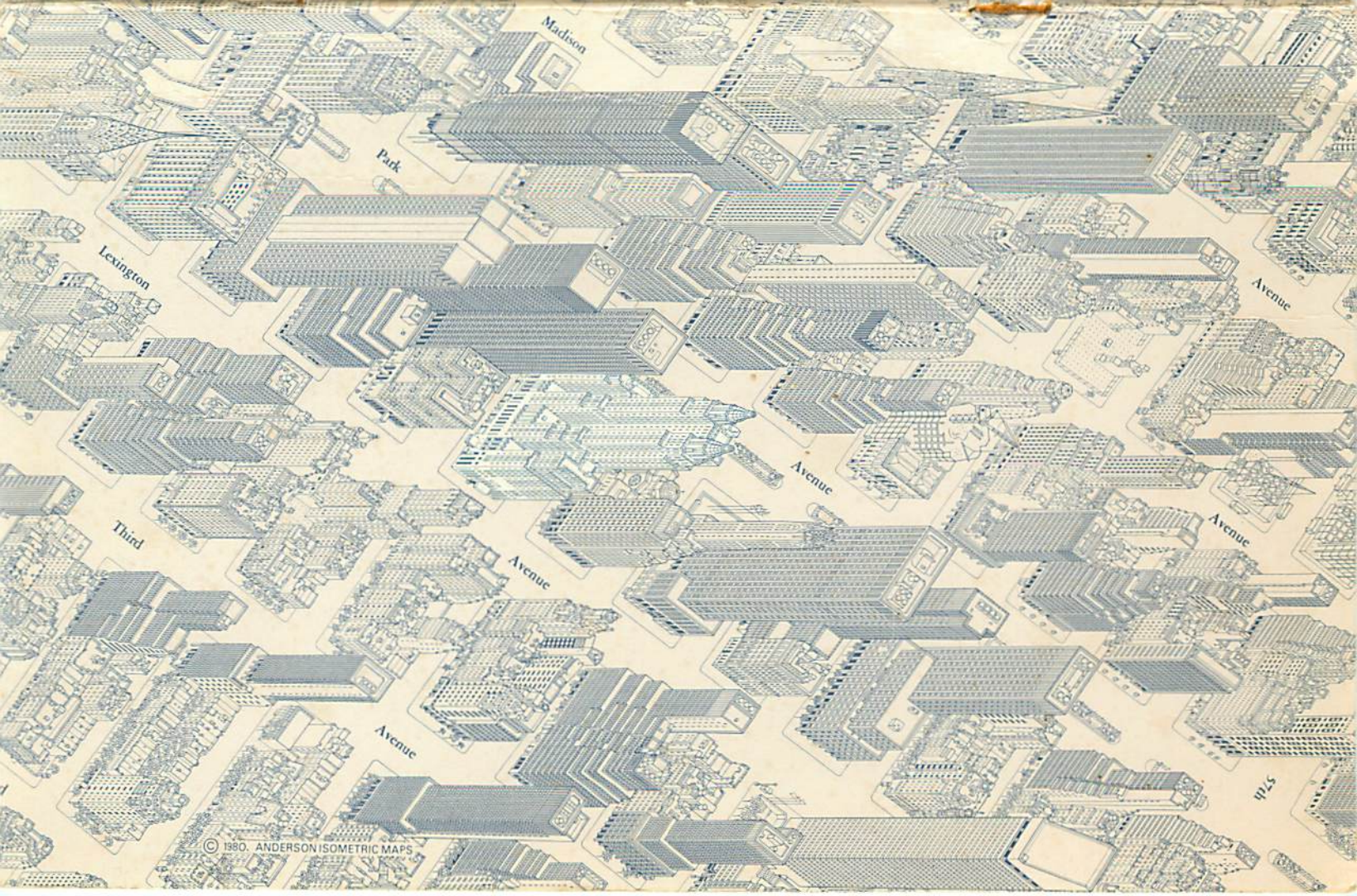
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JOINT MEETING 1980

Deutsche Gesellschaft Für Neurochirurgie
The American Academy of Neurological Surgery

The Waldorf-Astoria
New York, New York
October 1-4, 1980

DEUTSCHE GESELLSCHAFT FÜR NEUROCHIRURGIE

1980
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Local Hosts

Dr. and Mrs. Russel H. Patterson, Jr.
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PROGRAM 1980

REGISTRATION (The Terrace Court-Off Park Avenue Lobby)

Wednesday, October 1	2:00 - 6:00 p.m.
Thursday, October 2	8:00 - 10:00 a.m. 2:00 - 4:00 p.m.
Friday, October 3	8:00 - 10:00 a.m.

WEDNESDAY, OCTOBER 1

6:00 - 8:00 p.m.	Welcoming Cocktail Party The Empire Room (Off Park Avenue Lobby)

THURSDAY, OCTOBER 2

–ALL SCIENTIFIC SESSIONS WILL BE HELD IN THE EMPIRE ROOM WHICH IS LOCATED O FF PARK AVENUE LOBBY
–POSTER SESSION – POSTER PRESENTATIONS WILL BE ON DISPLAY OUTSIDE THE SCIENTIFIC MEETING AREA FOR YOUR INSPECTION THROUGHOUT THE MEETING.

7:00 - 8:15 a.m.	Breakfast and Business Meeting (Academy Members Only) The Conrad Salon, 4th Floor
8:30 - 8:40 a.m.	Opening Remarks by Hans-Peter Jensen, Pres., Deutsche Gessellschaft Für Neurochirurgie and by Eben Alexander, Jr., Pres., American Academy of Neurological Surgery (The Empire Room)
8:40 - 10:20 a.m.	Scientific Session
10:20 - 10:40 a.m.	Coffee Break
10:40 a.m. - 12:20 p.m.	Scientific Session
12:20 - 2:00 p.m.	Luncheon (Academy and German Society Members and Guests) The Hilton Room (Off Park Avenue Lobby)
2:00 - 3:40 p.m.	Scientific Session

3:30 - 3:50 p.m.	Coffee Break
3:50 - 4:50 p.m.	Scientific Session
6:00 - 11:00 p.m.	Dinner and Theatre Party: West Side Story Bus Departure 5:45 p.m., 49th Street Entrance

FRIDAY, OCTOBER 3

7:00 - 8:15 a.m.	Breakfast and Business Meeting (Academy Members Only) The Conrad Salon, 4th Floor
8:30 - 10:10 a.m.	Scientific Session
10:10 - 10:30 a.m.	Coffee Break
10:30 - 11:30 a.m.	Scientific Session
11:30 a.m. - 12:00 noon	Presidential Address
6:30 - 7:30 p.m.	Cocktail Reception The Hilton Room (Off Park Avenue Lobby)
7:30 - 11:30 p.m.	Dinner Dance (Black Tie) The Starlight Roof, 18th Floor

SATURDAY, OCTOBER 4

8:00 - 9:00 a.m.	Breakfast and Business Meeting (Academy Members Only) The Conrad Salon, 4th Floor
9:15 - 10:35 a.m.	Scientific Session
10:35 - 10:55 a.m.	Coffee Break
10:55 a.m. -12:35 p.m.	Scientific Session

LADIES PROGRAM 1980

REGISTRATION (The Terrace Court-Off Park Avenue Lobby)

Wednesday, October 1	2:00 - 6:00 p.m.
Thursday, October 2	8:00 - 10:00 a.m. 2:00 - 4:00 p.m.
Friday, October 3	8:00 - 10:00 a.m.

WEDNESDAY, OCTOBER 1

6:00 - 8:00 p.m.	Welcoming Cocktail Party The Empire Room (Off Park Avenue Lobby)

THURSDAY, OCTOBER 2

8:00 - 11:00 a.m.	Ladies Hospitality The Terrace Court (Off Park Avenue Lobby)
9:30 a.m. - 12:00 noon	Manhattan Tour I Meet at 9:15 a.m., Park Avenue Entrance Manhattan Tour II Meet at 8:30 a.m., Park Avenue Entrance
12:00 noon - 1:45 p.m.	Luncheon - Windows on the World Departure for P.M. Tours I & II from here
1:45 - 3:30 p.m.	P.M. Tour I
1:45 - 4:30 p.m.	P.M. Tour II
6:00 - 11:00 p.m.	Dinner and Theatre Party: West Side Story Bus Departure 5:45 p.m., 49th Street Entrance Dinner at The Rainbow Grill, Rockefeller Center

FRIDAY, OCTOBER 3

8:00 - 11:00 a.m.	Ladies Hospitality The Terrace Court (Off Park Avenue Lobby)
9:15 a.m. - 12:00 noon	Behind-the-Scenes Tour of Fifth Avenue Shops and Radio City Music Hall Departure 9:00 a.m., Park Avenue Lobby

11:30 a.m. - 12:00 noon	Presidential Address The Empire Room (Off Park Avenue Lobby)
6:30 - 7:30 p.m.	Cocktail Reception The Hilton Room (Off Park Avenue Lobby)
7:30 - 11:30 p.m.	Formal Dinner Dance The Starlight Roof, 18th Floor

SATURDAY, OCTOBER 3

8:00 - 11:00 a.m.	Ladies Hospitality The Terrace Court (Off Park Avenue Lobby)

SCIENTIFIC PROGRAM

SCIENTIFIC PROGRAM
DEUTSCHE GESELLSCHAFT FÜR NEUROCHIRURGIE
THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
JOINT MEETING
New York, New York
October 1 - 4, 1980

MODERATORS: Hans-Peter Jensen
Eben Alexander, Jr.

THURSDAY, OCTOBER 2

8:30 a.m. Opening Remarks
Hans-Peter Jensen, President, Deutsche Gesellschaft Für Neurochirurgie
Eben Alexander, Jr., President, The American Academy of Neurological Surgery

8:40 a.m.

