THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY



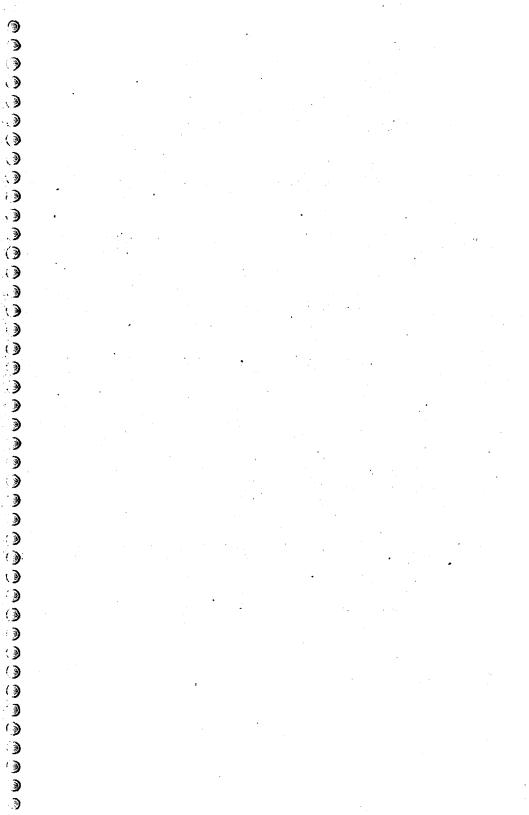
58th Annual Meeting



September 18 - 22, 1996



Jointly Sponsored by The American Association of Neurological Surgeons



THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY



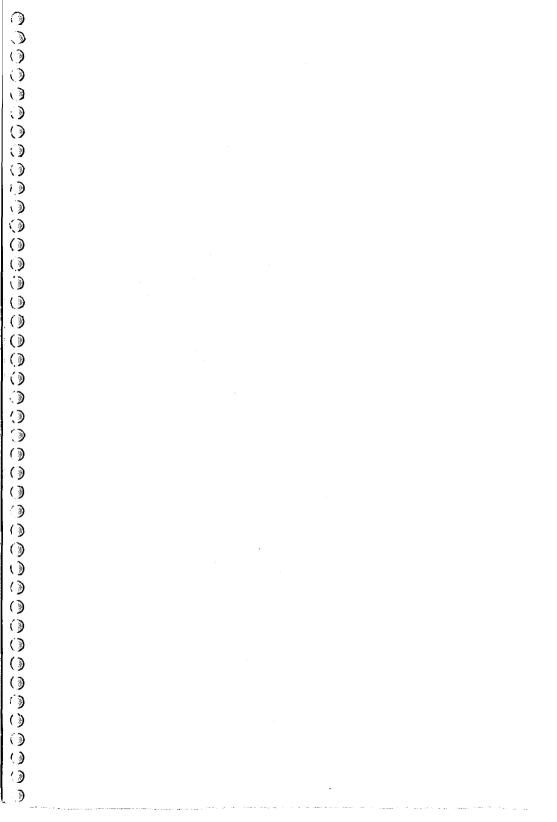
58th Annual Meeting



September 18 - 22, 1996



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HISTORIAN

Byron C. Pevehouse

PROGRAM COMMITTEE

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LOCAL HOSTS

L. Nelson (Nick) and Bonnie Hopkins

THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

Activities Program

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Tuesday, September 17			
4:00 PM - 7:30 PM	ABNS Exam Committee Review Pierce Room		
7:30 PM - 9:30 PM	ABNS Dinner Main Dining Room		
Wednesday, September 18			
8:00 AM - 3:00 PM	ABNS Primary Exam Committee Pierce Room		
2:00 PM - 5:00 PM	Registration Lower Lobby, Area B		
2:00 PM - 5:00 PM	Speaker Ready Room Editing Room (across from Chesapeake Room)		
2:00 PM - 3:00 PM	Executive Committee Buchanan Room		
3:00 PM - 5:00 PM	Membership Committee Buchanan Room		
6:30 PM - 8:00 PM	Welcome Reception* Cameo Ballroom		
7:30 PM - 10:00 PM	Dinner* Crystal Room		
7:30 PM - 10:00 PM	Executive Committee Dinner* Taft Room		
* Evening Wear:			

Gentlemen - Business suits, jackets and slacks, neckties or black tie Ladies - Dresses (short or long), evening suits

Thursday, September 19	
6:00 AM - 2:00 PM	Speaker Ready Room Editing Room
7:00 AM - 8:00 AM	Breakfast/Business Meeting (Members only)* Crystal Room
* Guest breakfast available Main Dining Room at 7:30	in Draper's Cafe at 7:00 AM or the OAM
8:00 AM - 1:00 PM	Registration Chesapeake Room Foyer
8:00 AM - 1:00 PM	Symposium & Scientific Session I Chesapeake Room
10:05 AM - 10:30 AM	Coffee Break Colonial Lounge & Terrace
1:00 PM - 2:30 PM	ABNS Advisory Council Wilson Room
1:04 PM - 6:00 PM	Golf Sign up at Registration Desk
2:45 PM - 5:00 PM	Journal of Neurosurgery Editorial Board Pierce Room
3:00 PM - 5:00 PM	Tennis Sign up at Registration Desk
6:30 PM - 8:00 PM	Reception Colonial Lounge & Terrace
7:30 PM - 10:00 PM	Dinner* Main Dining Room
	

^{*} Sign up for 7:30 or 8:00 PM seating at Registration Desk

Friday.	Septemb	er 20
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6:00 AM - 2:00 PM	Speaker Ready Room Editing Room
7:00 AM - 8:00 AM	Breakfast/Business Meeting (Members only)* Crystal Room
 Guest breakfast available in Main Dining Room at 7:30 	Draper's Cafe at 7:00 AM or the AM
8:00 AM - 12:30 PM	Registration Chesapeake Room Foyer
8:00 AM - 12:30 PM	Symposium & Scientific Session II Chesapeake Room
10:05 AM - 10:35 AM	Coffee Break Colonial Lounge & Terrace
11:45 PM - 12:30 PM	Presidential Address: John M. Tew, Jr., MD Chesapeake Room
1:12 PM - 6:00 PM	Golf Tournament Sign up at Registration Desk
3:00 PM - 5:00 PM	Tennis Sign up at Registration Desk
6:30 PM - 8:00 PM	Reception and Putting Contest* Chesapeake Bay & Terrace

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* Sign up for contest at Registration Desk

7:30 PM - 10:00 PM Dinner*
Main Dining Room

* Sign up for 7:30 or 8:00 PM seating at Registration Desk

)		
))	Saturday, September 21	
))	6:00 AM - 12:45 PM	Speaker Ready Room Editing Room
)	7:00 AM - 8:25 AM	Breakfast for Members and Guests Individual arrangements*
))	* Draper's Cafe at 7:00 AM or	the Main Dining Room at 7:30 AM
)	7:30 AM - 8:25 AM	The Society of Neurological Surgeons Lee Room
•		
)	8:25 AM - 12:45 PM	Registration Chesapeake Room Foyer
))	8:25 AM - 12:45 PM	Scientific Session III & IV Chesapeake Room
•	9:45 AM - 10:30 AM	Keynote Address: Leadership Without Easy Answers
)		Ronald Heifetz, Kennedy School of Government, Harvard University
))		Chesapeake Room
)	10:30 AM - 11:00 AM	
)	*Group Photograph to be taken durin Sign up at Registration Desk for a c	
)	1:04 PM - 6:00 PM	Golf
))	1.04 1 141 - 0.00 1 141	Sign up at Registration Desk
))	3:00 PM - 5:00 PM	Tennis Tournament Sign up at Registration Desk
)	6:30 PM - 7:30 PM	Reception Colonial Lounge & Terrace
)))	7:30 PM - 12:00 AM	Banquet - Dance Black tie Chesapeake Room
)	Sunday, September 22	Departures
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SCHEDULE OF ACTIVITIES FOR SPOUSES

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Wednesday, September 18

6:30 PM - 8:00 PM Welcome Reception* Cameo Ballroom

7:30 PM - 10:00 PM Dinner* Crystal Room

7:30 PM - 10:00 PM Executive Committee Dinner* Taft Room

* Evening Wear:

Gentlemen - Business suits, jackets and slacks, neckties or black tie Ladies - Dresses (short or long), evening suits

Thursday, September 19

Walking Course on your own; maps available at the Meeting Registration Desk

7:00 AM - 10:00 AM Breakfast - Individual Arrangements*

* Draper's Cafe from 7:00 - 10:00 AM or Main Dining Room from 7:30 - 10:00 AM

9:00 AM - 11:00 AM Hospitality Suite Spring Room

10:00 AM - 11:00 AM Book Review

Fair and Tender Ladies by Lee Lewis

Spring Room

1:04 PM - 6:00 PM Golf

Sign up at Registration Desk

3:00 PM - 5:00 PM

Sign up at Registration Desk

3:00 PM - 5:00 PM

Hospitality Suite* Spring Room

* Card tables available; bridge and backgammon tables are in the nearby Green and Trellis Lobbies

