

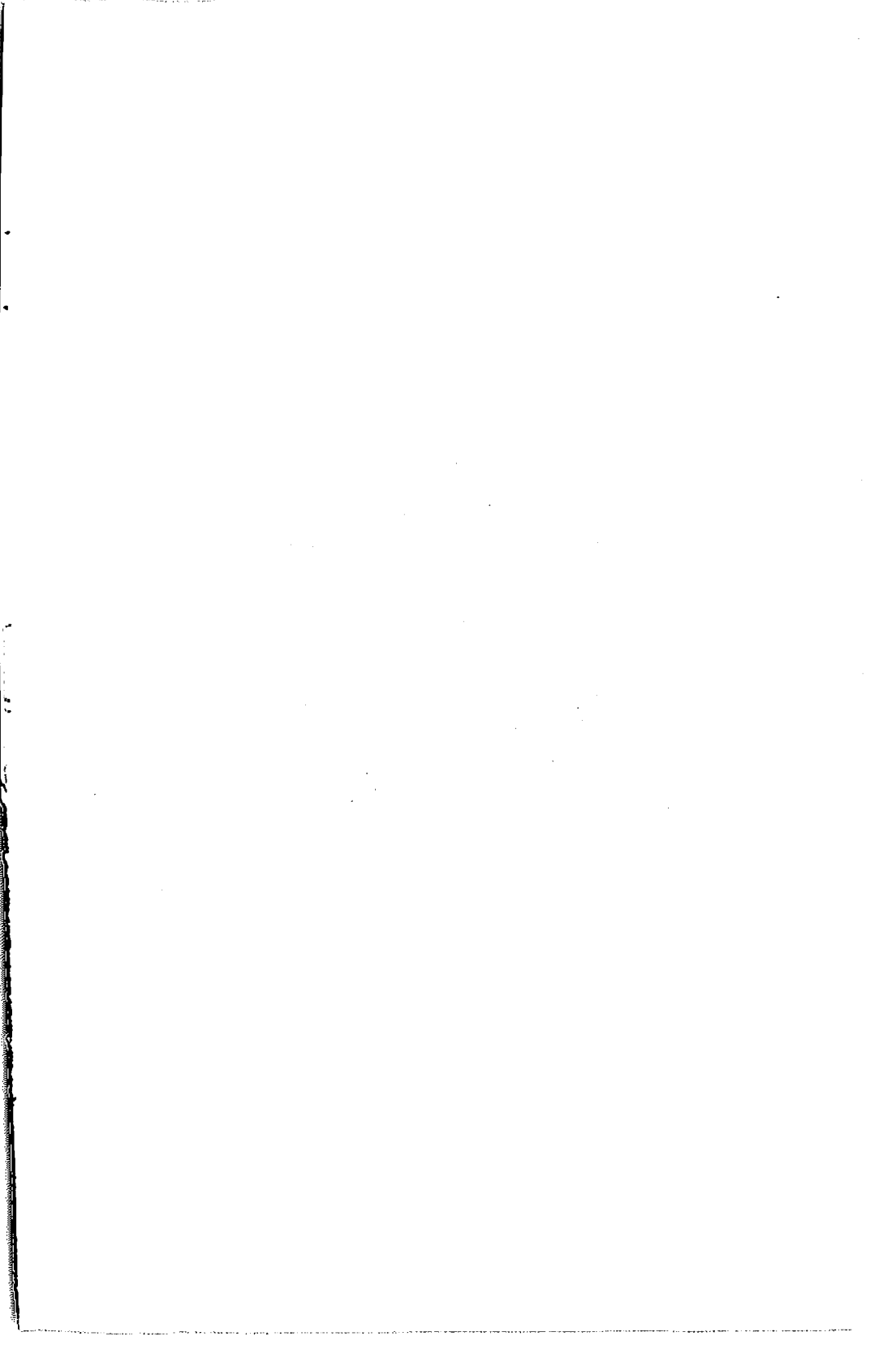
American Academy



Annual Meeting
Key Biscayne, Florida
November 11-14, 1964







The American Academy of Neurological Surgery

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Scientific Program

THURSDAY MORNING, NOVEMBER 12, 1964

9:00 A.M.

1. Review of Surgical Treatment of Aneurysms of the Anterior Communicating Artery.

Alfred Uihlein, Colin B. Holman, John Cleary, Russell L. Thomas, and Arthur G. Waltz

In 72 cases where angiography had demonstrated aneurysm of the anterior communicating artery, surgical intervention was undertaken at the Mayo Clinic in the 7 year period 1957 through 1963.

The angiographic findings and the presence of evident arterial spasm are evaluated for their prognostic significance.

The operative results are analyzed with regard to the number of preoperative subarachnoid hemorrhages, the preoperative medical and neurologic status, the surgical approach, the possibility of definitive treatment, the ancillary aids employed (profound or blanket hypothermia, intravenous mannitol or urea, drainage of spinal fluid, dexamethasone), the operative mortality, and the quality of survival.

Morbidity and mortality figures are analyzed in an effort to establish criteria for dealing with this problem.

9:15 A.M.

2. Intracranial Arterial Aneurysms in Childhood

Donald D. Matson

It has been pointed out frequently that although congenital saccular aneurysms of the intracranial arterial system probably represent incomplete resorption of vestigial vessels of the primitive circulation, they do not often bleed or become otherwise symptomatic during childhood. It also has been recognized widely that spontaneous intracranial hemorrhage in early childhood is much more apt to be due to bleeding from an arterial venous malformation, a malignant neoplasm, a choroid plexus papilloma or a blood dyscrasia than it is from a saccular arterial aneurysm.

Any one surgeon's experience with arterial aneurysms in childhood is, therefore, limited.

The clinical features, arteriographic characteristics and surgical treatment of 12 arterial aneurysms in young children seen in our clinic will be discussed in this presentation.

9:30 A.M.

3. Methyl Methacrylate Investment of Intracranial Aneurysms: A Report of Seven Years' Clinical Experience

George J. Hayes

An analysis of the results in the treatment of aneurysms by

investment with methyl methacrylate is presented. Forty patients were treated. There were four deaths related to operation. No invested aneurysm caused a death within the period of study which varies from three to seven years. There have been no signs of recurrent hemorrhage from encased aneurysms during this time span. The advantages and disadvantages of the technique are briefly discussed.

9:45 A.M.

4. Ruptured Mycotic Cerebral Aneurysms: A Report on 5 Cases

Charles G. Drake and M. R. Roach

Rupture of a mycotic cerebral aneurysm is a rare cause of subarachnoid hemorrhage. Published descriptions are few and meagre and fail to emphasize the importance of making the clinical diagnosis. Bacterial endocarditis may be unapparent for long periods until the unheralded rupture of a cerebral aneurysm. Even then the diagnosis may not be suspected in the turmoil associated with a serious neurological illness. However, the site and appearance of these aneurysms are usually quite different from the typical "berry" aneurysms and this should lead to vigorous attempts to establish the presence of endocarditis which is the commonest cause.

Six ruptured mycotic aneurysms in 5 patients have been treated surgically. This is an incidence of 2.6% of verified ruptured intracranial aneurysms on our service. Two are well, one is dead and two have serious neurologic deficit. None has had clinically significant heart disease.

Mycotic cerebral aneurysm should be suspect if (1) an obvious infection is present, (2) there is a heart murmur, splenomegaly, petechiae, microscopic hematuria, fever or elevated sedimentation rate, (3) the aneurysm is in an unusual situation — especially peripheral, or (4) recurrent aneurysms develop.

9:55 A.M.

5. Aneurysm of the Posterior Inferior Cerebellar Artery, Filling the Fourth Ventricle

Eben Alexander, Courtland Davis, Jr., and Louis Pikula

There is, judging from a review of the literature, a rarity of aneurysms presenting in the fourth ventricle.

The case herein discussed is one presenting with a typical picture of a posterior fossa tumor with a typical ventriculographic picture.

The lesion presented as a large mass completely filling the fourth ventricle and arising from the right posterior inferior cerebellar artery. The aneurysm was surrounded by a "pseudomembrane" which facilitated its removal without apparent damage.

The aneurysm was completely clotted and laminated, its only symptom producing quality being that of a mass obstructing the flow of cerebrospinal fluid.

10:00 A.M.

Discussion (Papers 1-5)

10:15 A.M.

Coffee Break

10:30 A.M.

