

Byron Seehouse

American Academy
of
Neurological Surgery
1981

PROGRAM
PALM SPRINGS, CALIFORNIA

Meeting of the
**AMERICAN ACADEMY OF
NEUROLOGICAL SURGERY**
1981

**Sheraton Plaza
Palm Springs, CA
Nov. 1-4, 1981**

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A. Earl Walker

Local Hosts

Dr. and Mrs. John T. Garner

PROGRAM 1981

REGISTRATION (Poolside)

Sunday, November 1	3:00 - 6:00 p.m.
Monday, November 2	8:00 - 10:00 a.m. 2:00 - 4:00 p.m.
Wednesday <i>Tuesday</i> November 3	8: - 10:00 a.m.

SUNDAY, NOVEMBER 1

6:00 - 8:00 p.m.	Welcoming Cocktail Party Poolside
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MONDAY, NOVEMBER 2

— ALL SCIENTIFIC SESSIONS WILL BE HELD IN THE PLAZA BALLROOM

7:00 - 8:00 a.m.	Breakfast and Business Meeting (Academy Members Only) Plaza Ballroom
8:20 - 8:30 a.m.	Opening Remarks by Joseph Ransohoff, Pres., American Academy
8:30 - 10:30 a.m.	Scientific Session
10:30 - 10:50 a.m.	Coffee Break
10:50 - 12:00 p.m.	Scientific Session
12:00 - 1:00 p.m.	Luncheon
1:00 - 3:00 p.m.	Scientific Session
3:00 - 3:20 p.m.	Coffee Break
3:20 - 4:40 p.m.	Scientific Session

6:30 - 11:00 p.m.

Western Steak Fry
Poolside

TUESDAY, NOVEMBER 3

7:00 - 8:00 a.m.

Breakfast and Business Meeting
(Academy Members Only)
Plaza Ballroom

8:30 - 10:10 a.m.

Scientific Session

10:10 - 10:30 a.m.

Coffee Break

10:30 - 11:00 a.m.

Presidential Address

6:30 - 7:30 p.m.

Cocktail Reception
Plaza Ballroom

7:30 - 11:30 p.m.

Dinner Dance (Black Tie)
Plaza Ballroom

WEDNESDAY, NOVEMBER 4

7:00 - 8:00 a.m.

Breakfast and Business Meeting
(Academy Members Only)
Plaza Ballroom

8:30 - 10:10 a.m.

Scientific Session

10:10 - 10:30

Coffee Break

10:30 - 12:10 p.m.

Scientific Session

LADIES PROGRAM 1981

REGISTRATION (Poolside)

Sunday, November 1

3:00 - 6:00 p.m.

Monday, November 2

8:00 - 10:00 a.m.

2:00 - 4:00 p.m.

Wednesday, November 3

8:00 - 10:00 a.m.

SUNDAY, NOVEMBER 1

6:00 - 8:00 p.m.

Welcoming Cocktail Party
Poolside

MONDAY, NOVEMBER 2

8:00 - 11:00 a.m.

Ladies Hospitality Brunch

9:30 a.m. - 2:00 p.m.

Aerial Tramway Tour

12:00 noon - 1:45 p.m.

Luncheon
Atop Mt. San Jacinto

6:30 - 11:00 p.m.

Western Steak Fry
Poolside

TUESDAY, NOVEMBER 3

8:00 - 11:00 a.m.

Ladies Hospitality Brunch

9:15 a.m. - 12:00 noon

Celebrity Homes Tour
By Arrangement

10:30 - 11:00 a.m.

Presidential Address
Plaza Ballroom

6:30 - 7:30 p.m.

Cocktail Reception
Plaza Ballroom

7:30 - 11:30 p.m.

Formal Dinner Dance
Plaza Ballroom

WEDNESDAY, NOVEMBER ⁴

8:00 - 11:00 a.m.

Ladies Hospitality Brunch

SCIENTIFIC PROGRAM

1980

Adenoma	25%	
Cushing's	4%	
Prolactin	38%	
Mixed	10%	
Inactive	23%	
		Oncocytoma 3%
		Inactive 5%
		abnormal substrate (glycoproteins) 15%

SCIENTIFIC PROGRAM
THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
ACADEMY MEETING
Palm Springs, California
November 1-4, 1981

MODERATOR: Courtland H. Davis, Jr.

MONDAY, NOVEMBER 2

8:20 a.m. Opening Remarks
 Joseph Ransohoff

8:30 a.m.

1. PITUITARY TUMORS SECRETING ONLY
THE ALPHA SUBUNIT OF THE
GLYCOPROTEIN HORMONES

E. Chester Ridgway, M.D.
Anne Klibanski, M.D.
Nicholas T. Zervas, M.D.

The pituitary glycoproteins, TSH, LH and FSH are composed of immunologically identical alpha subunits and biologically specific beta subunits. Pituitary tumors commonly secrete prolactin, GH, or ACTH, and, in some patients, co-existent abnormalities in alpha subunit secretion may occur. The evaluation of patients with "non-functioning chromophobe adenomas is difficult because these patients lack a specific tumor marker. We have recently identified four patients previously characterized as having "non-functioning chromophobe adenomas" who were found to have high serum concentrations of only the alpha subunit. In these patients, measurement of the alpha subunit was valuable in diagnosing a pituitary tumor and in following the patients' following therapy. The serum alpha levels ranged from 5.0 to 9.6 ng/ml (normal 0.5 to 2.5 ng/ml). Alpha secretion was autonomous in two patients; unresponsive to TRH (thyrotropin releasing hormone) stimulation. Analysis of the pituitary tumor tissue revealed only the alpha subunit which was immunologically and chromatographically

identical to serum alpha. Following transsphenoidal hypophysectomy, serum alpha levels decreased. We conclude (1) pituitary tumors secreting only the alpha subunit have been identified and (2) the alpha subunit is a useful tumor marker in some patients previously thought to have non-functioning pituitary tumors.

8:50 a.m.

2. "TRANSPHENOIDAL SURGERY FOR PITUITARY TUMOR: THE IOWA EXPERIENCE"

8 had hydrocephalus John C. VanGilder, M.D.
9 ↑ intracran. pressure.

1% mortality

Two hundred sequential patients, at the University of Iowa, between the ages of 5 and 87 years with a pituitary tumor > 3.5 cm. diameter removed by transphenoid operation were reviewed to assess the advantages of this extracranial approach. One hundred forty-two tumors were nonhormone secreting and presented with symptoms of parasellar mass effect and/or insufficiency of one or more pituitary hormones. Fifty-eight patients had evidence of hypersecretion of one or more pituitary hormones.

The series is reviewed to assess postoperative visual acuity and field changes, status of pituitary hormone function, mortality and morbidity of surgery, and recurrence incidence in a 1-5 year follow-up period.

3 cases with large frontal extensions were done supra tentorial.

9:10 a.m. *Subtotal Trans. sphen. approach treated by X-ray.*

3. ADJUVANT THERAPY FOR THE MANAGEMENT OF FUNCTIONAL PITUITARY TUMORS

Martin H. Weiss, Calvin Ezrin, and Charles M. March

A six-year follow-up of patients with prolactin secreting microadenomas of the pituitary reveals that the vast majority of these patients do not show significant change in the size of their tumor during this period of observation. We have subsequently found that it is reasonable to treat patients with Stage I prolactin secreting tumors desirous of restitution or fertility with bromocriptine in an effort to reduce prolactin and restore normal gonadal function. Only one of 43 patients so treated has undergone significant growth of a pituitary tumor during the course of her pregnancy;

this growth was controlled symptomatically by re-institution of bromocryptine therapy during the pregnancy with a subsequent resection of the pituitary tumor after delivery. This same agent has proven effective in reducing the size of *non-cystic* contrast enhancing prolactin secreting tumors therein simplifying their surgical resection. None of these tumors in our series, however, has undergone cure by medical treatment alone in that withdrawal of the agent has resulted in a return of hyperprolactinemia.

Although constituting only 25 to 30% of all acromegalics, those growth hormone secreting tumors that show stimulation to the administration of TRH have shown a significant response to bromocryptine administration in the form of reduction of the size of the tumor although growth hormone appears to be reduced by only about 50% with continuation of abnormal growth hormone dynamics. Once again, in this group, withdrawal of the bromocryptine results re-evaluation of growth hormone values.

It seems apparent that this agent may contribute significantly to our capacity to manage these complex neuro-endocrine problems with a spirited cooperative interaction between endocrinologists and neurosurgeons.

9:30 a.m.

