. All patients had postop diabetes insipidus. One child is in a chronic care hospital. Total removal was felt to be obtained in nine cases; four children with subtotally removed tumors subsequently had x-ray therapy.

Our results indicate that neither prior operation nor x-ray therapy is an insurmountable obstacle to successful re-operation. The limits of reoperation will be described. Children with initial subtotal removal of craniopharyngioma are potentially curable, and radiation may be avoided or deferred.

9:30 AM Discussion

9:45 AM Recess

SCIENTIFIC SESSION VII
MODERATOR: Stewart B. Dunsker, M.D.

10:00 AM

29. The Effect of Blocker of Excitory Amino Acid Neurotransmitter on Focal Cerebral Ischemia
Keiji Sano, M.D., Akira Tamura, M.D., Takaaki Kirino, M.D.

The potential role of excitatory amino acid neurotransmitter on ischemic neuronal injury has recently come to be widely realized. We examined the effect of a non-competitive antagonist of excitatory amino acid receptor, dibenzocycloheptenemine (MK-801), on focal cerebral ischemia model in the rat. Twenty-four hours following ischemia, 4 coronal sections of the rat were stained with triphenyltetrazolium chloride method. The areas of infarction were measured

using a planimeter. In 7 rats, MK-801 (10mg/Kg) was given i.p. 30 minutes prior to ischemia, and saline was injected in the other 7 animals. The area of infarction in cerebral cortex was significantly decreased in the MK-801 treated group. These results show that the excitotoxic mechanism may be responsible for ischemic injury in the cortical neurons.

10:15 AM

30. Vasospasm: A Light at the End of the Tunnel
Bryce Weir, M.D.

In the past two decades the existence of angiographic vasospasm has been incontrovertibly demonstrated and its role in delayed ischemic neurologic deficits clearly delineated. Clinically it is now possible to ameliorate the effects of vasospasm in some cases by using hypotension and hypervolemia. It has also recently been demonstrated that the clinical outcome of patients following subarachnoid hemorrhage can be improved by the use of a calcium antagonist — Nimodipine. On the experimental front a primate model of chronic cerebral vasospasm which sometimes produces delayed ischemic neurological deficit has permitted the systematic testing of putative therapeutic agents. One such agent U-74, 006F has been shown to reduce the severity of chronic vasospasm. We have also been able to prevent vasospasm by mechanically removing blood within 48 hours or by using intrathecal tissue plaminogen activitator. We anticipate that clinical studies will shortly be underway for these agents.

10:30 AM

31. Injuries to the Vertebral Artery in Patients with Trauma to the Head and Neck
Willis E. Brown, M.D., Rebecca Barrett-Tuck, M.D., Holger E.I. Skerhut, M.D., Jim Story, M.D.

In patients sustaining injuries to the neck, injuries to the vertebral artery are relatively unusual; however, they may account for as many as 19% of vascular injuries in this setting. We are presenting our experience with 5 such patients and a review of 177 cases reported in the literature. Our experience and that of others confirms that the diagnosis and management of these injuries can be difficult, and, without the aggressive use of angiography, may not be achieved until the patient develops delayed symptoms and signs. Our illustrative cases include a patient with multiple

stab wounds who presented with delayed bleeding, a patient with gunshot wound and a spinal cord injury superimposed upon an arteriovenous fistula, a patient with a gunshot wound and a spinal cord injury in addition to occlusion of the vertebral artery, a patient with a gunshot wound that produced a pseudoaneurysm of the carotid artery as well as a vertebral arteriovenous fistula, and a patient with a C5-6 fracture dislocation who developed a delayed syndrome of vertebrobasilar insufficiency and a brain stem stroke related to occlusion of the vertebral artery.

The literature reports 122 cases of penetrating injury to the vertebral artery. 43% due to gunshot wounds, 22% to shell fragments, 22% to stab wounds, 10% to percutaneous angiography, and 3% to anterior cervical disectomy. There were 47 lacerations or transections, 53 arteriovenous fistulae, and 24 pseudoaneurysms. Fifty five cases of non-penetrating injury were encountered: chiropractic manipulation accounted for 40%; motor vehicle accidents and sports-related injuries were also frequent causes. As might be expected, dissection or occlusion of the vertebral artery was seen most frequently.

