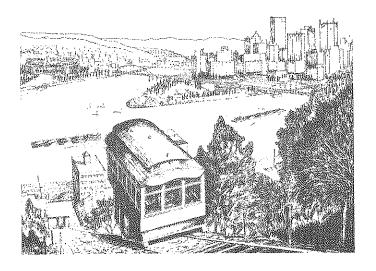
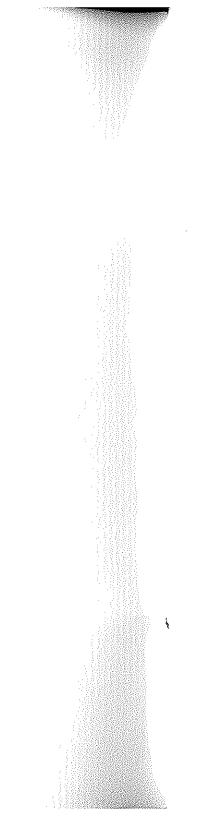
Section of Pediatric Neurological Surgeons of the American Association of Neurological Surgeons

15th ANNUAL MEETING



Hyatt Pittsburgh at Chatham Center Pittsburgh, Pennsylvania December 2-5, 1986





PROGRAM SUMMARY

Paolo Raimondi Lecturers

Pediatric Section Chairmen

Pediatric Annual Meeting Sites

Exhibitors

15th Annual Meeting Scientific Program

15th Annual Meeting Scientific Abstracts

Pediatric Section Member Listing

This program has been approved by the Joint Committee on Education of the American Association of Neurological Surgeons and Congress of Neurological Surgeons for a maximum of 13 hours of Category I credit toward the Continuing Education Award in Neurosurgery.

PAOLO RAIMONDI LECTURERS

E. Bruce Hendrick	1978
Paul C. Bucy	1979
Floyd Gilles	1980
(Panel Discussion)	1981
(Panel Discussion)	1982
Derek Harwood-Nash	1983
Anthony E. Gallo, Jr.	1984
William F. Meacham	1985

PEDIATRIC SECTION CHAIRMEN

Robert L. McLaurin	1972-73
M. Peter Sayers	1973-74
Frank Anderson	1974-75
Kenneth Shulman	1975-76
E. Bruce Hendrick	1976-77
Frank Nulsen	1977-78
Luis Schut	1978-79
Fred Epstein	1979-81
Joan L. Venes	1981-83
Harold J. Hoffman	1983-85
William R. Cheek	1985-87

PEDIATRIC ANNUAL MEETING SITES

Cincinnati	1972
Columbus	1973
Los Angeles	1974
Philadephia	1975
Toronto	1976
Cleveland	1977
Philadelphia	1978
New York	1979
New York	1980
Dallas	1981
San Francisco	1982
Toronto	1983
Salt Lake City	1984
Houston	1985
Pittsburgh	1986

The Pediatric Section of Neurological Surgery of the American Association of Neurological Surgeons gratefully recognizes the support of the following exhibitors for the 1986 Pediatric Annual Meeting:*

American V. Mueller - Niles, Illinois
Codman & Shurtleff, Inc. - Randolph, Massachusetts
The Greenery Rehabilitation Hospital - Boston, Massachusetts
Holter-Hausner International - Bridgeport, Pennsylvania
Philadelphia Medical Specialties - Voorhees, New Jersey
P.S. Medical - Goleta, California
U.S. Army Medical Center - Frederick, Maryland

All registrants are encouraged to visit the exhibit area frequently during the meeting.

^{*}A complete listing of all exhibitors will be distributed to all participants at the meeting.

PROGRAM

PEDIATRIC SECTION

AMERICAN ASSOCIATION OF NEUROLOGICAL SURGEONS

Hyatt Pittsburgh

Pittsburgh, Pennsylvania

December 2-5, 1986

TUESDAY, DECEMBER 2, 1986

6:00 - 8:00 p.m. Registration - Foyer (2nd Floor)

7:00 - 9:00 p.m. Reception - Foyer (2nd Floor)

WEDNESDAY, DECEMBER 3, 1986

7:00 a.m. Registration - Foyer (2nd Floor) 8:00 a.m. Meeting - Ball Room (2nd Floor)

> Welcome - William R. Cheek - Chairman AANS Pediatric Section

Opening Remarks - Robert L. McLaurin First Chairman AANS Pediatric Section

8:15 a.m. 7th Annual Special Lecture

"Reflections on 45 years of Pediatric

Neurological Surgery"

William Meacham

CHIARI MALFORMATION - Moderators: William R. Cheek, Steven L. Wald

9:00 a.m. 1. "Genesis of Brainstem and Cerebellar

Anomalies in the Chiari Type II Malformation" Peter W. Carmel

New York, New York

9:15 a.m. 2. "The Sulcal Pattern of the Brain in Type II

Chiari Malformation"

W. Jerry Oakes, R. E. McLendon

Durham, North Carolina

9:30 a.m. 3. "Arnold-Chiari Malformation: Packed Posterior

Fossa Syndrome"

Edward J. Kosnik, Martin P. Sayers

Columbus, Ohio

9:45 a.m. 4. "The Management of the Chiari Malformation and Management of the Concomitant Syringomyelia" Robert Jones, Vimala Navanar Randwick, N.S.W. Australia 10:00 a.m. 5. "Pediatric Arnold Chiari Malformation Without Myelodysplasia" Gregg N. Dyste, Arnold H. Menezes Iowa City, Iowa 10:15 a.m. COFFEE BREAK - Meeting Room (2nd Floor) MYELOMENINGOCELE AND TETHERED SPINAL CORD- Moderators: Jack E. McCallum, Patricia A. Aronin 10:45 a.m. 6. "Improved Pregnancy Outcome for Meningomyelocele Following Cesarean Section Delivery" David B. Shurtleff, J. Timothy Stuntz, Mitchell Berger, John Loeser, Robert Kropp Seattle, Washington 11:00 a.m. 7. "Glucose Metabolism and Oxidative Metabolism in the Experimental Tethered Cord" Thomas Purtzer, Terry Andrade, Shokei Yamada, John Patrickson, Takaharu Fuse Loma Linda, California 11:15 a.m. 8. "Tethered Cord Syndrome in Children: Clinical and Oxidative Metabolic Correlation" Shokei Yamada, David Knierim, Thomas Purtzer Loma Linda, California 11:30 a.m. 9. "The Role of MRI in Tethered Cords" Walter A. Hall, James Brunberg, A. Leland Albright Pittsburgh, Pennsylvania 10. "Spinal Cord Surgery in Fetal Rats" 11:45 a.m. A. Leland Albright Pittsburgh, Pennsylvania 12:00 noon LUNCH - (2nd Floor)

HIDROCKIIIAL	Moderators, Samuer S. Rasorr, Scephen S. Marnes
1:00 p.m.	11. "Changing Water Content and Ultrastructural Morphology Before and After Shunting in Hydrocephalus" Kenneth Shapiro, Futoshi Takei, Asao Hirano, Ira Kohn, Arno Fried Bronx, New York
i:15 p.m.	12. "Brain Specific Gravity Changes in Experimental Hydrocephalus" Marc R. Del Bigio, J. Edward Bruni Winnipeg, Manitoba, Canada
1:30 p.m.	13. "Distribution of Hematoporphyrin Derivative (Photofrin II) Following Intraventricular Injection in Rabbit" Shigeyo Hyman, J. Gordon McComb, Michael Apuzzo Los Angeles, California
1:45 p.m.	14. "The Effect of Acute Kaolin Hydrocephalus on Lymphatic Drainage Pathways in the Rabbit" J. Gordon McComb, Shigeyo Hyman, Floyd H. Gilles, Martin H. Weiss Los Angeles, California
2:00 p.m.	15. "The Foramen of Monro as a Control Element in Ventricular Volume Regulation" Jason A. Brodkey, Harold L. Rekate, Howard J. Chizeck, W. A. Elsakka Cleveland Heights, Ohio
2:15 p.m.	COFFEE BREAK - Meeting Room (2nd Floor) View Exhibits
Moderators:	Alexa I. Canady, Tadanori Tomita
2:45 p.m.	16. "The Neuropathology of Shunts" Randell G. Powell, Sydney S. Schochet, Jr., Vincent C. Traynelis Morgantown, West Virginia
3:00 p.m.	17. "The Slit Ventricle: A Complication or Goal of Shunting" Arno Fried, Marion L. Walker

HYDROCEPHALUS- Moderators: Samuel S. Kasoff, Stephen J. Haines

Salt Lake City, Utah

- 3:15 p.m.

 18. "The Treatment of Hydrocephalus in Preterm Infants with Intraventricular Hemorrhage"

 John D. Reeves, Edward C. Benzel,

 Theresa A. Hadden

 Shreveport, Louisiana
- 3:30 p.m.

 19. "External Ventricular Drainage (EVD) for Initial Treatment of Neonatal Posthemorrhagic Hydrocephalus (PHH): Surgical and Neurodevelopmental Outcome"

 Torunn T. Rhodes, William H. Edwards, Robert E. Harbaugh, Richard S. Saunders, Michael Scott Hanover, New Hampshire
- 4:00 p.m. Annual Business Meeting Members Only

THURSDAY, DECEMBER 4, 1986

7:00 a.m. Registration - Foyer (2nd Floor)

VASCULAR AND INTRACRANIAL PRESSURE - Moderators: Barry N. French,
Bruce C. Bressler

- 8:00 a.m. 20. "Giant Intracranial Aneurysms in Children and Adolescents"
 Shigeru Nemoto, Sydney J. Peerless,
 Charles G. Drake
 London, Ontario, Canada
- 8:15 a.m. 21. "Pediatric Cerebral Aneurysms"
 Siggi A. Stephensen, Edward J. Kosnik
 Columbus, Ohio
- 8:45 a.m.

 23. "Subcortical Cavernous Angioma: Intraoperative Ultrasound as an Adjunct to Localization and Resection"

 Martin J. Buckingham, Kerry R. Crone, Thomas S. Berger, Bokyung K. Han Cincinnati, Ohio

- 9:00 a.m.

 24. "Increases in Cerebral Interstitial Fluid and Cerebrospinal Fluid Adenosine Concentration During Hypoxia in Neonatal Piglet"

 T. S. Park, David G. L. Van Wylen, Rafael Rubio, Robert M. Berne Charlottesville, Virginia
- 9:15 a.m. 25. "In Vivo NMR Studies of Increased Intracranial Pressure in the Cat"

 Leslie N. Sutton, A. McLaughlin, W. Kemp,
 B. Cho, B. Chance, T. W. Langfitt, M. Schnall Philadelphia, Pennsylvania
- 9:30 a.m. 26. "CSF Pulsatility: Respiratory Effect on Wave Slope and as an Index of Intracranial Pressure" Eldon L. Foltz, Jeffrey Blanks Orange, California
- 9:45 a.m. 27. "Influence of ICP Monitoring on Outcome after Near Drowning"

 Jack Stern, David Reynolds, Samuel S. Kasoff Vaihalla, New York
- 10:00 a.m. 28. "Volumetric Analysis, Radiographic Findings, and ICP Changes in Craniosynostosis"

 E. Larry McCleary, Lee A. Harvey, Robert Hendee
 Denver, Colorado
- 10:15 a.m. COFFEE BREAK Meeting Room (2nd Floor)
 View Exhibits

MISCELLANEOUS PROBLEMS - Moderators: Arthur E. Marlin, Leslie N. Sutton

- 10:45 a.m.

 29. "Some Comments on Skull Growth after Craniofacial Repair"

 Arthur B. Eisenbrey, John Spolyar, William Vasileff, Alexa I. Canady Detroit, Michigan
- 11:00 a.m.

