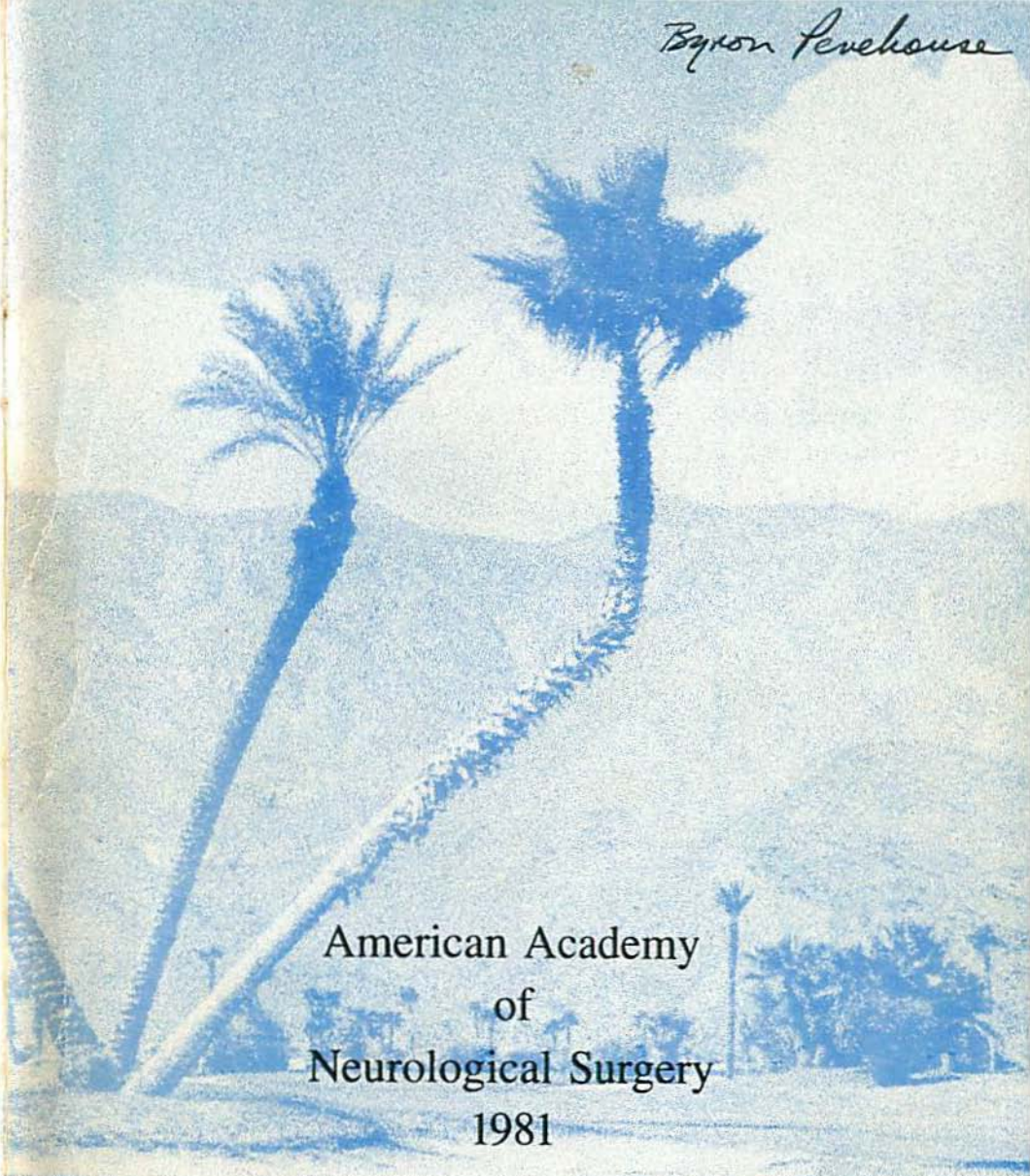
Byron Pevehouse



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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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.
Tuesday ~~Wednesday~~, November 3	8: - 10:00 a.m.