1. TRANSFRONTAL PITUITARY TUMOR OPERATION

Results 20 years before and 10 years after the microsurgical operation technique
O. Wilcke
Köln

All patients with suprasellar and parasellar developed pituitary tumors were operated on transfrontally; 441 patients were operated on without the microscope and beginning in 1970, 186 patients were operated on with the aid of the microscope, whereby in complete as possible removal of the tumor, including extirpation of the tumor capsule, was attempted. Before the use of the microscope, a recurrence of 23.5% was observed. After the microsurgical operation, anl 1% recurrence appeared. The mixed adenomas had the highest rate of recurrence with 34% in comparison to a rate of 19% among chromophobe adenomas and 9.6% among eosinophil adenomas. The operation mortality rate is 15% and with recurring adenomas, 27%. Two-thirds of the recurrences have been observed within the first five years. After the microsurgical operation the most (15%) appeared with chromophobe adenomas. The tendency to recur will be discussed as well as the possibility of recurring prophylaxis and rehabilitation.

(Discussion)

9:00 a.m.

2. SPECIAL PROBLEMS IN THE TREATMENT OF GIANT PITUITARY ADENOMAS

E. Halves, M. Gaab and K.–A. Bushe
Würzburg

Advancing endocrinological methods of examination induced new aspects of the pathophysiological conditions of pituitary tumors. Especially the control of our functional results in the treatment of hypersecreting microadenomas led to better and better microsurgical techniques of selective operations, so that for the time being there are less problems treating micro- than macroadenomas and the "giant adenomas" are still the most problematic ones.

During the last 4 years we gained experience with a combination of operative techniques in the treatment of about 70 macroadenomas including 20 giant tumors. The characteristic technical details are:

–an upright sitting positioning of the patient
–a transrhinoseptal approach
–a perioperative x-ray image intensifier and camera (70mm)
–operation microscope and special endoscopic and microsurgical equipment
–controlled lumbar air insufflation
–two-stage operations (transsphenoidal – transcranial).

In correlation to this technique we want to discuss the following aspects:

–indications for the different techniques in correlation to radiological and endocrinological findings
–advantage, limits and special risks of operation techniques
–indications for two-stage operations
–diagnosis and treatment of CSF-leakage
–postoperative treatment (irradiation, Bromocriptine therapy, replacement).

(Discussion)

9:20 a.m.

3. MICROADENOMECTOMY IN CUSHING'S DISEASE

R. Fahlbusch, F. Marguth
München

In the last eight years 31 patients with ACTH-dependent Cushing's disease and normal or slightly enlarged sellae were operated upon the pituitary via the transsphenoidal route.

Microadenomas could be removed selectively in 28 patients, whereas in 3 patients no adenoma could be detected – one patient with hyperplasia was hypophysectomized, two other patients underwent bilateral adrenalectomy.

Further, 3 patients were adrenalectomized in whom no clinical remission occurred although microadenomectomy was performed. All the other 25 patients showed clinical remission with hormonal remission – up to now documented in 20 patients – without necessity of long term hormonal replacement therapy.

ACTH-monitoring (O.A. Müller, Medizinische Klinik Innenstadt der Universität Munchen): Perioperative ACTH-levels (N=20) are helpful for the prognosis of operative therapy of pituitary ACTH-excess. In the majority of patients in remission a transitory secondary adrenal insufficiency was observed whereas still elevated ACTH-levels suggested an operative failure.

Immunocytochemistry (H.L. Fehm, K.H. Voigt, Medizinische Klinik der Universität Ulm): Besides light – and electron-microscropy the immunocytochemical occurence of ACTH, β-endorphin/β-lipotropin, α-MSH and 16-K-fragment were studied in tissue of the adenoma and of its "normal" surroundings (N=13). The results show that the removed microadenomas contained the active corticotrophic cells, responsible for the inappropriate ACTH-and β-endorphin hypersecretion in Cushing's disease.

Further observations of the patients may solve the problem if the remaining corticotrophs in the pituitary will be able to develop a recurrent Cushing's disease. Up to now in the majority of cases a primary pituitary defect has to be assumed.

(Discussion)

9:40 a.m.

4. **IN VITRO ASSAY OF CHEMOSENSITIVITY IN PATIENTS WITH MALIGNANT GLIOMAS**

D.G.T. Thomas, J.L. Darling, and D.E. Bullard
London

Clinically it has frequently been noted that even histopathologically similar gliomas do not uniformly respond to chemotherapeutic agents. This may be due to heterogeneity in response of tumor cells rather than to variation in the tumor bearing patients.

A technique has been developed, using scintillation autofluorescence, for measuring the chemosensitivity of gliomas in vitro. Samples of tumor were obtained at the time of surgery from patients with glioma. These were tested against a panel of twelve chemotherapeutic drugs including three, Vincristine, Procarbazine, and CCNU, which were used in subsequent chemotherapy of the patients with glioma. A wide variation apparent in in vitro chemosensitivity between similar grades of glioma was observed. The clinical response was evaluated in patients with glioma undergoing chemotherapy with the triple agent

regime, and classified as 'Responder' or 'Non- responder'. The correlation between in vitro and clinical response was determined and in preliminary analysis in a small series, there appears to be close correlation between response in vivo and in vitro.

(Discussion)

10:00 a.m.

5. COMPREHENSIVE REVIEW OF POSTOPERATIVE THERAPEUTICS FOR MALIGNANT GLIOMAS, WITH REPORT OF A CONTROLLED RANDOMIZED CLINICAL TRIAL OF LEVAMISOLE IMMUNOSTIMULATION

M.S. Mahaley, Jr.
Durham

The various postoperative adjunctive measures for therapy of malignant gliomas will be briefly reviewed: radiotherapy (conventional and interstitial), chemotherapy (systemic and regional), immunotherapy (non-specific and specific), and hyperthermia. A randomized, controlled clinical trial involving 100 patients with anaplastic gliomas will be reported. Each patient was treated with whole head radiotherapy and systemic bimonthly BCNU chemotherapy. Patients were randomly selected for treatment with levamisole immunostimulation. The results of the baseline immunological screening studies will be reported, which demonstrate the degree of cellular and humoral immune impairment characteristic of these patients. Serial immune screening studies every two months during treatment revealed no significant improvement in delayed hypersensitivity reactions, peripheral blood lymphocyte counts, peripheral blood T-lymphocyte quantities, or serum immunoglobulins in patients receiving levamisole versus those who did not. The median survival times for the two groups were similar. Those patients with glioblastoma multiforme tended to have shorter survival times than those with other types of malignant gliomas, much as one would expect. An important and potentially lethal toxic side effect of BCNU chemotherapy--pulmonary interstitial fibrosis--was studied in depth in this series of patients and has resulted in the generation of a discriminant functional analysis which predicts with 80% accuracy those patients who are at risk; an algorithm has been formulated to guide what is now felt to be a more logical and safe utilization of BCNU. A currently ongoing Phase I clinical protocol which involves active immunization of patients with malignant gliomas will be described and the results to date reported. This clinical study currently includes 10 patients with malignant glioma who are being actively immunized with a combination of irradiated glioma cells, BCG-cell wall preparation, and levamisole immunostimulation in a protocol also incorporating radiotherapy and BCNU chemotherapy.

(Discussion)

10:20 a.m. Coffee Break

10:40 a.m.
6. **THE THERAPY OF MALIGNANT GLIOMAS: WHERE DO WE GO FROM HERE?**

Charles B. Wilson
San Francisco

Prospects for the patient harboring a malignant glioma today are slightly better than in 1970, but it is sobering that the difference is not obvious without the application of statistical analysis. A treatment that is highly effective would not require large-scale trials and the application of statistical methods.

Although the past decade has brought disappointingly little in the way of an improved outlook for the patient with a newly diagnosed glioblastoma, lessons learned in the laboratory and in the course of clinical trials place today's investigators in a position to move forward with some assurance that rational, as opposed to empirical, approaches can be pursued. At this point it does not seem likely that any future clinical trials will be regressive.

Looking ahead into developments anticipated within the next few years, our group is counting heavily on the implantation of radioactive seeds, intratumoral chemotherapy, the pursuit of radiosensitizers, and a major effort to encourage the development of drugs that have pharmacological characteristics that we believe are important. Using currently available methods, it is now possible to carry out limited sensitivity studies on a freshly removed tumor, and the matching of specific treatment to specific tumors has become a reality.

While the rate of progress has been disappointing, it has been progress nonetheless. Although those of us involved in brain tumor research do not see any dramatic developments around the corner, we have little doubt that over the course of the next several years, the patient found to harbor a malignant brain tumor will look forward to a better and longer life than we can offer patients whom we are treating now.

(Discussion)

11:00 a.m.
7. **RESECTION OF VENTRICULAR TUMORS IN TUBEROUS SCLEROSIS**

E. Kazner, F. Marguth and
A. Kollmannsberger
Berlin

The charecteristic ventricular tumors found in tuberous sclerosis are most

often located in the anterior portions of the lateral ventricles and sometimes cause a sudden increase in intracranial pressure due to blockage of the foramen of Monro. The classic triad of symptioms comprises headache, vomiting and papilledema. The tumors, classified histologically as subependymal giant cell astrocytomas, are not necessarily associated with the fully developed clinical picture of tuberous sclerosis. Thus there may be no history of seizures.

Today the diagnosis is established by means of computerized tomography, which demonstrates the tumor and its relations to the head of the caudate nucleus and the foramen of Monro. Hydrocephalus is also evident, and some cases show calcification at the ventricular walls.

In the past five years we have treated 9 patients (8 children and adolescents, 1 adult) with tumors located in the anterior horn of the lateral ventricles and orginating in the region of the head of the caudate nucleus. Complete resection of the tumor was attempted in every case and achieved in 7, as CT follow-up studies demonstrated. In 8 cases the tumor was approached through the frontal cortex and through the corpus callosum in one case with a tumor on the left side.

Results were satisfactory to excellent in 8 cases. One child with an exceptionally large tumor died of hypothalamic dysfunction several weeks after direct surgery with total resection of the tumor.

Microsurgical technique allows total removal of giant cell astrocytomas almost always with complete preservation of adjacent structures, since the tumors are often loosely connected to their matrix and demonstrate a clear demarcation line between tumor and normal brain structures. The only sequelae directly related to the removal of the tumor in our series were slight temporary hemiparesis in one case and the disorders of hypothalmic function in the case mentioned above. Associated hydrocephalus is often severe and usually requires at least temporary external if not permanent CSF drainage.

