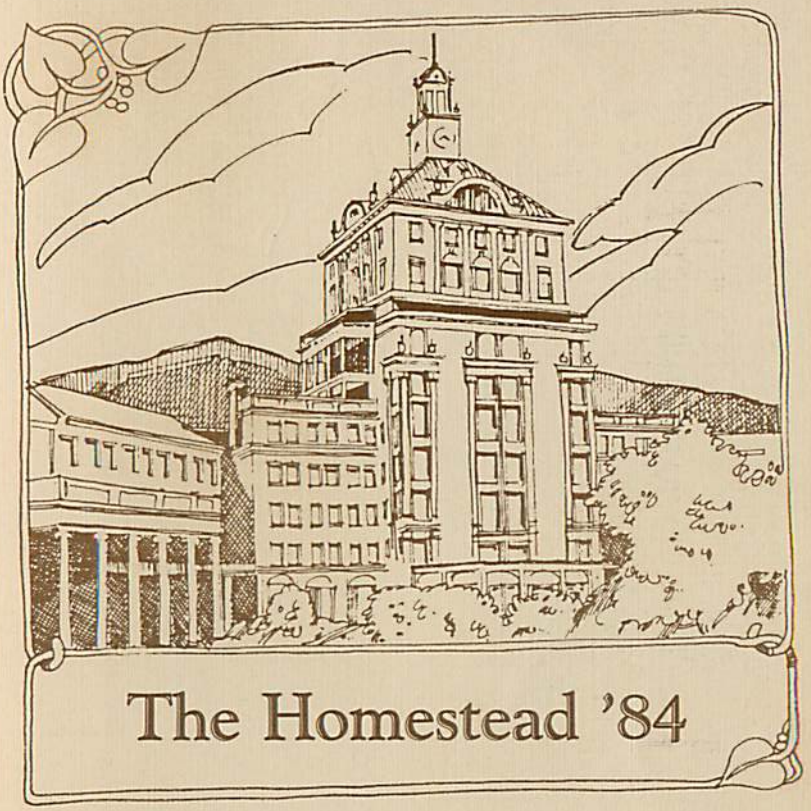

The American Academy of Neurological Surgery Program

Hot Springs, Virginia

ANNUAL MEETING OF THE
AMERICAN ACADEMY OF
NEUROLOGICAL SURGERY
1984

THE HOMESTEAD
HOT SPRINGS, VIRGINIA
OCTOBER 17-20, 1984

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THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

October 17 - 20, 1984
The Homestead
Hot Springs, Virginia

WEDNESDAY, OCTOBER 17

3:00 - 6:00 p.m.	Registration — Lobby Annex
4:30 - 5:30 p.m.	Tea Time — Hotel Lobby
6:00 - 7:30 p.m.	Reception — Crystal Room
7:30 - 8:30 p.m.	Dining — Main Dining Room (Coat and Tie dress)
	Late Dining — Grille Room

THURSDAY, OCTOBER 18

7:00 - 8:30 a.m.	Breakfast — Dining Room or Grille Room
8:00 - 10:00 a.m.	Registration — Lobby Annex
8:30 - 12:00 noon	Scientific Meeting - Commonwealth Room (with mid-morning coffee break)
12:00 noon	Group photograph — Outside or Commonwealth Room
12:00 - 1:30 p.m.	Lunch — Grille Room or Casino
1:30 - 4:30 p.m.	Scientific Meeting — Commonwealth Room (with mid-afternoon coffee break)
2:00 - 4:00 p.m.	Registration — Lobby Annex
4:30 - 5:30 p.m.	Business Meeting — Georgian Room (members only)
4:30 - 5:30 p.m.	Tea Time — Hotel Lobby

6:30 p.m.	Reception and Dinner – Ski Lodge (Casual dress)
10:30 a.m. - 4:30 p.m.	Ladies Hospitality – Valley Room

FRIDAY, OCTOBER 19

7:00 - 8:30 a.m.	Breakfast – Dining Room or Grille Room
8:00 - 10:00 a.m.	Registration – Lobby Annex
8:30 - 11:30	Scientific Meeting – Commonwealth Room
11:30 - noon	Business Meeting – Georgian Room (members only)
noon - 6:30 p.m.	Free afternoon
3:30 p.m.	Special Seminar for husbands and wives: Gems and Precious Stones (Dr. Joel Arem) – Georgian Room
4:30 - 5:30 p.m.	Tea Time – Hotel Lobby
9:00 a.m. - 4:30 p.m.	Ladies Hospitality – Valley Room
6:30 - 7:30 p.m.	Reception – Crystal Room
7:30 p.m.	Banquet – Commonwealth Room (Black Tie)

SATURDAY, OCTOBER 20

7:00 - 8:30 a.m.	Breakfast – Dining Room or Grille Room
8:30 - noon	Scientific Meeting – Commonwealth Room
9:00 - noon	Ladies Hospitality – Valley Room

NOTE: All breakfasts, the Wednesday dinner, and part of the charge for the Thursday and Friday dinners are included on the American Plan (modified) daily room charge.

LADIES ACTIVITIES

THURSDAY, OCTOBER 18

7:00 - 8:30 a.m.	Breakfast – Dining Room or Grille Room
9:30 - 10:30 a.m.	Homestead History – Tower Lounge
10:30 - 4:30 p.m.	Hospitality Room – Valley Room
2:00 - 3:30 p.m.	Nature Walk

FRIDAY, OCTOBER 19

7:00 - 8:30 a.m.	Breakfast – Dining Room or Grille Room
9:30 a.m.	The Antiquity of Jewelry – Empire Room
9:00 a.m. - 4:30 p.m.	Hospitality Room – Valley Room
3:30 p.m.	Gems and Precious Stones (Dr. Joel Arem) – Husbands and Wives – Georgian Room

SATURDAY, OCTOBER 20

9:00 a.m. - noon	Hospitality Room – Valley Room

SCIENTIFIC PROGRAM

Thusday, October 18

SCIENTIFIC SESSION I
MODERATOR - T.M. SUNDT, JR.

8:05 - 8:35 **SPECIAL LECTURE - MICHAEL THORNER "RECENT DEVELOPMENTS IN NEUROENDOCRINOLOGY AS RELATED TO HYPOTHALAMIC PITUITARY DISEASE"**

8:35 - 8:50

1. CLINICAL SILENT GROWTH HORMONE HYPERSECRETION IN PATIENTS WITH PITUITARY ADENOMAS

Nicholas T. Zervas, M.D. and Anne Klibanski, M.D.

Pituitary adenomas which hypersecrete growth hormone (GH) typically result in clinical acromegaly. GH measurements in acromegaly are critical for the diagnosis of these patients and serve as a marker of tumor growth and recurrance. We now report six patients thought to have prolactinomas or non-functioning pituitary tumors who were found to have hypersecretion of GH and/or abnormal GH dynamics without clinical features of acromegaly. Six patients between the ages of 26 and 37 years (media 32 years) were studied. The presenting symptoms were amenorrhea and/or galactorrhea in 5 patients and visual loss in 1. Four patients complained of mild to moderate fatigue. None of the patients had physical stigmata of acromegaly. All patients had macroadenomas with suprasellar extension and were treated with transsphenoidal adenomectomy. Two patients received conventional radiotherapy postoperatively. Immunocytochemical analysis was performed on the tumor tissue and positive staining for GH and prolactin (PRL) was found in all cases. GH concentrations were determined in the tumor tissue of 3 patients and ranged from 10,909 to 115,385 ng/mg protein. Three patients (Group I) had elevated and non-suppressible GH concentrations during oral glucose tolerance testing and 2 patients had elevated somatomedin-C concentrations of 7.9 and 2.6 U/ml., respectively. Three patients (Group II) had abnormal GH dynamics with marked GH elevations following thyrotropin-releasing-hormone administration and/or borderline GH suppression. All 6 patients had mildly elevated basal prolactin (PRL) levels from 18.5 to 50.2 ng/ml (normal < 15 ng/ml) with a median of 22. All patients had normal thyroid and adrenal function.

We have identified a group of patients with pituitary adenomas secreting GH in addition to PRL with: 1) marked hypersecretion of GH, 2) GH non-suppressibility and/or 3) abnormal GH dynamics

without clinical features of acromegaly. Symptoms of prolactin excess and fatigue were noted in most patients. GH levels in these patients represented an important tumor marker. We conclude that clinically silent GH excess may occur in patients with pituitary tumors. These patients represent a new subset of patients thought to have prolactinomas or non-functioning pituitary adenomas.

8:50 - 9:05

2. LONG-TERM FOLLOW-UP OF PROLACTINOMAS IN FEMALE PATIENTS TREATED BY TRANSSPHENOIDAL MICROSURGICAL RESECTION

G. Bertrand, M.D., G. Tolis, M.D., J. Montes, M.D. and Y. Zegeye

Seventy-nine female patients presenting with hyperprolactinaemia believed to be due to a prolactin secreting pituitary adenoma were operated upon by a transsphenoidal microsurgical technique at the Montreal Neurological Hospital between 1973 and December 1980.

(92%) presented with amenorrhea and, or galactorrhoea and only 3 patients (4%) had visual field defects. 54 patients (69%) were considered to have microadenomas by radiological criteria (Vezina & Sutton, 1974) and 16 grade II tumors (88% non-invasive tumors). Three had grade III and 1 grade IV tumors (12% invasive tumors). In 50 patients, prolactinaemia did not exceed 20 ng/ml. 18 patients ranged between 200 and 500 ng/ml, and 11 more than 500 ng/ml and up to 12,000 ng/ml.

