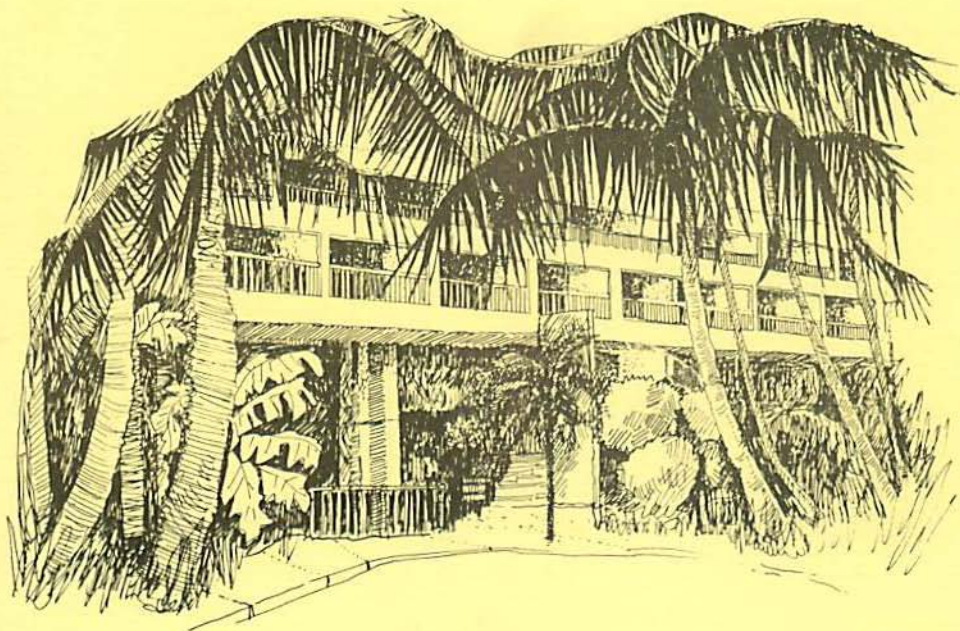


American Academy of
Neurological Surgery
ANNUAL MEETING

Kamuela, Hawaii
November 2-5, 1977



ANNUAL MEETING 1977



Mauna Kea Beach Hotel

Kamuela, Hawaii

November 2 - 5, 1977

THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

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Brain Death
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AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

MAUNA KEA BEACH HOTEL

Registration for Meeting	LOBBY LOUNGE
Aloha Cocktail Party	NORTH GARDEN
Breakfast Business Meeting	GARDEN PAVILION
Scientific Session	AUDITORIUM
Coffee Break	GARDEN PAVILION
Evening Hawaiian Luau	NORTH POINTE
Banquet	BATIK ROOM

PROGRAM 1977

WEDNESDAY, NOVEMBER 2

- 4:00 - 7:00 P.M. Registration - Lobby Lounge
6:30 - 8:00 P.M. Aloha Cocktail Party - North Garden
(Dinner on your own - jackets required)

THURSDAY, NOVEMBER 3

- 7:30 A.M. Breakfast and Business Meeting - Garden Pavilion
Members Only
8:20 - 10:30 A.M. Scientific Session - Auditorium
10:30 - 10:50 A.M. Coffee Break - Garden Pavilion
10:50 - 12:10 P.M. Scientific Session - Auditorium
12:10 P.M. Lunch - on your own
1:30 - 3:00 P.M. Scientific Session - Auditorium
(Academy Award Winner Presenting at 1:30)
3:00 - 3:20 P.M. Coffee Break - Garden Pavilion
3:20 - 4:00 P.M. Scientific Session - Auditorium
6:30 P.M. Cocktails - North Pointe
8:00 P.M. Luau Hawaiian Feast - North Pointe
(Aloha attire - entertainment)

FRIDAY, NOVEMBER 4

- 7:30 A.M. Breakfast and Business Meeting - Garden Pavilion
Members Only
8:30 - 10:30 A.M. Scientific Session - Auditorium
10:30 - 10:50 A.M. Coffee Break - Garden Pavilion
10:50 - 12:00 Noon Scientific Session - Auditorium
(Presidential Address at 11:30)
12:00 Noon Lunch - on your own
1:00 P.M. Tennis and Golf
7:00 P.M. Cocktails - Batik Room
8:15 P.M. Banquet - Batik Room

SATURDAY, NOVEMBER 5

- 8:00 A.M. Breakfast and Business Meeting - Garden Pavilion
Members Only
9:00 - 10:40 A.M. Scientific Session - Auditorium
10:40 - 11:00 A.M. Coffee Break - Garden Pavilion
11:00 - 12:30 P.M. Scientific Session - Auditorium

LADIES PROGRAM

WEDNESDAY, NOVEMBER 2

4:00 P.M. - 7:30 P.M. Registration - Lobby Lounge

6:30 P.M. - 8:00 P.M. "Aloha" cocktail party - North Garden

THURSDAY, NOVEMBER 3

10:00 A.M. - 12:00 Noon Hawaiiana demonstration - North Pointe

6:30 P.M. - 8:00 P.M. Cocktails - North Pointe

8:00 til Luau - Hawaiian Feast - North Pointe
and entertainment

FRIDAY, NOVEMBER 4

1:00 P.M. Tennis & Golf

7:00 P.M. Cocktails - Batik Room

8:15 P.M. til Banquet - Batik Room



Scientific Program

THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

Kameula, Hawaii

November 2 - 5, 1977

12-7-77

MODERATOR: William H. Sweet

THURSDAY, November 3

8:20 a.m.

Welcoming Remarks and Announcements

8:30 a.m.

1. Management and prognosis of intracranial germinomas
-- Follow-up study of 69 cases --

Ryuichi Tanaka, Hiroshi Kameda
and Komei Ueki
Niigata, Japan

42 part of
13 suppressor
other - hist +
in the region

In a series of 103 pineal tumors including so-called "ectopic pinealomas" there were 69 germinomas, 13 teratomas, 6 mixed teratoma and germinomas, 3 tumors with origin of the pineal parenchyma, and others.

For diagnosis of germinomas, clinical and neuro radiological findings and tumor cell diagnosis in CSF were very useful. The adequacy of the diagnosis was confirmed in most cases retrospectively after radiotherapy to recognize tumor shrinkage.

Follow-up study of 47 germinomas treated with radiotherapy revealed that the 5 year and 10 year survival rates were 44.7% and 23.4% in the whole germinomas, and 61.5% and 38.5% in the germinomas of the pineal region. Seventeen out of 26 irradiated cases with germinoma in the pineal region are still surviving, leading useful lives, 4 cases more than 15 years, 5 cases 10 to 15 years, and 6 cases 5 to 10 years. In the series of the pineal region germinomas, the smaller tumors responded very well to radiotherapy, whereas the larger ones showed a tendency to recurrence.

Since results of radiotherapy are fairly good and even palliative operations can be a hazard, the most promising management of germinomas may be radiotherapy before any surgical treatment except shunting operation if necessary.

Only 2 pineal germinomas
(17 cases - 100%)

(Discussion)

10 yr survival rate 22.9
x 1.5 = 38.5

Pattern: Tumor = hist +
always use whole brain irradiation.

Wilson: P>T reduces T lymphocyte competence.
Tumor bulk important - the larger the
8:50 a.m. poorer the response by lymphocytes

2. Appearance and Evolution of Immuno-deficiency in Brain Tumor Bearing Rats

Hachiki Sobue, Keiichi Inoue, Makoto Minagawa,
Kenichi Tanimura, and Komei Ueki
Miigata, Japan

ENU-induced rat glioma cells were transplanted to allogeneic 93 WKA rats, 35 subcutaneously and 58 intracerebrally. Intracerebrally transplanted tumor cells (1×10^6) developed into large mass with high incidence of take (89.7%). Tumor increased in size most rapidly 20-30 days post-implant. Subcutaneously transplanted tumor cells (1×10^6) were rejected rapidly or regressed spontaneously 12-15 days post-implant with temporary growth.

Lymphocyte infiltration, developed by the transplantation immunity, appeared in and around the tumor at the brain, and was poor compared with that at the subcutaneous tissue. The loss of quantitative balance between the tumor cells and the lymphocytes mobilized to the lesion, seemed to be one of the most important factors for the take and growth of tumor.

Microcytotoxicity assay in vitro showed that peripheral lymphocytes in both subcutaneously and intracerebrally tumor bearing rats destroyed the tumor cells specially 7-15 days post-implant. In intracerebrally tumor bearing rats, this ability was gradually lost along with the tumor growth, especially in the final stage (30 days-).

In intracerebrally tumor bearing rats, blastogenesis of lymphocytes to PHA tended to fall from the beginning of tumor growth (7-10 days), although the tumors were not large enough to decline the physical conditions of the hosts.

The suppression of cytotoxicity and blastogenesis with tumor growth, seems to be another factor to destine the continuous proliferation of tumor cells in the brain.

(Discussion)

9:10 a.m.