4. PRESENT PITUITARY PICTURE

Calvin Ezrin

(Discussion)

10:30 a.m.

Coffee Break

MODERATOR: James T. Robertson

10:50 a.m.

**5. REGIONAL METABOLISM OF RAT BRAIN
IN HYPOGLYCEMIA**

Robert A. Ratcheson, M.D.

James A. Ferrendelli, M.D.

Regional CNS levels of glucose reserves, glycolytic intermediates, and

high energy phosphate reserves were measured in insulin-treated, hypoglycemic rats and correlated with EEG activity. Intravenous administration of insulin to paralyzed, ventilated animals causes concomitant reduction of blood glucose levels and progressive abnormality and eventual loss of EEG activity. In all regions of brain examined glucose and glycogen levels decrease until they are depleted, and glucose-6-phosphate and fructose-1, 6-bisphosphate fall approximately 80%. Pyruvate levels decrease 50% in cerebral cortex and brain stem and a lesser amount in striatum, hippocampus, thalamus and cerebellum. Lactate levels fall 50-60% in all regions except cerebellum, where no change is observed. ATP and P-creatine levels remain normal until the EEG is isoelectric, and then decrease in all regions except cerebellum. Intravenous administration of glucose to hypoglycemic animals with isoelectric EEGs did not improve EEG activity, but increased blood and glucose levels, increased glycolytic flux in brain and elevated high energy phosphate reserves. The results of the present study lead to the following conclusions: (1) Hypoglycemia does not have a uniform effect on brain glucose and energy metabolism, and cerebellum seems to be relatively protected. (2) Depletion of energy reserves in brain is not responsible for the neurological dysfunction produced by hypoglycemia, but may contribute to its neuropathological effects. (3) Hypoglycemic encephalopathy is reversible (with glucose treatment) until EEG activity becomes isoelectric and/or CNS energy reserves diminish, and then it becomes irreversible.

11:10 a.m.

6. MODIFICATION OF VISUAL HALLUCINATIONS WITH THALAMIC STIMULATION

Joseph H. Goodman, M.D.
William E. Hunt, M.D.

Hallucinations resulting from damage to the visual system may occur as a release phenomenon. This is to be distinguished from seizure activity which is episodic and responsive to anti-convulsant medication. A patient with continuous non-formed hallucinations involving a hemianopic visual field is presented.

Stimulation of thalamic nuclei resulted in enhancement or suppression of the visual abnormality depending on current frequency, voltage and site of the stimulating electrode. Adequate suppression of the images was

obtained by permanent implantation of a monopolar electrode in the lateral geniculate body. Stimulation parameters and surgical technique are discussed.

11:30 a.m.

7. ACADEMY AWARD

**ELECTRONIC AND DENDRITIC SPINE ANALYSIS
OF CA3 AND DENTATE HIPPOCAMPAL NEURONS**

Dennis A. Turner

12:00 noon

LUNCHEON

MODERATOR: Joseph Ransohoff

1:00 p.m.

**8. POSITRON EMISSION TOMOGRAPHY
FOR DETECTION AND DYNAMIC ANALYSIS
OF BRAIN TUMORS**

William Feindel, M.D., Lucas Yamamoto, M.D.,
N. Arita, M.D., J-G Villemure, M.D.

The detection of brain tumours by positron emitting tracers was first proposed by Wrenn and his associates in 1951. The localization and lateralization of meningiomas and gliomas were demonstrated by this method with the technique of Sweet and Brownell and associates, using the positron emitting arsenic-74. But the rectilinear scanning gave limited topographic information with the technology then available. Over the past five years, the use of improved topographic imaging devices, with rapid scanning and computer analysis has provided a means of examining the early appearance of brain tumours, the spatial and temporal compartmentation of the administered positron tracers, and the nature of the blood-tumour barrier. An outline of the PET results in our series of tumours will be presented and the potential of PET for metabolic studies as well as for the kinetics of chemotherapeutic agents will be discussed.

1:20 p.m.

9. **STEREOTACTIC GUIDED CT-BIOPSY
OF DEEP BRAIN LESIONS**

Blaine S. Nashold, Jr., M.D., Dennis E. Bullard, M.D.,
Phillip Dubois, M.D., Sandra H. Bigner, M.D.

The ability to interface the traditional stereotactic frame with computerized tomography could potentially provide for better diagnosis and treatment of intracranial lesions. To further investigate this area, a lucite stereotactic frame has been adapted for use with wide aperture CT scanners, and a computer program designed which allows transformation of CT data into three-dimensional stereotactic coordinates. Utilizing this system, we have safely biopsied five patients with intracranial lesions. Three patients had deep-seated astrocytomas, one had a mycotic abscess and one had radionecrosis. Intraoperative evaluation of needle placement was possible in each case. Adequate histological preparations were obtained using thin needle aspirate staining techniques. No significant intraoperative or postoperative complications occurred. This system provided a safe means for obtaining tissue from intracranial lesions and has the potential for further therapeutic and diagnostic development.

1:40 p.m.

10. **SERUM ANTIBODIES IN PATIENTS
ACTIVELY IMMUNIZED FOR MALIGNANT GLIOMAS**

M.S. Mahaley, Jr., M.D.

Twenty patients with anaplastic gliomas have been actively immunized with a tissue-cultured glioma cell line. The initial immunization was accompanied by a BCG cell wall injection and subsequent monthly immunizations were performed with the cells alone. Each patient was also placed on levamisole immunostimulation. Monthly assays were carried out on the serum from each patient looking for anti-HLA antibodies as well as anti-glioma cell antibodies. The anti-glioma antibodies were further studied by absorption to determine the degree of antibody production against fetal bovine serum (part of the tissue culture media), against HLA antigens, and against glioma cells themselves. The results indicate that this active im-

munization protocol results in a rather large quantity of HLA antibodies in the majority of patients. Also there is a large production of antibody against fetal bovine serum from the tissue-cultured cell medium. In a smaller number of patients, there is a relatively small amount of antibody produced that seems to be distinctive for the glioma antigens. The methodology for these assays and the implication of these results will be discussed.

For future: monoclonal specific antibody

2:00 p.m.

11. IDENTIFICATION OF AN UNUSUAL AMINO ACID IN THE RIBOSOMES OF BRAIN AND DERIVATIVE TUMOURS

Wolff M. Kirsch, M.D.

John J. Van Buskirk

Certain drugs, such as the coumarin anticoagulants, specifically inhibit ribosomal RNA maturation in malignant glial tumor cells. In analyzing the effect of this drug, we have demonstrated in the ribosomes of a variety of tissues, to include human brain and malignant glial tumors, an unusual amino acid termed γ -carboxyglutamic acid (Gla). This acid occurs in the translational machinery of the cell, namely the ribosome, which is a complex component synthesizing protein. Thus, the blue and the H & E stained tumor section is a composite of a complex piece of cell machinery which converts data in the form of nucleotide sequences to polypeptides. The ribosome is a feasible therapeutic control point for malignant glial tumors, and there is evidence which indicates that the effect of certain steroids is exerted through this control point. This work has also led to the elucidation identification of a new amino acid, β -carboxyaspartic acid (Asa). This new amino acid, the first to be identified since 1974, has further implications in other important biological processes relative to neurologic disorders.

2:20 p.m.

12. INTRACEREBRAL MASSES IN PATIENTS WITH INTRACTABLE PARTIAL EPILEPSY

Dennis D. Spencer, M.D., Susan S. Spencer, M.D.,
Peter D. Williamson, M.D., Richard H. Mattson, M.D.

Approximately 10% of 1700 epileptic patients referred to the West

Haven V.A. Seizure Unit since 1972 have had medically refractory partial seizures of a degree sufficient to interfere significantly with their lives. Twenty-six of these patients (15%) were eventually found to harbor an intracranial mass. The masses included 17 neoplasms, two calcified AVM's, three arachnoid cysts, one granuloma, and three calcifications of unknown etiology. In these 26 patients, average age of onset of seizures was 18, ranging from 1 to 56 years. Seizures were of 9 years' average duration before discovery of the mass (range of 0.5 to 21 years). Five patients had only simple partial seizures; 13 had complex partial seizures, and 8 had both simple and complex partial seizures, with or without secondary generalization. 12 masses arose in the temporal lobe, 10 in the frontal lobe, 2 in the occipital lobe, and one each in the parietal lobe and insula. Nineteen patients had one or more previous evaluations for mass lesions before the diagnosis was made. The computerized axial tomographic scan was the only radiographic test that detected ten of the masses, and never failed to identify a mass demonstrated by other studies. Focal neurological findings or evidence of increased intracranial pressure developed prior to final diagnosis in ten patients, all of whom had neoplasms. The EEG was abnormal in 25 patients and correctly identified the cerebral lobe with the mass in nine. In the remainder, it was either normal (one patient), diffusely abnormal (13 patients), or falsely localizing (three patients).