Following surgery, 53 (67%), had serum prolactin levels reduced to normal at 20 ng/ml or less. In 4 patients, no discrete tumor was found. In the 46 patients with preoperative PRL levels of 2000 ng/ml or less and in whom a tumor was found, 40 had normal postoperative prolactin (87%). 6 additional patients had a postoperative PRL between 20 and 25 ng/ml.

At follow-up these 6 patients experienced a rapid recurrence of hyperprolactinaemia, indicating that a postoperative PRL level between 20 and 25 ng/ml probably indicates an incomplete removal of the tumor.

By contrast, the recurrance of hyperprolactinaemia was very infrequent in the 53 patients in whom prolactin was 20 ng/ml or less after surgery. So far, only 7 patients are known to have shown a recurrence in this group and only 1 with postoperative PRL less than 10 ng/ml.

Some patients have been lost to follow-up but 17 of the original 53, initially normalized, have been followed 5 years or more, serum PRL

has risen above 20 ng/ml in only 3 patients of this group, (18%). The rise was noted at 4 years in one patient and at 5 years in the two others. 12 patients are still normal after 6 years (mean 6.5 ± 0.45, up to 7.5) and no recurrence has been noted during that interval.

9:05 - 9:20

3. TRANSSPHENOIDAL SURGERY FOR NELSON'S SYNDROME

Lawrence H. Pitts, M.D. and Charles B. Wilson, M.D.

Until transsphenoidal removal of pituitary adenomas became routine over the past fifteen years, adrenalectomy often was performed for ACTH hypersecretion in Cushing's syndrome. Since more than two-thirds of patients with Cushing's syndrome harbor pituitary adenomas, adrenalectomy removed the source of cortisol elevation but did not prevent continued ACTH secretion from the pituitary tumor, thus sometimes producing Nelson's syndrome (ACTH hypersecretion from a pituitary adenoma in adrenalectomized patients) with its striking hyperpigmentation.

Since 1970, we have removed pituitary adenomas transsphenoidally in twenty-two patients with Nelson's syndrome. All patients were hyperpigmented and at least half had headaches and/or had a Cushingoid appearance. Half the women with Nelson's syndrome had menstrual irregularities. Cranial nerve palsies (III, V, VI) were present occasionally. Preoperative serum ACTH levels were 150-10,000 picograms/cc.

Surgery improved headaches and hyperpigmentation in half the patients, although preoperative weakness and easy fatiguability improved in almost all. As opposed to patients with Cushing's disease in whom ACTH levels fall to normal (below 100 pg/ml) after surgery, ACTH levels in Nelson's syndrome patients fell substantially after surgery but only occasionally were normal although clinical symptoms and signs improved more than the ACTH values.

We now have followed these patients 2-10 years since surgery and will present a clinical and endocrinologic summary of the late outcome of transsphenoidal removal of their ACTH secreting tumors.

9:20 - 9:30 Discussion

9:30 - 9:45 Coffee Break

9:45 - 10:00

4. ARTERIOVENOUS MALFORMATIONS OF THE CEREBRAL HEMISPHERES: A SURGICAL CLASSIFICATION

H.D. Garretson, M.D., Ph.D.

Currently available classifications of arteriovenous malformations (AVMs) have not given in our series a satisfactory feel for the actual surgical risk or technical difficulty involved in their surgical removal. There are five major elements of importance in determining the ease of malformation removal: (a) the relative involvement of the epicerebral, the transcerebral, and the subependymal and choroidal arterial supply in the arterial input to the malformations; (b) the number of proximal branches of the circle of Willis contributing to the malformations; (c) the location of the malformation with respect to areas of critical cortical function; (d) the presence of arterial feeders to the malformation measuring 3 mm or greater in size within 3 cm of the point of entry into the malformations; and (e) the age of the patient.

Using these factors, we developed a shorthand description of AVMs that reflects the relative technical difficulty and clinical gravity of the malformation, together with an associated surgical index.

A letter designation indicating the presence of epicerebral, transcerebral, and/or subependymal and choroidal arterial inflows followed by an Arabic number indicating the number of proximal branches of the circle of Willis with, finally, a Roman numeral indicating the decade of life of the patient, has proven to be a useful brief descriptor of the lesion when coupled with a modifier word (frontal, central, parietal, temporal, or occipital). Left is always presumed to be dominant unless specifically stated otherwise. A surgical index is also derived from simple addition of the numerical values of the classification ratings with a value of 1 assigned to the epicerebral, 2 to the transcerebral, and 3 to the choroidal/subependymal arterial circulations. The applications of these descriptors to our AVM series was reviewed. This is a useful and a very quickly applied descriptor of the lesions in our series with significant reflection of their relative surgical technical difficulty.

10:00 - 10:15

5. ARTERIOVENOUS MALFORMATIONS OF THE BRAIN IN CHILDREN

Robin P. Humphreys, M.D., E. Bruce Hendrick, M.D.
and Harold J. Hoffman, M.D.

Children can suffer ischemic or hemorrhagic cerebrovascular events, but on a basis somewhat different than those occurrences in adults. With the latter, a ruptured saccular aneurysm would be 6.5 times more likely than an arteriovenous malformation (AVM) to account for that hemorrhage. The ratio differs in children, where, in the experience to be reported from the Hospital for Sick Children, Toronto, AVM was three times more prevalent as the cause for the spontaneous subarachnoid/intraventricular hemorrhage than aneurysm. This report will review the 30 years experience with the treatment of traditional AVMs of the brain in 100 children 18 years of age and under.

Several features distinguish the child's AVM from that occurring in adults. A greater number (77%) of children first declare their malformation with hemorrhage, but a lesser number with chronic epilepsy (12%), as compared to adults. The mortality for the first hemorrhage is 22%, double that reported in adults. As with intracranial aneurysms, AVMs in children can be scattered throughout the brain, often in awkward locations. By contrast with adult experience, there is an unusual preponderance (22%) below the tentorium. The operative mortality is 8% and occurs exclusively in patients with subarachnoid hemorrhages Grades IV or V. Sixty-four percent of patients receiving operative care can be neurologically intact following surgery.

The authors conclude that it is inappropriate to downgrade experience with this neurosurgical condition in adults and presume that the clinical appearance and definition of the hemorrhagic stroke problem in children and their remarkable recuperative powers, distinguish the child who has suffered a hemorrhagic ictus.

10:15 - 10:30

6. SEIZURES IN PATIENTS WITH INTRACRANIAL VASCULAR MALFORMATIONS: RESPONSE TO SURGICAL TREATMENT

Edwin B. Boldrey, M.D. and Byron C. Pevehouse, M.D.

Since 1940, the authors have had at some time under their care 280 patients with intracranial vascular malformations. Of these, 142 patients received some form of surgical treatment. Total surgical removal of the malformation was achieved in 97 patients: 53 of these patients had had one or more preoperative seizures; 34 of the 53 had a history of preoperative intracranial hemorrhage. The response of seizures to excisive surgery is related in detail to factors including the location of the vascular malformation, the size of the malformation, the number of preoperative seizures, and the medical control of preoperative seizure. Control of seizures in the series, in general, exceeded 50%. The less satisfactory response without preoperative attacks who underwent surgery are discussed.

10:30 - 10:45 Discussion

10:45 - 11:00

7. ARTERIOVENOUS MALFORMATIONS WHICH FOLLOW THE TENTORIAL REGION

Bennet M. Stein, M.D.

From a series of over 200 AVM's the ones which were located around the incisura of the tentorium were selected for analysis. These comprised approximately 15% of the entire series. They were unique in the aspect that the surgical approach to these lesions required different maneuvers and not all of the incisural malformations could be approached through the same approach. The various approaches used were subtemporal, interhemispheric, and posterior fossa. The location of the malformation is stressed in determining which operative approach is used. The operative approaches will be detailed.

Although these malformations lay in obscure areas, the results of surgery including surgical morbidity and mortality were quite good.

11:00 - 11:15

8. **SPINAL AND CRANIAL AV MALFORMATIONS OF THE DURA**
Superselective Diagnosis and Surgical Treatment

E. Grote, K. Voigt and A. Thron

Clinical symptoms result from raised intracranial pressure and impaired venous drainage. The most frequent cerebral localization is around the transverse sinus, fed exclusively by branches of the external carotid. They immediately arterialized the sinuses, the homolateral one is very often occluded. Primary sinus thrombosis as etiological factor and reactive AV malformation is considered.

Radical excision of malformation, sinus and tentorium is mandatory for cure, superselective embolization effective only in dural spontaneous carotid cavernous sinus fistula.

Increasing neurological deficit in spinal manifestations require dural resections of one root entering zone and adjacent collaterals, venous dorsal dilated drains should be preserved. Surgical treatment seems to be more effective and safer than embolization.

11:15 - 11:30

9. **SURGICAL OCCLUSION OF ARTERIOVENOUS MALFORMATIONS OF THE LATERAL SINUS AND OF THE VEIN OF GALEN**

Sean Mullan, M.D.

Five lateral sinus arteriovenous malformations were eliminated by packing the sinus between occlusive balloons. One vein of Galen malformation was eliminated by internal packing. Two falx tentorial malformations which drained into the vein of Galen were eliminated by injection of glue into their feeding arteries. The techniques and problems will be discussed.

11:30 - 11:45 Discussion

SCIENTIFIC SESSION II
MODERATOR - RUSSEL PATTERSON

1:30 - 2:00 **ACADEMY AWARD PAPER**

2:00 - 2:15

10. ASPECTS OF ANEURYSM SURGERY IN THE LIGHT OF MODERN PHYSIOLOGICAL CONCEPTS

Professor Lindsay Symon TD FRCS

One of the main problems in the management of intracranial aneurysms with or without subarachnoid haemorrhage, has always been the handling of fragile high pressure sacs and their preparation for the application of suitable clips or ligatures. A variety of techniques have been employed to enable safe reduction of intravascular pressure to be achieved, ranging from total cardiac arrest under hypothermia combined with extracorporeal perfusion, to extreme hypotension. For many years the application of temporary vascular clips was thought unsatisfactory, but with the advent of microscopic surgery and more suitable clips, many have reverted to the use of a temporary vascular occlusion in the preparation particularly of large aneurysms for clipping or ligature.

