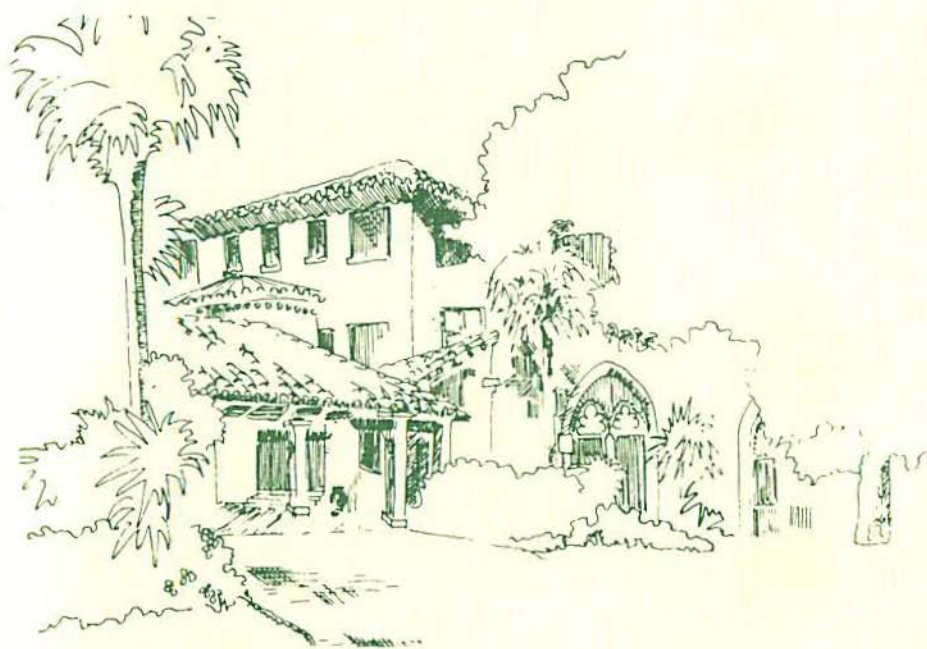


The American Academy of Neurological Surgery Program



**The Cloister
Sea Island, Georgia
1986**

**ANNUAL MEETING OF THE
AMERICAN ACADEMY OF
NEUROLOGICAL SURGERY
1986**

The Cloister
Sea Island, Georgia
November 5-8, 1986

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THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

November 5-8, 1986

The Cloister

Sea Island, Georgia

Wednesday, November 5

1:00 PM-5:00 PM Registration - Plantation Lounge
6:30 PM-8:00 PM Reception - Plantation Lounge
7:30 PM-9:00 PM Dining - The Cloister (Coat and Tie Dress)

Thursday, November 6

7:00 AM-8:30 AM Breakfast Business Meeting (Members Only)
South Georgian Room - The Cloister
8:30 AM-12:00 NOON Scientific Meeting - Ballroom
Plantation Center
12:00 NOON-12:30 PM Reception (Members, Spouses and Guests)
Plantation Lounge
12:30 PM-1:30 PM Lunch - The Cloister Restaurants
1:30 PM-5:00 PM Scientific Meeting - Ballroom
Plantation Center
6:15 PM-10:00 PM Plantation Oyster Roast - (Trams begin leaving the Plan-
tation Center 6:15 PM) Casual Dress.

Friday, November 7

7:00 AM-8:30 AM Breakfast Business Meeting (Members Only)
South Georgian Room - The Cloister
8:30 AM-12:00 NOON Scientific Meeting - Ballroom
Plantation Center
12:00 NOON Luncheon - The Cloister Restaurants
6:30 PM Reception and Dinner - Plantation Lounge
(Black Tie)

Saturday, November 8

7:00 AM-8:30 AM Breakfast - Cloister Dining Rooms
8:30 AM-12:00 NOON Scientific Meeting - Ballroom Plantation Center

SEA ISLAND SENIORS PROGRAM

Tuesday, November 4

2:00 PM-5:00 PM Registration Plantation Center
6:30 PM Reception Dr. Frank Mayfield's Cottage #30

Wednesday, November 5

8:30 AM Breakfast Buffet - The Cloister
South Georgian Dining Room
10:00 AM-11:30 AM Nature Lecture and Jeep Ride. Jeeps leave from River-
side (across from Plantation Center).
12:30 PM Buffet Luncheon - South Georgian Dining Room

LADIES ACTIVITIES

Wednesday, November 5

6:30 PM-8:00 PM Plantation Lounge Reception

Thursday, November 6

8:30 AM-5:00 PM Ladies Hospitality - Cumberland Room
Plantation Center
10:00 AM-12:00 NOON Tour of Historical St. Simons. Buses leave from front of
Riverhouse.
12:00 NOON-12:30 PM Reception (Members, Spouses and Guests)
Plantation Lounge
6:15 PM Tram Makes First Run To Plantation
Oyster Roast - Leaves from Plantation Center.
(Casual Dress)

Friday, November 7

10:00 AM-12:00 NOON Shopping Trip to Galleries and Boutiques.
Buses leave from Riverhouse.
11:00 AM-12:00 NOON Tour of Cloister Kitchens (Limited)
Meet in Main Lobby.
1:00 PM-5:00 PM Golf Tournament
2:00 PM-5:00 PM Tennis Tournament
6:30 PM Reception and Dinner
Plantation Center (Black Tie)

Saturday, November 6

8:30 AM-12:00 NOON

**Ladies Hospitality - Cumberland Room
Plantation Center**

Note: All meals and part of Thursday and Friday dinners are included in the American Plan of daily room charges.

SCIENTIFIC PROGRAM

1954

Thursday, November 6

SCIENTIFIC SESSION I

MODERATOR: Thoralf M. Sundt, Jr., M.D.

8:30 WELCOME: Phanor L. Perot, M.D., Ph.D. - President

8:35

1. ENDOTHELIUM AND VASOSPASM

T. Bun, M.D., J. Peterson, Ph.D., *N. Zervas, M.D.
Massachusetts General Hospital

The endothelium of cerebral arteries appears to lose its function following subarachnoid hemorrhage.