6. Studies in the Protection Against Cerebral Damage from Intracarotid Injection of Hypaque

Lonnie L. Hammargren, August W. Geise, and Lyle A. French

This study was designed in an attempt to investigate the toxic action of contrast media used in cerebral angiography on the central nervous system and to evaluate methods to prevent complications. The protective action of low molecular weight dextran (Rheomacrodex) and dexamethasone (Decadron) on cerebral damage following intracarotid injection of 90% Hypaque was studied in rabbits.

This study attempts to simulate cerebral angiography in a controlled manner. Cerebral damage and death often resulted from injections of large doses of Hypaque into the carotid artery. Pre-medication with LmDx or with Decadron resulted in a significant reduction in cerebrovascular permeability to dyes, cerebral edema and mortality. Premedications with both drugs simultaneously produced almost complete protection from cerebral damage.

10:45 A.M.

7. Low Molecular Weight Dextran (Rheomacrodex) for Improving Microcirculation of the Brain

J. Lawrence Pool

Rheomacrodex 10% in Dextrose has been administered to patients in poor condition neurologically because of a ruptured intracranial aneurysm, with the hope of improving the cerebral circulation. Its use is based on studies which show that it has a twofold action:

1. Prevention of aggregation of red blood cells by altering their electrical charge.
2. Improvement of the circulation because it expands the plasma volume.

These two effects are supposed to improve the capillary or microcirculation of various tissues. Therefore this type of dextran solution has been used with the aim of improving the microcirculation of the brain. Our experience with a small series of cases will be described.

11:00 A.M.

8. Radioisotopic Studies of Human Cerebral Circulation

William Feindel, Henry Garretson and Y. Lucas Yamamoto

Radioactive isotopes have been injected by a catheter placed in the internal carotid artery at the time of craniotomy. Using multiple miniature gamma ray probe detectors, various sites on the exposed surface of the brain can then be monitored during the rapid passage of the isotope through the cerebral blood vessels.

The "visual field" of the detectors is sufficiently small to provide differentiation between arteries, veins and regions of cortex not covered by large vessels. A preliminary report of this method was given at the 1963 meeting; we now wish to review additional examples studied by this technique, illustrating its application to the handling of angiomas and in the investigation of local changes in the cerebral circulation.

11:15 A.M.

Discussion (Papers 6-8)

11:25 A.M.

9. Selective Blood-Brain Barrier Damage

C. Norman Shealy (By invitation of Frank Nulsen)

We have selectively increased blood-brain barrier (BBB) damage in brain lesions without obvious brain edema or neurologic damage. Bilateral symmetrical focused ultrasonic lesions were placed in the cerebral hemispheres of cats. Three or four days later the blood-brain barrier is almost completely repaired so that there is little staining with trypan blue. If the BBB of one side is now challenged with intracarotid 15% ethanol or 50% Hypaque, there is a striking increase in subsequent trypan blue staining of the ultrasound lesion in that hemisphere. The animals survive this procedure without neurologic deficit and there is no BBB damage to any brain tissue so perfused but lying beyond the area of ultrasonic damage. Further studies with radio-isotopes are now under way and other drugs are being used to increase selectively the barrier damage at the lesion site.

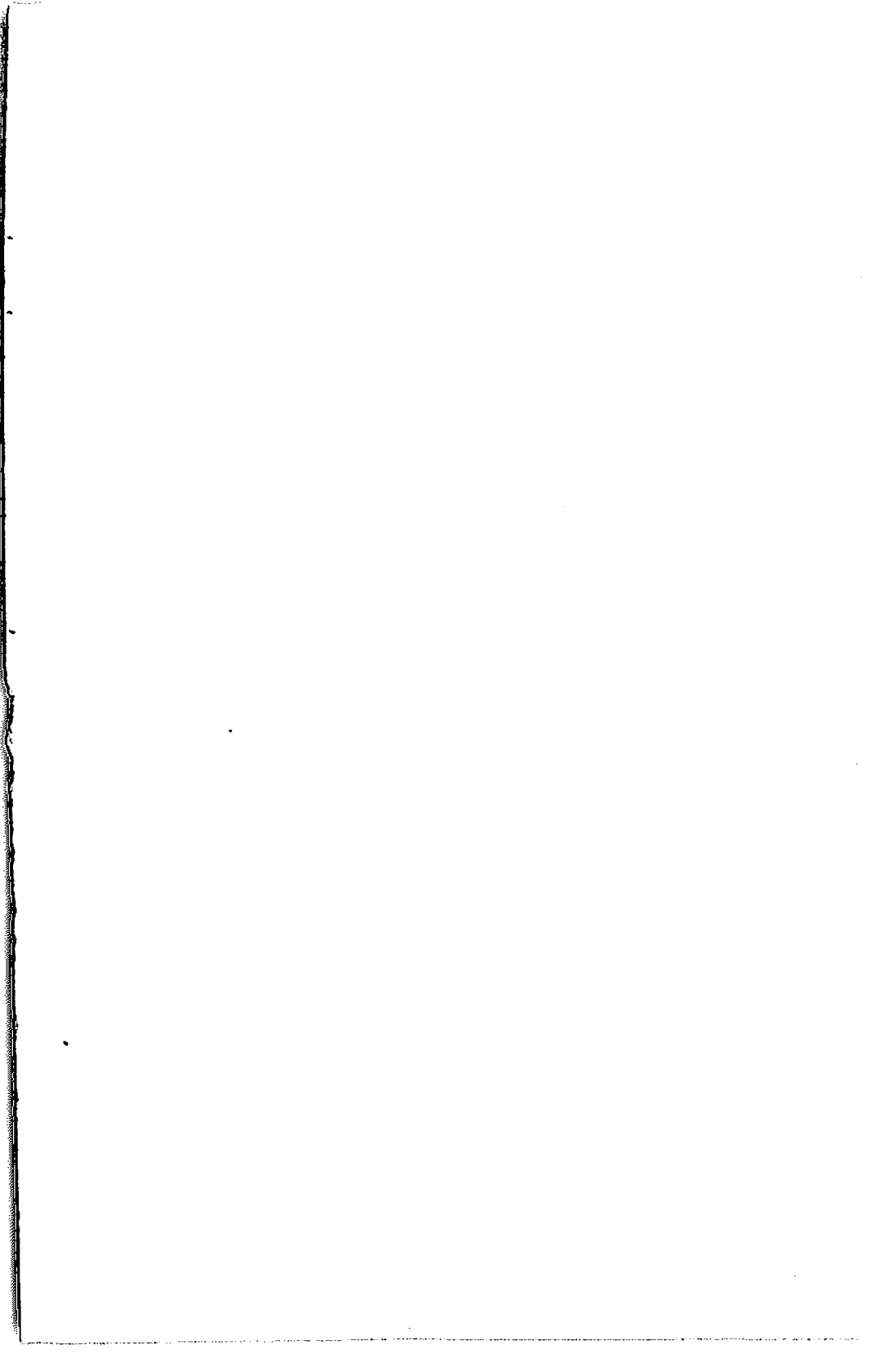
There is evidence that a differential uptake of 50 to 1 (tumor to normal brain) is needed for chemotherapy to be effective. Probably a 10 to 1 ratio is satisfactory for radio-isotopic diagnostic studies. Conceivably, an increase in BBB damage in tumors could improve diagnostic and therapeutic effectiveness. The study has further implications in relation to complications from carotid arteriography.

11:40 A.M.

10. Experimental and Clinical Profound Isolated Cerebral Hypothermia

R. White, J. Verdura, M. Albin, and H. Brown (By invitation of Frank Nulsen)

To simplify and yet retain the operative advantages of profound hypothermia of the central nervous system a modified arterial-arterial



perfusion has been developed allowing reductions of brain temperature to 15° C. or less with minimal reductions in body core temperature.

Following the elimination of the posterior circulation to brain via ligation of the basilar artery, the brain was selectively cooled in 21 anesthetized, heparinized dogs by pumping blood from the femoral artery through a small metal heat exchanger to 2 "T" cannulae positioned in the carotid arteries. Utilizing flow rates of 100 - 130 cc./min. and carotid cannula pressures of 120 - 140 mm. Hg. intracerebral temperatures of 15° C. were produced within 25 minutes or less while right atrial temperature remained above 31° C. Cerebral ischemia was produced by the temporary closure of the major branches of the external carotid and the internal carotid arteries; during this period the head was packed in ice. Cerebral rewarming resulted from systemic perfusion after the carotid ligatures were released. 18 dogs survived without neurological defect. 1 animal died and 2 others had temporary hind leg paralysis.