Since 1978, temporary vascular occlusion has been used in 27 cases in a series of 36 giant aneurysms, and it is now routinely employed in difficult dissections in the middle or anterior cerebral circulation either when difficulty is expected, or when the aneurysm ruptures in the course of dissection.

A number of physiological observations are available to guide us in the applications of temporary vascular occlusion. The characteristics of intravascular pressure change in the primate circulation above the level of the circle of Willis are such that intravascular pressure is maintained at about 60% of normal with branch middle cerebral occlusions, at about 20% of normal with complete middle cerebral occlusion and at somewhat higher levels with terminal carotid artery occlusion. The main determinant of exsanguination of the peripheral circulation is the failure to use distal clips in association with proximal clips and excessive bleed-out of the collateral circulation. Work from our own and other laboratories has established that with a complete middle cerebral occlusion in the experimental primate, failure of the evoked response will occur when regional CBF falls below 20 ml/100g/min but that provided reperfusion is instituted promptly within 15 minutes the evoked response will reappear. The threshold for failure of the evoked response is appreciably higher than the established thresholds in similar experiments for movements of extracellular ions suggesting disruption of

membrane homeostasis, (around 10 ml/100g/min), and this last, ionic, threshold appears, when sustained, to be similar to the level at which infarction of the tissue is inevitable.

Over the last few years, monitoring of the somatosensory evoked response has been applied particularly to surgery of aneurysms in the terminal carotid, middle cerebral and basilar distribution as a monitor of induced ischaemia on the principle that the electrical threshold when crossed, will be a premonitor of impending ischaemia which may yet be recoverable on reperfusion.

Results of experience of such monitoring in a series of 70 aneurysm cases will be presented, with some correlations of evoked response change and neurological outcome.

2:15 - 2:30

11. HEPARIN REDUCES MORBIDITY AND MORTALITY IN SUBARACNOID HEMORRHAGE
Rationale and Results

John P. Kapp, M.D., Walter R. Neill, M.D.,
James V. Salter, B.S. and Thomas Y. Barnes, M.S.

Based on recent data concerning changes in cerebral arteries which follow subaracnoid hemorrhage, the cerebral vasospasm complex can be divided into three separate but overlapping phases: a muscular contraction phase; an arterial injury phase which is initiated by desquamation of endothelium followed by formation of platelet carpet, mural thrombi, and distal embolization; and a healing or proliferative phase which results in structural stenosis and loss of compliance of the cerebral artery. Herapin activates plasma antithrombin III, reduces the platelet aggregation which is stimulated by thrombin, neutralizes platelet factor IV, and inhibits the activity of platelet derived growth factor, a factor which may be important in the development of proliferative angiopathy. Herapin would therefore appear to be a logical treatment for phases 2 and 3 of the cerebral vasospasm complex. The records of 104 patients treated prophylactically with herapin during gradual carotid ligation following rupture of an intracranial aneurysm, 11 patients who received herapin after development of an ischemic neurological deficit during clamp closure, and 46 patients who did not receive herapin at any time have been reviewed. These patients were treated by a single neurosurgical group between 1966 and 1983, and have not been previously reported. The incidence of ischemic neurologic deficit, both permanent and transient, was 19.2% in the prophylactic herapin group compared with 38.6% in the control group. The incidence rate of recur-

rent intracranial hemorrhage was 9.6% in the herapin group vs. 26.1% in the control group, and the mortality was 9.6% in the herapin group vs. 30.4% in the control group. All differences were statistically significant at or below $p < .01$ by the Mantel-Haenszel statistical test.

2:30 - 2:45

12. DEPLETION OF CIRCULATING BLOOD VOLUME IN PATIENTS AFTER SUBARACNOID HEMORRHAGE: IMPLICATIONS FOR THE MANAGEMENT OF SYMPATOMATIC VASOSPASM

Robert A. Solomon, M.D., Kalmon D. Post, M.D.
and James G. McMurtry, M.D.

Recent efforts to treat ischemia associated with cerebral vasospasm have been directed towards augmentation of intravascular volume. This technique attempts to increase cerebral blood flow above critical ischemic levels by elevating cardiac output. The substantial benefits associated with this mode of treatment have spurred interest in the association between circulating blood volume and the development of cerebral ischemia after subarachnoid hemorrhage.

Maroon and Nelson in 1979 first measured blood volume directly in 15 nonselected patients after subaracnoid hemorrhage and documented a modest hypovolemia in these patients as a group (1). Kudo et al published a report of three cases where reduced blood volume accompanied cerebral ischemia related to vasospasm, the clinical improvement ensued with restoration of normal blood volume (2).

These studies and others suggest a correlation between hypovolemia and subarachnoid hemorrhage. However, the existing data do not adequately document the role of decreased blood volume in the genesis of ischemic deficits related to vasospasm. To address these issues further, we began to evaluate systematically in a prospective manner the blood volume of all patients admitted to the Neurological Institute of New York with the diagnosis of spontaneous subarachnoid hemorrhage.

We are presenting the results of 42 blood volume determinations made with autologous RBC's labeled with chromium-51. The subjects consisted of 11 controls and 25 patients with recent subarachnoid hemorrhage. The mean red blood cel volume (RBCV) and total blood volume (TBV) for female patients after SAH were significantly lower than corresponding control values ($p < .01$). No depression of blood volume was found in males as a group. 72% of females had had angiographic vasospasm; 8 of these patients were asymptomatic and 7

patients demonstrated signs of cerebral ischemia. Only 1 patient with asymptomatic vasospasm had below normal RBCV or TBV, whereas 6 of 7 patients with symptomatic vasospasm had subnormal RBCV and TBV. The mean RBCV and mean TBV for patients with symptomatic vasospasm were significantly lower than corresponding control values ($p < .02$) and lower than values for patients with asymptomatic vasospasm ($p < .05$) (see Figure 1).

The data suggest that volume status may be the important differential between asymptomatic and symptomatic vasospasm. Patients with normal blood volume are far less likely to experience cerebral ischemia, even if vasospasm develops. The blood volume data gathered during this study provide rational support for the use of volume expansion therapy in the treatment of patients with symptomatic vasospasm after SAH. Further clinical studies are needed to determine if prophylactic normalization of blood volume parameters on all patients after SAH is warranted.

2:45 - 3:00

13. CALCIUM CHANNELS IN CEREBRAL ARTERIOLAR SMOOTH MUSCLE CELLS

Gerald D. Silverberg, M.D.

Voltage-dependent calcium channels in rat pial arterioles were explored with intracellular microelectrodes. Voltage and current clamping were used to study the inward calcium current associated with depolarization. Two separate calcium channels were defined: 1) a long duration, low amplitude, low threshold current that increases linearly with depolarization, and 2) a high amplitude, rapidly inactivating current that appears only at high levels of depolarization. The long duration, low amplitude current seems responsible for constriction while the brief, high amplitude current appears to propagate the action potential. Two potassium currents are associated with the depolarization as well: a voltage-dependent membrane rectifier and a calcium-dependent potassium channel. These four currents are reflected in the muscle action potential.

Voltage-dependent calcium channels appear to be related to perivascular noradrenergic innervation. Where there are stainable noradrenergic nerves, voltage-dependent calcium channels are found. Where there are no nerves, calcium channels are not activated by depolarization. The voltage-dependent calcium channels are blocked by manganese, cobalt and the organic calcium channel blocking agents, such as verapamil and nifedipine.

3:00 - 3:30 Coffee Break

SCIENTIFIC SESSION III
MODERATOR - BURTON ONOFRIO

3:30 - 4:30 **SPECIAL SEMINAR ON CHYMOPAPAIN WITH OPEN ACADEMY DISCUSSION —CONTROVERSIES AND COMPLICATIONS—**
Panel Members: J. Robertson, C. Watts and D. Long

Friday, October 19

SCIENTIFIC SESSION IV
MODERATOR - S. MAHALEY

8:30 - 8:45

14. THE EVOLUTION OF SURGICAL CORRECTIVE PROCEDURES FOR UNILATERAL CORONAL SYNOSTOSIS: DESCRIPTION OF A NEW PROCEDURE

David D. McCullough, M.D.

Plagiocephaly due to unilateral stenosis of a coronal suture is one of the most deforming craniofacial anomalies observed in pediatric neurosurgical practice. The condition demands correction and surgery should be performed during infancy. The past three decades have witnessed an evolution of surgical procedures from simple synostectomy to unilateral canthal advancement and then to bilateral correction involving the skull base and frontal bone. The trend toward increasingly radical approaches appears to reflect the lack of satisfaction with procedures previously recommended in standard textbooks.

This presentation traces the development of surgical procedures and results culminating in the description of another modification of technique-unilateral floating canthal advancement. A philosophy related to the management of unilateral coronal synostosis will be outlined.

8:45 - 9:00

15. CRANIOFACIAL RESECTION OF TUMORS AT THE ANTERIOR SKULL BASE

Willis E. Brown, Jr., George A. Gates,
Ashwani Kapila and Jim L. Story

This paper reviews our experience since 1978 in the evaluation and management of seven patients whose tumors in the region of the anterior skull base required neurosurgical intervention to facilitate the resection. Three patients had juvenile angiofibromas, arising in the nasopharynx; two patients had an esthesioneuroblastoma, arising in the cribriform plate; two patients had carcinoma, arising in the paranasal sinuses.