Dogs were subjected to two separate subarachnoid hemorrhages which caused severe basilar artery constriction within one week. The arteries were then studied angiographically and, after isolation in a "heart-lung device" which permitted:

1. simultaneous perfusion of the luminal surface at a chosen distending pressure and superfusion of the adventitial surface with the physiologic solutions;
2. electrophysiologic recording of intracellular smooth muscle membrane potential;
3. biomechanical studies of contractility; and
4. scanning of electron microscopic examination of luminal and adventitial surfaces.

The behavior of the vessel segments *in vitro* were examined *with* and *without* the endothelium.

The following observations were made:

1. Vasoconstriction induced *in situ* by subarachnoid blood is accompanied by a partial depolarization of the smooth muscle cell membrane potential.

2. Perfusion of the lumen with solutions containing serotonin or the endothelium-dependent vasodilator Substance-P result in *transient* constriction or prolonged vasodilation in normal control vessels, but *prolonged* constriction and transient vasodilation in vessels chronically constricted *in-situ*.

3. Removal of the endothelium of normal vessels results in the same electrophysiological and biochemical properties seen in chronically constricted vessels.

It thus appears that the endothelium of vessels exposed to SAH becomes functionally inactivated, disrupting its ability to provide the media relaxing factors and to prevent the entry of vasoactive factors from blood.

8:55

2. **IMPACT OF EARLY ANEURYSM SURGERY ON THE NATURAL HISTORY OF SUBARACHNOID HEMORRHAGE**

*Douglas Chyatte, M.D., Thoralf M. Sundt, Jr., M.D., Nicolee C. Fode, R.N., M.S.

Yale University Medical Center

The management results for 195 patients admitted to our institution within 3 days of aneurysmal subarachnoid hemorrhage from the years 1979 to 1984 were analyzed with respect to the timing of surgical intervention. Twenty-six patients had no surgery, 53 patients were operated on within 0-3 days after hemorrhage, 72 patients 4-9 days after hemorrhage, and 44 patients 10 or more days post hemorrhage. Overall, 53% of patients had an excellent outcome in follow up, 10% good outcome, 15% a poor outcome, and 24% died. Eighty-seven percent of patients who were Botterell Grade I on admission had an excellent result. Sixty percent of the Grade II patients had an excellent result while Grades III and IV patients had a less satisfactory outcome. The timing of surgical intervention did not affect management outcome in most instances, however, Grade II patients operated on within 10 days did somewhat better than those who were operated on later. The risk of rebleeding after hospital admission was relatively small and increased only modestly with time suggesting that a uniform policy of early surgery would have only a minor impact on the overall management results. Operative and postoperative complications were similar in all treatment groups suggesting that early surgery is safe and technically feasible.

Based on these observations, we recommend operating on "good" grade patients at the earliest convenient time and thus capitalizing on the small benefit of reduced rebleeding. Surgical intervention may be delayed in "poor" grade patients until their condition stabilizes.

9:15

3. **WHAT IS CEREBRAL VASOSPASM?**

*N.F. Kassell, M.D., H. Joshita, M.D., R.M. Lehman, M.D., B.K.A. Weir, M.D., G.N. Nazar, M.D., T. Nakagomi, M.D., T. Sasaki, M.D.

University of Virginia School of Medicine

It has yet to be determined whether vasospasm is caused by a thickening of the arterial wall or by vasocontraction. To elucidate the mechanism of the arterial narrowing, morphometric analyses of cerebral arteries evidencing vasospasm was conducted. Six cynomolgus monkeys has instillation of blood around middle cerebral artery, the contralateral side serving as control. Four groups of six rabbits each were also studied.

The monkey's middle cerebral artery was reduced in diameter 55% anglographically and 60% morphometrically. In the rabbits, BaCl₂ narrowed the artery 50% morphometrically. Acute subarachnoid hemorrhage produced 45% morphometric narrowing. Chronic SAH produced 45% anglographic and 50% morphometric narrowing. There was no significant difference in the medial cross sectional area or percent of the media occupied by smooth muscle cells between the control and SAH sides in the monkeys or between the BaCl₂, acute and chronic SAH rabbit groups. Furthermore, there was no appreciable encroachment upon the lumen by intimal thickening in the monkeys or rabbits, although there were alterations in the endothelial morphology.

These data suggest that vasospasm is due to profound (normal or abnormal) contraction or failure of relaxation of smooth muscle cells and that is not produced by thickening of the arterial wall.

The following study was conducted to further characterize the arterial narrowing. A vasodilator "cocktail" consisting of adenosine, nitroprusside, and papaverine has been shown to produce maximal dilatation in a variety of vascular beds.

Control: normal animals. **Control Plus Vasodilatation:** normal animals perfused with the cocktail prior to fixation. **Chronic SAH:** animals sacrificed 72 hours subsequent to intracisternal injection of 1cc/kg of blood. **Chronic SAH Plus Vasodilatation:** SAH plus perfusion with cocktail. **Vasoconstriction:** animals sacrificed 30 minutes subsequent to intracisternal injection of BaCl₂. **Vasoconstriction Plus Vasodilatation:** BaCl₂ animals perfused with cocktail. All animals were sacrificed by perfusion fixation. The vasodilation animals were perfused with the cocktail for 5 minutes prior to fixation.

The diameter of the basilar arteries in the **Control** and the **Control Plus Vasodilatation Groups** was not significantly different. BaCl₂ reduced the diameter 50% and SAH reduced the diameter 30%. There were no significant differences between the diameter of the **barium chloride plus vasodilatation group** and the **chronic SAH plus vasodilatation group** when compared to the **control** or the **control plus vasodilatation**. These data demonstrate that chronic vasospasm is fully reversible.

We conclude that experimentally produced cerebral vasospasm is a result of profound, but reversible, vasoconstriction. Caution must be exercised in extrapolating these results in experimental SAH to humans with ruptured aneurysms.

9:40 COFFEE BREAK

10:00

4. COMPLEX ANEURYSMS OF THE BASILAR ARTERY TREATED WITH CIRCULATORY ARREST, HYPOTHERMIA, AND BARBITUATE CEREBRAL PROTECTION

*Robert F. Spetzler, M.D., Mark N. Hadley, M.D.