4:15 PM - 5:00 PM Readings of Essays, Poetry, and Short Stories Spring Room

Tea and Concert

4:15 PM - 5:00 PM Main Lobby

6:30 PM - 8:00 PM Reception Colonial Lounge & Terrace

7:30 PM - 10:00 PM Dinner* Main Dining Room

* Sign up for 7:30 or 8:00 PM seating at Registration Desk

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•	Friday Sentember 20	
•	Friday, September 20 Walking Course on your own	
•	7:00 AM - 10:00 AM	Breakfast - Individual Arrangements*
•	* Draper's Cafe from 7:00 - 10:00	AM or
)	Main Dining Room from 7:30 -	· 10:00 AM
)	9:00 AM - 11:00 AM	Hospitality Suite Spring Room
)	10:30 AM - 11:30 AM	Interior Tour of The Greenbrier* Meet guide at the Spring Room
•	* Sign up at Registration Desk	, ,
)	1:12 PM - 6:00 PM	Golf Tournament Sign up at Registration Desk
•	3:00 PM - 5:00 PM	Tennis
)		Sign up at Registration Desk
))	3:00 PM - 5:00 PM	Hospitality Suite Spring Room
)	4:15 PM - 5:00 PM	Tea and Concert Main Lobby
))	6:30 PM - 8:00 PM	Reception and Putting Contest*
)	* Sign up for contest at Registra	Chesapeake Bay & Terrace ation Desk
) .	7:30 PM - 10:00 PM	Dinner*
)	• Sign up for 7:30 or 8:00 PM	Main Dining Room
)	• •	scaling at Registration Desk
)	Saturday, September 21	
)	Walking Course on your own	
)	7:00 AM - 10:00 AM • Draper's Cafe from 7:00 - 10:0	Breakfast - Individual Arrangements*
	Room from 7:30 - 10:00 AM	NAME OF TAKEN DIRECTOR
)	9:00 AM - 11:00 AM	Hospitality Suite
)	1 04 PM 4 6 00 PM	Spring Room
)	1:04 PM - 6:00 PM	Golf Sign up at Registration Desk
)		
))	4:15 PM - 5:00 PM	Tea and Concert Main Lobby
•	3:00 PM - 5:00 PM	Tennis Tournament Sign up at Registration Desk
))	6:30 PM - 7:30 PM	Reception Colonial Lounge & Terrace
)	7:30 PM - 12:00 AM	Banquet - Dance
)		Black tie
)		Chesapeake Room
)	Sunday, September 22	Departures
)		9

SCIENTIFIC PROGRAM AMERICAN ACADEMY OF NEUROLOGICAL SURGERY 1996 LEARNING OBJECTIVES September 18-22, 1996

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Jointly Sponsored by The American Association of Neurological Surgeons

Following the Scientific Sessions, the participants will be able to:

Critique the value of the recommended surgical and nonsurgical options presented in the scientific papers.

Evaluate the relevance of the research methodologies, the findings, and the potential usefulness in practice of the topics presented for cerebrovascular, neoplastic, spinal and developmental and functional nervous system diseases.



The American Association of Neurological Surgeons is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

The American Association of Neurological Surgeons designates this continuing medical education activity for 14.7 credit hours in Category I of the American Medical Association.

) SCIENTIFIC PROGRAM AMERICAN ACADEMY OF NEUROLOGICAL SURGERY September 19-22, 1996 Jointly Sponsored by The American 7 Association of Neurological Surgeons Thursday, September 19 WELCOME - John Tew. President 8:00-8:05 8:05-10:05 SYMPOSIUM - Evaluating and Improving **Neurosurgical Results** Moderators: Julian Hoff, Martin Camins 8:05-8:25 Critical Pathways - Charles Rich 8:25-8:45 Assessing and Reporting Complications -Roberto Heros Outcome Analysis - Stephen Haines 8:45-9:05 Institutional Guidelines - Joseph Hahn 9:05-9:25 9:25-9:45 Federal Regulations - Paul Nelson

Discussion

Coffee Break

9:45-10:05

10:05-10:30

Thursday, September 19 Cont'd		
	SCIENTIFIC SESSION I Moderators: Edward Connolly, Harry van Loveren	
10:30-10:55	Pathobiology of Cerebral Cavernous Malformations: An Integrative Paradigm Issam A. Awad, Murat Gunel, KM Desai, John Pizzonia, Richard P. Lifton	
10:55-11:20	Posteroventral Pallidotomy: Indications, Surgical Technique and Clinical Outcome Kim Burchiel, Jacques Favre, Jamal Taha	
11:20-11:45	Pathophysiology of Syringomyelia Associated with Chiari I Malformation JD Heiss, H DeVroom, A Eidsath, T Talbot, N Patronas, EH Oldfield	
11:45-12:10	Differentiation between Cortical Atrophy and Hydrocephalus using ¹ H MRS Stefan Blum, J. Gordon McComb, Brian D. Ross	
12:10-12:35	The Risk of Major Morbidity after Stereotactic Radiosurgery <u>Douglas Kondziolka</u> , John C. Flickinger, L. Dade Lunsford, David J. Bissonette	
12:35-12:50	Academy Award Presentations, Bryce Weir, Chair Drug-Regulated Apoptotic Death in a Rat Glioma Model Mediated by a Novel Retroviral Vector Academy Award Paper John S. Yu, Massachusetts General Hospital, Boston, Massachusetts	
12:50-1:00	Academy Award Honorable Mention In Vivo Angioplasty Prevents the Development of Vasospasm in Canine Carotid Arteries: Pharmacological and Morphological Analyses Joseph F. Megyesi, McKenzie Health Sciences Center, Edmonton, Alberta	

8:00 AM - 10:00 AM	SYMPOSIUM - New Directions in Image- Guided Minimally Invasive Neurosurgery Moderators: John Tew, Kalmon Post
8:00-8:20	Frameless Sterotaxis - Peter Heilbrun
8:20-8:40	Surgery in the MRI - Peter Black
8:40-9:00	Intravascular Therapies – L. N. Hopkins
9:00-9:20	Spinal Endoscopy - Volker Sonntag
9:20-9:40	Video-based Systems - David Thomas
9:40-10:05	Discussion
10:05-10:35	Coffee Break
0:35-11:00	SCIENTIFIC SESSION II Moderators: Roberto Heros, Volker Sonntag Extra-foraminal Lumbar Disc Herniations V. Rajaraman, N. Swami, W. S. Tobler, S. B. Dunsker The Miami Project – Scientific Achievements Barth A. Green, Richard P. Bunge
11:20-11:45	Clinical and Experimental Studies of the Value of Surgical Decompression in Spinal Cord Injury Charles H. Tator
11:45-12:30	Presidential Address: John Tew Introduced by Edward Connolly

Saturday, September 21

	SCIENTIFIC SESSION III Moderators: David Piepgras, Fredric Meyer
8:25-8:45	AVM Radiosurgery: Truths and Consequences L.D. Lunsford, B. E. Pollock, D. Kondziolka, A. Maitz, J.C. Flickinger
8:45-9:05	Mechanism of Radiation Effect On Arterial Smooth Muscle Cells and Endothelium Marc M. Mayberg
9:05-9:25	The Role of Neuronal NO in Collateral and Normal rCBF Christopher M. Loftus, Scott C. Robertson
9:25-9:45	Repair of Carotid Endarterectomy with Collagen Dacron Graft Fredric B. Meyer, Wanda L. Windschitl
9:45-10:30	Keynote Address: Leadership Without Easy Answers Ronald Heifetz
10:30-11:00	Coffee Break

Saturday, September 21 Cont'd		
	SCIENTIFIC SESSION IV Moderators: L.N. Hopkins, Keith Black	
11:00-11:20	Drug Delivery by Intracerebral Clysis in a Rat Glioma Model Jeffrey Bruce, B. Birch, J. Johnson, J. Yoon, C. Lycette, A. Falavigna	
11:20-11:40	Comprehensive Image-Guided Surgery of Sellar Tumors Michael A. Schulder, Peter Carmel	
11:40-12:00	Dorsal Cervical-Medullary Bulge in Chiari I Malformations <u>Peter W. Carmel</u> , Jacqueline A. Bello	
12:00-12:20	Brain Tumor Invasion – Biological Rationale and Preclinical Studies Mark L. Rosenblum, Tom Mikkelsen, Klaus Edvardsen, Kevin Nelson, Elise Kohn, Robert Smith, Donna Spencer, David Rasnick, Bonnie Sloane	
12:20-12:40	Immunobiology of Primary Brain Tumors WH Brooks, RL Roszman, LE Elliott	
12:45	Adjourn	
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Thursday, September 19 10:30 AM

Pathobiology of Cerebral Cavernous Malformations: An Integrative Paradigm

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Awad I, Gunel M, Desai KM, Pizzonia J, Lifton R

The cerebral cavernous malformation (CCM) is a common vascular lesion of the brain, affecting 0.5% of the population, and manifesting myriad neurologic sequelae including headache, epilepsy, focal neurologic deficits and hemorrhagic stroke. We review epidemiologic data from a multi-institutional data base of cases evaluated during one decade. There is marked difference in probability of lesion behavior among the genders, and in different age groups. Lesions which have hemorrhaged previously are more likely to hemorrhage again and cause clinical disability. Probability of untoward clinical events is driven by host and lesion factors, and does not follow a simple lesion-year hemorrhagic risk model. In collaboration with twelve centers across the United States, we have examined the genetics of the lesion, and mapped a gene causing CCM to 7q11.2-q21. Linkage dysequilibrium studies in Hispanic Americans have demonstrated a preserved haplotype in the region of the CCM gene in familial and apparently sporadic cases, indicating that all CCM cases in this population are likely genetic and inherited from a common ancestor. We have uncovered evidence of genetic locus heterogeneity among non-Hispanic patients, with one additional gene likely causing the disease. Vascular biology studies have included immunohistochemical staining for markers of vessel wall maturity, and expression of angiogenesis growth factors. Studies on lesion epileptogenicity have focused on iron assays and impact of heme breakdown products on glial and neuronal excitability. Data from these studies is integrated into a disease paradigm with potential clinical applications including genetic screening, prediction of penetrance and clinical sequelae, and novel strategies for molecular modification of lesion behavior.

