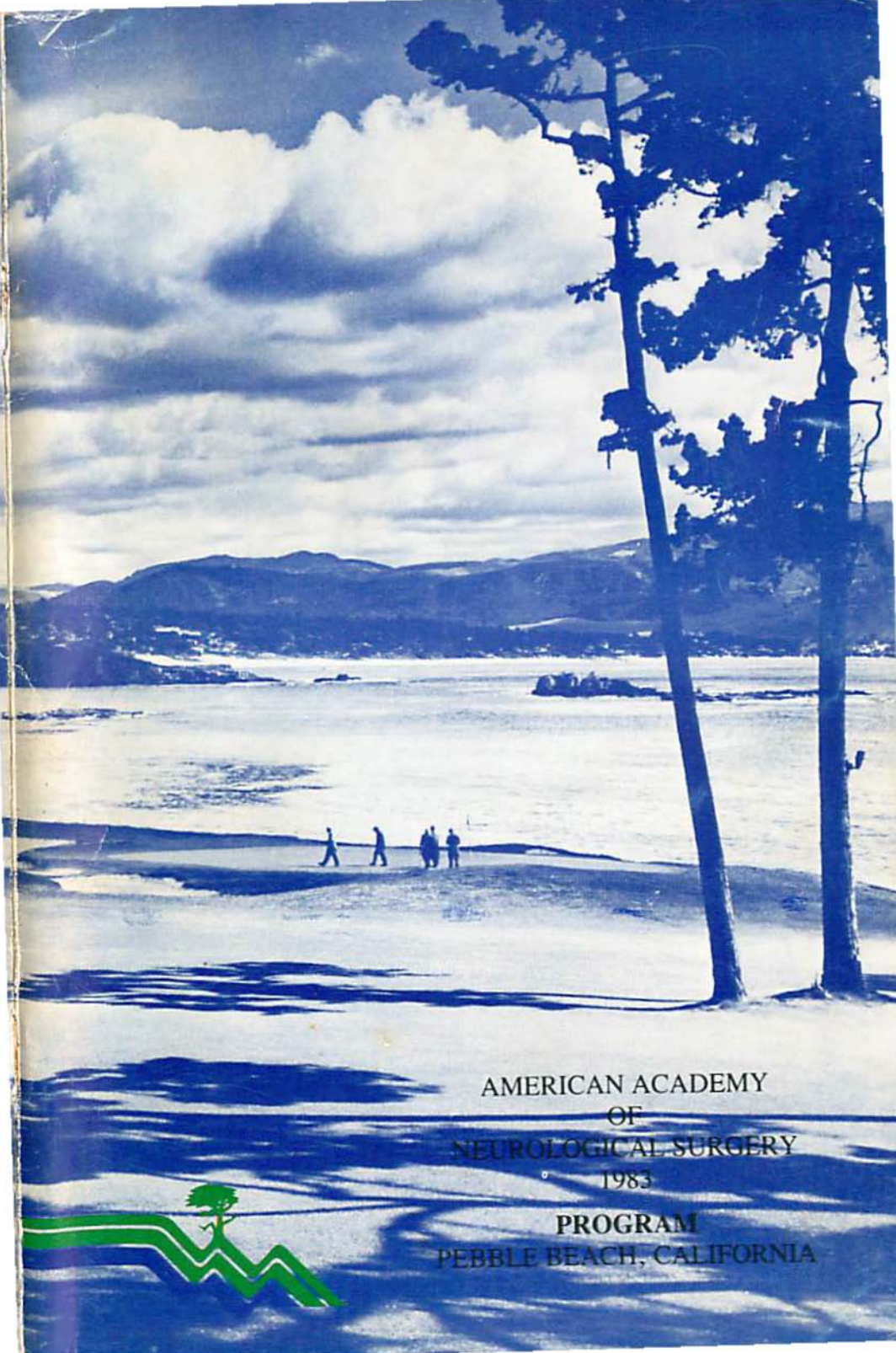

AMERICAN ACADEMY
OF
NEUROLOGICAL SURGERY
1983

# PROGRAM

PEBBLE BEACH, CALIFORNIA

ANNUAL MEETING OF THE
**AMERICAN ACADEMY OF**
**NEUROLOGICAL SURGERY**
**1983**

THE LODGE
PEBBLE BEACH, CA
OCTOBER 23–26, 1983

1983 Officers and Committees

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**Representative to International Committee on Neurosurgical Implants**
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**Representative to the Inter-Agency Committee on Irreversible Coma and Brain Death**
A. Earl Walker

**Local Hosts**
Debby and Marty Weiss

## PROGRAM 1983

**REGISTRATION (Conference Center, Council Room)**

| | |
|---|---|
| Sunday, October 23 | 1:00 - 6:00 p.m. |
| Monday, October 24 | 8:00 - 5:00 pm. |
| Tuesday, October 25 | 8:00 - 10:30 a.m. |

**SUNDAY, OCTOBER 23**

| | |
|---|---|
| 6:00 - 6:45 p.m. | Welcoming Cocktail Party — The Lodge (Pebble Beach Room) |
| 6:45 p.m. | Dinner — The Lodge (Pebble Beach Room) |

**MONDAY, OCTOBER 24**

— ALL SCIENTIFIC SESSIONS WILL BE HELD IN THE CONFERENCE CENTER

| | |
|---|---|
| 7:00 a.m. | Breakfast & Business Meeting (Academy Members Only) The Lodge (Pebble Beach Room) |
| 8:00 a.m. | Opening Remarks, Sidney Goldring President, American Academy |
| 8:05 - 10:00 a.m. | Special Lectures on Neuroscience Moderator: Martin Weiss |
| 10:00 - 10:15 a.m. | Coffee Break |
| 10:15 - 11:35 a.m. | Scientific Session Moderator: Hugo Rizzoli |
| 11:35 - 12:00 p.m. | Academy Award David S. Baskin |

| | |
|---|---|
| 12:00 - 1:30 p.m. | Luncheon |
| 1:30 - 2:50 p.m. | Scientific Session<br>Moderator: Sidney Goldring, Jr. |
| 2:50 - 3:10 p.m. | Coffee Break |
| 3:10 - 4:50 p.m. | Scientific Session<br>Moderator: M. Stephen Mahaley, Jr. |
| 6:00 p.m. | Buses Leave from The Lodge for<br>Dinner in Delmonte Forest<br>(Indian Village) |

**TUESDAY, OCTOBER 25**

| | |
|---|---|
| 7:00 a.m. | Breakfast & Business Meeting<br>(Academy Members Only)<br>Pebble Beach Room |
| 8:00 - 10:00 a.m. | Scientific Session<br>Moderator: Nicholas Zervas |
| 9:00 - 10:00 a.m. | Pituitary Tumors |
| 10:00 - 10:15 a.m. | Coffee Break |
| 10:15 - 11:15 a.m. | Scientific Session<br>Moderator: Russell Patterson |
| 11:15 - 12 noon | Presidential Address - Sidney Goldring |
| 6:30 - 7:30 p.m. | Cocktail Reception<br>Beach and Tennis Club |
| 7:30 - 11:30 p.m. | Dinner Dance (Black Tie)<br>Beach and Tennis Club |

**WEDNESDAY, OCTOBER 26**

| | |
|---|---|
| 7:00 a.m. | Breakfast<br>Pebble Beach Room |
| 8:00 - 11-20 a.m. | Symposium on Vascular Surgery<br>Moderator: Thoralf Sundt |
| 9:40 - 10:00 a.m. | Coffee Break |
| 11:30 a.m. | Meeting Adjourns |

## LADIES PROGRAM 1983

**SUNDAY, OCTOBER 23**

| | |
|---|---|
| 6:00-6:45 p.m. | Welcoming Cocktail Party<br>The Lodge — Pebble Beach Room |
| 6:45 p.m. | Dinner<br>The Lodge — Pebble Beach Room |

**MONDAY, OCTOBER 24**

| | |
|---|---|
| 8:00-5:00 p.m. | Ladies Hospitality Suite<br>The Lodge — Card Room |
| 9:30-4:00 p.m. | Guided Bus and Train Tour through<br>Roaring Camp and Redwoods<br>Lunch at the Greenhouse at the Farm<br>Return through Del Monte Forest |
| 6:00 p.m. | Buses leave from the Lodge for Indian Village<br>in Del Monte Forest |

**TUESDAY, OCTOBER 25**

| | |
|---|---|
| 8:00-5:00 p.m. | Ladies Hospitality Suite<br>The Lodge — Card Room |
| 6:30-7:30 p.m. | Cocktail Reception<br>Beach and Tennis Club |
| 7:30-11:30 p.m. | Dinner Dance (Black Tie)<br>Beach and Tennis Club |

**WEDNESDAY, OCTOBER 26**

8:00-11:30 a.m. Ladies Hospitality Suite
The Lodge — Card Room

# SCIENTIFIC PROGRAM

# AMERICAN ACADEMY
# OF
# NEUROLOGICAL SURGERY
# PEBBLE BEACH, CALIFORNIA
# OCTOBER 23–26, 1983
# SCIENTIFIC PROGRAM

## MONDAY, OCTOBER 24

| | |
|---|---|
| 8:00 a.m. | Opening remarks, Sidney Goldring<br>President, American Academy |
| 8:05 - 10:00 a.m. | Special Lectures on Neuroscience<br>Moderator: Martin Weiss |
| 8:05 - 8:45 a.m. | Floyd Bloom<br>"Molecular Genetics and the<br>Search for New Neuro-Transmitters" |
| 8:45 - 9:25 a.m. | Eric Shooter<br>"Nerve Growth Factor Gene Expression" |
| 9:25 - 10:00 a.m. | Discussion |
| 10:00 - 10:15 a.m. | Coffee Break |

MODERATOR: HUGO RIZZOLI

10:15 a.m.

## 1. SELECTION OF SURGICAL APPROACHES TO THIRD VENTRICULAR TUMORS

James I. Ausman, M.D., Ph.D. and Jeffrey Pearce, M.D.

