



**BRITISH SOCIETY
OF
NEUROLOGICAL SURGEONS
AND
AMERICAN ACADEMY
OF
NEUROLOGICAL SURGERY**

BERMUDA

NOVEMBER 6-9, 1974

**SOCIETY OF
BRITISH
NEUROLOGICAL SURGEONS**

AND

**AMERICAN ACADEMY
OF
NEUROLOGICAL SURGERY**

JOINT MEETING

**SOUTHAMPTON PRINCESS HOTEL
SOUTHAMPTON, BERMUDA**

NOVEMBER 6-9, 1974

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OF
BRITISH NEUROLOGICAL SURGEONS**

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PROGRAM 1974

WEDNESDAY, NOVEMBER 6

- 12 - 6:00 p.m. Registration
Atlantic Lobby
- 6:30 - 8:30 p.m. Reception and Cocktail Party
Atlantic Lobby

THURSDAY, NOVEMBER 7

- 8:00 a.m. - 5:00 p.m. Registration
Atlantic Lobby
- 8:15 a.m. - 12:00 noon Scientific Session
Atlantic Room
- 12:00 noon Lunch
Imperial Room
- 1:30 - 5:00 p.m. Scientific Session
Atlantic Room
- 5:00 - 6:00 p.m. Business Meeting
King Room
- 7:00 p.m. Beach Party—Dinner and
Entertainment—*Beach Club*

FRIDAY, NOVEMBER 8

- 8:00 a.m. - 5:00 p.m. Registration
Atlantic Lobby
- 8:30 a.m. - 12:00 noon Scientific Session
Atlantic Room
- 6:30 - 7:30 p.m. Cocktails
Atlantic Lobby
- 7:30 p.m. Dinner Dance
Atlantic Room

SATURDAY, NOVEMBER 9

- 8:30 a.m. - 12:00 noon Scientific Session
Atlantic Room
- 12:00 noon - 1:00 p.m. Business Meeting
King Room

SCIENTIFIC PROGRAM

THURSDAY MORNING, NOVEMBER 7

Moderator, Benjamin B. Whitcomb

8:20 a.m.

Welcoming Remarks and Announcements

**ANEURYSMS: SPASM, TREATMENT AND
SEQUELAE**

1

8:30 a.m.

The Successful Treatment of Delayed Arterial Spasm in the Rhesus Monkey Following Subarachnoid Hemorrhage

Christopher W. Norwood, G. Joseph Poole, Dixon Moody and Eben Alexander, Jr.

A successful treatment for delayed cerebral vasospasm following subarachnoid hemorrhage has been reported. Of our Rhesus monkey population subjected to subarachnoid hemorrhage, 62.5% developed delayed cerebral vasospasm. Seven were treated with a *beta*-adrenergic drug alone, and five of the seven responded to the drug, representing a 71% response rate. Four other monkeys with delayed cerebral vasospasm were treated with a combination of a phosphodiesterase inhibitor and a *beta*-adrenergic stimulator; 100% of these animals responded with complete relief of the delayed cerebral vasospasm.

When the two groups of monkeys were treated as one group, nine of 11 animals with delayed cerebral vasospasm had relief of the vasospasm, representing an 81% response rate. The pharmacology of *beta*-adrenergic stimulators and phosphodiesterase inhibitors is discussed and a rationale for their synergistic effect postulated.

A Study of Cerebral Arterial Spasm: *In vitro* contractile activity of various vasoactive agents on the human basilar and anterior cerebral arteries.

George S. Allen, Shelley N. Chou and L. A. French

In vitro experiments were previously reported in which a small volume chamber was used to determine the contractile activity of various vasoactive agents on canine basilar and middle cerebral arteries.¹ This report describes similar experiments carried out on human basilar and anterior cerebral arteries. Segments of arteries were removed within one hour of death from patients dying of a variety of causes including subarachnoid hemorrhage from an aneurysm. Cumulative dose-response curves were obtained for most of the agents tested including serotonin, norepinephrine, and F₂^α prostaglandin. These human arterial segments gave several grams of contraction and would react to concentrations of some of these agents as low as 1×10^{-12} Molar. Serotonin produced a 90% maximal contraction of these arterial segments at a concentration 10-30 times less than that known to be in blood. The cumulative dose-response curves for the human arteries were similar to the canine arteries; the canine cerebral artery may therefore be a good experimental model for the study of the etiology of cerebral arterial spasm.

¹Allen, George S., et.al: A Study of Cerebral Arterial Spasm, Part I, II, III. *Journal of Neurosurgery*. Vol. XXXX, No. 4, April 1974.

Hypothalamic Influence on Intracranial Arterial Spasm

Robert H. Wilkins

It is postulated that the hypothalamus plays a vital role in the development of delayed intracranial arterial spasm.

The evidence for this hypothesis is as follows. First, ruptured aneurysms on the anterior circle of Willis are more often associated preoperatively with such spasm than those on the vertebrobasilar system. Second, postoperative intracranial arterial spasm is more common after craniotomy for carotid and anterior cerebral aneurysms than for aneurysms in other locations, and is more common after craniotomy for sellar and suprasellar tumors than for

tumors in other locations. Third, subarachnoid hemorrhage is frequently accompanied by autonomic disturbances, such as electrocardiographic changes, which are reported to be more common in patients who exhibit cerebral vasospasm. And fourth, intracranial arterial spasm can be induced experimentally by the injection of hypothalamic extracts (but not cerebral cortical extracts) into the cisterna magna of dogs.

At least three mechanisms may be involved in this relationship. First, hypothalamic injury may stimulate sympathetic function, with impulses passing from the hypothalamus through the brainstem and spinal cord to the stellate and cervical ganglia and back to the cerebral vessels, or it may interfere with parasympathetic function and permit this same type of sympathetic predominance. Second, hypothalamic injury may stimulate a widespread sympathetic discharge, thus increasing the levels of circulating catecholamines, which may affect the cerebral arteries, especially those that are hypersensitive following denervation by subarachnoid hemorrhage. And third, hypothalamic injury may result in the liberation of various vasoactive chemical agents into the cerebrospinal fluid bathing the intracranial arteries. Each of these three mechanisms may act in addition to, or synergistically with the effects of blood in the intracranial subarachnoid space. Furthermore, since intracranial arterial spasm is most marked in the anterior part of the circle of Willis, such spasm may cause ischemia and further injury to the hypothalamus, thus perpetuating the period of spasm.

The clinical and experimental evidence for and against these assertions will be presented.

9:15 a.m.

Discussion of #1, 2 and 3

Guy Odom

4

9:25 a.m.

Twenty-five Years Experience with Middle Cerebral Aneurysms

John Gillingham

81 cases of middle cerebral aneurysms treated by the author. The results are shown in the following table. The majority of cases were small and were treated by the author.

5

9:45 a.m.

The "A" Principle - A New Approach to the Treatment of Intracranial Aneurysms

W. J. Atkinson

Attention is directed to the fact that the "A" principle is a new approach to the treatment of intracranial aneurysms. It is based on the fact that the majority of cases are small and are treated by the author.

6

10:05 a.m.

Late Results on Carotid Ligation for Posterior Communicating Aneurysms

Alan Richardson, Richard Winn and John Jane

Attention is directed to the fact that the results of carotid ligation for posterior communicating aneurysms are shown in the following table.

10:25 a.m.

COFFEE BREAK

Sweet - proton beam, single session Rx -
will traverse meninges, the small vessel
malformations -

7

10:50 a.m.

Surgery of Anterior Communicating Aneurysms

B.H. Dawson

*115 p. 1000
10:50 - 11:10
797 - 10:50 - 11:10*

8

11:10 a.m.

Direct Surgery of Anterior Communicating Aneurysms and its Effect on Intellect and Personality

R.P. Sengupta

OTHER FORMIDABLE VASCULAR LESIONS

9

~~11:25 a.m.~~

8:15 AM Today

Intracranial Arteriovenous Malformations: A 26 Year Experience

Edwin B. Boldrey, Byron C. Pevehouse

Over the past 26 medical record years, the authors have been involved in the care of 150 arteriovenous malformations. 132 of these were supratentorial and 18 infratentorial.

Patients under care in the earlier years were seen because of hemorrhage and had in general larger lesions than those coming to our attention more recently. This is doubtless related to the expansion of angioencephalography as a diagnostic tool during this period—especially with respect to patients with convulsive seizures, the other more common presenting symptom.

54 patients were operated upon by the authors and

2 mm silastic spheres —
7 intracranial AVM reported —

another by 17 surgeons with whom the patient had been seen in consultation. Surgical intervention was felt to be contraindicated in 40 patients and was declined though advised in the others. Radiation therapy was administered 3 times by 1000 KV source and 2 times by Synchrotron (70 thousand KV) without benefit.

Concomitant saccular aneurysm was noted 9 times, all with supratentorial lesions. There was an associated hemangioblastoma on 3 occasions.

The results of therapy and observations of the natural history of this malady as observed in the series will be discussed.

10

11:40 a.m.

Embolization in the Preparation for Surgery of Large Cerebral Arteriovenous Malformations

Bennett M. Stein, Richard A. R. Fraser, Samuel Wolpert

Embolization of small silastic emboli through a femoral arterial catheter in the treatment of arteriovenous malformations of the cerebrum and cervical region has been utilized in 13 patients. In malformations fed by the external carotid circulation embolization has been curative or significantly ameliorative. In the cerebral malformations, embolization has been utilized in five cases of large arteriovenous malformations which otherwise might have been considered inoperable. While embolization has not been curative it has significantly reduced the flow through these arteriovenous malformations making them easier to resect. The extent and location of the cerebral malformation as well as the features which made them particularly amenable to embolization as a presurgical preparation will be discussed. Technical complications encountered during surgery and the failure of embolization in one case to significantly facilitate surgery will be discussed.

The presentation is illustrated by pre- and post-embolization arteriography as well as pre- and postsurgical angiography, gross and microscopic histological specimens and operative photographs showing the location of emboli.

11:55 a.m.

Discussion of 9 and 10

Eben Alexander, Jr

Sweet -

25% unhappy about the number of
new conversion -

22% recurrence rate in pts followed

2-6 yrs -

12:00 p.m.

LUNCH

THURSDAY AFTERNOON, NOVEMBER 7

Moderator, Valentine Logue

PAIN AND NEUROSURGICAL CONTRIBUTIONS TO PHYSIOLOGY

11

1:30 p.m.

Analgesia Dolorosa Following Differential Retrogasserian Thermal or Mechanical Rhizotomy; Tactics to Decrease its Incidence

W. H. Sweet

Following differential thermal retrogasserian rhizotomy with sparing of some touch fibers there may be a state of continuing pain described in the same terms as those used by patients with the anesthesia dolorosa seen after total division of rootlets. This has occurred in patients with idiopathic trigeminal neuralgia often enough so that we stop with a smaller lesion than formerly in any patient in whom any subjective numbness gives him concern. In patients with any other cause of facial pain, analgesia dolorosa has occurred even oftener. Hence for the past 18 months we have included in the preoperative appraisal a temporary differential lidocaine block in the retrogasserian rootlets to give such a patient a trial of the feel of hypalgesia or analgesia without loss of touch. Clinical results will be described in the treatment of 1) idiopathic trigeminal neuralgia; 2) symptomatic trigeminal neuralgia; 3) periodic migrainous neuralgia and facial pain due to 4) trauma; 5) neoplasm; 6) herpes zoster and 7) no demonstrable cause. The results will be correlated with the various grades and types of sensory loss in each clinical category.

Medical Modification of Sensory Loss after Operative Denervation

Charles J. Hodge, Jr. and Robert B. King

Denervation of the head and neck by sectioning the descending tract of the trigeminal nerve, the nervus intermedius, the ninth and upper parts of tenth cranial nerves, as well as the upper cervical dorsal roots would be expected to provide adequate relief of cranial cervical pain in light of current anatomical knowledge. The results of this type of surgery are often poor, however.

Detailed sensory examination of two patients after such procedures and after having received L-DOPA, alpha-methyl-dopa and nitrous oxide reveal that the sensory loss from extensive denervation is variable in a predictable fashion. Further, the return of preoperative pain associated with administration of L-DOPA has been correlated with return of sensation to areas presumed to be completely denervated. The subjective and objective decrease in sensory loss could be reversed by alpha-methyl-dopa and nitrous oxide. We have concluded that sensory overlap via neighboring dorsal roots is more extensive than previously described in man and further that the pathways involved in return of sensation and subjective pain do not develop as a result of the denervation, but rather are always present; the quantitative amount of sensory information these overlapping pathways transmit depends in some extent on the pharmacologic balance of the individual's catecholamine metabolism, presumably reflecting subliminal interneuron activation by aminergic pathways.

Surgery for Motor Tics of the Face and Neck

William Beecher Scoville

A presentation is made of various facial tics including hemifacial, bilateral blepharo, and platysma spasm with appropriate surgical approaches for their correction.

These motor tics can be totally disabling and psychiatric and drug therapy is often of no avail. Selective microsurgical section of the proximal submastoid portion of the 7th nerve is simple and safe. A mild partial return of the tic may occur in two or more years. Operation may be repeated.

Janetta - 44 yrs @ hemispical spasm -
sponge prosthesis put between artery &
facial nerve -

These operations can be done unilaterally or bilaterally at one sitting without cosmetic scar and with a simpler approach than selective operations distal to the parotid gland.

Discussion is made of Dr. Peter Jannetta's innovative approach by microsurgical decompression (from abnormal arterial impingement) of the 7th nerve within the cerebellar angle. Dr. Jannetta will attend this meeting and hopefully may enter the discussion of this subject.

The writer has assisted Dr. Jannetta in three of his procedures and unquestionably his approach is valid and highly successful but it requires expert microsurgical technique and runs some risk of unilateral loss of hearing because of the vulnerability of the blood supply to the internal auditory artery in persons having hemifacial spasm.