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Thursday, September 19 10:55 AM

Posteroventral Pallidotomy: Indications, Surgical Technique and Clinical Outcome

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Burchiel K. Favre J. Taha J

From January 1993 through May 1996, we performed 113 posteroventral pallidotomies (PVP) on 82 patients. In this series, two patients had probable "Parkinson's plus" syndromes, one had hemiballismus from cerebral palsy, and one had a combination of simultaneous pallidotomy with a thalamotomy performed contralaterally. The remainder (78) were patients with previously Sinemet-responsive Parkinson's disease, now medically intractable despite maximal adjunctive medications. Indications for PVP included in order of decreasing importance: Predominant symptoms of Levodopa-induced dyskinesias, bradykinesia, rigidity, dystonia, severe on-off fluctuations, freezing, gait disturbance, and tremor. Patients underwent Fast Spin Echo Inversion Recovery (FSE IR) MRI imaging for stereotactic localization using the Leksell frame system. Pallidotomy target parameters of 19-22 mm lateral, 2 mm anterior to the midcommissural point and 1-7mm below the intercommissural plane were used. Microelectrode recording in GPe/GPi, and in optic tract was used to confirm target localization. Anatomic (MRI) and physiologic (microelectrode) target localizations differed by <2mm in 98% of cases. Thus, MRI directed targets were rarely altered by microelectrode recordings. Furthermore, macrostimulation was used to rule out target localization within the internal capsule and optic tract. Typically three sequential lesions were created at 840 C for 60 seconds with a 2 X 1.3mm lesion electrode. Outcome measures showed that the majority of patients were satisfied with the procedure, and that excellent to good results were obtained in 65% of patients. Age appeared to be an important determinant of outcome in that patients over 70 years had a good/excellent recovery 37% of the time, while those under 70 had good/excellent results 72% of the time. Predictors of good outcome included age < 70, dyskinesias, rigidity, bradykinesia, and tremor. Dyskinesias, bradykinesia, rigidity, tremor, and walking were all significantly improved. Some deterioration in voice volume and articulation occurred in the majority of patients, and speech deterioration was quantitatively worse with bilateral pallidotomy. Overall, in comparing unilateral and bilateral pallidotomy, improvements (e.g., dyskinesia, rigidity, tremor and walking were better after bilateral pallidotomy, whereas complications with bilateral pallidotomy were more prominent (e.g., decreased voice volume and articulation, increased drooling). In summary, pallidotomy is a valuable surgical treatment for parkinsonian symptoms, but complications, particularly with bilateral procedures, are not negligible.

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NOTES

Thursday, September 19 11:20 AM

Pathophysiology of Syringomyelia Associated with Chiari I Malformation

Heiss JD, DeVroom H, Eidsath A, Talbot T, Patronas N, Oldfield, EH

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Recent investigations using anatomic and cine MRI combined with intraoperative ultrasound (IOUS) in patients with syringomyelia associated with a Chiari I malformation of the cerebellar tonsils suggest that syringomyelia may result from occlusion of the subarachnoid space (SAS) at the foramen magnum by limiting the free pulsatile movement of CSF between the cranial and spinal SAS, partial entrapment of the CSF in the spinal SAS, and excess pulsatile pressure waves in the spinal SAS. To investigate this further, we performed a clinical protocol in which clinical assessment, anatomic and cine MRI, preoperative pressures measured simultaneously from the syrinx and the cervical and lumbar SAS at rest and with Valsalva and Queckenstedt maneuvers, intraoperative pressure measurements from the same sites (and a cerebral ventricle for the intraoperative component of the study) combined with IOUS monitoring of the spinal cord, syrinx, and tonsils, and postoperative MRI and pressure testing similar to the preoperative testing was performed. The pressure waves in the syrinx and SAS at the various sites were recorded digitally and the cine MRI and IOUS were related to the cardiac cycle (EKG) so that the physiological and anatomic changes that occur over the course of the cardiac cycle could be related and compared. The results, which confirm the hypothesis of the study. and their clinical significance will be presented.

Thursday, September 19 11:45 AM

Differentiation between Cortical Atrophy and Hydrocephalus using ¹H MRS

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Blum S, McComb JG, Ross BD

Quantitative ¹H MRS to determine cerebral metabolite patterns, and MRI to determine CSF flow, were applied to 12 patients with ventricular dilatation - Group A, cortical atrophy (N=5), or Group B, hydrocephalus (N=7) - and in 9 normal controls.

While mean brain water (Group A = $80\% \pm 6$; Group B = $86\% \pm 5$; normal = $95\% \pm 4$) did not differ between the 2 groups of patients and controls, ¹H MRS distinguished those patients with cortical atrophy (Group A) (*NAA/Cr = 0.69 ± 0.17 , vs normal = 1.06 ± 0.16 ; P < 0.002; [NAA] = 5.9 ± 1.3 mmoles/kg. vs normal 8.0 ± 1.4 ; P < 0.02) from those with hydrocephalus (Group B) (NAA/Cr = 1.16 ± 0.11 ; [NAA] = 9.2 ± 1.2 ; P > 0.13 and P > 0.07). Lactate levels were elevated in 3/5 patients with cortical atrophy, but 0/7 of those with hydrocephalus. Mean absolute concentrations (mmoles/kg) of the five major cerebral osmolytes were 41 ± 4 (Group A), 43 ± 6 (Group B), and 42 ± 4 (normal), so that despite massive brain deformation, constant osmolality was maintained.

Thinning of the cortical mantle in hydrocephalus may result from osmotically driven reduction in individual cell-volumes, (shrinkage), rather than brain-compression.

^{*}N-acetylaspartate/creatine

Thursday, September 19 12:10 PM

The Risk of Major Morbidity After Stereotactic Radiosurgery Kondziolka D, Flickinger JC, Lunsford LD, Bissonette DJ,

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Are current dose-selection guidelines acceptable for radiosurgery? We performed an analysis of major morbidity after Gamma Knife radiosurgery from a population of 1860 patients, to define the risks for parenchymal or cranial nerve deficits related to lesion type, location, size, and radiation dose. We selected prospectively a dose based on a theoretical risk of < 3% for permanent tissue injury. Morbidity occurred within 3 years.

1600 patients were evaluable at least 12 months after radiosurgery. Diagnosis included AVM (n=531), cavernous malformation (CM) (n=49), acoustic tumor (n=318), meningioma (n=204), pituitary tumor (n=63), malignant tumor (metastasis/glioma) (n=309), other tumor (n=94), and trigeminal neuralgia (n=32). We determined that major morbidity impacted function (e.g., hemiparesis, sensory loss, ataxia, hemianopsia, dysphasia, visual loss, dementia, lower cranial nerve palsy) or death. Acoustic, facial, or trigeminal deficits were excluded. Morbidity was defined by clinical exam and neuroimaging. Major morbidity developed in 30 patients (1.9%): 16 AVM (3.0%), 5 CM (10%), 1 acoustic (0.3%), 4 meningioma (2.0%), 1 pituitary (1.6%), 2 malignant tumors (0.6%), 1 other (1.1%), and no trigeminal neuralgia (0%). 11/16 AVMs were located in the brainstem or diencephalon. Lesion volume was related significantly to risk. 8 of 30 patients (27%) had received prior irradiation. 26 of 30 patients improved (87%). 14 of 1600 patients (0.9%) developed permanent major neurologic deficits after radiosurgery. Death following radiation-related neurologic morbidity occurred in 2 patients (.12%).

Dose-selection guidelines proved accurate. Major morbidity was 1.9% (below the 3% predicted risk), but was increased in patients with large lesion volumes, and those who received prior irradiation. The relative risks of radiosurgery should be compared to the risks of other surgical approaches during selection of management options.

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Thursday, September 19 12:35 PM

Drug-Regulated Apoptotic Death in a Rat Glioma Model Mediated by a Novel Retroviral Vector

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John S. Yu, Miguel Sena-Esteves, Werner Paulus, Xandra O. Breakefiled, Steven A. Reeves

IL-1B-converting enzyme (ICE) is a member of a growing family of cysteine proteases shown to be a crucial component in the activation of a genetic program that leads to autonomous cell death in mammalian cells. In this study a murine ICE-lacZ fusion gene was introduced into a novel retroviral vector designed to achieve regulated ectopic expression of a foreign gene in mammalian cells. By delivering the ICE-lacZ gene within a retroviral vector and under the control of a tetracycline regulated promoter, we were able to utilize the intrinsic cell death program of ICE as a means for tumoricidal therapy in a rat brain tumor model. Both in culture and In vivo suppression of ICE-lacZ expression was extremely tight in the presence of tetracycline, as determined by the lack of X-gal positive tumor cells and by cell viability. When tetracycline was withdrawn, ICE-lacZ gene expression was rapidly turned on and apoptosis-mediated cell death occurred in essentially all tumor cells.