Standard skull films and polytomography were useful in the evaluation of these patients, but computerized tomographic scanning offered the best depiction of the tumor's location and extent. Angiography was indispensable in locating the blood supply of the neoplasm, in demonstrating displacements of the carotid system, and in establishing the need for preoperative embolization of highly vascular tumors. This adjunctive measure dramatically reduces the transfusion requirements in patients with juvenile angiofibromas; forty-eight hours appears to be the optimum time for preoperative embolization. The techniques we have employed (antibiotic coverage, elective tracheostomy, rotation of vital dural grafts, and careful reconstruction of surgical defects with vital tissues) have been successful in preventing intracranial infection. The combined neurosurgical and otorhinolaryngological approach to these tumors has been very effective in the management of these patients.

Finally, we will present our initial experience with a new technique —the transfacial approach to the pterygopalatine fossa, in which a skin incision is made in the preauricular and submandibular region (to turn a skin flap based at the midline of the face) and the facial and masticatory muscles are reflected from the facial midline laterally (to maintain their innervation). This approach provides direct access to the anterior wall of the middle cranial fossa, the superior orbital fissure, the foramen rotundum, and the foramen ovale, and the region of the pterygoid plates.

9:00 - 9:10 Discussion

9:10 - 9:25

16. USE OF INTRAOPERATIVE AUDITORY EVOKED POTENTIALS TO PRESERVE HEARING IN UNILATERAL ACOUSTIC NEUROMA REMOVAL

Robert G. Ojemann, M.D., Robert A. Levine, M.D. and
William M. Montgomery, M.D.

Thirty-four patients with unilateral acoustic neuromas and preoperative speech discrimination scores of 35% or more had intraoperative monitoring of the electrocochleogram (ECoG) using a transtympanic electrode, and of the brainstem auditory evoked potentials (BAEPs) using scalp electrodes. Rapid feedback was provided about the status of the cochlear microphonics from the hair cells of the inner ear (CM of the ECoG), the compound action potential of the auditory nerve (N-1 of the ECoG or Wave I of the BAEPs) and the potentials from the lower brainstem (Wave V of the BAEPs). In 31, the cochlear nerve was anatomically preserved, and 31 had good postoperative facial nerve function. Correlation of tumor size with postoperative hearing was as follows: Discrimination scores of more than 35% in five of seven patients with 1 cm tumors, six of fourteen with 1.5 cm tumors, two of nine with 2 to 2.5 cm tumors, and one of four with tumors of 3 cm or more; three other patients had discrimination scores of less than 35%. In two patients, the discrimination scores improved. At the end of the operation, all patients with hearing had a detactable N-1, and, when recorded, CM. All but one patient with no hearing had lost N-1, and CM was absent or reduced. Unless Wave V was unchanged, it was a poor predictor of postoperative hearing, and its absence did not preclude preservation of good hearing.

The electrophysiological changes during each stage of the operation were analyzed and correlated with events during surgery. Areas in which there was an increased risk of loss of the potentials were determined. In some patients monitoring was unnecessary, because no recovery occurred. However, in other patients monitoring alerted the surgeon to a possible problem and the method of dissection was altered. Possible mechanisms of hearing loss were suggested from the changes in the recordings.

9:25 - 9:40

17. **RETROLABRYNTHINE TRANSECTION OF THE VESTIBULAR NERVE FOR INTRACTABLE VERTIGO**

Julian T. Hoff, M.D.
John L. Kemink, M.D.

Forty patients with intractable vertigo localized to one vestibular apparatus have undergone vestibular nerve transection by the retrolabrynthine approach in the past two years. Thirty-six have had relief of their symptons while four have remained vertiginous. None were made deaf through mild sensorineural hearing loss was documented in three after operation. Hearing improved in three patients with Meniere's disease after operation. There were no facial nerve complications and no deaths. A wound infection developed in three patients and three developed CSF leak.

Videotape records were made in twenty-one patients to assess the role of transneural compression of the vestibular nerve by vascular structures. Two of the twenty-one had large veins overlying the nerve and two had separation of the facial and cochleovestibular nerves by large arterial structures. None had arterial compression accounting for symptoms. Videotape examples of the transection procedure by this approach will be shown.

9:40 - 9:50 Discussion

9:50 - 10:15 Coffee Break

10:15 - 10:30

18. **CT ADAPTOR FOR FUNCTIONAL STEREOTACTIC NEUROSURGERY**

Lauri V. Laitinen, M.D., Ph.D.
Bengt Liliequist, M.D., Ph.D.
Markku Fagerlund, M.D.
Tommy A. Eriksson, M.D.

Until present, CT guided stereotaxis has been used for morphological neurosurgery (biopsy and isotope therapy of brain tumors) and only exceptionally for implantation of chronic depth electrodes. The instruments have often been very expensive.

We have designed an inexpensive and versatile adapter for CT guided stereotaxis. In addition to morphological neurosurgery it also permits ablative stereotactic surgery such as thalamotomy with a high degree of accuracy.

The adapter consists of an aluminium frame which can be fixed to the patient's head by means of two ear plus and a nasal support. The brain target is related to the adapter with rectilinear x, y and z coordinates from the CT pictures. The coordinates can any suitable time be transferred to a stereotactic instrument. The adapter is fitted to most stereotactic guides and can be used in all adult patients. For small children a smaller nasal support part is needed. The adapter is as such suitable for external stereotactic irradiation of brain targets with a conventional linear accelerator.

Comparison between CT and ventriculography guided determinations of thalamic targets showed a mean difference of 0.6 and 0.7 mm for the x and y coordinates, respectively. The z coordinates seldom showed any measureable difference. Therefore, we now perform all types of functional stereotaxis (implantation of depth electrodes, thalamotomy, hypothalamotomy, dentatotomy, cingulotomy, etc.) with CT guidance only. The short-term results have been at least as good as after previous ventriculography guided surgery.

10:30 - 10:45

19. THE USE OF THE ON/OFF (PORTNOY REVERSIBLE OCCLUSION VALVES IN SHUNTS FOR HYDROCEPHALUS AND OTHER CEREBRAL PROBLEMS)

Eben Alexander, Jr., M.D. and J.M. Whorter, M.D.

The on/off device, either in association with a Portnoy valve itself, or in association usually with a Holter Valve, has been used over thirty times in our institution in the last ten years. It has been used in certain instances in which it is felt that the shunt may not always be necessary, and in place of removing it, the shunt can be occluded to test the need for it. In those patients who are at risk for development of subdural hematoma, such as low pressure hydrocephalus in adults; it has been particularly useful since it can be closed, a drill hole made, the subdural evacuated and the brain allowed to expand, following which the valve can be reopened.

In certain children with large subdural hematomas, with small ventricules, the on/off device can be closed, the ventricles dilated, the subdural evacuated and obliterated.

In one patient with a cholesteatoma, with cholesterol intraventricularly, it has been used as a shunt of hydrocephalus and at the same time to occlude the valve until the cholesterol can be aspirated through a Rickham reservoir.

An account of the successes, failures and potential usefulness of this device will be given with the appropriate illustrations.

10:45 - 11:00 Discussion

11:00 - 11:30 **PRESIDENTIAL ADDRESS** Introduction
Russel Patterson James Correll

Saturday, October 20, 1984

SCIENTIFIC SESSION V

MODERATOR - B. STEIN

8:30 - 8:45

20. **SINGLE STAGE MENINGOMYELOCELE REPAIR AND SHUNT**

W.O. Bell
E.A. Arbit
R.A.R. Fraser

Thirty-seven consecutive neonates who underwent surgical repair of meningomyeloceles (M-M) and ventricular shunting shortly after birth were reviewed. An analysis of the first 13 patients in the series and their computed tomography (CT) scans revealed that head circumference was a poor indicator for the presence of ventriculomegaly. All 13 neonates had ventriculomegaly on CT scan despite head circumferences that were below the 90th percentile, and all eventually required ventriculoperitoneal (V-P) shunting after M-M repair. These observations suggested that ventriculomegaly on initial CT scan has predictive value for the development of overt hydrocephalus.

The remaining 24 neonates were managed by a treatment protocol consisting of preoperative CT scan and then repair of the M-M with simultaneous placement of a V-P shunt if ventriculomegaly was present on CT scan. If ventricular enlargement was not present, ventricular size was followed by either CT scan or real-time

ultrasonography and V-P shunt placed for evolving hydrocephalus when present.

Using this management protocol, 18 neonates underwent a one stage operation of M-M repair and V-P shunt placement. The remaining 6 patients had normal sized ventricles on initial CT and only M-M repair was performed. Of these, 4 neonates evenutally required V-P shunting for control of hydrocephalus. In total 34 of 37 neonates (92%) required V-P shunting. Three patients required biventricular shunts. Complications were 5 in number consisting of malfunctioning shunt hardware necessitating shunt revision. Hospital stay averaged 21 days.

Simultaneous M-M repair and V-P shunting treats hydrocephalus earlier and reduces wound and shunt complications secondary to cerebrospinal fluid accumulation under the M-M wound.

8:45 - 9:00

21. RECURRENT SYMPTOMATIC LESIONS AT THE SITE OF PREVIOUS CAROTID ENDARTERECTOMY

J.W. Correll
C. Loftus
T.K. Tatemichi
F.T. Nichols
W.J. Kupsky
J.P. Mohr

Since 1974, 51 operations have been carried out on 43 patients to remove a recurrent lesion at the site of previous carotid endarterectomy. Each patient had had a recurrence of ischemic symptoms felt to be due to the lesion. The shortest interval between the initial and the 2nd operation was 6 months, while the longest was 15.5 years; the average being 4.9 years. In 4 patients the initial operations had been carried out elsewhere; the remainder had been in a series of 1866 consecutive endarterectomies, making the incidence of recurrent symptomatic carotid lesions 2.5%. The recurrent lesions have been found to be predominantly 1 of 3 types: mural thrombus in various stages of organization, fibroendothelial hyperplasia or atheromatous deposition usually associated with mural thrombus. Factors of possible importance in the pathogenesis of these lesions will be discussed.