In one patient with known metastatic brain tumor, differential cooling of the brain to 11° C. with maintenance of intravascular temperature of 35.5° C. was accomplished in 19 minutes utilizing an identical arterial-arterial perfusion circuit. The perfusion cannulae were positioned in the external carotid arteries and with the common carotid arteries occluded flow rates of 450-200 cc./min. were used, depending on intracerebral temperature. The patient survived 30 minutes of carotid artery occlusion and removal of the tumor at this temperature.

11:55 A.M.

11. Isolated Profound Hypothermia of the Brain — A Description of Technique

B. C. Llewellyn, D. E. Richardson, and C. W. Pearce

The clinical application of the animal interests of two of the authors (Dr. Richardson and Dr. Pearce) allows description of a technique that affords profound brain cooling in man without systemic (heart) hypothermia. This technique cools one hemisphere of the brain to 10-15° centigrade while the body temperature remains within 4-6° centigrade of normal. The presentation will include a description of the apparatus used and the surgical technique required to achieve brain cooling. The physiological changes observed in the brain and in the body during the procedure will also be described. The presentation will include, finally, the various clinical problems that are particularly suited for the use of this method of achieving profound brain cooling.

12:05 P.M.

Discussion (Papers 9-11)

12:15 P.M.

Lunch

THURSDAY AFTERNOON, NOVEMBER 12

2:00 P.M.

12. Repair and Replacement of Small Arteries Using Microsuture Technique

Ghahreman Khodadad and William Lougheed

The experiments were carried out on cats under general anesthesia. The femoral or carotid arteries were exposed, and their external diameter was measured (1 to 2 mm.). The circulation was then stopped, and the following procedures completed:

1. Repair of a straight incision.
2. Repair of a transectional incision.
3. Repair of an arterial defect with venous patch.
4. Segmental arterial replacement by a segment of vein.
5. Bypass (end to side), using a segment of vein.

The patency of the arteries was evaluated by angiography at different intervals. Some of the animals were sacrificed, the site of operation exposed and examined, and finally the arteries were removed for histological examination. The following table shows the number of the arteries which were repaired, and their results:

Type of Repair	Number of Arteries	Arteri- ography	Patent.	Follow-up
Straight incision	30	All	All	7 months
Transect. incision	15	All	All	5 months
Venous patch	15	All	All	3.5 months
Segment. replacement	15	All	14	3 months
Bypass	5	All	All	1.5 months

The autopsy specimens of 25 of these arteries were histologically examined (majority of the animals are still alive), and showed a mild degree of endothelial hyperplasia and fibrosis at the site of the incision.

2:15 P.M.

13. The Effect of Increased Intracranial Pressure Upon Flow in the Internal Carotid Artery — Studies in 14 Patients

Guy L. Odom, George T. Tindall, Joseph C. Greenfield, Jr.

Continuous simultaneous measurements of blood flow (electromagnetic flowmeter) and arterial pressure were obtained from one internal carotid artery in a group of 14 patients in whom the intracranial pressure was artificially raised to a level of approximately 1000 mm. H₂O by the intrathecal infusion of Ringer's solution. Blood flow was determined with a Kolin-Kado type electromagnetic flowmeter. Arterial and CSF pressures were each measured with a Statham transducer. Each patient had a surgically established diagnosis of brain tumor, and infusion of the internal carotid artery with

a cancer-chemotherapy drug was carried out following this study. Craniotomy and subtotal resection of the tumor had been previously performed in each patient.

Results:

1. A quantitative relationship between internal carotid flow and CSF pressure was established in each patient. There was no significant change in flow in the internal carotid artery until the CSF pressure reached a level of approximately 350 mm. H₂O. Further increases in CSF pressure caused a progressive reduction in flow in all subjects.

2. In six patients in whom the elevated CSF pressure was sustained for 2 to 5 minutes, there was a slight tendency for internal carotid flow to return to the control value.

3. There were no significant changes in either arterial pressure, heart rate, electrocardiogram or electroencephalogram, in patients with a CSF pressure of 1000 mm. H₂O.

The clinical significance of these results is discussed.

2:30 P.M.

14. Neural Mechanisms for Fat Mobilization

James W. Correll (By invitation of J. Lawrence Pool)

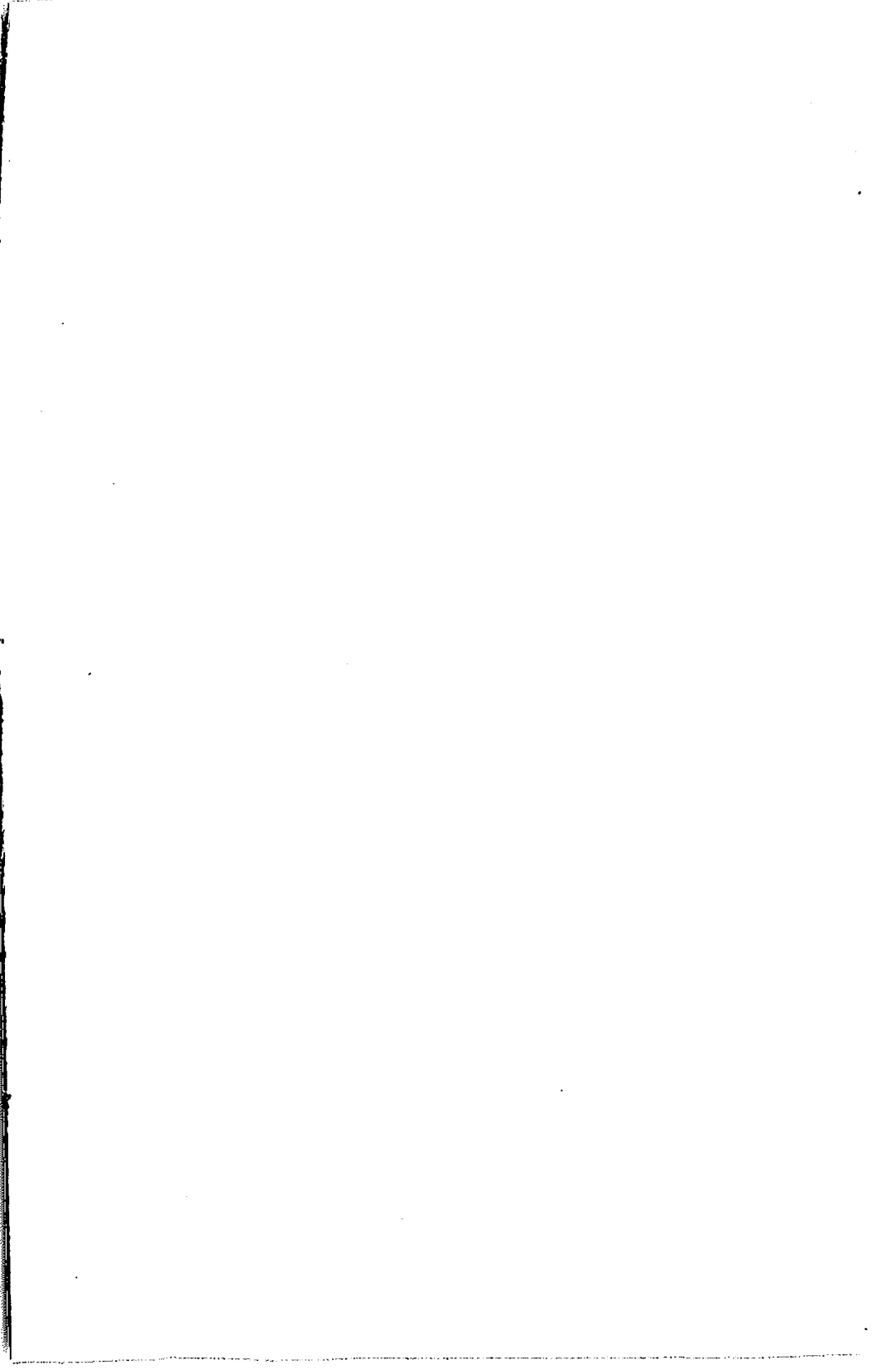
Fat mobilization is dependent on the liberation of free fatty acids (FFA) from the adipose tissue reservoirs. The experimental studies here reported demonstrate that adipose tissue has the capacity to function as an effector organ under direct control of the nervous system. Electrical stimulation of the nerve supply to an isolated nerve-fat preparation, in vitro, has shown that fat responds by the liberation of FFA (Correll, *Science* 140: 387, 1963). Structures important for FFA mobilization are in the basal forebrain and in the diencephalon with pathways descending into the spinal cord (Correll, *Fed. Proc.* 22: 575, 1963). It has also been found that the FFA mobilization which follows the intravascular administration of norepinephrine is in part dependent on the stimulation of the brain by the catecholamine (Correll, Cantu and Manger, *The Physiologist* 1964). These investigations provide direct evidence of the ability of the central nervous system to regulate the metabolism of adipose tissue and influence blood lipids.