(Discussion)

11:20 a.m.

8. THE PROBLEM OF MULTIPLE INTRACRANIAL TUMORS

W. Piotrowski, K. Tornow and M. Kröger
Mannheim

Several cases with multiple intracranial tumors of the same or of different blastodermic layers will be analyzed considering the known literature on the subject. The angiograms and CT scans will be presented. Among these cases is a woman with bilateral pontine-angle tumors and an additional suprasellar tumor. The clinical course and the indications for surgery will be described in particular. The differential diagnosis between original brain tumors and metastatic tumors in patients who were not operated on will be discussed. Finally the survival time will be reported.

(Discussion)

11:40 a.m.

9. **STEREOTAXIC BIOPSY AND RADIOSURGERY WITHIN THE CT-SCANNER (with a new targeting device)**

W. Huk
Erlangen

Computerized tomography (CT) provides us with detailed information about the localization and the shape of even small intracranial lesions. In combination with stereotaxy this information enables us to remove biopsy specimens from different parts of the same lesion for more reliable histological diagnoses in inoperable brain tumors. In the same manner the accurate interstitial implantation of radioactive isotopes is made possible.

With the help of a new targeting device, these stereotaxic procedures can be performed within the CT-scanner (head or body) under CT-control. By this means the origin of the biopsy specimen and the localization of the radioactive implants can be controlled and documented easily, and intraoperative complications can be detected immediately.

So far, no complications were observed due to the fact that these operative procedures were performed outside the operating room in the CT-unit.

A description of the procedure and the first results in about 30 cases are presented.

(Discussion)

12:00 noon

10. **GLIAL MEMBRANE POTENTIALS AND THEIR RELATIONSHIP TO (K+)o IN GUINEA PIG AND MAN: A COMPARATIVE STUDY OF INTRACELLULARLY MARKED NORMAL, REACTIVE AND NEOPLASTIC GLIA**

S. Picker, C.F. Pieper, and S. Goldring
St. Louis

Cells were studied in vitro using physiologically viable brain slices. After recording the resting membrane potential (RMP) or the relationship of RMP to changes in (K+)o in the cells with very stable RMPs, the cell was injected iontophoretically with HRP for later visualization and correlation with the physiologic data.

Normal glial cells were studied in cortical tissue obtained from guinea pig (GP), and in man from cortex overlying tumor tissue or from an epileptogenic focus where the tissue was microscopically normal but electrically abnormal.

Reactive glial cells were studied in cortical slices from human epileptogenic foci which showed neuronal drop-out and astrocytosis. Neoplastic cells were studied in tissue obtained from one human glioblastoma multiforme and one mixed glioma.

The mean RMP of 416 GP glia was -70 ± 7 mV. In 80 cells from normal human cortex the mean RMP was -69 ± 8 mV. The cells in both GP and man were protoplasmic astrocytes. The average RMP of 135 reactive glia was -67 ± 9 mV. The cell population consisted of both protoplasmic and fibrous astrocytes. The latter had long tortuous processes exhibiting varying degrees of swelling and irregular nodules.

In the glioblastoma multiforme the mean RMP from 53 cells was -32 ± 7 mV. These cells were large, varied in size, and resembled more the fibrous astrocytes. The labeled cells from the mixed glial tumor showed oligodendroglia, and astroglia resembling some of the glioblastoma cells. The average RMPs of the astroglial and oligodendroglial cells were -50 ± 11 mV and -53 ± 3 mV respectively.

In 22 GP protoplasmic astrocytes, the RMP was recorded while the perfusate (4 mM K+) was exchanged for solutions having (K+) of 7-12-20 and 40 mM. The slope of the RMP vs. log (K+)o was 59 ± 2 mV/dec, approximately the value predicted by the Nernst equation if glia are permeable only to K+. Similarly, in 12 human protoplasmic astrocytes from normal cortex a slope of 57± 5 mV/dec was obtained when (K+)o was changed from 4-12-20-40 mM. In 12 human reactive glia, however, a similar change in (K+)o yielded a slope of 32 ± 8 mV/dec.

The accumulating data demonstrate the utility of combining the in vitro brain slice technique with intracellular marking for characterizing physiological properties of normal, reactive and neoplastic glia in the human brain.

(Discussion)

12:20 p.m. **Luncheon**

MODERATORS: Hermann Dietz
George Ehni

2:00 p.m.

11. VISUAL FIELD DEFECTS IN CASES OF CEREBRAL GIANT ANEURYSMS

G. Lausberg
Bochum

Among the 123 patients affected by cerebral aneurysms at the Neurosurgical University Clinic Bochum-Langendreer from 1976 to March 1980, eight presented with giant aneurysms (more than 25 mm in diameter). Three of

the cases involved the anterior communicating artery, the rest of them were localized in the region of the internal carotid artery. As a result, ocular symptoms were clearly predominant in the clinical picture of 5 cases, and in 4 cases they constituted the primary symptoms. Three cases presented a unilateral amaurosis of five to ten years standing. In another 3 cases psychic changes had developed. Subarachnoid haemorrhage as a minor episode without disturbances of consciousness was only found twice in the medical history. Only in one case was a cerebral seizure the primary symptom. All eight cases were operated on following computerized tomography and angiography; two of the patients died. The problems associated with late dignosis and the cause of death in relation to the localization of the giant aneurysm are discussed.

(Discussion)

2:20 p.m.

12. TRANSVASCULAR TREATMENT OF VASCULAR ABNORMALITIES OF THE CAROTID, VERTEBRAL, AND INTRACEREBRAL CIRCULATIONS

Alex Berenstein, Joseph Ransohoff,
Eugene Flamm, and Irvin I. Kricheff
New York

The management of vascular abnormalities involving the carotid, vertebral, and intracerebral circulations has undergone a radical change thanks to the introduction of transvascular occlusive techniques of embolization. The use of balloon catheters of different designs permits cannulation of distal tortuous vessels previously unreachable. The use of balloons that can detach at a predetermined site and can occlude abnormal arteriovenous communications or large surgically unreachable aneurysms, and the introduction of low viscosity tissue adhesives such as isobutyl-2-cyanoacrylate, which polymerizes immediately after contact with ionic solutions such as blood or with the intima of a vessel, have permitted the occlusion of high flow lesions in a more precise manner. Particulate emboli such as silicone spheres still remain a valuable tool for palliation in some large high sump malformations when subselective catheterization cannot be accomplished.

This report is based on 63 patients who underwent 83 embolization procedures in the last 3 years. Seven complications occurred, including two deaths, one permanent supratentorial infarct, one permanent posterior fossa stroke and two transitory infarcts with 90% recovery in one patient and complete recovery in the other. A spinal cord infarction occurred in one case.

This group includes patients with 35 cerebral arteriovenous malformations in whom 57 embolization procedures were performed; 2 with aneurysmal dilatations of the vein of Galen, 2 with purely dural AVMs, 3 with spinal cord

AVMs, 3 with hemangiomas of a vertebral body, 10 with carotid cavernous fistulas, 6 with hemangiomas of a vertebral body, 10 with carotid cavernous fistulas, 1 with a giant aneurysm of the vertebral artery and 1 with a cavernous carotid aneurysm.

The catheterization capabilities and occlusion will be demonstrated, the complications will be analyzed.

(Discussion)

2:40 p.m.

13. OPERATIVE INDICATIONS IN COMPLETE STROKE

C.U. Sprick, W.-J. Bock, M. Chirmer, H.U. Thal, D.P. Lim
Duesseldorf

In spite of the general restriction to do extra-intra-cranial bypass operations in complete stroke, we performed a number of these operations in our department. Follow-up studies showed good results and clinical improvements in a high percentage of cases.

Since January 1979, our procedure in Duesseldorf is as follows: we attempt to admit patients for diagnostic measures as early as possible to our department; angiography and cranial computerized tomography are always performed.

Should routine investigations reveal extracranial stenosis of the internal carotid artery the vascular surgeon conducts disobliteration of the vessel. In complete occlusion of the middle cerebral artery operative disobliteration should always be attempted by the neurosurgeon within six hours after the incident. In all other cases of TIA, RIND, PRIND and even progressive and complete stroke extra-intracranial bypass operations are done.

In cases of complete stroke thrombocyte aggregation is reduced by acetylsalicylic acid. Should CT reveal a fresh hypodense area corresponding to the occluded vessel extra-intracranial anastomosis is not carried out immediately because of the risk of hemorrhagic infarction.

Looking at the good results of our procedure we would like to discuss whether the operative indications of extra-intracranial bypass surgery should not be handled more generously especially in consideration of the relatively low risk of this operation.

(Discussion)

ACADEMY AWARD

3:00 p.m. **NEURAL SYSTEMS WHICH ACTIVATE THE SUBSTANTIA GELATINOSA: NEW DEVELOPMENTS IN THE PHYSIOLOGY OF PAIN SUPPRESSION**

David Dubuisson, M.D., C.M., M.Sc.
Montreal

3:30 p.m. Coffee Break

3:50 p.m. 14. **CLINICAL CLASSIFICATION OF CEREBRAL ANGIOMATOUS MALFORMATIONS**

H.-P. Jensen, H. Klinge, U. Muhtaroglu
Kiel

Histologically, ZÜLCH (1956) classified the angiomatous malformations of the brain as arteriovenous angiomas, cavernous, capillary (telangiectasias), venous, or capillary and venous angiomas. Our clinical classification chiefly reflects symptoms and signs as well as the appearance of the malformations in angiography and computed tomography, with regard to the possibilities of surgery for the various types according to localization and size.

In the first group we summarized as macroangiomas all those vascular malformations which can easily be seen angiographically or in a CT scan. They are characterized by enlarged feeding arteries, huge draining veins and rapid blood flow. Clinically they may act as the source of intracranial hemorrhage, or of cerebral ischemia like a steal-syndrome or as a mass lesion.

In one variety of central macroangiomas, particularly in children, we find a direct shunt from the arterial system into the vein of Galen.