3. Radioimmunoassay of astroprotein (an astrocyte-specific cerebroprotein) in cerebrospinal fluid

K. Morimoto, T. Mori, Y. Ushio, T. Hayakawa and H. Mogami
Osaka, Japan

5. Appearance and Evolution of Immuno-Responsiveness in Brain Tumor Bearing Mice

Hiroki Soga, Kazuo Inoue, Masao Minagawa, Kazuo Tamura, and Kazuo Imai
Mitsui, Japan

Brain-tumor-bearing mice were transplanted with allogeneic B16 melanoma cells. The tumor-bearing mice were transplanted with B16 melanoma cells and B16 melanoma cells were transplanted with B16 melanoma cells. The tumor-bearing mice were transplanted with B16 melanoma cells and B16 melanoma cells were transplanted with B16 melanoma cells. The tumor-bearing mice were transplanted with B16 melanoma cells and B16 melanoma cells were transplanted with B16 melanoma cells.

Lymphocyte proliferation was induced by the transplantation of tumor cells. The tumor-bearing mice were transplanted with B16 melanoma cells and B16 melanoma cells were transplanted with B16 melanoma cells. The tumor-bearing mice were transplanted with B16 melanoma cells and B16 melanoma cells were transplanted with B16 melanoma cells.

Immunological assay in vivo showed that lymphocytes were transplanted with B16 melanoma cells and B16 melanoma cells were transplanted with B16 melanoma cells. The tumor-bearing mice were transplanted with B16 melanoma cells and B16 melanoma cells were transplanted with B16 melanoma cells.

In immunological assay in vivo showed that lymphocytes were transplanted with B16 melanoma cells and B16 melanoma cells were transplanted with B16 melanoma cells. The tumor-bearing mice were transplanted with B16 melanoma cells and B16 melanoma cells were transplanted with B16 melanoma cells.

The appearance of cytotoxicity and lymphocytes was transplanted with B16 melanoma cells and B16 melanoma cells were transplanted with B16 melanoma cells. The tumor-bearing mice were transplanted with B16 melanoma cells and B16 melanoma cells were transplanted with B16 melanoma cells.

(Continued)

3. Radioimmunoassay of Adrenocortical Steroids in Serum of Mice

K. Takahashi, T. Mori, Y. Ishida, T. Hayakawa and H. Higashi
Osaka, Japan

4,000 Med. wt.

An attempt was made to apply astroprotein (Benda et al 1970 and Mori 1970) to immunological diagnosis of central nervous system disorders. Using radioimmunoassay, astroprotein concentration in cerebrospinal fluid (CSF) was measured in 81 patients with intracranial disease (60 brain tumors and 21 miscellaneous) and 4 patients without central nervous system disorders. Astroprotein concentration in CSF was below 25 ng/ml in all patients who were free from intracranial disorders. In 9 out of 14 patients (64%) with glioblastoma or astrocytoma, the level of astroprotein in CSF was more than 25 ng/ml. In 3 patients with glioblastoma, astroprotein level elevated more than 500 ng/ml. Of 46 patients with intracranial tumors other than glioblastoma or astrocytoma, 10 patients (22%) showed astroprotein level more than 25 ng/ml of CSF. Of 21 patients with central nervous system disorders other than tumors, 4 patients (19%) showed astroprotein level more than 25 ng/ml of CSF.

We conclude that measurement of astroprotein level in CSF might be useful not only as a screening test of brain tumors such as glioblastoma or astrocytoma, but also an indicator of the degree of brain damage.

neutron

Sketches + brain contours (Discussion) *neutron capture therapy - and other*
"markers" (3-4 mentioned) *after using the astroprotein*
9:30 a.m. *lects only 1 wk. ? proportional to tumor size?*

4. A FOREFATHERS' TRAIL - BORON NEUTRON CAPTURE THERAPY

Hiroshi Hatanaka
Tokyo, Japan

neutron
brain tissue

neutron
how to
measure
large

Application of neutron capture reaction, of either Lithium-6 or Boron-10 which emits alpha particle as secondary radiation to cancer treatment was first proposed by Locher in 1936. Sweet, Javid, Zervas and others considered such a therapy will be suited to treat brain tumors specifically, because the brain tissue is not penetrated by the boron compounds that are easily taken up by tumor cells. After the earlier series at Brookhaven National Laboratory and MIT reactors between 1953 and 1961 conducted by Sweet and Farr, Hatanaka joined the project group in 1964 and back in Tokyo he and Sano resumed treatment with revised protocol by using mercaptoundecahydrododecaborate which was originally studied by Soloway, Hatanaka and Davis. His earlier series between 1968 and 1974 was mostly conducted at Hitachi Training Reactor with minimal yield of thermal neutron flux and only 15 patients were treated, mainly due to short supply of boron-10 isotope and lack of a medical radiation facility. The average survival for glioblastoma patients, most of whom had been treated elsewhere with conventional therapies has exceeded 28 months. Some patients are fully active in their occupation

7.
neutron delivery therapy: boron level

author has average of long survival (3-4 years)

even after several years. Since March 1977, Musashi Institute of Technology reactor specifically re-designed for this purpose has been employed, and at least twelve patients' treatment is expected before March 1978. Use of epithermal neutron for deep-seated tumors and development of simultaneous dose monitoring device are the new features of current studies. The Boron-10 enrichment and production of the compound by the Shionogi Research Laboratory will make it possible to treat two dozen patients in the coming year.

Chondro sarcoma was benign also.
(Discussion)

9:50 a.m.

5. Short Course Irradiation of Glioblastoma Multiforme

Robert L. McLaurin, Vicharn Lorvidhaya,
and Bernard S. Aron
Cincinnati, Ohio

During the past two years, nine consecutive patients with glioblastoma multiforme have been treated with "short course irradiation" following biopsy for partial tumor removal. The purpose of this study was to determine whether there was justification for subjecting the patient to more prolonged irradiation in view of the limited survival time of such patients. A total of 2,500 or 2,700 rads were delivered in five daily treatments. Decadron was administered during irradiation.

The results in this group of patients were compared with previously treated patients in whom surgery alone or surgery plus prolonged irradiation had been used. The results indicate that the present group of patients showed a survival rate similar to those who had been treated with more prolonged radiation and distinctly better than those treated with surgery alone. It is concluded that short course irradiation is well tolerated, is equally as effective as more prolonged irradiation following surgery, but is less effective than other recently reported therapeutic regimens.

(Discussion)

Monday: 50% with prolonged survival. Some with 10-15% by post-irradiation.

10:10 a.m.

6. Excision of Craniopharyngioma: Surgical technique and operative morbidity

yr	66%	Russel H. Patterson, Jr. and
2		Alec Danylevich
5	72%	New York, New York
10	89%	8.

new study

During the past two years, 9 patients were subjected to a presumed total removal of a craniopharyngioma. Removal was accomplished with least trauma through a subfrontal approach behind the optic chiasm and between the optic tracts to free the tumor from the hypothalamus. Once freed from behind, large tumors were extracted between the optic nerves after removal of the tuberculum sellae. not from

The postoperative morbidity in this group was compared with 20 consecutive operations limited to partial excision, and the results are summarized below.

	Subtotal	Total
patients	16	9
operations	20	9
mortality	0	0
postoperative stay	16 days	23 days
acuity better	55%	11%
acuity worse	10%	22%
difficult water balance	0	22%
steroid dependent	88%	100%

Since our earlier reported experience demonstrated a recurrence rate after subtotal removal without radiation therapy of 72% at 5 years and 89% at 10 years, we believe that total removal should be attempted in all cases with the exception of children who are still growing.

(Discussion)

10:30 a.m.
Coffee Break

10:50 a.m.
7.

Cushing's Disease Revisited

(Sella normal in a lot also)

Charles B. Wilson and Blake Tyrrell
San Francisco, California

adrenals have been normal - absent to subnormal.

In 1932 Cushing delivered his classic paper on pituitary basophilism. Convincingly and with characteristic clarity he described the clinical and pathological findings in hypercortisolism caused by pituitary basophilic adenomas, and that form of Cushing's Syndrome has been designated Cushing's Disease.

9.

20 yrs. using fec cortisol over 100 is best test for Cushing's disease.

Smallest 1.5mm found in total gland removal.

The authors have evaluated 30 patients with Cushing's Disease, all of whom have undergone transsphenoidal exploration of the pituitary gland. The usual finding has been a 2-6 mm microadenoma, and in 85% of cases normal function of both pituitary and adrenal glands has been restored. Presented will be preoperative diagnostic testing, operative technique and results.

Had to back out of 2 because of large benign
dura. all patients = Cushing probably have
bicyclic adenoma.
(Discussion)

11:10 a.m.

8. SYMPTOMATIC PITUITARY TUMOR ENLARGEMENT AFTER INDUCED PREGNANCY

P.B. Nelson, A.G. Robinson, D.F. Archer
and J.C. Maroon
Pittsburgh, Pa.

2 pituitary
1 "Epilepsy"

Conscious patient's
tumor

A case is presented and 11 cases are reviewed in which symptomatic pituitary tumor enlargement occurred during pregnancies which resulted from induced ovulation. This complication is being reported more commonly with the availability of gonadotropins and bromocriptine for induction. The syndrome is usually characterized by headache and visual disturbances with bitemporal field cuts, but may present with ocular muscle palsies. The cases are divided into two groups. One group had a shorter duration of amenorrhea, 3.8 years, developed symptoms before the 14th week of pregnancy, and were usually treated by tumor removal. The second group had a longer period of amenorrhea, 10.1 years, and developed symptoms after the 24th week of pregnancy and were usually treated by delivery of the infant. Tumor removal or termination of the pregnancy both resulted in resolution of symptoms. All pregnancies resulted in normal infants. This series provides guidelines for management of future cases.