 30. "The Spectrum of Cerebellar Dysgenesis and Posterior Fossa Cysts Demonstrated by MRI"

 R. Michael Scott, Samuel M. Wolpert,

 Mary L. Anderson, Eddie S. K. Kwan,

 Val M. Runge

 Boston, Massachusetts

11:15 a.m.	31.	"Administration of Indomethacin for the Prevention of Intraventricular Hemorrhage in High-Risk Newborns" William C. Hanigan, Gail Kennedy, Frank Roemisch, Robert Anderson, Tom Cusack, Tim Miller Peoria, Illinois
11:30 a.m.	32.	"Optic Nerve Decompression for Osteopetrosis in Early Childhood" Stephen J. Haines, Donald L. Erickson Minneapolis, Minnesota
11:45 а.ш.	33.	"Surgical Treatment of Pediatric Intracranial Arachnoid Cysts" J. B. Delashaw, William C. Broaddus, T. S. Park Charlottsville, Virginia
12:00 noon	34.	"Selection of Spastic Cerebral Palsy Patients for Selective Posterior Rhizotomy" Warwick J. Peacock Los Angeles, California
		FREE AFTERNOON
6:30 p.m.		Reception
7:30 p.m.		Banquet
9:00 р.ш.		Entertainment
FRIDAY, DECEMBE	R 5, 19	36
SPINE/INFECTION	- Mo	derators: Marion L. Walker, Michael Scott
8:00 а.т.	35.	"The Significance of Lateral Odontoid Displacement in the Diagnosis of Atlantoaxial Subluxation" David M. Klein, Jerald P. Kuhn Buffalo, New York
8:15 a.m.	36.	"Evaluating Craniovertebral Junction Anomalies with Flexion-Extension MRI" Conrad Pappas, Harold Rekate Phoenix, Arizona
8:30 a.m.	37,	"Atlas Assimilation: Implications and Management" Arnold H. Menezes Iowa City, Iowa 10

8:45 a.m.	38.	"Sinusitis and Intracranial Infection in Children" Dennis L. Johnson, David C. McCullough Washington, DC
9:00 a.m.	39.	"Rasmussen's Encephalitis - Surgical Managemen Revisited" Benjamin S. Carson, Donlin M. Long Baltimore, Maryland
9:15 a.m.	40.	"CSF Formation in Acute Ventriculitis" Robert E. Breeze, J. Gordon McComb, Shigeyo Hyman, Floyd H. Gilles, Martin H. Weiss Los Angeles, California
9:30 a.m.	41.	"CT Evaluation and Management of H Flu Subdural Effusions" Fraser C. Henderson, Steven K. Gudeman, Phanor L. Perot, Jr. Charleston, South Carolina
9:45 a.m.		COFFEE BREAK - Meeting Room (2nd Floor) View Exhibits
TRAUMA- Moderat	ors:	Fred Epstein, Arnold H. Menezes
10:15 a.m.	42.	"Pediatric Spinal Trauma" Mark N. Hadley, Joseph M. Zabramski, Volker K. H. Sonntag, Harold Rekate Phoenix, Arizona
10:30 a.m.	43.	"Outcome of Pediatric Depressed Skull Fractures" Ann M. Flannery, Yoon S. Hahn, Martha J. Barthel, David G. McLone Chicago, Illinois
10:45 a.m.	44.	"Outcome of Pediatric Head Injuries in Children Under 36 Months of Age" Yoon Sun Hahn, David G. McLone, Chee Hong Chyung, Ann M. Flannery, Martha Barthel Chicago, Illinois
11:00 a.m.	45.	"Head Trauma in Infants" Maurice Choux, Lorenzo Genitori, Alberto Yanez Gabriel Lena Marseille, France
		3.3

46. "Profile of Pediatric Head Injury: 11:15 a.m. A Prospective Study of 1906 Patients Less than Fifteen Years of Age" Thomas G. Luerssen, Melville R. Klauber, Lawrence F. Marshall, Howard M. Eisenberg, Michael E. Miner San Diego, California TUMOR - CRANIOPHARYNGIOMAS - Moderators: Harold Hoffman, Shokei Yamada 11:30 a.m. 47. "CT and MRI Features of Craniopharyngiomas in Children and Adults" Robert B. Snow, Robert D. Zimmerman, Richard D. Becker, Steven Albert New York, New York 11:45 a.m. 48. "Neurobehavioral Syndromes Following Translamina Terminalis Resection of Large Craniopharyngiomas in Children" Dachling Pang, Christopher Ryan, Ellen O. Hesky, Paul Polinko Pittsburgh, Pennsylvania 12:00 noon 49. "Glioma Arising in the Field of Radiation Therapy for Craniopharyngioma" John Shillito, Jr. Boston, Massachusetts 12:15 p.m. LUNCH - (2nd Floor) TUMORS - Moderators: Richard A. Coulan, Jr., Edward J. Kosnik 50. "Pituitary Tumors in Children: Endocrinologic. 1:15 p.m. Radiologic, and Pathologic Findings Related to Pituitary Stalk Size" Kerry R. Crone, Thomas S. Berger, John M. Tew, Jr. Cincinnati, Ohio 1:30 p.m. 51. "Choroid Plexus Papillomas in Neonates, Infants and Children" Tadanori Tomita, David G. McLone Chicago, Illinois 1:45 p.m. 52. "Pediatric Diencephalic Gliomas - A Review of 1'8 Cases" Eric W. Scott, J. Parker Mickle

2:00 p.m.	53.	"Factors Affecting Survival in Malignant Glioma of Children" Ossama Al-Mefty, Naef R. Al-Rodhan, John L. Fox, Andrew D. Parent Jackson, Mississippi
2:15 p.m.	54.	"Head 'Lumps' of Infants and Children" John R. Ruge, Tadanori Tomita, Thomas P. Naidich, Yoon S. Hahn, David G. McLone Chicago, Illinois
2:30 p.m.	55.	"Radiation-Induced Meningiomas Presenting in the Pediatric Patient" S. David Moss, Gaylan Rockswold, Douglas Yock, Edward L. Seljeskog
2:45 p.m.		COFFEE BREAK - Meeting Room (2nd Floor) View Exhibits
TUMORS - Modera	tors	: David M. Klein, Marion L. Walker
3:15 p.m.	56.	"Sensitivity of PNET Derived Cell Lines to in vitro Cytolysis by Lymphokine-Activated Killer Cells" Richard E. George, Richard P. Moser Houston, Texas
3:30 p.m.	57.	"Bromodeoxyuridine as a Photosensitizer and a Radiosensitizer" Corey Raffel, Michael S. B. Edwards, Dennis Deen San Francisco, California
3:45 p.m.	58.	"Cerebellar Astrocytoma in Childhood" Masaharu Yasue, Tadanori Tomita, David G. McLone Chicago, Illinois
4:00 p.m.	59.	"Cerebellar Astrocytomas: The Significance of the Contrast Enhancing Cyst Wall" Mitchel S. Berger, Harold Hoffman, Robin Humphreys, E. Bruce Hendrick Seattle, Washington
4;15 p.m.	60.	"Post-Operative Myelography for the Evaluation of Malignant Pediatric Posterior Fossa Tumors" Ann M. Flannery, Tadanori Tomita, Mary Ann Radkowski, David G. McLone Chicago, Illinois

Gainesville, Florida

4:30 p.m. 61. "Medulloblastoma in Children: Factors
Determining Survival"
John M. McGregor, Edward J. Kosnik
Columbus, Ohio

SCIENTIFIC ABSTRACTS

1. GENESIS OF BRAINSTEM AND CEREBELLAR ANOMALIES IN THE CHIARI TYPE II MALFORMATION

Peter W. Carmel (New York, NY)

Sagittal MRI and operative microscopy have allowed new analysis of five recent Type II Chiari malformations, including a cystic malformation. The brainstem was elongated, in each case projecting rostrally into the posterior IIIrd ventricle and caudally into the upper cervical canal. Changes were most notable in the pontine region, which was straightened, with its anterior portion markedly flattened. Cerebellar deformity was characterized by vermian prolongation caudal to the tonsils, which were typically hypoplastic. The choroid plexus was the most caudally displaced cerebellar structure in three cases, and arrangement found early in cerebellar embryological development.

In the cystic case, typical Chiari II brainstem anomalies were found, while cerebellar structures were in fairly normal position. Pathological examinations of the cyst membrane was consistent with posterior medullary velum.

We believe that the primary cause of the Chiari II malformation is failure of the pontine flexure to form during the 4th embryonic week (as proposed by Daniel and Stritch). Our new findings suggest that the posterior medullary velum is therefore carried caudally with the hindbrain. Cerebellar development then progresses caudally along this membrane. Because the choroid and vermis are prevented from invaginating into the ventricle, they maintain their early embryological relationship.

The cerebellar anomalies are therefore a secondary phenomenon occurring at a later embryological period than the primary brainstem dysgenesis. Our new findings make it unlikely that the genesis of the malformation is from either hydrocephalus (Gardner) or from neuroschisis (Padget).

- 2. THE SULCAL PATTERN OF THE BRAIN IN TYPE II CHIARI MALFORMATIONS
- W. Jerry Oakes, R.E. McLendon (Durham, NC)

The brains of fifteen patients with the Type II Chiari malformation were examined at post mortem for the character of their gyral pattern. These patterns were compared to ten normal pediatric control brains. The quantification of the sulcal pattern was performed by a computer assisted digitizer program and grouped into primary, secondary, and tertiary sulci. The density of the sulcal pattern per unit area was calculated. A subset of six brains with the Chiari malformations were identified with a statistically significant more complex gyral pattern (polygyria).

The study documents the abnormal gyral pattern in Type II Chiari malformation patients and demonstrates that polygyria associated with myelodysplasia is compatible with normal intellectual function.

 ARNOLD-CHIARI MALFORMATION: PACKED POSTERIOR FOSSA SYNDROME

Edward J. Kosnik, Martin P. Sayers (Columbus, OH)

Over the past 30 years we have treated over nine hundred children with the diagnosis of myelomeningocele. Somewhere in the range of 75-100% of these children will be found anatomically to have the Arnold-Chiari Malformation (generally type II).

We have had occasion to treat many children with symptoms we feel directly referable to a "packed posterior fossa." It has been the policy of this clinic not to routinely open the dura during the course of decompression.

Our experience in 80 operative cases with regard to the symptoms, evaluation, treatment and long-term results will be reviewed and discussed.

Emphasis will be placed on the pre CT scan and MRI evaluation and long-term follow-up of these patients. Changing perspective of operative care guided by newer diagnostic imaging will be reviewed.

4. THE MANAGEMENT OF THE CHIARI MALFORMATION AND MANAGEMENT OF THE CONCOMITANT SYRINGOMYELIA

Robert Jones, Vimala Nayanar, Graham Wise (Randwick N.S.W. Australia)

A personal series of eight children (six with myelomeningocoele) with progressive syringomyelia in the presence of normal intracranial pressure have been treated by craniocervical exploration and various intradural procedures.

	Age in years	Successful	Unsuccessful
Dural graft only	4		1
Freeing of Tonsils	5,8		2
Plug in obex unsutured	3,5	1	1
Plug in obex sutured	6.9.14	3	-

A recent example is a previously normal 14 year old male with scoliosis recognized for six months and minimal neurological signs.

He was shown to have normal head CT, a syrinx and tonsils to C1. On MRI the apparent distance between 4th ventricle and the syrinx was 5cms.

At operation, the obex of the 4th was at Cl. An attempt to demonstrate the communication using lacrymal probes was unsuccessful. Muscle was sewn in place over the obex. This caused transient bradycardia. Postoperatively there was worsening of his long tract signs for a few days and a permanent diminution in size of the syrinx on uncontrasted CT scan.

Despite the general excellence of modern imaging, the caudal end of the 4th ventricle may not be shown and the proximity of the obex to the syrinx not appreciated.

Plugging of the central canal at the obex appears to be a safe and effective procedure especially if the muscle is sutured in place.

It is also applicable to patients who today are diagnosed at an earlier stage by MRI.

5. PEDIATRIC ARNOLD CHIARI MALFORMATION WITHOUT MYELODYSPLASIA

Gregg N. Dyste, Arnold H. Menezes (Iowa City, IA)

The Arnold Chiari Malformation (ACM) withouth myelodysplasia was infrequently encountered in the pediatric age group. However. with current neurodiagnostic modalities, it is being seen more frequently. Unfortunately, the prognosis is not clear because publications have included a number of different entities, used a variety of surgical approaches, and lacked long term follow-up.

In the past ten years, 16 patients under the age of 20 have been treated for ACM without myelodysplasia. Eleven had Chiari I, 3 Chiari II and 2 Chiari III. Average age was 11 years and average symptom duration was 20 months. The common symptoms were: motor weakness (56%), pain (37.5%) and sensory loss (25%). Signs consisted of motor deficit (81%), sensory loss (50%), scoliosis (50%) and cranial nerve palsies (50%).