Discussion is made of the surgical treatment of spastic torticollis. Stereotactic lesions of the thalamus have proved unsatisfactory. Anterior cervical rhizotomy and separate spinal accessory nerve section is the treatment of choice in intractable cases while selective unilateral sectioning of the spinal accessory nerve in the neck is sufficient in early cases.

2:30 p.m.

COFFEE BREAK

14

3:00 p.m.

Human Ventrolateral Thalamic Stimulation Improves Performance in Some Dysnomic States

George Ojemann

The effect of 2-4 day previous stimulation of the human ventrolateral thalamus during object naming was determined in 2 dysnomic states: 1) the transient dysnomia that sometimes follows ventrolateral thalamotomy, especially on the dominant side (in 11 patients), and 2) in a single case of preexisting dysnomia from a left hemispherical cerebrovascular accident. These findings were derived from a study of the acute effects of thalamic stimulation on language and memory carried out during the course of stereotaxic operations for the treatment of dyskinesias. As part of that acute study, stimulation occurred during object naming on randomly selected trials. Patients were retested 2-4 days after thalamotomy. The presence of an anomic state after thalamotomy was determined by an increase in object

naming errors at postoperative testing on those trials where no stimulation had occurred at the time of operation. In an unselected series of 11 patients showing such a dysnomia following thalamotomy, 10 showed fewer naming errors on the objects where stimulation had occurred at operation than would be expected from performance on objects without stimulation at operation ($T = 1, p < .01$). The mean error rate for naming of objects where stimulation had occurred at operation was 56% of that expected from the error rate of objects where no stimulation occurred at operation. The patient with the preexisting anomia made errors on naming of 21% of objects where no stimulation occurred at operation. No change in this percentage occurred following placement of a left pulvinar lesion for spasticity (also a residual of his stroke). However naming errors occurred on only 7.7% of the objects where stimulation had occurred at operation, 34% of the errors expected from performance on objects where stimulation had not occurred at operation. These observations suggest that human lateral thalamic stimulation at the time of input of verbal information enhanced the accuracy of identifying this same material several days later. This effect seems to be present even with the preexisting dysnomia and may prove useful in the treatment of some language disorders.

15

3:20 p.m.

Computer Mapping of Human Subcortical Sensory Pathways During Stereotaxis

R. R. Tasker, I. H. Rowe, P. Hawrlyshyn, L. W. Organ

A computer technique has been developed for on-line display of physiological data collected for target localization during stereotaxic surgery performed for the relief of intractable pain or involuntary movement. First, atlas detail for the appropriate brainstem plane is plotted in terms of stereotaxic frame coordinates, corrected for the patient's thalamic height and length. Then as the electrode is introduced and serial threshold stimulation carried out in 2 mm steps, the trajectories, stimulation sites, and thresholds are added together with the quality and body location for all responses in the form of a Woolsey-type figurine chart. Data is stored so that the results from every patient can be scanned permitting a computerized plotting on appropriate brainstem sections of all responses of any particular type ever obtained. Illustrative data will be presented charting the course of the human auditory, vestibular and somatosensory pathways.

X-ray Rx

Warfarin - anticoagulant

no improvement in glioblastoma -

? some benefit in metastatic tumours

to the brain -

EEG's showed slow waves & theta
abnormalities, usually focal in expected
brain regions - most LP's were negative -
brain scans were usually positive -
half of patients had family history of
migraine -

16

3:35 p.m.

Six Years Experience with Combination Chemotherapy for Malignant Tumors of the Neuroaxis

Wolff Kirsch

17

3:55 p.m.

Trauma-Triggered Migraine and Juvenile Head Trauma Syndromes

David Haas, Gergorio S. Pineda, Herbert Lourie

The clinical spectrum of trauma-triggered migraine (TTM) was derived from an analysis of 42 attacks in 23 patients. Attacks were grouped into 4 clinical types: I. hemiparesis; II. somnolence, irritability, and vomiting; III. blindness; IV. brain stem signs. All attacks followed mild head trauma after a latent interval, generally of 1 to 10 minutes. Forty of the 42 attacks occurred in patients under 14 years of age. Full recovery occurred after a variable time in all but one instance. This patient, and one other patient, had an angiographically demonstrable occlusion of a branch of the middle cerebral artery. In its clinical and laboratory features, TTM is similar to spontaneous neurologic migraine. TTM is a common phenomenon after head trauma in children.

18

4:10 p.m.

Surgery of Craniofacial Deformities

John A. Jane

Until approximately ten years ago the outlook for rehabilitation of a child with major craniofacial deformity involving malposition of the eyes and significant asymmetry

of facial and cranial bones was quite dismal. Although multiple attempts were often made by plastic surgeons to improve the symmetry of the features, the malposition of the foundation, especially the orbit, made the final result disappointing to the patients' families and surgeons. Similarly in the field of Neurosurgery attention has been toward prematurely fused suture lines and an effort to provide ample room for brain enlargement. Again, serious secondary deformities of the skull and face were seen developing in many of these children during the years of growth after apparently successful maintenance of brain function. Our experiences in the correction of major craniofacial anomalies have supported the encouraging efforts of a group comparison with the leadership with Dr. Paul Tessier, and because of this we have been operating at a progressively earlier age. The advantages of early approach to these anomalies is avoidance of psychological trauma, both to the patients and the parents, and in addition the possibility of more normal growth and achievement of such physiological functions as binocular fusion. In a series of over twenty cases operated under the age of six including Crouzons, encephalocetes and complex craniofacial anomalies, there has been one operative mortality. The results of the surgery on this group as well as a discussion of theories of dural and skull reconstruction will be presented as will experiences gained on a series of over thirty cases over the age of six.

19

4:30 p.m.

Dissecting Aneurysms of the Internal Carotid

James W. Correll, Ernest H. Wood, and Martin B. Camins

Dissecting aneurysms of the internal carotid may be more frequent than heretofore thought. Two patients, each with proven dissecting aneurysms of the internal carotid and treated surgically will be presented. In both, evidence indicating that the symptoms were due to repeated episodes of embolism to the brain will be demonstrated. The arteriographic findings in one case were quite different from those in the other and similar to the findings in a number of patients studied in the past where the diagnosis was not made. These latter cases will be presented, pointing out that a dissecting aneurysm should at least have been strongly suspected, possibly resulting in modification of management.

Both patients treated surgically recovered; one following resection of the lesion with end to end anastomosis of the

internal carotid and the other after ligation of the internal carotid to prevent continued embolism.

20

4:45 p.m.

Surgical Repair of High Cervical Carotid Artery Aneurysms

Howard Morgan and James T. Robertson

Aneurysm formation of the cervical carotid arteries is not a common entity although well-documented cases have been reported secondary to a variety of causes. During the past twenty years with advances in vascular surgery, surgical correction of carotid artery aneurysms has become more common with preservation of blood flow to the brain. Numerous such cases have been reported, most dealing with lesions in and about the carotid bifurcation. However, there remains a difficult group of carotid artery aneurysms to repair, those which involve the high cervical carotid arteries just below the base of the skull. The present report reviews three cases of carotid artery aneurysms and details the authors' surgical approach to high cervical carotid artery lesions. The first case was a false internal carotid artery aneurysm following a gunshot wound at the C-12 level. The second case involved surgical repair of a dissecting aneurysm of the internal carotid artery extending to the base of the skull from the carotid bifurcation. In the third case, a large aneurysmal formation of the distal internal maxillary branch of the external carotid artery was resected along with an associated arteriovenous fistula. With careful sharp dissection exposure can safely be gained anterio-laterally above the styloid process to the base of the skull. This necessitates dissection and mobilization of the superficial lobe of the parotid gland and care not to injure the surrounding cranial nerves.

FRIDAY MORNING, NOVEMBER 8

Moderator, Richard L. DeSaussure, Jr.

CEREBROSPINAL FLUID DYNAMICS AND
HYDROCEPHALUS

21

8:30 a.m.

A Mathematical Model of Cerebro-Spinal Fluid
Dynamics

A. Talalla

Bla

22

8:50 a.m.

Chronic Measurement of Epidural Pressure with an
Induction-Powered-Oscillator Transducer

H. Grady Rylander, H. Lyndon Taylor, John P. Wissinger,
Jim L. Story

An induction-powered-oscillator transducer (IPOT) was designed for the chronic measurement of epidural pressure. The IPOT combined the durability and long-lifetime of the passive-resonant-circuit type transducers with the sensitivity and telemetry capabilities of battery powered devices. The IPOT was completely implantable so no tubes or wires were required to penetrate the skin. It had a sensitivity of 2 mm H₂O, and had very low zero drift. Zero drift has been a substantial problem with most other transducers. This problem was eliminated by hermetic sealing of the transducing element. The IPOT was evaluated by implantation in eleven dogs. Epidural pressure and intraventricular pressure were measured simultaneously over periods of hours to twelve weeks. Optimal parameters for insertion were determined. Epidural pressure was found to be a linear function of intraventricular pressure with a correlation of 0.998 in both acute and chronic implants. Epidural pressure

ventricular size index -
width of portion 1 + width of portion 3
over width of skull at level of lower
end of septum pellucidum

was found to be higher than intraventricular pressure in the chronic implants because of fibrosis of the dura underneath the transducer which created a stress force on the transducer. This dural stress created a fictitiously high epidural pressure. Dural fibrosis not only caused absolute pressure differences but also decreased the measured epidural pressure response to changes in intraventricular pressure because the transducing membrane became stiff. An accurate correlation of epidural pressure with intraventricular pressure in chronic implants will depend on the use of special materials to minimize dural fibrosis. Despite the problems with dural fibrosis, the IPOT performed flawlessly and provided reliable and sensitive measurement of epidural pressure in chronic implants.

23

9:10 a.m.

Traumatic External Hydrocephalus

Walpole Lewin

[Handwritten notes in cursive script, mostly illegible]

24

9:30 a.m.

Benign Intracranial Hypertension: Results of Treatment by Dehydrating Agents

A.A. Jefferson

[Handwritten notes in cursive script, mostly illegible]

head wrapping
14 infants - 1-3 yr FU - 1.5 cm vertical
9 compensated @ 3.5 cm vertical month -
5 " @ less than " " " " &
were shunted @ on-off shunt -

on-off shunt -

6 min drainage of 1 hr.

18 children - 1-4 yr FU - 3 deaths
in pts @ meningomyelocele -

13 - now shunt independent

5 - still need on-off shunt intermittently

subtemporal decompression -

41 children - 9 mo to 6 yrs - 4-13 periods
of intermittent hydrocephalus -

Nelson

weakly echo scans instead of head wrapping
fewer cases remission rate - 1

no obstructive hydrocephalus become shunt
independent -

The Avoidance of Shunt Dependency

Joseph Ransohoff and Fred Epstein

Hydrocephalus is commonly treated by a variety of shunting procedures the goal of which is to reduce the ventricular volume to normal or near normal. While these procedures have preserved the life of many infants and children it is becoming increasingly clear that total shunt dependency with all of its sequelae is a frequent complication and the long term results have been far from satisfactory. Most authors with extensive experience in caring for these children report a 25 to 30% 'success rate' over a ten to fifteen year followup.

On the basis of extensive clinical and laboratory investigation the authors now propose a new approach in the neonate with hydrocephalus, which is designed to eliminate or minimize shunt dependency. In the first therapeutic endeavor, an attempt is made to avoid implanting a shunting system. Rather an effort is made to arrest the disease by cranial compression, with resultant increases in spinal fluid absorption.

If this form of therapy is contraindicated or unsuccessful an "on-off" shunt is inserted which drains CSF electively and an attempt is made over an extended period of time to gradually discontinue utilization of the shunting system. In other words an attempt is made to achieve a state of compensated hydrocephalus. If this is not successful and it becomes evident that the child will indeed remain dependent on the shunt an effort is made to maintain the ventricles at a larger volume than normal while preserving a minimum of 3 cm of frontal cortical mantle.

It is believed that this will make future obstructions of the ventricular end of the shunt less common and revisions easier to accomplish. When despite all of these measures the child becomes shunt dependent with small ventricles, sub-temporal craniectomy is advocated to again make the child less dependent on the shunt. With this procedure the ventricle on the side of the craniectomy increases in volume and therefore makes catheter obstruction less likely. In addition increased intracranial pressure is vented and the resultant signs and symptoms are less fulminant.

CSF: "Pulse Pressures" in Hydrocephalus

Eldon L. Foltz and Scott Lederhaus

Ventricular CSF pressures and subdural "brain" pressures have been recorded in 16 normal dogs and 2 hydrocephalic dogs as well as 10 hydrocephalic humans. The recordings were done in each instance to record the "stable" compartment conditions under these two conditions, as well as to record these compartment pressures under certain volume loading conditions which included cerebral venous volume changes, cerebral CSF volume changes, and increased resistance to skull expansion (compression of skull), and increased or decreased systemic blood pressure.

The overall goal of this study was to record the characteristics of transcerebral pressure of the pulse wave generated in the ventricle by the choroid plexus (presumptive) and to record the CSF "pulse pressure" relationship to mean CSF pressure relative to peak-to-peak pressure ranges, characteristic waveform and duration, and latency of appearance of the pulse pressure as related to the cardiac QRS complex.

Results in the normal animals show that the ventricular CSF pulse wave is proportionally increased in its peak-to-peak value with mean CSF pressure increase when this increase is secondary to intracranial venous volume increase, increased CSF volume,—but may also be increased without mean CSF pressure increase when pulse pressure falls concomitant with mean pressure until at maximum CSF release, the CSF and brain pulse pressure suddenly increase remarkably. At the same time, the pulse wave profile shows a sharp reduction in duration and loss of the diastolic polyphasic characteristics.