We shall discuss management of these patients and point out that increased awareness of these injuries in patients with trauma to the head and neck has led to more frequent use of angiography, earlier diagnosis, more appropriate definitive management, and significantly reduced morbidity and mortality.

10:45 AM

32. Hemifacial Spasm: Follow-up Assessment More than Five Years After Microvascular Decompression

Robert Wilkins, M.D., Charles Rawlings, M.D.

Forty-one patients treated for hemifacial spasm by a retromastoid craniectomy with microvascular decompression of the facial nerve were followed from 5 to 12 years postoperatively (average, 8.1 years). Of these patients, 29 have had complete relief of hemifacial spasm with no recurrence (excellent result), and 6 have had only occasional mild twitching, usually about the eye (good result). Three patients had initial relief of hemifacial spasm followed by recurrence beginning at 1, 6, and 54 months after operation, respectively; one of these patients was reexplored with mild improvement. One patient had partial improvement; after a second operation, complete relief of hemifacial spasm was

achieved. Two patients had no improvement; one of them obtained temporary relief with a second operation, but subsequently experienced a recurrence. At the time of final follow-up of the 41 patients, 30 had an excellent result, 6 had a good result and 5 had significant residual or recurrent hemifacial spasm. The results of treatment seem to hold up with time — there were only 3 recurrences of hemifacial spasm among the 41 patients who had an initially excellent or good result, 2 of which occurred within 6 months after the operation.

Of the surgical complications, all were temporary except for one instance of a residual hemiparesis and contralateral facial weakness that resulted from intraoperative air embolism with hypotension, two instances of postoperative anosmia (for which we have no explanation), three instances of moderate to profound ipsilateral acoustic nerve dysfunction, and one instance of an ipsilateral vocal cord paralysis. Since we have instituted intraoperative monitoring of auditory evoked potentials, no further patients have experienced a significant loss of hearing.

11:00 AM

33. Intraspinal Opiates — Results in Fifty Patients

Burton Onofrio, M.D.

Since the 1st patient treated with constant infusion of subarachnoid morphine for pain of malignant origin we have had experience with 49 others. Since 1981 some modifications in technique have diminished the risk of the procedure and expanded its use to the more terminally ill patients. Some criteria used for predicting value of its efficacy have been developed and with Tony Yaksh some speculation as to the failure of extremely high dose intrathecal opiates has been drawn.

11:15 AM

34. A New Philosophy in the Invasion Treatment of Trigeminal Neuralgia

William Sweet, M.D., D.Sc.

The extraordinary lability of the mechanisms responsible for trigeminal neuralgia is attested by the facts that minor intracranial manipulations in either the middle or posterior cranial fossa produce gratifying protracted periods of complete relief in most patients. Seven publications reporting 927 cases

treated by various "compression" or "decompression" procedures in the middle cranial fossa with long term followup are available to compare with 9 publications reporting 1232 patients with microvascular decompression (MVD) in the posterior fossa with somewhat shorter average followup. The totals of initial failures plus significant recurrences averaged 26% after the middle fossa procedure and 22.5% after MVD. Our own followups after even more minor manipulations extracranially, as well as intracranially in middle and posterior cranial fossas, with extraordinarly long periods of relief will also be described.

The peculiar unexplained propensity for the pain in this nonlethal disorder to stop upon modest to minor disturbances of tissues at or near the trigeminal pathways demands an initial conservative approach. The debat re the cause of the disorder need not be resolved in order logically to lead to this conclusion. It is immaterial whether the cause is demyelination of sensory trigeminal fibers, or extrinsic pressure against them, or both or neither. The simplest, safest way to perform the first minor manipulation seems to be the conservative percutaneous operation.

11:30 AM

35. Pain and Spinal Cysts in Paraplegia

B.S. Nashold, M.D., Jose Vieira, M.D.