Diffuse macroangiomas may appear more or less localized or widespread over the brain as multiple malformations. They do not have enlarged feeding arteries and veins and there is no rapid blood flow. On the contrary, the arteries are sometimes narrowed and the blood flow is diminished so that a collateral vascular network is built up. Then there may be an appearance aniographically which Japanese neurosurgeons call "Moya-Moya-disease".

Since the introduction of computed tomography, the so called "angiographically occult angiomas" have become of special interest. They are detected by CT examination as mass lesions which are very often mistakenly diagnosed as gliomas.

The initial clinical signs of microangiomas "cryptic angiomas," are caused by spontaneous intracranial hemorrhage frequently seen in children and young adults. Otherwise clinically they remain completely dormant. Angiographically, they may be detected by careful analysis of the individual vessels, appearing as

minute vascular malformations sometimes up to the size of a peanut or an olive, either they are seen during surgery in the wall of the hematoma, or they are found in the surgically removed blood clot through careful histological examination.

The last group of our classification is the encephalo-facial angiomatosis of Sturge-Weber-disease.

In an analysis of 67 cases with cerebral angiomatous malformations we saw 16 typical arteriovenous macroangiomas, 4 angiomas with vein of Galen malformation, 6 diffuse angiomas, 3 angiographically occult angiomas, 36 microangiomas and 2 encephalo-facial angiomas.

Relevant cases are presented.

(Discussion)

4:10 p.m.

15. COMPLICATIONS AND IMPROVEMENT IN ISOBUTYL-CYANOACRYLATE (IBCA) TREATMENT OF ARTERIO-VENOUS MALFORMATIONS

Basil Harris
Seattle

Surgical excision in accessible arterio-venous malformations (AVM) remains a choice method, however some are inaccessible or unresectable. In this report direct injection of IBCA during craniotomy or arterial exposure into the distal branches of arteries leading to the arterio-venous shunts in malformations of the brain, face and scalp have been done in 21 operations on 14 patients as an adjunct to surgery and as a partial or complete obliterative substitute for excision of AVM. Data on complications and innovations for further advancement of the technique will be presented.

AVM's are constructed of multiple arterio-venous shunts wherein each small artery drains into individual veins. Yet, unresolved problems using IBCA in intracranial and extracranial AVM's will be illustrated. These include large direct shunts, critical IBCA polymerization times, extracranial/intracranial connections and retrograde thrombotic emboli. New techniques will be discussed including time-phase intraoperative arteriography and selective direct exposure retrograde occlusions with IBCA of large AV shunts.

(Discussion)

4:30 p.m.

16. **FURTHER EXPERIENCE IN THE TREATMENT OF ARTERIOVENOUS MALFORMATIONS OF THE BRAIN**

B.M. Stein
Boston

We now have experience with over 100 cases of arteriovenous malformations of the brain, most of which have been treated by combined techniques of embolization and surgical resection. The anatomical factors which designate cases for embolization prior to surgery can now be enumerated in some detail. Embolization techniques most commonly used are those of silastic ball emboli delivered by a femoral catheter. However, other techniques have been utilized, including small balloon catheters and direct injection of acrylic substances at the time of operative intervention. These latter techniques have certain pitfalls which can be detailed in this discussion.

The surgical removal of the these lesions has generally been carried out in a single-stage operation. There are certain aspects of the technique of removal, especially after embolization, that bear emphasis. The mortality and morbidity of these operative procedures, even though the lesions are generally large and located in vital areas, has been acceptably low.

On the basis of this material, we strongly recommend a team and combined approach to large arteriovenous malformations of the brain, and feel that the results of treatment, specifically the mortality and morbidity are at acceptable levels so that this approach is recommended in the majority of cases.

(Discussion)

FRIDAY, OCTOBER 3

MODERATORS: W.–Joachim Bock
Julian Hoff

8:30 a.m.

17. **SYRINGOMYELIA TREATED SURGICALLY**

Gilles Bertrand
Montreal

This report presents a review of 48 cases of "hydro-syringomyelia" operated upon personally by the author between 1964 and 1980. A Chiari malformation was found in 64% of cases. Adhesive arachnoiditis was the chief pathological process in the others. Four cases had arachnoiditis along the dorsal cord; in two of these, there was antecedent trauma to the spine. The various

procedures used to correct the condition are described together with their complications. There was no immediate mortality but three deaths occurred from late complications or failure to arrest the disease seven months to two years after operation. Other complications such as aseptic meningitis were frequent and means to prevent them are discussed. Overall results were: 25% objectively improved, 50% stabilized and 25% continued deterioration.

(Discussion)

8:50 a.m.

18. CERVICAL DISC REPLACEMENT BY POLYMETHYLMETHACRYLATE (PMMA) - A CLINICAL AND RADIOLOGICAL LONG-TERM FOLLOW-UP STUDY IN 331 PATIENTS WITH TRAUMATIC AND DEGENERATIVE DISC DISEASES

K. Roosen and W. Grote
Essen

In order to evaluate the use of bone cement instead of autogenous or homologous bone grafts for anterior cervical interbody fusion the authors present the late results of 331 patients treated from 1969 until 1977.

100 patients had suffered injuries of the cervical spine, 231 patients had degenerative disc diseases, such as cervical myelopathy (75) or radicular and discogenic syndromes (167). In all patients, pathological discs were removed by the ventral approach (CLOWARD, 1956; DEREYMAEKER, 1956; ROBINSON and SMITH, 1955) and according to the method of GROTE (1967) replaced by an allograft (PMMA).

Similar to the clinical graduation of ODOM (1958), the authors present a clinical evaluation scheme, which regards the neurological status as well as the patients' subjective symptoms. Six different grades allow an exact classification of all clinical courses.

The long-term results are anlayzed in correlation to the following parameters: age and sex of patients; preoperative duration of the disease; type and intensity of the clinical status, depending on the direction of the disc prolapse or protrusion; degree of osteochondrosis; duration of the postoperative period; social status of the patients; the influence of postoperative physical therapy. This analysis enables one to compare our own results with those published in the literature.

Radiological examinations in anteroposterior, lateral and oblique views before and after operative therapy were performed in 70 patients. They were completed by x-ray studies in functional positions of the spine and by lateral and AP tomograms. The clinical value of radiological criteria correspond with other published observations (ARONSON, 1970; CLOWARD, 1963; CONNALLY, 1965; LAIN, 1974; MURPHY, 1972; RILEY, 1969; RISH, 1976;

ROBINSON, 1962; TAHERI, 1972; WHITE, 1973; WILLIAMS, 1968; WILSON, 1977).

Bone grafts result in a better morphological integration within the spine; however, the PMMA-allografts are mechanically stronger and become surrounded by a bone cuff, usually within two years after implantation.

The comparison between our own and the published clinical and radiological results as well as the rates of complications demonstrate that the use of bone cement for cervical spondylodeses is up to now an equivalent operative procedure.

(Discussion)

9:10 a.m.

19. COLLAGENASE IN THE TREATMENT OF LUMBAR DISC PROTRUSION

Jaime G. Gomez
Bogota

Preliminary results of treatment of lumbar disc protrusion by intervertebral injection of Collagenase are presented. Patients with lumbar and sciatic pain lasting over two months, not relived by two weeks of strict bed rest in hospital, analgesics and muscle relaxants, who had neurological signs of deficit, one level myelographic defects and no previous surgical treatment were selected for Collagenase discolysis.

The procedure was performed under neuroleptoanalgesia and under televised fluoroscopic control. Follow up has been between six and twelve months with excellent results in twenty of twenty one cases. One patient with an extruded disc had to be operated 48 hours after discolysis.

Our initial impression is highly satisfactory and a larger number of patients are new being treated.

(Discussion)

9:30 a.m.

20. SURGICAL TREATMENT OF INSTABILITY OF THE SPINE AFTER EXTENDED LAMINECTOMY

K. Schürmann
Mainz

It is a well-known fact that after an extended laminectomy, as indicated in certain cases of intramedullary gliomas or arteriovenous malformations, an instability of the spine is not present in the early phase after the operation but

will follow in most cases after some years. This instability of the spine can be followed by a more or less severe progressive deformity of the spine as kyphoscoliosis or lordotic deformity which may produce new neurological symptoms. Particularly severe deformities are to be seen in the cervical spine. Therefore, a fusion operation is recommended in the same procedure in which the extended laminectomy was done. Fusion is to be performed lateral to the area of laminectomy by denuding the articular processes and by destroying the cartilaginous space with diamond-drill. The destroyed articular space is then filled with spongious bone material and as the essential act a strong bone graft, obtained from the iliac crest, is laterally inserted and fixed by steel wires with the articular processes. Additional fixation with the cranial and with the distal vertebral arch is useful. The mobility of the cervidal spine is in fact hardly limited after this fusion technique but a postoperative progressive deformity of the endangered cervical spine is definitely prevented. The described fusion technique seems to be the method of choice for the cervical spine, whereas in the thoracic and lumbar level the well-known postero-lateral spondylosyndesis after the HARRINGTON technique is sufficient. Relevant cases are presented.

(Discussion)

9:50 a.m.

21. LATE RESULTS OF OPERATIVE CASES OF LUMBAR SPONDYLOLISTHESIS WITH RUPTURED DISC

William Beecher Scoville
Hartford

In the Journal of Neurosurgery, Volume 40, April 1974, I published 20 cases of spondylolisthesis with concomitant signs of a ruptured intervertebral disc treated by radical removal of the disc without fusion and with early mobilization and strenuous exercise regime. It is well known that there is no further slippage of a true spondylolisthesis after teenage and if the case shows unilateral leg pain it is probably due to an accompanying ruptured disc. Hence the above regime has been continued to date, now with 38 cases.

Late results have shown complete collapse of the spondylolisthesis interspace, still without further slippage, and with results exactly comparable to simple operative discs.

(Discussion)

10:10 a.m. **Coffee Break**

10:30 a.m.

22. **EXPERIMENTAL SPINAL CORD LESIONS: PROPERTIES AND POSSIBLE VALUE OF A LESION – SPECIFIC CORD POTENTIAL**

J. Schramm, R. Krause
Berlin

In 38 cats various modes of experimental spinal cord injury were employed: acute concussive injury, fast graded compression, subacute graded compression, weight bearing injury, pharmacological injury and chronic cord compression.