NOTES 27

Thursday, September 19 12:50 P.M.

In Vivo Angioplasty Prevents the Development of Vasospasm in Canine Carotid Arteries: Pharmacological and Morphological Analyses
Joseph F. Megyesi, J. Max Findlay, Bozena Vollrath, David A. Cook,
Ming H. Chen

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Background:

In order to study the effects of *in vivo* transluminal balloon angioplasty (TBA) on the structure and function of the arterial wall, a new canine model of hemorrhagic cerebral vasospasm of the high cervical internal carotid artery (ICA) was developed. This model was used to determine if TBA performed prior to clot placement could prevent the development of vasospasm.

Methods:

Twelve dogs underwent surgical exposure of both distal cervical ICAs, followed by baseline angiography. One randomly-selected ICA in each dog was then subjected to in vivo TBA and repeat angiography. Both distal ICAs were then surrounded with blood clots held by silastic sheaths. Seven days later angiography was repeated and all animals sacrificed. The ICAs in four animals were perfusion-fixed in situ for morphological analysis by electron microscopy, and the arteries in the remaining eight animals were removed, and immediately immersed in oxygenated Krebs solution. Contractile responses of isolated arterial rings from each ICA were recorded after treatment with potassium chloride (KCI), noradrenaline (NA), serotonin (5-HT), and prostaglandin F, (PGF,) while relaxations were obtained after pre-constriction with NA, in response to the calcium ionphore A, and bradykinin. The morphology and pharmacological responses of ICAs that had been exposed to blood with or without prior TBA, were compared with data obtained from control arterial segments of intact, more proximal regions of the ICAs from each animal.

Results:

TBA resulted in immediate angiographic enlargement of the ICA lumen that was still evident seven days later despire the placement of clotted blood around the artery. Scanning and transmission electron microscopy demonstrated flattening of the intima and internal elastic lamina in these dilated arteries, associated with patchy losses of endothelial cells. In contrast, ICAs which had been exposed to clotted blood but had not undergone prior TBA, developed consistent angiographic and morphological vasospasm. In comparison with control vessels and non-dilated vasospastic vessels, vessels dilated with TBA and then exposed to clotted blood showed significantly diminished responses to all compounds tested, with the exception of PGF_{2a} and bradykinin.

These results indicate that in vivo TBA results in a degree of functional impairment of vascular smooth muscle that persists for at least seven days. This result is consistent with previous observations of the acute effects of TBA in isolated arteries. Furthermore, these results support the hypothesis that normal smooth muscle function is required for the development of vasospasm. Finally, these results indicate that TBA performed prior to the onset of vasospasm prevents its development.

29

NOTES

Friday, September 20, 1996 10:35 AM Extra-Foraminal Lumbar Disc Herniations Rajaraman V, Swami N, Tobler WS, and Dunsker SB

Because of the advent of CT and MRI imaging, extra-foraminal or extreme lateral lumbar disc herniations are diagnosed more easily. To approach these lesions, some individuals continue to perform a unilateral laminectomy and facetectomy, but far lateral (extra-spinal) approaches are being performed more frequently. Moreover, recent biomechanical studies indicate there is increased translational movement following facetectomy alone, and the movements are worsened by combining facetectomy and discectomy.

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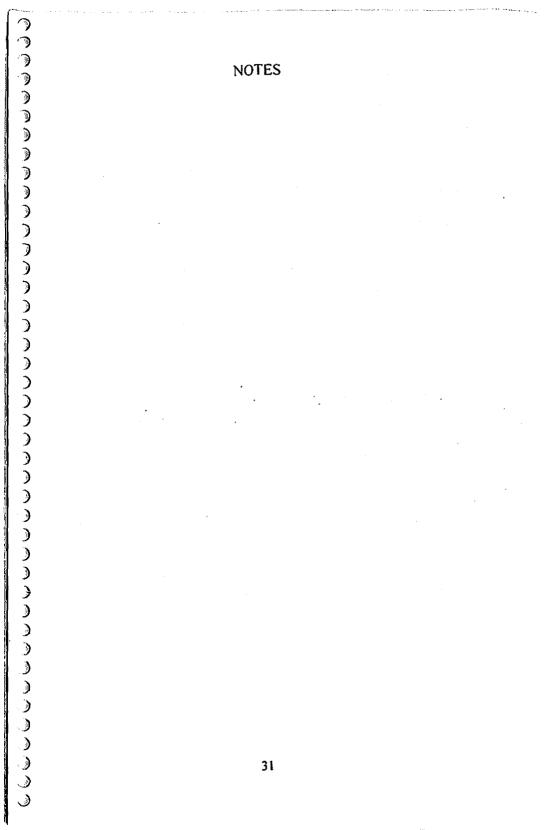
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We will present the important anatomy to know when performing these extra-spinal procedures, including the demonstration of an arterial arcade which encircles the main nerve trunk, and which endangers it. We believe that proper attention to this arcade and to the attached posterior primary ramus of the nerve will prevent the painful burning dysesthesias that commonly follow the extraspinal approach.

We will also present our clinical experience with the procedure.



Friday. September 20 11:00 AM The Miami Project - Scientific Achievements Green B. Bunge RP (

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The Miami Project to Cure Paralysis was established in 1985 with support from the University of Miami Department of Neurological Surgery, generous philanthropic gifts, and with a determination to find more effective treatment for patients with spinal cord injury. The faculty are members of the Department of Neurological Surgery who lead 15 laboratory groups, with expertise ranging from molecular biology to clinical physiology and rehabilitation, and including a comprehensive study of human spinal cord pathology and physiology. This unique group of basic and clinical scientists concentrating on a single clinical entity has: 1) shown that central cord syndrome may result from bilateral damage to the lateral corticospinal tract at the cervical level, rather than central cord cavitation; 2) developed a new method for stimulus evoked EMG monitoring during transpedicular lumbosacral spine instrumentation that reliably detects screw misplacement and decreases complications; 3) documented that assisted ejaculation and fertilization can allow many spinal cord injured men to successfully father children; 4) demonstrated that a spinal cord based central pattern generator for locomotion can be detected in selected cases of human spinal cord injury; 5) established that axons of the long tract of the spinal cord can be induced to regenerate by favorable cellular environment at the site of injury (for example transplantation of Schwann cells to midthoracic spinal cord injury; 6) devised reliable methods for the isolation and cultivation of Schwann cells from adult human nerve; 7) generated neuronal cell lines by genetic manipulations and established that some lines can express neuronal morphology appropriate to the brain region into which they are transplanted. This work and other studies will be presented.

NOTES 33

Friday, September 20 11:20 AM

Clinical and Experimental Studies of the Value of Surgical Decompression in Spinal Cord Injury

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There is a need for a randomized control trial of surgical decompression in patients with persisting compression of the cord after spinal cord injury. There is no definite evidence that decompression improves neurological recovery after either complete or incomplete acute spinal cord injury.

Several retrospective clinical studies have been performed including one by the author, which have failed to show improved recovery following surgical decompression. In contrast, several well controlled experimental studies including two from the author's laboratory have shown improved recovery following posttraumatic decompression of the spinal cord in various species. This presentation will review the past and current clinical and experimental studies of posttraumatic decompression of the cord. In addition, the results will be described of a recent retrospective analysis of the current incidence (66%) of surgical treatment of spinal cord injury in 36 U.S. and Canadian centres. Many of these operations were performed for purposes of decompression. The current protocol will be discussed for a proposed prospective control trial of surgical decompression in patients with persisting acute traumatic compression of the spinal cord.

Saturday, September 21 8:25 AM

AVM Radiosurgery: Truths and Consequences

Lunsford LD, Pollock BE, Kondziolka D, Maitz A, Flickinger JC

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Introduction:

To clarify recent pejorative reports about outcomes of AVM radiosurgery, we reviewed our experience in 315 patients evaluable at least 3 years afterwards. The obliteration and risk rates were assessed.

Methods & Materials:

Follow-up was available 98%; 197 patients (63%) completed 3 year angiography. MRI was used serially to assess AVM flow and to detect complications.

Results:

The overall angiographically confirmed obliteration rate was 72%. Depending on volume, the obliteration rate varied from 41% to 98%. The minimum nidus dose was the only significant predictor of obliteration. The major cause for failure was poor nidus resolution during intraoperative imaging. MRI proved to have a 96% sensitivity and a 100% specificity of detecting residual AVM flow. No difference in outcomes or obliteration rates were detected in patients who had only MRI follow-up. Deep AVMs had a 5x increased risk of neurological sequelae. A recent AVM hemorrhage before radiosurgery increased the risk of a repeat hemorrhage during the latency interval prior to obliteration. No AVM bled after MRI demonstrated obliteration.