2:45 P.M.

15. Sensory and Motor Interactions in the Ventral Anterior Nucleus of the Thalamus

A. G. MacIntyre, F. Ervin and V. H. Mark (By invitation of William Sweet)

Using cats, we were able to record evoked potentials from the ventral anterior nucleus of the thalamus (V.A.) after application of electric shocks either to the cortex or to the paws. Then, using various pairs of stimuli adjustably separated in time, it was shown



that whether it originates in the cortex or in the periphery, the potential produces a prolonged inhibitory effect on any other potential subsequently arriving. This effect extends far beyond the time refractory period and lasts about 50 to 300 milliseconds depending on stimulation parameters.

The results describe two phenomena in this part of the extralaminar systems. Central mechanisms controlling sensory transmission involve V. A. and sensory impulses modify cortically induced responses passing through this same nucleus. The latter finding may explain one means by which increased sensory input aggravates disorders of movement.

3:00 P.M.

Coffee Break

3:15 P.M.

16. Ultrasonic Scanning of the Brain

J. H. Galicich, C. T. Lombroso and D. D. Matson

Ultrasonic B-scanning of the brain as well as A-scope midline determinations were carried out in 78 neurosurgical patients and 20 neurologically normal controls. Recognition of echoes from midline brain structures was found to be more reliable with intensity modulated scanning than with A-scope presentation. In the control groups, deviation of midline interfaces from the mid-sagittal plane of the skull averaged less than 0.5 mm. on B-scans with a range of 0 to 1 mm. In neurosurgical patients, midline positions determined by ultrasonic scanning correlated well with those determined by contrast studies in 31 of 31 cases. Enlarged lateral ventricles were clearly demonstrated on scans of 29 of 31 proven cases of hydrocephalus. Eleven of 14 supratentorial tumors were visualized by scanning.

3:30 P.M.

17. Five Years' Experience in the Treatment of Infantile Hydrocephalus by Auriculo-Ventricular Shunting

Samuel R. Snodgrass

During a five year period beginning in 1959, 52 infants with hydrocephalus have been treated with the Spitz-Holter or Pudenz-Heyer valve. 95 operative procedures were performed; there were four wound infections, three of which were accompanied by septicemia and three additional patients developed septicemia. Four patients have been lost to follow-up and thirteen are known to have died. Discussion with lantern slides showing etiology of the hydrocephalus, age at onset of treatment, number of operations per patient, interval since last operation, complications of operation and cause of death will be presented.

3:45 P.M.

18. The Cause and Consequence of Acromegaly in Salmon

Henry L. Heyl

During the spawning journey up the river from the ocean, salmon, particularly the males, develop large ugly lower jaws and humpbacks. This is associated in both sexes with overall enlargement of the pituitary and early preponderance of eosinophilic chromophiles, hypertrophy of adrenal tissue, and increase of plasma corticosteroids. In the Pacific species, the journey invariably ends with death, due, according to the evidence of O. H. Robertson, to auto-intoxication with cortisone. Our study is stimulated by the fact that many Atlantic salmon survive the journey and may even repeat the cycle several times in subsequent years. We are searching for clues to the cause of this difference and the possible relation of the phenomenon to pituitary function in general. During the last part of the journey, the pituitary of Pacific species shows a shift to basophiles that is much less dramatic in the Atlantic species. Atlantic fish show less adrenal proliferation and lower maximal plasma corticosteroid levels which actually have returned to normal by the time the fish re-enters the ocean after spawning. The related microscopic changes in certain other endocrine structures are also discussed.

4:00 P.M.

19. Academy Award Presentation

Stereotoxic Investigation of Hypothalamic Sexual Cycling

Earle E. Crandall

5:00 P.M.

Executive Meeting

FRIDAY MORNING, NOVEMBER 12

9:00 A.M.

20. Cordotomy for Pain Following Cauda Equina Injury

Robert W. Porter, George Hohmann and Ernest Bors

Severe, persistent, lancinating pain in the lower extremities requiring surgical intervention was a sequela of traumatic injury of the cauda equina in 34 of 637 patients treated. These patients were subjected to 47 bilateral high thoracic cordotomies and were observed for periods of 8 to 20 years following operation. All but one patient received significant relief from their symptoms in the immediate post-operative period. When evaluated 4-6 years later, however, it was noted that 62 per cent remained free of pain or were sufficiently improved so that no further treatment was considered. Thirty-eight per cent were considered unsuccessful as additional cordotomies or

narcotics were required to control their pain. As might be expected, it was observed that when the level of analgesia induced fell to within four segments of the level of injury, the failure rate was higher than in those patients in whom the level remained five segments or above. A substantially higher percentage of failures were noted following cordotomies performed within the first eighteen months after injury than following those done after four to six years had elapsed before operation was required. It was concluded that although cordotomy may be a useful and at times a necessary procedure for the relief of shooting extremity pain of traumatic origin, it should only be performed after an exhaustive and critical evaluation of the patient's over-all course of rehabilitation.

9:15 A.M.

21. Unilateral Cordotomy for Intractable Pain Problems — Multiple Section Technique

George S. Baker and R. H. Miller

Multiple sections of the spinothalamic tracts for unilateral pain has been used to good advantage by the authors for ten years or more. The favorable results and the maintenance of good levels for the control of pain over this period warrants a report and follow-up study from a clinical standpoint. The selection of cases for its use and the technique employed will be discussed.

9:30 A.M.

22. Frontal Cingulotomy for Disabling Mood Disturbances: A Preliminary Report

H. Thomas Ballantine, Jr., Walter L. Cassidy
and Norris B. Flanagan

In 1962, Foltz and White published a paper entitled "Pain Relief by Frontal Cingulotomy." They described a technique of electrode cauterization of the cingulum bilaterally in 16 patients. Significant in their paper was the statement that "patients who had anxiety and/or depression in one of various combinations had the best results from cingulotomy."

Drawing on this experience and that of Cairns and others who have performed "open" cingulectomy for mental illness, we have cingulotomized ten patients with intractable mood disturbances — usually characterized by the symptoms of depression. The technique employed has been a slight modification of that described by Foltz and White. Multiple coagulations have been performed in most instances.

One patient received so little electrocoagulation that it is doubtful that a lesion was created: she showed no post-operative change. One patient committed suicide after an initial period of improvement. The other eight have shown various degrees of improvement, includ-

ing at least one "cure". As nearly as we can determine, no patient has been made worse by the surgery.

The results of these initial efforts have been sufficiently gratifying to warrant this preliminary report. Detailed descriptions of patient types and procedures employed will be presented.

9:45 A.M.

**23. Compression Procedure for Trigeminal Neuralgia:
Review and Clinical Appraisal of Ten Years' Experience**

C. Hunter Shelden

10:00 A.M.

Discussion (Papers 20-23)

10:15 A.M.

Coffee Break

10:30 A.M.

**24. Anthropometric Encephalometry (Brain Measurements
and Stereotaxy)**

Hannibal Hamlin

Stereotactic experience continues to reiterate discrepancy in correlation between traditional points and planes of the head and skull and favorite intracerebral landmarks. Studies of cranial growth during early life (Moss and Young) and clinical examples from post-adolescent years indicate that change in head size and form coincides with homologous alteration of the expanding or shrinking brain and its capsule. A method of brain marking has been developed both for the intact cadaver head and the fixed postmortem cerebrum. The AC-PC plane has been utilized as the primary midsagittal axis of a 3-plane interperpendicular system of coordinates to construct a tri-dimensional system of anthropo-encephalometry which should be of major heuristic importance as well as of interest to stereotactic neurosurgery.

10:40 A.M.

Discussion

10:45 A.M.