(Discussion)

Transsphenoidal removal
recommended.

11:30 a.m.

9. POSTERIOR FOSSA MENINGIOMAS, OUR EXPERIENCE WITH 31 CASES

John Cuff, Eben Alexander
Winston-Salem, North Carolina

Thirty-one patients operated on for posterior fossa meningiomas at North Carolina Baptist Hospital between 1949-1977 are reviewed. These include 12 lateral convexity tumors, 8 in the cerebello-pontine angle, 6

foramen magnum, 3 primarily involving the tentorium, and 2 clivus tumors.

The initial symptoms, all symptoms at the time of diagnosis, length of time between onset of symptoms and diagnosis, and physical signs are presented and discussed.

A review of the neuroradiological studies obtained is presented with a discussion of the role of computerized cranial tomography. 6 cases are presented which made use of this diagnostic tool. The impact on treatment and diagnosis of these patients, especially in terms of early diagnosis and surgery, is discussed.

Morbidity and mortality are discussed and correlation with tumor location and clinical presentation is made. Suggestions for anticipating the high risk patient and for decreasing the surgically related morbidity and mortality are presented.

Finally, a discussion of our long-term surgical experience is presented with respect to total or partial removal, location of tumor, and other clinical or surgical parameters.

8 operating deaths

(Discussion)

11:50 a.m.

Des. number 4 Fisher gives. This is different than the order of malignancy

10. Cerebral Metastatic Chorionic Carcinoma: A Unique Case

Robert G. Fisher, Scott Bennion,
Dan Daniel Frimmer, Robert L. Malatesta
Plainfield, New Jersey

This individual case report is most unique. The patient presented with having a typical course for this type of lesion -- bleeding into the subarachnoid space from a mass lesion readily diagnosed by neuroradiologic means. However, at no time was any demonstrable mass in either the pelvis or the lung found. The human gonadotrophic hormone was markedly elevated.

Combined surgical, cobalt irradiation, and chemotherapy has effected a cure.

The literature will be discussed and pertinent points learned both from the experience of others and the author will be pointed out.

(Discussion)

12:10 p.m.

Lunch

MODERATOR: Robert S. Knighton

1:30 p.m.

Wass: blood or unit of plasma

11.

ACADEMY AWARD

Medial hypothalamic neurons and their connections:
A neural network regulating pituitary function.

*Howel
pitted
electrode*

Howard W. Blume
Montreal, Quebec, Canada

4+

2:00 p.m.

12. On the occurrence of rupture of cerebral aneurysms during intracranial operation.

Yuji Miyazaki
Sapporo, Japan

Even today when remarkable advances have been achieved in intracranial direct operation techniques of cerebral aneurysm, there is always a possibility of rupture during the operation. Thus in attempt to cope with this insidious problem with the dual aim of finding preventive measures and procedure to be followed in the case of ruptures during operation, a study was made on cerebral aneurysm ruptured cases occurring during operations.

Up to 1976 I have conducted intracranial direct operation of 376 cerebral aneurysms (337 cases), out of which 48 aneurysms ruptured during operation (12.8%), and among these cases internal carotid aneurysms showed the highest incidence (20.0%).

It was found that no age level correlation was seen with rupture and cases with 3 previous ruptures showed the highest frequency of 25.0%. Cases within one month after the last rupture showed a high frequency of 21.0%.

The causal factor of rupture of cerebral aneurysm during operation in 44 cases (91.7%) was clearly attributable to operative procedures and these factors will be described in detail.

On the other hand, rupture of the cerebral aneurysm during operation without the aid of a surgical microscope showed a frequency of 21.0%, while in contrast the incidence of rupture under a surgical microscope was 11.9%, and I wish to emphasize that even in the latter the frequency of rupture during operation is far from low.

The state of the ruptured site of cerebral aneurysm will be described in detail. The value of intracranial interruption of cerebral blood flow under

*Microncope was little difference
provisional temporary occlusion this 3x best effect*

hypothermia and artificial hypotension for prevention of rupture of cerebral aneurysm will be discussed.

(Discussion)

2:20 p.m.

13. SURGICAL MANAGEMENT OF INTRACRANIAL GIANT ANEURYSMS

Yoshio Hosobuchi
San Francisco, California

The introduction of microsurgical techniques has greatly improved the surgical management of intracranial aneurysms. However because of their size, direct surgical attack on giant aneurysms still presents a considerable challenge.

Obliteration of the aneurysmal cavity by a mass of coiled wire was achieved in a single case by Werner and colleagues in 1941. In 1971, J.F. Mullan reintroduced this technique for the electrometallic thrombosis of cerebral aneurysms. Its purpose is to induce intramural thrombosis of the aneurysm by inserting fine (copper, copper-alloy, or stainless-steel) wires into the aneurysm and reinforcing the aneurysmal wall with large quantities of packed, fine wire.

During the past seven years, the author has operated on 40 cases of giant intracranial aneurysms, 21 of which were treated by the electrometallic thrombosis technique; the others were treated by clipping the neck of the aneurysm, entrapment, or carotid ligation. This report discusses the indications for the electrometallic thrombosis technique, and presents findings which indicate that it is the optimal management technique for giant aneurysms of the basilar artery and carotid bifurcation.

(Discussion)

Does not give complete thrombosis - at aneurysms

2:40 p.m.

14. THE INFLUENCE OF NITROPRUSSIDE ON CEREBRAL AUTOREGULATION

Martin H. Weiss, John Spence and Theodore Kurze
Los Angeles, California

Ten cats were studied to assess the question of abolition of cerebral autoregulation attendant to the use of nitroprusside for hypotensive anesthesia. The animals were anesthetized with pentobarbital, 30 mg./kg., following which appropriate cannulas were placed enabling continuous

40 cases 30% 10% to mortality 15%

monitoring of arterial and central venous pressures. Animals were ventilated on a constant volume ventilator; end tidal CO₂ content was continuously monitored and maintained at 4%. Core temperature was maintained at 38 degrees centigrade. A cannula was stereotaxically placed in the lateral ventricle enabling continuous monitoring of intracranial pressure utilizing appropriate transducers and amplifying systems.

After the establishment of stable baseline parameters as described above, a continuous infusion of sodium nitroprusside was begun in a dose sufficient to maintain a mean systemic arterial pressure (SAP) of 65 mm. Hg. The infusion was continued for incremental periods of 30 seconds to 10 minutes, increasing the time of infusion by 30 seconds for each subsequent trial. At 10 seconds following the cessation of nitroprusside administration, intravenous dopamine was infused to raise the systemic arterial pressure to a mean of 100 mm. Hg., and the subsequent response in intracranial pressure was recorded in each instance.

In no animal was a loss of cerebral autoregulation noted when the nitroprusside infusion was maintained for 3 minutes or less. When the infusion was maintained for 4 minutes or longer, cerebral autoregulation was lost in each animal, and the length of time to return of cerebral autoregulation correlated with the duration of nitroprusside infusion. Infusions of duration of 3½ to 4 minutes evoked variable responses; in some, cerebral autoregulation was lost, in some, autoregulation persisted until infusions beyond 4 minutes in duration were established.

It is apparent that sodium nitroprusside disturbs the integrity of cerebral autoregulation and that the onset and extent of this disturbance is a dose-dependent phenomenon.

(Discussion)

3:00 p.m.
Coffee Break

3:20 p.m.

15. VASCULAR CHANGES IN ARTERIOVENOUS MALFORMATIONS OF THE BRAIN

Bennett M. Stein, and Samuel M. Wolpert
Boston, Massachusetts

*1.5 to 3 mm
subcuti*

A review of our experience with 48 arteriovenous malformations of the cerebral hemisphere will be presented. To be stressed are the aspects relating to the vascular changes accompanying therapeutic embolization and post operative vascular changes which are seen in a dynamic state by serial angiography.

14.

*next to bow out
stop each.*

The majority of the cases have had embolization and surgery as a form of treatment. We have observed dynamic vascular changes as a response to occlusion of feeding vessels and the interstices of these arteriovenous malformations. These vascular changes following either embolization or surgery are indicative of an evolving change in the circulation when a high flow arteriovenous shunt is partially or totally eliminated and the arterial supply associated with this shunt returns to its normal function.

The changes which are observed through frequent angiographic studies include the following:

1. Initial increase in caliber of the feeding arteries when occlusion of these vessels occurs followed by a very gradual decrease in the size of feeding arteries with apparent stasis and prolonged ectasia,
2. A variable course of events associated with the size of coincidental aneurysms on the feeding vessels when these vessels are eliminated from their role as contributors to an arteriovenous malformation,
3. Cerebral edema either short term or prolonged is assumed to be associated with neurological symptomatology appropriate for the changing arterial and venous patterns.

The paper will be illustrated by 35 mm slides, primarily of angiographic material.

(Discussion)

3:40 p.m.