From 1978 to 1983 at Henry Ford Hospital, 21 patients were operated for third ventricular tumors. A variety of surgical approaches was utilized: transcallosal, transfrontal transventricular, transventricular trans-choroidal fissure, parietal transcallosal, occipital transtentorial, and supracerebellar. Thirteen anterior third and eight posterior third ventricular lesions were treated. Patient positions included: sitting, supine, and ¾ prone.

The advantages and disadvantages to each approach in the selection of the appropriate position will be detailed for lesions in the anterior, mid, and posterior third ventricle. Transoperative and postoperative techniques to aid recovery will be illustrated. A new approach to the pineal and posterior third ventricular region will be presented (the ¾ prone operated side down position). This position minimizes occipital lobe retraction, allows two surgeons to work microscopically, is optimum to approach supracerebellar, pineal region, and posterior third ventricular lesions.

In this series clinical presentation and radiologic tests were not diagnostic of the varied pathology found. Specific treatments were fashioned for each tumor which could not have been accomplished with radiation therapy and shunting or "biological biopsy" as therapeutic choices.

Surgical mortality was 0% and permanent morbidity was 10%. The morbidity included a mild hemiparesis in one patient previously shunted and irradiated years before. A tissue diagnosis of benign teratoma was made. This may have been a complication of the patient's previous amphetamine abuse and vasculitis.

Surgical approaches to the third ventricle are safe, provide tissue diagnosis and tumor debulking for optimization of all therapeutic modalities.

10:35 a.m.

## 2. PLATEAU WAVES: OBSERVATIONS AND A GENERAL THEORY

Michael J. Rosner, M.D.

Recent laboratory observations made upon fluid percussion injured cats suggest plateau waves are causally related to systemic blood pressure variation and require intact autoregulation. While this seems contrary to earlier observations related to plateau waves, we have found that each wave was preceded by a 10-20 mmHg decline in mean blood pressure. When perfusion pressure reaches 70-80 mmHg, the rate of vasodilation to further, small decrements in CPP is known to increase suddenly, and is accompanied by an even greater increase in cerebral blood volume.

A "cascade" of vasodilatation, cerebral blood volume increase, and subsequent elevation of ICP result in further decrements in perfusion pressure. This CPP decrement promotes further vasodilatation and maintains the cycle until vasodilatation is maximal, which of necessity, will be at the lower limit of autoregulation. Cerebral blood volume will be maximal at this point as will ICP. Plateau pressures continued as long as CPP remained constant. If CPP declined, a rise in systemic pressure occurred in response to cerebral ischemia and CPP was restored; this was always at 40-50 mmHg. If the restoration of CPP was adequate for vasoconstriction to occur, ICP declined. CPP increased further and the "cascade" produced rapid autoregulatory vasoconstriction and a rapid decline in ICP. ICP stabilized at a level corresponding to the maximal vasoconstriction possible at the perfusion pressure attained.

We have identified four phases in the development of a plateau wave. The first is the "drift" phase where SABP declines slowly and ICP rises slowly. ICP and SABP correlate -.90 during this phase. The next is the plateau phase proper which begins at a CPP of 70-80 mmHg; ICP becomes maximal at a CPP of about 50 mmHg and remains elevated until CPP is restored. CPP restoration occurs in the third or "ischemic response" phase and is the result of ischemia (CPP$\approx$40) stimulating a sympathoadrenal discharge, thereby elevating SABP. The "resolution" phase is marked by a rapid decline in ICP associated with restoration of CPP to >80 mmHg.

Since the response is to perfusion pressure, systemic circulatory change becomes but a single event capable of initiating the vasodilatory cascade. Other stimuli such as hypoxemia, hypercarbia or venous compression may also induce a CPP decrement and initiate a plateau wave. Similarly, restoration of CPP will abort such a wave.

These responses require mostly intact autoregulation and appear to represent the logical result of an unstable perfusion pressure acting upon a generally autoregulating cerebral vasculature in the face of decreased compliance.

10:55 a.m.

## 3. VEIN OF GALEN ANEURYSMS IN THE FIRST YEAR OF LIFE

Derek A. Bruce, M.D.

In the last two years, we have treated 8 children less than one year of age for arteriovenous shunts in the region of the vein of Galen with dilatation of the vein of Galen. All but 1 of these children presented at birth with heart failure and cardiomegaly. All but 1 had a cranial bruit and in all 8 the diagnosis of a vein of Galen aneurysm was made on CT scan. The cardiac failure was initially controlled in all the children and no emergency surgery performed in the first few days of life. Four of the children developed uncontrollable, high-output cardiac failure: 3 at three weeks of age and 1 at seven months of age. Three of these children underwent surgical procedure for occlusion of shunting arteries and 1 had surgery plus embolization. The fourth child was not operated on and died of heart failure at four weeks of age. Surgery was performed with monitoring of cardiac output and sent for venous $PO_2$. As vessels were occluded, repeated cardiac outputs were performed until the cardiac index was in the normal range. Two of these children are developing normally at four months of age; 1 now almost three years old remains six months to one year behind developmentally.

Two children developed the acute onset of seizures at three months of age with a loss of milestones and apparent acute blindness. In 1 child, the CT scan showed complete loss of cerebral tissue secondary to progressive steal through the AVM and no intervention was felt to be indicated. The second child had severe cerebral steal on arteriogram and surgery was performed with occlusion of many feeding vessels. The first child was untreated, has a flat EEG, and is severely retarded. The second child after poor initial development now has a developmental quotient of approximately 70% at the age of two years.

Two children have developed normally with easily controllable heart failure and have normal CT scans at six months and nine months. Neither of these children had any surgical intervenion.

It is clear that children with arteriovenous shunts into the vein of Galen are at risk for progressive cerebral steal leading to significant loss of cerebral tissue, neurological deterioration, and blindness. Those children in whom intractable heart failure occurs clearly become candidates for some kind of surgical or embolization procedure. They are probably the group in whom therapy is most clearcut. The children who go on to develop progressive cerebral steal appear to do this independent of cardiac failure and without any external signs of progressive atrophy until some acute event occurs. The major question of therapy in these children is whether early intervention with improvement in the cerebral steal would run the likelihood of improving their neurological outcome and preventing progressive

cerebral ischemia. Finally, there is a group of children who apparently require minimal cardiac drugs whose failure is easily controlled, and who go on to develop normally without any apparent cerebral steal and without the requirements of surgical intervention. Several major problems then face the pediatric neurosurgeon in decisions for and against operative intervention in this group of children. It is clear that with good cardiac medication, most of these children got through the first few weeks of life, the period when surgical mortality approached 90%. Some of the children will redevelop cardiac failure which cannot be controlled with medication and surgery at the age of three weeks on appears to be safe. Of major concern, however, is whether surgery is likely to be able to prevent progressive neurological deterioration or whether, because of the in utero steal and early steal, the likelihood of being able to improve intellectual outcome with any intervention at all is zero, bringing up the question as to whether any therapy is indicated for these children. These points will be discussed in light of our last two years' experience.

11:15 a.m.

## 4. CSF SHUNT FLO-CONTROL VALVE: USE IN SHUNTED HYDROCEPHALIC PATIENTS WITH CSF HYPOTENSION OR "SLIT VENTRICLES"

Eldon L. Foltz, M.D. and Jeffrey Blanks, B.S.

CSF shunt systems have been designed in general to work via an arbitrary pressure threshold. CSF pressure measurements over several hours in hydrocephalus patients, however, have shown that CSF pressure is markedly variable. There frequently are large amplitude, short duration pressure increases which far exceed the arbitrary pressure thresholds of the CSF shunts. This strongly implies "over-shunting" probability. A shunt system which functions on a pressure threshold AND a flow rate base has been needed.

Two groups of hydrocephalic patients have now been treated with a new Flo-Control valve for CSF shunts which functions on a pressure threshold but limits flow rate as well. These patient groups include:

1. Twenty-five patients with "slit ventricles" in whom danger of shunt obstruction appeared present. Some patients in this group showed low CSF pressures; others showed near normal mean pressure but high pulse pressures.
2. Five patients with significant neurological deficits apparently produced by LOW CSF pressure (negative), and associated with or without "slit ventricles".

The results in Group 1 indicate the Flo-Control valve is effective in enlarging slit ventricles which are not as yet associated with stiffened walls. The results have been followed by serial CSF pressure measurements and CT scans for ventricular size. Twelve of the twenty-five patients showed favorable ventricular enlargement without clinical symptoms associated in spite of increasing the CSF pressure. In thirteen, the pressure valves were replaced with higher threshold valves, or "stiff ventricles" prevented ventricular enlargement. No adverse clinical effects were observed in any of these. Followup is up to three years.

In the second group, clinical deficits ordinarily associated with an obstructed CSF shunt were present in all patients, apparently associated with significant negative CSF pressure as measured by transducer/polygraph techniques. All five showed prompt clinical improvement and near normal CSF pressures following Flo-Control valve placement. Followup is up to two years.

Though not conclusively proved as yet, this Flo-Control valve is useful in preventing over-shunting from rapid, intermittent, repetitive CSF pres-

sure elevations as occurs in valsalva maneuvers which occur naturally. It seems, therefore, that shunts for hydrocephalus may need not only a pressure threshold capability but a flow control characteristic as well.

11:35 a.m.

**ACADEMY AWARD**

"The Role of the Endogenous Opioid System in the Pathophysiology of Cerebral Ischemia"

DAVIS S. BASKIN
University of California, San Francisco

12:00

**LUNCHEON**

Afternoon Scientific Session

MODERATOR: SIDNEY GOLDRING

1:30 p.m.

## 5. A SURGICAL APPROACH TO LIPOMAS OF THE CONUS MEDULLARIS

Paul H. Chapman, M.D.

Of the various forms of occult spinal dysraphism, spinal lipoma is the most difficult to manage surgically. The usual leptomyelolipoma has a broad indistinct interface with conus medullaris and is intermittently related to emerging nerve roots. Dissection directed at "untethering" the cord is accordingly difficult with the possibility of inadequate release or injury of functioning neural elements. Based on experience with thirty cases the author presents an operative approach reflecting the predictable anatomic features of these lesions. It is important to properly identify the relationship of emerging nerve roots and dural attachments to the spinal cord — lipoma interface. One can anticipate these relationships on the basis of the predominant site of attachment of lipoma to conus — either caudal or dorsal. Preoperative radiographic studies are quite helpful in this regard. In spite of a systematic operative approach, the pitfalls of this surgery remain inad-

equate untethering and injury to functional tissue. These, as well as other persistent management issues, will be discussed in the context of the authors experience.