25. Considerations in the Management of Ependymomas

John W. Hanbery

Whether an ependymoma arises in the posterior fossa, the cerebral hemispheres, or the spinal canal it is frequently impossible

erally accepted that postoperative irradiation to the area of known residual neoplasm is of value. It is well recognized that distant seeding of an ependymoma via the cerebrospinal fluid pathway may occur. There is a marked discrepancy in the reported incidence of this type of neoplastic spread with figures ranging between one and 50 per cent.

A review of our last 14 cases of histologically proven ependymoma at the Stanford Medical Center has demonstrated that within this relatively small series of cases the incidence of spread via the cerebrospinal fluid pathway has been sufficiently high that it has altered our plan of management as it pertains to postoperative irradiation.

Distant spread of neoplasm within the central nervous system occurred in four of our cases. These were all of clinical significance and were demonstrated by myelography, ventriculography, or operation. In another infant, autopsy studies revealed an ependymoma originating in the hypothalamic region with subarachnoid seeding within the fourth ventricle. In two other cases, neoplastic cells were clearly identified in the lumbar cerebrospinal fluid with the primary lesion being present in one case within the fourth ventricle and in the other within one lateral ventricle.

At the present time, it is our practice to irradiate the entire neural axis in all cases of ependymoma arising within the fourth ventricle or within the supratentorial ventricular system. We would follow the same plan of irradiation following the removal of an ependymoma of the cauda equina if it were felt that cellular contamination of the cerebrospinal fluid had occurred. Furthermore, we have felt that it was important to search for neoplastic cells in ventricular and lumbar subarachnoid fluid, using a millipore technique and to use unequivocal presence of neoplastic cells as an indication for generalized irradiation.

11:00 A.M.

26. Steel Mesh Tunnel for Repair of Severe Meningomyeloceles with Hydrocephalus

William B. Scoville

To date surgical repair of severe infantile meningomyeloceles in the new born have been disappointing because of a frequent progressive hydrocephalus and/or a progression of paraparesis. This latter results from a congenital absence of both roof and walls of the spinal canal and a consequent direct compression of the terminal cord by the fascial-skin repair.

A report is made of nine severe cases of totally exposed spinal cords with rachischisis and three additional cases of infiltrating large lipomata with spina bifida in which artificial tunnels were made using a molded convex covering of extra heavy steel mesh, extending well above and below the spina bifida to permit future growth. Its

The results have been better than anticipated with a continuing improvement in neurological status and, to our surprise, a regression or absence of hydrocephalus in 7 out of 8 cases, in spite of grossly exposed cords.

11:10 A.M.

27. Cerebral Atrophy, Clinical and Roentgenologic Aspects

Robert G. Fisher

All cases of cerebral atrophy from 1951 through 1963 were analysed from the standpoint of clinical and roentgenologic aspects. Their particular features involved in individual cases were studied as far as possible. Polyp examinations were analysed and long-range studies were made to indicate variation as far as progress of the condition was concerned. The majority of cases did not deteriorate rapidly, convulsions were frequent, but were generally controlled with anti-convulsive medication. The majority did not require state hospitalization. Suggestions about new methods of approach are being made in an attempt to find out the specific factors involved in cerebral atrophy cases of the future.

11:25 A.M.

28. Focal Encephalitis as a Cause of Unilateral Progressive Epilepsy

James W. Kernohan, David D. Daly, and John R. Green

A seven-year-old boy developed focal seizures and a progressive severe left hemiparesis over a period of fourteen months. Excision of a portion of the motor cortex had produced a remission of seizures for one week. Examination revealed a spastic left hemiplegia, left homonymous hemianopsia, impaired sensation on the left side of his body, and frequent episodes of status epilepticus. Electroencephalography disclosed multiple foci of epileptiform discharges throughout the right hemisphere and dilatation of right lateral and third ventricles. A right hemispherectomy was performed. The boy has had no seizures during the year since surgery was performed and has returned to school. The neuropathological study of the surgical specimens forms the basis of this report. In some areas the entire cortex had been destroyed, in others there were numerous foci of subacute encephalitis of unusual appearance. There were some findings suggestive of a viral origin for the inflammatory process.

11:45 A.M.

Presidential Address

Some Physiological Implications in Focal Epilepsy

Theodore B. Rasmussen

SATURDAY MORNING, NOVEMBER 14

9:00 A.M.

29. **Complications from the Anterior Approach for Cervical Spine Disease**

Stuart N. Rowe and Anthony F. Susen

The first one hundred consecutive patients on whom we have operated for cervical spine disease via the anterior route were subjected to a critical review with respect to postoperative complications.

We have broken down these complications into three broad groups:

- A. Anterior soft tissue complications – recurrent laryngeal paralysis, sympathetic paralyses, and rupture of the esophagus.
- B. Cord and nerve complications – neuropathy and spinal cord dysfunction.
- C. Bony element complications – failure of fusion, extrusion of plug, and collapse of vertebra due to infection.

As our experience increased, we were able to prevent many of these complications, and these measures will be discussed.

9:15 A.M.

30. **Anterior Cervical Decompression (Without Fusion)**

Edwin B. Boldrey

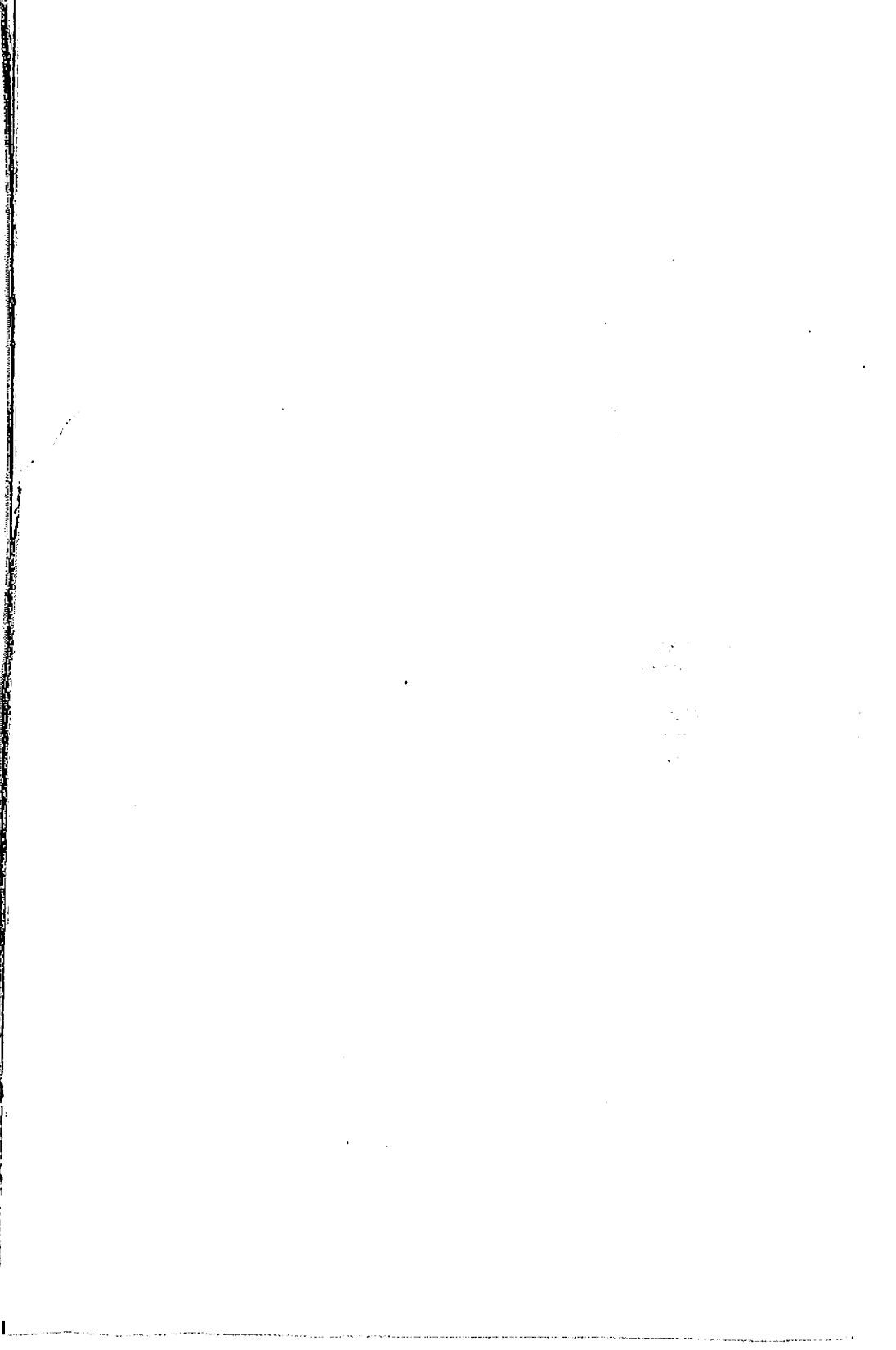
Over the past four years transarticular removal of osseous ridges ("bar discs") from the anterior surface of the cervical canal and of proliferative excrescences from the anterior margin of the intervertebral foraminae have been carried out in a select group of patients with a high incidence of, but not one hundred per cent, success. By omitting the introduction of foreign (bony) material as in the anterior cervical fusion, the complications therein attendant are avoided. The approach limited to the cleft remaining after removal of the intervertebral disc material and sparing as much of the annulus fibrosus as possible is not as generous as when a bony plug is removed, but is adequate and is felt to leave a more substantial anatomic basis for a firm fibrous bond. The reasons for the procedure are evidence of clinical involvement of the cervical cord by the bar disc, and/or evidence of irritation of the root at or near the foramen.