Conclusions:

AVM radiosurgery is an effective strategy for many AVMs. Intraoperative MRI and angiography are critical to define the target volume. MRI alone can be used effectively to detect success, failure, and complications. AVMs with volumes that prevent minimal marginal doses > 16Gy should be considered for other treatment options, including staged radiosurgery.

NOTES

Saturday, September 21 8:45 AM Mechanism of Radiation Effect on Arterial Smooth Muscle Cells and Endothelium Mayberg, M (

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To determine by which radiation modulates injury for vascular smooth muscle cells (SMC) and endothelium, 108 rats were divided into three groups: (A) unilateral irradiation to cervical common carotid artery, (B) unilateral carotid balloon catheter injury, and (C) bilateral balloon injury with unilateral irradiation. Animals received from 100-2000 cGy (rads) and carotid arteries were processed for immunohistochemistry at 0-72 hours after treatment with terminal transferase end labeling (TUNEL - to detect apoptosis) and in situ nick translation (ISNT - to detect necrosis). Cultured rat aortic SMC and endothelium were placed in either serum-free medium (growth-arrested) or whole blood serum (growth-stimulated), radiated with 500-2000 cGy, and processed for TUNEL and ISNT at 0-72 hours.

Balloon catheter injury only (group B) produced immediate necrosis in SMC on the abluminal aspect of the media. The addition of radiation to balloon injury (group C) did not affect either TUNEL or ISNT labeling. Radiation alone (group A) caused dose-dependent apoptosis in endothelium, but not SMC; no necrosis was observed at any dose. Similarly, irradiated cultured SMC in either media showed mild morphologic changes compared to controls, but no evidence of necrosis or apoptosis up to 2000 cGy. Endothelium in vitro, on the other hand, showed considerable sensitivity to radiation with significant apoptosis at doses as low as 500 cGy, in both media. These data suggest that the primary effect of gamma radiation on medium-sized arteries involves apoptosis in endothelial cells. Strategies to either inhibit or augment the effect of radiation on arterial endothelium may be useful in future treatment of intracranial lesions.

NOTES

Saturday, September 21 9:05 AM The Role of Neuronal NO in Collateral and Normal rCBF Loftus CM, Robertson SC 6

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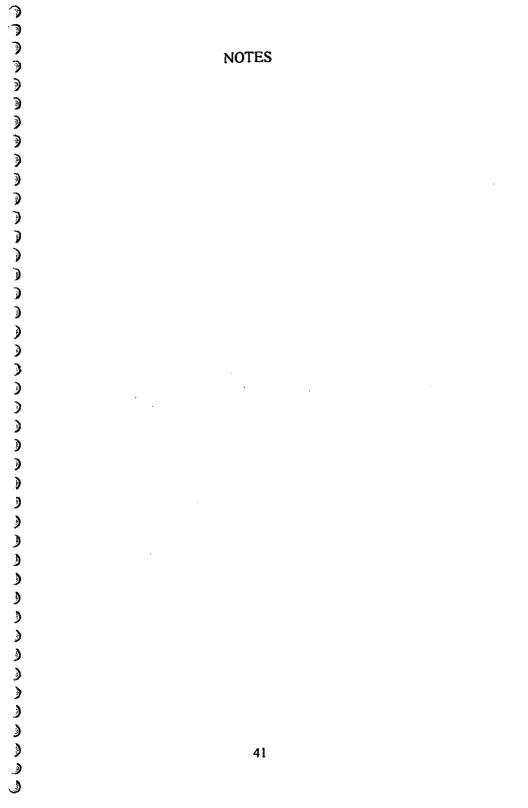
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N-Methyl-D-aspartate (NMDA) produces cerebral vasodilation in response to receptor activation, probably mediated by the neuronal isoform of nitric oxide (NO) synthase. Our experiments were studied the effects of NMDA and selective inhibition and neuronal NO synthase with 7-nitroindazole (7-NI) on collateral and normal cerebral blood flow (rCBF) following MCA occlusion. In 9 dogs under halothane anesthesia, a left craniotomy was performed. An MCA branch was cannulated for determination of collateral dependent tissue (CDT). CBF was measured using radioactive microspheres and cerebral vascular pressures were measured with a glass micropipette.

NMDA (300 u M) increased flow to DCT by 54.6%, while normal ipsilateral and contralateral cerebrum increased by at least 35% from baseline. 7-NI caused a significant drop in rCBF, with the greatest drop of 33.8% occurring in CDT. Normal ipsilateral and contralateral rCBF were reduced by 32.3% and 20.2% respectively. The response to NMDA was significantly attenuated following 7-NI administration as rCBF actually decreased further from baseline. Vascular resistance decreased in response to NMDA; 7-NI caused a significant increase in cerebral vascular resistance.

In our study agonism at NMDA receptors following MCA occlusion increased blood flow significantly to CDT and normal cerebrum, probably due to the production of neuronal NO resulting in vasodilation. Blockade of neuronal NO synthase activity with 7-NI reduced rCBF to all regions of the brain from increase usually seen in response to NMDA administration. Neuronal NO production appears to play an important role in regulating vasculating vascular tone and CBF and may be especially important in maintaining flow to CDT following MCA occlusions.



Saturday, September 21 9:25-9:45 AM Repair of Carotid Endarterectomy with Collagen Dacron Graft Meyer FB, Windschitl WL 6

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Closure of the arteriotomy following carotid endarterectomy with a saphenous vein patch graft may decrease the risk of acute postoperative occlusion and recurrent stenosis. However, disadvantages of a vein patch include postoperative rupture (0.15%) and pseudoaneurysm formation. This report analyzes preliminary results using a Hemashield microvel knitted double velour graft. From 1/93 through 7/95, 190 consecutive carotid endarterectomies were repaired with the synthetic graft. The surgical management included intraoperative monitoring with selective shunting, infusion of heparin 5000 units prior to cross clamping, pre and postoperative aspirin 325 mg qd, and perioperative antibiotics. All patients had postoperative noninvasive assessment of vessel patency prior to discharge with either carotid ultrasound or oculoplethysmography. There were no wound hematomas, perioperative occlusions, or graft infections. There was 1 major (embolic) and 1 minor (hemodynamic) stroke occurring in Sundt Grades 3 and 4, respectively. There was 1 death from cardiac arrest which occurred 10 days after surgery at home following combined cardiac and carotid revascularization. There were 2 patients involving 3 vessels who developed recurrent stenosis. The 1 patient who developed bilateral recurrent stenosis 9 months after the second surgery appeared to have a dense fibrotic reaction to surgery as evidenced by hypertrophic scar formation of the skin and myointimal hyperplasia of the endarterectomy site. The second patient developed a focal proximal stenosis underneath the graft site 6 months after surgery. These preliminary results suggest that this synthetic graft may be a viable alternative for those surgeons who prefer to repair a carotid endarterectomy with a patch graft.

Saturday, September 21 11:00 AM

Drug Delivery by Intracerebral Clysis in a Rat Glioma Model Bruce J, Birch B, Johnson J, Yoon J, Lycette C, Falavigna A (

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To circumvent the limitations of a systemic chemotherapy for malignant brain tumors, a novel local drug delivery method known as intracerebral clysis has been developed which involves the use of high flow microinfusion directly into brain tumors by means of a controllable pump. To establish baseline parameters for preclinical, in vivo drug investigations of intracerebral clysis in a rat glioma model, we have studied the effects of varying infusion parameters on intracranial pressure, infusate distribution and clinical therapeutic response.

C6 rat glioma cells were stereotactically implanted into the frontal lobe of male Fischer rats. Between 10 to 25 days later, catheters were stereotactically implanted into the tumors and 100 ul of 25% albumin was infused at flow rates from 0.5 to 4 ul/min while monitoring ICP. Fluoresceinated dextran (MW 20,000 D) was then infused under similar conditions and the distribution volumes were calculated after brain sectioning and photography under UV light.

Flow rates less than 1 ul/min were tolerated well even after 25 days of tumor growth. The increase in ICP with increasing flow volumes was progressively greater as the flow rate was increased. Flow rates greater than 3 ul/min were associated with morbidity and mortality. Volumes of distribution at tolerated flow rates were sufficient to provide adequate drug delivery to tumors. Preliminary results demonstrating efficacy of several anti-tumor compounds delivered by intracerebral clysis will also be presented.

NOTES

Saturday, September 21
11:20-11:40 AM
Comprehensive Image Guided Surgery of Sellar Tumors
Schulder MA, Carmel P

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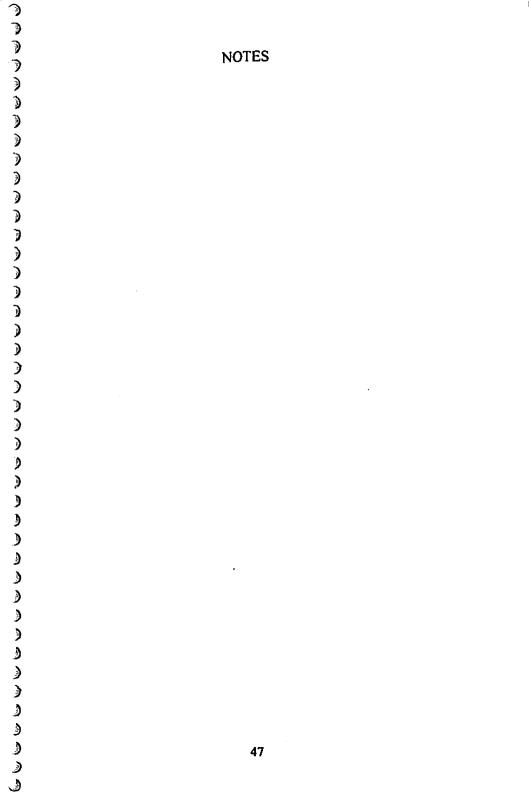
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More than 25 years ago, the advent of the operating microscope and fluoroscopic C-arm rehabilitated the transsphenoidal approach to the sella, making it the route of choice for most patients with pituitary tumors. However, significant obstacles still limit the radical removal of these tumors, including; 1) uncertainty of limits of cavernous sinus extension; 2) lack of lateral landmarks, especially in patients with larger, more invasive tumors; 3) "tunnel" vision of the operating microscope does not provide angles needed to visualize portions of tumor.