1:50 p.m.

## 6. MANAGEMENT OF LIPOMYELOMENINGOCELES: EXPERIENCE AT THE HOSPITAL FOR SICK CHILDREN, TORONTO — 1967–1982

Harold J. Hoffman, Chopeow Taecholarn, E. Bruce Hendrick, and Robin P. Humphreys

Controversy continues to surround the issue of management of the patient with a lipomyelomeningocele. Matson in his 1969 textbook stated "operation is usually principally for cosmetic reasons." He went on to say that the "operation, if performed at all, should be delayed until pre-school age."[1] Recently, Chapman[2] pointed out the mechanism by which lipomyelomeningoceles can produce progressive deterioration of neurologic function through the tethering phenomenon.

We reviewed the management of 73 patients with lipomyelomeningoceles who have been treated in our institution over the years 1967-1982. We only included those patients who had more than a cosmetic procedure for their lipomyelomeningocele. Namely, their spinal cord had been freed up from the lipomatous connection to overlying skin and the fully freed up spinal cord had been left encased in a secure dural envelope usually aided by the use of fascia or cadaver freeze-dried dura. Thirty-five of these patients had been treated early in infancy before the age of 6 months, 17 between 6 months and 3 years, 15 between 4 years and 12 years and 6 were older than 12 years at the time of repair.

In order to assess these patients before and after treatment, we devised a functional grading scheme — Grade 0 patients had no significant neurological, orthopedic or urological deficit. Grade 1 patients had minimal weakness and/or muscle wasting and/or foot deformity affecting only one leg without significant gait disturbance and with normal bladder and sphincter function. Grade 2 patients had a neurogenic bladder alone or combined with minimal weakness of one leg or they had intact bladder function but weakness affecting both legs without significant gait disturbance. Grade 3 patients had moderate to severe weakness of one leg with gait disturbance with or without a neurogenic bladder or minimal weakness of both legs combined with a neurogenic bladder. Grade 4 patients were paraparetic

requiring aids for walking with or without a neurogenic bladder. Grade 5 patients were unable to ambulate.

Twenty of the neonates were Grade 0, 9 were Grade 1 and 6 were Grade 2. Among the 17 patients between 6 months and 3 years of age at the time of therapy, 7 were Grade 0, 4 Grade 1 and 6 Grade 2. Among the 15 patients between 4 years and 12 years, none were Grade 0, 5 were grade 1, 7 were grade 2, 3 were Grade 3. Among the 6 patients more than 12 years of age at the time of therapy, none were Grade 0 or 1, 3 were Grade 2, 2 were Grade 3 and one was Grade 4. We found that the neurologic deficit in patients with a lipomyelomeningocele seemed to increase with increasing age probably due to a combination of tethering of the spinal cord combined with direct trauma to the conus produced by a water-hammer effect exerted through lipoma and skin through to the continuous lipoma in spinal cord. We found that the likelihood of improving the functional grade of the patient was best in the infants and decreased with increasing age. We further found that the likelihood of increasing neurological deficit with surgical management of the lipomyelomeningocele was minimal and was the same in all age groups.

Lipomyelomeningoceles are benign lesions which can lead to a progressive deterioration in neurological function if left untreated. Early prophylactic repair of a lipomyelomeningocele ideally within the first weeks of life can effectively prevent loss of neurologic function and ensure a good outcome in these patients. Adequate knowledge of the anatomy of these lesions is absolutely necessary to allow for appropriate surgical management of these lesions.

[1] Matson, DD: Neurosurgery of Infancy and Childhood. Second edition, Charles C. Thomas, publisher, pg 46, 1969.

[2] Chapman, PH: Congenital intraspinal lipomas. Anatomic considerations and surgical treatment. Child's Brain 9: 37-47, 1982.

2:10 p.m.

## 7. MALFORMATIONS RELATED TO THE NEURENTERIC CANAL

Eben Alexander, III, M.D., W. Jerry Oakes, M.D.
and Dennis R.S. Osborne, M.D.

Neurenteric cysts have been described under many different names and in many different clinical manifestations ever since Roth's description in 1881 of a patient with mesenteric abnormalities and a mediastinal cyst which was firmly adherent to the vertebral bodies. Most commonly these involve the cervicodorsal vertebral column, occasionally with hemiverte-

brae and enteric cysts, or diverticuli in the mediastinum or abdomen. A more controversial group of cases involve cysts in the lumbosacral area of the vertebral column. Differentiation of these cysts from "teratomatous cysts" often relies on the clinical situation and overall anatomic arrangement as much as on the histopathology of the cyst itself. Neurenteric cysts are related by their common embryologic origin from defects involving the neurenteric canal, and may even involve totipotential stem cells leading to a "teratoma".

Our seven cases exemplify the wide range of malformations which result from abnormal embryologic development of the neurenteric canal. They include two patients with high cervical intradural extramedullary cysts, two with high thoracic cysts (one of whom had an associated abdominal enteric duplication cyst and butterfly vertebrae), and three with high lumbar cysts dorsal to the spinal cord or roots.

Many proposals have been made concerning the embryology of these cysts, ranging from a cleft notochord, endo-ectodermal adhesion or blastopore retardation with persistent neurenteric canal to compression of the yolk-sac fluid space causing adhesion to the dorsal ectoderm. Resultant ectopic migration of toti-potential cells may lead to teratoma formation. The wide variety of final clinical manifestations is due to the multitude of possible repairs at different times of this developmental error, which occurs at such an early stage in embryonic life.

2:50-3:10 p.m. **Coffee Break**

MODERATOR: M. STEPHEN MAHALEY, JR.

2:30 p.m.

8. **HpD-PHOTORADIATION THERAPY FOR MALIGNANT CEREBRAL TUMORS LABORATORY AND CLINICAL (PHASE I) STUDIES**

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

Hematoporphyrin derivative (HpD) effectively concentrates in experimental and human brain tumors, and it can be activated by red or blue light to produce a cytoxic reaction within tumor cells. A method for quantitating HpD in tissue has been developed, standardized, and applied to human and experimental (rat) brain and tumor tissue. HpD does not transgress the blood-brain barrier, providing an element of protection in clinical use. The time course of concentration of HpD in brain tumor tissue has been deter-

mined and is maximal at 4 hours post-infusion when a dose of 5 mg/Kg is given intravenously.

Optimal wavelengths of light for producing cytotoxicity in cell culture have been determined, and dose calculations have been made; so that minimal concentration of HpD for red and blue light activation are known and are readily achievable. Light exposure necessary to produce the cytoxic effect has been measured in Joules for varying power densities of both red and blue light.

Clinical trials have been carried out in 13 patients harboring malignant brain tumors — all had failed prior conventional therapy. HpD at 5 mg/Kg was well tolerated with no adverse effects in patients who were protected from sunlight. Stereotactic HpD activation with Argon-dye laser (633 nm) light delivered through a fiberoptic probe was utilized for deep and cystic tumors. Post-resection surface irradiation with blue or red light was utilized for superficial lesions. The major practical difficulties are lack of penetration of light and heat effects. Results will be discussed, but are difficult to evaluate in this varied group of patients. Although heat effects can usually be controlled, penetration remains the limiting factor with achievable concentrations of HpD. Recommendations for further development of this promising method for the management of malignant brain tumors will be made.

3:10 p.m.

## 9. CURRENT STATUS: THALAMOTOMY FOR PARKINSONISM

R.R. Tasker, M.D.

Common in the 1960s, this procedure is seldom mentioned in the 1980s. To define current status, the 190 consecutive prospectively quantitatively assessed procedures performed 1965–1981 were reviewed, selecting three groups of 25 from the pre-, mid-, and late-, dopa eras. Physiological criteria defining the target by stimulation and micro-electrode recording were determined. A computer graphics program constructed histograms of average function pre- and three months, one and two years post-operatively for each group and the entire seventy-five. Contralateral and ipsilateral: tremor and rigidity in each muscle group, manual function (five tests) and facial movement as well as speech and gait were examined. Scattergrams plotted each individual patient's pre- versus post-operative function in such a way that maximum post-surgical improvement would cause all data to fall on the 'O' ordinate, maximum post-surgical deterioration on the 'O' abscissa, and no change along a 45° angle through origin in order to detect

deterioration in any function caused by the surgery itself.

No ipsilateral nor any effect on speech, facial movement, gait, writing, or finger-nose movement occurred and no function tested showed consistent post-operative deterioration. Two years after surgery, 82% still had no, 7% almost no contralateral hand tremor (by far the most consistent and prominent site). Improvement in rigidity was also striking, but less complete. There was striking improvement in the ability to touch fingers to thumb and to pat and rotate the wrist repetitively contralaterally, presumably because of lessened tremor. There was no mortality. Eight per cent suffered persisting complications; in one patient each:-thalamic haemorrhage with sensory ataxia, aggravation of dysarthria, arm ataxia, and three with dystonic foot inversion. The differences between the three separate eras was slight, compatible with changes in technical expertise and lesion size. Tremor recurrence was a problem with lesions under three millimeters in diameter.

Thalamotomy is the treatment of choice in Parkinsonian patients under the age of seventy-five with tremor as the major and the disabling symptom not adequately controlled by drugs.

3:30 p.m.

## 10. ELECTRICAL STIMULATION OF THE BRAIN FOR TREATMENT OF CHRONIC PAIN

Ronald F. Young, M.D.