Mean age 15 yr.

Of 23 patients who came to surgery, three were treated with biopsy and irradiation, nine with mass removal, and eleven with mass removal plus lobectomy for seizure control. Two of the nine patients treated with only mass removal have had no recurrent seizures. Two had no change in their seizure frequency. Good results (75% seizure frequency reduction) were obtained in most of the others. Ten of the 11 patients treated with lobectomy are seizure free and the remaining patient's seizure frequency was reduced by 90%.

Thus, of patients with partial seizures who are incompletely controlled with medical management approximately 15% are eventually found to harbor masses. 73% of our patients had one or more negative radiographic studies before the discovery of a mass, indicating the need for comprehensive periodic diagnostic evaluation, especially CT scanning, in patients with poorly controlled partial epilepsy. Treatment directed at the mass diminishes the patient's seizure frequency regardless of EEG localization, seizure type, or tumor locale. Seizure control is further improved as more of the mass and surrounding gliotic brain are removed.

Complex partial - temporal lobe, limbic system
Simple partial - focal/motor-sensor

2:40 p.m.

13. BRAIN TUMOR SURGICAL MORTALITY — FORTY YEARS' EXPERIENCE

Mortality and Morbidity — Significance and Improvement

James Greenwood, Jr., M.D.

Over the 40-year period, 1935 through 1975, the effect of improved technique is recorded (bipolar coagulation, radical removal, microsurgery), new diagnostic tests (C.A.T. scan, etc.), and dilution of cases and subspecialization (partial specialization)

Seven hundred and thirty-four brain tumor cases are divided into five-year periods showing the reduction of mortality from 21% in the initial five years to 2.4% for the last ten years. This was related to technique as above and by radical removal to 5.9% preceded by transient rise in the beginning from the average 10% to 14% but immediately followed with a drop to 5.9% showing that radical removal reduced the mortality and also the morbidity. With magnification and the microscope, our young men are doing excellent work. In my own group of cases which are reported here, we had 83 consecutive radical cases done without a death and with lowered morbidity including formerly inoperable cases. The last ten years in which I was doing surgery, I had 83 tumors with two deaths, giving a mortality of 2.4%. One of these was in poor condition and perhaps should not have been done and the other was a reoperated pituitary tumor who developed uncontrollable inappropriate ADH which might have been corrected by a procedure at the base of the stalk of the pituitary to produce diabetes insipidus.

The mortality in brain tumor surgery approaches 0 with proper selection of cases but will increase as formerly inoperable tumor cases are admitted to operation.

*44 yr. Chief of Methodist Hosp.
1935 -
no surgery
after 1976*

3:00 p.m.

Coffee Break

MODERATOR: George Ehni

3:20 p.m.

**14. AN APPROACH TO NONINVASIVE MEASUREMENT
OF INTRACRANIAL PRESSURE**

Robert G. Fisher, M.D., Ph.D.
John Semmlow, PhD.

The ability to noninvasively monitor intracranial pressure would be a valuable aid to the diagnosis and treatment of hydrocephalus and related disorders. An experiment is described which measures the acoustic properties of the skull in animals, under normal and intracranial pressures. Results show intracranial pressure strongly influences the acoustic response and at least one feature of the response is related to intracranial pressure in a consistent manner. Clinical studies are being conducted at present.

3:40 p.m.

**15. RELATIONSHIP BETWEEN VISUAL EVOKED
POTENTIALS AND INTRACRANIAL PRESSURE**

Clark Watts, M.D.
Donald York

The relationship between intracranial pressure (ICP) and latency of visual evoked potentials (VEP) was investigated in patients with cerebral edema following head trauma and in hydrocephalic patients with suspected shunt malfunction. The evoked potentials were elicited by flash stimulus delivered at 1/sec for 60-100 repetitions. The recording electrodes were placed at Cz or Oz according to the international 10-20 system. Signals were analyzed by a Nicolet CA 1000 clinical signal averager and entered into a microprocessor for further analysis. In the patients with cerebral edema, evoked potentials were analyzed simultaneously with intracranial pressure measurements obtained by a fiberoptic epidural monitoring system. Intracranial pressure was measured in the hydrocephalic patients by direct puncture of the ventricle or the shunt. A linear positive correlation of increase in latency of wave N2 (normal latency = ± 9.2 msec) of the VEP with elevations in ICP was observed. Grouped data from the initial 12 patients with head injuries and elevated pressures, resulting in 57 data points, allowed the construction of a normogram with a linear regression

coefficient of .84. From the normogram, prediction of intracranial pressure by measurement of VEP latencies was accurate in 15 patients with head trauma and 6 patients with hydrocephalus. Early experience suggests this correlation is probably not due to ventricular size or alterations in cerebral perfusion pressure.

4:00 p.m.

16. **THE NERVE ALLOGRAFT RESPONSE —
AN IMMUNOLOGICAL ASSESSMENT
OF REJECTION AND PRETREATMENT METHODS**

A.R. Hudson, S.E MacKinnon,
R.E. Falk, D. Hunter

The nerve allograft response was studied between closely and distantly related inbred strains of rats. Lewis rats (RT1^l) served as the recipient animals; three other strains provided the donor nerves; (Fischer rats (RT1^f) have minor histocompatibility differences, while Buffalo (RT1^b) and ACI (RT1^a) differ from the Lewis rat at the major histocompatibility locus.) 51 Cr release cytotoxicity assay was used to assess antigen recognition. This assay allows quantitation of the rejection phenomenon. An index of recognition (I.R.) was calculated:

$$IR = \frac{\text{counts per minute (c.p.m.)/gm. spleen test animal}}{\text{c.p.m./gm. spleen control animal}}$$

Thus, the index was inversely proportional to the degree of rejection. The results showed sensitization with ACI and Buffalo animals as early as day 8 (IR = .20 p ± .001). The difference between Fischer sensitized animals and control animals did not reach significance until day 80 (IR = .5 P ± 0.05). Cell-serum transfer studies showed that the nerve allograft response was mediated by both cellular and humoral factors. In the pretreatment study, ACI nerves were pretreated with irradiation (200r, 10,000r, 35,000r); lyophilization, freezing (-70°C.) and predegeneration (1, 3, 6, 12 wks.). Using the ⁵¹Cr release assay it was found that lyophilization or irradiation (35,000r) significantly decreased the antigenicity of the nerve allograft (IR = .23) P ± 0.001). The other methods of pretreatment did not alter the immunogenicity of the grafts (IR = .98).

4:20 p.m.

17. **INTRAVENTRICULAR FOETUS IN FETU**

F. Afshar, M.D., F.R.C.S.

A unique case of an intraventricular 14 cm. foetus in fetu is described in a 6-week old child presenting with enlarging head size. The entire intracranial foetus with clearly recognisable limbs, trunk, head and exomphalos was removed totally from the lateral and third ventricles with excellent recovery of the infant, who is now 18 months old. The foetus has been subjected to cytogenetical studies and tissue histology. Discussion of operative procedure, management, prognosis, embryology, as well as a review of this extremely rare pathology are discussed. A series of photographs of the diagnostic tests and operative and post-operative stages are presented.

MODERATOR: William Sweet

TUESDAY, NOVEMBER 3

8:30 a.m.

18. **CONTINUOUS LOW-DOSE INTRATHECAL MORPHINE
ADMINISTRATION IN THE TREATMENT
OF CHRONIC PAIN OF MALIGNANT ORIGIN**

Burton M. Onofrio, M.D.

Tony L. Yaksh, M.D.

Phillip G. Arnold, M.D.

A new approach to the control of pain of malignant origin is described. The merits of chronic intrathecal morphine infusion, as compared with the standard destructive neurosurgical procedures, include the preservation of motor and sensory modalities while achieving a pain-free state. Intrathecal morphine in the dose range needed to achieve a clinical effect on pain, unlike parenteral narcotics, does not lead to suppression of supraspinal centers.

8:50 a.m.

**19. PERCUTANEOUS EPIDURAL STIMULATION:
PREDICTABILITY OF PAIN RELIEF**

R.R. Tasker, M.D., T. Tsuda, M.D.