The incidence of traumatic syringomyelia in paraplegics ranges from 0.3 to 2.3%. The most common sign of a developing spinal cord cyst may be an increase in the patient's neurological deficit. We would like to bring to the attention of neurosurgeons that the appearance of pain after spinal injury is often associated with cyst development and this can occur from 6 months to 12 years after the injury. The DREZ operation has been done in 81 paraplegics with intractable pain with improvement in 60% and 22% had an associated spinal cyst which was also drained. Four patients had multiple spinal cysts which were only detected by intraoperative ultrasound studies. Each separate cyst requires surgical treatment. The common neurosurgical practice in patients with spinal cysts is drainage of the cyst by various methods. When pain is a prominent part of the symptomatology, however, we do not think drainage alone is sufficient to give long lasting pain relief. The combination of DREZ plus drainage of the cyst has the best chance of long term pain relief.

11:45 AM Discussion

12:00 Adjourn

RESIDENTS PAPER AWARD WINNERS

WINNER

James T. Rutka, M.D.

Dept. of Neurosurgery
University of Toronto

"Isolation and Partial Purification of Transforming Growth Factors from Human Malignant Gliomas: Implications for the Pathogenesis of the Human Gliosarcoma"

RUNNERS UP

John H. Neal, M.D.

Dept. of Neurosurgery
University of Southern California in Los Angeles

"N-Methyl-D-Aspartic Acid Neurotoxicity during Development"

Donald R. Ross, M.D.

Dept. of Neurosurgery
University of California at San Francisco

"Expression of c-myc, n-myc, int-1 and c-arc protooncogenes in meduloblastoma: Implications for neurohistopathology"

ACADEMY AWARD WINNERS

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
Terry Lichtor 1985
Michael G. Nosko 1986
Joseph R. Madsen 1987

GUEST	*GUESTS OF*
Michael Apuzzo Los Angeles, California	Martin Weiss
Hunt Batjer Dallas, Texas	Kemp Clark
Henry Brem Baltimore, Maryland	Don Long
Richard B. Budde Cincinnati, Ohio	The Academy
Elizabeth Bullitt Durham, North Carolina	Frank Mayfield
Peter Carmel New York, New York	Russel H. Patterson, Jr.
Wu Cheng-yuan Jinan, China	M. Peter Heilbrun
Allan Friedman Durham, North Carolina	Blaine Nashold
Cordell Gross Burlington, Vermont	Robert King
Jeffrey Keller Cincinnati, Ohio	The Academy
Gerry King Albuquerque, New Mexico	Wolff Kirsch
Steven Kitai Memphis, Tennessee	The Academy
William Kuhn New Orleans, Louisiana	Raeburn Llewellyn
Sanford Larson Milwaukee, Wisconsin	Stewart B. Dunsker
Paul Lin Jenkintown, Pennsylvania	William Buchheit
Timothy Mapstone Cleveland, Ohio	Robert Ratcheson
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Dennis Spencer New Haven, Connecticut	William Collins
Charas Suwanwela Bangkok, Thailand	The Academy
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Harry vanLoveren Cincinnati, Ohio	John Tew
Allen Wyler Memphis, Tennessee	James Robertson
Hwa-shain Yeh Cincinnati, Ohio	The Academy

PAST PRESIDENTS

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

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William S. Keith 1942
John Raaf 1943
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John Raaf 1947
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F. Keith Bradford 1949
David L. Reeves 1950
Henry G. Schwartz 1951
J. Lawrence Pool 1952
Rupert B. Raney 1953
David L. Reeves 1954
Stuart N. Rowe 1955
Jess D. Herrmann 1956
George S. Baker 1957
Samuel R. Snodgrass 1958
C. Hunter Shelden 1959
Edmund Morrissey 1960
Donald F. Coburn 1961-62
Eben Alexander, Jr. 1963
George L. Maltby 1964
Robert Pudenz 1965
Francis A. Echlin 1966
Benjamin Whitcomb 1967
Homer S. Swanson 1968
Augustus McCravey 1969-70
Edward W. Davis 1971
John R. Green 1972
George J. Hayes 1973
Richard L. DeSaussure 1974
Ernest W. Mack 1975
Frank E. Nulsen 1976
Robert S. Knighton 1977
Robert G. Fisher 1978

Robert B. King 1979	H.T. Ballantine, 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	James W. Correll 1984
Thomas Langfitt 1985	E.B. Hendrick 1985
Phanor L. Perot, Jr. 1986	Griffith R. Harsh III 1986
Shelley N. Chou 1987	Ellis B. Keener 1987