The alterations of spinal and cortical evoked responses were studied. In chronic compression (over several months) a correlation to the clinical findings was made.

The properties of a lesion-specific potential, to be recorded at the injury site, are described. It is a monophasic positive potential, well discernible from the usual triphasic cord potential, and may be picked up rostral from the injury site. Common reference recordings proved to be more sensitive than bipolar techniques. In slowly developing lesions a gradual development of the lesion-specific alteration is found. These alterations may be reversible and are a very sensitive indicator of localized impaired spinal cord conduction. In serial recordings it was possible to localize the injury or compression site in the order of millimeters.

Several aspects concerning the possible value of this neurophysiological alteration in spinal cord pathology may be mentioned.

First, the local , lesion-specific alterations occur earlier than cortically recorded changes.

Second, a precise topographic localization of the lesion is possible.

Third, the application of intraoperative monitoring of spinal cord function by means of cortical evoked responses could gain in value by using serial spinal cord recordings.

(Discussion)

10:50 a.m.

23. **SPINAL CORD BLOOD FLOW IN EXPERIMENTAL TRAUMA AND IN SPINAL TUMORS**

R. Wüllenweber and H. Collmann
Bonn

In order to simulate an acute lesion of the spinal cord comparable to that produced by an accident in man – we used the weight – dropping model according to Allen.

Materials and methods:
20 beagle dogs were anesthetized with piritramide, curarized, intubated and artificially ventilated with a $N_20/0_2$-mixture. Cardiovascular and respiratory functions were monitored by recording the arterial and venous blood pressure, the ECG, the endexpiratory $C0_2$ concentration and by repeated blood gas analyses. Following cervical laminectomy the spinal cord was exposed from C_3 to C_6 . Using the principle of heat-clearance, two needle-shaped thermoprobes were placed into the spinal cord 3-4 cm apart. Relative changes of local spinal blood flow were continuously registered. Between the 2 thermoprobes a standardized dropped-weight trauma (450 g/cm) was applied to the dorsal surface of the cord. Following the impact, the regional spinal blood flow was monitored for a period up to 5 hrs. The vascular reactivity to hypercapnia and norepinephrine–induced hypertension was assessed.
Results:
Morphological findings are reported with regard to hemorrhagic necrosis, perifocal edema and deformations of neuronal cells.
Pathophysiological findings are described with regard to the following topics: hyperemia, CO_2-response and autoregulation after the impact. These results are compared with the findings of our earlier studies in man during surgical procedures where we have used disc-shaped thermoprobes. They confirm that there is no significant difference between the regulation of spinal and cerebral blood flow.

(Discussion)

11:00 a.m.

24. **DECOMPRESSION AND STABILIZATION OF LUMBAR AND THORACIC FRACTURES**

Wesley A. Cook, Jr.
Durham

A variety of surgical procedures have been utilized to manage fractures of the lumbar and thoracic spine. These include laminectomy, with or without fusion, vertebral body resection followed by anterior interbody fusion, reduction and fixation with Harrington rod instrumentation and various combinations of these procedures. In an attempt to eliminate some of the disadvantages of these procedures, we have utilized a posterolateral approach to decompress the contents of the spinal canal followed by Harrington rod instrumentation for reduction and fixation.

This report summarizes our experience with 19 patients with fracture of the lumbar or thoracic spine who were operated on during 1977 or 1978. Indications for surgery included compromise of the neural elements within the spinal canal and actual or potential instability of the spine. Preoperative evaluation included AP and lateral tomograms, myelography and in selected cases, transaxial tomography. During surgery, spinal cord function was monitored by evoked spinal cord potentials or by awakening the patient during

the reduction. Postoperatively the patients were placed in thoracolumbar corsets and mobilized within 7-10 days.

Preoperatively, two patients were neurologically intact and three patients demonstrated complete and immediate paraplegia. The neurologic status of these patients was not altered by the surgical procedure. The remaining fourteen patients had sustained partial but varying degrees of spinal cord injury. Of these patients three reverted to a normal neurologic status, seven were improved, four were unaltered and one was worse following surgery.

The discussion of these cases will emphasize patient selection, technique of the procedure and the advantages and disadvantages of this form of management with the following goals in mind: 1) preservation of remaining neural function 2) decompression of potentially functional neural elements 3) restoration of normal spine alignment and stability and 4) early ambulation and rehabilitation.

(Discussion)

11:30 a.m.

PRESIDENTIAL ADDRESS: THE INFLUENCE OF GERMAN NEUROSURGERY ON HARVEY CUSHING

Eben Alexander, Jr.

SATURDAY, OCTOBER 4

MODERATORS: Frank Marguth
John T. Garner

9:15 a.m.

25. BRAINSTEM AUDITORY EVOKED RESPONSE RECORDING DURING NEUROLOGICAL SURGERY

Andrew G. Shetter and Peter A. Raudzens
Phoenix

Hearing loss following operative manipulation is a significant risk of surgical procedures involving dissection in the region of the 8th cranial nerve complex. We monitored intraoperative brainstem auditory evoked response potentials (BAER) in 17 patients undergoing posterior fossa or middle fossa operations near the acoustic nerve in an attempt to lessen or prevent this complication. The initial five waveforms comprising the BAER were satisfactorily recorded in all instances. Eleven patients had no alteration in the BAER and no change in postoperative auditory function. Three patients demonstrated intraoperative changes in waveform latency and amplitude that persisted and were associated with subsequent hearing deficits. An additional three patients experienced temporary deterioration in their evoked response patterns that returned to normal with adjustments in retraction positioning. These individuals had no postoperative hearing loss and represent the group who

may have benefited from intraoperative evoked response monitoring. On the basis of our initial experience we believe the BAER can be reliably recorded in an operating room environment and may be valuable in alerting the surgeon to changes in acoustic nerve function that are potentially reversible.

(Discussion)

9:35 a.m.

26. EXPERIMENTAL COLD LESION AND LYSOSOMAL MEMBRANE STABILITY OF THE CAT BRAIN UNDER BARBITURATE ANESTHESIA

D. Stokle, H. Dietz
Hannover

Barbiturates are know to bring about a beneficial effect on brain ischemia and on intracranial pressure caused by severe head injury. This experimentally well established effect has been assumed to be primarily a consequence of the reduction in cerebral metabolic rate.

In experimental studies the influence of thiopental and pentobarbital on the stability of lysosomal membranes was investigated. The gray and white matter of the brain tissue were assayed after barbiturate pretreatment as well as after cold lesion under barbiturate anesthesia compared to another anesthesia procedure.

Lysosomal enzymes are subcellular organelles involved in the degradation of cellular components, contain acid hydrolytic enzymes and are surrounded by a lipoprotein membrane. To indicate lysosomal membrane stability the amount of free activity of the lysosomal enzyme β-glucuronidase was assayed.

Our results indicate that after barbiturate pretreatment there is an increase in lysosomal membrane stability in the white (after pentobarbital-pretreatment) as well as in the gray matter (after thiopental-pretreatment) compared to the control group. Under barbiturate anesthesia further results demonstrate an increased lysosomal membrane stability after cold lesion in both hemispheres in gray and white matter compared to the group of animals subjected to the same procedure under ketamine anesthesia.

This increase in lysosomal membrane stability is regarded as an important protective effect of the barbiturates not only against brain ischemia but also against brain edema and increased intracranial pressure induced by experimental brain lesion.

(Discussion)

9:55 a.m.

27. RENAL DIURETICS IN THE TREATMENT OF ACUTE EXPERIMENTAL CRANIAL HYPERTENSION

Harold A. Wilkinson
Worcester

Three different diuretics have been studied in three separate experimental projects involving three forms of experimentally induced intracranial hypertension. A total of 57 dogs and 5 baboons have been used in this study to date. Forty-five dogs were used to study the diuretic synergy between ethacrynic acid and mannitol in the treatment of acute experimental cerebral edema created by the intracarotid injection of sodium lauryl sulphate. A second group of nine dogs was used to study the effects of "Megadose" Diamox, with and without Mannitol in the treatment of mixed intracranial mass lesions (balloons) with inflation/reinflation induced brain edema. The third and still ongoing series has incorporated to date five baboons and three dogs to study the effect of furosemide on intracranial hypertension induced by subdural balloon inflation.

Results: All three renal diuretics induced a large volume urinary diuresis but little or no effect on intracranial hypertension. Ethacrynic acid had no effect when used alone but synergized nicely with mannitol to effect a more rapid and sustained decrease in ICP. Megadose Diamox consistently caused an increase in ICP, which was only partly counteracted by concomitant mannitol administration. The Lasix studies to date are incomplete but have failed to show a striking reduction in ICP acutely.

(Discussion)

10:15 a.m.

28. IMPAIRMENT OF MOTOR RECOVERY AFTER LATE SECONDARY NERVE SUTURE

H.–P. Richter, U.P. Ketelsen, D. Frösch
Günzburg

It still is a matter of controversy, if the bad motor recovery after a late secondary nerve suture is related to the nerve or the muscle.

To study this question, we sectioned the peroneal nerve in rabbits and performed a secondary nerve suture after denervation periods up to 12 months. Six months later, clinical testing and electromyography were done and nerve and muscle tissue removed for histological, histochemical and ultrastructural examination. The results were obtained from 47 animals and 75 nerve-muscle preparations.

With longer lasting presuture denervation the animals became less able to

spread their toes, the amplitude of the elctrically evoked muscle potential decreased, the potentials became more polyphasic and the morphological alterations of the muscle increased. Concerning the neurotization of the peroneal nerve distal to the suture site, individual variations were more obvious that a decrease with longer lasting denervation. The identification of motor endplates for ultrastructural investigations was difficult after a late secondary nerve suture, but in all neuromuscular junctions studies in this group of animals we found important morphological changes.

Although we were unable to define a more precise interval, a denervation of 8-10 months is a critical period in rabbit peroneal nerve. After longer lasting denervation the nerve suture is followed by an insufficient motor recovery.