SUNDAY, NOVEMBER 1

6:00 - 8:00 p.m.	Welcoming Cocktail Party Poolside

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, NOVEMER 4

8:00 - 11:00 a.m.	Ladies Hospitality Brunch

SCIENTIFIC PROGRAM

1980

Adenoma	25%
Cushing	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 & ↑ intracran. pressure. John C. VanGilder, M.D. 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-sph. 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 bromocyptine 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 fructos-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 cerbellum 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 unusal 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 15 yr. age

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-sensory

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). 729

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 McThodist 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 ($RT1^l$) 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 ($IR = .20\ p \pm .001$). The difference between Fischer sensitized animals and control animals did not reach significance until day 80 ($IR = .5\ P \pm 0.05$). 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 ^{51}Cr release assay it was found that lyophilization or irradiation (35,000r) significantly decreased the antigenicity of the nerve allograft ($1R = .23)\ P \pm 0.001$). 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 levorpharnol (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 ED_0 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 theses 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.

MODERATOR: Nicholas Zervas

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. INTACT ARCH SPONDYLOLESTHESIS

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.

Barrister 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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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 (TERRYE) 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
W. Germany

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 Neurochirugia
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
Universität München
Marchioninistrasse 15
8000 München 70, West Germany

RAUL MARINO, JR. 1977
Rua Itaoeva
490, 11 Andar
01000 Sao Paula, SP
Brazil

HELMUT PENZHOLZ
Director Neurochirurgischen
Universitat Heidelberg
Gebaudes 110 im Neuenheimer Feld
6900 Heidelberg, West Germany

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

B. RAMMAMURTHI 1966
2nd Main Road G.I.T. Colony
Madras 4, India

KURT SCHURMANN 1978
Director
Neurochirurg
Univ-Klinik Mainz
Langenbeckstr 1
6500 Mainz, West Germany

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
Universitatsklinik
Kantonsspital
8000 Zurich, Switzerland

Active Members		ELECTED
JAMES I. AUSMAN Henry Ford Hospital 2799 West Grand Blvd. Detroit, Michigan 48202	(CAROLYN)	1978
GILLES BERTRAND Montreal Neurological Inst. 3801 University Street Montreal, Quebec, Canada	(LOUISE)	1967
WILLIAM A. BUCHHEIT, M.D. 3401 North Broad Street Philadelphia, Pennsylvania 19140	(HELEN)	1980
JERALD S. BRODKEY 2065 Adelbert Rd. Cleveland, Ohio 44106	(ARIELLE)	1977
BARTON A. BROWN 2001 Union Street San Francisco, California 94123	(MARTHA)	1968
SHELLEY CHOU University of Minn. Med. Ctr. Minneapolis, Minnesota 55455	(JOLENE)	1974
GALE G. CLARK University of California Medical Center San Francisco, California 94143	(MARIAN)	1970
W. KEMP CLARK 5323 Harry Hines Blvd. Dallas, Texas 75235	(FERN)	1970
WILLIAM F. COLLINS, JR. Yale Univ. School of Med. 333 Cedar Street New Haven, Connecticut 06510	(GWEN)	1963

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 Med.
Winston-Salem, North Carolina 27103 Vice-P. '81

RICHARD L. DeSAUSSURE (PHYLLIS) 1962
920 Madison Avenue
Memphis, Tennessee 38103

DONALD F. DOHN (CAROLYN) 1968
9500 Euclid Avenue
Cleveland, Ohio 44106

R.M. PEARDON DONAGHY (FRANCES) 1970
Mary Fletcher Hospital
Burlington, Vermont 05401

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

STEWART B. DUNSKER (ELLEN)d 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 Vice P '80

WILLIAM H. FEINDEL (FAITH) 1959
Montreal Neurological Institute Pres. '76
3801 University Street
Montreal, Quebec, Canada

ROBERT G. FISHER (CONSTANCE) Vice P '78 1956
Rutgers Medical School
Piscataway, New Jersey 08854

EUGENE FLAMM (SUSAN) Acad Award '82 1979
N.Y.U. Medical Center
550 First Avenue
New York, New York 10016