In the hydrocephalic study, the pulse pressure of the ventricular CSF transmits directly through brain with little alteration to the surface of the brain. Transmission is very rapid and the shape of the pulse wave is unaltered. The ventricular and subdural pressure recordings change simultaneously whenever the venous or CSF volume change occurs, and is present in *early* hydrocephalus. However in chronic, longer term hydrocephalus, this relationship was occasionally found to be absent and the ventricular space and the subdural space acted as separate compartments relative to pressure changes induced by CSF or blood volume alteration.

In infants with expansile skulls and progressing hydrocephalus, the pulse pressure wave showed a surprising *reduction* in amount when the skull was compressed, even though the mean pressure was strikingly elevated.

Conclusions from this combined laboratory-clinical study indicate that the intracranial CSF compartment acts normally as a dampening-absorbing system to modify the "water hammer" action of the pulse pressure which presumably originates from the choroid plexus in the

Malis - chondomas

9 pts operated upon trans nasally -
8 surviving & apparently complete
removal - 1 nose reflected via rhinotomy -

Rapport

extradural cobalt implantation in cats -

ventricles. This CSF compartment apparently does this in part by expanding at its limiting membranes, possibly including the pacchionian granulations. This may represent part of the so-called "ventricular compliance" which is an early compensatory factor in hydrocephalus. The venous volume of the brain also acts in a similar manner to reduce the impact of this pulse pressure on brain, a previous concept which this study supports. Since head compression in the expansile head of infants with hydrocephalus produces an increased mean pressure but a reduced pulse pressure, this may be indirect evidence supporting the concept that the amount of pulse pressure is a critical factor in the progressing ventriculomegaly of hydrocephalus since "head wrapping" has been shown to occasionally be effective in reducing advancing ventriculomegaly of hydrocephalus.

10:30 a.m.

COFFEE BREAK

27

10:50 a.m.

Tumors of the Clivus of Blumenbach

Edward S. Connolly and James Domingue

This is a review of the problem of clivus tumors based on both the recent experience at the Ochsner Clinic with clivus tumors and with a review of the world literature. The differential diagnosis, clinical course, pathology, and therapy will all be discussed. Specific problems of therapy will be discussed.

28

11:10 a.m.

Results and Complications of Translabrynthine and Transtentorial Approaches to 60 Acoustic Nerve Tumours

T.T. King

11:30 a.m.

ACADEMY AWARD

Richard L. Rapport

SATURDAY MORNING NOVEMBER 9

Moderator, John Hankinson

SURGERY OF THE SPINAL CORD

29

8:30 a.m.

Effect of Trauma on Spinal Cord Blood Flow in Monkeys

W. George Bingham, Jr.

By means of indicator fractionation techniques utilizing antipyrine ^{-14}C , spinal cord blood flow was determined in normal and bluntly traumatized spinal cords of adult male rhesus monkeys. Injury was inflicted by dropping a 20 gram weight 15 cm onto the exposed T₆ segment with dura intact. The T₂ segment was also exposed and served as uninjured control tissue. Following injury the animals were allowed to survive for varying periods from 5 minutes to 4 hours. Arterial pressure, blood gasses and endexpiratory CO₂ were monitored throughout the experiment. At the end of the experiment, T=1 min, the animal was heparinized and collection of arterial blood at one second intervals was begun in order to determine cardiac output. At T=0, twenty-five microcuries of antipyrine ^{-14}C were injected intravenously and at T=40 seconds the animal was killed by injecting a 5 ml bolus of saturated KCl. Four animals underwent laminectomy but no cord trauma and served as laminectomy controls. Eight animals had no surgery and were used to study normal flow rates in several cord segments. In both the experimental group and the unoperated control group of animals the gray matter was dissected from the white matter utilizing a dissecting microscope and separate flow rate determinations were made for the gray and white matter.

Flow rates in traumatized tissue demonstrated marked differences in regional perfusion of the white matter and gray matter. Gray matter perfusion was nearly obliterated while white matter blood flow persisted and in fact was higher than uninjured controls. Cardiac output dropped precipitously following injury and returned only to approximately half its pretrauma value during the four hour experiment. Circulatory blood volume also decreased following injury. Mean arterial blood pressure rose instantly following injury but returned to normal within 5 to 10 minutes.

Ballantyne - layer of fat on dura materally
occurs adhesions of muscle to dura after
laminectomy -

The findings do not support the concept of ischemia as a factor in white matter failure. If toxic pathobiochemical alterations are induced by trauma it may be possible to reverse these changes by exploiting the preserved white matter blood flow for chemotherapeutic intervention.

30

8:50 a.m.

A Report on 100 Cases of Syringomyelia

Studied over 15 yr period -
J. Hankinson *done in the*

31

9:10 a.m.

Syringomyelia and the Venous Drainage of the Spinal Cord

done in 1911
A.R. Taylor

32

9:30 a.m.

Spinal Dural Patch Grafts in Experimental Animals

K. Kurakawa, S. Dunsker and T. Mayfield

33

9:50 a.m.

Extention of Carcinoma in the Cervix to the Lumbar Spine

Robert G. Fisher, Steve Acker and Ralph W. Day

Carcinoma of the cervix is a lesion that is generally not considered to involve the spinal column. Two recent cases attest to the fact that this lesion due to aorta iliac node metastases may compress the lumbar vertebrae and cause paralysis. Laminectomy with decompression followed by radiation therapy alters only temporarily the course of the lesion.

The portals of irradiation are inadequate with present techniques to prevent this lesion.

34

10:05 a.m.

Myelo-vascular Complications of Cervical Rhizotomy (A Surgical Hazard with Malpractice Threat)

H. Hamlin & W. H. Sweet

During the early development of neurosurgery the rich intrinsic vascularity of the spinal cord seemed to provide an experiential margin of safety (most of us started our operative training within the spinal canal)—until the crucial importance became evident of the extrinsic collateral blood supply to the dorsolumbar and especially the cervical segments. Separate rhizotomy operations in the latter sector on two patients were competently performed by the authors of this report. Both patients intraoperatively sustained irreversible patterns of funicular tract damage, clinically consonant with infarction. Both cases generated sizeable malpractice lawsuits. Fortuitous angiographic studies on one of the patients, coupled with experimental data on myelitic blood flow, demonstrated an insidious inherent hazard that pertains to the vertebral and radicular vascularity of the human *chorda cervicalis*.

35

10:20 a.m.

Neurosurgery in Singapore

G. Baratham

Mayfield -

must remove subchondral plate in order
to get prompt interbody fusion -

10:35 a.m.

COFFEE BREAK

SOME MICROSURGICAL CONTRIBUTIONS

36

11:00 a.m.

Anterior Cervical Discectomy – A Microsurgical Approach

John M. Tew, Jr. and Frank H. Mayfield

In 1970, we decided to perform a prospective study to determine the value of iliac bone graft placement in the treatment of the herniated disc operated by the anterior interbody approach. It was agreed that only patients with minimal degenerative changes (spondylosis) and a single herniated disc would be included in the study. Subsequently, 30 young patients have been operated and followed by one of us (JMT). In order to minimize the removal of the bone subjacent to the disc, the surgical microscope has been used in all cases. The illumination and magnification provided has proved a valuable adjunct in this procedure.

This report will review the pertinent clinical features but emphasize the points of surgical technique which we have found to be of value. 16 mm. movie and microsurgical dissections will supplement the illustrations of technical points and documentation of disc herniation.

Analysis of post operative clinical and radiographic status has been achieved in all cases. We believe that this study will demonstrate that the herniated cervical disc can be effectively removed by the interbody approach; that the microsurgical technique facilitates the procedure; and that bone grafting is not essential to a successful result.

37

11:20 a.m.

Bipolar Diathermy

J.L. Robinson

Sterility During Micro-Surgery

Theodore Kurze, Martin H. Weiss, Michael L. J. Apuzzo

There is a considerable mythology as to the preferential method of preserving sterility in the operative area during microsurgery. Little or no experimental evidence exists as to the effectiveness of these various methods beyond the subjective impressions as to the frequency of wound infections. The authors performed controlled bacteriologic studies on various methods and will report the data of these experiments. In addition, it was also learned that a conventional ethylene oxide technique is highly satisfactory; the details of our experience with this technique will be discussed.

ACADEMY AWARD WINNERS

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Hubert L. Rosomoff	1956
Byron C. Pevehouse	1957
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Earle E. Crandall	1964
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John P. Kapp	1967
Yoshio Hosobuchi	1968
Gary G. Ferguson	1970
Richard L. Pressley	1971
David G. McLone	1972
Arden F. Reynolds, Jr.	1973
Richard L. Rapport	1974

ACADEMY AWARD

1974

RICHARD L. RAPPORT

**Dept. of Neurological Surgery
University of Washington Hospital
Seattle, Washington 98105**

**"The Effects of Prophylactically
Administered Diphenylhydantoin
on the Development of Chronic
Cobalt-Induced Epilepsy in
the Cat"**

GUEST LIST 1974

GUEST

HOST

✓ George S. Allen Minneapolis, Minnesota	Lyle French
✓ Edgar Bering Oxford, Maryland	Anthony Susen
✓ W. George Bingham, Jr. Columbus, Ohio	William E. Hunt
✓ Charles E. Brackett Kansas City, Kansas	R. C. Llewellyn
William Cooke Hamilton, Bermuda	Academy
✓ Brian Counsel Hamilton, Bermuda	Academy
✓ Howard S. Dunbar New York, New York	Russel H. Patterson, Jr.
✓ Stewart B. Dunsker Cincinnati, Ohio	Frank Mayfield
Gary Fischer Boston, Massachusetts	John Tew
Stevenson Flanigan Little Rock, Arkansas	Benjamin B. Whitcomb
Roberto C. Heros Newton Center, Massachusetts	William H. Sweet
✓ Edgar M. Housepian New York, New York	James Correll
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Charlottesville, Virginia

David Yashon ✓

Peter Jannetta
Pittsburgh, Pennsylvania

Hannibal Hamlin ✓

Joseph Maroon
Pittsburgh, Pennsylvania

Lyle French

Dagoberto Sosa Montalvo
Guatemala, Central America

Nicholas T. Zervas

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George Ojemann
Seattle, Washington

Robert G. Ojemann

Richard L. Rapport
Seattle, Washington

Academy ✓

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Farmington, Connecticut

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Richard Rovit
New York, New York

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Grady Rylander
San Antonio, Texas

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James H. Salmon
Springfield, Illinois

Robert McLaurin ✓

Gerard A. Sava
Stamford, Connecticut

Donald Dohn

Michael Scott
Philadelphia, Pennsylvania

Augustus McCravey

James Simmons
Memphis, Tennessee

Richard L. De Saussure

John Stubbs
Hamilton, Bermuda

Academy ✓

Clark C. Watts
Dallas, Texas

Kemp Clark

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Dean H. Echols	1938-39
Spencer Braden	1940
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Francis Murphey	1942
Frank H. Mayfield	1943
A. Earl Walker	1944
Barnes Woodhall	1946
William S. Keith	1947
Howard A. Brown	1948
John Raaf	1949
E. Harry Botterell	1950
Wallace B. Hamby	1951
Henry G. Schwartz	1952
J. Lawrence Pool	1953
Rupert B. Raney	1954
David L. Reeves	1955
Stuart N. Rowe	1956
Arthur R. Elvidge	1957
Jess D. Herrmann	1958
Edwin B. Boldrey	1959
George S. Baker	1960
C. Hunter Shelden	1961-62
Samuel R. Snodgrass	1963
Theodore B. Rasmussen	1964
Edmund J. Morrissey	1965
George Maltby	1966
Guy L. Odom	1967
James G. Galbraith	1968
Robert H. Pudenz	1969-70
William B. Scoville	1971
Robert L. McLaurin	1972
Lyle A. French	1973

PAST VICE-PRESIDENTS

Francis Murphey	1941
William S. Keith	1942
John Raaf	1943
Rupert B. Raney	1944
Arthur R. Elvidge	1946
John Raaf	1947
Arthur R. Elvidge	1948
F. Keith Bradford	1949
David L. Reeves	1950
Henry G. Schwartz	1951
J. Lawrence Pool	1952
Rupert B. Raney	1953
David L. Reeves	1954
Stuart N. Rowe	1955
Jess D. Herrmann	1956
George S. Baker	1957
Samuel R. Snodgrass	1958
C. Hunter Shelden	1959
Edmund Morrissey	1960
Donald F. Coburn	1961-62
Eben Alexander, Jr.	1963
George L. Maltby	1964
Robert Pudenz	1965
Francis A. Echlin	1966
Benjamin Whitcomb	1967
Homer S. Swanson	1968
Augustus McCravey	1969-70
Edward W. Davis	1971
John R. Green	1972
George J. Hayes	1973

PAST SECRETARY-TREASURERS

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

PAST MEETINGS OF THE ACADEMY

- Hotel Netherlands Plaza,
Cincinnati, OhioOctober 28-29, 1938
- Roosevelt Hotel,
New Orleans, LouisianaOctober 27-29, 1939
- Tudor Arms Hotel,
Cleveland, OhioOctober 21-22, 1940
- Mark Hopkins Hotel, San Francisco,
and Ambassador Hotel,
Los Angeles, California November 11-15, 1941
- The Palmer House,
Chicago, IllinoisOctober 16-17, 1942
- Hart Hotel,
Battle Creek, MichiganSeptember 17-18, 1943
- Ashford General Hospital,
White Sulphur Springs,
West VirginiaSeptember 7-9, 1944
- The Homestead,
Hot Springs, VirginiaSeptember 9-11, 1946
- Broadmoor Hotel,
Colorado Springs, Colorado October 9-11, 1947
- Windsor Hotel,
Montreal, CanadaSeptember 20-28, 1948
- Benson Hotel, Portland, OregonOctober 25-27, 1949
- Mayo Clinic,
Rochester, MinnesotaSeptember 28-30, 1950
- Shamrock Hotel, Houston, Texas October 4-6, 1951
- Waldorf-Astoria Hotel,
New York CitySeptember 29-October 1, 1952
- Biltmore Hotel,
Santa Barbara, CaliforniaOctober 12-14, 1953
- Broadmoor Hotel,
Colorado Springs, ColoradoOctober 12-14, 1953