From our investigation we conclude that the quality of motor restitution after a secondary nerve suture depends largely on the motor end-organs and not mainly on the nerve, confirming the results of GUTMANN and YOUNG (1944). The original pattern of muscular innervation, however, never can be restored by a nerve suture. Even after primary nerve suture a type grouping and transformation of muscle fibers is observed in histochemical reactions, a phenomenon typical for a reinnervated muscle.

Although the mentioned intervals cannot be transferred to man, the course and quality of muscular restitution probably are comparable in animals and humans.

(Discussion)

10:35 a.m.

29. **THE ROLE OF THE BLOOD/NERVE BARRIER (BNB) IN NORMAL AND INJURED PERIPHERAL NERVE**

A.R. Hudson, F. Gentili, D.G. Kline & D. Hunter
Toronto

The management of a major peripheral nerve injury remains a considerable therapeutic challenge. This is, in part, related to our lack of understanding of the critical parameters that determine functional nerve regeneration following injury. Recently, it has been suggested that nerve fibres in a peripheral nerve function in a unique environment created and maintained by special barrier mechanisms (blood/nerve barrier) analogous to the blood/brain barrier in the central nervous system. This barrier plays an important role in determining optimal nerve fibre function.

The present experimental study was designed to examine the role of the BNB in both normal nerve and in nerves under various pathological conditions. The sciatic nerves of over 500 adult rats have been studied.

The function and alterations in the BNB were assessed with both a fluorescent microscopic tracer technique, using 5% bovine serum albumin (M.W. 69,000), labelled with 1% Evans' blue (EBA), and an electron microscopic tracer

technique, using horseradish peroxidase (HRP) (MW. 40,000) as the tracer proteins. Light and electron microscopic studies, as well as motor nerve conduction velocities, were carried out.

The right sciatic nerves of five groups of animals were subjected to (1) nerve injection injury with a variety of antibiotic, steroid and local anaesthetic agents (400 animals); (2) internal neurolysis (90) animals; (3) nerve transsection, followed by epineural suture (50 animals); (4) nerve transsection with neuroma formation (25 animals).

The left sciatic nerve was used as the control, and nerves were studied at varying periods from one hour to eight months after injury.

Results revealed that the anatomic sites of the BNB appeared to be the tight junctions of the endoneurial capillary endothelium and the perineurial cells of the perineurium.

1. Nerve injection injury resulted in a significant breakdown in the BNB with marked endoneurial edema, which was felt to contribute to the nerve damage seen with these injuries.
2. Following internal neurolysis, there was a transient disturbance of the BNB during the first week only, which appeared to correlate with a reduced nerve conduction velocity in the nerve. Damage to the perineurium resulted in a more persistent alteration in the BNB.
3. Following nerve section and suture, there was an immediate breakdown of the BNB at the site of anastomosis which, over the following week, spread throughout the distal segment undergoing degeneration. Restitution of the BNB occurred in stages in a proximodistal direction comparable to the process of regeneration, but lagged four to six weeks behind axonal regeneration.
4. Our results also suggest that a peripheral nerve at the site of a traumatic end-bulb neuroma lacks a normal BNB. The disturbed endoneurial environment may contribute to the painful symptoms often seen with these lesions. The authors conclude that the concept of the BNB appears to be of major significance in our overall understanding of the variables affecting the results of peripheral nerve surgery.

(Discussion)

10:55 a.m. **Coffee Break**

11:15 a.m.

30. POSTERIOR INTEROSSEOUS NERVE LESIONS

D.G. Kline and A.R. Hudson
New Orleans

Operative experience with 700 patients with serious nerve lesions has included 45 with posterior interosseous paralysis:

Entrapment	LAC	FX	Iatrogenic	Forearm Crush	GSW	Tumor	Volkmann's
8	16	6	5	4	3	2	1

Diagnosis can be missed since wrist drop is not a feature but rather radial drift with dorsiflexion since branches to extensor carpi radialis either arise from radial before posterior interosseous or more frequently traverse the proximal portion of the superficial sensory branch. Thus, by the time the need for exploration was obvious, denervation by EMG was quite severe not only in the group of focal lesions such as laceration, fracture, and gunshot wound but also in the entrapment group. In the latter group of patients, radial sensory NAP was always present but due to associated injury was not always present with a focal lesion. Non-invasive conduction studies can be misleading making exploration and more direct stimulation and recording paramount in cases where either serious sustained entrapment or injury is suspected. Thus in 27 lesions in continuity where operative recording was done, there were five instances where despite complete denervation of distal muscles by EMG an NAP across the lesion was present and in three of these cases there was no distal muscular response to nerve stimulation. In two cases felt to be incomplete or recovering by clinical and EMG study, there was no response to stimulation nor an NAP.

In	EMG	Stim	NAP					
Continuity	C	I	O	Ab	P	Ab	P	Neurolysis/Result	Suture/Result	Graft/Result	Split/Result
27	15	9	3	13	14	10	17	17/15	5/3	2/1	3/2

Exposure beneath brachioradialis, through the two heads of the supinator and to deep dorsal forearm requires thorough mobilization of brachioradialis, section of volar supinator and careful dissection of the rich vasculature found in the pre-supinator space and surrounding the posterior interosseous nerve. Three of the 8 entrapments occurred at the dorsal forearm rather than the supinator level. These, as well as those with the more classic supinator level entrapment recovered with neurolysis, although one requiring graft did not.

Other results included 5 transections repaired by suture with recovery to grade 3 or better, 5 iatrogenic lesions usually secondary to fracture manipulations where recovery was variable due to length of lesion and severe scars.

Pertinent anatomy as outlined by Spinner and others, as well as selected electrophysiologic traces and operative slides will also be shown.

(Discussion)

11:35 a.m.

31. MICROSURGICAL VASCULAR DECOMPRESSION OF THE TRIGEMINAL ROOT FOR THE TREATMENT OF SO-CALLED IDIOPATHIC TRIGEMINAL NEURALGIA

H. Penzholz and A. Kühner

Heidelberg

The hitherto usual operations for trigeminal neuralgia are able to remove the typical pain attacks in most cases. But this success can only be achieved with

a defect of sensibility which is disturbing for many patients. It may still be much more annoying for the patients, if they get painful dysesthesias or even an "anesthesia dolorosa". A further disadvantage is the rather high recurrence rate, which reached over 50% in our material within 10 years.

Therefore it seemed to us justified to seek for further improvements in our therapeutic possibilities. In doing so the microsurgical vascular decompression of the root entry zone of the trigeminal nerve in the posterior fossa as recommended by JANETTA seemed especially interesting.

We are now able to report the results of this operation in 23 patients with a maximum follow up of 2½ years.

It seems to us, that these results are encouraging and that indeed in many cases a really etiologic therapy may be possible. Indications, technique and risks are discussed.

(Discussion)

11:55 a.m.

32. THE ROLE OF THE DORSAL ROOT ENTRY ZONE IN THE CONTROL OF CENTRAL PAIN

Blaine S. Nashold, Jr., Roger Ostdahl and Elizabeth Bullitt
Durham

The dorsal root entry zone (DREZ) represents the first integrative step for afferent impulses entering the spinal cord. Recent anatomic and physiologic data show that there is a definite organization for the small fiber input into the Rexed zones 1 and 5 and that these areas contain high amounts of endorphin and substance P, two neurohumors which have been postulated to modulate noxious input.

In 1976, we reported on the focal coagulation of the dorsal root entry zone (DREZ) for relief of central pain following brachial plexus avulsion. Since that time 30 additional patients with intractable central pain syndromes due to spinal injury and post herpetic pain have been treated by this new technique of multiple focal coagulations of the DREZ.

Twenty-one patients suffering from complete avulsion of the brachial plexus had been unsuccessfully treated with various neurosurgical operations (cordotomy, rhizotomy, dorsal column stimulation, stereotactic mesencephalotomy); however, following dorsal root entry zone lesions of the cervical cord, 67% have been relieved of pain from 1 to 4 years. Ten patients with complete traumatic paraplegia and intractable central pain at or below the level of the spinal cord transection were also treated with dorsal root entry zone lesions by segmental bilateral dorsal root entry zone lesions in the spinal cord segments just above the traumatic transection followed by a 50% relief rate for 1 to 2 years. In addition, six patients with post herpetic arm or chest pain have

also been treated by local segmental dorsal root entry zone lesions corresponding to dorsal roots from the herpetic cutaneous lesions with a 50% reduction of pain with a followup of 6 months to 1 year.

(Discussion)

12:15 p.m.

33. RADIOFREQUENCY RHIZOTOMY: NEUROPATHOLOGICAL CORRELATION IN A CLINICAL MODEL

Harold P. Smith, Joe M. McWhorter, Venkata R. Challa
Winston-Salem

In 1968, Letcher and Goldring demonstrated the effects of radiofrequency and heat on the action potentials of peripheral nerves in cats. Their demonstration that radiofrequency and heat lesions blocked the action potentials of smaller delta and C fibers before those of the alpha beta group formed the neurophysiological basis for use of radiofrequency lesions in the treatment of trigeminal neuralgia, chronic pain, and spasticity. However, in 1977, Uematsu reported, in a preliminary histological study of radiofrequency thermocoagulation lesions in the sciatic nerve of the cat, that he found indiscriminate destruction of both the large myelinated and the small fibers, rather than selective destruction of just the smaller myelinated delta and unmyelinated C fibers. To date, no neuropathological confirmation of Uematsu's work has been reported in cats or in a clinical rhizotomy model, nor have the effects of graded temperature lesions been studied.