Surgical procedures consisted of foramen magnum decompression (3 transoral clivus odontoid resections and 15 posterior fossa decompressions with dural grafting), alteration of CSF pathways at the cervicomedullary junction (plugging foramen caecum and 4th ventricle to subarachnoid shunt with posterior fossa decompressions), and ventriculoperitoneal shunting (2). In follow-up 37.5% are asymptomatic, 50% improved and 12.5% stable after an average follow-up of 43 months. The asymptomatic group was younger (9.3 years) and had a shorter symptom duration (7.2 months) than both the improved (11.9 years, 16.4 months) and stable groups (15 years, 20 months).

As we have shown, correction of the compressive pathology and the abnormal CSF pathways leads to excellent results in a problem which had been previously described as having a dismal prognosis.

6. IMPROVED PREGNANCY OUTCOME FOR MENINGOMYELOCELE FOLLOWING CESAREAN SECTION DELIVERY

David B. Shurtleff, J. Timothy Stuntz, Mitchell Berger, John Loeser, Robert Kropp (Seattle, WA)

Twenty-four cases of meningomyelocele with or without hydrocephalus were diagnosed prenatally and delivered by cesarean section. Four were diagnosed before 25 weeks gestational age, and the parents were informed there was no information about the natural history in utero and little data on the course of hydrocephalus. An additional six were similarly informed, and four others with multiple anomalies and a poor prognosis chose voluntary termination. Three of eight diagnosed after 24 weeks as having thoracic level lesions (no leg movement) and far advanced hydrocephalus were delivered by cesarean section rather than the recommended cephalocentesis. Three of these other five were stillborn prematurely, and the other two died shortly after birth. Twenty-one (four diagnosed before and 17 after 24 weeks gestational age) were told cesarean section may prevent loss of function in the legs by eliminating the trauma of labor. Five (including the three with no leg movement) (21%) are currently thoracic or high level lesions, four (19%) are mid or low lumbar. nine (38%) are sacral level, and six (25%) have no evident loss of function. The 15 with sacral levels of paralysis or less (63%) compre favorably with the 13% of 83 patients born after labor during the same time period (Fisher's exact, P = 0.000006). The lower proportion of more severely paralyzed patients in the group born by cesarean section may be due to selection of the least paralyzed, elimination of trauma from labor, or immediate postnatal treatment.

7. GLUCOSE METABOLISM AND OXIDATIVE METABOLISM IN THE EXPERIMENTAL TETHERED CORD

Thomas Purtzer, Terry Andrade, Shokei Yamada, John Patrickson, Takaharu Fuse (Loma Linda, CA)

Tethered cord syndrome is manifested by motor and sensory deficits in the legs, and incontinence. Oxidative metabolic derangement is a known underlying mechanism. Utilizing Sokoloff's 2-(Cl4) Deoxyglucose (2-DG) autoradiographic method, the authors studied glucose metabolism of the cat spinal cord with and without experimental tethering, parallel to oxidative metabolic study.

Cats were anesthetized and laminectomy was done for exposure of the lumbosacral cord. A ligature was placed around the filum terminale and was connected to either no traction (control), 3 g or 5 g traction. Forty-five minutes after IV administration of 2 DG (125 mCi/Kg), the animal was sacrificed. Perfusion of the aorta with glutaraldehyde and formaldehyde allowed fixation of the lumbosacral cord. Autoradiography of the L7 and S2 cord segments was done.

2 DG uptake was significantly less at the L7 and S2 cord segments with 5 g traction. Significant decrease in 2 DG uptake was noted at L7 and S2 cord segments which was subjected to 5 g traction; greater in the S2 than L7 segments. 2 DG uptake in the cords subjected to 3 g was equal to the control.

These findings were well correlated with impairment of oxidative metabolism, i.e., severely impaired in the S2 segment and mildly impaired in the L7 under 5 g traction; and only mildly impaired in S2; and not impaired in L7 under 3 g traction.

The mechanism involved in oxidative metabolic changes in mitochondria respiration in the tethered cord is explained partly by the impairment of glucose metabolism as in the ischemic brain or spinal cord.

8. TETHERED CORD SYNDROME IN CHILDREN: CLINICAL AND OXIDATIVE METABOLIC CORRELATION

Shokei Yamada, David Knierim, Thomas Purtzer (Loma Linda, CA)

Of 35 pediatric patients with tethered cord syndrome 28 underwent intraoperative spectrophotometric reduction/oxidation ratio (redox) measurements of cytochrome a,a3 before and after untethering procedures. Double beams of light (605 nm for sampling and 590 nm for reference) were projected to the lumbosacral cord. The reflected beams collected into the photomultiplier and redox changes were recorded. The mitochondrial cytochrome reactivities to low F102 between the pre and post tethering stages were compared. This permitted a clear division of the metabolic status of the lumbosacral cord into three categories.

- Type 1. mild derangement of redox activity which changed to normal status after untethering;
- Type 2. moderate to severe derangement which changed to normal status:
- Type 3. severe derangement which showed only partial restoration of normal state.

Type 1 patients regained normal neurological functions; Type 2 showed dramatic neurological improvement but with some minor deficits; while Type 3 had limited neurological improvement.

The oxidative metabolic results reflected a parallel to the eventual neurological improvement which usually occurred during the two week to three month period after the untethering procedures. It is therefore logical to assume the validity between the neurological improvements and the correction of the metabolic dysfunction of the gray matter.

9. THE ROLE OF MRI IN TETHERED CORDS

Walter A. Hall, James Brunberg, A. Leland Albright (Pittsburgh, PA)

Magnetic Resonance Imaging (MRI) was used to evaluate 15 children with spine bifida suspected of having tethered cords. Ten had myelomeningoceles closed at birth and five had spine bifida occulta. Tethering was suspected most frequently because of back pain (4), leg pain (4), and urinary incontinence (4). Signs of tethering included lumbar masses (5), cutaneous hemangiomas (3), and foot inversion (3). MRI scans were obtained preoperatively in 13/15 patients in sagittal and axial projections, using a GE 1.5 Tesla unit. Scans demonstrated tethering in all 13 patients, intraspinal fat in 10, and distal syrinxes in 4. In every case, all radiographic findings were confirmed at operation.

Routine postoperative MRI scans were performed within one year in 11/15 patients. Those scans showed that the position of the untethered cord remained at the preoperative level in 9 patients and ascended in only two. Unexpected retethering was found in three patients at the site of the cadaver dural graft. Ultrasound documented that the retethered cord was non-pulsatile and the patients were reoperated.

We conclude that: 1) MRI imaging is the diagnostic procedure of choice for tethered cord because it accurately identifies the pathology in nearly all patients without the use of myelography; 2) the untethered cord ascends less frequently than expected; and 3) MRI scans obtained after release of tethered cords show an unexpectedly high frequency of retethering to cadaver dura.

10. SPINAL CORD SURGERY IN FETAL RATS

Leland Albright (Pittsburgh, PA)

Operations for myelomeningocele do not restore neurologic function. If function is ever to be restored, the nonfunctional plaque will probably have to be replaced with functional, i.e., transplanted, tissue. Such transplants would have a better likelihood of functioning if done in utero.

We developed techniques for spinal cord injury and transplantation in fetal rats, where term gestation is 22 days. Fetal rats were operated on at 18-19 or 20-21 days. Lumbosacral spinal cords were exposed and were either a) transected-to evaluate cord reconnection, b) transected and the lumbosacral cord removed-to evaluate distal cord regeneration, c) transected, the distal cord removed briefly, then replaced (auto-transplant), or d) transected, and the distal cord transplanted into another fetus (iso-transplant). Surviving animals were videotaped at 4-6 weeks to document gait, were then killed, and the spinal cords removed for histologic examination.

Fetal mortality was related to the timing of operation (higher in 18-19 day fetuses than in 20-21 day) and to the procedure, lowest in cord cuts and highest in auto transplants. Walking was characterized as normal, paraparetic or paraplegic. Results are as follows:

	Normal	Paraparetic	Paraplegic
Cord transected	5	2	5
Cord removed	7	3	1
Autotransplant	2	2	6
Isotransplant	3	0	3

Normal gait in animals undergoing cord procedures was unexpected, and apparently due mainly to the level of transection. Although the lumbosacral cord anlage was cut in utero, the cord at that low level often developed into cauda equina, giving in essence a peripheral nerve lesion. Cord reconnection apparently occurred occasionally; photographs will be shown. Regeneration from the distal cord stump was never observed.

These techniques can be utilized for transplantation in higher species.

11. CHANGING WATER CONTENT AND ULTRASTRUCTURAL MORPHOLOGY BEFORE AND AFTER SHUNTING IN HYDROCEPHALUS

Kenneth Shapiro, Futoshi Takei, Asao Hirano, Ira Kohn, Arno Fried (Bronx, NY)

In order to correlate ultrastructure and water content of white matter in progressive hydrocephalus, cisternal kaolin was administered in two groups of cats. Group I had only the calvarium removed and Group II had both the calvarium and dura removed. The animals were sacrificed at 2. 3 and 6 weeks after kaolin instillation. Water content was determined 1mm, 2mm and 3mm from the lateral ventricle using conventional dry/wet weight techniques and microgravimetric analysis. Time matched controls were perfused in vivo and sections corresponding to sites of H20 content analysis were taken for electron microscopy. Another group of shunted hydrocephalic cats had water content determined or in vivo perfusion. Two patterns of water content emerged in the untreated hydrocephalic animals. In the Group II animals, there were early and progressive increases in water content (2-4% per week) at all sites which continued through 6 weeks. In Group I, significant increases in water content were only observed adjacent to the ventricle 3 and 6 weeks after kaolin, but not in remote sampling sites. These differences in H20 content between the two models correlated with differences in the rate of ventricular enlargement. Group II preparations showed an early rapid increase in ventricular size which slowed after 2 weeks. Ventricular size in Group I increased at a constant rate during the 6 weeks. Significant differences of ventricular size were seen between these groups at 2 and 3 weeks after kaolin, but was similar in both groups at 6 weeks. These changes in water content were mirrored in ultrastructural observations. In early phase expansion of extracellular space adjacent to the ventricular was seen in both groups. However, this expansion was more prominent in Group II than Group I. Gliotic changes were found close to the ventricle, but the gliosis was most prominent in Group I animals. Both ependymal and astrocytic junctions were intact in both preparations. Following insertion of the shunts, the water content was indistinguishable from normal control values in cats studied 2-3 weeks after shunting. However, the ultrastructural studies showed progression of gliosis. The implication of these changes on the clinical treatment ofhydrocephalus are discussed.

12. BRAIN SPECIFIC GRAVITY CHANGES IN EXPERIMENTAL HYDROCEPHALUS

Marc R. Del Bigio, J. Edward Bruni (Manitoba, Canada)

To examine changes in the water content of the hydrocephalic brain, hydrocephalus was induced in 4-5 month old rabbits by injection of silicone oil into the cisterna magna. At 3 days (n=4) and 1 (n=5) and 4 (n=6) weeks hydrocephalics along with appropriate controls (n=4) were decapitated and the brains quickly dissected over dry ice. Bilateral frozen tissue samples extending from the pial to the ventricular surface of the frontal horn were sectioned serially at 0.5 mm intervals and the specific gravity (SG) of the wet tissues was determined in kerosene/bromobenzene gradient columns. In 3 day hydrocephalics, the SG of the tissue 0.5 mm from the cortical surface was higher (P 0.01) whereas SG was lower at the ventricular surface. In 1 and 4 week hydrocephalic animals, SG values were similar and higher (P 0.02) than controls at all cerebral depths except at the ventricular surface. SG of comparable dessicated tissue samples was lower (P 0.001) in white than gray matter, but no differences existed between controls and hydrocephalics. The calculated water content decreased from 78.6% to 77.3% in gray matter and from 72.7% to 70.9% in white matter by 4 weeks post-injection,

These data suggest that, during acute stages of hydrocephalus when transependymal CSF flow is presumed to occur, water content in the periventricular white matter increases. At the same time, water is lost from the cortical regions. During chronic stages, water content decreases in both gray and white matter. We conclude that brain volume alterations in moderate hydrocephalus are due primarily to loss of tissue water and not loss of solid tissue components.

(Supported by grants from MHRF and MRC of Canada.)

13. DISTRIBUTION OF HEMATOPORPHYRIN DERIVATIVE (PHOTOFRIN II) FOLLOWING INTRAVENTRICULAR INJECTION IN THE RABBIT

Shigeyo Hyman, J. Gordon McComb, Michael L. J. Apuzzo (Los Angeles, CA)

The fluorescent characteristics and binding properties of hematoporphyrin derivative (Photofrin II) are well suited to tracing CSF flow patterns within and beyond the ventricular system.