- The Homestead,
Hot Springs, VirginiaOctober 27-29, 1955
- Camelback Inn, Phoenix, Arizona November 8-10, 1956
- The Cloister,
Sea Island, Georgia November 11-13, 1957
- The Royal York Hotel,
Toronto, CanadaNovember 6-8, 1958
- Del Monte Lodge,
Pebble Beach, CaliforniaOctober 18-21, 1959
- Hotel Sheraton Plaza,
Boston, Massachusetts October 5-8, 1960
- Royal Orleans,
New Orleans, Louisiana November 7-10, 1962
- El Mirador,
Palm Springs, CaliforniaOctober 23-26, 1963
- The Key Biscayne,
Miami, Florida November 11-14, 1964
- Terrace Hilton Hotel,
Cincinnati, OhioOctober 14-16, 1965
- Fairmont Hotel & Tower,
San Francisco, CaliforniaOctober 17-19, 1966
- The Key Biscayne,
Miami, Florida November 8-11, 1967
- Broadmoor Hotel,
Colorado Springs, Colorado October 6-8, 1968
- St. Regis Hotel, New York City September 21, 1969
- Camino Real Hotel,
Mexico City November 18-21, 1970
- Sahara-Tahoe Hotel,
Stateline, NevadaSeptember 26-29, 1971
- New College, Oxford, EnglandSeptember 4-7, 1972
- Huntington-Sheraton Hotel,
Pasadena, California November 14-17, 1973

1974
MEMBERSHIP LIST
THE AMERICAN ACADEMY
OF NEUROLOGICAL SURGERY

HONORARY - 5	Elected
Guy Lazorthes 26 Rue d'Auriol 31 Toulouse, France	1973
Gosta Norlen Neurokirurgiska Kliniken Sahlgrenska Sjukhus Goteborg, SV Sweden	1973
Sixto Obrador (Alcalde) Eduardo Dato 23 Madrid 10, Spain	1973
Wilder Penfield Montreal Neurological Institute 3801 University Street Montreal 2, Quebec, Canada	1960
R. Eustace Semmes 20 S. Dudley Street, Suite 101-B Memphis, Tennessee 38103	1955
SENIOR MEMBERS - 32	
George S. Baker 200 First Street, S.W. Rochester, Minnesota 55901	1940
E. Harry Botterell Faculty of Medicine Queens University Kingston, Ontario, Canada	1938

- Howard A. Brown 1939
2001 Union Street
San Francisco, California 94123
- Harvey Chenault 1949
2370 Nicholasville Road
Lexington, Kentucky 40503
- Donald F. Coburn 1938
6400 Prospect Avenue, Room 204
Kansas City, Missouri 64132
- ✓ Edward W. Davis 1949
Providence Medical Office Bldg.
545 N.E. 47th Avenue
Portland, Oregon 97213
- Francis A. Echlin 1944
100 East 77th Street
New York, New York 10021
- Arthur Elvidge 1939
Montreal Neurological Institute
3801 University Street
Montreal 2, Quebec, Canada
- Theodore C. Erickson 1940
University Hospitals
1300 University Avenue
Madison, Wisconsin 53706
- Joseph P. Evans Founder
Edificio El Dorado
Cr. 34 x Calle 11, Apt. 304
"El Poblado"
Medellin, Columbia S.A.
- ✓ Everett G. Grantham 1942
234 East Gray Street
Louisville, Kentucky 40202
- ✓ James Greenwood, Jr. 1952
1117 Hermann Professional Building
6410 Fannin Street
Houston, Texas 77025

Wesley A. Gustafson Rt. 1, Box 125 Sewall's Point Jensen Beach, Florida 33457	1942
Wallace B. Hamby 3001 N.E. 47th Court Fort Lauderdale, Florida 33308	1941
Hannibal Hamlin 270 Benefit Street Providence, Rhode Island 02903	1948 ✓
Jess D. Herrmann P.O. Box 135 Mountain Pine, Arkansas 71956	1938
Henry L. Heyl Dartmouth Medical School Hanover, New Hampshire 03755	1951
William S. Keith Toronto Western Medical Building Suite 207 25 Leonard Avenue Toronto, Ontario, Canada	Founder
George L. Maltby 31 Bramhall Street Portland, Maine 04102	1942 ✓
Augustus McCravey 1010 East Third Street Chattanooga, Tennessee 37403	1944 ✓
Edmund J. Morrissey 450 Sutter Street, Suite 1504 San Francisco, California 94108	1941
Francis Murphey 20 S. Dudley Street, Suite 101-B Memphis, Tennessee 38103	Founder

- J. Lawrence Pool** 1940
Box 31
West Cornwall
Connecticut 06796
- Robert H. Pudenz** 1943
744 Fairmount Avenue
Pasadena, California 91105
- Stuart N. Rowe** 1938
302 Iroquois Building
3600 Forbes Street
Pittsburgh, Pennsylvania 15213
- C. Hunter Shelden** 1941
744 Fairmount Avenue
Pasadena, California 91105
- Samuel R. Snodgrass** 1939
Rural Route 3, Box 13
Nashville, Indiana 47448
- ✓ **Homer S. Swanson** 1949
1938 Peachtree Road, N.W.
Atlanta, Georgia 30309
- A. Earl Walker** 1938
Johns Hopkins Hospital
Division of Neurological Surgery
601 N. Broadway
Baltimore, Maryland 21205
- Exum Walker** 1938
490 Peachtree Street, N.E.
Atlanta, Georgia 30308
- ✓ **Thomas A. Weaver, Jr.** 1943
146 Wyoming Street
Dayton, Ohio 45409
- Barnes Woodhall** 1941
Duke University Medical Center
Durham, North Carolina 27706

CORRESPONDING MEMBERS - 17

- Karl August Bushe** 1972
Chirurgie Universitat
Gosler-Strasse 10
34 Goettingen, W. Germany
- Fernando Cabieses** 1966
Instituto Peruano de Fomento Educativo
Av. Arenales 371, Of. 501
Apartado 5254
Lima, Peru
- Juan Cardenas y C.** 1966
Av. Insurgentes Sur 594
Mexico, D.F.
- Juan C. Christensen** 1970
Ave. Quintana 474 8° A
Buenos Aires, Argentina
- Giuseppe Dalle Ore** 1970
Dipartimento di Neurochirurgia
Ospedale Maggiore 37100
Verona, Italy
- Hans E. Diemath** 1970
Prim. Univ. Doz.
Neurochir. Abt. d. Landersnervenklinik
Salzburg, 5020, Austria
- John Gillingham** 1962 ✓
Boraston House, Ravelson
Edinburgh 4, Scotland
- John Hankinson** 1973 ✓
Department of Neurosurgery
Newcastle General Hospital
Newcastle-upon-Tyne 4
England
- Kenneth G. Jamieson** 1970
131 Wickham Terrace
Brisbane, Queensland,
Australia 4000

Katsutoshi Kitamura 1970
University Kyushu Hospital
Faculty of Medicine
Fukuoka, Japan

Kristian Kristiansen 1962
Oslo Kommune
Ullval Sykehus
Oslo, Norway

Lauri Laitinen 1971
Neurokirurgiska Kliniken
Toolo Sjukhus
Helsinki, Finland

✓ Walpole S. Lewin 1973
Department of Neurosurgery
Addenbrooke's Hospital
Hills Road
Cambridge, England

William Luyendijk 1973
Pr. Bernhardlaan 60
Oegstgeest, Netherlands

B. Ramamurthi 1966
2nd Main Road, C.I.T. Colony
Madras 4, India

Charas Suwanwela 1972
Chulalongkorn Hospital Medical School
Bangkok, Thailand

Kjeld Vaernet 1970
Rigshospitalets Neurokirurgiske afdeling
Tagensvej 18, 2200
Copenhagen, N., Denmark

ACTIVE MEMBERS — 3

✓ Eben Alexander, Jr. (Betty) 1950
Bowman-Gray School of Med.
Winston-Salem, N.C. 27103
(1941 Georgia Ave., Winston-Salem, N.C. 27104)

- James R. Atkinson (Lona) 1970 ✓
 302 W. Thomas Road
 Phoenix, Ariz. 85013
 (5806 East Lewis Ave., Scottsdale, Ariz. 85257)
- H. Thomas Ballantine, Jr. (Elizabeth) 1951 ✓
 Massachusetts General Hosp.
 Boston, Massachusetts 02114
 (30 Embankment Road, Boston, Mass. 02114)
- Giles Bertrand (Louise) 1967 ✓
 Montreal Neurological Inst.
 3801 University St.
 Montreal, Quebec, Canada
 (385 Lethbridge, Montreal 16, Quebec, Canada)
- Edwin B. Boldrey (Helen) 1941 ✓
 University of Calif. Hosp.
 3rd Avenue & Parnassus
 San Francisco, Calif. 94143
 (924 Hayne Road, Hillsborough, Calif. 94010)
- Barton A. Brown (Martha) 1968 ✓
 2001 Union Street
 San Francisco, Calif. 94123
 (1648 8th Avenue, San Francisco, Calif. 94122)
- Gale G. Clark, Capt. USN MC (Marian) 1970 ✓
 12621 Brookpark Road
 Oakland, California 94619
 (12621 Brookpark Road, Oakland, Calif. 94619)
- W. Kemp Clark (Fern) 1970
 5323 Harry Hines Blvd.
 Dallas, Texas 75235
 (3909 Euclid Avenue, Dallas, Texas 75205)
- William F. Collins, Jr. (Gwen) 1963 ✓
 Yale Univ. School of Med.
 333 Cedar Street
 New Haven, Conn. 06510
 (403 St. Ronan Street, New Haven, Conn. 06511)
- Edward S. Connolly (Elise) 1973 ✓
 1423 State Street
 New Orleans, Louisiana 70118
 (1423 State St., New Orleans, LA 70118)

- ✓ James W. Correll (Cynthia) 1966
 Neurological Institute
 710 West 168th St.
 New York, New York 10032
 (Algonquin Trail, Saddle River, NJ 07458)
- Courtland H. Davis, Jr. (Marilyn) 1967
 Bowman-Gray School of Med.
 Winston-Salem, N.C. 27103
 (921 Goodwood Rd., Winston-Salem, NC 27106)
- ✓ Richard L. DeSaussure (Phyllis) 1962
 20 S. Dudley Street
 Memphis, Tenn. 38103
 (4290 Heatherwood Lane, Memphis, Tennessee 38117)
- ✓ Donald F. Dohn (Betty) 1968
 2020 East 93rd Street
 Cleveland, Ohio 44106
 (3010 Huntington Road, Shaker Heights, Ohio 44120)
- R. M. Peardon Donaghy (Frances) 1970
 Mary Fletcher Hospital
 Burlington, Vermont 05401
 (466 S. Prospect St., Burlington, Vermont 05401)
- Charles G. Drake (Ruth) 1958
 111 Waterloo Street, Suite 211
 London, Ontario, Canada
 (R.R. 3, Medway Heights, London, Ontario, Canada)
- Dean H. Echols (Fran) Founder
 Ochsner Clinic
 1514 Jefferson Highway
 New Orleans, Louisiana 70121
 (1550 Second Street, New Orleans, Louisiana 70130)
- George Ehni (Velaire (Larry)) 1964
 1531 Hermann Professional Bld.
 6410 Fannin Street
 Houston, Texas 77025
 (16 Sunset, Houston, Texas 77025)
- ✓ William H. Feindel (Faith) 1959
 Montreal Neurological Institute
 3801 University Street
 Montreal, Quebec, Canada
 (39 Thornhill Ave., Westmount, P.Q., Canada)

- Robert G. Fisher (Constance) 1957 ✓
~~800 N. E. 13th Street~~
~~Oklahoma City, Oklahoma 73104~~
~~(107 Lake Atuma Drive, Oklahoma City, Okla. 73121)~~
- Eldon L. Foltz (Catherine) 1960 ✓
 Chairman, Div. of Neurosurgery
 Univ. of Calif. School of Med.
 Irvine, California 92664
 (2480 Monaco Drive, Laguna Beach, Calif. 35213)
- John D. French (Dorothy) 1951
 The Center for the Health Sciences
 University of California
 Los Angeles, Calif. 90024
 (12841 Sunset Blvd., Los Angeles, Calif. 90049)
- Lyle A. French (Gene) 1954
 University of Minn. Medical Center
 Minneapolis, Minn. 55455
 (85 Otis Lane, St. Paul, Minn. 55104)
- James G. Galbraith (Peggy) 1947
 University of Alabama Med. Cent.
 1919 Seventh Avenue, South
 Birmingham, Alabama 35233
 (4227 Altamont Road, Birmingham, Ala. 34213)
- John T. Garner (Barbara) 1971 ✓
 744 Fairmount Avenue
 Pasadena, Calif. 91105
 (3075 Monterey Rd., San Marino, Calif. 91108)
- Henry Garretson (Marianna) 1973 ✓
 Dept. of Neurosurgery
 University of Louisville
 Louisville, Kentucky
 (517 Tiffany Lane, Louisville, Kentucky 40207)
- Sidney Goldring (Lois) 1964 ✓
 Barnes Hospital Plaza
 Division of Neurosurgery
 St. Louis, Missouri 63110
 (11430 Conway Road, St. Louis, Missouri 63131)
- Philip D. Gordy (Elizabeth Ann (Lisa)) 1968
 1025 Walnut Street
 Philadelphia, Pennsylvania 19107
 (420 N. Rose Lane, Haverford, Pennsylvania 19041)