16. Acquired and Congenital Vascular Malformations of the Middle Meningeal Artery

James T. Robertson, B. King Tipton, J.D.,
and James Langston
Memphis, Tennessee

Arteriovenous malformations of the middle meningeal artery are rare. The incidence and significance of such lesions have been debated. In recent years, with the advent of advanced neuroradiologic techniques, increasing numbers of these lesions have been reported. In this paper, the authors review the available literature and recount their own surgical experiences with nine cases of pseudoaneurysm and arteriovenous fistulae of the middle meningeal artery.

Dural vascular malformations can be associated with sudden or gradual neurological deterioration after head injury. Rarely, such lesions appear to be unassociated with craniocerebral trauma, i.e. of developmental origin. Emphasis has been placed on the variable natural

Resistance to his: 1 million sent to
show anticoagulants, no good
glucose - but neurologists
continue to resist.
∴ I will continue
to do bypass, no
matter what.

- I degree of risk (small)
- II efficacy (present study)
- III controlled trial (being initiated
as a group trial now).
- IV general use.

Sundt: clinic neurologists believe it a
very useful operation.

history of this disease, the need for proper radiologic technique allowing clearer delineation, and the value of careful followup allowing timely surgical intervention. A method of approaching middle meningeal artery arteriovenous fistulae and post-traumatic aneurysms is outlined.

(Discussion)

Friday, November 4

MODERATOR: Arthur A. Ward, Jr.

8:30 a.m.

17. **CEREBRAL REVASCULARIZATION -
PRESENT VALUE AND FUTURE TRENDS**

James I. Ausman, Myoung C. Lee,
Shelley N. Chou, and Lyle A. French
Minneapolis, Minnesota

*Protocol number
newly set in post
grading evaluation*

Fifty consecutive patients undergoing anterior and posterior circulation cerebral revascularization in the last three years have had detailed pre- and postoperative neurologic, neuropsychologic and angiographic assessment. In STA/OA-MCA anastomosis there has been no operative mortality, 4% postoperative morbidity, 2% long term mortality from MI and 2% from stroke. An independent neurologist found no patients with increased deficit after surgery, while 40% were improved objectively and 80% stated they were better subjectively in operations performed at least 3 months after ischemic infarction. Neuropsychologic evaluation six months or more after the procedure indicated several cases of significant documented improvement and none who were worse. In 100 pre- and postoperative cerebral angiograms there was no mortality and a 1% mild permanent morbidity. In 80% of patients with postoperative angiograms, 98% of the anastomoses were patent. Cerebral filling through the anastomoses evaluated by a new method of angiographic analysis correlated with the clinical result.

surgical New approaches to posterior circulation ischemia including OA-PICA, STA-superior cerebellar artery and vertebral artery-PICA using a vascular graft will be shown. For patients with ipsilateral common and ICA occlusions, subclavian to external carotid followed by STA-MCA bypasses have been employed for intracranial revascularization.

(Discussion)

8:50 a.m.

18. **The Effect of STA-MCA Anastomosis on rCBF
Following Experimental MCA Occlusion.**

Richard S. Kramer, and
W. Jerry Oakes
Durham, North Carolina

Eight of twelve mongrel dogs subjected to trans-orbital occlusion of the proximal left cerebral artery underwent left superficial temporal artery-middle cerebral artery (STA-MCA) anastomosis 5 to 38 days after occlusion. Instantaneous rCBF was determined in all animals prior to MCA occlusion ("control rCBF") by trans-femoral left ventricular (LV) injection of polystyrene microspheres labelled with Sr-85 under light general anesthesia. The effect of subsequent left MCA occlusion on the redistribution of rCBF was determined by LV injection of microspheres labelled with Sc-46 immediately prior to STA-MCA anastomosis.

The anastomoses were either evaluated acutely after 2 hours (3 dogs) or allowed to mature for 2 to 21 days (5 dogs) prior to final determination of rCBF by LV injection of microspheres labelled with Ce-141, again under controlled conditions of arterial blood pressure, pH, pO₂, and pCO₂; the patency of each anastomosis was evaluated angiographically prior to sacrifice. The diameter of the microspheres (15+ 3 microns) assured that each determination reflected nutrient, rather than non-nutrient (e.g. "A-V shunt"), flow to uniform volumes of cerebral tissue perfused by both left and right anterior cerebral (ACA), middle cerebral (MCA), and posterior cerebral (PCA) arteries in each dog. Every animal demonstrated persistent clinical evidence of left hemispherical ischemia (e.g. right hemiparesis, circling gait) after MCA occlusion. Following sacrifice and fixation, the cerebral hemispheres were dissected according to primary arterial perfusion patterns (ACA, MCA, and PCA) and analyzed for each of the three differentially-labelled microspheres, employing a 3-channel auto-gamma spectrometer; spectral "cross-talk" was corrected by computerized matrix analysis.

Regardless of the interval (5 to 38 days) following left MCA occlusion, nutrient CBF was redistributed so as to maintain control-level perfusion in the distribution of the occluded artery at the expense of the ipsilateral PCA and (to a lesser degree) ACA flow. This phenomenon has not heretofore been documented, and is considered to reflect the intensity and longevity of the local hyperemic, leptomenigeal collateral, and angioproliferative response to regional cerebral ischemia; in essence, this observation suggests that a patho-physiologic intra-cerebral "steal" routinely accompanies cerebro-vascular occlusion.

Left STA-MCA anastomosis was observed to exert a mildly deleterious effect on left hemispherical perfusion when measured acutely (2 hours after bypass), presumably as a consequence of operative vascular manipulation. Mature anastomoses (2 to 21 days) enhanced left hemispherical flow only in those animals in which MCA flow was significantly reduced following occlusion; in animals demonstrating successful maintenance of homeostatic left MCA flow after occlusion (by virtue of focal hyperemia, collateralization, etc.), STA-MCA anastomosis actually resulted in decreased rCBF in the MCA distribution.

Our experiments suggest that:

1. Following proximal MCA occlusion, canine rCBF is redistributed so as to maintain effective perfusion of the "ischemic" zone by re-directing flow from the ipsilateral PCA, and ACA, territories.
2. The STA-MCA bypass procedure cannot be usefully evaluated, either in metabolic or rheologic terms, during the first few hours after anastomosis.
3. The effectiveness, after "maturation", of an STA-MCA anastomosis is inversely related to flow in the MCA distribution pre-operatively; an innovative clinical method for determining regional MCA flow will be discussed.
4. While post-operative angiograms will define the patency of STA-MCA anastomosis they do not necessarily reflect the hemodynamic effects of this procedure.

Co-metabolites
EM infarct
few records

(Discussion)

9:10 a.m.

19. Effect of Lasix on Experimental Traumatic Cerebral Edema

Robert L. McLaurin, Patricia Tornheim Brown
and Raymond Sawaya
Cincinnati, Ohio

The present study was designed to evaluate the effect of Lasix on traumatic cerebral edema. Anesthetized cats received craniocerebral trauma delivered by the Remington Humane Stunner. Following injury the animals were given a fixed fluid and electrolyte intake. Nine animals received Lasix and compared with a control group of 9 animals given no medication. All animals were sacrificed at 48 hours and the heads immersed in liquid nitrogen. Only animals with unilateral cerebral contusion were used. The amount of edema in the contused and non-contused hemispheres was determined by a specific gravity technique employing a density gradient.

The results in these groups of animals were also compared with non-impacted controls and with impacted animals given ad lib fluid intake. The results indicate that despite some degree of systemic dehydration in the controlled-intake animals, the uncontused hemispheres of treated and untreated animals were similar in water content to the control non-impacted cats. The contused hemispheres of the treated animals had a significantly lower water content than those of untreated animals. In addition the pattern of edema distribution in treated animals was more restricted than in the untreated animals. It is concluded that Lasix appears to have a specific reducing effect on traumatic cerebral edema that does not depend simply on bodily dehydration.

(Discussion)

As expected is in brain surrounding to contusion.

why is reduced filtration pressure

or
? ischemic choroidal epithelium
transient changes

Dopamine & pentobarbital used because
↑ or ↓ effects are cardiac - not
peripheral vascular (like epinephrine
& asford).

9:30 a.m.

20. MODULATION OF CSF PRODUCTION BY ALTERATIONS IN CEREBRAL PERFUSION PRESSURE

Martin H. Weiss, Nancy Wertman
and Theodore Kurze
Los Angeles, California

Forty-eight cats have been studied to assess the interrelationship between intracranial pressure (ICP) and systemic arterial pressure (SAP) with respect to CSF production. Adult cats were anesthetized with intraperitoneal pentobarbital, 30 mg./kg., and appropriate cannulas were placed to enable continuous monitoring of systemic arterial and central venous pressures. The animals were ventilated on a constant volume ventilator; end tidal CO₂ content was continuously monitored and maintained at 4%. Core temperature was maintained at 38 degrees. A ventriculocisternal perfusion was established according to the technique of Pappenheimer, et al., using warmed Elliott's B. solution containing RISA¹²⁵ as a nondiffusible indicator. Intracranial pressure was set at a predetermined level (2 or 20 mm. Hg.) by adjusting the height of the outflow cannula. Blood pressure was continuously monitored and maintained at means of 70 and 115 mm. Hg. Four groups were studied in which cerebral perfusion pressure (CPP) was set at 50, 70, 95, and 115 mm. Hg. Calculations of CSF production (CSF_p) were made according to the formulas derived by Pappenheimer, et al..