9:00 - 9:15

22. ANTERIOR CEREBRAL ARTERY MICROVASCULAR RECONSTRUCTION

James I. Ausman, M.D., Ph.D.
F.G. Diaz, M.D., Ph.D.
M. Dujovny, M.D.
A. Yokoh, M.D.
J. Cabezudo, M.D.
F. Gomez, M.D.

Microsurgical dissection of 20 unfixed human brains was performed through the bifrontal interhemispheric approach or the bifrontal approach. Both main trunks of the anterior cerebral artery (ACA) and its branches were identified and the diameter and length of each of them were recorded. The mobility of the arteries and the number of perforating branches were also measured.

End-to-end, end-to-side, side-to-side anastomoses as well as autografting procedures were performed to evaluate different potential reconstructive procedures for the anterior cerebral system.

The proximal half of the A1 segments was difficult to reach with the interhemispheric approach because significant brain retraction was necessary, however, the bifrontal approach made this area accessible. For other portions of the ACA, the usual interhemispheric fissure aperture with ordinary degrees of retracation, permitted these procedures to be performed. Reconstruction was accomplished between the A1 and A2 segments, recurrent artery of Heubner, fronto-orbital and frontopolar with end-to-end or end-to-side anastomosis to the A1 segment. Side-to-side anastomosis between both A2 segments were completed without difficulty. The anatomical and experimental reconstruction data were used to perform two surgical reconstructive procedures for large or giant aneurysms in this area. An A1 to A2 end-to-end anastomosis and an A2 end-to-side anastomosis have been performed with no technical difficulties or complications.

The feasibility of these microvascular reconstructive procedures involving the anterior cerebral artery in the antero-basal part of the interhemisphere, where cerebral aneurysms frequently develop has been demonstrated. Furthermore, for corrective surgery in cases of focal occlusive disease or tumors, vascular reconstruction or reimplantation can be accomplished using these procedures.

9:15 - 9:30 Discussion

9:30 - 10:00 Coffee Break

10:00- - 10:15

23. **BASAL ARTERIAL OCCLUSIVE DISEASE OBSERVATIONS**

D.O. Quest
J.W. Correll

Seventeen patients with basal occlusive disease have been seen over the past four years. Nine of these have had a classic Moya-Moya appearance on angiography. Of these nine, seven were adults and two were children. Eight had ischemic episodes and one had a hemorrhage. One had neurofibromatosis with a chiasmal glioma and had received radiotherapy. Eight patients underwent STA-MCA bypass and one refused surgery. Six patients improved, one had a stroke on the opposite side, and one died.

Eight patients had unilateral basal arterial occlusive disease. Of these, three had ischemic episodes, four had hemorrhages, and one had only headaches. Four were adults and four were children. Five underwent STA-MCA bypass with improvement, one with headaches had an aneurysm treated, and two were not operated upon. Considerations regarding etiology, therapeutic options, and disease course in these patients will be discussed.

10:15 - 10:30

24. **RECOVERY OF FUNCTION FOCAL BRAIN INJURY**

Howard M. Eisenberg, M.D.
Andrew C. Papanicolaou, Ph.D.
Georg Deutch
Harvey S. Levin, Ph.D.

In 1975, Jenett and Bond predicted that the major determinant of long-term outcome after head injury would be the extent of recovery of cognitive and behavioral deficits rather than the presistence of motor or cranial nerve abnormalities. This idea is supported by our studies of outcome, and we have become particularly interested in recovery of cognitive deficits attributable to focal lesions. When we grouped our patients on the basis of the presence and localization of focal injuries by CT scan, we found that patients who had predominantly left temporal lobe injuries generally recovered from verbal memory deficits more completely than did other patients classified as having diffuse injury (Dissociation of verbal and visual memory has been demonstrated in

patients after temporal lobectomy). The reason for this preferential recovery was not clear, but we considered shift in hemispheric dominance a possible mechanism. To test this hypothesis, we developed an evoked potential paradigm that enabled us to assess lateralization of verbal as well as non-veral cognitive operations. This paradigm was first validated in normal subjects. When we tested patients with CT demonstrable left temporal lobe lesions who had impairment of language and memory and who had recovered, we found that verbal encoding was localized in the right hemisphere; all were adults and none had been left handed. We interpreted these findings as evidence that hemispheric reorganization contributes to the recovery of language and of verbal memory after focal injury, even in adults.

Because this evoked potential paradigm lacks regional specificity, we have developed methods of assessing intra as well as inter-hemispheric reorganization during recovery, using measurments of regional cerebral blood flow. Further, our studies of cognitive recovery are also being extended to assess the effect of focal lesions visualized by NMR scanning. Recently, we found discrete lesions that were not seen in CT scans. These NMR lesions were present even years after injury.

10:30 - 10:45

25. **THE CONTRIBUTION OF ORNITHINE DECARBOXYLASE ACTIVITY TO THE PATHOPHYSIOLOGY OF CEREBRAL ISCHEMIA**

Robert J. Dempsey, M.D.
Mark W. Roy, Ph.D.
Kathleen Meyer, M.D.
Jack W. Olson, Ph.D.

The process in which focal cerebral ischemia proceeds rapidly to infarction remains poorly understood. Cerebral tissue's exquisite sensitivity to ischemia as compared to other organs has been investigated at the molecular level for the relative contribution of prostaglandins, leukotrienes, opiates, kinins, and hyperglycemia in the pathophysiology of stroke without developing a comprehensive understanding or a unifying factor.

Polyamines are a group of molecules derived from ornithine by the acton of the rate limiting enzyme, ornithine decarboxylase (ODC). Previously studied by cell biologists for their role in cell differentiation, there is suggestive evidence that they could contribute to the development of cerebral infarction after ischemia. They are found in all

eukaryotic cells examined, are stimulated by ischemia, and are implicated in the development of vasogenic edema, the stimulus-response coupling of Ca++ dependent endocytosis (allowing early rapid increase in cellular water content - cytotoxic edema) and also inhibit platelet phospholipase C (needed for later prostacyclin production).

24 cats were studied to determine if tissue ODC activity could be shown to correlate with regional levels of ischemia or edema after a focal cerebral ischemic insult. 8 cats underwent R MCA occlusion under general anesthesia by occlusion of a prolene sling placed transorbitally 1 week prior. These were monitored for rCBF by H2 electrode technique at 4 sites (R temporal, R parietal, R occipital, L parietal). Nine flows were measured at each site over a period 4 hours prior and 6 hours post-occlusion. Each underwent craniectomy at the 4 above sites. Tissue was rapidly removed and frozen for radioimmunoassay of ODC activity. A separate sample of grey matter was studied for enema formation (water content) using a variable density kerosene-bromobenzene liquid gradient column.

Regional CBF was anatomically distributed at 0 to 6 hr post-occlusion. The R temporal lobe which was found to be infarcted was the area of densest ischemia (15.9 ± 1.6 cc/100gm/min). A surrounding penumbra of partial ischemia (R parietal 18.5 ± 1.5 cc/100gm/min) and decreased flow (R occipital 27.0 ± 2.4 cc/100gm/min) contrasted with the nonischemic L parietal region (56.2 ± 2.1 cc/100gm/min).

Grey matter edema, % water, was greatest at the R temporal site (dense ischemia) and increased with time (3 hr edema $83.5 \pm 0.4\%$ vs 6 hr $85.2 \pm 0.5\%$; significantly greater than control site $p < 0.001$). The R parietal site (partial ischemia) showed intermediate edema (3 hr $82.8 \pm 0.7\%$ vs 6 hr $82.7 \pm 0.8\%$; significantly greater than control $p < 0.05$). The R occipital site (decreased flow) showed less edema (3 hr $80.3 \pm 1.2\%$ vs 6 hr $81.3 \pm 1\%$) similar to the control L parietal site (nonischemic) (3 hr $80.7 \pm 0.9\%$ vs 6 hr $80.9 \pm 0.3\%$).

Ornithine decarboxylase (ODC) was increased with time after ischemia and was marked elevated in penumbra or marginally ischemic regions. In densely ischemic R temporal, ODC was 3 hr 1289 ± 392 vs 6 hr 1637.6 ± 376 pmole/gm/hr. In the penumbra region, R parietal, ODC was 3 hr 1492.5 ± 265 vs 6 hr 3812 ± 1049 and R occipital 3 hr 2713 ± 457 vs 6 hr 3041 ± 511 pmole/gm/hr. Nonischemic L parietal ODC activity was 3 hr 1935 ± 181 vs 6 hr 2509 ± 373 pmole/gm/hr.

ODC activity at 6 hr post-MCA occlusion, therefore, reflects the distribution of ishemic but not infarcted tissue. Densely ischemic brain is lowest while marginally perfused brain is highest in ODC acvitity. Increasing ODC activity over time if found in the area of brain in which infarction is most likely to proceed. ODC activity at 6 hr correlated with regional edema outside of the densely ischemic region. This suggest that

polyamines, the end product regulated by ODC activity, are present in ischemic brain, co-distributed with ischemia and ischemic edema, and are a potentially important contributor to the pathophysiology of infarct and endema development in marginally survivable areas of brain after a focal ischemic lesion. Further studies with selective blocking of ODC may show benefit in increasing cerebral tolerance to partial ischemia.