Although epidural spinal stimulation is a useful tool for treating chronic pain, neither the underlying mechanism of action nor the indications for patient selection are understood. In a review of 44 patients so treated with deafferentation pain associated with sensory loss caused by nerve or root lesions, or else with amputation stump or phantom pain, did better (67% initial relief) than those with deafferentation caused by cord lesions (38% initial relief), failed back syndrome (57% initial relief falling off rapidly with time), or pain of unknown cause (20% initial relief). Patients whose pain was most readily relieved by less than 100 mg IV sodium thiopental (morphine was ineffectual) and those with enhanced late components in the scalp evoked potential did best, effective stimulation, which required production of paraesthesiae in the painful area, being tightly correlated with suppression of these late components. Elapsed time since onset of pain did not affect the result.

It is suggested that *peripheral* deafferentation pain usually produces denervation neuronal hypersensitivity in the dorsal horn, an effect reflected in the evoked potential changes, which is known to be sensitive to the barbiturate-type drugs and presumably also to stimulation of these same affected dorsal horn areas. Deafferentation at the cord or brainstem level, and after some peripheral injuries, causes similar changes at the level of the mesencephalic tegmentum or medial thalamus, evidence for which has been obtained during stereotactic operations, an effect less sensitive to thiopental or to chronic spinal stimulation.

9:10 a.m.

**20. THE OPIATE ANTAGONIST NALOXONE REVERSES
THE ISCHEMIC ENUROLOGIC DEFICIT PRODUCED BY
UNILATERAL CAROTID LIGATION IN GERBILS**

Y. Hosobuchi, D. Baskin,
S. Woo, H.H. Loh

Because of anatomic anomalies commonly found in the circle of

Willis, homolateral ischemic brain damage and a consequent neurologic deficit (stroke) are produced in 30 to 50% of gerbils that undergo unilateral common carotid ligation. Under pentobarbital anesthesia (40mg/kg), the right common carotid artery was occluded in 70 adult gerbils. Within 4 hours, 23 of them developed stroke (2 died within 4 hours) (Group A); 47 gerbils that were ligated did not develop stroke (Group B). In 14 of 15 Group A gerbils, 1mg/kg of naloxone injected ip reversed the neurologic deficit within 2 to 5 minutes; 1 gerbil required an additional 1 mg/kg of naloxone to obtain reversal. Typically, reversal lasted 20 to 30 minutes, after which the neurologic deficit returned. However, reversal could be obtained repeatedly by injection of naloxone. 8 of 11 Group B gerbils injected i.p. with 15mg/kg of morphine developed naloxone-reversible stroke within 10 minutes. Naloxone-reversible stroke was produced in the other 3 gerbils by i.p. injection of an additional 15mg/kg of morphine. The stereoisomeric opiate agonist levorphanol (5mg/kg i.p.) was given to 10 Group B gerbils, 8 of which developed mild stroke symptoms that were reversed by naloxone. Dextrophan (15mg/kg), the enantiomer of levorphanol, was given i.p. to 10 Group B gerbils, none of which developed stroke. The delta receptor-specific enkephalin analogue Sandoz #FK33824 was given i.p. to 10 Group B gerbils at a dose of 15mg/kg, which is 80 times the mouse ED₀ analgesic dose. None developed stroke, although all were totally analgesic over 24 hours.

0.4 mg. IV - lasted for 30 minutes
Levo-Naloxone reversal of stroke induced by cerebral ischemia is a novel observation. Our results strongly suggest that the action of opiates and naloxone on this neurologic deficit are stereospecific and mu receptor specific. Recent findings of naloxone reversal of neurologic deficit caused by cerebral ischemia in a small group of patients are in accord with these findings in gerbils.

9:30 a.m.

21. EXPERIMENTAL TRANSCEREBRAL FISTULA FOR HYDROCEPHALUS: SECOND REPORT

Eldon L. Foltz, M.D., Jeffery Blanks

In the continuing study of hydrocephalus treatment by operative transcerebral fistula, ten normal dogs have been submitted to a 5-stage protocol to evaluate this treatment modality. The five-step protocol consisted of:

1. Proof of normal CSF system:
 - a. CSF pulse pressure study;
 - b. Isotope flow study including peri-neural olfactory flow;
2. Production of hydrocephalus by cisternal Kaolin;
3. Proof of hydrocephalus by:
 - a. CSF pressure study;
 - b. Isotope flow study including absent olfactory peri-neural CSF flow;
4. Transcerebral fistula operation to produce CSF flow from lateral ventricle to convexity subarachnoid space; special arachnoid seal technique;
5. Fistula patency proof by:
 - a. CSF pressure study;
 - b. Isotope CSF flow via fistula imaging and peri-neural olfactory flow reestablished;
 - c. Vital dye CSF marker pre-postmortem;
 - d. Histology postmortem.

Five animals completed the protocol. All five had functioning fistulas and relief of hydrocephalus (clinical and morphologic and physiologic) proved up to three months postoperative. The operative technique including gelfilm pads to seal the arachnoid will be emphasized. Clinical application seems imminent.

9:50 a.m.

22. REOPERATION FOR FAILED ANEURYSM SURGERY

C.G. Drake, M.D., S.J. Peerless, M.D.

Twenty years ago we recognized the seriousness of incomplete obliteration of cerebral aneurysms and have made an effort to "redo" as many of our failures as possible; as well, we have had the opportunity to attempt definitive retreatment of a number of referred cases, ~~67~~⁷¹ in all, 16 from our Unit and 51 referred.

They fall into four groups:

1. Clipping abandoned (20)
2. Clipping abandoned for reinforcement incompletely with gauze or plastic (15) / 17
3. "Slipped" or imperfectly applied clip (22) 24
4. Failure of carotid ligation (esp. common) to control the aneurysm (10)

Anterior 41
Posterior 31

24 giant aneurysms

Ethicon Co. will provide
glue for "life-death" situation

The most common reason for reoperation was simply an attempt to improve an imperfect clip (34 cases) but recurrent SAH (16 cases), mass effect (16 cases) and the revelation of a long-delayed follow-up angiogram (5 cases) accounted for the remainder. The timing of reoperation was highly varied but tended to be shortest after abandoning clipping or after early angiograms revealed a "slipped" or imperfect clip. Longer delays occurred after gauze or plastic coating suggesting that the surgeons had initial faith in this form of restraint. Only 2 of the 10 reoperated after failed carotid ligation were seen within a year, the rest varying from 4 to 16 years, suggesting some initial restraint of the sac. Eight recurred after common carotid occlusion but 2 after internal carotid occlusion, although in 1 the internal carotid was deliberately narrowed only to a 90% stenosis.

Treatment was most straightforward when done within a few days or a week or two from the original unsuccessful procedure. Longer delays caused some heavy scarring, although unpredictably. Usually, however, the clip, gauze or plastic were out on the dome and/or waist of the sac leaving virgin vessels and tissues around the neck.

Treatment was effective in completing the obliteration of the aneurysm in all but 6 cases of which all but 1 were giant aneurysms. There were 8 poor results but only 3 of these were worsened by treatment. Five postoperative deaths occurred, 1 rebleed from an imperfect clip, 1 from arterial spasm, 1 after pulmonary embolus but 2 from enigmatic thrombosis of the carotid artery opposite to that harbouring the treated aneurysm.

55 of 71 were excellent results
5 dead - various reasons

10:00 a.m.

Coffee Break

10:30 a.m.

23.

PRESIDENTIAL ADDRESS

Joseph Ransohoff, II, M.D.

WEDNESDAY, NOVEMBER 4

8:30 a.m.

**24. CEREBRAL VASCULAR MALFORMATIONS:
CASES FOR OPERATION AND RESULTS**

Charles B. Wilson, Neil A. Martin

One hundred consecutive cerebral vascular malformations (AVM's) treated by operation have been reviewed. Lesions managed by interruption of afferent vessels without excision were excluded.

The majority were arteriovenous fistulas that either bled or produced ischemic manifestations by shunting, and these lesions were identified by angiography whether large or small. A minority of pathologically documented malformations were angiographically occult cavernous or racemose angiomas. Not included in this series were venous angiomas, because, while readily identified by angiography, with rare exceptions they are benign.

The indications for staged operations will be given in addition to results and complications. With proper selection of cases, outcome after operative treatment is clearly superior to the natural history of these lesions untreated.

8:50 a.m.

**25. SURGICAL EXPERIENCE WITH
ARTERIOVENOUS MALFORMATIONS OF THE
BRAIN AND SPINAL CORD**

Bennett M. Stein, M.D.

One hundred operated cases of arteriovenous malformation of the brain and twenty operated spinal AVM's form the basis of this review.