PAST SECRETARY-TREASURER

Francis Murphey 1938-40	Eben Alexander, Jr 1954-57
A. Earl Walker 1941-43	Robert L. McLaurin 1958-62
Theodore C. Erickson 1944-47	Edward W. Davis 1963-65
Wallace B. Hamby 1948-50	Robert G. Fisher 1966-68
Theodore B. Rasmussen .. 1951-53	Byron C. Pevehouse 1969-72

PAST SECRETARY

Byron C. Pevehouse 1973
Russel H. Patterson, Jr ... 1974-76
Phanor L. Perot, Jr 1977-80
John T. Garner 1981-83
James T. Robertson 1984-86
Nicholas Zervas 1987----

PAST TREASURER

Russel H. Patterson, Jr 1973
Phanor L. Perot, Jr 1974-76
John T. Garner 1977-80
James T. Robertson 1981-83
Nicholas T. Zervas 1984-86
William Buchheit 1987----

PAST MEETINGS OF THE ACADEMY

Hotel Netherland Plaza, Cincinnati, Ohio ..... October 28-29, 1938
Roosevelt Hotel, New Orleans, Louisiana ..... 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 21-23, 1954
The Homestead, Hot Springs, Virginia ..... October 27-29, 1955
Camelback Inn, Phoenix, Arizona ..... November 8-10, 1956
The Cloister, Sea Island, Georgia ..... 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
The Key Biscayne, Miami, Florida ..... November 11-14, 1964
Terrace Hilton Hotel, Cincinnati, Ohio ..... October 14-16, 1965
Fairmont Hotel & Towers,
San Francisco, California ..... October 17-19, 1966
The Key Biscayne, Miami, Florida ..... November 8-11, 1967
Broadmoor Hotel, Colorado Springs, Colorado ..... October 6-8, 1968
St. Regis Hotel, New York City ..... September 21, 1969
Camino Real Hotel, Mexico City ..... November 18-21, 1970
Sahara-Tahoe Hotel, Stateline, Nevada ..... September 26-29, 1971
New College, Oxford, England ..... September 4-7, 1972
Huntington-Sheraton Hotel, Pasadena, California .. November 14-17, 1973
Southampton Princess Hotel,
Southampton, Bermuda ..... November 6-9, 1974
The Wigwam (Litchfield Park), Phoenix, Arizona ... November 5-8, 1975
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 Springs, 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
The Lincoln Hotel Post Oak, Houston, Texas ..... October 27-30, 1985
The Cloister, Sea Island, Georgia ..... November 5-8, 1986
Hyatt Regency, San Antonio, Texas ..... October 7-10, 1987

1988
MEMBERSHIP LIST
AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
FOUNDED OCTOBER, 1938

HONORARY MEMBERS	*ELECTED*
GUY LAZORTHES 26 Rue D Auriol 31 Toulouse, FRANCE	1973
VALENTINE LOGUE (Anne) 16 Rowan Road Hammersmith London W6 7DU ENGLAND	1974
GOSTA NORLEN (Gunvor) Linnegaten 35 IV 11447 Stockholm, SWEDEN	1973
BERNARD PERTUISET (Francoise) Hospital dela Pitie 83 Boulevard de l'Hospital 75651 Paris, Cedex 13 FRANCE	1986
KEIJI SANO (Yaeko) TEIKYO University Hospital 2-11-1 Kaga, Itabashi-ku Tokyo 173, JAPAN	1975