9:30 A.M.

31. **Anterior Approach to Decompress the Spinal Cord and Nerve Roots: Clarification of Some Problems**

Ludwig Kempe (By invitation of George Hayes)

This is a report of the observation made on 100 patients having had the anterior surgical approach for cervical myelopathy and radi-



culoneuropathy. The shortest time of observation was twelve months. It was seen that the tendency to anterior angulation at the level of fusion could be reduced by a more prolonged postoperative external fixation. Delayed postoperative appearance of myelopathy is believed to be due to onset of this angulation. Hypertrophic osteoarthritic spur formation does not seem to disappear at the level of fusion. Removal of the spurs with visualization of the nerve roots is recommended. Postoperative progressive anterior angulation combined with hypertrophic osteoarthritic spurring is believed to be the cause of recurrence of symptoms.

9:45 A.M.

32. The Transoral Approach to Some Neurosurgical Problems

Sean Mullan and Ralph Naunton

Although the nasopharyngeal approach to the pituitary has been widely explored, the possibilities of a transoral approach to the basiocciput, the basisphenoid, the anterior portion of the first and second vertebrae as well as to intradural tumors anterior to the medulla and upper spinal cord, have received relatively little attention.

Recently by this route we have totally removed the chordoma infiltrated body and odontoid process of the second cervical vertebra of a patient aged 39. For a 3-year-old child we have made an intracapsular removal of an anteriorly placed cranio-cervical neurofibroma.

Our paper relates our experiences with these two patients and discusses some of the possibilities and limitations of the method.

9:55 A.M.

Discussion (Papers 29-32)

10:15 A.M.

Coffee Break

10:30 A.M.

33. Myelographic Diagnosis of Spinal Adhesive Arachnoiditis

D. E. Richardson and R. C. Llewellyn

During the past four years sixteen patients evaluated in our teaching services at the Veterans Administration Hospital and Charity Hospital in New Orleans have had final diagnoses of adhesive spinal arachnoiditis.

A review of these patients reveals a rather clear division into two groups. The first was caused by infection or previous surgery resulting in paraparesis, paresthesia, hypalgesia or lower extremity pain. Myelography in these patients revealed the typical picture of arachnoiditis with irregular, streaked and globulated obstruction to the contrast media. (Eight patients).

The second group was patients who had had previous myelography and/or spinal anesthesia for disc surgery with recurrence of symptoms at a later date. The symptoms were usually sciatica and back pain, and clinical findings were for the most part consistent with recurrent disc rupture. The myelographic findings were confusing with smooth spinal block often mimicking a large disc rupture and even an intradural tumor. The picture of a smooth, conical, non-deviated myelographic block overlying a vertebral body rather than a disc space in a patient previously operated should bring up the likelihood of arachnoiditis. (Eight patients).

Myelographic points of interest, typical cases, and difficult cases will be presented by projection slides. All diagnoses were either obvious or surgically confirmed.

10:40 A.M.

34. Three Dimensional Myelography

Ernest W. Mack

In an attempt to secure additional information from lumbar myelography we have performed a series of cases in which very small amounts of Pantopaque have been dispersed in varied amounts of spinal fluid. The pictures thus obtained have been of interest and examples of these will be presented. A satisfactory technique has been secured and an optimum concentration has been decided upon. It is felt that while this technique probably could not be considered as worthy of displacing the standard technique for Pantopaque myelography it, nevertheless, does offer some interesting additional information which might be useful in certain types of myelography.

10:50 A.M.

35. Review of Discitis Cases

Edmund J. Morrissey

Postoperative intravertebral disc space infections have been relatively rare but have always caused considerable concern. The clinical findings and X-rays characteristics in a series of postoperative discitis as well as a series of primary discitis cases will be reviewed. Surprisingly good prognosis under proper treatment will be emphasized.

11:00 A.M.

36. Thrombosis of the Meningorachidian Plexus

Augustus McCravey

A twelve-year-old negro girl with acute onset of pain in back and lower extremities with progressive paralysis. Myelogram showed complete block at D-4. Large thrombotic lesion removed with very slow return of motor and sensory components in lower extremities.

11:05 A.M.

Discussion (Papers 33-36)

11:20 A.M.

37. **Metabolic Response to Cranial Surgery and Trauma**

Robert L. Mc Laurin and Lionel King

Thirty balance studies have been performed on 29 patients following intracranial surgery or trauma and the results have been subjected to statistical analysis. The factors studied included effects of variable inputs of water, sodium, potassium, nitrogen and calories, and of age, sex, and degree of trauma on balances of water, sodium, potassium, and nitrogen. Age, sex and severity of trauma were found to have no effect on metabolic balances. The responses of water, nitrogen, and electrolyte balances are similar qualitatively to those observed after general surgery or bodily trauma. Sodium retention occurred during the initial period and between 2 and 4 days following injury sodium diuresis appeared. An initial water retention also was noted and was related to water input during the first 3 days. A relationship of water to sodium occurred on the third day only with any statistical significance. Corrected potassium balances were slightly positive during the 5-day observation period. Nitrogen loss of 2-6 Gm. was induced by trauma and nitrogen utilization was apparently not impaired, indicating that excess nitrogen intake may be of benefit. There was no evidence of cerebral salt-wasting or cerebral salt-retention in the series. The conclusions are useful in planning parenteral therapy following head injury or surgery.

11:45 A.M.

Final Executive Meeting

Guests of the Academy 1964

- Dr. Walter Cassidy *Wellesley, Massachusetts*
Dr. James W. Correll *New York, New York*
Dr. Earle E. Crandall *San Diego, California*
Dr. Frank Ervin *Boston, Massachusetts*
Dr. Frank Espy *Greenville, South Carolina*
Dr. Joseph Galicich *Boston, Massachusetts*
Dr. Henry Garretson *Montreal, Quebec*
Dr. Tracy Haverfield *Miami, Florida*
Dr. Ludwig G. Kempe *Washington, D. C.*
Dr. James W. Kernohan *Rochester, Minnesota*
Dr. Ghahreman Khodadad *Toronto, Ontario*
Dr. Paul H. Lewis *Cincinnati, Ohio*
Dr. Alexander MacIntyre *Boston, Massachusetts*
Dr. Vernon Mark *Boston, Massachusetts*
Dr. Louis Pikula *Winston-Salem, North Carolina*
Dr. David Reynolds *Miami, Florida*
Dr. Lamar Roberts *Gainesville, Florida*
Dr. James R. St. John *Santa Barbara, California*
Dr. C. Norman Shealy *Cleveland, Ohio*
Dr. Anthony F. Susen *Pittsburgh, Pennsylvania*
Dr. George Tindall *Durham, North Carolina*
Dr. Ian Turnbull *London, Ontario*
Dr. Robert J. White *Cleveland, Ohio*
Dr. Charles Wilson *Lexington, Kentucky*

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John Raaf	1949
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Wallace B. Hamby	1951
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David L. Reeves	1955
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George Baker	1960
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C. Hunter Shelden	1959
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Donald Coburn	1961-62
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Past Secretary-Treasurers

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Past Meetings of the Academy