New Zealand white rabbits had a 0.1 ml bolus of Photofrin II injected into one lateral ventricle. The distribution of the Photofrin II was then examined under ultraviolet light at 1, 24 and 72 hours.

In all rabbits, irrespective of time, fluorescence was confined to the injected ventricle with no dye being present in the contralateral ventricle. Intense fluorescence was present in the third ventricle, aqueduct of Sylvius, fourth ventricle, basal cisterns and spinal subarachnoid space; to a lesser degree in the olfactory, pineal and espiscleral regions; and only faintly over the convexities and cortical dura.

The distribution pattern of Photofrin II beyond the ventricular system was similar to that of other large tracers such as RISA and carbon black. These tracer studies indicate little CSF bulk flow over the dorsal surface of the rabbit brain. The findings also emphasize the importance of injecting both lateral ventricles during experimental studies.

- 14. THE EFFECT OF ACUTE KAOLIN HYDROCEPHALUS ON LYMPHATIC DRAINAGE PATHWAYS IN THE RABBIT
- J. Gordon McComb, Shigeyo Hyman, Floyd H. Gilles, Martin H. Weiss (Los Angeles, CA)

Physiologic and anatomic studies indicate that the majority of CSF drainage in the rabbit occur via the lymphatic system rather than by the arachnoid villus route. The present study examines the changes that result in these lymphatic drainage pathways in acute kaolin hydrocephalus.

New Zealand white rabbits had kaolin injected into both lateral ventricles. Carbon black was mixed with the kaolin to aid visual identification. Immediately thereafter, a four hour ventricular infusion was begun using artificial CSF to which \$125I-RISA\$ and indigo carmine was added for half of the studies. The animals were then killed and either \$125I-RISA\$ distribution measured or the CNS examined for kaolin/carbon black distribution, grossly or microscopically.

125I-RISA concentration in the deep cervical lymph nodes was reduced and the residual \$125I-RISA\$ remaining in the CNS higher compared with controls. Grossly, carbon black was highly concentrated within the ventricular system, basal cisterns and at the root exit zones of the cranial nerves; little was present over the hemispheric surface. Microscopically kaolin/carbon black was seen to be present in high quantity where the olfactory fila and optic nerves exited the subarachnoid space.

It is concluded that known major CSF egress pathways into lymphatic systems are occluded in acute kaolin hydrocephalus.

15. THE FORAMEN OF MONRO AS A CONTROL ELEMENT IN VENTRICULAR VOLUME REGULATION

Jason A. Brodkey, Harold L. Rekate, Howard J. Chizeck, W.A. Elsakka (Cleveland Heights, OH)

Using a mathematical model and computer simulation of the dynamics of the cerebrospinal fluid (CSF) system, it has been suggested that in order to maintain constant ventricular volume under physiologic conditions, the resistance provided by the foramina of Monro does not remain constant, or near zero levels, but varies according to pressure and flow rate. The foramina of Monro have been long misunderstood and often times wrongfully neglected in modeling attempts of the CSF system. We have shown that they provide a significant resistance to CSF flow and therefore may play a critical role in regulating ventricular volume. At CSF flow rates near, or slightly greater than the CSF production rate in dogs, the foramina of Monro provide maximum resistance. At even higher flow rates, the resistance drops to near zero levels thus approaching the characteristics of an opening and closing ball-type valve. The operating curve (resistance versus flow) for the foramina is of hyperbolic nature with the apex located at or slightly higher than physiologic norms for the CSF production rate. This apex may be the operating point about which the resistance of the foramina vary thus regulating ventricular volume. This physiologic controller of ventricular volume will be discussed in relation to classical experiments of the pathophysiology of hydrocephalus and post-shunt ventricular asymmetries.

16. THE NEUROPATHOLOGY OF SHUNTS

Randell G. Powell, Sydney S. Schochet, Jr., Vincent C. Traynelis (Morgantown, WV)

Materials consisting variously of shunt parts, intralumenal tissue fragments, surrounding tissues and fluids from 22 cases of shunted hydrocephalus who presented with shunt malfunction and/or infection were studied by standard neuropathological techniques. Analysis revealed a broad spectrum of responses of the hosts to the shunt parts including mild and marked foreign body reactions, extensive mineralization of the shunt tube and tract and spallation of the silastic tubing, occasional hemorrhage, and even aseptic eosinophilia suggesting an allergic response. The concept that silastic shunts are basically "inert" and passive participants in the pathophysiology of shunt complications is unjustifiably simplistic as demonstrated by our cases. Literature regarding neuropathology of shunt failure is reviewed and the impact of the insight gained on the management of shunt dysfunction and shunt infection is discussed.

17. THE SLIT VENTRICLE: A COMPLICATION OR GOAL OF SHUNTING

Arno Fried, Marion L. Walker (Salt Lake City, UT)

The management of the shunted hydrocephalic child with slit ventricles remains a problem for most neurosurgeons. Treatment options have varied from revising shunts in an effort to avoid the presence of slit ventricles to operating on only the most problematic cases. In order to understand the expected ventricular and clinical response to shunting, this study was designed to establish a hydrocephalus data base, to describe the "natural history" of shunted hydrocephalus and the slit ventricle syndrome.

The charts and CT scans of 370 shunted hydrocephalic children (mean follow-up 5.5 years ± 53 months) were reviewed and a computer data base generated. The main categories of data collection consisted of: 1) Etiology of hydrocephalus; 2) initial shunt data (age, valve, placement); 3) follow-up of ventricular size and head circumference; 4) follow-up of outcome and complications; 5) shunt malfunction data (revision types, number, ventricular response); 6) presence and treatment of slit ventricle problems.

Slit-like ventricular size after shunting is present in 63.4% of the total population. When those with severe brain anomalies are omitted, slit ventricles were seen in 79.5% of shunted children. In contrast, the incidence of the symptomatic slit ventricle was 11.5% of all shunted children with only 6.4% requiring surgical treament (p < .001 asymptomatic versus symptomatic slit ventricles). It took a mean of 4.5 months to develop slit ventricles after initial shunting with 60% developing slit ventricles after the initial shunt.

The symptomatic slit ventricle syndrome consisted of several categories of different presentations. These included: a) on and off recurring problems; b) postural headaches alone; c) recurrent shunt malfunction with acutely elevated ICP; d) school and behavior problems and; e) subdurals/synotosis problems. Only 6.4% of the total population required shunt treatment for symptomatic slit ventricles, most commonly antisiphon device and valve

17. THE SLIT VENTRICLE: A COMPLICATION OR GOAL OF SHUNTING (cont'd)

upgrade together. Sixty-nine percent of all shunted children required at least one shunt revision with 1.9 revisions per child in those with slit ventricles compared to 1.8 revisions per child in those never developing small ventricles (p>.1).

This hydrocephalus data base documents: 1) slit ventricles are common and should be the goal of hydrocephalus treatment; 2) the various symptomatic slit ventricle syndromes are much less common; 3) children with slit ventricles do not have increased shunt related problems compared to those without slit ventricles; 4) only the occasional child with unmanageable slit ventricle syndrome should have surgical efforts to increase the ventricular size and; 5) shunted children in whom there is not a prompt reconstitution of the cortical mantle with small ventricles should be re-evaluated for suboptimal shunt function.

18. THE TREATMENT OF HYDROCEPHALUS IN PRETERM INFANTS WITH INTRAVENTRICULAR HEMORRHAGE

John D. Reeves, Edward C. Benzel, Theresa A. Hadden (Shreveport, LA)

In order to evaluate the optimum treatment of premature infants with intraventricular hemorrhage, we retrospectively evaluated 19 patients who were surgically treated at LSU, Shreveport for hydrocephalic complications related to intra-ventricular hemorrhage.

The diagnosis of intra-ventricular hemorrhage was made on the basis of ultrasonographic or CT appearance of blood adjacent to or within the ventricular system. All shunting procedures were performed on patients with Grade III (15 patients) and Grade IV (4 patients) hemorrhages. The progressive included indications for surgery ventriculomegally and/or thinning of the cortical mantle in the face of inadequate control with repeated lumbar punctures. Six Rickham or McComb reservoirs were placed in the early neonatal period (in less than 1250 gram babies) allowing a safe but intermittent ventricular access. These were subsequently converted to ventriculo-peritoneal shunts in all but one of the patients. The remainder of the patients were initially treated with ventriculo-peritoneal shunts. They were older and more mature (greater than 1250 grams).

Six of the Grade III patients achieved a normal or near normal outcome, whereas three had a moderate developmental delay. Five were severely impaired and one died. The patients with a Grade IV hemorrhage had a poorer outcome with one moderate developmental delay, one severe developmental delay and two deaths. Thirty-two percent of the patients incurred a shunt infection and 58% ultimately required a shunt revision. These results compare favorably with the published literature.

It appears that the early placement of ventricular reservoirs is acceptable as an alternative to the early placement of ventriculo-peritoneal shunts. This approach may reduce the incidence of shunt infection as well as non infectious shunt complications.

19. EXTERNAL VENTRICULAR DRAINAGE (EVD) FOR INITIAL TREATMENT OF NEONATAL POSTHEMORRHAGIC HYDROCEPHALUS (PHH): SURGICAL AND NEURODEVELOPMENTAL OUTCOME

Torunn T. Rhodes, William H. Edwards, Robert E. Harbaugh, Richard S. Saunders, Michael Scott (Hanover, NH)

External ventricular drainage using a subcutaneously tunneled catheter has been our initial treatment for neonatal posthemorrhagic hydrocephalus since 1977. We have studied the surgical outcome of 38 infants treated with EVD through 1985, and the neurodevelopmental outcome in the survivors. The mean age at EVD placement was 13 days; mean duration of drainage was 21 days, and an average of 1.8 drains was used per patient. Complications associated with placement were apnea (10%) and hemorrhage (8%). Ventriculitis occurred in 6% of the patients. Ten infants died from pulmonary disease unrelated to the surgery. Reasons for removal of the drains were: elective in 35%, occlusion in 41%, dislodgement in 13% and proven or suspected infection in 11%. Sixty-eight percent of the survivors required a permanent shunt.

Neurodevelopmental outcome was evaluated at a mean age of 42 months. Eighteen of the 27 children (67%) had normal cognitive function, 3 had borderline and 6 (22%) had moderate to profound mental retardation. Nine of the children (33% had a normal neuromuscular exam. One child had mild, 11 had moderate and 6 had severe cerebral palsy. Four children had serious visual impairment. The worse outcomes were in the children with parenchymal or large intraventricular hemorrhages.

Our results compare favorably with other outcome reports of PHH. Early surgical decompression may have a beneficial effect on developmental outcome, although proof would require a controlled trial. We have found EVD to be a reliable method for treating PHH, with a low rate of serious complications.

20. GIANT INTRACRANIAL ANEURYMS IN CHILDREN AND ADOLESCENTS

Shigeru Nemoto, Sydney J. Peerless, Charles G. Drake (London, ON, Canada)

Forty-seven patients, age 20 years or less, with giant intracranial aneurysms (> 25 mm in diameter) have been treated in our unit. Thirty-two were age 15 or younger. Sixty-five percent of these aneurysms arose from vessels of the posterior circulation, with aneurysms of the basilar trunk being most common (28%). Twenty-four percent of the aneurysms were fusiform. Thirty-eight percent of the patients presented with subarachnoid hemorrhage; 36% presented with signs of neural compression and the remainder with headache. Forty-five patients underwent surgical treatment, with the neck of the aneurysm being clipped in only three patients and proximal vessel occlusion in 35 patients. Excellent results were obtained in 85% of the patients who were operated on in a good risk state, with an operative mortality of 7% and an overall management mortality of 11%. This experience with the treatment of giant intracranial aneurysms in children and adolescents is compared to the treatment of giant aneurysms in adults, to emphasize the difference in location, pathology and surgical management of these uncommon lesions.

21. PEDIATRIC CEREBRAL ANEURYSMS

Siggi A. Stephensen, Edward J. Kosnik (Columbus, OH)

The authors present 16 cases of pediatric cerebral aneurysms seen at Columbus Children's Hospital between 1954 and 1985. There were 11 males and 5 females, ranging in age from 3 months to 17 years. The mean age was 11.6 years. The Hunt/Hess grades on admission were as follows: Grade 0 - 2, I - 0, II - 7, III - 3, IV - 2, and V - 2. There were 5 anterior communicating, 6 middle cerebral, 4 internal carotid and 1 posterior communicating artery aneurysms. Seven patients had antiographic evidence of vasospasm. Four patients developed ischemic neurologic deficit, presumably secondary to vasospasm.

