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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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.

2 **8:45 a.m.**

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 $F_2\alpha$ 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.

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

3 **9:00 a.m.**

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

5 **9:45 a.m.**

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

W. J. Atkinson

6 **10:05 a.m.**

Late Results on Carotid Ligation for Posterior Communicating Aneurysms

Alan Richardson, Richard Winn and John Jane

10:25 a.m.

COFFEE BREAK

Sweet – proton beam, single session R_x – will thrombose many of the small vessel malformations –

7 **10:50 a.m.**

Surgery of Anterior Communicating Aneurysms

B.H. Dawson

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.**

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 sphere —
? 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 Synchetron (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 newness of
new sensation —

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.

12 1:50 p.m.

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-methyldopa 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-methyldopa 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.

13 2:10 p.m.

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 ♀ hemifacial 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 hemispheral 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 tumors

to the brain –

EEG's showed slow waves & theta

abnormalities, usually focal in injured

brain region – 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, encephaloceles 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

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 H_2O, 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 shell 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

24 **9:30 a.m.**

Benign Intracranial Hypertension: Results of Treatment by Dehydrating Agents

A.A. Jefferson

head wrapping
14 infants – 1–3 yrs FU – 1.5 cm vertical mantle
9 compensated ē 3.5 cm vertical mantle –
5 " ē less than " " "
were shunted ē on-off shunt –

on-off shunt –

6 min drainage ā 1 hr.
18 children – 1–4 yrs FU – 3 deaths
in pts ē meningomyelocele –
13 – now shunt independent
5 – still need on-off shunt intermittently

sub temporal decompression –
4 children – 9 mos to 6 yrs – 4–13 periods
of intermittent hydrocephalus –

Nulsen
weekly echo scans instead of head wrapping
pts same mission rate – 1
no distinction hydrocephalus became shunt
independent –

25 9:50 a.m.

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 thereapy 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.

26 10:10 a.m.

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

Mahi – chordomas
9 pts treated upon transnasally –
8 surviving ē apparently complete
removal – 8 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 Translabyrinthine 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 T_6 segment with dura intact. The T_2 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 CO_2 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 oblitèrated 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 markedly lessens 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

J. Hankinson

31 **9:10 a.m.**

Syringomyelia and the Venous Drainage of the Spinal Cord

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

38 **11:40 a.m.**

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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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
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Samuel R. Snodgrass	1963
Theodore B. Rasmussen	1964
Edmund J. Morrissey	1965
George Maltby	1966
Guy L. Odom	1967
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Robert H. Pudenz	1969-70
William B. Scoville	1971
Robert L. McLaurin	1972
Lyle A. French	1973

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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

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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, Ohio . . . . . .October 28-29, 1938

Roosevelt Hotel,
New Orleans, Louisiana . . . . . .October 27-29, 1939

Tudor Arms Hotel,
Cleveland, Ohio . . . . . .October 21-22, 1940

Mark Hopkins Hotel, San Francisco,
and Ambassador Hotel,
Los Angeles, California . . . . . November 11-15, 1941

The Palmer House,
Chicago, Illinois . . . . . .October 16-17, 1942

Hart Hotel,
Battle Creek, Michigan . . . . . .September 17-18, 1943

Ashford General Hospital,
White Sulphur Springs,
West Virginia . . . . . .September 7-9, 1944