Of the cerebral arteriovenous malformations, the ones offering the greatest challenge are those located deep in the region of the third ventricle or in the medial side of the temporal and parietal regions. In these instances, the surgeon finds himself looking around corners or working through transcortical incisions via normal cortex and white matter to encounter the AVM on its "blind side." I am presenting a modest experience with lesions located in such areas. To be reviewed is the preliminary evaluation of the lesion and

the surgical approach and the result of these lesions. The operative mortality of cerebral AVM's is under 2% and the morbidity under 6%.

Spinal arteriovenous malformations present different technical problems. It has been our feeling that although spinal angiography may be helpful in visualizing the arteriovenous malformation that it has been more often misleading in determining the exact location of the malformation. The frequent intramedullary component of the spinal AVM's will be discussed in some detail as this aspect presents a special problem.

The surgical technique in the removal of lesions with intramedullary components and results in 5 number of cases will be presented.

9:10 a.m.

26. Degenerative spondylo with intact arch **INTACT ARCH SPONDYLOLESTHESIS** *1854 first reported*

Eben Alexander, Jr., M.D.,
C.H. Davis, Jr., M.D.,
David Kelly, Jr. M.D.

The experience gained in the past 8 years in finding and operating on patients with severe back and leg pain due to intact arch spondylolesthesis has been one the most gratifying to patients and surgeons alike. The analysis of 35 cases, the small details of diagnosis and of operative technic are most important and are illustrated in some original drawings.

9:30 a.m.

27. THE CHRONIC CENTRAL CORD SYNDROME

Deborah Hyde-Rowan, M.D.
Teresa Ruch, M.D.
Jerald S. Brodkey, M.D.

Recent experience with the acute central cord syndrome following trauma has shown that there is often an initial period of neurological improvement lasting four to six weeks. Subsequently the neurological status will often remain static unless the almost invariable anterior bar compressing the cervical spinal cord is removed. Cases of the chronic central cord syndrome caused by cervical spondylosis will be presented. The onset is

insidious and gradual and not associated with an episode of trauma. The anterior compressing bars in our cases have been very large leading in one case to a complete block on myelography. The location of the bars has been mid-cervical well above the lower cervical segments innervating the hands. As in many cases of the acute syndrome, there was immediate postoperative improvement within 24 hours after operation in several of the cases of the chronic syndrome. The suggestion is made that the mechanism for neurologic dysfunction in both the acute and chronic central cord syndromes (after the acute effects of trauma subside) is the same — namely vascular.

9:50 a.m.

28. SUBDURAL AND EPIDURAL EMPYEMA IN CHILDHOOD

W.C. Hendrick, H. Smith,
R.P. Humphreys, H.J. Hoffman

This paper represents the total experience with subdural and epidural empyema at The Hospital for Sick Children during the period of 1954 and 1981. There were 31 cases encountered, 22 of which were subdural, 6 epidural and 3 with combined subdural and epidural sites.

All epidural empyema occurred in patients between the ages of 12 and 16 years. The subdural empyemas occurred between the age group of 6 to 12 years and 12 to 16 years. The subdural empyemas develop from any sources, although the paranasal sinuses, the ear and mastoid predominated. Five patients were infected following craniotomy or shunt procedures. The epidural empyemas developed from infections in the paranasal sinus and middle ear in almost all cases. The combined infections in the subdural and epidural spaces occurred from paranasal sinuses and in one instance following craniotomy. The bacterial flora varied, but were common to both sites.

Presentation in the subdural empyemas was with a focal seizure in the older group. The older group at presentation were seriously ill and also had more focal deficits. Epidural empyema showed few focal deficits and delayed diagnosis occurred in most cases. Clinical diagnosis, investigative procedures, and treatment will be discussed in detail.

*Zannister review
of literature
is recommended*

10:10 a.m.

Coffee Break

MODERATOR: S.J. Peerless

10:30 a.m.

**29. VERTEBRAL TO COMMON CAROTID ARTERY
TRANSPOSITION IN THE TREATMENT
OF VERTEBROBASILAR INSUFFICIENCY**

Jim L. Story, Willis E. Brown, Jr., Moustapha Abou-Samra

Since 1977 we have performed vertebral artery to common carotid artery transposition in 15 patients with vertebral-basilar artery insufficiency (VBI). The VBI syndrome resulted from symptomatic subclavian steal in six patients and from stenosis of a vertebral artery at its origin in nine patients. (Of these 9 patients with symptomatic stenosis at the origin of a vertebral artery, 7 had total occlusion of the contralateral vertebral artery. The remaining 2 patients in this group had a small contralateral vertebral artery with advanced atherosclerotic disease in the distal segments, just proximal to the vertebral-basilar junction). Two patients with symptomatic subclavian steal and 4 patients with vertebral artery stenosis required simultaneous ipsilateral carotid endarterectomy.

In 8 of the 15 patients, direct anastomosis of the vertebral artery to the common carotid artery was performed. Seven of the 15 patients required an interposition polytetrafluoroethylene tube graft.

Electromagnetic blood flow determination was made intraoperatively in a number of patients in this series. These studies in patients with subclavian steal syndrome consistently demonstrated not only reversal of flow in the vertebral artery, but remarkable net increase in vertebral flow. In the patients with vertebral artery stenosis, the intraoperative blood flow data documented the considerable augmentation of vertebral blood flow that results when a stenotic vertebral artery is transposed to the common carotid artery.

Postoperative angiograms revealed patent anastomoses and excellent vertebral artery flow in all patients.

There was one death in the series. It resulted from an acute myocardial infarction on the second postoperative day. Although there was no neurological deterioration related to the surgical procedure in this patient, this death

from acute myocardial infarction does demonstrate the challenge we accept when undertaking the care of patients with widespread atherosclerotic disease.

With regard to clinical outcome, the results of this series have been gratifying. The postoperative courses in these patients have been remarkably smooth. One patient has an occasional minor episode of vertigo; however, all of the remaining patients are asymptomatic, alive, and well.

10:50 a.m.

30. **SURGICAL CORRECTION OF LESIONS
AFFECTING THE VERTEBRAL ARTERY
FOR VERTEBRAL BASILAR INSUFFICIENCY**

James I. Ausman, M.D., Ph.D., Fernando G. Diaz, M.D., Ph.D.,
R.A. de los Reyes, M.D., Hooshang Pak, M.D.,
Jeffrey Pearce, M.D., Behrat Mehta, M.D.

Vertebral basilar insufficiency can be caused by stenosis or occlusion of the proximal subclavian artery or the vertebral artery at any level from its origin to the vertebral basilar junction. The cases of 23 patients presenting with vertebral basilar insufficiency and lesions in various segments of the vertebral artery will be reviewed. *Three patients had proximal subclavian occlusion with subclavian steal*: two were treated with vertebral to carotid transposition, one, with vertebral artery ligation. *Eighteen patients had vertebral origin stenosis*; one, was treated by endarterectomy, one, by vertebral to subclavian and another by vertebral to thyrocervical anastomosis. Twelve underwent vertebral to carotid transpositions (VCT). There was one failed operation early in the series because of a low lying vertebral origin. Two patients underwent simultaneous ipsilateral VCT and carotid endarterectomy for ipsilateral carotid and vertebral lesions. All patients were asymptomatic or improved postoperatively. There was no mortality and only transient morbidity which included ipsilateral Horner's syndrome in four and elevated ipsilateral diaphragm in four. No intravascular shunts were used. Of the 19 patients with vertebral origin surgery, 18 demonstrated postoperative patency.

Two patients had intracranial vertebral endarterectomy, one of which was occluded postoperatively.

A discussion of the preferred approaches and techniques for lesions in each of these areas will be given. The series will be compared with non-surgical alternatives which are presently available.

11:10 a.m.

**31. EFFECT OF STA-MCA BYPASS, TEMPORALIS
MUSCLE GRAFT AND OMENTAL TRANSPOSITION
ON CORTICAL BLOOD FLOW
IN MCA OCCLUSION**

James M. Blue, M.D.

The above revascularization procedures were carried out on 24 mongrel dogs with 8 animals serving as controls. Cortical blood flow (CoBF) was then measured using the microsphere technique before and after retro-orbital middle cerebral artery (MCA) occlusion, and again at 4-18 days post-occlusion (recovery period).