Surgery was performed in 12 patients. Eleven patients underwent aneurysmorrhaphy, 8 by clip application and 3 by silk ligature. One patient had a trapping procedure done. The outcome was excellent in 5 cases, good in 2 and fair in 3 cases. There were 2 post-operative deaths. Four patients died within 24 hours of admission from massive subarachnoid hemorrhage.

Using our experience with adults, we have developed an approach to the perioperative managment of pedicatric aneurysmal subarachnoid hemorrhage. This is based on manipulating central venous pressure and/or pulmonary wedge pressure in selected cases. The methods used, as well as the potential risks of these techniques, will be discussed. Interesting cases will be briefly presented.

22. INTRACRANIAL VASCULAR ANOMALIES OF INFANCY: MODERN CONCEPTS

John C. Godersky, Arnold H. Menezes, David Warner, Wendy R.K. Smoker (Iowa City, IA)

A variety of cerebrovascular abnormalities may become manifest in the first years of life. Their multisystem involvement demands attention to heart failure, hydrocephalus, and cerebral ischemia throughout the course of their evaluation and management. We present our recent experience with 9 children, ages 1 week to 2½ years (mean 6 months) who presented with congestive heart failure (4), progressive head enlargement (3), seizures and microcephaly (1) and subarachnoid hemorrhage (1). The anomalies included vein of Galen aneurysm (3), complex arteriovenous fistulae (2), posterior fossa and dural arteriovenous malformations (2), and arterial aneurysms (2).

CT scanning was invaluable in delineating the site of the lesion while arterial, digital subtraction angiography with general endotrachial anesthesia was utilized to obtain the high quality images, reduced dye volume and rapid filming, mandatory in these evaluations.

Direct surgical intervention was accomplished in 7 cases, while embolization was used to reduce flow through the 2 posterior dural AVMs. Swan-Ganz catheters were utilized in cases where CHF was present, for both the intra- and postoperative fluid management.

CHF resolved in all operated cases and improved in those undergoing embolization. Hydrocephalus improved in 2 cases with the elimination of the vascular anomaly, obviating shunt placement. Morbidity included seizures in 2 cases and transient diabetes insipidus in 1. Neurologic outcome was satisfactory and there was no surgical mortality.

These infants present challenges in evaluation and management which must be tailored to the individual. We will discuss our guidelines for management of these unusual problems.

23. SUBCORTICAL CAVERNOUS ANGIOMA: INTRAOPERATIVE ULTRASOUND AS AN ADJUNCT TO LOCALIZATION AND RESECTION

Martin J. Buckingham, Kerry R. Crone, Thomas S. Berger, Bokyung K. Han (Cincinnati, OH)

Intracranial cavernous angiomas are vascular malformations which occur uncommonly in adults and rarely in children.

We report two cases of cavernous angioma in children both of whom presented with seizures. One patient had a progressive seizure disorder with growth of the lesion documented on C.T. Preoperative diagnosis was suggested by characteristic C.T. findings, which included a hyperdense lesion without surrounding edema, slight enhancement following administration of contrast, and normal angiography.

Both lesions were located subcortically in the left frontal lobe and at operation the cortex appeared normal. We found intraoperative ultrasound to be useful in localizing these lesions and in documenting complete removal. Both patients were without neurological deficit and seizures free postoperatively.

24. INCREASES IN CEREBRAL INTERSTITIAL FLUID AND CEREBROSPINAL FLUID ADENOSINE CONCENTRATION DURING HYPOXIA IN NEONATAL PIGLET

T.S. Park, David G.L. Van Wylen, Rafael Rubio, Robert M. Berne (Charlottesville, VA)

Hypoxic-ischemic brain injury is the most important neurological problem during the perinatal period. To understand the pathogenesis of this condition, it is necessary to study the precise roles played by metabolic mediators in regulating blood flow of the perinatal brain. In this study, the effects of arterial hypoxia on interstitial fluid adenosine concentrations were investigated in the frontal cortex and thalamus by the brain dialysis technique (1) and in CSF from the cisterna magna of the newborn piglet. Under control conditions the interstitial adenosine concentrations were 0.75 \pm 0.31 (SEM) μ M in the frontal cortex and 1.09 \pm 0.29 μM in the thalamus. Acute hypoxia (Pa02=20 ± 1 mmHg) increased interstitial adenosine by 2.4-fold in the frontal cortex and 2.5-fold in the thalamus. Interstitial inosine and hypoxanthine also increased significantly during hypoxia. The adenosine concentration in the cisterna magna CSF under control conditions was 0.04 ± 0.01 (SEM) μM , which increased significantly to $0.17 \pm 0.04 \mu M$ with hypoxia (Pa02=4.7 ± 1.2mmHg). Cisterna magna CSF inosine levels did not change significantly during the severe hypoxia. Adenosine concentrations found in the interstitial space and CSF of newborn piglets under normal and hypoxic conditions are within the vasodilator range. These results thus suggest that in the neonatal brain adenosine may play a role in regulating blood flow during hypoxia.

1. VanWylen DGL, Park TS, Rubio R. Berne RM: Increases in cerebral concentration during hypoxia, local potassium infusion and ischemia. J Cereb Blood Flor Metabol (in press, 1986)

Supported by NIH grants NS-21045, NS-00924, $\rm HL$ -10384 and United Cerebral Palsy Foundation grant R-350.

25. IN VIVO NMR STUDIES OF INCREASED INTRACRANIAL PRESSURE IN THE CAT

Leslie N. Sutton, A. McLaughlin, W. Kemp, B. Cho, M. Schnall, T.W. Langfitt, B. Chance (Philadelphia, PA)

Management of ICP in the clinical setting is really an attempt to maintain brain energy metabolism, but until recently this could not be directly assessed. Studies over the past two decades have shown that as the ICP rises, the mean arterial blood pressure also rises in an attempt to maintain the cerebral perfusion pressure constant (cerebral perfusion pressure is defined as the difference between the mean arterial blood pressure and the ICP). At high values of ICP, however, this autoregulation fails and ischemia results. We have used 31P and 1H NMR techniques to study two important questions: 1) What is a "critical ICP" which, if untreated, results in the failure of energy metabolism? 2) What are the limits of reversibility of metabolic failure due to increased ICP?

Five cats were anesthetized, cannulated with arterial and venous lines, and artificially ventilated. Arterial blood pressure and ICP were monitored, and arterial blood samples were withdrawn for analysis of blood gases and pH. The ICP was increased by the gradual infusion of mock CSF into the cisterna magna. The temporal muscle was resected and a double tuned surface coil placed on the skull. 31P and 1H spectra were acquired simultaneously using a PhospoEnergetic spectrometer operating at a 2.2 T.

Threshold ischemia, manifested by elevated cortical lactate, occurred at 60, 50, 28 and 55 torr cerebral perfusion pressure. Loss of phosphocreatine occurred at lower cerebral perfusion pressures (15,18,25,33,55 torr). This wide variability among different animals reflects different autoregulatory curves and emphasizes that there is no universally "safe" cerebral perfusion pressure. Metabolic recovery was possible even with complete loss of ATP.

26. CSF PULSATILITY: RESPIRATORY EFFECT ON WAVE SLOPE AND AS AN INDEX OF INTRACRANIAL PRESSURE

Eldon L. Foltz, Jeffrey Blanks (Orange, CA)

CSF pulse pressure and wave slope has previously been reported as an index of intracranial compensation for pressure elevation. Further refinement of this concept implies that the slope of CSF pulse does correlate with intracranial compliance. Intracranial compliance is expressed as a slope unit at any point on the graph line produced by the ratio of intracranial pressure increase to intracranial pressure volume increase, the familiar delta pressure/delta volume CSF relationship. The flatter this slope, the greater the intracranial compliance.

In 30 patients under study to identify the degree of remaining intracranial compliance (i.e., how severe is the hydrocephalus, or to what degree has ICP increase been compensated), CSF pulse wave forms were analyzed for slope changes during normal inspiration and expiration. Slope measurements for ten pulses were analyzed during normal inspiration and expiration respectively.

Three groups of patients were identified: 1) those patients with full compliance showed an I (slope during inspiration)/E (slope during expiration) of 2.0 or greater; clinical review showed these patients were normal and nonhydrocephalic patients; 2) those with moderate compliance showed I/E of 1.3-1.7; clinically these patients were slowly progressing hydrocephalus, shunted hydrocephalus, or compensated hydrocephalus; 3) those with lack of compliance, an I/E of less than 1.3; clinically, these patients were suffering from high ICP symptoms, progressing hydrocephalus, or brain tumors.

This simple technique does not rely on mean pressure. It is a simple and quick technique to quantitate remaining intracranial compliance and its attendant ICP implications.

27. INFLUENCE OF ICP MONITORING ON OUTCOME AFTER NEAR DROWNING

Jack Stern, David Reynolds, Samuel S. Kasoff (Valhalla, NY)

Accidental drowning is the leading cause of death and severe neurological morbidity in children. Conn has classified near drowning victims into three groups: A. awake, B. blunted consciousness, C. comatose. Comatose patients were further divided into three subgroups: C1, decorticate, C2 decerebrate, C3 flaccid. Nussbaum concluded that there were no significant prognostic parameters to distinguish C3 patients who recovered (29%) from those with significant brain damage (23%). The patients that expired (48%) all had initial ICP's greater than 20 mm. Hg and CPP's less than 50 mms. Hg. Conn's group came to similar conclusions and Bohn's group at the Toronto Childrens Hospital and has abandoned the use of ICP monitoring as part of the treatment protocol for near drowning. We present 14 children treated at over the past three years. Children falling into Conn's A or B classification where not monitored and therefore excluded from this analysis. There were 11 males and 3 females with a mean age of 2.6 years. All children had epidural monitors and continuous arterial and CVP measurements. Four children had initial ICP's greater than 20 mm Hg. and CCP's less than 50. These children had GCS's of 3 or less and were in the C3 group - all expired. This despite the administration of Manitol and of Pentobarbitol. The remaining 10 children all had normal or minimally elevated ICP's and CPP's. Their GCS's range from 3-6 and most were in Conn's category Cl and C2. Only 3 of 10 made full recovery. The remainder remained vegetative or severely impaired. We conclude the following: 1. Elevated ICP correlates with the patient's demise, 2. This relationship was unaltered by aggressive treatment with Manitol or Pantobarbitol, 3. "Peak Swelling" at days 2-4 was not observed in any patient and in fact there was little variation, in any individual patient from the initial ICP or CPP. 4. Normal ICP correlated with survival but not with degree of neurological function, 5. Specific portions of GCS i.e. the initial clinical exam, may be a better indicator of recovery than the score as a whole, 6. ICP monitoring and treatment did not alter survival or outcome.

28. VOLUMETRIC ANALYSIS, RADIOGRAPHIC FINDINGS, AND ICP CHANGES IN CRANIOSYNOSTOSIS

E. Larry McCleary, Lee A. Harvey, Robert W. Hendee, Jr. (Denver, CO)

Conventional teaching has been that craniofacial dysmorphism resulting from calvarial sutural abnormalities was a purely cosmetic problem. This might not be entirely correct.

Early observers noted severe visual loss in children with malformed heads. Recent evidence has accumulated to suggest that intracranial pressure may be elevated in this group of children and probably was the likely etiology for the visual loss which was described in these children several hundred years ago.

Over the past several years we have been asked to evaluate a large population of children for possible craniosynostosis involving the anterior cranial vault. These children have been evaluated radiographically and in selected instances ICP monitoring was carried out. Results of these studies support prior claims documenting the presence of increased intracranial pressure in certain of these children.

Intracranial pressure monitoring data will be presented and will be compared with the clinical course and the radiographic findings. Preliminary results of cranial vault volumetric analysis which has been done in selected cases will also be discussed.

29. SOME COMMENTS ON SKULL GROWTH AFTER CRANIOFACIAL REPAIR

Arthur B. Eisenbrey, John Spolyar, William Vasileff, Alexa I. Canady (Detroit, MI)

The results of the surgical procedures carried out for craniofacial dysostosis and the various syndromes associated with premature closure of the sutures, have not always been entirely satisfactory.