In the first portion of this study, radiofrequency lesions of 85°C. for 2 minutes each were made by placing a temperature monitoring electrode under direct vision into the lumbar intervertebral foramina of beagle dogs. That a satisfactory rhizotomy leison had been made was confirmed by comparing thresholds of motor response to pre-and post-lesion stimulation. The dogs were killed at 1-, 2-, 3-, 5-, and 6-week intervals, the spinal cords were perfused, and sections of the lesion, dorsal root, ventral root, dorsal root ganglion, and spinal cord were removed for light and electron microscopic study. Sixty tissue blocks from 15 lesions in 5 dogs as well as blocks of normal nerve tissue were obtained. In all lesions, there was total loss of non-myelinated fibers and near-total loss of myelinated fibers; the remaining few fibers were mostly large myelinated fibers. The lesions at 1, 2, 3, 5, and 6 weeks post-rhizotomy were not qualitatively different, but they differed quantitatively in the amount of myelin debris and the number of macrophages and Schwann cells present. Ventral roots were involved in 2 of the 15 lesions.

In the second portion of the study, graded temperature lesions of 45°C. 55°C., 65°C., and 75°C. were made and 25 blocks of tissue from 4 lesions were obtained one week later, processed, and studied as in Part I. They were closely similar to the lesions made at 85°C.

This study demonstrates that at temperatures of 45° to 85°C. (those used clinically) there is no selective destruction of unmyelinated C fibers and small myelinated fibers as hypothesized by Letcher and Goldring, but rather there is indiscriminate destruction of both small and large myelinated fibers, as Uematsu had found.

The techniques and results of both parts of this study are presented, and the implications of our findings on the future clinical use of radiofrequency lesions in management of pain and spasticity are discussed.

(Discussion)

POSTER SESSION

POSTTRAUMATIC STEROID TREATMENT AND ITS EFFECT ON MEMBRANE STABILITY.

A. Weidner, D. Stolke, and H. Dietz.

EXPERIMENTAL AND CLINICAL INVESTIGATIONS OF PENETRATION OF ANTIBIOTICS INTO THE CEREBRO-SPINAL FLUID.

H. Friedrich, G. Hansel-Friedrich, and J. Potel.

ON THE DIAGNOSIS AND TREATMENT OF TUMORS OF THE JUGULAR FORAMEN.

J. Menzel, H.J. Denecke, and Th. Rommel.

NORMAL AND PATHOLOGIC ANATOMY OF THE BASILAR ARTERY.

Marc Mayberg, Theodore Liszczak, and Nicholas T. Zervas.

MONITORING DURING CAROTID ENDARTERECTOMY: EVIDENCE THAT AN INTERNAL SHUNT IS NOT NECESSARY.

Gary G. Ferguson, J.K. Farrar, and W.T. Blume.

POSTER PRESENTATIONS WILL BE ON DISPLAY OUTSIDE THE SCIENTIFIC MEETING AREA (THE EMPIRE ROOM) WHICH IS LOCATED OFF MAIN LOBBY FROM OCTOBER 2-4, 1980.

1980
ACADEMY AWARD

DAVID DUBUISSON, M.D., C.M., M.Sc.
Montreal Neurological Institute and Hospital
Montreal, Quebec, Canada

"Neural Systems Which Activate the Substantia Gelatinosa:
New Developments in the Physiology of Pain Suppression"

HONORABLE MENTION

ACADEMY AWARD

1980

I

Kim J. Burchiel, M.D.
University of Washington
Seattle, Washington

"Abnormal Impulse Generation in Focally Demyelinated Trigeminal Roots"

II

Stephen J. Haines, M.D.
University of Pittsburgh
Pittsburgh, Pennsylvania

"Prophylactic Methicillin for Shunt Operations:
Effects on Incidence of Shunt Malfunction and Infection"

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Hotel Netherlands Plaza, Cincinnati, Ohio ..... October 28-29, 1938
Roosevelt Hotel, New Orleans, Louisiana ..... October 27-29, 1939
Tutor 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
The Key Biscayne, Miami, Florida ..... November 11-14, 1964
Terrace Hilton Hotel, Cincinnati, Ohio ..... October 14-16, 1965
Fairmont Hotel & Tower, San Francisco, California . October 17-19, 1966
The Key Biscayne, Miami, Florida ..... November 8-11, 1967
Broadmore 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
The Mills Hyatt House, Charleston,
South Carolina ..... November 10-13, 1976
Mauna Kea Beach Hotel, Kameula, Hawaii ..... November 2-5, 1977
Hotel Bayerischer Hof, Munich Germany ..... October 22-25, 1978
Hyatt Regency, Memphis, Tennessee ..... November 7-10, 1979

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FRANCIS A. ECHLIN 100 East 77th Street New York, New York, 10021	(LETITIA)	1944
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234 East Gray Street
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JOHN R. GREEN Barrow Neurological Inst 302 West Thomas Street Phoenix, Arizona 85013	(GEORGIA)	1953
JOHN W. HANBERY Division of Neurosurgery Stanford Medical Center Palo Alto, California 94304	(SHIRLEY)	1959
MAJ. GEN. GEORGE S. HAYES MC USA 303 Skyhill Road Alexandria, Virginia 22314	(CATHERINE)	1962
E. BRUCE HENDRICK Hospital for Sick Children 555 University Avenue, Rm. 1502 Toronto, Ontario, Canada 1X8	(GLORIA)	1968
JULIAN HOFF Department of Neurosurgery University of California San Francisco, California 94143	(DIANNE)	1975
EDGAR M. HOUSEPIAN 710 West 168th Street New York, New York 10032		1976
ALAN R. HUDSON St. Michaels Hospital 38 Shutter Street Toronto, Ontario, Canada M5B LA6	(SUSAN)	1978

WILLIAM E. HUNT (CHARLOTTE) 1970
Division of Neurological Surgery
University Hospital
410 West 10th Avenue
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370 Winn Way, #201
Decatur, Georgia 30030

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

WILLIAM A. KELLY 1977
University of Washington
School of Medicine
Seattle, Washington 98195

GLENN W. KINDT (CHARLOTTE) 1977
University of Michigan
Medical Center
Ann Arbor, Michigan 48104

ROBERT B. KING (MOLLY) 1958
University Hospital
Upstate Medical Center
750 East Adams Street
Syracuse, N.Y. 13210

WOLFF M. KIRSCH
University of Colorado Medical Center (MARIE-CLAIRE) 1971
4200 East 9th Avenue
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DAVID G. KLINE (CAROL) 1972
Louisiana State University Medical Center
1542 Tulane Avenue
New Orleans, Louisiana 70012

ROBERT S. KNIGHTON (LOUISE) 1966
9388 Avenida San Timeteo
Cherry Valley, California 92223

RICHARD S. KRAMER Duke Hospital Durham, North Carolina 27710	(ROBIN)	1978
THEODORE KURZE 10 Congress Street Suite 340 Pasadena, California 91105		1967
THOMAS W. LANGFITT Hospital of the Univ. of Pennsylvania 34th and Spruce Streets Philadelphia, Pennsylvania 19104	(CAROLYN)	1971
RAEBURN C. LLEWELLYN 9661 Lake Forest Blvd Suite 350 New Orleans, Louisiana 70127	(CARMEN)	1963
WILLIAM M. LOUGHEED Medical Arts Bldg. Suite 430 170 St. George Street Toronto 5, Ontario, Canada	(GRACE ELEANOR)	1962
HERBERT LOURIE 713 East Genesee Street Syracuse, New York 13210	(BETTY)	1965
ALFRED J. LUESSENHOP Georgetown University Hospital Washington, D.C. 20007	(BETSY)	1976
ERNEST W. MACK 505 S. Arlington Avenue Suite 212 Reno, Nevada 89502	(ROBERTA)	1956
M. STEPHEN MAHALEY, JR. Univ. of N.C., Room 229H 148 Clinical Sciences Bldg. Chapel Hill, North Carolina 27514	(JANE)	1972
LEONARD MALIS 1176 Fifth Avenue New York, New York 10029	(RUTH)	1973

FRANK MAYFIELD 506 Oak Street Cincinnati, Ohio 45219	(QUEENEE)	Founder
ROBERT L. McLAURIN Division of Neurosurgery Cincinnati General Hospital Cincinnati, Ohio 45229		1955
WILLIAM F. MEACHAM Vanderbilt University Hospital Division of Neurosurgery Nashville, Tennessee 37203	(ALICE)	1952
JOHN F. MULLAN, M.D. Univ. of Chicago Clinics Department of Neurosurgery 950 East 59th Street Chicago, Illinois 60637	(VIVIAN)	1963
BLAINE S. NASHOLD, JR. Duke University Med. Center Durham, North Carolina 27706	(IRENE)	1967
FRANK E. NULSEN Division of Neurosurgery University Hospital 2065 Adelbert Road Cleveland, Ohio 44106	(GINNEY)	1956
GUY L. ODOM Duke University Medical Center Durham, North Carolina 27706	(MATALAINE)	1946
GEORGE OJEMANN University of Washington Dept. of Neurosurgery Seattle, Washington 98195	(LINDA)	1975
ROBERT G. OJEMANN Massachusetts General Hospital Division of Neurological Surgery Boston, Massachusetts 02114	(JEAN)	1968

BURTON ONOFRIO Mayo Clinic Rochester, Minnesota 55901	(JUDITH)	1975
RUSSEL H. PATTERSON, JR. 525 East 68th Street New York, New York 10021	(JULIET)	1971
S.J. PEERLESS P.O. Box 5339 Terminal A University Hospital London, Ontario Canada N6A 5A5	(ANN)	1977
PHANOR L. PEROT, JR. Department of Neurosurgery Medical University of South Carolina 171 Ashley Avenue Charleston, South Carolina 29403	(ELIZABETH)	1970
BYRON C. PEVEHOUSE 2001 Union Street San Francisco, California 94101		1964
ROBERT W. PORTER 5901 East 7th Street Long Beach, California 90804	(AUBREY DEAN)	1962
JOHN RAAF 1120 N.W. 20 #100 Portland, Oregon 97209	(LORENE)	Founder
AIDEN A. RANEY 125 North Las Palmas Los Angeles, California 90004	(MARY)	1946
JOSEPH RANSOHOFF II New York Univ. Med. Center 550 First Avenue New York, New York 10016	(RITA)	1965
THEODORE B. RASMUSSEN Montreal Neurological Institute 3801 University Street Montreal 2, Quebec, Canada	(CATHERINE)	1947