In the control group (no revascularization), all animals circled post-occlusion, 3 of 8 animals died, and cortical infarction was evident in all gross brains. CoBF ipsilateral to occlusion was 69 cc/100g/min pre-occlusion, dropping by 49% post-occlusion, and recovering by 55% at 4-18 days in surviving animals. In the STA-MCA bypass group, no animals circled post-occlusion, none died, and cortical infarction was seen in only 2 brains. CoBF in the ipsilateral MCA distribution was 64 cc/100g/min pre-occlusion, dropping by 59% post-occlusion, but recovering 113% over post-occlusion values. Animals with temporalis muscle grafts had a higher incidence of post-occlusion circling (5 of 8 dogs) with 3 deaths and gross cortical infarction in 6 brains. CoBF prior to occlusion averaged 50 cc/100g/min, with a drop of 50% post-occlusion. However, in surviving animals, CoBF recovered by 173%. In animals with an omental transposition, circling was present in 3 of 8, gross infarction in 5 of 8 brains, but there were no deaths. Pre-occlusion CoBF was 64 cc/100g/min and dropped by 48%, with only 50% recovery, both values no better than controls. The above findings suggest that no revascularization procedure prevents the abrupt drop in CoBF following MCA occlusion. However, STA-MCA bypass affords the best protection against immediate infarction and death with excellent potential of CBF recovery.

11:30 a.m.

**32. COMPUTERIZED EEG MONITORING
 DURING CEREBRO-VASCULAR SURGERY**

J.W. Correll, I.J. Rampil,
J.A. Holzer, N.T. Smith

Monitoring with the EEG is a direct, sensitive and clinically useful measure of the adequacy of cerebral blood flow. However, the classical approach to EEG monitoring is cumbersome and expensive, requiring specially trained personnel.

An inexpensive compact device for analyzing and monitoring the EEG, making use of computer technology, has been developed (Fleming & Smith, *Anesthesiology*, 1979 50:456-460). This involves spectral analysis which consists of the mathematical determination of the frequency distribution of energy in the EEG. The computer preprocessing produces a highly compressed, legible and convenient display on the standard strip chart recorder (or television screen). Operation of the computer and interpretation can be learned in less than 1 hour, allowing the anesthesiologist and surgeon to easily incorporate the EEG into other routine physiological monitoring.

We have used this method to monitor more than 70 patients (over 60 were carotid endarterectomies). In some cases conventional EEG was obtained simultaneously for comparison with the computerized technique. Six patients were observed to have a post-operative deficit (transient in 5). All 6 showed striking EEG changes which persisted for more than 10 minutes. Many EEG events of less than 10 minutes duration were observed. Temporal correlation of EEG events with the clinical situation is revealing and will be analyzed.

11:50 a.m.

**33. POSITIVE END EXPIRATORY PRESSURE —
 AN EFFECTIVE PREVENTATIVE OF AIR EMBOLISM
 IN NEUROSURGICAL PROCEDURES**

Richard A.R. Fraser, M.D., Alan Van Poznak, M.D.,
Rand Voorhies, M.D.

Venous air embolism may occur in any operation in which there exists a

sufficient negative pressure gradient from the site of surgery to the heart. Neurosurgical procedures frequently include such a hemodynamic gradient. As a consequence, air embolism has been reported during craniotomies, cervical laminectomies and most frequently during posterior fossa procedures. Intracardiac catheter placement for air withdrawal combined with a precordial Doppler monitor has been recommended by several authors as necessary in these procedures. Air embolism under such monitoring conditions has been reported in 21 to 60% of patients undergoing cervical laminectomy and transsphenoidal surgery, with the highest frequency described in posterior fossa procedures.

We wish to report a simple, effective but hitherto unreported maneuver (in this setting) which allows for any patient position and eliminates the hemodynamic gradient that places patients at risk for air embolism. Positive end expiratory pressure (PEEP) has been routinely employed in all patients in this institution where surgery has been employed in the sitting position for the past 24 months. Forty-one patients, 11 cervical laminectomies and 30 suboccipital craniectomies are included. PEEP is instituted at the beginning of surgery with controlled ventilation. PEEP levels from 5 to 15 cm of water have been utilized. Atrial catheters were not placed in any patient. The appropriate PEEP level was determined by increasing PEEP in 5 cm increments till venous bleeding was just observed, usually from emissary veins, or mild epidural venous distention in cervical laminectomies. In no case did bleeding interfere with the surgery. In one instance Doppler monitoring suggested an air embolus. Increasing PEEP and covering the wound was quickly followed by normal Doppler recording. No instance of significant circulatory changes was observed in this entire group.

We believe this maneuver eliminates the risk of air embolism in neurosurgical procedures. Simultaneously it eliminates the need for an atrial catheter and the attendant risks of such a procedure. (A complication rate of 0.4-9.9% has been reported with subclavian vein CVP line placement.) Finally, PEEP allows unrestricted positioning of the patient and retains the anatomical and technical advantages of the sitting position or its variants by providing the hemodynamic equivalent of the supine position. A special advantage occurs in the rare circumstance when a major cranial venous sinus must be opened.

1981

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DENNIS A. TURNER, M.D.
Department of Neurosurgery
Washington University School of Medicine
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**“Electrotonic and Dendritic Spine Analysis of
CA3 and Dentate Hippocampal Neurons”**

HONORABLE MENTION

ACADEMY AWARD

1981

Frederick A. Lenz
University of Toronto
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**“The Effect of Cortical Lesions on Reflex Responses
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1980

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✓ JAMES GREENWOOD, JR. 1117 Hermann Professional Bldg. 6410 Fannin Street Houston, Texas 77025	(MARY)	1952
WALLACE B. HAMBY 3001 N.E. 47th Court Fort Lauderdale, Florida 33308	(ELEANOR)	1938
HANNIBAL HAMLIN 270 Benefit Street Providence, Rhode Island 02903	(MARGARET)	1941
JESS D. HERRMANN Post Office Box 135 Mountain Pine, Arkansas 71956	(MARY JO)	1948
WILLIAM S. KEITH 55 St. Leonards Crescent Toronto, Ontario, Canada M4N 3 A 7	(ELEANOR)	Founder
JOHN J. LOWREY P.O. Box 4302 Kawaihae, Hawaii 96743	(CATHERINE "Katy")	1965
GEORGE L. MALTBY 470 Black Point Road Scarsborough, Maine 04074	(ISABELLA "Sim")	1942
FRANK MAYFIELD, M.D. 506 Oak Street Cincinnati, Ohio 45219	(QUEENEE)	Founder

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

✓ WILLIAM F. MEACHAM, M.D. (ALICE) 1952
Vanderbilt University Hosp.
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. (MADALINE) 1946
Duke University Med. Ctr.
Durham, North Carolina 27706

J. LAWRENCE POOL (ANGELINE) 1940
Box 31
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. 20 #100
Portland, Oregon 97209

THEODORE B. RASMUSSEN, M.D. (CATHERINE) 1947
Montreal Neurological Instit.
3801 University Street
Montreal 2, Quebec, Canada

	R.C.L. ROBERTSON (MARJORIE)	1946
	2210 Maroneal Blvd. Shamrock Professional Bldg. Suite 404 Houston, Texas 77025	
	STUART N. ROWE (ELVA)	1938
	302 Iroquois Bldg. 3600 Forbes Street Pittsburgh, Pennsylvania 15213	
	HENRY G. SCHWARTZ (REEDIE)	1942
	Barnes Hospital Plaza Division of Neurological Surgery St. Louis, Missouri 63110	
✓	WILLIAM B. SCOVILLE (HELEN)	1944
	85 Jefferson Street Hartford, Connecticut 06106	
	WILLIAM H. SWEET (ELIZABETH)	1950
	1 Longfellow Place Suite 201 Boston, Massachusetts 02114	
✓	C. HUNTER SHELDEN (ELIZABETH)	1941
	734 Fairmont Avenue Pasadena, California 91105	
	HOMER S. SWANSON (LaMYRA)	1949
	1951 Mount Paran Rd., N.W. Atlanta, Georgia 30327	
	JOHN TYTUS (VIRGINIA "Gina")	1967
	Mason Clinic Seattle, Washington 98107	
	ALFRED UIHLEIN (IONE)	1950
	200 Frist Street S.W. Rochester, Minnesota 55901	

WARD, Arthur Pres '78

- A. EARL WALKER (TERRY) 1938
Johns Hopkins Hospital
Division of Neurological Surgery
601 North Broadway
Baltimore, Maryland 21205
- 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
85 Jefferson Street
Hartford, Connecticut 06106
- BARNES WOODHALL (FRANCES) 1941
Duke University Medical Center
Durham, North Carolina 27706