- ✓ **John R. Green (Georgia) 1953**
Barrow Neurological Institute
302 West Thomas Street
Phoenix, Arizona 85013
(2524 E. Crittendon Ln., Sutton Place, Phoenix, Ariz. 85016)
- ✓ **John W. Hanbery (Shirley) 1959**
Division of Neurosurgery
Stanford Medical Center
Palo Alto, California 94304
(70 Mercedes Lane, Atherton, Calif. 94025)
- ✓ **Maj. Gen. George S. Hayes, MC USA (Catherine) 1962**
Principal Deputy
Office of the Assistant Sec. of
Defense (Health & Environment)
Washington, D.C. 20301
(303 Skyhill Road, Alexandria, VA 22314)
- ✓ **E. Bruce Hendrick (Gloria) 1968**
Hospital for Sick Children
555 University Avenue
Toronto, Ontario, Canada
(63 Leggett Avenue, Weston, Ontario, Canada)
- ✓ **William E. Hunt (Charlotte) 1970**
410 West 10th Avenue
Columbus, Ohio 43210
(1000 Urlin Avenue, Columbus, Ohio 43212)
- ✓ **Robert B. King (Molly) 1958**
University Hospital
Upstate Medical Center
750 East Adams Street
Syracuse, New York 13210
(408 Maple Drive, Fayetteville, NY 13066)
- ✓ **Wolff M. Kirsch (Marie-Claire) 1971**
University of Colorado Medical Center
Denver, Colorado 80220
(635 Bellaire, Denver, Colorado 80220)
- ✓ **David G. Kline (Carol) 1972**
Louisiana State Univ. Medical Center
1542 Tulane Avenue
New Orleans, Louisiana 70012
(46 Thrasher St., Lake Vista, New Orleans, LA 70124)

- Robert S. Knighton (Louise) 1966
Henry Ford Hospital
2799 W. Grand Blvd.
Detroit, Michigan 48202
(27486 Lathrup Blvd., Lathrup Village, Mich. 48075)
- Theodore Kurze 1967 ✓
Los Angeles County – U.S.C. Medical Center
1200 North State Street
Suite 5046
Los Angeles, California 90033
(13856 Bora Bora Way, #306-C,
Marina Del Rey, California 90291)
- Thomas W. Langfitt (Carolyn) 1971
Hospital of the Univ. of Penn.
34th and Spruce Streets
Philadelphia, Penn. 19104
(71 Merbrook Bend, Merion, Pennsylvania 19066)
- Raeburn C. Llewellyn (Carmen) 1963 ✓
Tulane University
1430 Tulane Avenue
New Orleans, La. 70012
(32 Versailles Blvd., New Orleans, La. 70124)
- William M. Lougheed (Grace Eleanor) 1962
Medical Arts Building, Suite 430
170 St. George St.
Toronto 5, Ontario, Canada
(67 Ridge Drive, Toronto, Ontario, Canada)
- Herbert Lourie (Betty) 1965 ✓
713 East Genesee Street
Syracuse, New York 13210
(101 Thomas Road, DeWitt, New York 13214)
- John J. Lowrey (Catherine (Kay)) 1965 ✓
Straub Clinic
888 S. King Street
Honolulu, Hawaii 96813
(2299-B Round Top Dr., Honolulu, Hawaii 96822)
- Ernest W. Mack (Roberta) 1956 ✓
505 S. Arlington Avenue, Suite 212
Reno, Nevada 89502
(235 Juniper Hill Road, Reno, Nevada 89502)
- M. Stephen Mahaley, Jr. (Janet) 1972
Duke University Med. Center
Durham, North Carolina 27706
(3940 Nottaway Road, Durham, North Carolina 27707)

- ✓ Leonard Malis (Ruth) 1973
 1176 Fifth Avenue
 New York, New York 10029
 (219-44 Peck Avenue, Hollis Hills, NY 11427)
- ✓ Frank Mayfield (Queenee) Founder
 506 Oak Street
 Cincinnati, Ohio 45219
 (1220 Roodwood Drive, Cincinnati, Ohio 45208)
- ✓ Robert L. McLaurin (Kathleen) 1955
 Division of Neurosurgery
 Cincinnati General Hosp.
 Cincinnati, Ohio 45229
 (2461 Grandin Road, Cincinnati, Ohio 45208)
- William F. Meacham (Alice) 1952
 Vanderbilt University Hosp.
 Division of Neurosurgery
 Nashville, Tenn. 37203
 (3513 Woodmount Boulevard, Nashville, Tenn. 37215)
- John F. Mullan (Vivian) 1963
 Univ. of Chicago Clinics
 Department of Neurosurgery
 950 E. 59th St.
 Chicago, Ill. 60637
 (6911 S. Bennett Ave., Chicago, Ill. 60649)
- Blaine S. Nashold, Jr. (Irene) 1967
 Duke University Medical Center
 Durham, North Carolina 27706
 (410 E. Forest Hills Blvd., Durham, North Carolina 27706)
- ✓ Frank E. Nulsen (Ginney) 1956
 Div. of Neurosurgery
 University Hospital
 2065 Adelbert Road
 Cleveland, Ohio 44106
 (31200 Fox Hollow Dr., Pepper Pike, Ohio 44124)
- ✓ Guy L. Odom (Matalaine) 1946
 Duke University Med. Center
 Durham, North Carolina 27706
 (2812 Chelsea Circle, Durham, North Carolina 27706)
- ✓ Robert G. Ojemann (Jean) 1968
 Massachusetts General Hospital
 Division of Neurological Surg.
 Boston, Mass. 02114
 (85 Nobscot Road, Weston, Mass. 02193)

- Russel H. Patterson, Jr. (Julie) 1971 ✓
 525 East 68th Street
 New York, New York 10021
 (535 East 86th St., New York, NY 10028)
- Phanor L. Perot, Jr. (Elizabeth) 1970 ✓
 Medical Univ. of South Carolina
 80 Barre St.
 Charleston, S. Carolina 29401
 (704 Willowlake Road, Charleston, S. Carolina 29407)
- Byron C. Pevehouse (Maxine) 1964 ✓
 2001 Union St.
 San Francisco, Calif. 94123
 (135 Mountain Spring Ave., San Francisco, Calif. 94114)
- Robert W. Porter (Aubrey Dean) 1962
 5901 E. 7th Street
 Long Beach, California 90804
 (5400 The Toledo, Long Beach, California 90803)
- John Raaf (Lorene) Founder
 833 S.W. 11th Avenue
 Portland, Oregon 97205
 (390 S.W. Edgecliff Road, Portland, Oregon 97219)
- Aiden A. Raney (Mary) 1946
 2010 Wilshire Blvd., Suite 203
 Los Angeles, California 90057
 (125 N. Las Palmas, Los Angeles, California 90004)
- Joseph Ransohoff II (Rita) 1965 ✓
 New York Univ. Medical Center
 500 First Avenue
 New York, New York 10016
 (140 Riverside Drive, New York, New York)
- Theodore B. Rasmussen (Catherine) 1947 ✓
 Montreal Neurological Institute
 3801 University Street
 Montreal 2, Quebec, Canada
 (29 Surrey Drive, Montreal 16, Quebec, Canada)
- David H. Reynolds (Marjorie) 1964
 1150 N.W. 14th Street, Suite 209
 Miami, Florida 33136
 (1701 Espanola Drive, Miami, Florida)

- ✓ Hugo Rizzoli (Helen) 1973
 2150 Penn Avenue, N.W.
 Washington, DC 20037
 (6100 Kennedy Drive, Kenwood, Chevy Chase, Md. 20015)
- ✓ James T. Robertson (Valeria) 1971
 20 S. Dudley
 Memphis, Tennessee 38103
 (628 N. Trezevant St., Memphis, Tennessee 38112)
- R. C. L. Robertson (Marjorie) 1946
 Shamrock Professional Building
 2210 Maroneal Boulevard, Suite 404
 Houston, Texas 77025
 (5472 Lynbrook Drive, Houston, Texas 77027)
- Richard C. Schneider (Madeleine) 1970
 C5135, Out-Pt. Building
 University Hospital
 Ann Arbor, Michigan 48104
 (2110 Hill Street, Ann Arbor, Michigan 48104)
- ✓ Henry G. Schwartz (Reedie) 1942
 Barnes Hospital Plaza
 Division of Neurological Surgery
 St. Louis, Missouri 63110
 (2 Briar Oak, Ladue, St. Louis, Missouri 63132)
- ✓ William B. Scoville (Helene) 1944
 85 Jefferson Street
 Hartford, Connecticut 06106
 (27 High Street, Farmington, Connecticut 06032)
- ✓ Bennett M. Stein (Doreen) 1970
 Department of Neurosurgery
 171 Harrison Avenue
 Boston, Massachusetts 02111
 (16 Tamarack Road, Weston, Massachusetts 02193)
- ✓ Jim L. Story (Joanne) 1972
 7703 Floyd Curl Drive
 San Antonio, Texas 78229
 (3211 Stonehaven Road, San Antonio, Texas 78230)
- Thoralf M. Sundt, Jr. (Lois) 1971
 200 First Street, S.W.
 Rochester, Minnesota 55901
 (1406 Weatherhill Court, Rochester, Minnesota 55901)

- Anthony F. Susen (Phyllis) 1965 ✓
 3600 Forbes Avenue
 Pittsburgh, Pennsylvania 15213
 (3955 Bigelow Boulevard, Pittsburgh, Pennsylvania 15213)
- William H. Sweet (Mary) 1950 ✓
 Massachusetts General Hospital
 Division of Neurological Surgery
 Boston, Massachusetts 02114
 (35 Chestnut Place, Brookline, Massachusetts 02146)
- Ronald R. Tasker (Mary) 1971 ✓
 Toronto General Hospital
 Room 121, U. W.
 Toronto, Ontario, Canada
 (12 Cluny Drive, Toronto 5, Ontario, Canada)
- John Tew, Jr. (Susan) 1973 ✓
 506 Oak Street
 Cincinnati, Ohio 45219
 (2145 East Hill Avenue, Cincinnati, Ohio 45208)
- George T. Tindall (Suzie) 1968
 Emory University School of Medicine
 Division of Neurosurgery
 Atlanta, Georgia 30322
- John Tytus (Virginia (Gina)) 1967 ✓
 Mason Clinic
 Seattle, Washington 98101
 (1000 N.W. Northwood Road, Seattle, Washington 98177)
- Alfred Uihlein (Ione) 1950
 200 First Street, S.W.
 Rochester, Minnesota 55901
 (Box 1127, Naples, Florida 33940)
- Arthur A. Ward, Jr. (Janet) 1953 ✓
 Department of Neurological Surgery
 University of Washington Hospital
 Seattle, Washington 98105
 (3922 Belvoir Place, N.E., Seattle, Washington 98105)
- W. Kasley Welch (Elizabeth) 1957 ✓
 Children's Hospital Medical Center
 300 Longwood Avenue
 Boston, Massachusetts 02115
 (25 Gould Road, Waban, Massachusetts 02168)

- ✓ Benjamin B. Whitcomb (Margaret) 1947
 85 Jefferson Street
 Hartford, Connecticut 06106
 (38 High Farms Road, West Hartford, Connecticut 06107)
- Lowell E. White, Jr. (Margie) 1971
 Professor & Chairman
 Division of Neurosciences
 Univ. of S. Alabama Medical School
 Mobile, Alabama 36688
 (912 Regency Drive West, Mobile, Alabama 36609)
- ✓ Robert Wilkins (Gloria) 1973
 Scott & White Clinic
 Temple, Texas 76501
 (3409 Aspen Trail, Temple, Texas 76501)
- Charles B. Wilson (Mary) 1966
 Department of Neurological Surgery
 University of California Medical Center
 Third and Parnassus
 San Francisco, California 94122
 (215 Round Hill Road, Tiburon, California 94920)
- ✓ Frank Wrenn (Betty) 1973
 123 Mallard Street
 Greenville, South Carolina 29601
 (712 Crescent Ave., Greenville, South Carolina 29601)
- ✓ David Yashon (Myrna) 1972
 410 W. 10th Avenue, N., #911
 Columbus, Ohio 43210
 (5735 Saranac Drive, Columbus, Ohio 43227)
- Nicholas T. Zervas (Thalia) 1972
 330 Brookline Avenue
 Boston, Massachusetts 02215
 (100 Canton Avenue, Milton, Massachusetts 02186)

DECEASED MEMBERS	Date	Elected
Dr. Percival Bailey 731 Lincoln Street Evanston, Illinois 60201	(Honorary) 8-10-73	1960
Dr. William F. Beswick Buffalo, New York	(Active) 5-12-71	1949
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Dr. W. Jason Mixer Woods Hole, Massachusetts	(Honorary) 3-16-58	1951
Dr. Rupert B. Raney Los Angeles, California	(Active) 11-28-59	1939
Dr. David L. Reeves Santa Barbara, California	(Senior) 8-14-70	1939
Dr. C. William Stewart Montreal, Quebec, Canada	(Corresponding)	1948
Dr. Glen Spurling	(Honorary) 2-7-68	1942
Dr. Hendrik Svien Rochester, Minnesota	(Active) 6-29-72	1957