The identification of the endogenous opiate system within the brain led to exploration of a variety of means to activate this system for the relief of chronic intractable pain in man. Since October 1978 we have employed electrical stimulation of a variety of brain loci in man for treatment of chronic pain states. Sixty-two electrode placements have been carried out in patients with chronic pain. A vigorous screening protocol including outpatient and inpatient pain clinic evaluation and treatment as well as psychiatric and psychological testing has been employed to select patients for the procedure. A variety of pain states related to both benign and malignant disease have been treated. Targets for electrode implantation have included the periaqueductal grey, periventricular grey, thalamus and internal capsule. Maximum follow up of these patients is five years and mean follow up is approximately two years. At last follow up 60% of patients described their pain as completely or nearly completely resolved. A 62% reduction in narcotics utilization has been achieved. In addition 35% of patients have returned to a completely normal daily living pattern and another 20% have had a marked improvement in activity pattern. No mortality and low morbidity has been encountered. Measurements of Beta-endorphin levels in

ventricular fluid before, during and after electrical stimulation, as well as a variety of clinical observations, suggest that the pain relief realized in these patients may be unrelated to the release of endogenous opiates. Electrical stimulation of the brain appears to be a useful method for treatment of chronic pain in man but the mechanism of action is at present uncertain.

3:50 p.m.

## 11. EXPERIMENTAL AND CLINICAL EXPERIENCE WITH MOTOR EVOKED POTENTIALS

Walter Levy, M.D. and Clark Watts, M.D.

Evoked potentials have assumed increasing importance in the diagnosis of disease and use in intraoperative monitoring to prevent damage to the nervous system. The established evoked potentials monitor one or another sensory modality, but are unable to monitor the motor system because stimulated peripheral nerves cannot transmit their motor signals backwards across synapses in the spinal cord to the brain. We are developing an evoked potential which monitors the motor pathways as a result of direct stimulation of them. Previously reported was direct stimulation of the motor tracts in the spinal cord when exposed at surgery or when percutaneously stimulated. A less invasive method is to place a stimulating electrode on the scalp over the motor cortex and transmit a current through the scalp to the brain. This will activate a sufficient number of cortex motor cells to produce a descending motor evoked potential which lies in the corticospinal tracts. Depth electrode and lesioning studies in the cat have shown that this has its major signal component in the dorsal lateral quadrant but some signal traveling in the anterior cord, probably in the anterior corticospinal tract, although perhaps in others. Under the appropriate stimulation conditions this signal can produce EMG activation or motor movements of the controlateral limbs. In clinical experience to date a number of cases have shown that it can monitor changes in the nervous system induced by manipulation at surgery and that to date it correlates better with the presence of a lesion as a result of trauma to the spinal cord than do somatosensory evoked potentials. This test has a number of developmental possibilities which can allow it to complement the existing evoked potentials in some areas and add entirely new information that has not been available in others.

4:10 p.m.

## 12. THREE—DIMENSIONAL COMPUTER RECONSTRUCTION OF THE IV VENTRICLE AND ITS SURROUNDING STRUCTURES

F. Afshar, B.Sc., M.D.

The three-dimensional concept of the anatomy of the IV ventricle and its surrounding adjacent structures has proved difficult to understand due to the spatial configuration complexity and close proximity of structures found within, and passing throughout, the tegmentum in this area of the brainstem. Improvement in neuroimaging and stereotaxic neurosurgery requires a knowledge of the precise three-dimensional localisation of structures found in this area of the brain.

This paper presents a method whereby material from two-dimensional serial sections of the IV ventricular area has been regenerated using a three-dimensional computer based recording technique.

The origin of the data base is the Stereotaxic Atlas of the Human Brainstem and Cerebellar Nuclei by Afshar et al.[1] This atlas utilised probability data derived from thirty human autopsy specimens in which the posterior fossa contents were fixed in situ, using the Corsellis technique.[2] Stereotaxic markings were undertaken and specific steps were made to minimise shrinkage artefacts at each step of the specimen preparation.

Using an internal rectilinear coordinate reference system, each nucleus and tract, as seen on a transverse section, was measured in relation to the reference planes and points. The IV ventricle and its adjacent structures were digitised for each of the 1-mm. serial sections of the statistically significant composite atlas using an x-y coordinate plotting microscope.[3]

The digital data thus generated, was used as input to computer programs, enabling scaling and rotation of each section to a common origin.

The x & y coordinates of the computer stored outlines for each 1-mm. section enables subsequent manipulation of the data. One of these manipulations allows the regeneration of the opposite half of hemibrainstem and IV ventricle. In order to obtain three-dimensional information, utilisation of the third, or z, coordinate is necessary and achieved as each section is 1-mm. apart.

Programs have been written to reconstruct traced transverse section outlines and incorporate any desired nuclei and tracts in the IV ventricular floor.

The operational modes for reconstruction permit either the presence or absence of hidden line removal techniques; the former permits one structure to hide an overlapping structure.

With the available data, rotation about any, or all of the three ortho-

gonal axes is possible and permits any view of the reconstructed specimen to be computer generated. The utilisation of colour graphic display facilities further help to improve delineation of both tracts and nuclei in relation to the ventricle. Programs have been written to allow the data to be presented as stereopairs. Examples of important stereotaxic targets, as well as the ventricle, will be illustrated following computer reconstruction in colour, after varying axial rotations.

[1]Afshar, F., Watkins, E.S., Yap, J.C.: Stereotaxic Atlas of the Human Brainstem and Cerebellar Nuclei: A Variability Study. New York, Raven Press (1978).
[2]Corsellis, J.A.B.: Individual Variation in the Size of the Tentorial Opening. J. Neurol. Neurosurg. Psychiatry 21: 279-283 (1958).
[3]Dykes, E., Clement, J.G.: The Construction and Applications of an X-Y Coordinate Plotting Microscope. J Dent. Res. 59 (Special Issue D. Part 1): 1800 Abstract (1980).

4:30 p.m.

## 13. BIPOLAR COAGULATION

Leonard I. Malis, M.D.

Since the invention of bipolar coagulation by James Greenwood in 1940, its use has steadily become an important part of neurosurgical technique. Particularly in the microneurosurgical era, spanning the last 15 years, utilization of bipolar coagulation has increased almost logarithmically.

Both for unipolar and bipolar coagulation, the effects of different electrical waveforms, frequencies, periodic and aperiodic repetitions, output impedances, leakage currents and power regulation, have all been investigated and the differences will be demonstrated. Temperature regulation, brief pulsed application and other modifications will be reviewed.

The relative advantages of bipolar and unipolar generators will be discussed. Bipolar operative techniques, forceps configuration, irrigation, suction, instrument selection and voltage settings will be shown in terms of operative technique for safe, rapid and reliable coagulation with avoidance of sticking, charring and vessel rupture.

## TUESDAY, OCTOBER 25

## SCIENTIFIC SESSION

MODERATOR: NICHOLAS T. ZERVAS

8:00 a.m.

## 14. THE EFFECTS OF SPINAL OPIATES ON MICTURITION IN UNANESTHETIZED INTACT AND SPINAL-TRANSECTED RATS

Charles R. Brent and Tony L. Yaksh

Urinary retention has been frequently reported in the clinical use of spinal and epidural opiates for analgesia. The pharmacology of spinal neurotransmitter/receptor systems involved in micturition is not well understood. A preparation was devised to examine the spinal pharmacology of opiates in unanesthetized rats. Indwelling polyethylene catheters were placed in the urinary bladders of rats and tunneled subcutaneously to exit through the scalp, allowing both ureters and urethra to remain intact. These animals were previously fitted with polyethylene intrathecal (IT) catheters directed to the lumbosacral enlargement. Rats were placed in a restraining cage over a collecting device attached to a strain gauge for measurement of urine volumes. The bladder catheters were attached to a transducer and pump for measurement of intravesicular pressure during continuous infusion of normal saline. At a constant rate of infusion, bladder contractions were observed at regular intervals and were associated with simultaneous and efficient expulsion (no residual volume) of discrete volumes of urine.

In unanesthetized animals, a complete inhibition of the volume-evoked micturition reflex (VEMR) was produced by IT administration of opiates. This effect was produced by both $\mu$ and $\delta$ receptor agonists (morphine, 15 $\mu$g and d-ala$^2$-d-leu$^5$-enkephalin: DADL, 0.8 $\mu$g). The $\kappa$ agonist, U-50488H (50-100 $\mu$g) failed to inhibit the VEMR. Systemic morphine (1mg/kg, i.p.), IT naloxone (15 $\mu$g), and IT thiorphan (70-200 $\mu$g) had no effect on volume-evoked micturition.

Following mid-thoracic spinal cord transection ($T_5$-$T_7$), rats acutely developed arreflexic bladders with urinary retention and overflow incontinence. Intrathecal opiate agonists and antagonists had no effect on these bladders. Between 7 and 14 days the automatic cord bladder state emerged during which infusion of saline evoked micturition at regular intervals but a significant residual urine volume remained. At this stage, the automatic VEMR could be blocked with opiate agonist (DADL, 15 $\mu$g) which was reversible with IT naloxone (15 $\mu$g). Naloxone alone administered intrathecally (15 $\mu$g) or systemically (1 mg/kg, i.p.) was observed to facilitate bladder emptying with an increase in urine volume and reduction in residual volume despite no change in frequency of urination.

We conclude that the supraspinally-mediated VEMR in intact animals as well as spinally-mediated micturition in chronic spinal animals is modu-

lated through μ and/or δ, but not κ opiate receptor systems at the lumbosacral spinal level. These experiments also suggest the possible therapeutic benefits of naloxone in the treatment of urinary retention following spinal injury.

8:20 a.m.

## 15. CHANGES IN FOOD INTAKE WITH ELECTRICAL STIMULATION OF THE VENTROMEDIAL HYPOTHALAMUS IN DOGS

Frederick D. Brown, M.D., Richard G. Fessler, M.D., Ph.D., Jacob R. Rachlin, M.D., and Sean Mullan, M.D.

Although the general phenomenon of appetite satiety following electrical stimulation of the ventromedial hypothalamic area (VMH) of the brain has been known for over two decades, direct clinical application of this knowledge has received little attention because, 1) the expense and size of the necessary equipment made routine use impractical, 2) results of stimulation in small mammals were frequently transient or complicated by undesirable side effects, and 3) a post-stimulation "rebound" of increased ingestion was frequently observed. However, recent technological advances in the use of electrical brain stimulation as a therapy for chronic pain syndromes has circumvented at least the first two of these problems. Therefore, the following experiments were conducted to determine whether repeated stimulation of the VMH could produce prolonged reduction in food intake.