We have employed new technical advances to optimize surgical planning and removal of sellar tumors. Techniques utilized included: 1) differential hysteresis image processing, which defines cavernous involvement more clearly than standard MR scans; 2) Frameless stereotactic navigation, which allows precise evaluation of lateral resection extent; and 3) working channel endoscopy, which allows visualization and removal of tumor which lies above the planum, upper clivus, and laterally within and above the sella.

These techniques will be illustrated. They have the potential to considerably enhance the scope and safety of transsphenoidal operations. We believe they can be shown to be cost effective in treating patients with these tumors.



Saturday, September 21 11:40 AM Dorsal Cervical-Medullary Bulge in Chiari I Malformations Carmel PW, Bello JA **(** ,

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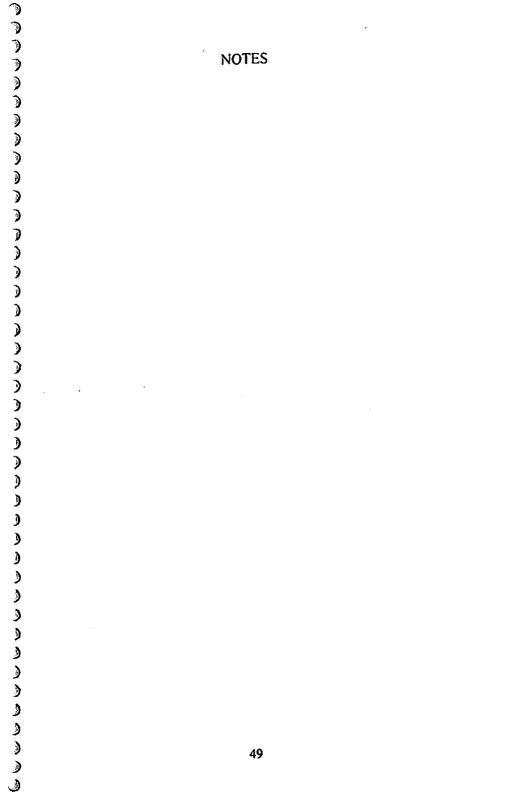
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A dorsal surface bulge underlying the tonsillar prolapse of Chiari I malformations has been reported by a number of authors. Surprisingly, there has been no systematic study of this associated deformity, correlation with clinical symptomatology, or explanation of its pathogenesis. This report details the pre- and post-operative MR findings, clinical and operative findings and incidence in a series of 137 patients with Chiari I malformations.

In adequately studied Chiari I patients, dorsal deformity was found in 19% (18/99). Patients with dorsal deformities had a higher incidence of lower cranial nerve problems, including a "medullary cough" syndrome. Bulges were infrequent in children and adolescents, and more frequent in adults.

Post-op MRI's showed progressive <u>resolution</u> of this deformity. These data indicate that the bulge is reversible. It is likely to be secondary to progressive tonsillar prolapse and is acquired, rather than congenital, in origin. It is not an anatomically "fixed" malformation.



Saturday, September 21 12:00 noon

Brain Tumor Invasion-Biological Rationale and Preclinical Studies

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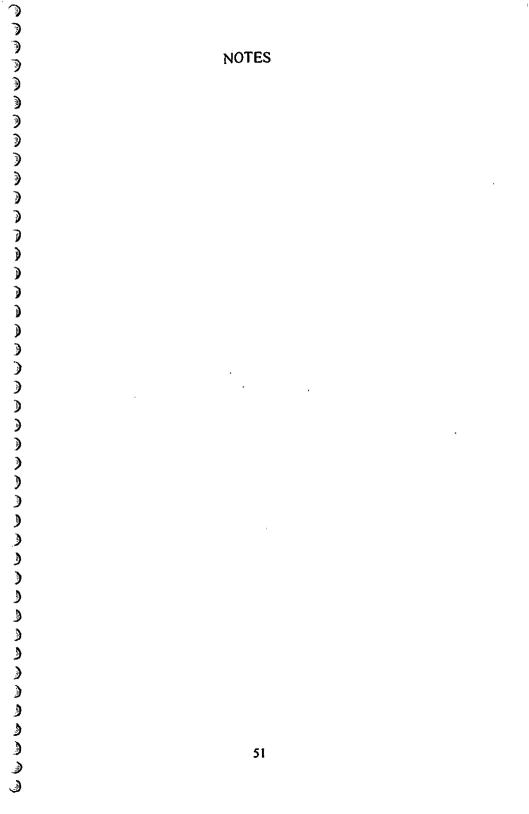
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Rosenblum ML, Mikkelsen T, Edvardsen K, Nelson K, Kohn E, Smith R, Spencer D, Rasnick D, Sloane B

The invasiveness of malignant glioma cells is a major cause of therapeutic failure both locally and at distant sites within the brain. We have evaluated the clinically relevant pathways of tumor spread and hypothesized that increased protease activity. cell receptor (integrin) interactions with specific extracellular matrix (ECM) proteins, and second messenger systems are the most logical targets against which to develop antiinvasive strategies. Cathepsin B (CB) is elevated in malignant gliomas, excreted into the extracellular space and prominent in invading cells (Cancer Res 54:6027-6031, 1994). We have utilized two types of specific, irreversible CB inhibitors to demonstrate a 10-70% inhibition of invasion for U251MGn, U87MG and HF66 malignant glioma cells using a Matrigel ECM invasion assay. A clinically relevant spheroid confrontation assay that demonstrates human brain tumor cell invasion into organotypic normal brain aggregates derived from fetal rats was used to show a marked inhibition of invasion using all the CB inhibitors. We have demonstrated the deposition of laminin by normal brain cells at the invading front of tumor cells in both the spheroid confrontation assay as well as an in vivo model for brain tumor invasion. A simultaneous increase in the expression of a laminin-associated integrin in tumor cells was also observed. Furthermore, antiintegrin antibodies were shown to decrease tumor cell invasion in the Matrigel ECM assay. Carboxy-amido-triazole (CAI) is an inhibitor of G-protein-mediated signal transduction and has been developed as an antimetastasis agent. We have shown that 2-20 uM CAI inhibits Matrigel invasion of glioma cell lines by an average of 60% without killing cells. CAI also inhibits tumor cell invasion in the spheroid confrontation model. We conclude that protease inhibitors and agents which block integrin-mediated adhesion and second messenger systems could be developed as novel antiinvasive strategies to treat malignant gliomas.



Saturday, September 21 12:20 PM Immunobiology of Primary Brain Tumors Brooks WH, Roszman TL, Elliott LE

Individuals harboring primary malignant tumors manifest a broad variety of immunological deficits. These anomalies are confined primarily to the T-helper cell subset and are linked to decreased production of IL-2 and failure to properly assemble the highaffinity receptor for IL-2 in the membrane of stimulated T lymphocytes. In this study, the early transmembrane signaling events on which normal T-cell function is contingent were examined. Concurrently, the mechanism(s) of how these and/or other biochemical and molecular abnormalities are induced in the T-cell was investigated. The results demonstrate that early transmembrane signaling via the T-cell receptor/CD3 complex of lymphocytes obtained from glioma bearing individuals is not normal: these cells are found to have an intrinsic defect which renders them unable to respond to appropriate proliferative stimuli. Concurrently, these investigations reveal that soluble factors secreted by malignant glial cells induce programmed cell death (apoptosis) in T cells. Collectively, the present study indicates that immune suppression observed in patients with malignant gliomas is contingent on and may be correlated with tumor size and the eliberation of glial T cell suppresser factors(s) (GSF). These findings further contribute to elucidating the immunobiological potential of these patients. The relevancy of this immunobiological model to immune modulation as a therapeutic option will be discussed.