Corresponding Members

ELECTED

- JEAN BRIHAYE 1975
1 Rue Heger-Bordet
B-1000 Brussels, Belgium
- KARL AUGUST BUSHE 1972
Neurochirurgischen Klinik
D-8700 Wurzburg
Josef-Schneider-Strasse 11
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- 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
 Ave. Quintana 474 80 A
 Buenos Aires, Argentina
- GIUSEPPE DALLE ORE 1970
 Dipartimento Di Neurochirurgia
 Ospedale Maggiore 37100
 Verona, Italy
- HANS ERICH DIEMATH 1970
 Prim. Univ. Doz.
 Neurochir. Abt. d. Landersnervenklink
 Salzburg, 5020, Austria
- JOHN GILLINGHAM 1962
 Boraston House 22, Ravelson Dykes Road
 Edinburgh, Scotland EH43PB
- JAIME G. GOMEZ 1975
 Transversal 4 No. 42-00
 Conmutador 2-32 4070
 Bogota 8, Columbia, South America
- JOHN HANKINSON 1973
 Department of Neurological Surgery
 Newcastle General Hospital
 Newcastle-Upon-Tyne 4
 England
- SHOZO ISHII 1975
 Department of Neurosurgery
 Juntendo Medical College
 Tokyo, Japan

- RICHARD JOHNSON** 1974
 Department of Neurological Surgery
 Royal Infirmary
 Manchester, England
- KATSUTOSHI KITAMURA** 1970
 University Kyushu Hospital
 Faculty of Medicine
 Fukuoka, Japan
- KRISTIAN KRISTIANSEN** 1962
 Oslo Kommune
 Ullval Sykehus
 Oslo, Norway
- LAURI LAITINEN** 1971
 Department of Neurosurgery
 5016 Haukeland Sykehus
 Norway
- WILLIAM LUYENDIJK** 1973
 Pr Bernhardlaan 60
 Oegstgeest, Netherlands
- FRANK MARGUTH** 1978
 Director, Department of Neurochirurgischen
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 Marchioninistrasse 15
 8000 München 70, West Germany
- RAUL MARINO, JR.** 1977
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- HELMUT PENZHOLZ**
 Director Neurochirurgischen
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- HANS-WERNER PIA** 1978
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 Zentrums für Neurochirurgie
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 Klinikstr 37
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- B. RAMMAMURTHI** 1966
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 Madras 4, India
- KURT SCHURMANN** 1978
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 Neurochirurg
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 Langenbeckstr 1
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- CHARAS SUWANWELA** 1972
 Chulalongkorn Hospital
 Medical School
 Bangkok, Thailand
- KJELD VAERNET** 1970
 Righospitalets Neurokirurgis
 Tagensvfj 18, 2200
 Copenhagen, Denmark
- SIDNEY WATKINS** 1975
 The London Hospital
 Whitechapel, London E 1
 England
- GAZI YASARGIL** 1975
 Neurochirurgische
 Universitätsklinik
 Kantonsspital
 8000 Zurich, Switzerland

Active Members

ELECTED

- ✓ JAMES I. AUSMAN (CAROLYN) 1978
 Henry Ford Hospital
 2799 West Grand Blvd.
 Detroit, Michigan 48202 *Program 1982*
- GILLES BERTRAND (LOUISE) 1967
 Montreal Neurological Inst.
 3801 University Street
 Montreal, Quebec, Canada
- ✓ WILLIAM A. BUCHHEIT, M.D. (HELEN) 1980
 3401 North Broad Street
 Philadelphia, Pennsylvania 19140
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 2065 Adelbert Rd.
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- ✓ BARTON A. BROWN (MARTHA) 1968
 2001 Union Street
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- ✓ SHELLEY CHOU (JOLENE) 1974
 University of Minn. Med. Ctr.
 Minneapolis, Minnesota 55455
- ✓ GALE G. CLARK (MARIAN) 1970 *Round Robin*
 University of California Medical Center
 San Francisco, California 94143
- W. KEMP CLARK (FERN) ~~1974~~ 1970
 5323 Harry Hines Blvd.
 Dallas, Texas 75235
- ✓ WILLIAM F. COLLINS, JR. (GWEN) 1963
 Yale Univ. School of Med.
 333 Cedar Street
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EDWARD S. CONNOLLY (ELISE) 1973
Ochsner Clinic
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✓ JAMES W. CORRELL (CYNTHIA) 1966
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✓ COURTLAND H. DAVIS, JR. (MARILYN) 1967
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Winston-Salem, North Carolina 27103 *Vice-P-'81*

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STEWART B. DUNSKER (ELLEN)d 1975
Mayfield Neurological Institute
506 Oak Street
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The Neurosurgical Group of Houston, Assoc.
6560 Fannin St., #1250, Scurlock Tower
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- ✓ WILLIAM H. FEINDEL (FAITH) 1959
 Montreal Neurological Institute
 3801 University Street
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Pres. '76
- ROBERT G. FISHER (CONSTANCE) 1956
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 Piscataway, New Jersey 08854
Vice P. '78
- ✓ EUGENE FLAMM (SUSAN) 1979
 N.Y.U. Medical Center
 550 First Avenue
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Acad Award '82
- ✓ ELDON L. FOLTZ (CATHERINE) 1960
 Division of Neurosurgery
 Univ. of Cal. School of Medicine
 Irvine, California 92664
- ✓ RICHARD A.R. FRASER 1976
 525 East 68th Street
 New York, New York 10021
Exec Comm. '82
- ✓ JOHN T. GARNER (BARBARA) 1971
 1127 East Green Street
 Pasadena, California 91106
*Sec. 80-83
Treas 77-80*
- ✓ HENRY GARRETSON (MARIANNA) 1973
 Health Sciences Center
 University of Louisville
 Louisville, Kentucky
- SIDNEY GOLDRING (LOIS) 1964
 Barnes Hospital Plaza
 Division of Neurosurgery
 St. Louis, Missouri 63110
President, '83
- PHILIP D. GORDY (SILVIA) 1968
 1727 East 2nd Street
 Casper, Wyoming 92601

- ✓ JOHN W. HANBERY (SHIRLEY) 1959
 Division of Neurosurgery
 Stanford Medical Center
 Palo Alto, California 94304
- ✓ GRIFFITH R. HARSH, III, M.D. (CRAIG) 1980
 Univ. of Alabama Med. 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, jrm. 1502
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 Department of Neurosurgery
 University of Michigan
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 710 West 168th Street
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 St. Michaels Hospital
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- ✓ WILLIAM E. HUNT (CHARLOTTE) 1970
 Division of Neurological Surgery
 University Hospital
 410 West 10th Avenue
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✓ ELLIS B. KEENER (ANN) 1978
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DAVID KELLY (SALLY) 1975
Bowman-Gray School of Medicine
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WILLIAM A. KELLY 1977
✓ University of Washington
School of Medicine
Seattle, Washington 98195

GLENN W. KINDT (CHARLOTTE) 1977
University of Michigan
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ROBERT B. KING (MOLLY) 1958
✓ University Hospital *Pres. '79*
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✓ University of Colorado Medical Center
4200 East 9th Avenue
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Louisiana State University Medical Center
1542 Tulane Avenue
New Orleans, Louisiana 70012 *Neurology Implants Rep*

✓ ROBERT S. KNIGHTON (LOUISE) 1966
9388 Avenida San Timeteo *Vice P '77*
Cherry Valley, California 92223

RICHARD S. KRAMER (ROBIN) 1978
Duke Hospital *Acad Award → '83*
Durham, North Carolina 27710

ROBERT L. McLAURIN 1955
Division of Neurosurgery
Cincinnati General Hospital
Cincinnati, Ohio 45229

JOHN F. MULLAN, M.D. (VIVIAN) 1963
✓ Univ. of Chicago Clinics *Vice Pres 182*
Department of Neurosurgery
950 East 59th Street
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BLAINE S. WASHOLD, JR. (IRENE) 1967
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Massachusetts General Hospital
Division of Neurological Surgery
Boston, Massachusetts 02114

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

✓ RUSSEL H. PATTERSON, JR. (JULIET) 1971
525 East 68th Street *Sec 74-76*
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Terminal A
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London, Ontario Canada N6A 5A5

✓ PHANOR L. PEROT, JR. (ELIZABETH) 1970
Department of Neurosurgery
Medical University of South Carolina
171 Ashley Avenue
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Info 74-76