10:45 - 11:00 Discussion

11:00 - 11:15

26. NEUROLOGIC MANIFESTATIONS OF THE ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS)

Mark L. Rosenblum, M.D.
Dale E. Bredesen, M.D.
Robert M. Levy, M.D.

We have evaluated 318 homosexual patients with AIDS or generalized lymphadenopathy (LAN) treated at the University of California, San Francisco over the 5-year period from 1979 to 1984. 124 of these 318 patients (39%) were neurologically symptomatic. Of these patients, approximately one-third presented with their neurologic complaints. Thus, 10% of all AIDS patients present with problems relating to the nervous system.

Central nervous system complications were encountered in 97 patients with AIDS or LAN and included the following viral syndromes: subacute encephalitis (31 patients), atypical aseptic meningitis (17), herpes simplex encephalitis (8), progressive multifocal leukoencephalopathy (2), viral myelitis (2) and varicella Zoster encephalitis (1). Non-viral infections in this population were found to be caused by toxoplasma gondii (11), cryptococcus neoformans (8), candida albicans (2), coccidioidomycosis (1) and treponema pallidum (1). Neoplasms seen in these patients included primary CNS lymphoma (6), systemic lymphoma with CNS involvement (2) and metastatic Kaposi's sarcoma (2). Cerebrovascular complications were seen in one patient with hemorrhage and in one patient with infarction due to non-bacterial thrombotic endocarditis.

Cranial or pereipheral nerve complications were encountered in 33 patients with AIDS or LAN. Cranial nerve syndromes included multiple cranial neuropathies secondary to chronic inflammatory polyneuropahty (5) and Bell's palsy (3). Peripheral nerve syndromes included chronic inflammatory polyneuropathy (12), distal symmetrical neuropathy (5), herpes zoster radiculitis (6) and persistent myalgias (2).

The unusual and varied nervous system diseases that afflict patients with AIDS and LAN should prompt an aggressive workup in any patient who suffers from neurological symptoms. The protean behavior of the disease and imprecise radiographic and serological studies suggest that biopsies of CNS space-occupying lesions be performed for tissue diagnosis before therapy is begun.

11:15 - 11:30

27. DEVELOPMENTAL CERVICAL STENOSIS WITH NEUROLOGICAL COMPLICATIONS

Carole A. Miller, M.D. and William E. Hunt, M.D.

The significance of the congenitally narrow cervical canal without spondylosis has not been fully appreciated. While canal narrowing due to acquired cervical osteoarthritis was recognized as early as 1911, the role of the developmentally narrow canal in myelopathy was not fully appreciated until 1966. It is now generally agreed that most patients with cervical spondylosis who develop cervical myelopathy also have a congenitally narrow canal system.

In the past eight years, we have seen 16 patients with a narrow cervical canal and spinal cord and/or nerve root symptomatology, without osteoarthropathy. Ages ranged from 16 to 63. Seven suffered cord injuries from hyperextension injuries. Two had flexion and rotation injuries. Five were thought to have obscure degenerative diseases of their spinal cord, one with a diagnosis of multiple sclerosis, two had recurrent stiff necks with Lhermitte's sign and fasciculations in the shoulder girdle and upper extremities.

There is a typical radiographic, CT scan appearance and myelograhic appearance in these patients. In fact, the lateral cervical spine films taken in the emergency room can usually alert the clinician to the diagnosis. CT scanning is very helpful because it demonstrates well the flattened laminae, the short pedicles and distortion of the spinal cord. Myelography may be dangerous in these patients because hyperextension of the neck may cause futher damage to the spinal cord.

The congenitally narrow canal is straight with flat laminae and short pedicles. The cortical line at the base of the spinous processes is superimposed on the line of the facets. There is straightening of the cervical spine, failure of funnelling of the upper cervical canal and often a congenital abnormality of C1.

The incidence of this condition in the general population is not known, but certainly some people are at hazard, particularly from hyperextension injuries. Coaches should particularly be alerted to the

youngster with a long slim neck without much muscular development and the possibility of the injuries associated with contact sports. Our cases also indicate that the symptoms may develop spontaneously.

Surgical procedures are adapted to the particular patient and will be discussed. We do not, as a rule, recommend prophylactic decompression.

11:30 - 11:45

28. DOES DENERVATION CAUSE "BURSTING" FIRING PATTERNS IN THE DORSAL HORN?

Charles J. Hodge, M.D.
A.V. Apkarian, M.S.
A. Fraser, M.D.
S. Martini, M.D.

Central to the current concepts of denervation pain is the hypothesis that removal of afferent input results in periodic hyperactivity or "bursting" of the denervated neurons. This study was designed to test this hypothesis. The first part of the study involved converting the qualitative definition of bursting, i.e. periodic high frequency activity, into a workable quantitative definition. Bursting activity was considered to occur when the actual firing pattern of a cell showed significant clustering of high frequency activity. This was determined by comparing a unit's actual distribution of shorter interspike intervals to the distribution that was predicted on the basis of a random (i.e. not clustered) distribution of such intervals.

Eleven cats, five of which were controls and six of which had unilateral extensive lumbar dorsal rhizotomies one month prior to recording, were used. Single unit dorsal horn cell recordings were made of the spontaneous activity of dorsal horn cells in both groups of animals (87 control cells, 47 cells from the dennervated animals). The laminar locations of the recording sites were determined for all units included in the study. Cells were excluded if they were located deeper than the dorsal horn (laminae 1-7) or if the recording sites were indeterminant.

Results of the analysis of the firing patterns of these cells indicates: 1) Rhizotomy results in a higher ratio of bursting to non-bursting cells than is seen in the control animals, 2) More cells from the denervated animals, when compared to the control group, had high mean frequencies, and 3) The presence of a bursting type activity was not related to the laminar location of the cells.

We conclude that denervation results in bursting firing patterns of

the appropriate cells. Whether this is the cause of denervation dysesthesiae and pain is unclear since none of the treated animals displayed overt behavior indicating any kind of discomfort. The method of defining bursting, however, will be useful in quantitatively assessing the effects of a variety of drugs on such unit activity, in determining if bursting activity occurs in humans, and determining the effects of both denervation and dorsal root entry zone lesions on more rostral sites such as the reticular core and the sensory thalamic nuclei.

11:45 - 12:00

29. **PARTIAL CHARACTERIZATION OF POSTTRANSLATIONAL AMINOACYLATION OF PROTEINS IN GUINEA PIG BRAIN**

David J. Gower, M.D.

David Kelly MD

The mechanism of axoplasmic transport is a highly ordered process rather than a diffusion of material down a concentration gradient. This structural organization in the axon requires proteins to be modified at the synapse so they may assume their final role in the synaptoplasm or the synaptic membrane. Several systems of macromolecule modification involving tubulin and the intermediate filaments are well described. In addition to the proteins undergoing axonal transport a relatively large quantity of transfer RNA is also moved by slow component B (scB) to the synapse. A 20 fold increase in tRNA content during a regenerative effort by the goldfish optic nerve is also observed. The role of this transfer RNA (tRNA) was unclear, since no ribosomes or other mechanisms of protein synthesis were concomitantly transported.

Work with squid axoplasm has recently shown that this tRNA may be acting to modify axonal macromolecules in an aminoacylation reaction, or the tRNA may hold a free amino acid so that it may be added to a protein chain. This manuscript demonstrates a similar postranslational modification of macromolecules in the high speed supernate of the guinea pig brain. The reaction is shown to be tRNA dependent, exhibits feedback inhibition and amino acid and acceptor protein specificity.

Changes in the content of transported tRNA during regeneration imply that this work may represent the first step in understanding the biochemistry of the mammalian CNS regenerative effort and post-translational processing of axonal peptides.

12:00 - 12:15 Discussion

RESIDENTS PAPER AWARD WINNERS

WINNER – KEVIN J. KIWAT, M.D.
MASSACHUSETTS GENERAL HOSPITAL

"Leukotriene Production in CNS During Experimental Ischemic Brain Insult, Subarachnoid Hemorrhage, And Concussive Brain Injury"

1ST RUNNER UP – DOUGLAS CHYATTE, M.D.
MAYO CLINIC

"Prevention Of Chronic Experimental Cerebrovasospasm With Ibuprofen And Methyl Prednisolone: The Role Of Prostaglandins In The Pathogenesis of Vasospasm"

HONORABLE MENTION – W. CRAIG CLARK, M.D., PH.D.
UNIVERSITY OF TENNESSEE

"Selective In Vitro Killing Of Human Glial Tumor Cells By A Monoclonal Antibody Ricin Immunotoxin"

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David S. Baskin	1983
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George S. Baker	1960	Edmund Morrissey	1960
C. Hunter Shelden	1961-62	Donald F. Coburn	1961-62
Samuel R. Snodgrass	1963	Eben Alexander, Jr.	1963
Theodore B. Rasmussen	1964	George L. Maltby	1964
Edmund J. Morrissey	1965	Robert Pudenz	1965
George Maltby	1966	Francis A. Echlin	1966
Guy L. Odom	1967	Benjamin Whitcomb	1967
James G. Galbraith	1968	Homer S. Swanson	1968
Robert H. Pudenz	1969-70	Augustus McCravey	1969-70
William B. Scoville	1971	Edward W. Davis	1971
Robert L. McLaurin	1972	John R. Green	1972
Lyle A. French	1973	George J. Hayes	1973
Benjamin B. Whitcomb	1974	Richard L. DeSaussure	1974
John R. Green	1975	Ernest W. Mack	1975
William H. Feindel	1976	Frank E. Nulsen	1976
William H. Sweet	1977	Robert S. Knighton	1977
Arthur A. Ward	1978	Robert G. Fisher	1978
Robert B. King	1979	H.T. Ballentine, Jr.	
Eben Alexander Jr.	1980	George Enhi	1980
Joseph Ransohoff II	1981	Courtland H. Davis, Jr.	1981
Byron C. Pevehouse	1982	John F. Mullan	1982
Sidney Goldring	1983	Hugo Rizzoli	1983