The mean rate of CSF_p at CPP of 70 mm. Hg. or above averaged 20 μ l./minute whereas reduction of CPP to 50 mm. Hg. (ICP 20 mm. Hg. and SAP 70 mm. Hg.) decreased CSF_p by approximately 50%. Statistical analysis, using an analysis of variance, reveals these to be significantly different ($P < .001$). Recent evidence indicates that this level of CPP exceeds the capacity of cerebral autoregulation to maintain constant cerebral blood flow, therein resulting in oligemic perfusion of choroid plexus. It is apparent that under conditions of stable SAP, CSF production may be significantly influenced by cerebral perfusion pressure.

(Discussion)

9:50 a.m.

21. CSF Antibiotic Levels During Treatment of Shunt Infections

Robert L. McLaurin and Steven L. Wald
Cincinnati, Ohio

no dose 2/3 injections without serum
shunt.

uses intraventricular dose
1x in lucasol - for prophylaxis
(+ systemic 2/3 days before).

Stop amn
opisth
E. Calc
etc.

During the past decade the treatment of shunt infections has been influenced by the observation that in some instances removal of the shunt is not necessary but rather that the infection can be controlled by a combination of intraventricular and systemic antibiotic treatment. The intraventricular agent has been administered in arbitrary dosages and at arbitrary intervals but no systematic observations on the validity of the regimens have been made.

In the past year we have made serial observations on the CSF level of antibiotic achieved in 8 patients. Six patients have had Holter shunt systems in place while 2 have had ventriculitis without a shunt system. In 2 patients multiple courses of treatment using different antibiotics have been used. The antibiotics included in these observations are methicillin, cephalothin, and gentamycin. The concentrations of antibiotics have been compared with the minimal inhibitory concentration (MIC) in each instance.

The observations have demonstrated that with the dosages being currently used in our clinic it is probably not necessary to administer intraventricular therapy more often than every other day since adequate levels are maintained during that interval even with a functioning shunt. A corollary to this conclusion is that testing for efficacy of treatment requires that the CSF sample be taken not sooner than 72 hours after cessation of treatment. It is recommended also that if facilities are available antibiotic concentrations and MIC determinations should be done during the course of treatment of each patient.

(Discussion)

10:10 a.m.

22. THE MANAGEMENT OF ENCROACHMENT OF THE CERVICAL SPINE

Mitsuo Tsuru
New York, New York

Hokaido

In recent years, many factors have been blamed for the myelopathy of cervical spondylosis, with no general agreement as to their individual importance.

Some of these factors are mechanical, and include the sagittal diameter of the spinal cord, the size and attachments of the dentate ligaments, the presence of root-sleeve fibrosis and of intradural adhesions.

Vascular factors may also play a part. because the main blood supply of the cervical cord usually comes from two or three radicular arteries which may be involved by spondylitic spurs.

I think it is probable that more than one factor is operating in any individual patient. In this report, I would like to show the importance of

the predisposed narrow canal in the development of cervical myelopathy, in the cases of cervical spondylosis, ossification of the posterior longitudinal ligaments, and the atlanto-axial dislocation.

In the cases of atlanto-axial dislocation, I would like to also propose the definition and the indication of surgical treatment of the atlanto-axial dislocation, using the predisposed canal size and the so-called instability index.

(Discussion)

10:30

Coffee Break

10:50 a.m.

23. Disc Protrusions in Adolescents

Robert G. Fisher
Plainfield, New Jersey

Thirty cases of lumbar disc protrusions in patients 21 years or younger were operated with uniformly successful results initially. An initial follow-up period indicated that 10% of the cases required reoperation within three years.

Follow-up studies ranging up to 25 years are disclosing results that suggest long standing follow-up studies and attending are necessary.

The symptoms, signs, myelogram and operative findings are for the most part similar to those of the adult. Prompt recognition of disc protrusions in the first and second decades in the past has been lacking.

(Discussion)

11:10 a.m.

24. Anterior Cervical Discectomy and Interbody Fusion With a Synthetic Calcium-Phosphate

T. Shima, F.H. Mayfield,
and S.B. Dunsker
Cincinnati, Ohio

Anterior cervical discectomy with and without fusion has been reported upon repeatedly. One major objection to the interbody fusion is that it produces increased morbidity from pain at a site remote from the disease area. To avoid this increased morbidity heterologous substances such as bovine bone have been used with varying degrees of acceptance.

We have studied the use of an entirely new type of biomaterial: a resorbable ceramic. It is a purified form of tricalcium phosphate. This substance has the advantage that as osteocytes and other new tissue proliferate and calcify within the matrix, the matrix itself is absorbed. This material has been used successfully in some dental work and in plastic surgery.

Cervical discectomies and interbody fusions were performed in adult mongrel dogs. Comparisons were made between autogenous bone and the synthetic bone matrix, using fluorescent histologic techniques. These studies demonstrate the method of fusion in autogenous bone and these will be compared with the synthetic substance.

(Discussion)

*now fused.
Soft tissue pushed
into canal.*

11:30 a.m.

**PRESIDENTIAL ADDRESS
NEW KNOWLEDGE IN NEUROSCIENCE:
EXAMPLE, THE NEUROPEPTIDES**

William H. Sweet

**Saturday, November 5
Moderator: John J. Lowrey**

9:00 a.m.

25. Intraspinal Neurenteric Cysts

Robert H. Wilkins
Durham, North Carolina

Intraspinal neurenteric cysts are unusual lesions resulting from abnormalities of embryonic development of the spinal cord, spine, and gut. Other related developmental anomalies, such as persistence of the neurenteric canal and neurenteric cysts in other locations, are also part of the split notochord syndrome. Forty-three previously reported cases and two personal cases of intraspinal neurenteric cysts have been reviewed: their clinical, radiographic, surgical and pathological features will be presented.

(Discussion)

22.

Onset: trauma. Big rather than in one case that operated on because "trauma" was not operated on.

9:20 a.m.

26. Epidemiological Studies on the Patients with Persistent Vegetative State.

Kenichiro Higashi, Yoza Sakata, Mitsunori Hatano,
Seisho Abiko, Kiyoshi Ihara, Sanao Katayama,
Yukio Wakuta, Tomomi Okamura,
Kiryuki Ueda, Michihiko Zenke and Hideo Aoki
Ube, Japan

Vegetative patients resulting from severe brain damage were investigated epidemiologically with co-operation of 69 clinics in 16 prefectures of western Japan. After inquiring with doctors in this area, we examined 193 cases reported to us and selected 110 patients. The criteria for the selection were 1) defect of communication, 2) loss of expression of intention, 3) urinary and fecal incontinence, 4) complete loss of self-supportability, 5) continuation of above conditions beyond 3 months.

The causes of brain damage in this survey were varied. More than one-third of the cases were due to trauma, and more than one-fifth were from vascular accidents. Duration of the vegetative state varied from 3 months to 17 years. Three-year observation revealed that 65% of the patients died during this period. Mean survival time for dead patients was 38 months. Among survivors, only 3 patients recovered from vegetative state.

Reactivity, clinical signs, EEG findings, methods of management and results of various trials of treatment were investigated in connection with the patient's prognosis.

(Discussion)

9:40 a.m.

27. Mechanism of Tremor Generation

H. Narabayashi
Tokyo, Japan

Through experiences in human stereotaxic surgery on various extrapyramidal symptoms, one of the most important and established findings is the existence of unitary rhythmic burst discharges in the ventralis intermedius nucleus of the thalamus in cases with tremor.

These cellular activities in burst fashion can be divided in to three sub-groups; rhythmic tremor-locked one, rhythmic but not tremor-locked

*100% not work assigned to 100% cases
each 100% treatment --4*

The rest moved out of the PVS definition.

*usable. 3/4 part!
only 1 comp. kind of
stroke &
could walk assisted*

one and the non-rhythmic one.

Either stimulation or lesion-making in the exact area of tremor-locked rhythmic bursts increases or abolishes tremor.

It seems almost definite this area is generating tremor. But the pattern of frequency (c/s) of tremor rhythm, i.e. its phenotype seems to depend on the peripheral factor as well. Tremor-locked bursts are the results of afferent projection of muscle sense, which might suggest a model of reverberating circuit producing a kind of resonance-effect.

(Discussion)

10:00 a.m.

28. EXPERIMENTALLY-BASED PROPOSAL FOR MODIFIED TEMPORAL LOBECTOMY IN TLE

E.C. Poletti, M. Sujatanond, M.A. Kinnard,
G.C. Creswell, F. Morrison, M. Kliot,
R.N. Kjellberg, N.T. Zervas, W.H. Sweet and
P.D. MacLean
Boston, Massachusetts

Approximately 80% of patients suffering from intractable temporal lobe epilepsy (TLE) have pathology limited to the anterior hippocampus (AH) and the uncus. The pathways by which sub-threshold stimulation and seizures spread from the AH have been investigated during the last nine years in five series of experiments.

First, 666 basal diencephalic (BD) units in the awake monkey were studied during shock stimulation and AH seizure activity. A total of 13% of hypothalamic (Hyp), 28% of preoptic (Pro), and 32% of basal forebrain (BF) units were responsive.