In order to better evaluate the growth of the skull in infants and children, bone markers have been placed in both control subjects and those with various developmental abnormalities. The continuing measurements of the displacement of these markers from fixed anatomical points has been followed in serial x-ray studies in a cephalometer with a standardized technique. A mannequin was used to test the validity of all the measurements to a ninety-five and ninety-nine percent accuracy.

In the evaluation of only one aspect of the total study which has been undertaken, it has been found that within the first thirty-six months of growth of the skull and of the skull base, that principle growth occurs as a posterior displacement of the occipitoparietal regions with a rolling back and unfolding of the posterior aspects of the head and ascending development of the frontal areas, with considerably less anterior displacement than posteriorly.

The techniques for the insertion of bone markers and subsequent measurements with developmental tracings will be shown.

It is our conclusion that a considerably better overall development of the skull and its shape can be obtained if more attention is directed to the lambdoid sutures as well as the sychondroses and the development of the occipital bone with more extensive surgery being directed at these areas in children having presumed diagnosis of Crouzon syndrome and other coronal and sphenoidal abnormalities.

- 30. THE SPECTRUM OF CEREBELLAR DYSGENESIS AND POSTERIOR FOSSA CYSTS DEMONSTRATED BY MRI
- R. Michael Scott, Samuel M. Wolpert, Mary L. Anderson, Eddie S.K. Kwan, Val M. Runge (Boston, MA)

Ten children or adolescents with retro- or supracerebellar fluid collections were studied by CT and MRI. CT scans demonstrated a spectrum of appearances ranging from "classic" Dandy-Walker syndrome to "classic" retrocerebellar cyst. MRI delineated an area of cerebellar dysgenesis or hypoplasia in many patients, however, suggesting that many cystic collections in the posterior fossa are associated with an underlying focal disturbance of cerebellar formation. Classic Dandy-Walker Syndrome may therefore represent the most florid manifestation of a cerebellar dysgenesis syndrome associated with many posterior fossa cystic collections.

31. ADMINISTRATION OF INDOMETHACIN FOR THE PREVENTION OF INTRAVENTRICULAR HEMORRHAGE IN HIGH-RISK NEWBORNS

William C. Hanigan, Gail Kennedy, Frank Roemisch, Robert Anderson, Tom Cusack, Tim Miller (Peoria, IL)

Over a two-year period, 203 preterm infants with a birth weight of 500 to 1499 g and gestational age of less than 34 weeks were admitted to the Neonatal ICU at the Saint Francis Medical Center. 122 children were enrolled in a randomized, double-blind trial using intravenous indomethacin (0.01 mg/kg) or placebo for the prevention of periventricular/intraventricular (PVH/IVH) hemorrhage. Medication was administered prior to 12 hours of age and at 24, 48, and 72 hours following birth. Over a seven-day period, cranial ultrasonography was used to identify the presence of a PVH/IVH.

Infants were stratified into low weight (501 to 1000 g) or high weight groups and randomized using a variable length, stratified block scheme. The study design used a group sequential analysis with asymmetric one-sided boundaries for hypothesis testing. Alpha of 0.05 and power of 90% was used with a projected 50% reduction in a 40% incidence of PVH/IVH.

31. ADMINISTRATION OF INDOMETHACIN FOR THE PREVENTION OF INTRAVENTRICULAR HEMORRHAGE IN HIGH_RISK NEWBORNS (Cont'd)

39 infants were included in the low weight group. 6/19 (32%) of these infants receiving placebo sustained a PVH/IVH while 5/20 (25%) of the infants receiving indomethacin sustained a PVH/IVH. 83 infants in the high weight group demonstrated that 9/41 (22%) receiving placebo and 4/42 (10%) receiving indomethacin sustained a PVH/IVH. Interim analysis did not indicate that the study should be terminated. In the analysis of the completed study, although there was a weak suggestion that indomethacin was beneficial in the high weight group (Fisher's exact test - 1 tail; p < 0.15), there was no statistically significant difference in either weight group.

Six infants (3 receiving placebo) were withdrawn from the study because of the development of oliguria or thrombocytopenia. These complications were not serious enough to warrant code breaks. It is concluded that the prophylactic administration of indomethacin at this dosage, in the premature newborn does not significantly decrease the frequency of PVH/IVH.

32. OPTIC NERVE DECOMPRESSION FOR OSTEOPETROSIS IN EARLY CHILDHOOD

Stephen J. Haines, Donald L. Erickson (Minneapolis, MN)

Progressive bony overgrowth secondary to defective osteoclast function in osteopetrosis can lead to encroachment upon foramina at the base of the skull causing cranial nerve dysfunction. Among the most disabling of these problems is visual loss secondary to optic atrophy related to compression in the optic canal. In aggressive forms of the disease, such encroachment may occur early in childhood leading to visual loss which, because of the difficulty of sensitive visual evaluation in young children, may go unnoticed and therefore untreated for relatively long periods of time. We have recently had the opportunity to decompress seven optic nerves in four children with osteopetrosis and report technical aspects of the evaluation, operation and outcome.

Four children (2 months, 8 months, 18 months and $3\frac{1}{2}$ years) underwent seven operations to decompress seven optic nerves. There was one bilateral operation and one patient in which decompression was incomplete and a seond operation was required. Five nerves were evaluated pre and postoperatively with flash visual evoked potentials. In two nerves, in which there was essentially no measurable preoperative response, there was no change 3 weeks postoperatively. In one there was no change 5 weeks postoperatively but 3 months postoperatively evoked potential had returned to normal. In one there was noticeable improvement in evoked potential 4 weeks postoperatively and one evoked potential showed mild deterioration 4 days postoperatively but measurable significant improvement 8 months postoperatively. One nerve was followed with serial evoked potentials but was not decompressed. Two nerves were followed with serial postoperative evoked potentials. None of these nerves showed significant changes in the evoked potentials over periods of 2, 3 and 8 months. Improvement in evoked potentials not seen in unoperated nerves occurred in the majority of nerves evaluated. The likelihood of improvement in evoked potentials may correlate generally with the severity of abnormality preoperatively. We conclude that flash visual evoked responses are a useful adjunct to the assessment of the need for and response to optic nerve decompression in early childhood.

33. SURGICAL TREATMENT OF PEDIATRIC INTRACRANIAL ARACHNOID CYSTS

J.B. Delashaw, William C. Broaddus, T.S. Park (Charlottesville, VA)

Neurological deficits due to intracranial arachnoid cysts present in children as a result of mass effect or obstruction of cerebral spinal fluid outflow. Surgical treatment of these cysts remains controversial.

A retrospective review was performed in 10 children treated for intracranial arachnoid cysts. Patient age ranged from 2 months to 18 years. Cyst locations included four in the frontal or temporal regions, four in the suprasellar area, and two in the posterior fossa. Craniotomy with fenestration was performed on 8 patients. One of them required repeat craniotomy for recurrence two years later and another underwent a cystoperitoneal shunt as a second procedure. Cystoperitoneal shunting as the first surgical therapy was performed on one child with a suprasellar cvst. Five months after her first procedure, her arachnoid cyst had recurred. She underwent craniotomy with fenestration and removal of her cystoperitoneal shunt which successfully reduced the size of the arachnoid cyst. Craniotomy with fenestration combined with cystoperitoneal shunting was performed on one child with a suprasellar cyst as the first means of therapy. This led to a persistent reduction in cyst size.

These results indicate that craniotomy with fenestration may be the procedure of choice to successfully treat intracranial arachnoid cysts.

34. SELECTION OF SPASTIC CEREBRAL PALSY PATIENTS FOR SELECTIVE POSTERIOR RHIZOTOMY

Warwick J. Peacock (Los Angeles, CA)

Where the spasticity of cerebral palsy has proved resistant to standard forms of therapy, selective posterior rhizotomy may be of value. Spasticity interferes with function by increasing resistance to movement. In selective posterior rhizotomy, the rootlets comprising the posterior roots of L2-S1 on the left and right sides are stimulated and those rootlets associated with an abnormal response are divided thereby decreasing spasticity. With intensive postoperative therapy postural and functional gains may then be made.

The diagnosis of cerebral palsy should be definitive. Cerebral palsy is a non-progressive motor disorder due to an insult to the brain before, during or shortly after birth. The patients should be purely spastic without evidence or choreoathetoid movements as dystonia does not appear to be influenced by posterior rootlet section. Good power should be evident as patients whose muscles are weak may be using spasticity to assist function. Spasticity should be interfering with function, which may not be the case if the patient has severe fixed contractures with limited range of movement or if there has been previous orthopedic surgery which has corrected postural abnormalities. Although older patients usually benefit from rhizotomy, pre-school children often improve more dramatically. Intelligence and motivation are important if major functional gains are to be made, but retarded spastic children should not be excluded as they benefit significantly from reduction in hypertonia. It is also extremely important that the patient receives carefully planned neurodevelopmental therapy after surgery to unlearn abnormal movement patterns used to overcome spasticity and to acquire more normal new patterns.

35. THE SIGNIFICANCE OF LATERAL ODONTOID DISPLACEMENT IN THE DIAGNOSIS OF ATLANTO-AXIAL SUBLUXATION

David M. Klein, Jerald P. Kuhn (Buffalo, NY)

Many types of atlanto-axial dislocation are unstable and can pose a serious threat to neurological function or to life, but the significance of "rotary subluxation", as is usually defined from plain x-rays, is questionable. We have previously reported that 5 patients given this radiographic diagnosis during episodes of acute wry neck showed normal clinical and radiographic findings within 4-56 days as symptoms subsided, excepting for persistence of unilaterial displacement of the odontoid (odontoid offset) in 4 of 5 follow-up x-rays.

Assessment of craniocervical motion was made as part of routine CT scanning in 121 patients, 3-18 years of age, having no known cervical or posterior fossa disease. Measurements of odontoid position relative to the midline of the atlantal ring were made directly on the films and confirmed with the CT cursor. In 26 patients (21.5%) there was odontoid offset of 1.0 mm. or more. With rotation, a consistent pattern of movement was generally observed between the odontoid and the atlantal ring.

We conclude that: 1) offset odontoid is a common normal variant in children as well as a product of normal rotation, 2) radiographic diagnosis of "rotary subluxation" frequently captures patients with cervical muscle spasms or normally offset odontoids, or both, 3) true "rotary fixation", if such a problem exists, cannot be identified by conventional radiographs.

36. EVALUATING CRANIOVERTEBRAL JUNCTION ANOMALIES WITH FLEXION-EXTENSION MRI

Conrad Pappas, Harold Rekate (Phoenix, AZ)

Precise radiological identification of craniovertebral junction anomalies is imperative for operative management. In the past, pluridirectional tomography combined with contrast myelography has been used to define the site and type of compression at the craniovertebral junction. However, flexion-extension MRI gives better definition of these abnormalities. For example, a $3\frac{1}{2}$ -year-old male born with cervical hemivertebrae presented with quadriparesis and bilateral sixth cranial nerve palsy. MRI showed brain stem compression, and flexion-extension MRI showed an abnormal range of odontoid movement constricting the cervicomedullary junction. MRI also demonstrated basilar invagination of the odontoid with marked bowing and compression of the cervical medullary junction in a 13-year-old female with impaired extraocular movements. Flexion-extension MRI studies showed abnormal angulation of the brain stem in flexion. Both patients underwent ventral and posterior decompression. Postoperatively, the patients had marked neurological improvement. MRI provides a thorough understanding of the static and dynamic anatomic relationships essential to therapeutic decisions intended to help reverse longstanding neurological deficits. By identifying specific structures, the site and nature of the compression can be identified definitively.

37. ATLAS ASSIMILATION: IMPLICATIONS AND MANAGEMENT

Arnold H. Menezes (Iowa City, IA)

Assimilation of the atlas is the most frequently encountered, but poorly understood, abnormality of the craniovertebral junction (CVJ). Progressive problems range from atlanto-axial instability (AAI) to severe basilar invagination (BI) and soft tissue mass compromising the medulla. Thirty-four symptomatic patients were investigated prospectively to provide insight into the pathological history and management.

Assimilation comprised 13% (34/270) of CVJ abnormalities treated. Presentation was 5-20 years: 12; 20-40 years: 15, 40-60 years: 5. Symptoms included nuchal rigidity, brain stem-cervical myelopathy and cranial nerve dysfunction. Sleep apnea 4, and acute trauma precipitated deficits 6. Basilar migraine and scoliosis was peculiar to children.