SENIOR MEMBERS	*ELECTED*
EBEN ALEXANDER, JR. (Betty) Bowman Gray School of Medicine 300 South Hawthorne Winston-Salem, North Carolina 27103	1950
GEORGE S. BAKER (Enid) 607 North Litchfield Road Litchfield Park, Arizona 85340	1940
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DONALD F. COBURN (Ellie) The Plaza 812 1303 Delaware Avenue Wilmington, Delaware 19806	1938
COURTLAND DAVIS (Carrie) Bowman-Gray School of Medicine Winston-Salem, North Carolina 27103	1967
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ELDON L. FOLTZ (Catherine) — 1960
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AUGUSTUS McCRAVEY (Helen) — 1944
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WILLIAM F. MEACHAM (Alice) — 1952
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FRANCIS MURPHEY (Marge) — Founder
3951 Gulf Shore Blvd. North
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WILLIAM H. SWEET (Elizabeth) 1950
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JOHN TYTUS (Virginia) 1967
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ALFRED UIHLEIN (Ione) 1950
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EXUM WALKER (Nelle) 1938
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W. KEASLEY WELCH (Elizabeth) 1957
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BENJAMIN B. WHITCOMB (Margaret) 1947
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ACTIVE MEMBERS	*ELECTED*
JAMES I. AUSMAN (Carolyn) Henry Ford Hospital 2799 West Grand Blvd. Detroit, Michigan 48202	1978
GILLES BERTRAND (Louise) Montreal Neurological Institute 3801 University Street Montreal, Quebec, Canada H3A 1B4	1967
ROBERT S. BOURKE (Marlene) 5802 Nicholson Lane Rockville, Maryland 20852-2967	1983
JERALD S. BRODKEY (Arielle) 24755 Chagrin Boulevard Suite #205 Beachwood, Ohio 44122	1977
WILLIS E. BROWN, JR. (Ann) Division of Neurosurgery The University of Texas Health Science Center 7703 Floyd Curl Drive San Antonio, Texas 78284-7843	1984
DEREK A. BRUCE (Frances) 7777 Forrest Lane, #C703 Dallas, Texas 75230	1984
WILLIAM A. BUCHHEIT (Lyn) 3401 North Broad Street Philadelphia, Pennsylvania 19140	1980
PAUL H. CHAPMAN (Tansy) Department of Neurosurgery Massachusetts General Hospital Boston, Massachusetts 02114	1983
SHELLEY N. CHOU (Jolene) University of Minnesota Medical Center 420 Delaware St. S.E., Box 96 Minneapolis, Minnesota 55455	1974
WILLIAM F. COLLINS, JR. (Gwen) Yale University School of Medicine 333 Cedar Street New Haven, Connecticut 06510	1963
EDWARD S. CONNOLLY (Elise) Ochsner Clinic 1514 Jefferson Highway New Orleans, Louisiana 70121	1973

JAMES W. CORRELL (Cynthia) 1966
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STEWART B. DUNSKER (Ellen) 1975
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HOWARD M. EISENBERG (Janet) 1985
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GRIFFITH R. HARSH, III (Craig) 1980
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MARK PETER HEILBRUN (Robyn) 1984
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E. BRUCE HENDRICK (Gloria) 1968
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ROBERTO C. HEROS (Deborah) 1985
Department of Neurosurgery
Massachusetts General Hospital
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CHARLES HODGE (Linda) 1982
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JULIAN HOFF (Diane) 1975
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Ann Arbor, Michigan 48104

HAROLD HOFFMAN (Jo Ann) 1982
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EDGAR M. HOUSEPIAN (Marion) 1976
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ALAN R. HUDSON (Susan) 1978
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JOHN A. JANE (Noella) 1982
Department of Neurosurgery, Box 212
University of Virginia
Charlottesville, Virginia 22908

JOHN P. KAPP (Lureese) 1985
406 North Main Street
Galax, Virginia 24333

ELLIS B. KEENER (Ann) 1978
915 East Lake Drive, NW
Gainesville, Georgia 30506

DAVID KELLY (Sally) 1975
Bowman Gray School of Medicine
300 S. Hawthorne
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WILLIAM A. KELLY (Joan) 1977
Department of Neurological Surgery
RI-20
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Seattle, Washington 98195

GLENN W. KINDT (Charlotte) 1977
Division of Neurosurgery
Box C-307
University of Colorado Medical Center
4200 East 9th Avenue
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WOLFF M. KIRSCH (Marie-Claire) 1971
531 Chamiso Lane, N.W.
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Louisiana State University Medical Center
1542 Tulane Avenue
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RICHARD S. KRAMER (Robin) 1978
Duke Hospital Medical Center
Durham, North Carolina 27710