Barrow Neurological Institute

Complete circulatory arrest, deep hypothermia, and barbituate cerebral protection are efficacious surgical adjuncts in the treatment of giant intracranial aneurysms. We have used these techniques in the treatment of five patients with complex (1) or giant (4) basilar artery aneurysms with good results. The rationale, indications, and specific anesthetic and operative techniques will be reviewed. Our perioperative morbidity and long-term results support the use of these techniques in selected patients with complex intracranial vascular lesions.

10:20

5. RISK OF CAROTID ENDARTERECTOMY: AN ANALYSIS

*Donald O. Quest, M.D., J.W. Correll, M.D., J.P. Mohr, M.D.,

L. Lennihan, M.D., T.K. Tatemichi, M.D.

Columbia University, College of Physicians & Surgeons

A prospective analysis of 425 carotid endarterectomies performed at the Neurological Institute of New York from 1983 to 1986 was carried out. Forty of the 425 operations were for asymptomatic disease - usually in patients with contralateral occlusion and severe stenosis on the asymptomatic side. 407 patients (95.8%) had no post-operative deficit. 18 patients developed a peri-operative stroke; 8 (1.9%) were minor deficits, 10 (2.4%) were major deficits. Four patients (.9%) had myocardial infarctions. Of the 10 patients with major deficits 7 had pre-existing risks factors such as recent stroke, coronary artery disease, or other medical problems. 3 patients (0.7%) had only a prior TIA and not risk factors. In the current climate of heightened criticism of carotid endarterectomy these figures demonstrate that with a team of internists, neurologists, anesthesiologists, and neurosurgeons dedicated to evaluation and treatment of patients with cerebral vascular disease, low morbidity and mortality can be achieved. When comparing risk versus benefits for this surgical procedure it is important to consider the level of expertise of those treating the problem and the selection criteria for surgery. In addition the pre-operative condition of the patient must be considered when discussing post-operative complications.

10:40

6. MICROSURGICAL ANATOMY OF THE LENTICULOSTRIATE SYSTEM FROM THE MIDDLE CEREBRAL ARTERY TO ITS TERMINAL SUPPLY AND ITS SURGICAL RECONSTRUCTION

*James I. Ausman, M.D., Ph.D., Manuel Dujovny, M.D., Moises Vasques, M.D., Fernando G. Diaz, M.D., Ph.D., Ghaus Malik, M.D., Jose Selmon, M.D.

Henry Ford Hospital

The importance of the lenticulostriate arterial system in cerebral ischemia, intracerebral hemorrhage, AVM and aneurysm surgery requires a thorough understanding of its anatomy. 773 lenticulostriate arteries (LSA) or perforating branches (PFB's) from 29 unfixed human brains (58 hemispheres) were injected with polyester resin and studied. The following parameters were evaluated: outer diameter (OD), site of origin, branching patterns, length, and their pattern of distribution in the anterior perforated substance (APS).

12% (91 vessels) of the PFB's originated from A-1 through the short central artery (ShCA) and 9% (69 vessels) from the Recurrent Artery of Heubner (RAH) both forming the MEDIAL GROUP OF PFB's. 79% (613 vessels) of the PFB's originated from the Middle Cerebral Artery (MCA). Almost all of the PFB's formed the LATERAL and INTERMEDIATE GROUP of perforators. 42% of these PFB's formed the LATERAL GROUP, most of which arose as single arteries while the remainder came from common stems. In the INTERMEDIATE GROUP most (72%) of the PFB's arose from common stems.

An inverse relationship in the diameters of the RAH and ShCA was found to relate to the dominance of either in supplying the APS. The INTERMEDIATE GROUP was composed mostly by trunks which divided into small vessels before entering the APS in contrast to the LATERAL GROUP whose branches were of greater diameter and had fewer divisions. Regardless of the variations in the origins of the PFB's, the microanatomy of the vessel entry points into the APS was constant. The presence of intracerebral anastomoses between the different groups was very frequent (18 of 28 hemispheres), in contrast with the rare finding of extracerebral anastomoses (4 hemispheres).

In 28 hemispheres diluted silicone rubber was injected selectively through small catheters microsurgically located close to the three groups of PFB's. After 10 days of fixation 5mm horizontal brain slices were made and the distribution of the different groups of perforators was noted.

The thalamus did not receive irrigation from these vessels. Great differences were found between one brain and the other as well as between hemispheres of the same brain. These differences could explain the variability of the clinical picture produced by their occlusion.

Surgical reconstruction of the INTERMEDIATE AND LATERAL GROUPS of perforators was accomplished with microsurgical techniques and milliwatt laser anastomosis and will be presented included a direct approach to the ostium of the lenticulostriate arteries through the MCA trunk.

11:00

**7. MIDDLE CEREBRAL ARTERY ANEURYSMS -
A 10 YEAR EXPERIENCE**

*Eugene S. Flamm, M.D.

New York University School of Medicine

Aneurysms of the middle cerebral artery (MCA) comprises 20-25% of intracranial aneurysms in recently reported series. They pose several particular problems; these are reviewed in this paper which examines our experience with 140 MCA aneurysms operated upon in the past 10 years. These occurred in 129 patients and represent 22.1% of 632 aneurysms operated upon during this period.

Of the 129 patients, 20 had bilateral MCA aneurysms that required separate craniotomies (15.5%); 13 of these patients had at least on additional aneurysm. Another 23 patients had a MCA aneurysm as one of multiple aneurysms (17.8%). Eight patients had 2 MCA aneurysms on the same side (6.2%) in 4 of these cases additional aneurysms were present. Although the incidence of bilateral MCA aneurysms is only 15.5%, the overall occurrence of multiple aneurysms including the MCA is 41.1% (53/129). This figure is considerably greater than the expected rate for multiple aneurysms.

Twenty-one patients had no SAH, 4 patients bled from an aneurysm other than the MCA, and 104 patients presented with SAH from the MCA aneurysm (80.6%). There were 4 deaths, all in Grade III and IV (Hunt) patients. There were 6 patients with significant postoperative neurological deficits. These 6 patients had complex MCA aneurysms, two of which had bled.

Particular problems that have been examined regarding MCA aneurysms include the management of complicated MCA aneurysms which may require the use of temporary occlusion of the MCA, suction decompression of the aneurysm prior to clipping, and EC/IC bypass.