Seven adult dogs were implanted stereotaxically with chronic indwelling Medtronic platinum tipped electrodes in the left VMH area; two dogs served as controls with electrodes placed in the subcortical white matter. Each dog was allowed to recover and to resume normal food and water intake. At that time each dog was food deprived for 24 hours and hooked to a harness, containing the stimulator, which allowed the animal complete freedom of movement. By random assignment each dog was either stimulated (100 μA, 1 msec pulse width, 50 pulse/sec) for 1 hour, or not stimulated. One week later, following resumption of normal food intake, each dog received the opposite treatment. None of the seven experimental dogs ate during stimulation, and five of seven dogs delayed their next meal a period ranging from 6 to 18 hours. When not stimulated, however, each dog ate immediately upon receiving its food and consumed greater than average daily intake ($p < .01$). The two control dogs ate immediately upon receiving food regardless of whether they were stimulated or not.

One week after these experiments, each dog received 1 hour of VMH stimulation on a q12 hour schedule, for 3 consecutive days. Food consump-

tion in the seven dogs ranged from 13% to 51% of normal baseline, and water consumption varied between 29% and 67% of the baseline. Both of these changes were statistically significant ($p < .01$). After cessation of stimulation food and water intake returned to normal within 24-72 hours. The two animals who received subcortical electrodes showed no change in food or water intake.

Using a similar schedule of stimulation over a 60 day period, these dogs have maintained 50 to 60% of baseline food intake and 80 to 90% of baseline water intake. Blood pressure, pulse, respiration, temperature, and gross behavior are not altered during or after stimulation. Serum Na, K, Cl, BUN, Cr, Hct, and protein also have not changed even after 60 days of stimulation. These results suggest that the use of electrical stimulation of the VMH may be a useful modality for regulating food intake, and deserves further examination as a potential alternative therapy for human morbid obesity.

8:40 a.m.

## 16. TISSUE PRESSURE GRADIENTS IN FOCAL AND DIFFUSE BRAIN SWELLING

J. Hoff, M.D. and G. Schielke, M.D.

We studied tissue pressure (TP) in focal and diffuse brain swelling to establish the role of hydrostatic pressure gradients in progressive focal ischemia. Brain TP, rCBF, and brain water were evaluated in two experimental groups of cats. One underwent middle cerebral arterial occlusion ($n = 45$); the other acute water intoxication ($n = 9$). Focal ischemic edema developed over four hours in the first group, characterized by a TP gradient of 7 mmHg between the lesion and the ventricle. The rising gradient was accompanied by a progressive fall in rCBF. Diffuse cytotoxic edema developed over three hours in the second group, without the appearance of a significant TP gradient and without a change in rCBF unless intracranial pressure exceeded 40 mmHg. We believe TP gradients play an early, significant role in the development of progressive ischemia after arterial occlusion.

## PITUITARY TUMORS

MODERATOR: NICHOLAS T. ZERVAS

9:00 a.m.

### 17. LONG-TERM FOLLOWUP OF TRANSSPHENOIDAL SELECTIVE ADENOMECTOMY FOR PROLACTINOMA

K.D. Post, M.D., E. Rodman, M.D.,
M.E. Molitch, M.D., S. Reichlin, M.D.

Transsphenoidal selective resection of prolactinoma is efficacious, having high cure rates and low morbidity but the frequency of late recurrence of adenomas is controversial. We now report our long-term followup of patients having had such surgery. Of 42 patients with microadenomas, 37 achieved normal prolactin (PRL) levels (<25ng/ml) immediately following surgery. Long term followup data was available on 29 of these 37, 24 being found to remain cured at 50 ± 3 months (range 11-81 months) followup. Five patients became hyperprolactinemic again after intervals of 6, 8, 12, and 16 months following surgery. The mean PRL levels of the patients relapsing were significantly greater than those remaining cured both before (176.0 ± 50.0 vs. 95.9 ± 14.0; ($p<0.05$) and 6 weeks following surgery (12.6 ± 1.4 vs. 5.7 ± 0.6; ($p<0.01$). Of 23 patients with macroadenomas 9 had normal PRL levels immediately following surgery. In 5 of these 9, long-term followup data was available for analysis and showed that four remained cured at 41 ± 3 months following surgery and one relapsed at 78 months following surgery. Neither basal PRL levels nor testing of PRL secretory dynamics at 6 weeks postoperatively were predictive of who would relapse: using TRH (500μg), blunted responses (<100% increment above basal) were found in 12 of 27 patients remaining cured and in 3 of 4 patients relapsing ($p>0.05$, Fisher's exact test); using insulin-induced hypoglycemia, blunted responses (<100% increment above basal) were found in 15 of 25 patients remaining cured and in 4 of 4 patients relapsing ($p<0.05$, Fisher's exact test). Retesting of 13 patients with microadenomas who remained cured at a mean of 57 ± 4 months following surgery showed increased numbers who had normal responses to insulin-hypoglycemia and TRH compared to immediate postoperative testing (insulin: early – 38%, late – 62%; TRH: early – 55%, late – 82%).

Conclusions: Transsphenoidal selective adenomectomy remains a highly efficacious mode of therapy for microadenomas and for selected patients with macroadenomas, initial cure rates of 88% being achieved for the former. However, there is a late relapse rate of 17.2% in patients with

microadenomas initially thought to be cured and these relapses occurred usually within the first year. A similar rate of relapse occurred in the patients with macroadenomas (20%), although only a small number were studied. It is not known whether the relapses are due to regrowth of tumor remnants left at surgery or represent new tumor formation, perhaps due to underlying hypothalamic dysregulation. The continued return to normal PRL secretory dynamics in most patients suggests that in these (and therefore in most prolactinoma patients) underlying hypothalamic regulation is normal. Further followup of the patients with continued abnormal secretory dynamics is being performed to see if they will suffer relapse.

9:10 a.m.

## 18. RECURRENT PITUITARY ADENOMAS: TREATMENT ALTERNATIVES

Gerald A. Silverberg, M.D.

Recent advances in endocrinology have increased our diagnostic accuracy in patients harboring pituitary adenomas. The transsphenoidal approach to these tumors has allowed for total excision with minimal risk for most patients. In many patients, total tumor excision may be accomplished with sparing of sufficient normal pituitary that the patient may not be dependent on exogenous hormones postoperatively. In a number of patients harboring large tumors, total excision may still be carried out, sparing the patient postoperative irradiation.

Some patients, however, are not cured of their tumor by the initial therapy. Subsequent treatment may then include watchful waiting, repeat transsphenoidal or transfrontal surgery, external beam irradiation, or interstitial irradiation. We have reviewed our last 100 patients with pituitary adenomas to assess the incidence of recurrence and the efficacy of therapy directed at the recurrence.

Our data suggests that in the patient harboring a hormone-producing adenoma still confined to the sella, repeat transsphenoidal surgery can effect a cure. In patients harboring larger tumors where pituitary tumor remains because of extension anatomically beyond safe surgical excision, external beam irradiation is probably in the patient's best interest. Symptomatic recurrent tumor following surgery and external beam irradiation may be treated by further surgical resection followed by interstitial irradiation with a gamma source, Cesium 137.

9:20 a.m.

## 19. PRE-OPERATIVE CONTRAST RADIOLOGICAL STUDIES OF SELLAR AND PARA-SELLAR TUMORS, DURING TRANSSPHENOIDAL PROCEDURES

Raul Marino, Jr.

This presentation will emphasize the interest of using contrast studies, during the operative procedure under fluoroscopy in 420 cases of transphenoidal procedures, as a control of anatomical preservation of the normal pituitary gland and as a means of planning further surgical tactics in the more difficult cases. We have used either Pantopaque, Dimer-X, Metrizamide, or Barium Sulphate embedded in Gelfoam or cotton in order to visualize the tumor bed after surgical removal of microadenomas or large sellar or para-sellar tumors, like chordomas or craniopharingeomas. In case of cystic tumors, air has also been used.

In intrapituitary microadenomas this technique will allow to visualize and document the exact anatomical and topographical position and size of the lesion, that can be measured in millimeters.

The author has found the technique of special aid mainly in the large cystic or solid sellar and para-sellar tumors, as a guide for evaluation and extent of surgical removal. We now find it indispensible for the planning of the surgical tactics during the removal of the more difficult supra-sellar lesions. A series of selected cases will be presented.

9:30-10:00 a.m. **DISCUSSION**

10:00-10:15 a.m. **Coffee Break**

## SCIENTIFIC SESSION

MODERATOR: RUSSELL H. PATTERSON, JR.

10:15 a.m.

## 20. NEUROLOGICAL CORRELATIONS IN CERVICAL MONORADICULAR SYNDROMES: TREATMENT BY THE MUSCLE SPLITTING APPROACH

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

Seventy-two patients were operated for acute cervical monoradicular syndrome, defined as the abrupt onset of brachalgia with sensorimotor and/or reflex changes, between 1969 and 1982. There were 49 males and 23 females with a mean age of 50 years. The levels were: C5 – 4 cases; C6 – 18 cases; C7 – 38 cases; C8 – 11 cases; and T1 – 1 case.

Only 5 patients with acute herniated disc had anterior discectomy and fusion. These were eliminated from this study. The rest were operated from posterior at one level, determined by the pattern of neurologic deficit, radiography and EMG.

At operation, 51 patients were found to have soft herniated discs with root compression; 19% had lateral "hard" disc at the level of operation. Equivocal findings at exploration were noted in 3 of cases. One patient had chronic granulomatous changes in the epidural space.

The neurologic examination proved to be most specific. A 98% accuracy was based upon careful assessment of 7 motor tests, 2 reflexes and the patients' description of distorted sensation. Pain patterns were unreliable as to level.

Radiographically, osteoarthrotic changes were common, but it was not unusual to find normal x-rays. Plain radiography identified the level in only 15 of 72 (20%). Cervical myelography was performed in all but 2 cases. The involved level was abnormal in all but 4 cases, but multiple defects, due to osteophytes, were frequent and often more obvious, at asymptomatic levels. The myelogram was sensitive in 95%, but specific in only 53%. In 4 patients the myelograms were normal at the level identified by neurologic examination. In all 4, a soft lateral herniated nucleus pulposus was found at exploration.