Patients were evaluated by dynamic myelotomography, CT, traction, MRI and at operation. Foramen Magnum effective sagittal diameter was always less than 20 mm. Segmentation failure C2-C3 (28/34) and 26 had irreducible basilar invagination surrounded by granulation necessitating ventral medullary decompression (22 had secondary dorsal fixation). Reducible AAI or BI was only in children below 15 years (8/34) where no granulation was seen. It was irreducible in 2 older teenagers. Associated Arnold-Chiari Malformation (ACM) in 12/34 and hydromyelia 3. 10/12 ACM had immediate improvement with ventral decompression of BI. All patients had posterior atlanto-axial arthrodesis. Improvement was the rule.

CONCLUSIONS: Assimilation with non-segmentation C2-C3 (43% ACM) leads to AAI in childhood. Subsequent odontoid upward invagination and granulation proliferation from instability, leads to irreducible neural compromise. To arrest the disease process, attention must be focused on the pediatric age group.

38. SINUSITIS AND INTRACRANIAL INFECTION IN CHILDREN

Dennis L. Johnson, David C. McCullough (Washington, D.C.)

At Children's Hospital National Medical Center 17% of children admitted with acute sinusitis will develop intracranial infection. We reviewed the treatment of 13 such children whose ages ranged from 11 to 18 years. All of the patients presented with headache and all but three of the patients presented with ipsilateral, frontal, percussion tenderness. Six children presented with behavioral change, and in four cases this diagnosis was heralded by a seizure.

The pansinusitis was diagnosed in most patients; but the posterior wall of the sinuses was intact in all cases but one. A potpourri of bacteria was found in both the sinuses and the intracranial abscesses, but the majority were anaerobic. Five children were found to have a brain abscess, four had a subdural empyema, and four had an epidural abscess. In nearly all cases there was radiographic evidence of disease progression in the face of medical management alone.

Surgical treatment consisted of simultaneous drainage of the involved sinuses and the intracranial abscess. The majority of children were treated with intravenous antibiotics for six weeks following surgical drainage.

All children returned to their premorbid neurologic condition. None of the four patients who had seizures preoperatively developed a persistent seizure disorder.

Although the introduction of CT has ushered in a new, improved era in the treatment of intracranial infection, our date conflicts with recent reports recommending nonsurgical treatment of intracranial infection associated with sinusitis.

39. RASMUSSEN'S ENCEPHALITIS - SURGICAL MANAGEMENT REVISITED

Benjamin S. Carson, Donlin M. Long (Baltimore, MD)

Rasmussen Encephalitis is characterized by progressive intractable seizures associated with atrophic changes in one cerebral hemisphere and progressive hemiparesis. The disease process usually presents in childhood and can progress to a point of total debilitation and even death.

In the last year we have treated six patients with this complex of signs and symptoms by way of cerebral hemispherectomy. All of the patients became seizure-free post-operatively and retained neurological function which improved with subsequent physical therapy. The morbidity has been small and there has been no mortality.

All of these patients were severely handicapped prior to undergoing hemispherectomy and were much more functional after surgery. In light of this, knowledge about this disease process and its successful surgical treatment should be emphasized in the medical community. The techniques of surgical management and post-operative care will also be presented.

40. CSF FORMATION IN ACUTE VENTRICULITIS

Robert E. Breeze, J. Gordon McComb, Shigeyo Hyman, Floyd H. Gilles, Martin H. Weiss (Los Angeles, CA)

Clinically there appears to be a significant reduction in cerebrospinal fluid (CSF) formation during acute ventriculitis, an observation that has not been confirmed by experimental studies.

New Zealand white rabbits had an inoculum of E. Coli injected into one or both lateral ventricles. Approximately sixteen hours later the survivors (40%) underwent a ventriculo-cisternal perfusion for three hours using 14_{C}-Dextran (MW-7x10 4) as a reference marker for CSF formation.

CSF formation on the average was reduced to one third of normal, confirming the clinical observation. Histologically the stroma of the choroid plexus was the site of an extensive inflammatory infiltrate. Meningitis, ependymitis and encephalitis completed the picture. Vasculitis was not present.

It is presumed that the alterations in the choroid plexus by the inflammatory process produced the diminished CSF formation in this acute setting.

41. CT EVALUATION AND MANAGEMENT OF H FLU SUBDURAL EFFUSIONS

Fraser C. Henderson, Steven K. Gudeman, Phanor L. Perot (Charleston SC)

H Flu is responsible for approximately 80% of bacterial meningitis in infants and children under two years of age. Subdural effusions complicate up to 50% of cases. The CT findings of subdural effusions have not been previously described. The history and CT scans of 31 infants under 24 months of age with culture proven H Flu meningitis were reviewed. A comparison of clinical findings of the infants with H Flu meningitis alone versus those with meningitis and subdural effusions, allowed us to identify major and minor criteria for the diagnosis of subdural effusions secondary to H Flu meningitis in infants. Major criterialincluded a finding of a new neurologic deficit, the new onset of seizures or a bulging fontanelle. Minor criteria included spasticity, poor feeding or emesis, grunting respirations or the presence of fever or lethargy for more than 3 days after the institution of antibiotic therapy. The presence of one major or 2 minor criteria was highly suggestive of a subdural effusion and in the authors' opinion justified a CT scan. Of the 19 CT scans performed, 16 of which met the criteria above, 15 revealed subdural effusions. Examination of those 15 scans revealed the following findings: low density extra axial, crescent shaped fluid collections 4mm-2cm in thickness (15/15), enhancing membranes (9/15), effacement of adjacent sulci (10/15), the appearance of cerebral atrophy (8/15), midline shift (2/15), ventriculomegaly (2/15) and encephalomalacia (1/15). The effusions were predominantly frontal (10/15) and bilateral (9/15), other locations were parietal (3/15), occipital (1/15) and temporal (1/15). Two years later, 8 of the 15 patients with subdural effusions including the 4 whose effusions that had been tapped were available for follow-up study. Follow-up CT scans were normal in all but 3 cases. One showed marked encephalomalacia with hydrocephalus and two mild cerebral atrophy. There were no persistent subdural effusions or contrast enhancing membranes. There was no significant difference between the tapped and untapped patients. The authors conclude that while identifying the nature of the effusions, percutaneous aspiration serves no diagnostic purpose that is not met by CT scan. The authors

41. CT EVALUATION AND MANAGEMENT OF H FLU SUBDURAL EFFUSIONS (Cont'd)

acknowledge that aspiration may serve a therapeutic function in certain instances. The authors recommend that for diagnostic purposes in infants with H Flu meningitis CT scan be performed on all patients meeting at least one major or 2 minor criteria to determine the presence of subdural effusion.

42. PEDIATRIC SPINAL TRAUMA

Mark N. Hadley, Joseph M. Zabramski, Volker K.H. Sonntag, Harold Rekate (Phoenix, AZ)

One hundred pediatric patients (2-months to 16-years-old) with spinal column injuries were evaluated over ten years. Males outnumbered females 3:1. Accordingly, the injuries were most commonly due to motor vehicle accidents, falls, and sports and diving injuries. The injuries ranged from occipitoatlanto sublaxation to lumbosacral fractures. A multivariate analysis included age, type and level of injury, neurological compromise the treament administered, and long-term outcome. Younger patients (ages 0-9 years) had more severe neurological injuries after spinal trauma: a 77% incidence of more severe neurological compromise compared to 38% for patients 10-13 years and 57% for 14-16 years. These same younger patients had a lower incidence of fractures and a higher incidence of subluxation only or of spinal cord injury without fracture or subluxation than their older counterparts. In addition, 71% of the injuries in the 0-9 age group involved the cervical spine; 43% were located between the occiput and C2. Overall, 79% of the patients with an incomplete neurological injury improved by at least one Frankel grade; however, only 10% of the patients with a complete neurological injury improved (median follow-up 25 months). Management recommendations for each patient as determined by the patient's age, level and type of injury, and the degree of associated neurological compromise will be presented.

43. OUTCOME OF PEDIATRIC DEPRESSED SKULL FRACTURES

Ann M. Flannery, Yoon S. Hahn, Martha J. Barthel, David G. McLone (Chicago, IL)

Since 1970, 141 cases of depressed skull fractures (DSF) in children under the age of 16 have been seen at the Children's Memorial Hospital, Chicago. Review of these cases show 121 were managed operatively. Sixty-nine percent of the 141 were male. Forty-one percent were compound (open) fractures, 59% were closed. Follow-up of one to forty-eight months reveals an over all mortality of 2%, 2.8% seizures and 0% infections.

Of the 121 patients treated surgically, 26 (21.5%) improved, 93 (76.9%), were unchanged, and 2 (1.6%) worsened. Only ten of 141 children were judged to have a poor outcome. These cases included 2 acute deaths, one patient who remained vegetative and later died, five who were moderately disabled physically, one who had a documented decline in I.Q., and one who remained severely disabled.

Approximately one-fourth of the fractures explored surgically showed evidence of dural laceration. We subdivided these patients with dural lacerations as either simple, without cortical damage, (10 cases) or as complex, with associated cortical damage (22 cases). The 22 cases with complex dural lacerations included two distinct clinical groups based on outcome. Sixteen of the 22 had a good outcome. Seven of these 16 improved following surgery. Six of the 22 had a bad outcome. These six cases represented 75% of all those operated on with a bad outcome. This same group also includes one of the two patients who worsened following surgery, as well as two of the three patients who succumbed.

This retrospective analysis of DSF reveals that the subgroup with complex dural lacerations represents a special clinical challenge. While most of this group will improve following surgery, a significant number will prove to be the least responsive to therapy. The authors will attempt to identify the criteria by which those with highest risk may be identified for special care.

44. OUTCOME OF PEDIATRIC HEAD INJURIES IN CHILDREN UNDER 36 MONTHS OF AGE

Yoon Sun Hahn, David G. McLone, Chee Hong Chyung, Ann M. Flannery, Martha Barthel (Chicago, IL)

The Glasgow Coma Scale as an objective assessment of neurological function has limited utility for children under the age of three years. To overcome this limitation a "Children's Coma Scale, (C.C.S.) (3-15)" was developed modifying the Glasgow Coma Scale and applied to those children 36 months or younger. The demography and clinical features of 36 months or younger and older children are compared. The results of treatment and outcome of these younger patients are analyzed.

In a five year study period from 1981 through 1985, there were 738 head injured patients (0-16 years of age) admitted to Children's Memorial Hospital in Chicago. Three hundred and eighteen patients (43.1%) were under 36 months of age.

Initial data on the outcome of these younger children showed the following observations:

- 1. Abnormal pupils, I.C.P. and C.C.S. seem to be prognostically significant. Seventeen children showed "abnormal pupils (anisocoria, fixed dilatation or small pupils)." Out of five children who had fixed dilated pupils, four died and one remained "vegetative". Conversely, children who had anisocoria or "small pupils" showed good prognosis. Seven out of 10 patients were "good" or "moderate", three remained "vegetative".
- 2. Children's Coma Scale (C.C.S.) was found to be as useful as the Glasgow Coma Scale (G.C.S.). Twenty-three children had C.C.S. under eight. Six out of seven children with C.C.S. of three or four subsequently became severely disabled or remained vegetative. All 11 children with C.C.S., seven or eight showed good recovery or were moderately disabled.
- 3. Other factors affecting to the outcome of these younger children will be presented.

45. HEAD TRAUMA IN INFANTS

Maurice Choux, Lorenzo Genitori, Alberto Yanez, Gabriel Lena (Marseille, France)

2190 infants with head trauma have been hospitalized in the Pédiatric Neurosurgical Départment of the Hospital de la Timone in Marseille, since 1972 to 1985. 57% were boys, and the étiology was: fall (80%), traffic accident (7.4%) and battered child (2%).

An initial loss consciousness was observed in 438 cases (20%). 67 were in comatose state more than 24 hours. Immédiate post traumatic seizures were observed in 67 patients (3%).

Skull lésions were present in 829 cases (37.8%). 744 were linear fracture. Topographically linear fractures were pariétal (62%), occipital (15%), pariéto-occipital (7%), frontal (6%), fronto-pariétal (3%), temporal (2%).

85 were depressed fractures: 55% parietal, 16% frontal, 9% occipital, 7% parieto-temporal, 6% parieto-occipital. 8 infants with linear fracture develop with a growing fracture.