The increased use of CT scanning and more recently the use of digital intravenous angiography has led to more frequent diagnosis of aneurysms before hemorrhage has occurred. These issues and the general surgical management of MCA aneurysms that has evolved with this experience will be presented.

11:20

8. SURGICAL RESULTS IN CHILDREN WITH MOYAMOYA DISEASE

Katsutoshi Kitamura, M.D., Masashi Fukui, M.D., Toshio Matsushima, M.D., Kanehiro Hasuo, M.D., Toru Kurokawa, M.D.
Kyushu University, Fukuoka, Japan

Encephalo-duro-arterio-synangiosis (EDAS) was carried out on 21 sides of 15 children with Moyamoya disease. EDAS alone was done on 19 sides, and Encephalo-myo-synangiosis (EMS) was added later on two of them. EDAS and EMS were done together as one-stage operation on the remaining two sides of the 21.

Results: Nine out of 11 patients with transient ischemic attack (TIA) preoperatively showed a clinical improvement after surgery. Of the nine patients, the TIA disappeared immediately or within six months after surgery in seven, the TIA disappeared but involuntary movement remained in one, and the TIA disappeared in the upper limb but not in the lower limb in one. No improvement was achieved in two patients. Postoperative angiography was done on 17 sides of 12 patients to evaluate the surgical results. Development of new collaterals was observed markedly on 10 sides, and to a minor degree on three sides. When the development of the collaterals after EDAS was only little, EMS was added with a better subsequent formation of collaterals from the muscle in some cases. There were also cases in which EDAS and EMS were done at the same time and the collaterals developed only from the muscle. The data of the pre- and postoperative CT scan, EEG and PET scan will also be presented. As postoperative complications, temporary worsening of the TIA occurred in three cases, reversible ischemic neurologic deficit (RIND) in one and convulsive seizure in one.

Conclusion: The majority of the patients with a good development of collaterals after EDAS showed an improvement in the clinical symptoms. In some patients, however, the involuntary movement or TIA in the lower limb remained. In some of the cases with a poor result after EDAS, EMS could induce a better collateral circulation.

11:40

9. ASSOCIATION OF ATRIAL NATRIURETIC PEPTIDE (ANP) WITH HYPOTENSION FOLLOWING EXPERIMENTAL BRAIN INJURY

*Michael J. Rosner, M.D., F.A.C.S., K.J. Chang, Ph.D., W.J. Gray, F.R.C.S., D. Esposito

University of North Carolina School of Medicine

Mechanical brain injury is associated with an intense sympathoadrenal discharge and immediate severe hypertension. This hypertension correlates closely with the release of circulating catecholamines for the first 500 seconds post-injury, but then hypotension, proportional to the degree of injury supervenes. This hypotension occurs when circulating epinephrine and norepinephrine are still elevated by orders of magnitude and does not represent sympathoadrenal collapse. Because atrial natriuretic peptide (ANP) is capable of reducing myocardial contractility and inducing hypotension, we tested the hypothesis that this hormone would be released as the result of mechanical brain injury.

In ten mongrel cats, weight 2.5-3.5 kg., underwent ventilation after methohexital induction of anesthesia and maintained using N₂O:O₂ (70:30) and pancuronium bromide. After obtaining a stable baseline the fluid percussion injury was induced with the cat in the sphinx position. Arterial and venous blood samples were obtained. These paired samples were immediately placed in stop solution, spun down, the plasma separated and frozen in liquid nitrogen. ANP was assayed by radioimmunoassay technique.

At one hour after injury (3600 seconds) plasma ANP was very highly related to post-injury hypotension. A similar relationship was developing by 1000 seconds though it did not quite reach statistical significance.

These results are consistent with ANP acting as a myocardial depressant and vasodilator which may block and reverse the effects of hypercatecholaminemia. In extreme circumstances, massive release may lead to a cardiovascular rather than cerebral death of the animal. ANP is also a natriuretic agent and may be partly responsible for post-injury natriuresis and hyponatremia (mimicking SIADH).

12:00 LUNCH

1:30 ACADEMY AWARD PRESENTATION

2:45 PANEL DISCUSSION, ETHICS IN MARKETING AND QUALIFICATIONS

Moderator: Bennett M. Stein, M.D.

Panel: W. Kemp Clark, M.D.

David Kidder, Esq.

Robert G. Ojemann, M.D.

Warren T. Reich, S.T.D.

Frank R. Wrenn, M.D.

3:30 COFFEE BREAK

3:45 SENIORS AT SEA ISLAND

Reminiscences

Friday, November 7

SCIENTIFIC SESSION II

MODERATOR: Phanor L. Perot, M.D., Ph. D.

8:30

10. SINGLE NEURONAL ACTIVITY IN HUMAN TEMPORAL CORTEX RELATED TO LANGUAGE AND MEMORY

*George A. Ojemann, M.D., Otto D. Creutzfeldt, M.D., Ettore Lettich, REEG

University of Washington

Dominant hemisphere cortical resections under local anesthesia for the treatment of medically intractable epilepsy provide a unique opportunity to investigate single neuron activity related to language and memory. Changes in mean firing rates of extracellular neuronal activity recording during visual stimuli presented during measures of two language functions, naming and reading, were compared to recordings obtained during identical stimuli presented in a recent verbal memory measure, and in a control spatial matching measure. These recordings were obtained from left lateral temporal cortex just within the margin of the planned resection in 16 consenting patients. Satisfactory recordings were obtained in 12 patients, from 16 neuronal populations. Four populations showed no relation to language or memory; 8 populations demonstrated statistically significant increases in mean firing rates related to naming or reading, and 8 to recent verbal memory; half of each group were related to both. Several specific patterns of firing related to language and memory were identified, including several populations of neurons that increased firing with memory input and during word reading, and populations that fired briefly during naming or reading and in a more prolonged fashion when part of the recent memory measure. A particular pattern of firing related to recent memory was frequently observed: a relative prolonged

period of increased firing at the time of input of information to memory and again at its retrieval, with a relative reduction of firing during the time the memory was stored. These findings provide additional evidence of the participation of areas of lateral temporal cortex in language and recent verbal memory, and provide some of the first evidence on the nature of the neuronal activity that generates these functions.