The EMG, if done more than three weeks after onset of symptoms, was helpful in about 50% of cases.

The operative technique was a unilateral muscle-splitting approach to the cervical spine (72 cases). This will be illustrated with a video tape, with emphasis on accurate interspace identification. There have been no compli-

cations, in contrast to anterior discectomy, with or without fusion.

Conclusions:

1) Acute cervical radiculopathy due to disc is almost invariably at a single interspace.
2) Radiolographic findings in isolation may be misleading.
3) The neurologic findings are the most reliable localizing criteria.
4) A muscle splitting incision with negligible morbidity is described.

10:35 a.m.

## 21. POSTERIOR INSTRUMENTATION FOR UNSTABLE THORACOLUMBAR FRACTURES

Sanford J. Larson, M.D.

One hundred fifty four patients with unstable thoracolumbar fractures involving the spinal cord were operated upon between 1968-81. Myelopathy was complete in 63 and incomplete in 91. The lateral extracavitary approach was used for reconstruction of the spinal canal and interbody fusion, which was achieved in each patient. Posterior metallic fixation was added as a part of a one stage, one incision procedure, allowing early mobilization. Initially, Harrington rods were used, and have been placed in 51 patients. In three, the rods dislocated in the immediate postoperative period. Late, rod related symptoms are common because Harrington rod fixation is a rigid system which does not adapt to rotation of the thoracic spine or to flexion/extension of the lumbar spine. To avoid the problems associated with rods while retaining the stabilizing effect, Weiss springs containing a cylindrical stainless steel rod were substituted and have been placed in 54 patients. Parham bands fix the springs to the spinous processes for lateral stability. Since rotation and flexion/extension movements are possible at each end of the system, the hooks can follow normal body movement. None of the springs have dislocated, caused symptoms, or been removed during a follow-up period of six months to six years and alignment achieved at surgery has been maintained. Luque rods have been applied in five patients. Neurological improvement was about the same in patients who had bone grafts only, Harrington rods, springs, or Luque rods. Prior to surgery, 33 patients were able to walk, 17 with assistance and 16 without. Following operation, 71 patients were able to walk, 18 with assistance and 53 without. Prior to surgery, voluntary control of micturition was present in 32 patients and afterward in 64. Our observations have suggested that Harrington rods are superior to springs in patients with complete myelopathy and severe mal-

alignment who need reduction and fixation without reconstruction of the spinal canal. The springs are superior when myelopathy is incomplete and the spinal column incompletely disrupted. Luque rods are the best choice for patients with incomplete myelopathy and complete disruption of the vetebral column because they provide fixation without distraction or compression.

10:55 a.m.

22. **COLLAGENASE DISCOLYSIS**

Robert G. Fisher, M.D., Ph.D.

A series of patients proven to have a lumbar intervertebral disc were subjected to Collagenase Discolysis. The patients were conservatively managed from four to thirty weeks. In this series Chymopapain was not used. In the USA, Collagenase is still under experimental protocol under the direction of the Food and Drug Administration.

In a series of patients there were no episodes of anaphylactic reaction, shock, death, or neurological deficit.

The results varied but the results proved to be good to excellent in 70-80% of the patients. The hospital stay varied from three to nine days, most being home in five days. It was noted that the discs were mostly at the 4th and 5th lumbar interspaces. All patients returned to their usual occupation in six weeks.

All injections were done under local anesthesia.

11:15-12 noon **Presidential Address — Sidney Goldring**

12 noon **Adjourn**

**WEDNESDAY, OCTOBER 26**

**SYMPOSIUM ON VASCULAR SURGERY**

MODERATOR: THORALF SUNDT

8:00 a.m.

## 23. INITIAL EXPERIENCE WITH COMPUTED TOMOGRAPHIC EVALUATION OF CERVICAL CAROTID ARTERY DISEASE

C.J. Hodge, E. Cacayorin, M. Leeson,
A. Culebras, and A. Iliya

Thin slice computed tomographic (CT) scans following intravenous infusion of a contrast agent were used to evaluate the extracranial carotid arteries in 15 patients with ischemic symptoms. Standard axial scans with reformatted coronal and sagittal images were compared to standard angiographic views of the carotid artery as well as to gross surgical and microscopic pathologic specimens in order to determine the sensitivity and accuracy of this method in depicting the pathologic changes associated with carotid disease. This group of patients included 14 cases of transient ischemic attack secondary to atherosclerotic carotid stenosis and/or ulceration and one case of unilateral cervical carotid dissecting aneurysm. Compared to standard angiography, CT scan more accurately delineated the rostral extent of the atherosclerotic plaque, most of which were calcified, thus allowing better preoperative determination of the amount of rostral carotid exposure needed to comfortably do the endarterectomy. In some patients the area of plaque causing stenosis was non-calcified and relatively lucent. These areas, on gross and histologic examination, were found to be areas of old intramural hemorrhage, thus raising the question of the role of such hemorrhage in the pathogenesis of symptomatic carotid disease. CT scan was superior to conventional arteriography for assessment of the degree of luminal compromise secondary to atheromatous changes.

Areas of ulceration were clearly defined by CT scan. CT scan, like angiography, was not sensitive enough to delineate the small collections of thrombotic material in these ulcers. In one patient with repeated amaurosis fugax and a normal carotid arteriogram, CT scan identified an intraluminal filling defect most likely representing intraluminal clot. Two patients with ulcerative carotid disease were found to have ulcers that penetrated the adventitial wall of the artery thus becoming essentially small aneurysms requiring surgical obliteration of the wall defect. This defect was clearly visable after standard endarterectomy.

The scan from the patient with a spontaneous dissecting carotid aneurysm was particularly interesting because the scan clearly demonstrated the extension of the dissection to the distal internal carotid artery beyond the region of angiographically demonstrated reconstitution of the artery lumen. While these cases represent only a small early experience with this technique, it appears that, in some aspects, CT scanning allows better definition than angiography of the details of carotid disease. The addition of CT

scanning techniques to standard angiography, particularly if coupled with three dimensional reconstruction paradigms, may allow a more accurate assessment of the pathology of carotid disease than angiography alone.

8:20 a.m.

## 24. RADIATION-INDUCED CAROTID ARTERY DISEASE

Gerald D. Silverberg, M.D.

Nine patients with atherosclerotic carotid artery disease associated with neck radiation were compared to forty control patients. The data suggests that significant differences in age, incidence of coronary and peripheral vascular disease, elevated lipids and serum cholesterol, and the angiographic incidence of disseminated atherosclerosis justify the description of radiation-induced carotid disease as a clinical entity. Elevated serum cholesterol and hyperlipidemia may contribute to the development of radiation-induced vascular disease. Successful surgical reconstruction does not appear to be influenced by the prior radiotherapy, although periarterial fibrosis and increased difficulty in separating the plaques from the vascular media was encountered.

8:40 a.m.

## 25. SYNTHETIC TUBE GRAFTS IN CEREBRAL REVASCULARIZATION: CLINICAL AND LABORATORY EXPERIENCE

Willis E. Brown, Jr., Jim L. Story, Lee V. Ansell and M. Abou-Samra

Since our 1978 report on the use of expanded polytetrafluoroethylene (PTFE) tube graft for common carotid to distal middle cerebral artery bypass, we have used various lengths and diameters of this synthetic material in 14 carefully selected patients. Angiographic follow-up (ranging from 2 weeks to 48 months) demonstrates patency and excellent flow in 10 of the 14 patients. In 4 patients, the grafts occluded for various reasons, which we shall discuss. In contrast to these encouraging clinical results with PTFE, we found that the canine model is less rewarding. Only 4 of 16 grafts (examined from 2 days to 18 months after placement) remained open. It appears that there is a notable disparity between patency rates possible in patients and patency rates possible in this canine model. Although synthetic tube grafts cannot be recommended in preference to autogenous vein grafts in cerebral vascular reconstructive procedures, our clinical results thus far lead us to cautious optimism regarding the suitability of PTFE tube grafts in selected patients.

9:00 a.m.

## 26. CONTROLLED HYPERVOLEMIA AS THERAPY FOR ACUTE ARTERIAL INSUFFICIENCY

Glenn W. Kindt, M.D. and Ted S. Keller, M.D.

Induced hypertension as well as hypervolemia in a controlled manner is presently being used at many clinics for acute cerebral insufficiency secondary to postoperative cerebral vasospasm. In some clinics it has been reported to be effective in reversing early neurologic deficits in up to 70% of cases.

The use of this regimen in face of the much more common acute arterial occlusion (thrombotic stroke, cerebral emboli) is presently in the process of being investigated in some clinics. Our experience in this field will be described.

Animal work using acute middle cerebral artery occlusions followed by normotensive hypervolemia have shown marked increases in cerebral blood flow in the ischemic brain with little change in the normal brain. Twelve patients with angiographically documented arterial occlusive disease were treated with intravascular volume expansion. Cardiac output monitoring was instituted with a goal of doubling the intravascular volume and cardiac output but stopping short of decreased cardiac function and well short of pulmonary edema. Eleven of these patients showed marked to dramatic reversal of their neurologic deficits which we thought was directly related to this therapy. There were no significant complications. A controlled study to verify the safety and effectiveness of this therapy is underway.

9:20 a.m.

## 27. REGIONAL CEREBRAL BLOOD FLOW AND NEUROPSYCHOLOGICAL ASSESSMENT IN RECONSTRUCTIVE CEREBROVASCULAR SURGERY

Lee V. Ansell, Willis E. Brown, Jr., and Jim L. Story

Within the spectrum of atherosclerotic cerebrovascular disease is a group of patients who suffer ischemic symptoms from flow-limiting lesions of multiple arteries. The value of surgical vascular augmentation in these patients is not clearly established. This paper presents the preliminary results of a pilot study of such patients. Before and after vascular reinforcement, patients were evaluated with rCBF measurements and extensive neuropsychological tests. Results were correlated with intraoperative blood

flow measurements, clinical progress, and serial angiographic studies.

Fourteen patients have been studied thus far. Vascular augmentation procedures included five superficial temporal to middle cerebral artery anastomoses, three common carotid to middle cerebral artery saphenous vein bypass grafts, five vertebral to common carotid artery transpositions, and one revision of an existing common carotid to common carotid bypass graft. All patients were clinically improved postoperatively. Follow-up angiography demonstrated a 93% patency rate of the vascular grafts and anastomoses at an average of 12.5 months after operation.