Second, the results of an anatomical fornix degeneration study showed direct projections to certain Hyp, Pro and BF structures. These structures were shown in the previous study to have unit responses driven at long latencies (≥ 20 msec.) This suggested the possibility of a new pathway from AH to BD independent of the fornix system.

Accordingly, third, in awake monkeys with complete lesions of the fornix system, 619 BD units were tested to AH stimulation and seizure activity. A total of 12% of Hyp, 5% of Pro, and 18% of BF units were affected, establishing the postulated non-fornix pathway.

A current study of 519 amygdala units in awake monkeys has not found any short-latency driven units to AH stimulation or seizures. These results suggest that the non-fornix pathway from AH to BD does not pass through the amygdala, stria terminalis, or ventral amygdalofugal projections.

Most recently, a fifth study, using the radioactive 2-deoxyglucose technique in the rat, suggests that AH seizures are relayed to Hyp, Pro and BF structures via the subiculum and entorhinal cortex.

These combined results showing specific pathways for spread of sub-seizure and seizure activity from AH to BD raise the possibility of treating unilateral TLE with a more limited and less destructive operative procedure than temporal lobectomy. Based on our experimental data, two alternative operations are proposed. If either procedure should prove effective for unilateral TLE, it might also permit surgical therapy for the larger population of patients suffering from bilateral independent foci.

Micro to select beyond (Discussion) 5:00 pm from tip.
10:20 a.m. *lots of things to work forward to discuss*
col + work forward to discuss
col + subiculum. Temp hippocampus

29. Investigation of Cortical and Thalamic Neural Activity in the Late Phases of Somatosensory Evoked Potentials

J.A. Kusske, J.W. Hutchison and M. Verzeano

Ucal *Stain*
Evidence for experimentally induced supraspinal neural phenomena that lasts for long periods of time has been derived from several different sources in the last few years. Several pathologic clinical states have been related to this abnormal central activity which is thought to represent an alteration in firing patterns of neurons rather than a lesion. We have been interested in investigating neuronal networks which may underlie pathologic changes in neural activity. Recent investigations in our laboratory, both in humans and experimental animals, have demonstrated evoked oscillations that were time locked to a stimulus, and which extended as long as 3500 msec after the stimulus. These oscillations could be driven at certain frequencies of stimulation and contained components harmonically related to each other and to the frequency of the stimulus. In the cat these late oscillations were recorded not only in the somatosensory cortex; but in the specific and nonspecific thalamic nuclei as well. In the work reported here evoked potentials and associated neuronal activity, induced by repetitive stimulation of the sciatic nerve were recorded simultaneously from the somatosensory cortex, the ventro posterolateral and centre median nucleus in cats. Only the late components of the response extending beyond the first 500 msec were studied under high amplification. Averaging and autocorrelation studies of the late part of the evoked potential and of the associated neuronal discharge, show that their highest amplitude of oscillation and their highest degree of periodicity are reached at specific frequencies of stimulation, in the cortex as well as the thalamus. Cross correlation studies of the relations between gross and neuronal activities within the cortex or within the thalamus, as well as studies of the relations between cortical and thalamic activities show that the highest degree of correlation occurs when the frequencies of stimulation are within the same range. This suggests that the development of the late oscillations of the evoked potential and of the associated

neuronal discharge is based on the activity of cortical, thalamic and cortico-thalamic loops which respond most effectively at frequencies of stimulations related to their resonance characteristics. These results will serve to further our work in chronic pain states, epilepsy and in behavioral studies where memory consolidation is involved.

(Discussion)

10:40 a.m.
Coffee Break

11:00 a.m.

30. Variability in The Localization Of One And Two Languages In Cerebral Cortex

George A. Ojemann and Harry Whittaker
Seattle, Washington

The extent of lateral language cortex has been identified by the technique of stimulation mapping during a naming task, at craniotomy under local anesthesia for the treatment of medically intractible epilepsy in patients known to be left hemisphere dominant for language by intracarotid Amytal testing. Stimulation was at a constant current, below the threshold of after discharge, for each patient. Three questions, not previously addressed with this technique, were considered: 1) Does language cortex in an individual patient cover the classical language cortex derived from pooled data? 2) How much variability is there between patients in the extent of language cortex? 3) Are multiple languages located in the same cortex?

In 3 patients the extent of lateral language cortex was mapped with a large number of sites (23-28/patient). The area involved in language in each patient was larger than that classically described. These cases were pooled with another 5 patients where language cortex had been sampled at fewer sites. The variability in the extent of language cortex between all these patients was large. Only a small area of inferior frontal lobe just anterior to motor strip, apparently involved in the motoric aspects of language, was common to all patients in whom it was sampled. No area of temporal or parietal lobe was invariably part of language cortex in all patients! With this degree of variation, some lesions in left posterior temporal or parietal lobes may be resected without risk of aphasia, if the pattern of language localization is known for that individual patient. One patient was bilingual in English and Dutch. The two languages were not localized to the same areas of cortex: periSylvian cortex was common to both languages, but surrounding this, both frontally and parietally, were areas with differential representation of the two languages.

(Discussion)

11:20 a.m.

31. Experience With Ultrasonic Aspiration In Neurosurgery

**Eugene S. Flamm, Joseph Ransohoff
David Wuchinich and Alan Broadwin
New York, New York**

During the past two years, we have explored the possibility of using a new ultrasonic aspiration device for the removal of intracranial tumors. The instrument consists of a hand piece with a small bore tube that vibrates at rates sufficient to fragment tissue, which is then removed by the self-contained suction and irrigation system. The instrument has been used successfully in 24 cases to date, including 12 meningiomas, 4 acoustic neuromas, and 6 gliomas. With this instrument, tumor mass can be readily reduced in size to facilitate removal without the use of cutting current. In some cases, the ultrasonic aspirator was effective when other modalities of tissue removal were not successful. The advantages of the instrument are the reduced risk of damaging major blood vessels which tend to resist fragmentation by the instrument, the tactile feedback that the surgeon has which is not present when a cutting loop is used, and the absence of heating of surrounding tissue. The laboratory experience with this instrument, as well as the clinical applications, will be illustrated.

(Discussion)

11:40 a.m.

**32. ENHANCED VISUALIZATION OF THE CT SCAN WITH
ALTERNATE PLANAR VIEWS AND 3-D DISPLAY**

**Clark Watts
Columbia, Missouri**

This is a preliminary report on one of several means of improving the visualization of CT data obtained from transverse overlapping scans. Techniques for the rapid generation of coronal and sagittal planar views from the basic data have been developed. These additional planes can be visualized in a 3 dimensional presentation with or without the use of stereoscopic pairs. These techniques of alternative visualization have been helpful to the neurosurgeon and the radiation therapist in more clearly defining the location and size of mass lesions.

(Discussion)

12:00 noon

Write for copy

33. "The Effect of Left Stellate Ganglionectomy
Upon Certain Cardiac Arrhythmias

Wolff M. Krisch, Bruce Coyer, Ray Pryor,
Jay Law and Sol Penn
Denver, Colorado

There have been notable pharmacological advances in the management of life-threatening cardiac arrhythmias, primarily through the use of beta-blocking agents. A small but definite number of patients afflicted with unusual cardiac arrhythmias (recurrent ventricular tachycardia, "prolonged QT interval syndrome") have proven to be refractory to drug therapy or cardiac pacing procedures. This abstract reports our experience with left stellate ganglionectomy on two young men; one afflicted with the prolonged QT interval syndrome and the other with recurrent ventricular tachycardia. During the course of surgery on both of these patients, stimulation and recording of electrical activity from the left phrenic nerve, left stellate ganglia and sympathetic chain, and heart was obtained. A correlation of electrical activity in the left phrenic and sympathetic chain has been detected and will be described. The patient with the prolonged QT interval syndrome has had reversion of his QT interval to normal and has been totally free of syncopal attacks for a follow-up period of seven months. The second patient is now only one month post-operative, too short an interval to ascertain the effect of ganglionectomy on this disorder. A discussion of neurosurgical implications for interruption of left sided cardiac sympathetic innervation will be presented.