8:50

11. THE CONTRIBUTION OF SPINAL MARGINAL NEURONS TO LATERAL THALAMIC NOCICEPTION

*Charles J. Hodge, Jr., M.D., A. Vania Apkarian, M.D., Steven Martini, M.D., Robert Martin, M.D.

State University of New York, Syracuse

Anatomic evidence using retrograde transport of horseradish peroxidase indicates that lamina I spinal neurons send their axons to the thalamus via the dorsolateral funiculus (DLF) ipsilateral to the thalamic termination site and contralateral to the lamina I cell body and the side of cutaneous innervation. This is of importance in understanding nociceptive mechanisms since the spinal lamina I thalamic projection is the major nociceptive specific input to the thalamus. The separation of the dorsolateral spinothalamic tract (DSTT) from the classical ventral spinothalamic tract (VSTT) allows experimental determination of the relative contributions of these two pathways to thalamic nociception, as described in this report.

Cats anesthetized with nitrous oxide and halothane were used for these experiments. Single unit recordings were made from the VPL and POm nuclei of the right sensory thalamus. Data was collected only from units responding to left hindlimb nociceptive stimulation (pinch or cutaneous thermal stimulation > 45 degrees C). A thoracic laminectomy was done and small cold probes used to reversibly block transmission through either the right ventrolateral quadrant, in which lies the VSTT, or the dorsolateral funiculus, in which lies the DSTT. Following determination of control thalamic nociceptive unit responses, the effects of separately blocking the DSTT and the VSTT were determined.

Recordings from 25 thalamic units were completed. The majority of the units were located in the shell region of VPL. Two units were located in POm. Only 2/25 units had their nociceptive responses decreased by blocking the VSTT. Nine of the 25 units demonstrated a dramatic decrease in nociceptive response during DSTT block.

These results indicate that lamina I input to the lateral thalamus is able to signal nociception, however not all nociceptive responses were dependent on this lamina I input. Clearly the DSTT, which is present in primates also, presents a paradox since anterolateral cordotomy, which spares this

pathway, results in hypalgesia and pain relief. The persistence of the DSTT following cordotomy may be one of the reasons that there is, at times, return of pain and nociception after cordotomy. Furthermore, the simplistic notion of a single effective spinal cord nociceptive signaling system (labeled line concept) is not tenable in view of this data.

9:10

12. COMBINED INJURIES OF THE CLAVICLE AND BRACHIAL PLEXUS - MECHANISMS OF INJURY AND SURGICAL MANAGEMENT

*Suzie C. Tindall, M.D.

Emory University School of Medicine

Mechanisms of injury to the underlying brachial plexus in cases of clavicular fracture include: 1. progressive callus formation with compression of the plexus against the first rib, 2. direct injury to underlying neural elements by bone spicules or sharp fractured bone edges, and 3. associated stretch or traction injuries on plexus elements, particularly the axillary nerve. The mechanisms apply either singly or in combination. Appropriate patient workup includes clinical and plain film evaluation. CT scanning usually adds little useful information.

This presentation will emphasize the principles of surgical care that include: 1. drilling away the exuberant callus and removing any indriven bone spicules, 2. evaluation of injured elements using intraoperative nerve recording techniques, 3. primary or graft repair of injured elements if indicated and 4. repair of the clavicular fracture. If handled appropriately, these cases usually have a favorable outlook. Four representative cases from the author's surgical experience will be used to illustrate these mechanisms and surgical principles.

9:30

13. MULTIMODALITY EVOKED POTENTIALS IN COMATOSE PATIENTS

*R. Firshing, M.D., J. Luther, M.D., F.A. Boop, M.D., E. Eidelberg, M.D., J.L. Story, M.D., W.E. Brown, Jr. M.D.

The University of Texas, San Antonio

In 29 comatose patients (head injuries, intracerebral hemorrhages and other disorders) somatosensory short -(SER-S) and long (SER-L) latency, visual (VER), brain stem (BAER) and 40 Hz auditory (40 Hz AER) evoked responses were investigated and correlated to coma grade and outcome.

Statistical evaluation showed that the best prognosticator of survival or non survival — not regarding the underlying cause of coma — is the combination of SER-S and age. Prognosis based on coma grade and age was less accurate. VER showed no correlation to outcome. The use of evoked potentials in comatose patients for assessment of prognosis and localization of the functional lesion is recommended.

9:50

14. ISOVOLEMIC HEMODILUTION IN EXPERIMENTAL FOCAL CEREBRAL ISCHEMIA

*R.C. Heros, M.D., Y-K Tu, M.D., D. Karacostas, M.D., N. Zervas, M.D., K. LeGree, M.D., A. Hyodo, M.D., G. Candia, M.D.
Harvard Medical School

We have performed a study of **isovolemic** hemodilution (phlebotomy plus dextran infusion to bring the Hct to about 30%) to see if cerebral protection can be achieved without the concomitant increase in blood volume, cardiac work and intracranial pressure inherent to hypervolemic hemodilution.

Sixty dogs divided into an acute and a chronic group were used for the studies. The acute group of dogs had multiple cerebral blood flow measurements by microspheres and the chronic group was kept alive for 1 week for detailed histologic examination and to ascertain whether hemodilution could be adequately maintained for at least 1 week after a single session of phlebotomy and dextran infusion. In all animals a variety of hemodynamic, cardiovascular, hematologic and rheologic parameters were measured. The ischemic insult consisted of a six hour period of occlusion of the right distal internal carotid and proximal middle cerebral arteries (twelve animals were sham operated-arterial manipulation but no occlusion).

Pertinent results can be summarized as follows:

1. Isovolemic hemodilution was of protective value in the model of experimental focal cerebral ischemia.
2. Such protection could be achieved without significant changes in blood volume, systemic arterial pressure, central venous pressure, pulmonary wedge pressure or cardiac index.
3. Isovolemic hemodilution did not result in an increase in ICP.

4. When compared with controls, hemodiluted animals experienced a significant increase in regional cerebral blood flow during the time of ischemia.

5. The cerebral protection and increased cerebral blood flow observed was most likely related to the reduction in viscosity consistently observed in the hemodiluted animals.