In reporting early data from a pilot study, we recognize the necessity to interpret results with great caution. We have noted, however, several interesting correlations. Patients whose resting cerebral hemispheral blood flows were abnormally low before surgery experienced a 17.5% increase in the surgically augmented hemisphere. Also, there was a positive correlation between reinforcements which provided high flow rates (as measured intraoperatively with an electromagnetic flow meter) and postoperative increases in mean resting cerebral blood flow in the augmented hemispheres. Finally, analysis of psychometric test results revealed statistically significant improvements in cognitive/intellectual function following surgery.

Although the data are limited, we are encouraged by these results. We are expanding the study and anticipate that additional data will strengthen our understanding of the benefit of surgical reinforcement in the treatment of complex atherosclerotic cerebrovascular disease.

9:40-10:00 a.m. **Coffee Break**

10:00 a.m.

## 28. MICROSURGICAL RECONSTRUCTION OF THE MIDDLE CEREBRAL ARTERY

E.O. Mora, M. Dujovny, F. Umansky, and J.I. Ausman

Surgical microscopic dissections of 20 unfixed human brain hemispheres were performed through the sylvian fissure. The main trunk of the middle cerebral artery (MCA) and its secondary divisions and tertiary branches were identified. The diameter and length of each of these were recorded. The number of perforating branches as well as the required aperture of the sylvian fissure to comfortably perform anastomoses between the trunks, divisions, and branches were determined. End-to-end, side-to-side, end-to-side anastomoses as well as multiple grafting procedures using segments of the frontopolar artery harvested from the same specimen were

performed. The brains were injected with tinted polyester resin in order to confirm the patency of the anastomosis. Schematic drawings and photographic reproductions of the procedures were obtained. The aperture of the sylvian fissure involved in the anastomotic procedures as well as the length, number of the perforating branches (degree of mobilization) were determined to be the main factors affecting successful performance of the anastomosis. This study has shown the feasibility of these microvascular reconstructive procedures in the insular region which can be applied to the treatment of cerebral aneurysms, focal stenosis, or occlusions of the middle cerebral artery. Furthermore, repair of iatrogenically-damaged vessels in the course of aneurysm dissection or tumor removal can be accomplished using these guidelines.

10:20 a.m.

## 29. PREVALENCE OF ASYMPTOMATIC INCIDENTAL ANEURYSMS: REVIEW OF 4,568 ARTERIOGRAMS

H.R. Winn, J. Taylor and D.L. Kaiser

The prevalence of unruptured cerebral aneurysms is unknown but is estimated to be as high as 5-8%. We studied all cerebral arteriograms performed during the past decade to document the incidence of incidental aneurysms. 4,568 arteriograms were reviewed, and 3,684 were adequate for evaluation. The average age of the population was 47 years, thus comparable to the average age of a population with ruptured aneurysms. Twenty-four patients were found to have an incidental aneurysm: 8 males and 16 females. The aneurysms in 22 of the patients were located in the anterior circulation. Thus, the patients with incidental aneurysms were similar to a ruptured aneurysm population in sex and aneurysm location. In summary, the prevalence of unruptured, incidental, asymptomatic aneurysms, which can be visualized by arteriography, is only 0.65%, or 1/10 of that suggested from autopsy series. Based on this prevalence figure, we have estimated that the rupture rate of intact aneurysms is between 1 and 2.5% per year.

10:40 a.m.

## 30. THE NATURAL HISTORY OF ARTERIOVENOUS MALFORMATIONS OF THE BRAIN: A CLINICAL STUDY

Dan Fults, M.D. and David L. Kelly, Jr., M.D.

A series of 131 patients with intracranial AVM's is presented and some conclusions are drawn regarding the natural history of this disease. Eighty-

three patients managed conservatively and 48 patients treated surgically were followed for an average of eight years and their health conditions were rated. When viewed collectively, patients with cerebral AVM's fared neither better nor worse when undergoing surgery than when managed conservatively without operation. Hemorrhage occurred in 61.8% of all patients. A second hemorrhage occurred in 67.4% of survivors of the first hemorrhage. The mortality associated with recurrent hemorrhage did not increase significantly with successive episodes of hemorrhage. The rate of rebleeding was initially 17.9% per yer but declined to 3% per year after five years and then 2% per year after ten years. The natural history of patients presenting with seizures was more favorable than that of patients presenting with hemorrhage. The seizure patients had a 26.9% chance of hemorrhaging and an 11.6% chance of dying from that hemorrhage. Surgery altered the natural history of cerebral AVM's more favorably in patients presenting with a hemorrhage than in patients presenting with seizures. Patients with posterior fossa AVM's carried a dismal prognosis with a mortality of 66.7% with the first hemorrhage. Recurrent posterior fossa hemorrhage was the rule and few survived a second hemorrhage. The prognosis among children with AVM's was no different from that of adults. Conservative management is recommended for patients with supratentorial AVM's whose mode of presentation is epilepsy and not hemorrhage. Surgical excision, when technically feasible, is recommended for patients who have hemorrhaged.

11:00 a.m.

## 31. POSTERIOR FOSSA ARTERIOVENOUS MALFORMATIONS

Bennett M. Stein, M.D.

Experience in the treatment of posterior fossa arteriovenous malformations comprising approximately ten percent of a series of two-hundred arteriovenous malformations of the brain will be reviewed. These malformations will be divided into those involving the cerebellum, brain stem and posterior fossa cisterns. The most common malformations involve the cerebellar hemispheres or vermis.

Arteriovenous malformations of the posterior fossa occur in a somewhat older age group than in the cerebral hemispheres. Detailed and preferably stereoscopic angiography is necessary to define the exact location of these lesions. CT scanning on fourth generation scanners has been helpful in determining the exact relationship of these malformations to the important brain stem structures. Until recently the only treatment to be offered for these malfunctions has been surgical since standard embolization tech-

niques are not applicable to these lesions. Surgical considerations involve the following:

1) The exact location and size of the lesion.
2) Operative positioning and techniques of hypotension to facilitate the surgery.

Complications related to the location of these malformations will be discussed in some detail.

Recently, using new techniques of interventional neuroradiology it has been possible to reduce the size of the posterior fossa malformations by techniques of selective embolization prior to surgery. This will be discussed briefly.

## 1983

## ACADEMY AWARD

DAVID S. BASKIN, M.D.
Chief Resident
University of California
at San Francisco

"The Role of the
Endogenous Opioid System in the
Pathophysiology of Cerebral Ischemia"

HONORABLE MENTION

Douglas Chyatte
Mayo Clinic
Rochester, MN

"Prevention of Chronic Experimental
Cerebral Vasospasm Using Ibuprofen
and High Dose Methylprednisolone

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

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| Dr. Floyd Bloom<br>San Diego, CA | The Academy |
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| Dr. Dan Fults<br>Winston Salem, NC | Dr. David Kelly |
| Dr. John Godersky<br>Iowa City, IA | Dr. John C. Van Gilder |
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Syracuse, NY

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London, England

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Vancouver, B.C., Canada

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Dr. Robert B. King

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Joseph P. Evans ..... 1941
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Lyle A. French ..... 1973
Benjamin B. Whitcomb .... 1974
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William H. Feindel ..... 1976
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Robert B. King..... 1979
Eben Alexander ..... 1980
Joseph Ransohoff II ..... 1981
Byron C. Pevehouse..... 1982

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Francis Murphey..... 1941
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John Raaf ..... 1947
Arthur R. Elvidge..... 1948
F. Keith Bradford ..... 1949
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Rupert B. Raney..... 1953
David L. Reeves..... 1954
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Jess D. Herrmann..... 1956
George S. Baker..... 1957
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C. Hunter Sheldon ..... 1959
Edmund Morrissey ..... 1960
Donald F. Coburn..... 1961-62
Eben Alexander, Jr..... 1963
George L. Maltby..... 1964
Robert Pudenz ..... 1965
Francis A. Echlin ..... 1966
Benjamin Whitcomb..... 1967
Homer S. Swanson..... 1968
Augustus McCravey.... 1969-70
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John R. Green..... 1972
George J. Hayes ..... 1973
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Robert S. Knighton ..... 1977
Robert G. Fisher..... 1978
H.T. Ballantine, Jr..... 1979
George Enhi ..... 1980
Courtland H. Davis, Jr. .... 1981
John F. Mullan ..... 1982

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

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|---|---|
| Russel H. Patterson, Jr. . | 1974-76 |
| Phanor L. Perot, Jr. .... | 1977-80 |

## PAST TREASURER

| | |
|---|---|
| Russel H. Patterson, Jr. .... | 1973 |
| Phanor L. Perot, Jr. .... | 1974-76 |
| John T. Garner ..... | 1977-80 |

## 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 Francisco, California..... October 17-19, 1966
The Key Biscayne, Miami, Florida ..... November 8-11, 1967
Broadmoor Hotel, Colorado Springs, Colorado ..... October 6-8, 1968
St. Regis Hotel, New York City..... September 21, 1969
Camino Real Hotel, Mexico City..... November 18-21, 1970
Sahara-Tahoe Hotel, Stateline, Nevada ..... September 26-29, 1971
New College, Oxford, England ..... September 4-7, 1972
Huntington-Sheraton Hotel, Pasadena, California ..... November 14-17, 1973
Southampton Princess Hotel, Southampton,
Bermuda ..... November 6-9, 1974
The Wigwam (Litchfield Park), Phoenix, Arizona..... November 5-8, 1975
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 Springs, California ..... November 1-4, 1981
Ritz-Carlton Hotel, Boston, Massachusetts..... October 10-13, 1982