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|---|---|
| <p>C. B. T. Adams, Esq., F.R.C.S.
Department of Neurological Surgery
Radcliffe Infirmary
Oxford</p> | M |
| <p>The Rt. Hon. Lord Adrian, O.M., F.R.S.
Trinity College
Cambridge</p> | E |
| <p>Professor Bashir Ahmad, F.R.C.S.
24-A, Bahawalpur House
Lahore, W. Pakistan</p> | C |
| <p>K. Lewer Allen Esq., F.R.C.S., Ch.M.
Princess Nursing Home
69, Esselen Street
Johannesburg, South Africa</p> | H |
| <p>Professor P. Almeida Lima
Centro de Estudos Egas Moniz
Avenida de Brasil 53
Lisboa, Portugal</p> | H |
| <p>Dr. F. Amaral Gomez,
Centro-De-Neurogrugia
Centro Hospitalar de Coimbra
Coimbra, Portugal</p> | H |
| <p>Professor N. Ameli
15, Khiaban Jami
Teheran, Persia (Iran)</p> | C |
| <p>J. Andrew Esq., F.R.C.S.
Department of Neurosurgery
Middlesex Hospital
London, W. 1</p> | M |

- Dr. Roman Arana-Iniguez H
 Cremona 6751
 Montevideo
 Uruguay, South America
- Professor Dr. N. B. Arias C
 Director de Estudios Quirurgicos
 Universidad de Santo Domingo
 Santo Domingo, Dominican Republic
- Roger Armour Esq., F.R.C.S. A
 88, Wymondley Road
 Hitchin, Herts, SG4 9PX
- G. Arthurs Esq., F.R.C.S. C
 149, Macquarie Street
 Sydney, N.S.W., Australia
- Dr. A. Asenjo H
 Instituto Central De Neurocirugia y Neuropatologia
 Gabbelow Cushing
 Hospital del Salvador
 Santiago, Chile
- R. L. Atkinson Esq., F.R.C.S.E. A
 Alexandra
 Wickham Tce. 4,000
 Brisbane, Queensland, Australia
- W. J. Atkinson Esq., M.S., F.R.C.S. M
 Hurstwood Park Hospital
 Haywards Heath, Sussex
- H. J. Bagnall Esq., F.R.C.S. C
 803, Waterloo Avenue
 Ottawa, Ontario, Canada
- Ian C. Bailey Esq., F.R.C.S. A
 5, Rosevale Avenue
 Drumbee, Dunmurry, Belfast, BT17 9LG
- Professor Percival Bailey E
 1601 W. Taylor Street
 Chicago, Illinois, U.S.A.
- Dr. R. A. Bailey C
 11315 - 46th Avenue
 Edmonton, Alberta, Canada

- Miss Carys Bannister, F.R.C.S.E. A
 Department of Neurological Surgery
 General Infirmary
 Great George Street, Leeds, 1
- G. Baratham Esq., F.R.C.S. A ✓
 Department of Neurosurgery & Neurology,
 Tan Tock Seng Hospital
 Singapore 11
- Professor J. J. Barcia Goyanes H
 Gran Via Marques del Turia, 62
 Valencia, Spain
- J. Bartlett Esq., F.R.C.S. A
 Brook General Hospital
 Shooters Hill Road
 London, S.E. 18
- Dr. E. A. G. N. Basha OA
 Department of Neurological Surgery
 Crumpsall Hospital
 Manchester, 8
- Dr. J. Beattie H
 Queen's College
 Cambridge
- Professor J. le Beau C
 107, Rue de l'Universite
 Paris VIIe, France
- Professor J. W. F. Beks C
 Groningen
 Oostersingel 59, Holland
- Dr. V. Benes C
 Neurosurgical Clinic
 Charles University
 Praha – Stresovice, Czechoslovakia
- A. H. M. Bennett Esq., F.R.C.S. A
 Elinlegh Court
 Stone Street
 Stellis Minnis
 Canterbury, Kent
- Dr. Claude Bertrand H
 1392 Sherbrooke Street East
 Montreal, Canada

- Michael J. Betty Esq., F.R.C.S. M
 Regional Neurological Centre
 Newcastle General Hospital
 Newcastle upon Tyne, NE4 6BE
- Dr. Y. S. Bhandhari A
 The Neurosurgical Unit
 Salford Royal Hospital
 Chapel Street
 Salford 3, Lancs
- J. Block Esq., F.R.C.S. M
 Department of Surgical Neurology
 Royal Infirmary
 Dundee, Angus, Scotland
- Professor S. M. Boctor C
 Department of Neurosurgery
 Alexandria Faculty of Medicine
 Alexandria, U.A.R.
- Professor Michael Bond M
 Department of Psychological Medicine
 Southern General Hospital
 1345, Govan Road
 Glasgow, G51 4TF
- A. E. Booth Esq., F.R.C.S. A
 34, Cressida Road
 London, N.19
- Dr. E. H. Botterell H
 Dean of the Faculty of Medicine
 Queen's University
 Kingston, Ontario, Canada
- J. Brice Esq., F.R.C.S. M
 The Wessex Region Neurological Centre
 Southampton General Hospital
 Shirley, Southampton, Hants
- M. Briggs Esq., F.R.C.S. A
 Department of Neurological Surgery
 Radcliffe Infirmary
 Oxford
- Dr. B. Broager C
 Bispebjerg Hospital
 Copenhagen, Denmark

- G. Brocklehurst Esq., M.Chir., F.R.C.S. M
Hull Royal Infirmary
Hull, HU3 2JZ
- T. F. Buckley Esq., F.R.C.S. M
St. Finbarr's Hospital
Douglas Road
Cork, Ireland
- Dr. Paul Bucy H
251 East Chicago Avenue
Chicago 11, Illinois, U.S.A.
- Professor Eduard Busch H
Righospitalets neurokururgiske afdeling
Tagensvej 18, Copenhagen, Denmark
- Professor K. A. Bushe C
Neurochirurg. Abt. D.
Chirurg. Univ.-Klinik
34, Gottingen, Gosler-Strasse 10, W. Germany
- D. P. Byrnes Esq., F.R.C.S. A
Department of Neurological Surgery
Royal Victoria Hospital
Grosvenor Road, Belfast, BT12 6BA
- Dr. F. Cabieses C
Talara 655
Lima, Peru, South America
- P. C. J. Carey Esq. M
27, Fitzwilliam Square
Dublin 2, Eire
- I. Cast Esq., F.R.C.S. A
Neurosurgical Unit
Morrison Hospital
Swansea, SA6 6NL
- Dr. Binoy Chakravorty, F.R.C.S. C
3, Allenby Road
Calcutta - 20, India
- Professor J. Chandy, M.Sc., F.R.C.S. C
Christian Medical College and Hospital
Vallore, S. India

- A. K. Chatterjee Esq., F.R.C.S. A
 Department of Neurosurgery
 Royal Infirmary
 Preston, PR1 6PS
- A. B. Chaudhari Esq., F.R.C.S. A
 48, Devonshire Rd., Ealing, London, W.5
- Professor Jerzy Chorobski H
 Czerwonego
 Krzyza 11N 28, Warsaw, Poland
- A. R. Choudbury Esq., F.R.C.S. A
 Department of Surgery, Unit IV
 Assam Medical College
 Dibrugarh, Assam, India
- U. M. Chowdhary Esq., F.R.C.S. A
 Department of Neurological Surgery
 St. Bartholomews Hospital
 London, E.C. 1
- Dr. J. C. Christensen H
 Avenida Alvear 1399
 Buenos Aires, Argentine
- P. R. R. Clarke Esq., F.R.C.S. M
 Department of Neurosurgery
 Ward 17
 Middlesbrough General Hospital
 Ayresome Green Lane
 Middlesbrough, Yorks
- G. Clark-Maxwell Esq., F.R.C.S. E
 Mackworth House, Derby
- T. J. Connelley Esq., F.R.C.S. A
 St. Vincent's Hospital
 Victoria Street
 Darlinghurst, N.S.W., Australia
- R. C. Connolly Esq., F.R.C.S. M
 Department of Neurological Surgery
 St. Bartholomew's Hospital
 London, E. C. 1
- A. G. L. Corkill Esq., F.R.C.S. A
 Department of Neurological Surgery
 School of Medicine
 Davis, California 95616, U.S.A.

- H.A. Crockard Esq., F.R.C.S. A
 Department of Neurological Surgery
 Royal Victoria Hospital
 Grosvenor Road, Belfast, BT12 6BA
- J. N. Cross Esq., F.R.C.S. A
 Department of Surgery
 University of West Indies
 Mona
 Kingston 7, Jamaica, West Indies
- Dr. H. J. Crow A
 Burden Neurological Institute
 Stoke Lane
 Frenchay, Bristol
- Bernard Crymble Esq., F.R.C.S. A
 30, The Martlet
 The Upper Drive
 Hove, Sussex
- B. H. Cummins Esq., F.R.C.S. A
 Department of Neurological Surgery
 Frenchay Hospital
 Bristol, BS16 1LI
- J. T. Cummins Esq., F.R.C.S. A
 23 Hood Street
 North Balwyn
 Victoria, Australia
- J. C. Currie Esq., F.R.C.S. M
 Department of Neurological Surgery
 St. Bartholomews Hospital
 London, E. C. 1
- J. B. Curtis Esq., F.R.C.S., F.R.A.C.S. C
 376, Albert Street
 East Melbourne
 Victoria 3002, Australia
- Professor M. David H
 4, Rue Galliere
 Paris XVIe, France
- Dr. Courtland Davis Jr. H
 Bowman Gray School of Medicine
 Winston-Salem, N.C. 27103, U.S.A.

Dr. Loyal Davis 700 North Michigan Avenue Chicago, Illinois, U.S.A.	H
R. A. Daws Esq., F.R.C.S. 22, Egerton Road Ashton, Preston, Lancs	M
✓ B. H. Dawson, Esq., F.R.C.S. Salford Royal Hospital Salford, Lancs. M6 9EP	M
Dr. De Gutierrez-Mahoney St. Vincent's Hospital New York 11, U.S.A.	H
Dr. De Vasconcellos Marques Rua do Prior, 22 Lisboa, Portugal	C
Dr. J. A. De Vera Reyes Alvarez de Luge 36 Santa Cruz de Tenerife, Canary Islands	A
Professor J. C. De Villers, F.R.C.S. Department of Surgery Medical School Observatory Cape Town, South Africa	C
Dr. M. H. Dhruva	C
Stevens Dimant Esq., F.R.C.S. Puget Sound Medical Building 1106 South Fourth Street Tacoma, Washington, U.S.A.	C
F. Donovan Esq., F.R.C.S. St. Vincent's Hospital Stephens Green East Dublin, Eire.	A
N. W. C. Dorch Esq., F.R.C.S. Department of Neurosurgical Studies The National Hospital Queen Square, London, WC1N 3BG	A

Graeme Duffy Esq., F.R.C.S.	M
J. Dutton Esq., F.R.C.S. University Department of Neurosurgery Royal Infirmary Manchester, 13	M
Dr. El Sayed Awad El Gindi, F.R.C.S. 13, Abdel Kader Hamdy Street El-Hay El-Khamisi Heliopolis, U.A.R.	A
Dr. Harold Elliott Montreal General Hospital Montreal 25, Canada	C
Mr. S. El Molla, F.R.C.S. The General Infirmary Great George Street, Leeds, 1	OA
Bernard Fairburn Esq., F.R.C.S. "Cranbrook" 34, Hogarth Avenue, Brentwood, Essex	M
Murray A. Falconer Esq., F.R.C.S. The Maudsley Hospital DeCrespigny Park London, SE5 8AZ	M
T. F. Fannin Esq., F.R.C.S. Department of Neurological Surgery Royal Victoria Hospital Belfast, BT12 6BA	A
V. Farrell Esq., F.R.C.S. 6, Bompas Road Dunkeld Johannesburg, S. Africa	A
D. M.C. Forster Esq., F.R.C.S. Department of Neurological Surgery The Royal Infirmary Sheffield, S6 3DA	A
R. J. A. Fraser Esq., F.R.C.S. Royal Infirmary Foresterhill, Aberdeen	M

- Keith Frazer Esq., F.R.C.S. A
 Department of Neurological Surgery
 University Hospital of Wales
 Cardiff
- Professor Piero Frugoni C
 Istituto di Neurochirurgia
 Via Giustiniani, 5, Padua, Italy
- Dr. R. Frykholm C
 Sodersjukhuset
 Stockholm, Sweden
- John Garfield Esq., F.R.C.S. M
 Wessex Neurological Centre
 Southampton General Hospital
 Shirley, Southampton
- J. R. Gibbs Esq., F.R.C.S. M
 Brook General Hospital
 Shooters Hill
 London, SE18 4LW
- R. M. Gibson Esq., F.R.C.S. M
 Department of Neurological Surgery
 General Infirmary
 Leeds, 1
- Professor F. J. Gillingham, F.R.C.S. M
 Department of Surgical Neurology
 Royal Infirmary
 Edinburgh, 3
- C. A. Gleadhill Esq., F.R.C.S. M
 Royal Victoria Infirmary
 Belfast, N. Ireland
- J. R. W. Gleave Esq., F.R.C.S. M
 Department of Neurological Surgery
 Addenbrooke's Hospital
 Hills Road, Cambridge
- D. Gordon Esq., F.R.C.S. M
 30, Deramore Park
 Belfast BT9 5JU
 (& Royal Victoria Hospital
 Belfast)
- P. Gortvai Esq., F.R.C.S. M
 Oldchurch Hospital
 Romford, Essex

- H. H. Gossman Esq., F.R.C.S. M
 South Devon and East Cornwall Hospital
 Freedom Fields, Plymouth, Devon
- Norman Grant Esq., F.R.C.S. A
 The Hospital for Sick Children
 Great Ormond Street
 London W. C. 1
- Dr. James Greenwood Jr., H
 718, Hermann Professional Building
 Houston, Texas 77025, U.S.A.
- H. B. Griffith Esq., F.R.C.S. M
 Department of Neurological Surgery
 Frenchay Hospital
 Bristol
- Professor C. Gros C
 Service de Neuro-chirurgie
 Centre Hospitalier Regional de Montpellier
 Montpellier, France
- Dr. B. Guidetti C
 Via Ximenes 7
 Rome, Italy
- Dr. G. Guiot C
 Hopital Foch
 40 Rue Worth
 Suresnes (Seine)
 France
- A. N. Guthkelch Esq., F.R.C.S. M
 Combined Neurological Service
 The Royal Infirmary
 Hull
- R. S. Gye Esq., F.R.C.S. M
 Department of Neurological Surgery
 Radcliffe Infirmary
 Oxford
- J. G. Hamilton Esq., F.R.C.S. M
 Midland Centre for Neurosurgery
 Holly Lane
 Smethwick, Warley, Worcs.
- Dudley Hancock Esq., F.R.C.S. M
 Stoke Mandeville Hospital
 Aylesbury, Bucks