THEODORE KURZE 1967
521 East 14th Street #11G
New York, New York 10009

EDWARD R. LAWS, JR. (Peggy) 1983
Geo. Washington Medical Center
2150 Pennsylvania Ave. NW
Washington, DC 20037

DONLIN M. LONG (Harriett) 1983
Department of Neurological Surgery
John Hopkins Medical School
601 N. Wolfe
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ALFRED J. LUSSENHOP 1976
Georgetown University Hospital
3800 Reservoir Rd.
Washington, D.C. 20007

ERNEST W. MACK (Bobbie) 1956
505 South Arlington Avenue
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M. STEPHEN MAHALEY, JR. (Jane) 1972
Division of Neurosurgery
UAB Station
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LEONARD MALIS (Ruth) 1973
1176 Fifth Avenue
New York, New York 10029

JOHN F. MULLAN (Vivian) 1963
5844 Stoney Isle Avenue
Chicago, Illinois 60637

FRANK E. NULSEN (Ginny) 1956
32 10th Avenue, South
Naples, Florida 33940

GEORGE OJEMANN (Linda) 1975
6424 E. Mercer Way
Mercer Island, Washington 98040

ROBERT G. OJEMANN (Jean) 1968
Neurosurgery Service
Massachusetts General Hospital
Boston, Massachusetts 02114

BURTON ONOFRIO (Judith) 1975
Mayo Clinic
Rochester, Minnesota 55905

RUSSEL H. PATTERSON, JR. (Julie) 1971
New York Hospital
525 East 68th Street
New York, New York 10021

S.J. PEERLESS (Ann) 1977
University Hospital
339 Windermere Road
London, Ontario, Canada N6A 5A5

PHANOR L. PEROT, JR. 1970
Department of Neurosurgery
Medical University of South Carolina
171 Ashley Avenue
Charleston, South Carolina 29425

BYRON C. PEVEHOUSE (Lucy) 1964
2351 Clay St.
San Francisco, CA 94115

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200 First Street, S.W.
Rochester, MN 55905

DONALD O. QUEST (Ilona) 1986
The Neurological Institute
710 West 168th Street
New York, New York 10032

ROBERT A. RATCHESON (Peggy) 1986
University Hospital
2074 Abington Road
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ALBERT L. RHOTON, JR. (Joyce) 1984
University of Florida, Box J265
Department of Neurosurgery
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J. CHARLES RICH, JR. (Jasmine) 1987
324 10th Ave. #206
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HUGO RIZZOLI (Helen) 1973
2150 Pennsylvania Avenue, N.W.
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THEODORE S. ROBERTS (Joan) 1976
Dept. of Neurological Surgery
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JAMES T. ROBERTSON (Valeria) 1971
Department of Neurosurgery
University of Tennessee, Memphis
956 Court Avenue
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FREDRICK A SIMEONE (Kate) 1981
Pennsylvania Hospital
800 Spruce Street
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JAMES C. SIMMONS (Vanita) 1975
920 Madison Avenue
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KENNETH R. SMITH, JR. (Marjorie) 1987
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JIM L. STORY (Joanne) 1972
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THORALF M. SUNDT, JR. (Lois) 1971
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RONALD R. TASKER (Mary) 1971
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DECEASED MEMBERS		*ELECTED*
SIXTO OBRADOR ALCALDE Madrid, Spain (Honorary)	4/1978	1973
JAMES R. ATKINSON Phoenix, Arizona (Active)	2/1978	1970
PERCIVAL BAILEY Evanston, Illinois (Honorary)	8/1973	1960
WILLIAM F. BESWICK Buffalo, New York (Active)	5/1971	1959
EDWIN B. BOLDREY San Francisco, California (Senior)	6/1988	1941
SPENCER BRADEN Cleveland, Ohio (Active)	7/1969	Founder
F. KEITH BRADFORD Houston, Texas (Active)	4/1971	1938
JUAN CARDENAS Mexico (Corresponding)	1987	1966
WINCHELL McK. CRAIG Rochester, Minnesota (Honorary)	2/1960	1942
FRANCIS ECHLIN New Paltz, New York (Senior)	4/1988	1944
GEORGE EHNI Houston, Texas (Senior)	9/1986	1964
ARTHUR ELVIDGE Montreal, Quebec, Canada (Senior)	1/1985	1939
THEODORE C. ERICKSON Madison, Wisconsin (Senior)	10/1986	1940