10:10 COFFEE BREAK

SCIENTIFIC SESSION III
MODERATOR: William F. Collins, Jr., M.D.

10:25

**15. HYPERFRACTIONATED RADIATION THERAPY FOR
BRAINSTEM GLIOMA**

*Michael S.B. Edwards, M.D

University of California San Francisco

Malignant gliomas involving the brainstem constitute 10-20% of pediatric brain tumors. While they can vary histologically from well-differentiated astrocytomas to glioblastoma multiforme, the majority are ultimately lethal regardless of histology. Controversy exists regarding the role of biopsy/resection in the management of these tumors. Radiation therapy may improve signs and symptoms in 75-90% of patients and has produced a 5 year survival in 20-30% of children. However, the vast majority of responses are under 18 months.

Because radiation is effective, albeit for a short period of time, we have undertaken the use of hyperfractionated radiation therapy as our primary treatment of brainstem gliomas. This technique allows the delivery of 25% more radiation to the tumor without an increase in normal tissue toxicity.

Thirty children/adolescents (range 2-18 yrs.) have been treated since February of 1984. The diagnosis was confirmed by biopsy in 15 patients (12 mod. anaplastic, 2 highly anaplastic, 1 glioblastoma multiforme). The other 15 patients had clinical and radiographic evidence of brainstem tumor diffusely enlarging the brainstem (the majority were confirmed on MR scans). The median followup of these thirty patients is 55 weeks. Ten patients have died tumor related deaths (one died due to shunt infection at the time of tumor progression). All ten patients showed an initial improvement following radiation therapy which was confirmed by clinical and radiographic evaluation (CT and/or MRI), but recurred within 6 months of radiation therapy.

These results are significantly better than any previous studies we have conducted or have been reported using conventional radiation with or without chemotherapy. Steroid dependence has been a significant problem and three episodes of opportunistic infections and severe myelosuppression have been observed. The reason for the myelosuppression is unclear.

The rationale for hyperfractionation and the clinical and radiographic results will be reviewed. A longer follow-up period will be necessary to determine the long term effects of hyperfractionation on survival and its effect on normal tissue toxicity.

10:45

16. ALPHA-1-ANTITRYPSIN IN HUMAN BRAIN TUMORS

*Raymond Sawaya, M.D., Mario Zuccarello, M.D., Robert Highsmith, Ph.D.,

University of Cincinnati

Proteases and protease inhibitors play an important role in tumor biology. Their role in brain tumors is unknown because of lack of data. In this study, we have measured alpha-1-antitrypsin (A_1AT) and alpha-2-macroglobulin (A_2MG) in 77 consecutive brain tumor extracts obtained freshly in the operating room. The results of these measurements were correlated with several clinical and laboratory parameters (age, Karnofsky, tumor size, PT, PTT, Fibrinogen, etc.), as well as with the content of the tissue in plasminogen activator (PA), a major proteolytic enzyme. A_1AT and A_2MG were qualitatively assessed by Ouchterlony immunodiffusion and PA was assayed electrophoretically on SDS gels. Appropriate controls were also included.

Sixty-eight percent of the samples were positive for A_1AT , while all specimens were negative A_2MG . The frequency of A_1AT positivity varied with the histological type and ranged from 100% for acoustic schwannoma to 50% for meningioma. Metastatic tumors and glioblastoma were 91.6 and 78.9% positive respectively. Clinical and biological parameters failed to show statistically significant differences between the group of patients with positive A_1AT and the group with negative A_1AT with the exception of the following three parameters:

PA activity ($P=0.001$), **peritumoral brain edema** as quantitated on CT scan ($P=0.05$) and the preoperative **serum fibrinogen level** ($P=0.025$), all three values being higher in the group with positive A_1AT .

This study supports the hypothesis that A_1AT is produced locally by tumor cells in proportion to the regional proteolytic activity. To further support this hypothesis, we have immunohistochemically demonstrated A_1AT intracellularly in various brain tumors.

11:00

**17. INTRACRANIAL GERM CELL TUMORS:
PATHOLOGY AND TREATMENT**

*Keiji Sano, M.D.

Teikyo University, Tokyo, Japan

If we admit the germ cell theory proposed by Friedman, Moore, Tellum and others, germinoma (seminoma), mature teratoma with malignant areas (teratocarcinoma), embryonal carcinoma, choriocarcinoma, endodermal sinus tumor, and so-called mixed germ cell tumors (tumors consisting of two or more of these tumor components, all belong to the category of germ cell tumors. We have experienced 93 cases of these tumors verified at surgery.

Among these tumors, 56 were pure types, such as mature teratoma or germinoma, and 37 were mixed tumors.

51 of these germ cell tumors were in the pineal region, 30 were in the suprasellar-hypothalamic region, and 12 were located in other sites. Cases of suspected germinoma treated by radiation only were excluded from this series. Two-cell-pattern tumors of probable pineal parenchyma origin were also excluded. 72 of 93 cases were below the age of 20 years and 79 were males.

Pathological examinations include labelling index studies using bromouridine and its monoclonal antibody, and immunohistochemical studies. Germinoma showed unexpectedly high values of labelling index, which may explain its high radiosensitivity.

As for the treatment, three modalities of therapy should be recommended for three different tumor categories.

1. For germinoma, radiation or surgery followed by radiotherapy (we prefer the latter) should yield good results. In our series the 10-year survival rate by this modality of therapy was more than 85%.
2. Mature teratoma should be treated by surgical excision. In our series, the 10-year survival rate by total surgical removal was more than 78%.
3. The other kinds of germ cell tumors, namely, malignant teratoma, embryonal carcinoma, choriocarcinoma, endodermal sinus tumor, and mixed germ cell tumors usually show poor prognosis if treated by surgery and radiation only because of dissemination and hematogenous metastases. Combination chemotherapy with cisplatin, vinblastine and bleomycin (PVB therapy) seems to be promising for the treatment of these malignant tumors.