The Homestead,
Hot Springs, Virginia . . . . . .September 9-11, 1946

Broadmoor Hotel,
Colorado Springs, Colorado . . . . . October 9-11, 1947

Windsor Hotel,
Montreal, Canada . . . . . .September 20-28, 1948

Benson Hotel, Portland, Oregon . . . . . .October 25-27, 1949

Mayo Clinic,
Rochester, Minnesota . . . . . .September 28-30, 1950

Shamrock Hotel, Houston, Texas . . . . . October 4-6, 1951

Waldorf-Astoria Hotel,
New York City . . . . . .September 29-October 1, 1952

Biltmore Hotel,
Santa Barbara, California . . . . . .October 12-14, 1953

Broadmoor Hotel,
Colorado Springs, Colorado . . . . .October 12-14, 1953

The Homestead,
Hot Springs, Virginia . . . . . .October 27-29, 1955

Camelback Inn, Phoenix, Arizona . . . . November 8-10, 1956

The Cloister,
Sea Island, Georgia . . . . . November 11-13, 1957

The Royal York Hotel,
Toronto, Canada . . . . . .November 6-8, 1958

Del Monte Lodge,
Pebble Beach, California . . . . . .October 18-21, 1959

Hotel Sheraton Plaza,
Boston, Massachusetts . . . . . October 5-8, 1960

Royal Orleans,
New Orleans, Louisiana . . . . . .November 7-10, 1962

El Mirador,
Palm Springs, California . . . . . .October 23-26, 1963

The Key Biscayne,
Miami, Florida . . . . . November 11-14, 1964

Terrace Hilton Hotel,
Cincinnati, Ohio . . . . . .October 14-16, 1965

Fairmont Hotel & Tower,
San Francisco, California . . . . . .October 17-19, 1966

The Key Biscayne,
Miami, Florida . . . . . .November 8-11, 1967

Broadmoor Hotel,
Colorado Springs, Colorado . . . . . October 6-8, 1968

St. Regis Hotel, New York City . . . . . .September 21, 1969

Camino Real Hotel,
Mexico City . . . . . November 18-21, 1970

Sahara-Tahoe Hotel,
Stateline, Nevada . . . . . .September 26-29, 1971

New College, Oxford, England . . . . . .September 4-7, 1972

Huntington-Sheraton Hotel,
Pasadena, California . . . . . November 14-17, 1973

1974
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THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

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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
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R. Eustace Semmes
20 S. Dudley Street, Suite 101-B
Memphis, Tennessee 38103 — 1955

SENIOR MEMBERS – 32

George S. Baker
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E. Harry Botterell
Faculty of Medicine
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Kingston, Ontario, Canada — 1938

Howard A. Brown — 1939
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Harvey Chenault — 1949
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Donald F. Coburn — 1938
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Medellin, Columbia S.A.

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James Greenwood, Jr. — 1952
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Barton A. Brown (Martha) 1968
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Gale G. Clark, Capt. USN MC (Marian) 1970
12621 Brookpark Road
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Edward S. Connolly (Elise) 1973
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Charles G. Drake (Ruth) 1958
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Dean H. Echols (Fran) Founder
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George Ehni (Velaire (Larry)) 1964
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William H. Feindel (Faith) 1959
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3801 University Street
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~~800 N. E. 13th Street~~
~~Oklahoma City, Oklahoma 73104~~
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Eldon L. Foltz (Catherine) 1960 ✓
Chairman, Div. of Neurosurgery
Univ. of Calif. School of Med.
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(2480 Monaco Drive, Laguna Beach, Calif. 35213)

John D. French (Dorothy) 1951
The Center for the Health Sciences
University of California
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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
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John T. Garner (Barbara) 1971 ✓
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Henry Garretson (Marianna) 1973 ✓
Dept. of Neurosurgery
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(517 Tiffany Lane, Louisville, Kentucky 40207)

Sidney Goldring (Lois) 1964 ✓
Barnes Hospital Plaza
Division of Neurosurgery
St. Louis, Missouri 63110
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Philip D. Gordy (Elizabeth Ann (Lisa)) 1968
1025 Walnut Street
Philadelphia, Pennsylvania 19107
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John R. Green (Georgia) 1953
Barrow Neurological Institute
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John W. Hanbery (Shirley) 1959
Division of Neurosurgery
Stanford Medical Center
Palo Alto, California 94304
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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
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Los Angeles, California 90033
(13856 Bora Bora Way, #306-C,
Marina Del Rey, California 90291)

Thomas W. Langfitt (Carolyn) 1971
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34th and Spruce Streets
Philadelphia, Penn. 19104
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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.
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(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)

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