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Tudor Arms Hotel, Cleveland, Ohio October 21-22, 1940
Mark Hopkins Hotel, San Francisco and Ambassador
Hotel, Los Angelas, 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 20-22, 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 21-23, 1954
The Homestead, Hot Springs, Virginia October 27-29, 1955
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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
Copley 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 & Towers,
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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, Mexico City November 18-21, 1970
Sahara-Tahoe Hotel, Stateline, Nevada September 26-30, 1971
New College, Oxford, England September 4-7, 1972
Huntington-Sheraton Hotel,
Pasadena, California November 14-17, 1973
Southampton Princess Hotel, Bermuda November 6-9, 1974

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Mills Hyatt House, Charleston,	
South Carolina	
Mauna Kea Beach Hotel, Kamuela, Hawaii November 2-5, 1977	
Hotel Bayerischer Hof, Munich, Germany October 22-25, 1978	
Hyatt Regency, Memphis, Tennessee November 7-10, 1979	
Waldorf Astoria, New York City October 1-4, 1980	
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Tucson, Arizona September 27-October 1, 1989	
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Salishan Lodge, Gleneden Beach, Oregon September 22-26, 1991	
Ritz-Carlton Hotel, Naples, Florida October 21-25, 1992	
The Wigwam, Phoenix, Arizona October 27-30, 1993	
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BERNARD PERTUISET Hospital de la Pitie 83 Bernard de l'Hopital 75651 Paris Cedex 13 FRANCE	1986
KEIJI SANO (Yaeko) Dept. of Neurosurgery Teikyo Univ. Hospital 2-11-1 Kaga, Itabashi-ku Itabasji-ku Tokyo 173 JAPAN	1975

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•	Wake Forest School of Medicine 300 S. Hawthorne	
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	DONALD BECKER (Maria)	1990
)	UCLA, Division of Neurosurgery	1770
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WILLIAM H. SWEET (Elizabeth) Massachusetts General Hospital Fruit Street Boston, MA 02114	1950
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	Dept. of Neurological Surgery	1900
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ACTIVE MEMBERS	Elected
MICHAEL APUZZO (Helene) 1200 N. State Street, Ste. 5046 Los Angeles, CA 90033	1988
JAMES AUSMAN (Carolyn) Univ. of Illinois-Chicago Dept. of Neuro/ M/C 799 912 S. Wood St. Chicago, IL 60612	1979
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•	WILLIS BROWN, JR. (Ann)	1984
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CHARLES H. TATOR (Carol) Toronto Western Hospital 399 Bathurst Street Toronto, ON M5T 2S8 CANADA	1991
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RUSSELL L. TRAVIS 1401 Herrodburg Rd., Suite 485B Lexington, KY 40504-3700	1994
JOHN VAN GILDER (Kerstin) Department of Neurosurgery University of Iowa School of Medicine Iowa City, IA 55242	1980
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•	Ford & Ferraro	
•	98 San Jacinto Blvd., Suite 2000	
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•	BRYCE K. WEIR (Mary Lou)	1984
•	Section of Neurosurgery, MC 3026	
•	University of Chicago	
)	5841 S. Maryland Ave.	
	Chicago, IL 60637	
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)	MADTIN II WEIGO (D. II)	1001
•	MARTIN H. WEISS (Debby)	1981
•	USC Medical Center, Box 786	
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•		
)	Dept. of Neurosurgery 325 Ninth ZA86	
)	Seattle, WA 98104	
)	Scattle, WA 70104	
)	FREMONT P. WIRTH (Penny)	1993
	4 Jackson Blvd.	1993
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)		
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	Epilepsy Center, Swedish Medical Center	1770
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)	Souther, Will Foret	
)		
)	A. BYRON YOUNG (Judy)	1989
)	University of Kentucky Medical Center	
	800 Rose Street, MN 268	
)	Division of Neurosurgery	
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		() ()
RONALD F. YOUNG (Christina)	1986	•
Northwest Hospital 1560 N. 115th St., #G5		()
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HAROLD F. YOUNG (Terry)	1994	()
Medical Col. of Virginia Station	.,,,	€
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`))	INACTIVE	Elected
)))	ROBERT BOURKE 5802 Nicholson Lane, St. 305 Rockville, MD 20852 (301) 881-4567	1983
))))	JOHN KAPP P.O. Box 448 Galax, VA 24333 (703) 236-2613	1985
)))	RICHARD S. KRAMER Duke University Medical Center Box 3255 Durham, NC 27710	1978
))))		
)))		
)		
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SENIOR CORRESPONDING	ELECTED
JEAN BRIHAYE (van Geertruyden) Belgium 98 avenue Des Franciscains Brussels, BELGIUM	1975
KARL-AUGUST BUSHE (Eva-Christa) Lerchenweg 8 D-97074 Wurzburg GERMANY	1972
FERNANDO CABIESES Peruano De Formento Educativo Av. Arenales 371, of. 501 Apartado 5254 Lima, PERU	1966
JUAN CARDENAS (Delores) Insurgentes Sur 594 Av. Insurgentes Mexico City, 40 MEXICO	1966
JUAN CHRISTENSEN (Diana Poli) José C. Paz 234 Acassusi (1641) Buenos Aires Province ARGENTINA	1970
HANS ERICH DIEMATH (Karin) Landesnervenklinik, Dept. of Neurosurgery 5020 Salzburg, Ignaz Harrer-StraBe 79 AUSTRIA	1970

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•		
•		
•	HERMANN DIETZ (Elfrun)	1970
•	Department of Neurosurgery Hannover School of Medicine	
•	30623 Hannover	
)	GERMANY	
)))		
•		
•	JOHN GILLINGHAM	1962
•	Royal Infirmary Lauriston Place	
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)	Scotland, United Kingdom	
)		
)		
•	JAIME G. GOMEZ (Lucy)	1975
•	5353 N. Federal Highway, #210 Fort Lauderdale, FL 33068	
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ý		
))))	JOHN HANKINSON (Nicole)	1973
Ś	Westacres	
•	Woolsington Hall	
3	Newcastle-Upon-Tyne, NE13 8DG ENGLAND	
•	ENGLAND	
•		
)	SHOZO ISHII (Akiko)	1975
	Juntendo University	
)	2-1 Hongo, Bunkyo-ku	
))	Tokyo 113, JAPAN	
)	HANS-PETER JENSEN	1980
)	Neurochirurgische	50
)	Universitatsklinik Kiel	
)	Welmarer Strasse 8	
)	Kiel D-2300	
)	GERMANY	
)		
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)		

RICHARD JOHNSON Dept. of Neurological Surgery Royal Infirmary Manchester, ENGLAND	1974
KATSUTOSHI KITAMURA (Yoshiko) 1-3-1 Kanada Kokurakita-Ku, Kitakyushu 803, JAPAN	1970
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B. RAMAMURTHI (Indira) Voluntary Health Services Adyar Madras-600 113 INDIA	1973
KURT-FRIEDRICH SCHURMANN Am Eselsweg 29 D-6500 Mainz I GERMANY	1978
CHARAS SUWANWELA Chulalongkorn Hospital Medical School Bangkog THAILAND	1972

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• • • • • • • • • • • • • • • • • • •	LINDSAY SYMON (Pauline) Gough-Cooper Dept. of Neurological Surgery Institute of Neurology, The National Hospital Queen Square London WCIN 3BG ENGLand	1982
))))	KJELD VAENET Department of Neurosurgery Rigshospitalet Copenhagen 2100 DENMARK	1970
))))	SIDNEY WATKINS The London Hospital Whitechapel London E I ENGLAND	1975
))))	M. GAZI YASARGIL (Dianne) Dept. of Neurosurgery University of Arkansas Little Rock, AR 72205	1975
))))		
))))		
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CORRESPONDING	Elected
LEIGH ATKINSON (Sally Anne) Alexandra, Suite, 2nd, Floor 201 Wickham Terrace, 4000 Brisbane 4000 AUSTRALIA	1989
LUC CALLIAUW (Dora) Dept. of Neurosurgery, University Hospital De Pintelaan Ghent, BELGIUM	1988
H. ALAN CROCKARD (Caroline) Dept. of Surgical Neurology, National Hosp. Queen Square London, WCIN 3BG, ENGLAND	1992
NOEL GEORGE DAN (Adrienne) Specialist Medical Center, Suite 302 235-285 New South Head Road Edgecliff, N.S.W. 2027 AUSTRALIA	1989
NICHOLAS DE TRIBOLET Service de Neurochirugie Centre Hospital Universite Vaudois Lausanne, CH1011 SUISSE	1995
JACQUES DEVILLIERS (Jeanne Marie Erica) Department of Neurosurgery University of Cape Town Observatory 7925 Cape 7 Republic of SOUTH AFRICA	1986
94	

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•	VINKO DOLENC	1988
•	Univ. of Ljubljana/Neuro.	1700
•	Clinical Ctr. Zaloska 7	
•	Ljubljana 61105	
•	YUGOSLAVIA	
•		
•	RUDOLPH FAHLBUSH (Hanna)	1991
•	Neurochirurgische Klinik	
•	University; Erlangen-Nurnberg	
)	Schwabachanlage 6	
)	91054 Erlangen GERMANY	
)	GERMAN	
)		
)	SALVADOR GONZALEZ-CORNEJO (Rosa)	1982
•	Av. Chapultepec Sur 130-204	
)	Guadalahara, 44140 MEXICO	
)	MEXICO	
•		
•		
)	ERNST GROTE (Juliana)	1984
)	Department of Neurosurgery University Kliniks Schnarrenberg	
)	Hoppe Seyler-Str. 3	
)	7400 Tubingen	
)	GERMANY	
)		
•	DAR HER HAN (Sung Soon Cho)	1991
)	DAE HEE HAN (Sung Soon Cho) SNU Hospital	1771
•	Seoul Nat'l, Univ., School of Medicine	
)	#28 Youngon-dong	•
)	Chongno-Gu, Seoul 110-744	
.) `	KOREA	
)		
)	HAMIME HANDA (Hiroko)	1985
)	Takeda General Hospital	
)	28-1 Moriminami-cho Ishida	
)	Fushimi-ku,	
)	Kyoto 601-13, JAPAN	
) }	95	
<i>3</i> }		