- ✓ Professor J. Hankinson, F.R.C.S. M
Regional Neurological Centre
Newcastle General Hospital
Newcastle upon Tyne, NE4 6BE
- Richard Hannah Esq., F.R.C.S. M
Walton Hospital
Liverpool, 9
- T. M. Hannigan Esq., F.R.C.S. M
Crumpsall Hospital
Crumpsall, Manchester, 8
- J. Hardman Esq., F.R.C.S. M
119, Millhouses Lane
Sheffield, S7 2HD
- D. G. Hardy Esq., S.R.C.S. A
The Neurosurgical Unit
The London Hospital
London E. 1
- Bernard Harries Esq., F.R.C.S. M
University College Hospital
Gower Street
London W.C. 1
- Phillip Harris Esq., F.R.C.S. M
Department of Surgical Neurology
Royal Infirmary
Edinburgh, 3
- ✓ R. K. Hay Esq., F.R.C.S. A
Department of Neurosurgery
St. Mary's and Vaughan
Winnipeg 1, Manitoba, Canada
- Dr. J. K. Henderson C
20 Collins Street
Melbourne, Victoria 3000, Australia
- W. R. Henderson Esq., F.R.C.S. E
Lane Head
Creskeld Garth, Bramhope, Leeds
- Dr. J. Heras A
Avda, Republica Argentina, 23
Leon, Spain

- T. A.H. Hide Esq., F.R.C.S. M
 Institute of Neurological Sciences
 Southern General Hospital
 1345, Govan Road, Glasgow, G51 4TF
- E. R. Hitchcock Esq., F.R.C.S. M
 Department of Surgical Neurology
 Royal Infirmary
 Edinburgh 3
- A. D. Hockley Esq., F.R.C.S. A
 Department of Neurological Surgery
 Addenbrooke's Hospital
 Hills Road, Cambridge, CB2 2QQ
- A. E. Holmes Esq., F.R.C.S. A
 Department of Neurological Surgery
 Addenbrooke's Hospital
 Hills Road, Cambridge, CB2 2QQ
- Professor E. B. C. Hughes, F.R.C.S. M
 Queen Elizabeth Hospital
 Edgbaston, Birmingham, 15
- Allan Hulme Esq., F.R.C.S. M
 Neurosurgical Unit
 Frenchay Hospital
 Bristol
- R.D. Illingworth Esq., F.R.C.S. A
 Department of Neurological Surgery
 Central Middlesex Hospital
 Park Royal
 London, NW10 7NS
- Harvey Jackson Esq., F.R.C.S. E ✓
 56, Fairacres
 Roehampton Lane
 London, SW15 5LY
- Ivan Jacobson Esq., F.R.C.S. M
 Department of Surgical Neurology
 Royal Infirmary
 Dundee
- Dr. K. Jacz C
 Monteverdilaan 235
 Zwolle, Netherlands

- Antony James Esq., F.R.C.S. C
5, Masefield Way
Karori, Wellington, New Zealand
- T. G. I. James Esq., F.R.C.S. H
1, Freeland Road
Ealing Common
London, W.5
- Dr. Khalil Jawad A
Institute of Neurological Sciences
Southern General Hospital
1345, Govan Road, Glasgow, G51 4TF
- ✓ A. A. Jefferson Esq., F.R.C.S. M
Department of Neurological Surgery
Royal Infirmary
Sheffield, 6
- R. V. Jeffreys Esq., F.R.C.S. A
Department of Surgical Neurology
Western General Hospital
Crewe Road, Edinburgh, EH4 2XU
- Professor W. Bryan Jennett, F.R.C.S. M
Institute of Neurological Sciences
Southern General Hospital
Glasgow, G51 4TF
- Richard Johnson Esq., O.B.E., F.R.C.S. M
Department of Neurological Surgery
Royal Infirmary
Manchester
- R. A. C. Jones Esq., F.R.C.S. M
Salford Royal Hospital
Chapel Street
Salford, Lancs, M60 9ED
- Omar V. Jooma Esq., F.R.C.S. C
Jinnah Central Hospital
Karachi, Pakistan
- M. J. Joubert Esq., F.R.C.S. C
15-17, St. Augustine's Medical Centre
333, Clark Road
Durban, South Africa
- Vijay Kak Esq., F.R.C.S. A
Department of Neurosurgery
P.G. Institute of Medical Education & Research
Chandigarh, U.P. India

- R. M. Kalbag Esq., F.R.C.S. M
Regional Neurological Centre
Newcastle General Hospital
Newcastle upon Tyne, NE4 6BE
- S. Kalyanaraman Esq., F.R.C.S. C
A147, Central Street
Kilpauk Garden Colony
Madras-10, India
- W. F. C. Kennedy Esq., F.R.C.S. A
15-17, St. Augustine's Medical Centre
333, Clark Road
Glenwood, Durban, South Africa
- A. J. Keogh Esq., F.R.C.S. A
Department of Neurological Surgery
The Royal Infirmary
Sheffield, S6 3DA
- A. S. Kerr Esq., F.R.C.S. M
Regional Neurosurgical Centre
Walton Hospital
Liverpool 9
- Professor F. K. Kessell C
Stadt. Krankenhaus
Ismaningerstr. 22
Munich 8, Germany
- T. T. King Esq., F.R.C.S. M ✓
The London Hospital
Whitechapel
London, E.1
- Dr. M. R. Klein C
9, Square de L'Alboni
Paris XVIe, France
- G. C. Knight Esq., F.R.C.S. M
106, Harley Street
London, W.1
- P. Kothandaram, F.R.C.S. A
Pais Hill
Mangalore 6, India
- Professor Hugo Krayenbuhl H
Neurochirurgische Universitätsklinik
Kantonsspital
8000 Zurich, Switzerland

Dr. K. Kristiansen Ullevål Sykehus Oslo, Norway	C
Dr. R. A. Krynauw 22, Alexandra Ave. Sea Point, Cape Province, South Africa	C
Professor Zdenek Kunc Neurosurgical Clinic Charles University Praha-Štřesovice, Czechoslovakia	H
Professor Adam Kunicki Ślawkowska 24A Krakow, Poland	H
Professor E. Laine 25, Boulevard Vauban 59 Lille, France	H
Miss Beryl Lander, F.R.C.S. Midland Neurosurgical Centre Holly Lane Smethwick, Warley, Worcs.	A
C. Langmaid Esq., F.R.C.S. 174, Lake Road East Roath Park, Cardiff, CF2 5NR	E
J. P. Lanigan Esq., F.R.C.S. Richmond Hospital Dublin	M
L. P. Lassman Esq., F.R.C.S. Regional Neurological Centre Newcastle General Hospital Newcastle upon Tyne, NE4 6BE	M
Mr. C. W. Law, F.R.C.S. Florence Nightingale Buildings Kotze Street Hospital Hill Johannesburg, South Africa	C
Professor Jean Lecuire Hospital Neurologique 39, Boulevard Pinel, Lyon-Bron (Rhône), France	C

- Professor L. Leksell H ✓
Karolinska Sjukhuset
Stockholm, Sweden
- Profesor C. H. Lenshoek C
Neurochirurgische Kliniek
Algemeen Zieken Huis
Groningen, Holland
- W. S. Lewin Esq., M.S., F.R.C.S. M ✓
Department of Neurological Surgery and Neurology
Addenbrooke's Hospital
Hills Road, Cambridge
- Dr. Adolfo Ley Gracia H
Muntaner 318
Barcelona-6, Spain
- Dr. T. A. Lie C
v. Ryckevorsellaan 10
Moergestel, Holland
- Professor R. Lipschitz, F.R.C.S. C
Department of Surgical Neurology
Baragwanath Hospital
Johannesburg, South Africa
- Professor Valentine Logue, F.R.C.S. M ✓
Maida Vale Hospital
London, W.9
- R. C. Lowe Esq., F.R.C.S. C
94, Pasley Street
South Yarra 3141, Australia
- Professor Nils Lundberg C
Neurokirurgiska Klinicen
Lanslasarettet
Lund, Sweden
- Professor W. Luyendijk C
Neurosurgery Department
University Hospital
Leiden, Holland
- J. J. Maccabe Esq., F.R.C.S. M
113, Dulwich Village,
London, S.E. 21

- A. G. MacIntyre Esq., F.R.C.S.** **A**
158, Victoria Street E.
Alliston, Ontario, Canada
- Donald Mackenzie Esq., F.R.C.S.** **H**
- I. R. McCaul Esq., F.R.C.S.** **M**
Whittington Hospital
St. Mary's Wing
Highgate Hill, London, N.19
- J. W. McIntosh Esq., F.R.C.S.** **M**
Department of Neurosurgery
North Staffordshire Royal Infirmary
Princes Road, Hartshill
Stoke-on-Trent, Staffs.
- Sir Wylie McKissock, F.R.C.S.** **E**
Camus Na Harry
Lechnaside, Gairloch
Wester Ross, Scotland
- Dr. R. L. McLaurin** **H**
Cincinnati General Hospital
3231 Burnet Avenue
Cincinnati, Ohio 45229, U.S.A.
- Dr. Farhad Mahjouri-Sabet** **OA**
Department of Neurological Surgery
Crumpsall Hospital
Manchester, 8
- Dr. R. Malmros** **H**
Den Neurokirurgiske Afdeling Ved.
Arhus Kommunehospital
Arhus, Denmark
- Professor Louis Mansuy** **C**
Hopital Neurologique
39, Boulevard Pinel
Lyon-Bron (Rhone), France
- W. J. M. Marshall Esq., F.R.C.S.** **A**
Division of Neurosurgery
Queen's University
82, Barrie Street
Kingston, Ontario, Canada
- H. A. Maslowski Esq., F.R.C.S.** **M**
Crumpsall Hospital
Manchester, 8

- Andrew Masson Esq., F.R.C.S. C
 University College of the West Indies
 Mona St. Andrew
 Jamaica, West Indies
- Dr. Frank Mayfield H
 506 Oak Street
 Cincinnati, Ohio 45219, U.S.A.
- H. Mendelow Esq., F.R.C.S. C
 Princess Nursing Home
 Johannesburg, South Africa
- J. B. Miles Esq., F.R.C.S. M
 Department of Neurosurgery
 Walton Hospital
 Liverpool
- Sir Douglas Miller, F.R.C.S. H
 149 Macquarie Street
 Sydney, N.S.W., Australia
- J. D. Miller Esq., F.R.C.S. M
 Institute of Neurological Sciences
 Southern General Hospital
 Glasgow, G51 4TF
- Rex Money Esq., F.R.C.S. H
 175, Macquarie Street
 Sydney, N.S.W. 2000, Australia
- Dr. Alan Mooney H
 33, Fitzwilliam Place
 Dublin, Eire
- Dr. Jean Morelle H
 Clinique Chirurgicale de l'hôpital St. Pierre
 Rue de Bruxelles 69
 Louvain, Belgium
- Dr. G. Morello C
 Viale Regina
 Giovanni 11, Milano, Italy
- F. P. Morgan Esq., F.R.C.S. H
 55, Collins Street
 Melbourne 3000, Australia
- T. P. Morley Esq., F.R.C.S. C
 906, Medical Arts Building
 Toronto 5, Canada

- Dr. Wm. H. Mosberg Jr. C
 803 Cathedral Street
 Baltimore, Md. 21201, U.S.A.
- Dr. Lester Mount H
 Neurological Institute
 710 W. 168th Street
 New York, N.Y. 10032, U.S.A.
- Dr. R. Murali OA
 Department of Surgical Neurology
 Western General Hospital
 Crewe Road, Edinburgh, EH4 2XU
- Professor P. Narendran, F.R.C.S. A
 Stanley Medical College
 Madras, India
- J. Neil-Dwyer Esq., F.R.C.S. A
 Wessex Neurological Centre
 Southampton General Hospital
 Southampton, SO9 4XY
- R. L. G. Newcombe, F.R.C.S. A
 Yew Cottage
 Blackbrook Lane, Bickley, Kent, BR1 2HP
- E. J. Newton Esq., F.R.C.S. M
 North Staffordshire Royal Infirmary
 Stoke-on-Trent, Staffs
- W. Martin Nichols Esq., F.R.C.S. M
 Aberdeen Royal Infirmary
 Foresterhill, Aberdeen AB9 2ZB
- Professor W. Noordenbos H
 Messchaertstraat 10
 Amsterdam Z, Holland
- Dr. G. Norlen H
 Sahlgrenska Sjukhuset
 Gothenburg, Sweden
- G. B. Northcroft, F.R.C.S. M
 Brook General Hospital
 Shooters Hill, London, S.E.18
- D.W. C. Northfield Esq., F.R.C.S. E
 Holly Tree Cottage
 Latimer, Chesham, Bucks