JOSEPH P. EVANS Kensington, Maryland (Senior)	5/1985	Founder
WESLEY A. GUFTAFSON Jensen Beach, Florida (Senior)	7/1975	1942
HANNIBAL HAMLIN (Senior)	6/1982	1941
HENRY L. HEYL (Senior)	3/1975	1951
OLAN HYNDMAN Iowa City, Iowa (Senior)	6/1966	1942
KENNETH G. JAMIESON Brisbane, Australia (Corresponding)	1/1976	1970
SIR GEOFFREY JEFFERSON Manchester, England (Honorary)	3/1961	1951
WILLIAM KEITH Toronto, Canada (Senior)	12/1987	Founder
HUGO KRAYENBUHL Zurich, Switzerland (Honorary)	1985	1974
WALPOLE S. LEWIN Cambridge, England (Corresponding)	1/1980	1973
HERBERT LOURIE Syracuse, New York (Senior)	3/1987	1965
GEORGE L. MALTBY Scarsborough, Maine (Senior)	1988	1942
DONALD D. MATSON Boston, Massachusetts (Active)	5/1969	1950
KENNETH G. McKENZIE Toronto, Ontario, Canada (Honorary)	2/1964	1960

JAMES M. MEREDITH Richmond, Virginia (Active)	12/1962	1946
W. JASON MIXTER Woods Hole, Massachusetts (Honorary)	3/1968	1951
EDMUND J. MORRISSEY San Francisco, California (Senior)	2/1986	1941
HANS-WERNER PIA West Germany (Corresponding)	7/1986	1978
WILDER PENFIELD Montreal, Canada (Honorary)	4/1976	1960
HELMUT PENZHOLZ West Germany (Corresponding)	1985	1978
RUPERT B. RANEY Los Angeles, California (Active)	11/1959	1939
DAVID L. REEVES Santa Barbara, California (Senior)	8/1970	1939
DAVID REYNOLDS Tampa, Florida (Active)	4/1978	1964
R.C.L. ROBERTSON Houston, Texas (Senior)	2/1985	1946
STEWART N. ROWE Pittsburgh, Pennslyvania (Senior)	10/1984	1938
RICHARD C. SCHNEIDER Ann Arbor, Michigan (Senior)	6/1986	1970
WILLIAM B. SCOVILLE Hartford, Connecticut (Senior)	2/1984	1944

R. EUSTACE SEMMES Memphis, Tennessee (Honorary)	3/1982	1955
SAMUEL R. SNODGRASS Nashville, Indiana (Senior)	8/1975	1939
GLEN SPURLING LaJolla, California (Honorary)	2/1968	1942
C. WILLIAM STEWART Montreal, Quebec, Canada (Corresponding)	1948	1948
HENDRIK SVIEN Rochester, Minnesota (Active)	6/1972	1957
HOMER S. SWANSON Atlanta, Georgia (Senior)	6/1987	1949
THOMAS A. WEAVER, JR. Dayton, Ohio (Senior)	1985	1943
BARNES WOODHALL Durham, North Carolina (Senior)	1985	1941

THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY 1988 ANNUAL MEETING EVALUATION

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

(1) Was the general context of the scientific program:
- ☐ Excellent
- ☐ Good
- ☐ Poor

(2) If you found it poor, was it because:
- ☐ Too much review of old knowledge?
- ☐ Too simple or elementary?
- ☐ Too complex or abstruse?
- ☐ Of little practical value?

(3) Did the speakers aim their talks:
- ☐ Too high
- ☐ Too low
- ☐ Just about right

SCIENTIFIC PROGRAM

Thursday's Sessions ☐ Excellent ☐ Good ☐ Poor
Comments _____

_____

Friday's Sessions ☐ Excellent ☐ Good ☐ Poor
Comments _____

_____

Saturday's Sessions ☐ Excellent ☐ Good ☐ Poor
Comments _____

_____

SOCIAL PROGRAM

Comments _____

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

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Changes of address and/or telephone
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