HUGO RIZZOLI 2150 Penn Avenue, NW Washington, D.C. 20037	(HELEN)	1973
THEODORE S. ROBERTS Division of Neurosurgery University of Utah Medical Center Salt Lake City, Utah 84132	(JOAN)	1976
JAMES T. ROBERTSON Department of Neurosurgery UTCHS, 956 Court Avenue Memphis, Tennessee 38163	(VALERIA)	1971
RICHARD C. SCHNEIDER C5135 Out-Patient Building University Hospital Ann Arbor, Michigan 48104	(MADELEINE)	1970
JAMES C. SIMMONS 920 Madison Avenue Memphis, Tennessee 38103	(VANITA)	1975
BENNETT M. STEIN New England Medical Center Hosp. 171 Harrison Avenue Boston, Massachusetts 02111	(DOREEN)	1970
JIM L. STORY, M.D. 7703 Floyd Curl Drive San Antonio, Texas 78229	(JOANNE)	1972
THORALF M. SUNDT, JR. 200 1st Street, S.W. Rochester, Minnesota 55901	(LOIS)	1971
ANTHONY F. SUSEN 3600 Forbes Avenue Pittsburg, Pa. 15213	(PHYLLIS)	1965
RONALD R. TASKER Toronto General Hospital Room 121, U.W. Toronto, Ontario, Canada	(MARY)	1971

JOHN TEW, JR. 506 Oak Street Cincinnati, Ohio 45219	(SUSAN)	1973
GEORGE T. TINDALL Emory Univ. School of Medicine Division of Neurosurgery 1365 Clifton Road, N.E. Atlanta, Georgia 30322	(SUZIE)	1968
ARTHUR A. WARD, JR. Department of Neurological Surgery Univ. of Washington Hospital Seattle, Washington 98105	(JANET)	1953
CLARK WATTS 807 Stadium Road Suite N521 Columbia, Missouri 65212	(PATTY)	1975
W. KEASLEY WELCH Childrens Hospital Medical Center 300 Longwood Avenue Boston, Massachusetts 02115	(ELIZABETH)	1957
BENJAMIN B. WHITCOMB 85 Jefferson Street Hartford, Connecticut 06106	(MARGARET)	1947
LOWELL E. WHITE, JR. University of Southern Alabama Division of Neuroscience Mobile, Alabama 36688	(MARGIE)	1971
ROBERT WILKINS Duke University Medical Center Box 3807 Durham, North Carolina 27710		1973
CHARLES B. WILSON Department of Neurological Surgery U. of California Medical Center Third and Parnasus San Francisco, California 94122	(ROBERTA)	1966

FRANK WRENN 123 Mallard Street Greenville, South Carolina 29601	(BETTY)	1973
DAVID YASHON 410 W. 10th Ave. N. 911 Columbus, Ohio 43210	(MYRNA)	1972
NICHOLAS T. ZERVAS Massachusetts General Hospital Department of Neurosurgery Boston, Massachusetts 02214	(THALIA)	1972

Deceased Members		Date	Elected
DR. SIXTO OBRADOR ALCALDE Madrid, Spain	(Honorary)	4/27/67	1973
DR. JAMES R. ATKINSON Phoenix, Arizona	(Active)	2/78	1970
DR. PERCIVAL BAILEY Evanston, Illinois	(Honorary)	8/10/73	1960
DR. WILLIAM F. BESWICK Buffalo, New York	(Active)	5/12/71	1949
DR. SPENCER BRADEN Cleveland, Ohio	(Active)	7/20/69	Founder
DR. F. KEITH BRADFORD Houston, Texas	(Active)	4/15/71	1938
DR. WINCHELL McK. CRAIG Rochester, Minnesota	(Honorary)	2/12/60	1942
DR. WESLEY A. GUSTAFSON Jensen Beach, Florida	(Senior)	7/16/75	1942
DR. HENRY L. HEYL Hanover, New Hampshire	(Senior)	3/01/75	1951
DR. OLAN R. HYNDMAN Iowa City, Iowa	(Senior)	6/23/66	1942
MR. KENNETH G. JAMIESON Brisbane, Australia	(Corresponding)	1/28/76	1970
SIR GEOFFREY JEFFERSON Manchester, England	(Honorary)	3/22/61	1951
DR. WALPOLE S. LEWIN Cambridge, England	(Corresponding)	1/23/80	1973
DR. DONALD D. MATSON Boston, Massachusetts	(Active)	5/10/69	1950

DR. KENNETH G. McKENZIE Toronto, Ontario, Canada	(Honorary) 2/11/64	1960
DR. JAMES M. MEREDITH Richmond, Virginia	(Active) 12/19/62	1946
DR. W. JASON MIXTER Woods Hole, Massachusetts	(Honorary) 3/16/58	1951
DR. WILDER PENFIELD Montreal, Canada	(Honorary) 4/05/76	1960
DR. RUPERT B. RANEY Los Angles, California	(Active) 11/28/59	1939
DR. DAVID L. REEVES Santa Barbara, California	(Senior) 8/14/70	1939
DR. DAVID REYNOLDS Tampa, Florida	(Active) 4/03/78	1964
DR. SAMUEL R. SNODGRASS Nashville, Indiana	(Senior) 8/08/75	1939
DR. C. WILLIAM STEWART Montreal, Quebec, Canada	(Corresponding)	1948
DR. GLEN SPURLING La Jolla, California	(Honorary) 2/07/68	1942
DR. HENDRIK SVIEN Rochester, Minnesota	(Active) 6/29/72	1957

AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
1980 ANNUAL MEETING

EVALUATION

Please complete this evaluation form (omit those sessions or events you did not attend) and return to the Secretary, John T. Garner, 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

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

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

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

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

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Please print Name:

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1127 East Green Street
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NOTES

NOTES

NOTES

Heading 1957

NOTES

Yes

Reagan
Rich
K. Smith

No

Hunnicutt
R. Smith

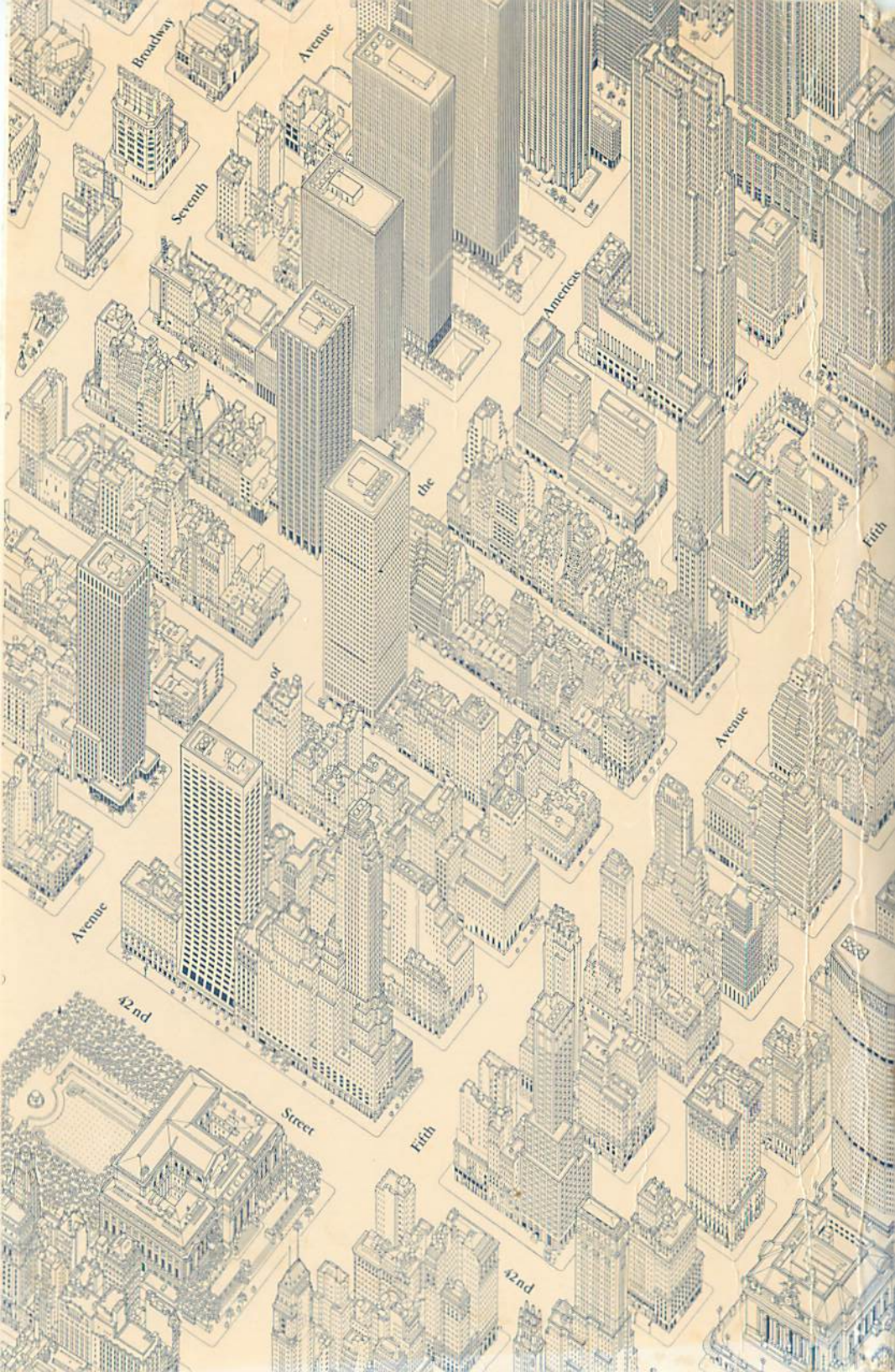
1958

- Peter Black
- Joe M. McWhorter

+

Harris
McCallum
Shotter
Carmel
B. Young
W. Shucart
Scott

- Bill Chandler
- Mike Aguirre
- Susie Tindall
- (J Silverberg)
- Allen Wyler
- Byron Young
- Wm. Shucart
- Morris Ray


Broadway
Avenue
Seventh
Americas
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Avenue
Avenue
42nd
Street
Fifth
42nd