Mr. Daryl H. Nye, F.R.C.S. 2/18 Murchison St. East St. Kilda 3183, Melbourne, Australia	OA
Dr. S. Obrador Eduardo Dato 23 Madrid 10, Spain	H
J. E. A. O'Connell Esq., M.S., F.R.C.S.	E
Professor Herbert Olivecrona Strandvagen 57, Stockholm, Sweden	E
L. C. Oliver Esq., F.R.C.S. 94, Harley Street London, W.1	M
Mr. A. A. Olumide, F.R.C.S. Department of Surgery Neurosurgery Unit University College Hospital Ibadan, Nigeria	OA
Ayub Ommaya Esq., F.R.C.S. N.I.N.D.B. National Institute of Health Bethesda 14, Maryland, U.S.A.	C
K. W. E. Paine Esq., F.R.C.S. Department of Neurosurgery University Hospital Saskatoon, Saskatchewan, Canada	C
Alexander R. Pate Esq., F.R.C.S. Ashley Hainault Road Foxrock, Co. Dublin, Eire	A
Sohana S. Patel Esq., F.R.C.S.E. Department of Surgical Neurology The Royal Infirmary Edinburgh, 3	A
A. Paterson Esq., F.R.C.S. Institute of Neurological Sciences Southern General Hospital Glasgow, G51 4TF	M
Professor Wilder Penfield, F.R.S. The Neurological Institute 3801, University Street Montreal, Canada	H

- J. B. Pennybacker Esq., C.B.E., F.R.C.S. E
 Creagandaraich
 Tighnabruaich
 Argyll, Scotland
- Professor D. Petit-Dutailis E
 (no address available)
- Professor R. Petr C
 Neurosurgical Clinic
 Hradec Kralove, Czechoslovakia
- Dr. Byron C. Pevehouse C
 2001 Union Street
 San Francisco, California 94123, U.S.A.
- D. G. Phillips Esq., F.R.C.S. M
 Neurosurgical Unit
 Frenchay Hospital
 Bristol, BS16 1LE
- Professor H. W. Pia H
 Neurochirurgische Universitats Klinik
 Klinikstrasse 37
 63 Giessen, Germany
- C. E. Polkey Esq., F.R.C.S. A
 Regional Neurosurgical Centre
 Brook Hospital
 Shooters Hill Road
 London, SE18
- Dr. J. Lawrence Pool H
 Box 31, West Cornwall, Connecticut 06796, U.S.A.
- Dr. James Poppen H
 The Lahey Clinic
 605 Commonwealth Ave.
 Boston, Massachusetts, U.S.A.
- J. M. Potter Esq., D.M., F.R.C.S. M
 Department of Neurological Surgery
 Radcliffe Infirmary
 Oxford
- M. J. Powell Esq., F.R.C.S. A
 263 West Street
 Braintree, Massachusetts, U.S.A.

- D. J. E. Price Esq., F.R.C.S.** A
Institute of Neurological Sciences
Southern General Hospital
Glasgow G51 4TF
- E. J. Radley-Smith Esq., F.R.C.S.** M
The Royal Free Hospital
Gray's Inn Road, London, W.C. 1
- Dr. Hamid Rahmat** A
Midland Centre for Neurosurgery
Holly Lane
Smethwick, Warley, Worcs
- B. Ramamurthi Esq., F.R.C.S.** C
2nd Main Road
C.I.T. Colony
Mowbray's Road, Madras-4, India
- Dr. Roberto E. Ramirez** OA
Hurstwood Park Hospital
Haywards Health, Sussex
- Dr. Joseph Ransohoff II** C
New York University Medical Centre
500 First Avenue, New York, N.Y. 10016, U.S.A.
- K. Ratnarajah Esq., F.R.C.S.** A
Ocean County Professional Building
1823 Route 88, Brick Town, N.J. 08723, U.S.A.
- J. M. Rice Edwards Esq., F.R.C.S.** A
Maida Vale Hospital
London, W.9
- Alan Richardson Esq., F.R.C.S.** M
Neurosurgical Unit
Atkinson Morley's Hospital
Copse Hill, Wimbledon, London, S.W. 20
- Dr. John Riishede** C
Rigshospitalets neurokirurgiske afdeling
Tagensvej 18, 2200 Copenhagen, Denmark
- J. G. Roberts Esq., F.R.C.S.** M
Central Middlesex Hospital
Acton Lane, Park Royal, London, N.W. 10

- D. B. Robertson Esq., F.R.C.S. C
Auckland General Hospital
Auckland, New Zealand
- J. M. Robertson Esq., F.R.C.S. M
589, Marton Road, Middlesbrough, Yorks
- J. Sloan Robertson Esq., O.B.E., F.R.C.S. H
The Anchorage
Carrick Castle
Loch Goil, Argyll, Scotland
- Julian Robinson Esq., M.D., M.S., F.R.C.S. A
34, Cote de la Fabrique
Quebec (4^e) P.Q., Canada
- R. G. Robinson Esq., F.R.C.S. C
Department of Neurosurgery
Dunedin Hospital
Dunedin, New Zealand
- Dr. A. N. Rocha Melo C
Rua Julio Diniz 745
Porto, Portugal
- Professor Sir J. Paterson Ross., M.S., F.R.C.S. E
14H John Spencer Square
Canonbury, London, N. 1
- G. F. Rowbotham Esq., F.R.C.S. E
Fairholme, Wylam, Northumberland
- Professor Dorothy Russell E
Holcombe End
Westcott, Dorking, Surrey
- J. A. Russell Esq., F.R.C.S. M
Institute of Neurological Sciences
Southern General Hospital
Glasgow, G51 4TF
- Dr. E. Scarff H
New York Neurological Institute
168th St. and Fort Washington Ave.
New York, N.Y., U.S.A.
- Harold Schaeffer Esq., F.R.C.S. A
Florey House
16, Bagot Street
North Adelaide 5006, South Australia

- J. Schorstein Esq. H
15, Dundonald Road, Glasgow, W.2
- Professor Dr. K. Schurmann C
Neurochirurgische Klinik
65, Mainz, Langenbeckstrasse 1, Germany
- P. H. Schurr Esq., F.R.C.S. M
Guy's-Maudsley Neurosurgical Unit
De Crespigny Park, London, S.E. 5
- Dr. W. B. Scoville C
85 Jefferson Street
Hartford, Connecticut 06106, U.S.A.
- C. B. Sedzimir Esq., F.R.C.S. M
Regional Neurosurgical Centre
Walton Hospital
Liverpool, 9
- Professor B. Selverstone H
New England Center Hospital
Corner Bennett Street and Harrison Ave.
Boston 11, Massachusetts, U.S.A.
- R. P. Sengupta Esq., F.R.C.S. A ✓
Regional Neurological Centre
Newcastle General Hospital
Westgate Road
Newcastle upon Tyne, NE4 6BE
- Dr. Alberto Serrats A
Hospital General de Asturias
Oviedo, Asturias, Spain
- M. M. Sharr Esq., F.R.C.S. A
Wessex Neurological Centre
Southampton General Hospital
Southampton, SO9 4XY
- J. Shaw Esq., F.R.C.S. M
Department of Surgical Neurology
Royal Infirmary
Edinburgh
- R. H. Shephard Esq., F.R.C.S. M
Department of Neurosurgery
Derbyshire Royal Infirmary
Derby DE1 2QY

- J. M. Small Esq., F.R.C.S. M
 Midland Centre for Neurosurgery
 Holly Lane
 Smethwick, Warley, Worcs.
- Dr. Honor Smith H
 25, Park Town, Oxford
- S. R. Soni Esq., F.R.C.S. A
 Department of Neurosurgery
 Morriston Hospital
 Morriston, Swansea, SA6 6NL
- Dr. N. C. Spanos A
 Department of Neurosurgery
 General Hospital
 Nicosia, Cyprus
- Professor F. A.R. Stammers, C.B., T.D., Ch.M., F.R.C.S. H
 56, Middle Park Road
 Wesley Hill, Birmingham 29
- Dr. W. E. Stern H
 Division of Neurosurgery
 University of California Medical Center
 Los Angeles 24, California, U.S.A.
- W. E. Strachan Esq., F.R.C.S. A
 Neurological Department
 General Hospital
 Freedom Fields, Plymouth, Devon
- F. R. Strang Esq., F.R.C.S. A
 Department of Neurosurgery
 Manchester Royal Infirmary
 Oxford Road, Manchester 13
- A. J. Strong Esq., F.R.C.S. A
 Department of Neurosurgical Studies
 The National Hospital
 Queen Square, London, WC1N 3BG
- Dr. William H. Sweet H
 Massachusetts General Hospital
 Boston, Massachusetts 02114, U.S.A.
- Lindsay Symon Esq., F.R.C.S. M
 The National Hospital
 Queen Square, London, WC1N 3BG

- Dr. P. Taarnhoj C
 Neurokirurgisk afdeling
 Kobenhavns Amts sygehus
 2600 Glostrup, Denmark
- A. Talalla Esq., M.D., F.R.C.S. A
 Department of Surgery
 McMaster University
 Hamilton, Ontario, Canada
- A. R. Taylor Esq., F.R.C.S. M
 Department of Anatomy
 Medical Sciences Institute
 Hawkhill, Dundee, DD1 4HN
- Julien Taylor Esq., F.R.C.S. M
 Department of Neurosurgery
 Royal Infirmary
 Derby
- G. M. Teasdale Esq., F.R.C.S. A
 Institute of Neurological Sciences
 Southern General Hospital
 1345, Govan Road, Glasgow, G51 4TF
- Dr. R. K. Thompson H
 803 Cathedral Street
 Baltimore, Maryland 21201, U.S.A.
- Kenneth Till Esq., F.R.C.S. M
 Hospital for Sick Children
 Great Ormond Street
 London, W.C. 1
- Dr. Esward Tolosa H
 Seccion de Neurocirugia del Policlínico
 Platon 7, Barcelona, Spain
- E. A. Turner Esq., F.R.C.S. M
 Department of Neurosurgery
 Queen Elizabeth Hospital
 Birmingham 15
- John Turner Esq., F.R.C.S. M
 Institute of Neurological Sciences
 Southern General Hospital
 Glasgow, G51 4TF
- G. K. Tutton Esq., F.R.C.S. M
 Department of Neurosurgery
 Royal Infirmary
 Preston, Lancs.

- R. Tym Esq., F.R.C.S. M
 Division of Neurosurgery
 Wellesley Hospital
 160 Wellesley St. East
 Toronto 5, Ontario, Canada
- Professor G. B. Udvarhelyi C
 Johns Hopkins University and Hospital
 Baltimore, Maryland, U.S.A.
- Dr. A. Uihlein C
 The Mayo Clinic
 Rochester, Minnesota, U.S.A.
- D. Uttley Esq., F.R.C.S. M
 Department of Neurosurgery
 Atkinson Morley Hospital
 31, Copse Hill, Wimbledon, London, S.W. 20
- C. K. Vanderfield Esq., F.R.C.S. C
 The Royal Prince Alfred Hospital Medical Centre
 100, Carillon Avenue
 Newtown 2042
 Sydney, N.S.W., Australia
- Dr. H. Verbiest H
 Neurosurgical Department
 The University
 Utrecht, Holland
- Dr. Arnold C. de Vet H
 's-Gravenhage
 Blauwe Kamerlaan, 2, Holland
- Dr. H. A. D. Walder C
 Klikiëk Voor Neurochirurgie
 Katholieke Universiteit
 Sint Radboudziekenhuis
 Nijmegen, Ziekenhuisweg, 3, Holland
- Dr. A. Earl Walker H
 The Johns Hopkins Hospital
 Baltimore, Maryland, U.S.A.
- A. E. Wall Esq., F.R.C.S. M
 Neurosurgical Department
 General Infirmary
 Leeds 1

- L. S. Walsh Esq., F.R.C.S. M
 Atkinson Morley's Hospital
 31, Copse Hill, Wimbledon, London, S.W. 20
- Professor E. S. Watkins M
 The London Hospital
 Whitechapel, London, E. 1
- R. D. Weeks Esq., F.R.C.S. M
 Department of Neurosurgery
 Royal Infirmary
 Cardiff
- Dr. A. J. M. Van der Werf C
 Parnassusweg 193
 Postgiro 343463
 Amsterdam-Z, Holland
- Dr. Aloys Werner C
 Hopital Cantonal
 Geneva, Switzerland
- Professor P. Wertheimer H
 41, Avenue de Saxe
 Lyon, France
- Dr. G. Whalen ✓
 53 Garden Road
 Shrewsbury, New Jersey, U.S.A.
- W. J. Whatmore Esq., F.R.C.S. A
 Walsgrave Hospital
 Clifford Bridge Road
 Walsgrave, Coventry, CV2 2DX
- Bernard Williams Esq., F.R.C.S. M
 Midland Centre for Neurosurgery
 Holly Lane, Smethwick, Warley, Worcs.
- Maurice Williams Esq., F.R.C.S. A
 Department of Neurological Surgery
 St. Bartholomew's Hospital
 London, E. C. 1
- P. J. E. Wilson Esq., F.R.C.S. M
 40 West Cross Lane
 Swansea, SA3 5LS

- Dr. J. M. D. Wissinger A
 Division of Neurosurgery
 University of Texas at San Antonio
 7703 Floyd Curl Drive
 San Antonio, Texas 78229, U.S.A.
- John Woodward Esq., F.R.C.S. A
 12, Collins Street
 Melbourne 3000, Australia
- Russell Worth Esq., F.R.C.S. OA
 Department of Neurology
 Adelaide Children's Hospital
 72, King William Road
 North Adelaide, S. Australia
- A. Dickson Wright Esq., M.S., F.R.C.S. E
 12, Wimpole Street, London, W.1
- Peter Wu Esq., F.R.C.S. C
 Rm. 338
 Rung Ying Building
 100, Nathan Road, Kowloon, Hong Kong
- Professor G. Yasargil C
 Neurochirurgische Universitätsklinik
 Kantonsspital
 8000 Zurich, Switzerland
- Professor J. Z. Young H
 Department of Anatomy
 University College
 London, W.C. 1

218 — 6

Theodor Rasmussen