(Discussion)

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Montreal General Hospital
Montreal, Quebec, Canada

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Benson Hotel, Portland, Oregon	October 25-27, 1949
Mayo Clinic, Rochester, Minnesota	September 28-30, 1950
Shamrock Hotel, Houston, Texas	October 4-6, 1951
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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
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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
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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

1977

MEMBERSHIP LIST

AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
Founded October, 1938

Honorary Members	Elected
HUGO KRAYENBUHL Neurochirurgische University Kantonsspital 8000 Zurich, Switzerland	1974
GUY LAZORTES 26 Rue D Auriol 31 Toulouse, France	1973
VALENTINE LOGUE Naida Vale Hospital London, W. 9, England	1974
GOSTA NORLEN Nuerokirurgiska Kliniken Sahlgrenska Sjukhus Goteborg, SV Sweden	1973
SIXTO OBRADOR ALCALDE Eduardo Dato 23 Madrid 10, Spain	1973
KEIJI SANO Dept. of Neurosurgery School of Medicine University of Tokyo Tokyo, Japan	1975
R. EUSTACE SEMMES 920 Madison Avenue Memphis, Tennessee 38103	1955

Senior Members

GEORGE S. BAKER 200 First Street, S.W. Rochester, Minnesota 55901	1940
E. HARRY BOTTERELL Faculty of Medicine Queens University Kingston, Ontario, Canada	1938
HOWARD A. BROWN 2001 Union Street San Francisco, California 94123	1939
HARVEY CHENAULT 2370 Nicholasville Road Lexington, Kentucky 40503	1938
DONALD F. COBURN 4740 Roanoke Parkway Apartment 1201 Kansas City, Missouri 64112	1938
EDWARD W. DAVIS Providence Med Office Bldg. 545 N.E. 47th Avenue Portland, Oregon 97213	1949
FRANCIS A. ECHLIN 100 East 77th Street New York, New York, 10021	1944
DEAN H. ECHOLS 1550 Second Street New Orleans, Louisiana 70130	Founder
ARTHUR ELVIDGE Montreal Neurological Institute 3801 University Street Montreal 2, Quebec, Canada	1939

THEODORE C. ERICKSON 425 North Livingston Street Madison, Wisconsin 53703	1940
JOSEPH P. EVANS American College of Surgeons 55 East Erie Street Chicago, Illinois 60611	Founder
JOHN D. FRENCH The Center for the Health Sciences University of California Los Angeles, California 90024	1951
JAMES G. GALBRAITH 2515 Crest Road Birmingham, Alabama 35223	1947
EVERETT G. GRANTHAM 234 East Gray Street Louisville, Kentucky 40202	1942
JAMES GREENWOOD, JR. 1117 Hermann Professional Bldg 6410 Fannin Street Houston, Texas 77025	1952
WALLACE B. HAMBY 3001 N.E. 47th Court Fort Lauderdale, Florida 33308	1938
HANNIBAL HAMLIN 270 Benefit Street Providence, Rhode Island 02903	1941
JESS D. HERRMANN Post Office Box 135 Mountain Pine, Arkansas 71956	1948
WILLIAM S. KEITH Toronto Western Med Bldg Suite 207 25 Leonard Avenue Toronto, Ontario, Canada	Founder

GEORGE L. MALTBY 31 Bramhall Street Portland, Maine 04102	1942
AUGUSTUS McCRAVEY 1010 East Third Street Chattanooga, Tennessee 37403	1944
EDMUND J. MORRISSEY 450 Sutter St. Suite 1504 San Francisco, California 94108	1941
FRANCIS MURPHEY 20 S. Dudley St. Suite 101-B Memphis Tennessee 38103	Founder
J. LAWRENCE POOL Box 31 West Cornwell, Connecticut 06796	1940
ROBERT H. PUDENZ Box 79, Route 1 Vineyard Drive Paso Robles, California 93446	1943
R.C.L. ROBERTSON 2210 Maroneal Blvd. Shamrock Professional Bldg Suite 404 Houston, Texas 77025	1946
STUART N. ROWE 302 Iroquios Bldg. 3600 Forbes Street Pittsburgh, Pennsylvania 15213	1938
WILLIAM B. SCOVILLE 85 Jefferson Street Hartford, Connecticut 06106	1944
HENRY G. SCHWARTZ Barnes Hospital Division of Neurological Surgery St. Louis, Missouri 63110	1942

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Division of Neurological Surgery
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THOMAS A. WEAVER, JR. 1943
146 Wyoming Street
Dayton, Ohio 45409

BARNES WOODHALL 1941
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Durham, North Carolina 27706

Corresponding Members

JEAN BRIHAYE 1975
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Brussels, Belgium 5

KARL AUGUST BUSHE 1972
Neurochirurgischen Klinik
D-8700 Wurzburg
Josef-Schneider-Strasse 11
W. Germany

- FERNANDO CABIESES** 1966
 Inst Peruano De Fomento Educa
 Av Arenales 371, OF 501
 Apartado 5254
 Lima, Peru
- JUAN CARDENAS** 1966
 Av. Insurgentes Sur 594
 Mexico, D.F.
- JUAN C. CHRISTENSEN** 1970
 Ave. Quintana 474 80 A
 Buenos Aires, Argentina
- GIUSEPPE DALLE ORE** 1970
 Dipartimento Di Neurochirurgia
 Ospedale Maggiore 37100
 Verona, Italy
- HANS ERICH DIEMATH** 1970
 Prim. Univ. Doz.
 Neurochir. Abt. d. Landersner
 Salzburg, 5020, Austria
- JOHN GILLINGHAM** 1962
 Boraston House 22, Ravelson Dykes Rd.
 Edinburgh, Scotland EH43PB
- J.A. GOMEZ** 1975
 Cra 12, No. 20-29
 Bogota, D.E., Columbia
 South Carolina
- JOHN HANKINSON** 1973
 Department of Neurosurgery
 Newcastle General Hospital
 Newcastle-Upon-Tyne 4
 England
- SHOZO ISHII** 1975
 Department of Neurosurgery
 Juntendo Medical College
 Tokyo, Japan

- RICHARD JOHNSON** 1974
 Department of Neurological Surgery
 Royal Infirmary
 Manchester, England
- KATSUTOSHI KITAMURA** 1970
 University Kyushu Hospital
 Faculty of Medicine
 Fukuoka, Japan
- KRISTIAN KRISTIANSEN** 1962
 Oslo Kommune
 Ullval Sykehus
 Oslo, Norway
- LAURI LAITINEN** 1971
 Neurokirurgiska Kliniken
 Toolo Sjukhus
 Helsinki, Finland
- WALPOLE S. LEWIN** 1973
 Department of Neurosurgery
 Addenbrookes Hospital
 Hills Road
 Cambridge, England
- WILLIAM LUYENDIJK** 1973
 Pr Bernhardlaan 60
 Oegstgeest, Netherlands
- B. RAMAMURTHI** 1966
 2nd Main Road G.I.T. Colony
 Madras 4, India
- CHARAS SUWANWELA** 1972
 Chulalongkorn Hospital
 Medical School
 Bangkok, Thailand
- KJELD VAERNET** 1970
 Rigshospitalets Neurokirurgis
 Tagensvfj 18, 2200
 Copenhagen, Denmark

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

GAZI YASARGIL 1975
Neurochirurgische
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Kantonsspital
8000 Zurich, Switzerland

Active Members

EBEN ALEXANDER, JR. (BETTY) 1950
Bowman-Gray School of Med
Winston-Salem, N.C. 27103

JAMES R. ATKINSON (LONA) 1970
302 W. Thomas Road
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H. T. BALLANTINE, JR. (ELIZABETH) 1951
Massachusetts General Hosp.
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Montreal Neurological Inst.
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SHELLEY CHOU (JOLENE) 1974
University of Minn. Med. Ctr.
Minneapolis, Minnesota 55455

GALE G. CLARK U. of Cal. Med. Center San Francisco, Cal. 94143	(MARIAN)	1970
W. KEMP CLARK 5323 Harry Hines Blvd. Dallas, Texas 57235	(FERN)	1970
WILLIAM F. COLLINS, JR. Yale Univ. School of Med. 333 Cedar Street New Haven, Conn 06510	(GWEN)	1963
EDWARD S. CONNOLLY Ochsner Clinic New Orleans, La. 70118	(ELISE)	1973
JAMES W. CORRELL 710 West 168th Street New York, N.Y. 10034	(CYNTHIA)	1966
COURTLAND H. DAVIS, JR. Bowman-Gray School of Med. Winston-Salem, N.C. 27103	(MARILYN)	1967
RICHARD L. DeSAUSSURE 920 Madison Avenue, Suite 201N Memphis, Tenn. 38103	(PHYLLIS)	1962
DONALD F. DOHN 9500 Euclid Avenue Cleveland, Ohio 44106	(PATTY)	1968
R.M. PEARDON DONAGHY Mary Fletcher Hospital Burlington, Vermont 05401	(FRANCES)	1970
CHARLES G. DRAKE University Hospital 339 Windermere Road London Ontario, Can. N6G 2K3	(RUTH)	1958
STEWART B. DUNSKER Mayfield Neurological Inst. 506 Oak Street Cincinnati, Ohio 45219	(ELLEN)	1975

GEORGE EHNI 6410 Fannin Street Houston, Texas 77025	(VALAIRE "LARRY")	1964
WILLIAM H. FEINDEL Montreal Neurological Inst. 3801 University Street Montreal, Quebec, Canada	(FAITH)	1959
ROBERT G. FISHER Muhlenberg Hospital Plainfield, N.J. 07061	(CONSTANCE)	1957
ELDON L. FOLTZ Division of Neurosurgery Univ. of Cal. School of Med. Irvine, California 92664	(CATHERINE)	1960
RICHARD A.R. FRASER, M.D. 525 East 68th Street New York, New York 10021	(GAIL)	1976
LYLE A. FRENCH University of Minn. Med. Ctr. Minneapolis, Minn. 55455	(GENE)	1954
JOHN T. GARNER 744 Fairmount Avenue Pasadena, California 91105	(BARBARA)	1971
HENRY GARRETSON Dept. of Neurosurgery Health Sciences Center University of Louisville Louisville, Kentucky 40201	(MARIANNA)	1973
SIDNEY GOLDRING Barnes Hospital Plaza Division of Neurosurgery St. Louis, Missouri 63110	(LOIS)	1964
PHILIP D. GORDY 1727 East 2nd Street Casper, Wyoming 82601	(ELIZABETH ANN "LISA")	1968