Hotel Netherland Plaza, <i>Cincinnati, Ohio</i>	October 28-29, 1938
Roosevelt Hotel, <i>New Orleans, Louisiana</i>	October 27-29, 1939
Tudor Arms Hotel, <i>Cleveland, Ohio</i>	October 21-22, 1940
Mark Hopkins Hotel, <i>San Francisco</i> , and Ambassador Hotel, <i>Los Angeles, Calif.</i>	November 11-15, 1941
The Palmer House, <i>Chicago, Illinois</i>	October 16-17, 1942
Hart Hotel, <i>Battle Creek, Michigan</i>	September 17-18, 1943
Ashford General Hospital, <i>White Sulphur Springs, West Virginia</i>	September 7-9, 1944
The Homestead, <i>Hot Springs, Virginia</i>	September 9-11, 1946
Broadmoor Hotel, <i>Colorado Springs, Colorado</i>	October 9-11, 1947
Windsor Hotel, <i>Montreal, Canada</i>	September 20-28, 1948
Benson Hotel, <i>Portland, Oregon</i>	October 25-27, 1949
Mayo Clinic, <i>Rochester, Minnesota</i>	September 28-30, 1950
Shamrock Hotel, <i>Houston, Texas</i>	October 4-6, 1951
Waldorf Astoria Hotel, <i>New York City</i>	Sept. 29 - October 1, 1952
Biltmore Hotel, <i>Santa Barbara, California</i>	October 12-14, 1953
Broadmoor Hotel, <i>Colorado Springs, Colorado</i>	October 21-23, 1954
The Homestead, <i>Hot Springs, Virginia</i>	October 27-29, 1955
Camelback Inn, <i>Phoenix, Arizona</i>	November 8-10, 1956
The Cloister, <i>Sea Island, Georgia</i>	November 11-13, 1957
The Royal York Hotel, <i>Toronto, Canada</i>	November 6-8, 1958
Del Monte Lodge, <i>Pebble Beach, California</i>	October 18-21, 1959
Hotel Sheraton Plaza, <i>Boston, Massachusetts</i>	October 5-8, 1960
Royal Orleans, <i>New Orleans, Louisiana</i>	November 7-10, 1962
El Mirador, <i>Palm Springs, California</i>	October 23-26, 1963

The American Academy of Neurological Surgery

Founded October 28, 1938

Honorary Members

	Elected
Dr. Percival Bailey <i>1601 West Taylor St. Chicago 12, Illinois</i>	1960
Dr. Wilder Penfield <i>Montreal Neurological Institute 3801 University St. Montreal 2, Quebec, Canada</i>	1960
Dr. R. Eustace Semmes <i>899 Madison Ave. Memphis 3, Tennessee</i>	1955
Dr. R. Glen Spurling <i>405 Heyburn Bldg. Louisville 2, Kentucky</i>	1942

Senior Members

Dr. Donald F. Coburn <i>221 Plaza Time Bldg. Country Club Plaza Kansas City 2, Missouri</i>	1938
Dr. Wallace B. Hamby <i>Cleveland Clinic 2020 East 93rd St. Cleveland 6, Ohio</i>	1941
Dr. Olan R. Hyndman <i>Veterans Administration Hospital Iowa City, Iowa</i>	1941

Corresponding Members

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Dr. Kristian Kristiansen <i>Oslo Kommune Ullevål Sykehus Oslo, Norway</i>	1962

Active Members

	Elected
Dr. Eben Alexander, Jr. <i>Bowman Gray Sch. of Medicine Winston-Salem 7, North Carolina</i>	Betty 521 Westover Ave. Winston-Salem, North Carolina 1950
Dr. George S. Baker <i>200 First Street, SW Rochester, Minnesota</i>	Enid Salem Road, Route 1 Rochester, Minnesota 1940
Dr. H. Thomas Ballantine, Jr. <i>Massachusetts General Hospital Boston 14, Massachusetts</i>	Elizabeth 30 Embankment Rd. Boston 14, Massachusetts 1951
Dr. William F. Beswick <i>685 Delaware Avenue Buffalo 9, New York</i>	Phyllis 59 Ashland Avenue Buffalo, New York 1949
Dr. Edwin B. Boldrey <i>University of Calif. Medical School San Francisco 22, California</i>	Helen 924 Hayne Road Hillsborough, California 1941
Dr. E. Harry Botterell <i>Faculty of Medicine Queen's University Kingston, Ontario, Canada</i>	Margaret Apt. 601, 150 Balmoral Ave. Toronto, Ontario, Canada 1938
Dr. Spencer Braden <i>1652 Hanna Building Cleveland 15, Ohio</i>	Mary 2532 Arlington Road Cleveland Heights, Ohio Founder
Dr. F. Keith Bradford <i>435 Hermann Professional Bldg. 6410 Fannin Street Houston 25, Texas</i>	Byra 3826 Linklea Drive Houston 25, Texas 1938
Dr. Howard A. Brown <i>Franklin Hospital 14th and Noe Streets San Francisco 14, California</i>	Dorothy 127 San Pablo Avenue San Francisco, California 1939
Dr. Harvey Chenault <i>200 West Second Street Lexington 6, Kentucky</i>	Margaret 2105 Nicholasville Road Lexington, Kentucky 1949
Dr. William F. Collins, Jr. <i>Medical College of Virginia 1200 E. Broad St. Richmond 19, Virginia</i>	Gwen 5105 W. Cary St. Richmond 27, Virginia 1963
Dr. Edward W. Davis <i>806 S.W. Broadway Portland 5, Oregon</i>	Barbara 1714 N.W. 32nd Avenue Portland 10, Oregon 1949
Dr. Richard L. De Saussure <i>Suite 101 B 20 S. Dudley Street Memphis, Tennessee</i>	Phyllis 74 Pinehurst Memphis 17, Tennessee 1962
Dr. Charles G. Drake <i>450 Central Avenue, Suite 301 London, Ontario, Canada</i>	Ruth R.R. 3, Medway Heights London, Ontario, Canada 1958
Dr. Francis A. Echlin <i>164 East 74th St. New York 21, New York</i>	Letitia 164 East 74th Street New York 21, New York 1944

		Elected
Dr. Dean H. Echols <i>Ochsner Clinic</i> 3503 Prytania Street New Orleans, Louisiana	Fran 1428 First Street New Orleans 13, Louisiana	Founder
Dr. Arthur R. Elvidge Montreal Neurological Institute 3801 University Street Montreal 2, Quebec, Canada	1465 Bernard Avenue, West Outremont, Quebec, Canada	1939
Dr. Theodore C. Erickson University Hospitals 1300 University Avenue Madison 6, Wisconsin		1940
Dr. Joseph P. Evans University of Chicago Clinics 950 East 59th Street Chicago 37, Illinois	Hermene 1234 East 56th Street Chicago 37, Illinois	Founder
Dr. William H. Feindel Montreal Neurological Institute 3801 University Street Montreal 2, Canada	Faith 492 Argyle Avenue Westmount, Province of Quebec Canada	1959
Dr. Robert G. Fisher Hitchcock Clinic Hanover, New Hampshire	Constance 11 Ledyard Lane Hanover, New Hampshire	1957
Dr. Eldon L. Foltz Div. of Neurosurgery University Hospital Seattle 5, Washington	Catherine 3018 E. Laurelhurst Drive Seattle 5, Washington	1960
Dr. John D. French The Medical Center University of California Los Angeles 24, California	Dorothy 1809 Via Visalia Palos Verdes Estates, California	1951
Dr. Lyle A. French University of Minnesota Hospitals Minneapolis 14, Minnesota	Gene 85 Otis Lane St. Paul 4, Minnesota	1954
Dr. James G. Galbraith 909 S. 18th Street Birmingham 5, Alabama	Peggy 4227 Altamont Road Birmingham 13, Alabama	1947
Dr. Everett G. Grantham 405 Heyburn Building Louisville 2, Kentucky	Mary Carmel 410 Mockingbird Hill Road Louisville 7, Kentucky	1942
Dr. John R. Green Park Central Medical Bldg. 550 West Thomas Road Phoenix, Arizona	Georgia 88 North Country Club Drive Phoenix, Arizona	1953
Dr. James Greenwood, Jr. 1117 Hermann Prof. Bldg. 6410 Fannin Street Houston 25, Texas	Mary 3394 Chevy Chase Blvd. Houston 19, Texas	1952
Dr. Wesley A. Gustafson First National Bank Bldg. McAllen, Texas	Jennie North Ware Road R.R. No 1, Box 296-A McAllen, Texas	1942