PAST SECRETARY-TREASURERS

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

PAST SECRETARY

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

PAST TREASURER

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

PAST MEETINGS OF THE ACADEMY

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 Fransicso, California ..... October 17-19, 1966
The Key Biscayne, Miami, Florida ..... November 8-11, 1967
Broadmoor Hotel, Colorado Springs, Colorado .... October 6-8, 1968
St. Regis Hotel, New York City ..... September 21, 1969
Camino Real Hotel, Mexico City ..... November 18-21, 1970
Sahara-Tahoe Hotel, Stateline, Nevada ..... September 26-29, 1971
New College, Oxford, England ..... September 4-7, 1972
Huntington-Sheraton Hotel, Pasadena, California ..... November 14-17, 1973
Southampton Princess Hotel, Southampton, Bermuda ..... November 6-9, 1974
The Wigwam (Litchfield Park), Phoenix, Arizona November 5-8, 1975

The Mills Hyatt House, Charleston,
South Carolina ..... November 10-13, 1976
Mauna Kea Beach Hotel, Kamuela, Hawaii ..... November 2-5, 1977
Hotel Bayerischer Hof, Munich, Germany..... October 22-25, 1978
Hyatt Regency, Memphis, Tennessee..... November 7-10, 1979
Waldorf Astoria, New York, New York ..... October 1-4, 1980
Sheraton Plaza, Palm Spring, California ..... November 1-4, 1981
Ritz-Carlton Hotel, Boston, Massachusetts ..... October 10-13, 1982
The Lodge at Pebble Beach, California ..... October 23-26, 1983

1984

MEMBERSHIP LIST
AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

Founded October, 1938

HONORARY MEMBERS	ELECTED
HUGO KRAYENBUHL Neurochirurgische University Kantonsspital 8000 Zurich, Switzerland	1974
GUY LAZORTHES 26 Rue D Auriol 31 Toulouse, France	1973
VALENTINE LOGUE 16 Rowan Road Hammersmith London W6 7DU U.K.	1974
GOSTA NORLEN Neurokirurgiska Kliniken Sahlgrenska Sjukhus Goteborg, SV Sweden	1973
KEIJI SANO Dept. of Neurosurgery School of Medicine University of Tokyo Tokyo, Japan	1975

SENIOR MEMBERS	ELECTED
EBEN ALEXANDER, JR. (BETTY) Bowman-Gray School of Medicine of Wake Forest University Winston-Salem, North Carolina 27103	1950
GEORGE S. BAKER (ENID) 607 North Litchfield Road P.O. Box 1234 Litchfield Park, Arizona 85340	1940
H. THOMAS BALLANTINE, JR. (ELIZABETH) Massachusetts General Hospital 275 Charles Street Boston, Massachusetts 02114	1951
EDWIN B. BOLDREY (HELEN) University of California Hospital 3rd Avenue and Parnassus San Francisco, California 94143	1941
E. HARRY BOTTERELL (MARGARET) 2 Lakeshore Boulevard Kingston, Ontario, Canada K7M 4J6	1938
HOWARD A. BROWN 2841 Ptarmigan Drive #1 Walnut Creek, California 94595	1939
HARVEY CHENAULT (MARGARET) 2370 Nicholasville Road Lexington, Kentucky 40503	1938
DONALD F. COBURN (ELLIE) The Plaza 812 1303 Delaware Avenue Wilmington, Delaware 19806	1938
EDWARD W. DAVIS (BARBARA) Providence Medical Office Building 545 N.E. 47th Avenue Portland, Oregon 97213	1949

RICHARD DE SAUSSURE (PHYLLIS) 1962
920 Madison Avenue
Memphis, Tennessee 38103

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

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

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

DEAN H. ECHOLS (FRAN) Founder
Ochsner Clinic
1514 Jefferson Highway
New Orleans, Louisiana 70121

ARTHUR ELVIDGE 1939
275 Brittany Avenue
Montreal HQR 2B3, Quebec, Canada

THEODORE C. ERICKSON (MARTHA) 1940
425 North Livingston St.
Madison, Wisconsin 53703

JOSEPH P. EVANS (HERMENE) Founder
Box 274
Kensington, Maryland 20895

ROBERT FISHER (CONSTANCE) 1956
909 Park Avenue
Plainfield, New Jersey 07060

JOHN D. FRENCH (DOROTHY) 1951
The Center for the Health Sciences
University of California
Los Angeles, California 90024

LYLE A. FRENCH, M.D. (GENE) 1954
University of Minnesota
Medical Center
Minneapolis, Minnesota 55455

JAMES G. GALBRAITH (PEGGY) 1947
2515 Crest Road
Birmingham, Alabama 35223

PHILIP D. GORDY (SILVIA) 1968
1727 East Second Street
Casper, Wyoming 82601

EVERETT G. GRANTHAM (MARY CARMEL) 1942
234 East Gray Street
Louisville, Kentucky 40202

JOHN R. GREEN, M.D. (GEORGIA) 1953
Barrow Neurological Institute
302 West Thomas Street
Phoenix, Arizona 85013

JAMES GREENWOOD, JR. (MARY) 1952
1839 Kirby Drive
Houston, Texas 77019

WALLACE B. HAMBY (ELEANOR) 1938
2001 N.E. 47th Court
Fort Lauderdale, Florida 33308

JESS D. HERRMANN (MARY JO) 1948
Post Office Box 135
Mountain Pine, Arkansas 71956

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

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

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

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

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

FRANK MAYFIELD, M.D. (QUEENEE) — Founder
506 Oak Street
Cincinnati, Ohio 45219

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

WILLIAM F. MEACHAM, M.D. (ALICE) — 1952
Vanderbilt University Hospital
Division of Neurosurgery
Nashville, Tennessee 37232

EDMUND J. MORRISSEY (KATE) — 1941
909 Hyde Street, Suite 608
San Francisco, California 94109

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

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

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

ROBERT H. PUDENZ (RITA) 1943
Box 79, Rt. 1
Vineyard Drive
Paso Robles, California 93446

JOHN RAAF, M.D. (LORENE) Founder
1120 N.W. 20th Avenue, #100
Portland, Oregon 97209

AIDEN A. RANEY, M.D. (MARY) 1946
2010 Wilshire Blvd.
Suite 203
Los Angeles, California 90057

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

R.C.L. ROBERTSON (MARJORIE) 1946
2210 Maroneal Blvd. Shamrock Professional Bldg.
Suite 404
Houston, Texas 77025

STUART N. ROWE (ELVA) 1938
605 Shady Avenue
Pittsburgh, Pennsylvania 15206

RICHARD C. SCHNEIDER (MADELEINE) 1970
Room 3605
Kregge Medical Research Bldg.
University of Michigan Medical Center
Ann Arbor, Michigan 48109

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

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

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

WILLIAM H. SWEET (ELIZABETH) 1950
309 Goddard Avenue
Brookline, Massachusetts 02146

JOHN TYTUS (VIRGINIA "Gina") 1967
Mason Clinic
Seattle, Washington 98107

ALFRED UIHLEIN (IONE) 1950
200 First Street S.W.
Rochester, Minnesota 55901

A. EARL WALKER (AGNES) 1938
1477 Wagontrain Drive, S.E.
Albuquerque, New Mexico 87123

EXUM WALKER (NELLE) 1938
490 Peachtree Street, N.E.
Atlanta, Georgia 30308

THOMAS A. WEAVER, JR. (MARY) 1943
146 Wyoming Street
Dayton, Ohio 45409

BENJAMIN B. WHITCOMB (MARGARET) 1947
50 Union Street
Ellsworth, Maine 04605

BARNES WOODHALL (FRANCES) 1941
Duke University Medical Center
Durham, North Carolina 27710

ACTIVE MEMBERS	ELECTED
JAMES I. AUSMAN (CAROLYN) Henry Ford Hospital 2799 West Grand Blvd. Detroit, Michigan 48202	1978
GILLES BERTRAND (LOUISE) Montreal Neurological Institute 3801 University Street Montreal, Quebec, Canada H3A 1B4	1967
ROBERT S. BOURKE (MARLENE) Division of Neurosurgery Albany Medical College Albany, New York 12208	1983
JERALD S. BRODKEY (ARIELLE) 24755 Chagrin Boulevard Suite #205 Beachwood, Ohio 44122	1977
WILLIAM A. BUCHHEIT, M.D. (LIN) 3401 North Broad Street Philadelphia, Pennsylvania 19140	1980
PAUL H. CHAPMAN (TANSY) Department of Neurosurgery Massachusetts General Hospital Boston, Massachusetts 02114	1983
SHELLEY CHOU (JOLENE) University of Minnesota Medical Center Minneapolis, Minnesota 55455	1974
GALE G. CLARK (MARION) University of California Medical Center San Francisco, California 94143	1970
W. KEMP CLARK (FERN) 5323 Harry Hines Blvd. Dallas, Texas 75235	1970

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

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

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

COURTLAND H. DAVIS, JR. (MARILYN) 1967
Bowman-Gray School of Medicine
Winston-Salem, North Carolina 27103

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

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

GEORGE EHNI (VALARIE "Lari") 1964
The Neurosurgical Group of Houston, Assoc.
6560 Fannin St., #1250, Scurlock Tower
Houston, Texas 77030