JOHN R. GREEN Barrow Neurological Inst 302 West Thomas Street Phoenix, Arizona 85013	(GEORGIA)	1953
JOHN W. HANBERY Stanford Medical Center Division of Neurosurgery Palo Alto, California 49304	(SHIRLEY)	1959
MAJ. GEN. GEORGE G. HAYES MC USA, Principal Deputy Office of the Asst. Sec. of Defense Health & Envir Washington, D.C. 20301	(CATHERINE)	1962
E. BRUCE HENDRICK Hospital for Sick Children 555 University Avenue Toronto, Ontario, Canada	(GLORIA)	1968
JULIAN HOFF Dept. of Neurosurgery Univ. of Cal.-San Fran. San Francisco, Calif. 94143	(DIANE)	1975
EDGAR M. HOUSEPIAN 710 West 168th Street New York, New York 10032	(MARION)	1976
WILLIAM E. HUNT University Hospital 410 West 10th Avenue Columbus, Ohio 43210	(CHARLOTTE)	1970
DAVID KELLY Bowman-Gray School of Medicine Winston-Salem, N.C. 27103	(SALLY)	1975

ROBERT B. KING University Hospital Upstate Medical Center 750 East Adams Street Syracuse, N.Y. 13210	(MOLLY)	1958
WOLFF M. KIRSCH University of Colorado Medical Center 4200 East 9th Avenue Denver, Colorado 80262	(MARIE-CLAIRE)	1971
DAVID G. KLINE Louisiana St. Univ. Med Ctr. 1542 Tulane Avenue New Orleans, Louisiana 70012	(CAROL)	1972
ROBERT S. KNIGHTON Henry Ford Hospital 2799 W. Grand Blvd. Detroit, Michigan 48202	(LOUISE)	1966
THEODORE KURZE Los Angeles County-U.S.C. Medical Center 1200 North State Street Los Angeles, Calif. 90033	(EMMA)	1967
THOMAS W. LANGFITT Hospital of the Univ. of Penn. 34th and Spruce Streets Philadelphia, Penn 19104	(CAROLYN)	1971
RAEBURN C. LLEWELLYN Tulane University 1430 Tulane Avenue New Orleans, La. 70012	(CARMEN)	1963
WILLIAM M. LOUGHEED Medical Arts Bldg, Suite 430 170 St. George Street Toronto 5, Ontario, Canada	(GRACE ELEANOR)	1962

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Straub Clinic
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950 East 59th Street
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(BETTY) 1965

(CATHERINE "KATY") 1965

(BETSY) 1976

(ROBERTA) 1956

(JANET) 1972

(RUTH) 1973

(QUEENEE) Founder

(KATHERINE) 1955

(ALICE) 1952

(VIVIAN) 1963

BLAINE S. NASHOLD, JR. Duke University Med. Center Durham, North Carolina 27706	(IRENE)	1967
FRANK F. NULSEN Div. of Neurosurgery University Hospital 2065 Adelbert Road Cleveland, Ohio 44106	(GINNEY)	1956
GUY L. ODOM Duke University Med. Ctr. Durham, N.C. 27706	(MATALAINE)	1946
GEORGE OJEMANN University of Washington Dept. of Neurosurgery Seattle, Washington 98195	(LINDA)	1975
ROBERT G. OJEMANN Massachusetts Gen. Hosp. Div. of Neurological Surg. Boston, Mass. 02114	(JEAN)	1968
BURTON ONOFRIO Mayo Clinic Rochester, Minn. 55901	(JUDITH)	1975
RUSSEL H. PATTERSON, JR. 525 East 68th Street New York, New York 10021	(JULIE)	1971
PHANOR L. PEROT, JR. Medical University of South Carolina 171 Ashley Avenue Charleston, South Carolina 29403	(ELIZABETH)	1970
BYRON C. PEVEHOUSE 2001 Union Street San Francisco, Calif. 94101	(MAXINE)	1964
ROBERT W. PORTER 5901 East 7th Street Long Beach, Calif. 90804	(AUBREY DEAN)	1962

JOHN RAAF 833 S. W. 11th Avenue Portland, Oregon 97205	(LORENE)	Founder
AIDEN A. RANEY 125 North Las Palmas Los Angeles, California 90004	(MARY)	1946
JOSEPH RANSOHOFF New York Univ. Med. Center 560 First Avenue New York, New York 10016	(RITA)	1965
THEODORE B. RASMUSSEN Montreal Neurological Inst. 3801 University Street Montreal 2, Quebec, Canada	(CATHERINE)	1947
DAVID H. REYNOLDS Section of Neurosurgery University of South Florida, Box 16 12901 N. 30th St. Tampa, Florida 33612	(MARJORIE)	1964
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 20 South Dudley Street Memphis, Tennessee 38103	(VALERIA)	1971
RICHARD C. SCHNEIDER C5135 Out Pt. Building University Hospital Ann Arbor, Michigan 48104	(MADELEINE)	1970
JAMES C. SIMMONS 920 Madison Memphis, Tennessee 38103	(VANITA)	1975

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New England Medical Center Hospital
171 Harrison Avenue
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7703 Floyd Curl Drive
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THORALF M. SUNDT, JR.
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Pittsburg, Pa. 15213

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Mason Clinic
Seattle, Washington 98107

ARTHUR A. WARD, JR.
Department of Neurol. Surg.
Univ. of Washington Hosp.
Seattle, Washington 98105

(DOREEN)
1970

(JOANNE)
1972

(LOIS)
1971

(PHYLLIS)
1965

(MARY)
1950

(MARY)
1971

(SUSAN)
1973

(SUZIE)
1968

(VIRGINIA "GINA")
1967

(JANET)
1953

CLARK WATTS Univ. of Missouri-Columbia N522 Medical Center Columbia, Missouri 65201	(PATTY)	1975
W. KEASLEY WELCH Childrens Hosp. Med. Ctr. 300 Longwood Avenue Boston, Mass. 02115	(ELIZABETH)	1957
BENJAMIN B. WHITCOMB 85 Jefferson Street Hartford, Conn. 06106	(MARGARET)	1947
LOWELL E. WHITE, JR. Professor & Chairman Division of Neurosciences Mobile, Alabama 36688	(MARGIE)	1971
ROBERT WILKINS Professor and Chairman of Neurosurgery Duke University Medical Center Box 3807 Durham, North Carolina 27710	(GLORIA)	1973
CHARLES B. WILSON Dept. of Neurol. Surgery University of California Medical Hospital Third and Parnassus San Francisco, California 94143		1966
FRANK WRENN 123 Mallard Street Greenville, S.C. 29601	(BETTY)	1973
DAVID YASHON 410 W. 10th Ave. N. 911 Columbus, Ohio 43210	(MYRNA)	1972
NICHOLAS T. ZERVAS 330 Brookline Avenue Boston, Mass 02215	(THALIA)	1972

Deceased Members	Date	Elected
DR. PERCIVAL BAILEY Evanston, Illinois	(Honorary) 8/10/73	1960
DR. WILLIAM F. BESWICK Buffalo, New York	(Active) 5/12/71	1949
DR. SPENCER BRADEN Cleveland, Ohio	(Active) 7/20/69	Founder
DR. F. KEITH BRADFORD Houston, Texas	(Active) 4/15/71	1938
DR. WINCHELL McK. CRAIG Rochester, Minnesota	(Honorary) 2/12/60	1942
DR. WESLEY A. GUSTAFSON Jensen Beach, Florida	(Senior) 7/16/75	1942
DR. HENRY L. HEYL Hanover, New Hampshire	(Senior) 3/01/75	1951
DR. OLAN R. HYNDMAN Iowa City, Iowa	(Senior) 6/23/66	1942
MR. KENNETH G. JAMIESON Brisbane, Australia	(Corresponding) 1/28/76	1970
SIR GEOFFREY JEFFERSON Manchester, England	(Honorary) 3/22/61	1951
DR. DONALD D. MATSON Boston, Massachusetts	(Active) 5/10/69	1950
DR. KENNETH G. McKENZIE Toronto, Ontario, Canada	(Honorary) 2/11/64	1960
DR. JAMES M. MEREDITH Richmond, Virginia	(Active) 12/19/62	1946
DR. W. JASON MIXTER Woods Hole, Massachusetts	(Honorary) 3/16/58	1951

DR. WILDER PENFIELD Montreal, Canada	(Honorary) 4/05/76	1960
DR. RUPERT B. RANEY Los Angeles, California	(Active) 11/28/59	1939
DR. DAVID L. REEVES Santa Barbara, California	(Senior) 8/14/70	1939
DR. SAMUEL R. SNODGRASS Nashville, Indiana	(Senior) 8/08/75	1939
DR. C. WILLIAM STEWART Montreal, Quebec, Canada	(Corresponding)	1948
DR. GLEN SPURLING La Jolla, California	(Honorary) 2/07/68	1942
DR. HENDRIK SVIEN Rochester, Minnesota	(Active) 6/29/72	1957

**AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
1976 ANNUAL MEETING**

EVALUATION

Please complete this evaluation form (omit those sessions or events you did not attend) and return to the Secretary, Phanor Perot, 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 _____

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

SOCIAL PROGRAM

Comments _____

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

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

Please print Name: _____

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171 Ashley Avenue
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