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 Exec comm. '82
New York, New York 10021

JOHN T. GARNER (BARBARA) 80-83 1971
1127 East Green Street Sec. Treas 77-80
Pasadena, California 91106

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

SIDNEY GOLDRING (LOIS) 1964
Barnes Hospital Plaza President '83
Division of Neurosurgery
St. Louis, Missouri 63110

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
Toronto, Ontario, Canada 1X8

JULIAN HOFF (DIANNE) 1975
Department of Neurosurgery
University of Michigan
Ann Arbor, Michigan 94143

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

ALAN R. HUDSON (SUSAN) 1978
St. Michaels Hospital
38 Shutter Street
Toronto, Ontario, Canada
M5B LA6

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

ELLIS B. KEENER (ANN) 1978
370 Winn Way, #201
Decatur, Georgia 30030

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

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

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

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

WOLFF M. KIRSCH (MARIE-CLAIRE) 1971
University of Colorado Medical Center
4200 East 9th Avenue
Denver, Colorado 80220

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

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

RICHARD S. KRAMER (ROBIN) 1978
Duke Hospital
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
Department of Neurosurgery
950 East 59th Street
Chicago, Illinois 60634

BLAINE S. NASHOLD, JR. (IRENE) 1967
Duke University Med. Center
Durham, North Carolina 27706

FRANK E. NULSEN (GINNEY) 1956
Division of Neurosurgery
University Hospital
2065 Adelbert Road
Cleveland, Ohio 44106

GEORGE OJEMANN (LINDA) 1975
University of Washington
Dept. of Neurosurgery
Seattle, Washington 98195

ROBERT G. OJEMANN (JEAN) 1968
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
New York, New York 10021

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

PHANOR L. PEROT, JR. (ELIZABETH) 1970
Department of Neurosurgery
Medical University of South Carolina
171 Ashley Avenue
Charleston, South Carolina 29403

BYRON C. PEVEHOUSE (LUCY) 1967
2001 Union Street
San Francisco, California 94101

ROBERT W. PORTER (AUBREY DEAN) 1962
5901 East 7th Street
Long Beach, California 90804

AIDEN A. RANEY (MARY) 1946
125 North Las Palmas
Los Angeles, California 90004

JOSEPH RANSOHOFF, II (RITA) 1965
New York Univ. Med. Center
550 First Avenue
New York, New York 10016

HUGO RIZZOLI (HELEN) 1973
2150 Penn Avenue, NW
Washington, D.C. 20037

THEODORE S. ROBERTS (JOAN) 1976
Division of Neurosurgery
University of Utah Medical Center
Salt Lake City, Utah 84132

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

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

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
170 St. George Street
Toronto 5, Ontario, Canada

HERBERT LOURIE (BETTY) 1965
713 East Genessee Street
Syracuse, New York 13210

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

ERNEST W. MACK (ROBERTA) 1956
505 S. Arlington Avenue
Suite 212
Reno, Nevada 89502

M. STEPHEN MAHALEY, JR. (JANE) 1972
Univ. of N.C., Room 229H
148 Clinical Sciences Bldg.
Chapel Hill, North Carolina 27514

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

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

RICHARD C. SCHNEIDER (MADELEINE) 1970
C5135 Out-Patient Building
University Hospital
Ann Arbor' Michigan 48104

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

BENNETT M. STEIN (DOREEN) 1970
New England Medical Center Hosp.
171 Harrison Avenue
Boston, Massachusetts 02111

JIM L. STORY, M.D. (JOANNE) 1972
7703 Floyd Curl Drive
San Antonio, Texas 78229

THORALF M. SUNDT, JR. (LOIS) Program 1984 1971
200 1st Street, S.W.
Rochester, Minnesota 55901

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

RONALD R. TASKER (MARY) 1971
Toronto General Hospital
Room 121, U.W.
Toronto, Ontario, Canada

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

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

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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.