✓ BYRON C. PEVEHOUSE (LUCY) 1967
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✓ ROBERT W. PORTER (AUBREY DEAN) 1962
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✓ AIDEN A. RANEY (MARY) 1946
125 North Las Palmas
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✓ JOSEPH RANSOHOFF, II (RITA) 1965
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550 First Avenue
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HUGO RIZZOLI (HELEN) 1973
2150 Penn Avenue, NW
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✓ THEODORE S. ROBERTS (JOAN) 1976
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✓ THEODORE KURZE 1967
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✓ RAEBURN C. LLEWELLYN (CARMEN) 1963
9661 Lake Forest Blvd.
Suite 350
New Orleans, Louisiana 70127

✓ WILLIAM M. LOUGHEED (GRACE ELEANOR) 1962
Medical Arts Bldg., Suite 430
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713 East Genessee Street
Syracuse, New York 13210

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Washington, D.C. 20007

✓ ERNEST W. MACK (ROBERTA) 1956
505 S. Arlington Avenue
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✓ M. STEPHEN MAHALEY, JR. (JANE) 1972
Univ. of N.C., Room 229H
148 Clinical Sciences Bldg.
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Exec Comm '81

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

✓	JAMES T. ROBERTSON Department of Neurosurgery UTCHS, 956 Court Avenue Memphis, Tennessee 38163	(VALERIA)	1971
	RICHARD C. SCHNEIDER C5135 Out-Patient Building University Hospital Ann Arbor' Michigan 48104	(MADELEINE)	1970
	JAMES C. SIMMONS 920 Madison Avenue Memphis, Tennessee 38103	(VANITA)	1975
✓	BENNETT M. STEIN New England Medical Center Hosp. 171 Harrison Avenue Boston, Massachusetts 02111	(DOREEN)	1970
✓	JIM L. STORY, M.D. 7703 Floyd Curl Drive San Antonio, Texas 78229	(JOANNE)	1972
✓	THORALF M. SUNDT, JR. 200 1st Street, S.W. Rochester, Minnesota 55901	(LOIS)	1971
✓	ANTHONY F. SUSEN 3600 Forbes Avenue Pittsburgh, Pennsylvania 15213	(PHYLLIS)	1965
✓	RONALD R. TASKER Toronto General Hospital Room 121, U.W. Toronto, Ontario, Canada	(MARY)	1971
✓	JOHN TEW, JR. 506 Oak Street Cincinnati, Ohio 45219	(SUSAN)	1973

*Program
1984*

- GEORGE T. TINDALL (SUZIE) 1968
 Emory Univ. School of Medicine
 Division of Neurosurgery
 1365 Clifton Road, N.E.
 Atlanta, Georgia 30322
- ✓ JOHN C. VAN GILDER, M.D. (KERSTIN) 1980
 Univ. of Iowa Hospital
 Iowa City, Iowa 55242
- ✓ ARTHUR A. WARD, JR. (JANET) 1953
 Department of Neurological Surgery
 Univ. of Washington Hospital
 Seattle, Washington 98105
- ✓ CLARK WATTS (PATTY) 1975
 807 Stadium Road
 Suite N521
 Columbia, Missouri 65212
- 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 1973
 Duke University Medical Center
 Box 3807
 Durham, North Carolina 27710

CHARLES B. WILSON (ROBERTA) 1966
Department of Neurological Surgery
U. of California Medical Center
Third and Parnassus
San Francisco, California 94122

✓ FRANK WRENN (BETTY) 1973
123 Mallard Street
Greenville, South Carolina 29601

DAVID YASHON (MYRNA) 1972
410 W. 10th Ave. N. 911
Columbus, Ohio 43210

✓ NICHOLAS T. ZERVAS (THALIA) 1972
Massachusetts General Hospital
Department of Neurosurgery
Boston, Massachusetts 02214
Program 1983

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	1949
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. HENRY L. HEYL (Senior) Hanover, New Hampshire	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. SAMUEL R. SNODGRASS (Senior) Nashville, Indiana	8/08/75	1939
DR. C. WILLIAM STEWART (Corresponding) Montreal, Quebec, Canada		1948
DR. GLEN SPURLING (Honorary) La Jolla, California	2/07/68	1942
DR. HENDRIK SVIEN (Active) Rochester, Minnesota	6/29/72	1957

**AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
1981 ANNUAL MEETING**

EVALUATION

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

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

- Excellent
- Good
- Poor

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

- Too much review of old knowledge?
- Too simple or elementary?
- Too complex or abstruse?
- Of little practical value?

(3) Did the speakers aim their talks:

- Too low
- Too high
- Just about right

SCIENTIFIC PROGRAM

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

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

Saturday's Sessions Excellent _____ Good _____ Poor _____
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SOCIAL PROGRAM

Comments _____

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

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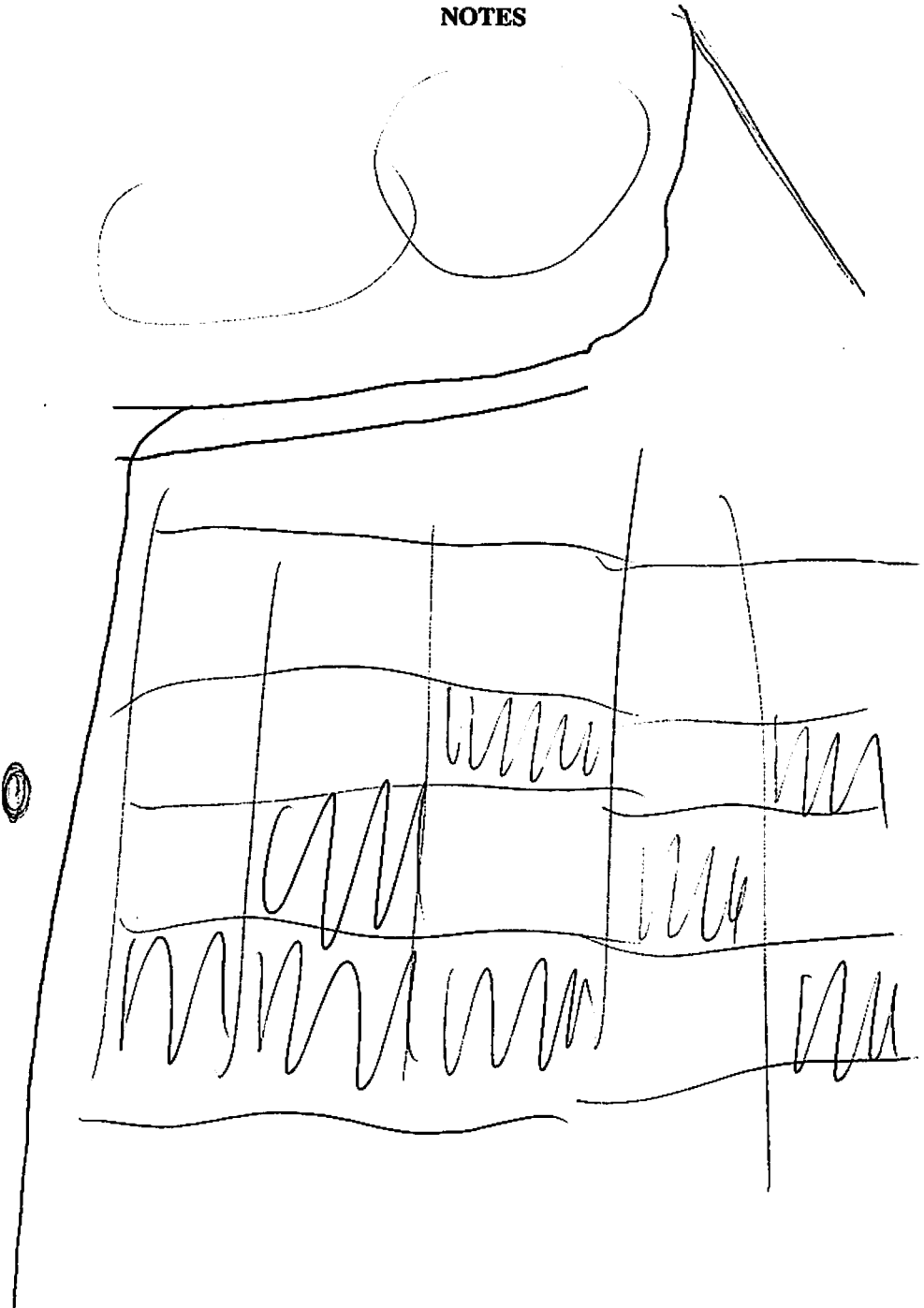
Return to: **John T. Garner, M.D.**
1127 East Green Street
Pasadena, California 91106

NOTES

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Eben

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Collected 1963-1980 - 35 cases
Begins age 40

see PH Newman
reference
or
Epstein

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