# 1983
# MEMBERSHIP LIST
# AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
**Founded October, 1938**

| HONORARY MEMBERS | ELECTED |
|---|---|
| HUGO KRAYENBUHL<br>Neurochirurgische University<br>Kantonsspital<br>8000 Zurich, Switzerland | 1974 |
| GUY LAZORTHES<br>26 Rue D Auriol<br>31 Toulouse, France | 1973 |
| VALENTINE LOGUE<br>Naida Vale Hospital<br>London, W. 9, England | 1974 |
| GOSTA NORLEN<br>Neurokirurgiska Kliniken<br>Sahlgrenska Sjukhus<br>Goteborg, SV Sweden | 1973 |
| KEIJI SANO<br>Dept. of Neurosurgery<br>School of Medicine<br>University of Tokyo<br>Tokyo, Japan | 1975 |
| R. EUSTACE SEMMES<br>20 S. Dudley, St. 101-B<br>Memphis, Tennessee 38103 | 1955 |

| SENIOR MEMBERS | ELECTED |
|---|---|
| EBEN ALEXANDER, JR. (BETTY)<br>Bowman-Gray<br>School of Medicine<br>of Wake Forest University<br>Winston-Salem, North Carolina 27103 | 1950 |
| GEORGE S. BAKER (ENID)<br>200 First Street, S.W.<br>Rochester, Minnesota 55901 | 1940 |
| H. THOMAS BALLANTINE, JR. (ELIZABETH)<br>Massachusetts General Hospital<br>275 Charles Street<br>Boston, Massachusetts 02114 | 1951 |
| EDWIN B. BOLDREY (HELEN)<br>University of California Hospital<br>3rd Avenue and Parnassus<br>San Francisco, California 94143 | 1941 |
| E. HARRY BOTTERELL (MARGARET)<br>2 Lakeshore Boulevard<br>Kingston, Ontario, Canada | 1938 |
| HOWARD A. BROWN (DOROTHY)<br>2001 Union Street<br>San Francisco, California 94123 | 1939 |
| HARVEY CHENAULT (MARGARET)<br>2370 Nicholasville Road<br>Lexington, Kentucky 40503 | 1938 |
| DONALD F. COBURN (ELLIE)<br>The Plaza 812<br>1303 Delaware Avenue<br>Wilmington, Delaware 19806 | 1938 |
| EDWARD W. DAVIS (BARBARA)<br>Providence Medical Office Building<br>545 N.E. 47th Avenue<br>Portland, Oregon 97213 | 1949 |

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

R.M. PEARDON DONAGHY (FRANCES) 1970
University Health Center
1 S. Prospect Street
Burlington, Vermont 05401

CHARLES G. DRAKE (RUTH) 1958
University Hospital
339 Windermere Road
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FRANCIS A. ECHLIN (LETITIA) 1944
100 East 77th Street
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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
Rutgers Medical School
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JOHN D. FRENCH (DOROTHY) 1951
The Center for the Health Sciences
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LYLE A. FRENCH, M.D. (GENE) 1954
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HOMER S. SWANSON (LaMYRA) 1949
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Ambulatory Care Center, Ste. 312
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JOHN TYTUS (VIRGINIA "Gina") 1967
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ALFRED UIHLEIN (IONE) 1950
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EXUM WALKER (NELLE) 1938
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THOMAS A. WEAVER, JR. (MARY) 1943
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BARNES WOODHALL (FRANCES) 1941
Duke University Medical Center
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Henry Ford Hospital
2799 West Grand Blvd.
Detroit, Michigan 48202

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3801 University Street
Montreal, Quebec, Canada

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University Neurosurgeons of Cleveland, Inc.
2074 Abington Rd.
Cleveland, Ohio 44106

WILLIAM A. BUCHHEIT, M.D. (HELEN) 1980
3401 North Broad Street
Philadelphia, Pennsylvania 19140

SHELLEY CHOU (JOLENE) 1974
University of Minnesota Medical Center
Minneapolis, Minnesota 55455

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

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

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
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
Medical Arts Plaza
Hospital Rd.
Pascagoula, Mississippi 39567

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

GEORGE EHNI (VELAIRE "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

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

ELDON L. FOLTZ (CATHERINE) 1960
2480 Monaco Drive
Laguna Beach, CA 92651

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525 East 68th Street
New York, New York 10021

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1127 East Green Street
Pasadena, California 91106

HENRY GARRETSON (MARIANNA) 1973
Health Sciences Center
University of Louisville
Louisville, Kentucky

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

PHILIP D. GORDY (SILVIA) 1968
1727 East 2nd Street
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JOHN W. HANBERY (SHIRLEY) 1959
70 Mercedes Lane
Atherton, CA 94025

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

MAJ. GEN. GEORGE S. HAYES (CATHERINE) 1962
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E. BRUCE HENDRICK (GLORIA) 1968
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555 University Ave., jrm. 1502
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CHARLES HODGE, M.D. 1982
Department of Neurosurgery
Upstate Medical Center
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JULIAN HOFF (DIANNE) 1975
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HAROLD HOFFMAN, M.D. 1982
The Hospital for Sick Children
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EDGAR M. HOUSEPIAN 1976
710 West 168th Street
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ALAN R. HUDSON (SUSAN) 1978
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M5B LA6

WILLIAM E. HUNT (CHARLOTTE) 1970
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University Hospital
410 West 10th Ave.
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JOHN A. JANE, M.D. (NOELLA) 1982
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ELLIS B. KEENER (ANN) 1978
370 Winn Way, #201
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DAVID KELLY (SALLY) 1975
Bowman-Gray School of Medicine
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WILLIAM A. KELLY (JOAN) 1977
15520-65th Place, N.E.
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GLENN W. KINDT (CHARLOTTE) 1977
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ROBERT B. KING (MOLLY) 1958
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WILLIAM M. LOUGHEED (GRACE ELEANOR) 1962
Medical Arts Bldg., Suite 430
170 St. George Street
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HERBERT LOURIE (BETTY) 1965
713 East Genessee Street
Syracuse, New York 13210

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

ERNEST W. MACK (BOBBIE) 1956
605 Juniper Hill Rd.
Reno, NV 89509

M. STEPHEN MAHALEY, JR. (JANE) 1972
University of North Carolina
148 Clinical Sciences Bldg.
Chapel Hill, North Carolina 27514

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

ROBERT L. McLAURIN 1955
Division of Neurosurgery
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| DECEASED MEMBERS | DATE | ELECTED |
|---|---|---|
| DR. SIXTO OBRADOR ALCALDE (Honorary)<br>Madrid, Spain | 4/27/67 | 1973 |
| DR. JAMES R. ATKINSON (Active)<br>Phoenix, Arizona | 2/78 | 1970 |
| DR. PERCIVAL BAILEY (Honorary)<br>Evanston, Illinois | 8/10/73 | 1960 |
| DR. WILLIAM F. BESWICK (Active)<br>Buffalo, New York | 5/12/71 | 1949 |
| DR. SPENCER BRADEN (Active)<br>Cleveland, Ohio | 7/20/69 | Founder |
| DR. F. KEITH BRADFORD (Active)<br>Houston, Texas | 4/15/71 | 1938 |
| DR. WINCHELL McK. CRAIG (Honorary)<br>Rochester, Minnesota | 2/12/60 | 1942 |
| DR. WESLEY A. GUSTAFSON (Senior)<br>Jensen Beach, Florida | 7/16/75 | 1942 |
| DR. HANNIBAL HAMLIN (Senior) | 6/28/82 | 1941 |
| DR. HENRY L. HEYL (Senior)<br>Hanover, New Hampshire | 3/01/75 | 1951 |
| DR. OLAN R. HYNDMAN (Senior)<br>Iowa City, Iowa | 6/23/66 | 1942 |
| MR. KENNETH G. JAMIESON (Corresponding)<br>Brisbane, Australia | 1/28/76 | 1970 |

| | | |
|---|---|---|
| SIR GEOFFREY JEFFERSON (Honorary)<br>Manchester, England | 3/22/61 | 1951 |
| DR. WALPOLE S. LEWIN (Corresponding)<br>Cambridge, England | 1/23/80 | 1973 |
| DR. DONALD D. MATSON (Active)<br>Boston, Massachusetts | 5/10/69 | 1950 |
| DR. KENNETH G. McKENZIE (Honorary)<br>Toronto, Ontario, Canada | 2/11/64 | 1960 |
| DR. JAMES M. MEREDITH (Active)<br>Richmond, Virginia | 12/19/62 | 1946 |
| DR. W. JASON MIXTER (Honorary)<br>Woods Hole, Massachusetts | 3/16/58 | 1951 |
| DR. WILDER PENFIELD (Honorary)<br>Montreal, Canada | 4/05/76 | 1960 |
| DR. RUPERT B. RANEY (Active)<br>Los Angeles, California | 11/28/59 | 1939 |
| DR. DAVID L. REEVES (Senior)<br>Santa Barbara, California | 8/14/70 | 1939 |
| DR. DAVID REYNOLDS (Active)<br>Tampa, Florida | 4/03/78 | 1964 |
| R. EUSTACE SEMMES (Honorary)<br>Memphis, Tennessee | 3/2/82 | 1955 |
| DR. SAMUEL R. SNODGRASS (Senior)<br>Nashville, Indiana | 8/08/75 | 1939 |
| DR. C. WILLIAM STEWART (Corresponding)<br>Montreal, Quebec, Canada | 1948 | 1948 |
| DR. GLEN SPURLING (Honorary)<br>La Jolla, California | 2/07/68 | 1942 |
| DR. HENDRIK SVIEN (Active)<br>Rochester, Minnesota | 6/29/72 | 1957 |

## AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
## 1983 ANNUAL MEETING

### EVALUATION

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

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

___Excellent
___Good
___Poor

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

___Too much review of old knowledge?
___Too simple or elementary?
___Too complex or abstruse?
___Of little practical value?

(3) Did the speakers aim their talks:

___Too low
___Too high
___Just about right

### SCIENTIFIC PROGRAM

Monday's Sessions Excellent_____Good _____ Poor _____
Comments _____
_____

Tuesday's Sessions Excellent_____Good _____ Poor _____
Comments _____
_____

Wednesday's Sessions Excellent_____Good _____ Poor _____
Comments _____
_____

**SOCIAL PROGRAM**

Comments_____

_____

_____

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

_____

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

_____

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Please Print Name:

Return to: John T. Garner, M.D.
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## NOTES

## NOTES

## NOTES

## NOTES

Photo by William C. Brooks, Pebble Beach, CA