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

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

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

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

JOHN T. GARNER 1971
185 South Euclid
Suite 6
Pasadena, California 91101

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

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

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

GRIFFITH R. HARSH III, M.D. (CRAIG) 1980
University of Alabama Medical Center
Birmingham, Alabama 35294

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

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

CHARLES HODGE, M.D. (LINDA) 1982
Department of Neurosurgery
Upstate Medical Center
Syracuse, New York 13210

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

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

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

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

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

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

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

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

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

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

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

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

DAVID G. KLINE (JEANIE) 1972
Louisiana State University Medical Center
1542 Tulane Avenue
New Orleans, Louisiana 70012

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

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

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

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

RAEBURN C. LLEWELLYN (CARMEN) 1963
9661 Lake Forest Blvd.
Suite 350
New Orleans, Louisiana 70127

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

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

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

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

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

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

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

JOHN F. MULLAN, M.D. (VIVIAN) 1963
University of Chicago Clinics
Department of Neurosurgery
950 East 59th Street
Chicago, Illinois 60634

BLAINE S. NASHOLD, JR. (IRENE) 1967
Duke University Medical Center
Durham, North Carolina 27710

FRANK E. NULSEN (GINNEY) 1956
University Hospital of Cleveland
2074 Abington Road
Cleveland, Ohio 44106

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

ROBERT G. OJEMANN (JEAN) 1968
Neurosurgical Service
Massachusetts General Hospital
Boston, Massachusetts 02114

BURTON ONOFRIO (JUDITH) 1975
Mayo Clinic
Rochester, Minnesota 55901

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

S.J. PEERLESS (ANN) 1977
P.O. Box 5339
Terminal A
University Hospital
London, Ontario, Canada N6A 5A5

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

BYRON C. PEVEHOUSE (LUCY) 1964
815 Eucalyptus Avenue
Hillsborough, California 94010

ROBERT W. PORTER (AUBREY DEAN) 1962
6461 Bixby Hill Road
Long Beach, California 90815

JOSEPH RANSOHOFF II (LORI ELLEN) 1965
New York University Medical Center
550 First Avenue
New York, New York 10016

HUGO RIZZOLI (HELEN) 1973
2150 Pennsylvania Avenue, N.W.
Washington, D.C. 20037

THEODORE S. ROBERST (JOAN) 1976
4375 Zarahemla Drive
Salt Lake City, Utah 84117

JAMES T. ROBERTSON (VALERIA) 1971
Department of Neurosurgery
UTCHS, 956 Court Avenue
Memphis, Tennessee 38163

FREDERICK A. SIMEONE (KATE) 1981
800 Spruce Street
Philadelphia, Pennsylvania 19107

JAMES C. SIMMONS (VANITA) 1975
920 Madison Avenue
Memphis, Tennessee 38103

BENNETT M. STEIN (DOREEN) 1970
710 West 168th Street
New York, New York 10034

JIM L. STORY, M.D. (JOANNE) 1972
7703 Floyd Curl Drive
San Antonia, Texas 78284

THORALF M. SUNDT, JR. (LOIS) 1971
200 1st Street, S.W.
Rochester, Minnesota 55901

ANTHONY F. SUSEN (PHYLLIS) 1965
3600 Forbes Avenue
Pittsburg, Pennsylvania 15213

RONALD R. TASKER (MARY) 1971
Toronto General Hospital
Room 7-221E
101 College Street
Toronto, Ontario, Canada
M5G 1L7

JOHN TEW, JR. (SUSAN) 1973
506 Oak Street
Cincinnati, Ohio 45219

GEORGE TINDALL (SUZIE) 1968
Emory University school of Medicine
Division of Neurosurgery
1365 Clifton Road, N.E.
Atlanta, Georgia 30322

JOHN C. VAN GILDER, M.D. (KERSTIN) 1980
University of Iowa Hospital
Iowa City, Iowa 55242

ARTHUR A. WARD, JR. (JANET) 1953
Department of Neurological Surgery
RI-20
University of Washington
Seattle, Washington 98195

CLARK WATTS (PATTY) 1975
One Hospital Drive
Ste. N522
Columbia, Missouri 65212

MARTIN H. WEISS (DEBBY) 1981
USC Medical Center
1200 N. State Street
Los Angeles, California 90033

W. KEASLEY WELCH (ELIZABETH) 1957
Childrens Hospital Medical Center
300 Longwood Avenue
Boston, Massachusetts 02115

LOWELL E. WHITE, JR. (MARGIE) 1971
University of Southern Alabama
Division of Neuroscience
Mobile, Alabama 36688

ROBERT WILKINS (GLORIA) 1973
Duke University Medical Center
Box 3807
Durham, North Carolina 27710

CHARLES B. WILSON 1966
Department of Neurological Surgery
University of California Medical Center
Third and Parnassus
San Francisco, California 94143

FRANK WRENN (BETTY) 1973
27 Memorial Medical Drive
Greenville, South Carolina 29605

DAVID YASHON (MYRNA) 1972
50 McNaughton Road
Columbus, Ohio 43213

NICHOLAS T. ZERVAS (THALIA) 1972
Massachusetts General Hospital
Boston, Massachusetts 02114

SENIOR CORRESPONDING MEMBERS **ELECTED**

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

SHOZO ISHII 1975
Department of Neurosurgery
Juntendo Medical College
Tokyo, Japan

KRISTIAN KRISTIANSEN (KARI) — 1962
Oslo Kommune
Uleval Sykehus
Oslo, Norway

WILLIAM LUYENDIJK — 1973
Pr Bernhardlaan 60
Oegstgeest, The Netherlands

KURT SHURMANN — 1978
Director
Neurochirurg
Univ-Klinik Mainz
Langenbeskstr 1
6500 Mainz, West Germany

CORRESPONDING MEMBERS — **ELECTED**

JEAN BRIHAYE — 1975
1 Rue Heger-Bordet
B-1000 Brussels, Belgium

FERNANDO CABIESES — 1966
Inst. Peruano De Formento Educativo
Av. Arenales 371, of. 501
Apartado 5254
Lima, Peru

JUAN CARDENAS, C. — 1966
Neurologo 4 Neurocirujano
Av. Insurgentes Sur 594, Desp. 402
Mexico 12 D.F.

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

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

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

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

JOHN GILLINGHAM 1962
Edinburg, Scotland EH43 PB

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

SALVADOR GONZALEX-COMEJO (ROSALIE) 1982
Av. Chapultepec Sur 130
Guadalajara, Mexico 44100

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

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

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

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

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

WILLIAM MARGUTH
Director, Department of Neurochirurgischen
Universität Munchen
Marchioninistrasse 15
8000 München 70, West Germany
1978

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

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

HANS-WERNER PIA
Director
Zentrums fur Neurochirurgie
Universitat Giessen
Kliniskstr. 37
6300 Giessen, West Germany
1978

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

CHARAS SUWANWELA
Chulalongkorn Hospital
Medical School
Bangkok, Thailand
1972

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

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

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

GAZI YASARGIL 1975
Neurochirurgische
Universitatsklinik
Kantonsspital
8000 Zurich, Switzerland

DECEASED MEMBERS		DATE ELECTED
DR. SIXTO OBRADOR ALCALDE (Honorary) Madrid, Spain	4/27/67	1973
DR. JAMES R. ATKINSON (Active) Phoenix, Arizona	2/78	1970
DR. PERCIVAL BAILEY (Honorary) Evanston, Illinois	8/10/73	1960
DR. WILLIAM F. BESWICK (Active) Buffalo, New York	5/12/71	1959
DR. SPENCER BRADEN (Active) Cleveland, Ohio	7/20/69	Founder
DR. F. KEITH BRADFORD (Active) Houston, Texas	4/15/71	1938
DR. WINCHELL McK. CRAIG (Honorary) Rochester, Minnesota	2/12/60	1942
DR. WESLEY A. GUSTAFSON (Senior) Jensen Beach, Florida	7/16/75	1942
DR. HANNIBAL HAMLIN (Senior)	6/28/82	1941
DR. HENRY L. HEYL(Senior)	3/01/75	1951
DR. OLAN R. HYNDMAN (Senior) Iowa City, Iowa	6/23/66	1942
MR. KENNETH G. JAMIESON (Corresponding) Brisbane, Australia	1/28/76	1970
SIR GEOFFREY JEFFERSON (Honorary) Manchester, England	3/22/61	1951

DR. WALPOLE S. LEWIN (Corresponding) Cambridge, England	1/23/80	1973
DR. DONALD D. MATSON (Active) Boston, Massachusetts	5/10/69	1950
DR. KENNETH G. McKENZIE (Honorary) Toronto, Ontario, Canada	2/11/64	1960
DR. JAMES M. MEREDITH (Active) Richmond, Virginia	12/19/62	1946
DR. W. JASON MIXTER (Honorary) Woods Hole, Massachusetts	3/16/58	1951
DR. WILDER PENFIELD (Honorary) Montreal, Canada	4/05/76	1960
DR. RUPERT B. RANEY (Active) Los Angeles, California	11/28/59	1939
DR. DAVID L. REEVES (Senior) Santa Barbara, California	8/14/70	1939
DR. DAVID REYNOLDS (Active) Tampa, Florida	4/03/78	1964
DR. WILLIAM B. SCOVILLE (Senior) Hartford, Connecticut	2/25/84	1944
DR. R. EUSTACE SEMMES (Honorary) Memphis, Tennessee	3/2/82	1955
DR. SAMUEL R. SNODGRASS (Senior) Nashville, Indiana	8/08/75	1939
DR. C. WILLIAM STEWART (Corresponding) Montreal, Quebec, Canada	1948	1948

DR. GLEN SPURLING (Honorary) LaJolla, California	2/07/68	1942
DR. HENDRIK SVIEN (Active) Rochester, Minnesota	6/29/72	1957