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___ Good
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___ Too much review of old knowledge?
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___ Too complex or abstruse?
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(3) Did the speakers aim their talks:

___ Too low
___ Too high
___ Just about right

SCIENTIFIC PROGRAM

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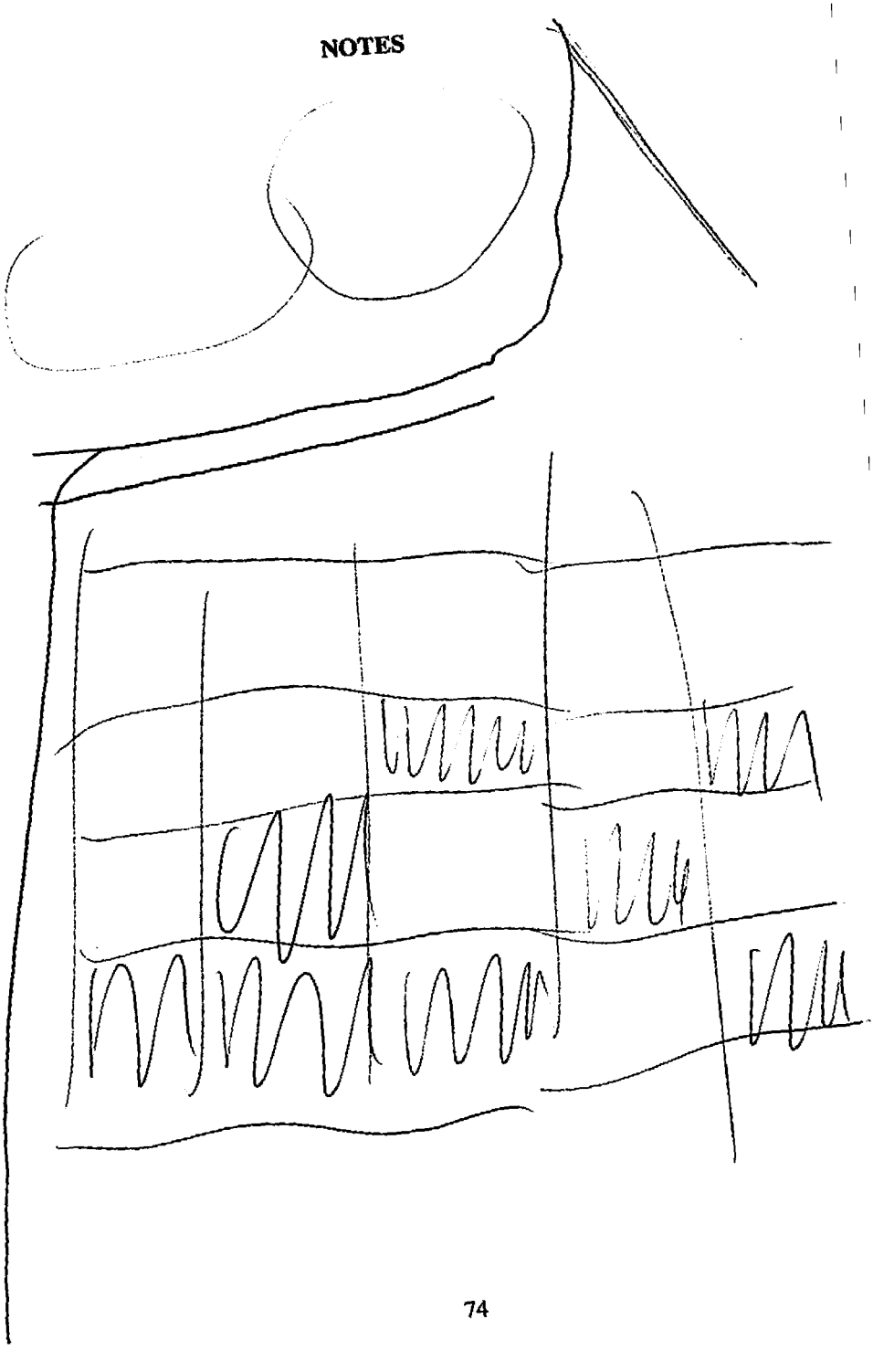

Eben

NOTES

Collected 1963 - 1980 - 35 cases see P H Newman reference or Epstein

Begins age 40

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