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Dr. Hannibal Hamlin <i>270 Benefit Street Providence 3, Rhode Island</i>	Margaret <i>270 Benefit Street Providence, Rhode Island</i>	1948
Dr. John W. Hanbery <i>Division of Neurosurgery Stanford Medical Center Palo Alto, California</i>	Shirley <i>70 Mercedes Lane Atherton, California</i>	1959
Dr. George J. Hayes <i>Box 236, Walter Reed Hospital Washington 12, D.C.</i>	Catherine <i>6932 - 15th Street, N.W. Washington 12, D.C.</i>	1962
Dr. Jess D. Herrmann <i>525 Northwest Eleventh Street Oklahoma City 3, Oklahoma</i>	Mary Jo <i>1604 Glenbrook Terrace Oklahoma City 14, Oklahoma</i>	1938
Dr. Henry L. Heyl <i>Hitchcock Foundation Hanover, New Hampshire</i>	Katharine <i>Norwich, Vermont</i>	1951
Dr. William S. Keith <i>Toronto Western Hospital 399 Bathurst Street Toronto 2B, Ontario, Canada</i>	Eleanor <i>55 St. Leonardi Crescent Toronto 12, Ontario, Canada</i>	Founder
Dr. Robert B. King <i>University Hospital Upstate Medical Center Syracuse 10, New York</i>	Molly <i>2 Clara Road Fayetteville, New York</i>	1958
Dr. Raeburn Llewellyn <i>Tulane University 1430 Tulane Avenue New Orleans, Louisiana</i>	Seleta <i>15 Colonial Club Drive New Orleans 23, Louisiana</i>	1963
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Dr. Donald D. Matson <i>300 Longwood Avenue Boston 15, Massachusetts</i>	Dorothy <i>44 Circuit Road Chestnut Hill 67, Massachusetts</i>	1950
Dr. Frank H. Mayfield <i>506 Oak Street Cincinnati 19, Ohio</i>	Queence <i>3519 Principio Ave. Cincinnati 26, Ohio</i>	Founder
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Dr. Robert L. McLaurin <i>Division of Neurosurgery Cincinnati General Hospital Cincinnati 29, Ohio</i>	Kathleen <i>2461 Grandin Road Cincinnati 8, Ohio</i>	1955

		Elected
Dr. William F. Meacham <i>Vanderbilt Hospital Nashville 5, Tennessee</i>	Alice <i>3513 Woodmont Blvd. Nashville 12, Tennessee</i>	1952
Dr. Edmund J. Morrissey <i>450 Sutter Street, Suite 520 San Francisco 8, California</i>	Kate <i>2700 Vallejo Street San Francisco 23, California</i>	1941
Dr. John F. Mullan <i>950 E. 59th Street Chicago 37, Illinois</i>		1963
Dr. Francis Murphey <i>Suite 101-B, Baptist Medical Bldg. 20 South Dudley Memphis 3, Tennessee</i>	Roder <i>1856 Autumn Avenue Memphis, Tennessee</i>	Founder
Dr. Frank E. Nulsen <i>Division of Neurosurgery University Hospitals 2065 Adelbert Road Cleveland 6, Ohio</i>	Ginny <i>21301 Shaker Blvd. Shaker Heights 22, Ohio</i>	1956
Dr. Guy L. Odom <i>Duke University School of Medicine Durham, North Carolina</i>	Suzanne <i>2812 Chelsea Circle Durham, North Carolina</i>	1946
Dr. J. Lawrence Pool <i>710 West 168th Street New York 32, New York</i>	Angeline <i>Closter Dock Road Alpine, New Jersey</i>	1940
Dr. Robert W. Porter <i>5901 E. 7th Street Long Beach 4, California</i>		1962
Dr. Robert Pudenz <i>744 Fairmount Avenue Pasadena 1, California</i>	Mary Ruth <i>3110 San Pasqual Pasadena 10, California</i>	1943
Dr. John Raaf <i>1010 Medical Dental Bldg. Portland 5, Oregon</i>	Lorene <i>390 S.W. Edgecliff Road Portland 19, Oregon</i>	Founder
Dr. Aidan A. Raney <i>2010 Wilshire Blvd. Los Angeles 57, California</i>	Mary <i>125 N. Las Palmas Los Angeles 5, California</i>	1946
Dr. Theodore B. Rasmussen <i>Montreal Neurological Institute 3801 University Street Montreal 2, Quebec, Canada</i>	Catherine <i>29 Surrey Drive Montreal 16, Quebec, Canada</i>	1947
Dr. David L. Reeves <i>316 West Junipero Street Santa Barbara, California</i>		1939
Dr. R.C.L. Robertson <i>437 Hermann Professional Bldg. 6410 Fannin Street Houston 25, Texas</i>	Marjorie <i>5472 Lynbrook Drive Houston, Texas</i>	1946
Dr. Stuart N. Rowe <i>302 Iroquois Building 3600 Forbes Street Pittsburgh 13, Pennsylvania</i>	Elva <i>6847 Reynolds Street Pittsburgh 8, Pennsylvania</i>	1938

Elected

Dr. Henry G. Schwartz 600 South Kingshighway St. Louis 10, Missouri	Reedie 2 Briar Oak, Ladue St. Louis 24, Missouri	1942
Dr. William B. Scoville 85 Jefferson Street Hartford 14, Connecticut	Helene 334 North Steele Road West Hartford, Connecticut	1944
Dr. C. Hunter Shelden 744 Fairmount Avenue Pasadena 1, California	Elizabeth 1345 Bedford Road San Marino, California	1941
Dr. Samuel R. Snodgrass John Sealy Hospital University of Texas Medical Branch Galveston, Texas	Margaret 1405 Harbor View Drive Galveston, Texas	1939
Dr. Hendrik J. Svien 200 First St., S.W. Rochester, Minnesota	Nancy 827 Eighth Street, S.W. Rochester, Minnesota	1957
Dr. Homer S. Swanson 384 Peachtree Street, N.E. Atlanta 3, Georgia	LaMyra 1951 Mt. Paran Road, N.W. Atlanta, Georgia	1949
Dr. William H. Sweet Massachusetts General Hospital Boston 14, Massachusetts	Mary 35 Chestnut Place Brookline 46, Massachusetts	1950
Dr. Alfred Uihlein 200 First Street, S.W. Rochester, Minnesota	Ione 21 Skyline Drive Rochester, Minnesota	1950
Dr. A. Earl Walker Johns Hopkins Hospital Division of Neurological Surgery 601 N. Broadway Baltimore 5, Maryland	Terrye 6007 Lakehurst Drive Baltimore 10, Maryland	1938
Dr. Exum Walker Suite 423, 340 Boulevard, N.E. Atlanta 12, Georgia	Frances 1819 Greystone Road, N.W. Atlanta, Georgia	1938
Dr. Arthur A. Ward, Jr. University of Washington School of Medicine Division of Neurosurgery Seattle 5, Washington	Janet 3922 Belvoir Place Seattle, Washington	1953
Dr. Thomas A. Weaver 146 Wyoming Street Dayton, Ohio	Mary 868 W. Alexandersville-Bellbrook Rd. Dayton 59, Ohio	1943
Dr. W. Keasley Welch 4200 E. Ninth Avenue Denver 20, Colorado	Elizabeth 744 Dexter Street Denver, Colorado	1957
Dr. Benjamin B. Whitcomb 85 Jefferson Street Hartford 14, Connecticut	Margaret 38 High Farms Road West Hartford, Connecticut	1947
Dr. Barnes Woodhall Duke University School of Medicine Durham, North Carolina	Frances 4006 Dover Road, Hope Valley Durham, North Carolina	1941

Deceased Members

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Dr. Winchell McK. Craig <i>Rochester, Minnesota</i>	(Honorary) 2-12-60	1942
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Dr. Kenneth G. McKenzie <i>Toronto, Ontario, Canada</i>	(Honorary) 2-11-64	1960
Dr. John M. Meredith <i>Richmond, Virginia</i>	(Active) 12-19-62	1946
Dr. W. Jason Mixer <i>Woods Hole, Massachusetts</i>	(Honorary) 3-16-58	1951
Dr. Rupert B. Raney <i>Los Angeles, California</i>	(Active) 11-28-59	1939
Dr. O. William Stewart <i>Montreal, Quebec, Canada</i>	(Corresponding)	1948