11:15

18. THERAPEUTIC EVALUATION OF A SPECIFIC ANTI-GLIOMA MONOCLONAL ANTIBODY (Mab) USING A SUBCUTANEOUS HUMAN GLIOMA XENOGRAFT MODEL

*Dennis E. Bullard, M.D., Yi Shing Lee, M.D., Ph.D, R. Edward Coleman, M.D., Darrell D. Bignar, M.D., Ph.D.
Duke University Medical Center

The development of Mabs reactive with human tumor-associated antigens (TAA) provides a potential means of delivering therapeutic agents selectively to human malignant gliomas. To evaluate the therapeutic potential of 81C6, an IgG_{2b} immunoglobulin, which defines a glioma extracellular matrix antigen, we radiolabeled and tested it in a subcutaneous athymic mouse xenograft model against D-54 MG, a human glioma-derived cell line (HGCL). In this well defined system, human tumors show progressive subcutaneous growth with a pattern of chemosensitivity roughly paralleling that seen in large human trials. D-54 MG subcutaneous tumors were passaged in nude mice until they developed progressively growing 200-500 mm³ tumors. Animals were then injected via the tail vein with either buffer, unlabeled 81C6, 131-I radiolabeled 81C6, or 131-I radiolabeled 45.6, a nonspecific control Mab of the same isotype. Specific activities of the Mab range from 6.0-15.5 mCi/mg with protein doses from 7.6-167 ug, the radiation doses delivered per animal for labeled 81C6 were 50, 250, 500, and 1000 uCi and 500 and 1000 uCi for 45.6. Tumors were measured thrice weekly until the individual tumor volume exceeded 5000 mm³. Tumor response was measured by growth delay, where the difference in days between the medians of the treatment and control groups to reach 1000 or 5000 mm³ was compared by the Wilcoxon Rank Sum test, and by comparing the number of tumors that had regression in volume after treatment using the Fisher Exact test. Statistically significant growth delays at 1000 mm³ were noted in 1/3 experiments with 500 uCi 81C6 (p=.000) and 2/3 for 1000 uCi 81C6 (p=.001, .000). In none of the other groups was a statistically significant response noted. At 5000 mm³, statistically significant growth delays were seen with radiolabeled 81C6 in 1/1 experiments at 250 uCi (p=.02), 4/4 at 500 uCi (p=.03-.000), and 2/2 at 1000 uCi (p=.001) and with 1/1 at 1000 uCi (p=.01). tumor regressors were noted in 0/39 animals treated with buffer and 0/20 with unlabeled 81C6; for radiolabeled 81C6, there were; 0/6 regressors at 50, 1/8 at 250, 7/39 at 500, and 15/28 at 1000 uCi. For radiolabeled 45.6, there were 0/10 regressors at 500 and 1/10 at 1000 uCi. Statically significant tumor regression was seen only at doses of 500 and 1000 uCi of 131-I-81C6. In this xenograft model, radiolabeled specific anti-glioma Mab

demonstrated therapeutic efficacy. The promising results obtained in this animal model suggested a potential value for this form of therapy against human malignant gliomas.

11:30 PRESIDENTIAL ADDRESS

Phanor L. Perot, M.D., Ph.D.

Introduction - Griffith R. Harsh, III, M.D.

Saturday, November 8

SCIENTIFIC SESSION IV

MALIGNANT BRAIN TUMORS, CURRENT CONCEPTS

MODERATOR: M.S. Mahaley, Jr., M.D.

**8:30 INNOVATIVE CURRENT THERAPIES
FOR MALIGNANT GLIOMAS**

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

9:00 CHROMOSOME AMPLIFICATION AND OENCOGENES

Sandra H. Bigner, M.D.

9:30 MONOCLONAL ANTIBODIES TO BRAIN TUMORS

Darrell D. Bigner, M.D.

10:00 PHOTOCHEMOTHERAPY

Stephen K. Powers, M.D.

10:30 COFFEE BREAK

10:45 REGIONAL ARTERIAL INFUSIONS

John R. Kapps, M.D.

11:15 CHEMOTHERAPY OF CHILDHOOD TUMORS

Henry Friedman, M.D.

11:45 INTERFERON AND ACTIVE IMMUNOTHERAPY

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

12:15 FINAL ANNOUNCEMENTS

12:30 ADJOURN

*Presenting Author

NOTES

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RESIDENTS PAPER AWARD WINNERS

— WINNER —

**MICHAEL G. NOSKO, M.D.
UNIVERSITY OF ALBERTA**

*"The Effect of Clot Removal at 24 Hours on Chronic Vasospasms
After Subarachnoid Hemorrhage in the Primate Model"*

— RUNNER UP —

**ERIC L. ZAGER, M.D.
MASSACHUSETTS GENERAL HOSPITAL**

*"Reducing Cellular Energy Requirements:
Screening for Agents That May Protect Against CNS Ischemia"*

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H. THOMAS BALLANTRINE, JR. (ELIZABETH) Massachusetts General Hospital 15 Parkman Street - ACC 312 Boston, Massachusetts 02114	1951
EDWIN B. BOLDREY (HELEN) University of California Hospital 3rd Avenue and Parnassus San Francisco, California 94143	1941
E. HARRY BOTTERELL (MARGARET) 2 Lakeshore Boulevard Kingston, Ontario, Canada K7M 4J6	1938
HOWARD A. BROWN 2841 Ptarmigan Dr., #1 Walnut Creek, California 94595	1939
HARVEY CHENAULT (MARGARET) 2370 Nicholasville Road Lexington, Kentucky 40503	1938
GALE C. CLARK (MARION) University of California Medical Center San Francisco, California 94143	1970
DONALD F. COBURN (ELLIE) The Plaza 812 1303 Delaware Avenue Wilmington, Delaware 19806	1938
EDWARD W. DAVIS (BARBARA) 27851 Sweetbriar Road Troutdale, Oregon 97060	1949
RICHARD DESAUSSURE (PHYLLIS) 920 Madison Avenue Memphis, Tennessee 38103	1962