FABIAN ISAMAT (Maria V. {Marivi}) Clinica Sagrade Familia Neurogrup, Torras y Pujalt, I 08022 Barcelona, SPAIN	1989
HARUHIKO KIKUCHI Dept. of Neurosurgery Kyoto Univ., Medicine 5-1 Kawahara-cho Shogoin Skyo-ku 606 Kyoto, JAPAN	1993
RAUL MARINO, JR. (Angela) Rua Maestro Cardim, 808 Instituto Nueurologico de S. Paulo S. Paulo-SP 01323-100, BRAZIL	1977
KINTOMO TAKAKURA Dept. of Neurosurgery Neurological Institute Tokyo's Women's Medical College 8-1, Kawadacho, Shinjuku-ku Tokyo 162, JAPAN	1988
DAVID G.T. THOMAS (Hazel) Institute of Neurology Queens Square London WC1 3BG ENGLAND	1995

DECEASED MEMBERS

	Deceased	Elected
JAMES R. ATKINSON Phoenix, Arizona (Active)	1978	1970
PERCIVAL BAILEY Evanston, Illinois (Honorary)	1973	1960
GEORGE BAKER Litchfield Park, AZ (Senior)	1993	1940
H. THOMAS BALLANTINE, JR. Boston, Massachusetts (Senior)	1996	1951
WILLIAM F. BESWICK Buffalo, New York (Active)	1971	1959
EDWIN B. BOLDREY San Francisco, California (Senior)	1988	1941
SPENCER BRADEN Cleveland, Ohio (Active)	1969	Founder
F. KEITH BRADFORD Houston, Texas (Active)	1971	1938
HOWARD BROWN San Francisco, California (Senior)	1990	1939
97		

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GALE CLARK Oakland, California (Senior)	1996	1970	, , , ,
DONALD COBURN Wilbington, Delaware (Senior)	1988	1938	
WINCHELL McK. CRAIG Rochester, Minnesota (Honorary)	1960	1942	
EDWARD DAVIS Portland, Oregon (Senior)	1988	1949	
PEARDON DONAGHY Burlington, Vermont (Senior)	1991	1970	
FRANCIS ECHLIN New POaltz, New York (Senior)	1988	1944	
DEAN ECHOLS New Orleans, Louisiana (Senior)	1991	Founder	
GEORGE EHNI Houston, Texas (Senior)	1986	1964	
ARTHUR ELVIDGE Montreal, Quebec, Canada (Senior)	1985	1939	
THEODORE C. ERICKSON Madison, Wisconsin (Senior)	1986	1940	% % %
JOSEPH P. EVANS Kensington, Maryland (Senior)	1985	Founder	
98			

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)			
)	JOHN FRENCH	1989	1951
•	Los Angeles, California		
)	(Senior)		
•	IOUN CREEN	1990	1052
) }	JOHN GREEN Phoenix, Arizona	1970	1953
))	(Senior)		
) }			
•	JAMES GREENWOOD, JR.	1992	1952
•	Houston, Texas (Senior)		
)	(Selliot)		
)	WESLEY A. GUSTAFSON	1975	1942
)	Jensen Beach, Florida		
)	(Senior)		
)	HANNIBAL HAMLIN	1982	1949
)	Providence, Rhode Island	1702	1747
)	(Senior)		
)			
)	JOHN W. HANBERY	1996	1959
•	Palo Alto, CA (Senior)		
)	(Semon)		
)	JESS HERRMANN	1994	1938
•	Oklahoma City, OK		
)	(Senior)		
•	HENRY L. HEYL	1975	1951
•	Hanover, New Hampshire	1773	1,51
)	(Senior)		
)	0		1040
•	OLAN HYNDMAN Iowa City, Iowa	1966	1942
•	(Senior)		
•	(Semon)		
}	KENNETH G. JAMIESON	1976	1970
•	Brisbane, Australia		
•	(Corresponding)		
•	SIR GEOFFREY JEFFERSON	1961	1951
)	Manchester, England		-
•	(Honorary)		
)	99		
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			(), (),
WILLIAM S. KEITH Toronto, Canada (Senior)	1987	Founder	
HUGO KRAYENBUHL Zurich, Switzerland (Honorary)	1985	1974	
KRISTIAN KRISTIANSEN Oslo, Norway (Senior corresponding)	1993	1967	
WALPOLE S. LEWIN Cambridge, England (Corresponding)	1980	1973	
HERBERT LOURIE Syracuse. New York (Senior)	1987	1965	
WILLEM LUYENDIJK Oegstgeest, the Netherlands (Senior Corresponding)	1996	1973	
M. STEPHEN MAHALEY Birmingham, Alabama (Active)	1992	1972	
GEORGE L. MALTBY Scarsborough, Maine (Senior)	1988	1942	
FRANK MARGUTH Munich, Germany (Senior Corresponding)	1991	1978	•
DONALD D. MATSON Boston, Massachusetts (Active)	1969	1950	()
FRANK MAYFIELD Cincinnati, Ohio (Senior)	1991	Founder	
	100		

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•				
•	AUGUSTUS M. OD AVEV		1990	1011
•	AUGUSTUS McCRAVEY Chattanooga, Tennessee		1990	1944
•	(Senior)			
)	(20,000)			
)	KENNETH G. McKENZIE		1964	1960
•	Toronto, Ontario, Canada			
•	(Honorary)			
)	JAMES M. MEREDITH		1962	1946
•	Richmond, Virginia		.,,,	1710
•	(Active)			
)				
)	J. DOUGLAS MILLER		1995	1988
)	Edinburgh, Scotland (Corresponding)			
•	(Corresponding)			
•	W. JASON MIXTER		1968	1951
•	Woods Hole, Massachusetts			
)	(Honorary)			
•	EDMUND J. MORRISSEY		1986	1941
)	San Francisco, California		1900	1741
)	(Senior)			
•				
•	FRANCIS MURPHEY		1994	Founder
)	Naples, FL			
)	(Senior)			
•	GOSTA NORLEN		1985	1973
)	Goteborg, Sweden		1703	.,,,
)	(Honorary)			
•				
)	FRANK NULSEN		1994	1956
•	Naples, Florida (Senior)			
)	(Sellor)			
)	SIXTO A. OBRADOR		1978	1973
)	Madrid, Spain			
)	(Honorary)			
•	PIETRO PAOLETTI		1991	1989
)	Milan, Italy		1771	1707
•	(Corresponding)			
		101		
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HANS-WERNER PIA	1986	1978
Giessen, West Germany (Corresponding)		
WILDER PENFIELD Montreal, Canada (Honorary)	1976	1960
HELMUT PENZHOLZ Heidelberg, West Germany (Corresponding)	1985	1978
RUPERT R. RANEY Los Angeles, California (Active)	1959	1939
BRONSON RAY New York, New York (Honorary)	1993	1992
DAVID L. REEVES Santa Barbara, California (Active)	1970	1939
DAVID REYNOLDS Tampa, Florida (Active)	1978	1964
R.C.L. ROBERTSON Houston, Texas (Senior)	1985	1946
STEWART N. ROWE Pittsburgh, Pennsylvania (Senior)	1984	1938
RICHARD C. SCHNEIDER Ann Arbor, Michigan (Senior)	1986	1970
WILLIAM B. SCOVILLE Hartford, Connecticut (Senior)	1984	1944
102		

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•			
•	R. EUSTACE SEMMES	1982	1955
)	Memphis, Tennessee	1702	1733
•	(Honorary)		
•		1055	
)	SAMUEL R. SNODGRASS	1975	1939
)	Galveston, Texas (Senior)		
)	(Selliot)		
•	GLEN SPURLING	1968	1942
)	LaJolla, California		
)	(Honorary)		
)	C. WILLIAM STEWART	1948	1948
)	Montreal, Quebec, Canada	1770	.,.0
)	(Corresponding)		
)	THORALE CUMPT IN	1002	1071
)	THORALF SUNDT, JR. Rochester, Minnesota	1992	1971
•	(Active)		
<i>,</i>	(,		
)	KENICHIRO SUGITA	1994	1988
)	Nagoya, Japan		•
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•	HENDRIK SVIEN	1972	1957
•	Rochester, Minnesota		
)	(Active)		
•	HOMER S. SWANSON	1987	1949
)	Atlanta, Georgia	1907	1949
•	(Senior)		
)	,		
•	ALFRED UIHLEIN	1990	1950
)	Rochester, Minnesota (Senior)		
•	(Sellor)		
)	A. EARL WALKER	1995	1938
•	Albuquerque, New Mexico		
•	(Senior)		
)	THOMAS A. WEAVER, JR	. 1985	1943
•	Dayton, Ohio	. 1703	1743
)	(Senior)		
))	· · · · ·	103	
)			

W. KEASLEY WELCH Waban, Massachusetts (Senior)	1996	1957
BARNES WOODHALL Durham, North Carolina (Senior)	1985	1941
FRANK WRENN Greenville, South Carolina (Senior)	1990	1973