DONALD F. DOHN (CAROLYN) Singing River Neurosurgical Associates 3003 Short Cut Road Pascagoula, Mississippi 39567	1968
R.M. PEARDON DONAGHY (FRANCES) P.O. Box 5035 RDI - Horn of the Moon Road Montpelier, Vermont 05602	1970
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FRANCIS A. ECHLIN (LETITIA) P.O. Box 342 New Paltz, New York 12561	1944
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THEODORE C. ERICKSON (MARTHA) 425 North Livingston Street Madison, Wisconsin 53703	1940
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JOHN R. GREEN (GEORGIA) Barrow Neurological Institute 2910 No. 3rd Avenue Phoenix, Arizona 85013	1953
JAMES GREENWOOD, JR. (MARY) 1839 Kirby Avenue Houston, Texas 77019	1952
WALLACE B. HAMBY (ELEANOR) 2001 N.E. 47th Court Fort Lauderdale, Florida 33308	1938
JESS D. .RRMANN (MARY JO) Post Office Bos 135 Mountain Pine, Arkansas 71956	1948
WILLIAM S. KEITH (ELEANOR) 55 St. Leonards Crescent Toronto, Ontario, Canada M4N 3A7	Founder
ROBERT S. KNIGHTON (LOUISE) 9388 Avenida San Tinetto Cherry Valley, California 92223	1966
WILLIAM M. LOUGHEED (GRACE) Room 219, 7th Floor Toronto General Hospital 101 College Street Toronto, canada M5G 1L7	1962
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FRANK MAYFIELD 506 Oak Street Cincinnati, Ohio 45219	Founder
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WILLIAM F. MEACHAM (ALICE) Vanderbilt University Hospital Division of Neurosurgery Nashville, Tennessee 37232	1952
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ROBERT H. PUDENZ (RITA) 574 Garfield Avenue South Pasadena, California 91030	1943
JOHN RAAF (LORENE) 1120 N.W. 20th Avenue, #100 Portland, Oregon 97209	Founder
AIDEN A. RANEY (MARY) 2010 Wilshire Blvd. Suite 203 Los Angeles, California 90057	1946

THEODORE B. RASMUSSEN (CATHERINE) 29 Surrey Drive Montreal, Quebec, Canada H3P 1B2	1947
HENRY G. SCHWARTZ (REEDIE) Barnes Hospital Plaza Division of Neurological Surgery St. Louis, Missouri 63110	1942
C. HUNTER SHELLEN (ELIZABETH) 414 Fairmont Avenue Pasadena, California 91105	1941
HOMER S. SWANSON (LaMYRA) 3649 Peachtree Rd., N.E. Unit 205 Atlanta, Georgia 30319	1949
WILLIAM H. SWEET (ELIZABETH) 309 Goddard Avenue Brookline, Massachusetts 02146	1950
JOHN TYTUS (VIRGINIA "Gina") Mason Clinic Seattle, Washington 98107	1967
ALFRED UIHLEIN (IONE) 200 First Street S.W. Rochester, Minnesota 55901	1950
A. EARL WALKER (AGNES) 1477 Wagontrain Drive, S.E. Albuquerque, New Mexico 87123	1938
EXUM WALKER (NELLE) 490 Peachtree Street, N.E. Atlanta, Georgia 30308	1938
BENJAMIN B. WHITCOMB (MARGARET) 50 Union Street Ellsworth, Maine 04605	1947

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WILLIS E. BROWN, JR. (ANN) Division of Neurosurgery 7703 Floyd Curl Drive San Antonio, Texas 78284	1984
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W. KEMP CLARK (FERN) 5323 Harry Hines Blvd. Dallas, Texas 75235	1970

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WILLIAM H. FEINDEL (FAITH) Montreal Neurological Institute 3801 University Street Montreal, Quebec, Canada H3A 2B4	1959
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DECEASED MEMBERS**DATE
ELECTED**

DR. SIXTO OBRADOR ALCALDE Madrid, Spain (Honorary)	4/27/67	
DR. JAMES R. ATKINSON Phoenix, Arizona (Active)	2/78	1970
DR. PERCIVAL BAILEY Evanston, Illinois (Honorary)	8/10/73	1960
DR. WILLIAM F. BESWICK Buffalo, New York (Active)	5/12/71	1959
DR. SPENCER BRADEN Cleveland, Ohio (Active)	7/20/69	Founder
DR. F. KEITH BRADFORD Houston, Texas (Active)	4/15/71	1938
DR. WINCHELL McK. CRAIG Rochester, Minesota (Honorary)	2/12/60	1942
DR. ARTHUR ELVIDGE Montreal, Quebec, Canada (Senior)	1/17/85	1939
DR. GEORGE EHNI (Senior)	9/2/86	1964
DR. JOSEPH P. EVANS Kensington, Maryland (Senior)	5/8/85	Founder
DR. WESLEY A. GUSTAFSON Jensen Beach, Florida (Senior)	7/16/75	1942

DR. HANNIBAL HAMLIN	6/28/82	1941
	6/28/ε	
DR. HENRY L. HEYL (Senior)	3/10/75	1951
DR. OLAN R. HYNDMAN Iowa City, Iowa (Senior)	6/23/66	1942
MR. KENNETH G. JAMIESON Brisbane, Australia (Corresponding)	1/28/76	1970
SIR GEOFFREY JEFFERSON Manchester, England (Honorary)	3/22/61	1951
HUGO KRAYENBUHL Zurich, Switzerland (Honorary)	1985	1974
DR. WALPOLE S. LEWIN Cambridge, England (Corresponding)	1/23/80	1973
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DR. KENNETH G. MCKENZIE Toronto, Ontario, Canada (Honorary)	2/11/64	1960
DR. JAMES M. MEREDITH Richmond, Virginia (Active)	12/19/62	1946
DR. W. JASON MIXTER Woods Hole, Massachusetts (Honorary)	3/16/68	1951

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DR. BARNES WOODHALL Durham, North Carolina (Senior)	1985	1941

— NOTES —

**AMERICAN ACADEMY OF
NEUROLOGICAL SURGERY
1986 ANNUAL MEETING
EVALUATION**

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

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

- Excellent
- Good
- Poor

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

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

(3) Did the speakers aim their talks:

- Too high
- Too low
- Just about right

— SCIENTIFIC PROGRAM —

Thursday's Sessions Excellent Good Poor
Comments _____

Friday's Sessions Excellent Good Poor
Comments _____

Saturday's Sessions Excellent Good Poor
Comments _____

— SOCIAL PROGRAM —

Comments _____

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

Changes of address and/or telephone (indicate office or home address):

Please Print Name: _____

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