In this series 31 extra-dural hematomas were operated (52.4% were large and extensive, 30% were localized in the temporal region, 8% were parietal, 6.4% occipital, 3.2% frontal). In 39 infants an acute or subacute hematoma was discovered. 63 brain lesions (contusion, cerebral hematoma) were observed.

14 infants died (0.63%). The sequellae are studied.

46. PROFILE OF PEDIATRIC HEAD INJURY: A PROSPECTIVE STUDY OF 1906 PATIENTS LESS THAN FIFTEEN YEARS OF AGE

Thomas G. Luerssen, Melville R. Klauber, Lawrence F. Marshall, Howard M. Eisenberg, Michael E. Miner (San Diego, CA)

A longitudinal prospective study, including three metropolitan areas, acquired data on 9272 patients who were admitted to the hospital for treament of a head injury. One thousand nine hundred six (20%) of these patients were fourteen years of age or less. This patient group provides the largest prospectively studied population of pediatric head injury reported so far, and yields an accurate profile of pediatric head injury. The overall mortality for this group of patients was 2.5%, with the highest mortality occurring in the youngest of children. The major cause of pediatric head injury was falls, followed by motor vehicle accident, bicycle accident, assault. motorcycle accident, and sports. However, the major cause of lethal head injury was motor vehicle accidents. The mortality for severe head injury (GCS \leq 8) was 29%, and the likelihood of death resulting from severe head injury generally fell throughout childhood. The mortality from moderate head injury (GCS GCS 9-12) was 1.4% and generally rose throughout childhood. No child with a mild head injury (GCS 13-15) died in this series. The occurrence of subdural hematoma fell throughout childhood, but the mortality was high at 40%. The occurrence of epidural hematoma rose throughout childhood but the mortality from this lesion was only 4%. Although pupillary abnormalities were a poor prognostic sign, only 532% of children with bilaterally fixed pupils died. Profound hypotension and the presence of chest or abdominal injury resulted in higher mortality. However, profound hypertension did not. The relationship of these factors to the severity of neurological injury will be discussed.

(This study was supported by Contracts N01-NS-9-2312, 2313, and 2314B of the National Institute of Neurological and Communicative Disorders and Stroke.)

47. CT AND MRI FEATURES OF CRANIOPHARYNGIOMAS IN CHILDREN AND ADULTS

Robert B. Snow, Robert D. Zimmerman, Richard D. Becker, Steven Albert (New York, NY)

Craniopharyngiomas account for 5-13% of childhood CNS tumors. They are less frequent, but not uncommon, in adults. Some investigators have separated adult and childhood craniopharyngiomas into two separate categories based on differences in clinical behavior and pathology. The CT features of craniopharyngiomas, irrespective of age, have been described as consisting of the triad of calcifications, cysts and contrast enhancement. The prognosis of these tumors differs between adults and children as well, with the adult form often acting as a bilogically indolent lesion with less predilection for post-operative recurrence.

In order to determine if there is a difference radiographically between craniopharyngiomas in adults and children, we studied the CT and MRI appearances of 25 adults and 10 children (<20 years of age) with these tumors.

We found there were two distinct types of tumors radiographically. In one group, the majority of patients were children and recurrence was high. Adults in this radiographic group also demonstrated a higher rate of recurrence. In the second group, the majority of patients were adults and the surgical cure rate was high.

The importance of these findings are that dividing craniopharyngiomas into "adult" and "childhood" -- type patterns on the basis of CT and MRI criteria, rather than on the basis of chronologic age, may be more accurate in determining prognosis.

48. NEUROBEHAVIORAL SYNDROMES FOLLOWING TRANSLAMINA TERMINALIS RESECTION OF LARGE CRANIOPHARYNGIOMAS IN CHILDREN

Dachling Pang, Christopher Ryan, Ellen Ormond Hesky, Paul Polinko (Pittsburgh, PA)

We studied the psychological profiles, IQ, memory, frontal lobe function, and psychosocial adjustment of 15 children, had undergone total excision craniopharyngiomas via the translamina terminalis route. Serial standard psychometric tests and detailed parental interviews were administered over a period of $1\frac{1}{2}$ to 5 years following surgery. Although all 15 children were classified as "neurologically normal" in that they were alert and had no language or sensorimotor deficits, several consistent neurobehavioral aberrations were noted when they re-entered their habitutal environments: (1) abulia minor -- 4 children showed mild degrees and 2 others showed moderate degrees of psychomotor hypoactivity and intertia, loss of motivation, enthusiasm and exploratory behaviors, cognitive slowing, and emotional flatness. (2) Hyperphagia -- 4 children had transient hyperphagia and no obesity; 2 had permanent but mild hyperphagia and moderate obesity that was controlled by dieting; 2 others had permanent hyperphagia and "uncontrolled" obesity. (3) Memory dysfunction-although all 15 children had normal IQ, 3 manifested significant recent-memory deficit and learning disability. The children with the worst abulia also had the most severe hyperphagia and memory dysfunction. suggesting a high correlation in the severity of involvement in the 3 syndromes. The composite psychobehavioral profile of the severely affected describes an obese child who overeats, refuses to participate in play or work, lacks enthusiasm and exploratory activities, avoids old and new friends, lacks initiative, impulsivity and emotional variability, performs poorly at school because of low motivation and memory problems, and feels all the more inadequate for it.

48. NEUROBEHAVIORAL SYNDROMES FOLLOWING TRANSLAMINA TERMINALIS RESECTION OF LARGE CRANIOPHARYNGIOMAS IN CHILDREN (Cont'd)

The abulia may be caused by injury to the dopaminerqic mesolimbic facilitator bundle as it passes through the medical septal region, or to the nucleus basalis of Neynert likely related to retractor pressure against the anterior perforated substance with deep subfrontal exposure. The hyperphagia may be caused by injury to the serotoniergic feeding-inhibitory pathway in the anterior medial hypothalamus; and the amnestic syndrome may be related to dissection around the mamillary bodies or the mediobasal forebrain nuclei. Other than these pathophysiological mechanisms, psychosocial dysfunction is also attributed to peer rejection, radical changes in body image following surgery, feelings of inadequacy, and parental psychopathology. The etiology prevention and management of these neurobehavioral complications following third ventricular operations are discussed.

49. GLIOMA ARISING IN THE FIELD OF RADIATION THERAPY FOR CRANIOPHARYNGIOMA

John Shillito, Jr. (Boston, MA)

Two cases have occurred within the past six months. The first is a boy radiated 20 years ago for craniopharyngioma explored and found inoperable. He developed first some cranial nerve signs and then a hemiparesis. CT and MRI studies indicated an intrinsic lesion in the brain stem and thalamus. Biopsy revealed active craniopharyngioma just outside of these areas but no tumor from the thalamus could be obtained. However, his neurological symptoms were reversed completely by radiation therapy to the areas of the presumed intrinsic tumor.

The second case was a patient radiated 8 years ago for a small craniopharyngioma fixed to the under surface of her chiasm which was explored but could not be excised. She developed a very similar neurological problem with hemiparesis and multiple cranial nerve deficits. CT and MRI studies showed a picture consistent with an intrinsic glioma of the brain stem. Radiation therapy to this area improved her but briefly. She died several months after the symptoms began. Autopsy was refused but a needle biopsy of the area was permitted. This showed tissue consistent with a malignant glioma of the brain stem and cerebellar structures.

50. PITUITARY TUMORS IN CHILDREN: ENDOCRINOLOGIC, RADIOLOGIC, AND PATHOLOGIC FINDINGS RELATED TO PITUITARY STALK SIZE

Kerry R. Crone, Thomas S. Berger, John M. Tew (Cincinnati, OH)

Pituitary tumors are uncommon in childhood. Since 1983, eight children have undergone surgical treatment (six transsphenoidal - two transcranial) for pituitary lesions. High resolution computed tomography was used to compare the size, shape, density, and post-contrast enchancement characteristics of the pituitary stalk and gland to the preoperative endocrine assessment. Measurement of the pituitary stalk (PS) to the basilar artery (BA) was used to determine the relative stalk size, PS/BA ratio.

Findings included five adenomas (four secreting, one non-secreting), one pituitary cyst, one germinoma, and one infiltrating inflammatory lymphoid process. Although size, shape, and density failed to distinguish the pathologic process, rim enchancement was observed in two of the four secreting tumors. The PS/BA ratio was less than one in all intrinsic pituitary gland abnormalities (five adenomas, one cyst) but markedly increased when extrinsic processes (germinoma, lymphoid infiltration) were present. Preoperative endocrine assessment revealed panhypopituitarism and diabetes insipidus in extrinsic conditions but absent or single hormonal abnormalities in intrinsic condition.

These preliminary findings suggest that elevated PS/BA ratios may be associated with extrinsic infiltrative lesions and best approached through transcranial procedures whereas ratios less than one are more likely to be associated with intrinsic gland lesions and best treated through the transsphenoidal route.

51. CHOROID PLEXUS PAPILLOMAS IN NEONATES, INFANTS AND CHILDREN

Tadonori Tomita, David G. McLone (Chicago, IL)

During the past 15 years, 20 patients with choroid plexus papillomas were diagnosed and treated. There were 15 males and 5 females. Ages at diagnosis ranged from 1 day to 14 years: 12 were treated during the first year of life and 3 during the second year of life. The location of choroid plexus papillomas were, the lateral ventricle in 13 (5 left, 7 right, 1 bilateral), the third ventricle in 6 and the IV ventricle in one. Angiography was performed in all patients and disclosed abnormal vascularity in the tumor. Computed tomography was obtained in 12 more recent patients and all showed enhancing masses. Total resection of papilloma was done in 19, but 1 patient died intraoperatively due to hemorrhage while undergoing resection of the third ventricular choroid plexus papilloma. Surgical mortality rate was 5%. Two other patients died 9 years and 10 years after surgery; one with malignant papilloma seeding in the subarachnoid space and another with sepsis (not related to the tumor). All other patients are alive and well except 2 patients with seizure disorder and mental retardation. In conclusion, choroid plexus papillomas are successfully removed with acceptable risks despite patients' young age and tumors' severe vascularity. Applied surgical techniques are also discussed.

52. PEDIATRIC DIENCEPHALIC GLIOMAS - A REVIEW OF 18 CASES

Eric W. Scott, J. Parker Mickle (Gainesville, FL)

Disagreement exists regarding the natural history and appropriate management of pediatric diencephalic gliomas (PDG). Eighteen cases of histologically verified PDG were analyzed retrospectively. Mean age was 8.3 years at presentation with a mean follow-up of $3\frac{1}{2}$ years.

The most common presentations were associated with increased intracranial pressure, (44%). Visual disturbances, though evident in most, were rarely the presenting complaint. Motor dysfunction/seizures were frequent in tumors involving the thalamus. One patient presented with precocious puberty, one with diabetes insipidus and two with the classical "diencephalic syndrome." Two children displayed evidence of neurofibromatosis. CT demonstrated mass lesions in all cases and hydrocephalus in 50%. Angiography, performed in 13 patients, was helpful in predicting tumor histology.

Sixteen patients underwent craniotomy with biopsy in 11 and subtotal resection in 5; two underwent sterotactic biopsy. Twelve patients required shunting for hydrocephalus and further debulking procedures were necessary in 3. Tumors involved the thalamus (39%). hypothalamus (61%) and optic chiasm (44%). Histology included 14 low grade astrocytomas, 1 mixed, low grade astrocytoma-oligodendroglioma and 3 high grade astrocytomas. Initial treatment included radiation therapy (RT) in 14 and observation in 3. Five patients received chemotherapy subsequent to RT. Disease remission, defined as clinical improvement or stabilization with radiological evidence of decrease in tumor size, was documented in 50% of patients receiving RT and in two untreated children. There were 4 deaths from tumor progression and 1 from surgical complications. Three year actuarial survival was 87% but fell to 67% at 5 years.

We conclude that: (1) diagnostic biopsy is necessary in cases of diencephalic tumors, (2) thalamic involvement portends a poor prognosis both in terms of histology and survival, (3) beneficial effects of RT are difficult to demonstrate and (4) therapy for PDG must be individualized and long-term spontaneous remissions may occur.

53. FACTORS AFFECTING SURVIVAL IN MALIGNANT GLIOMA OF CHILDREN

Ossama Al-Mefty, Naef R. Al-Rodhan, John L. Fox, Andrew D. Parent (Jackson, MS)

Forty-four patients (23 males and 21 females) aged 2 to 20 years (mean 9.6), harboring pathologically proven malignant glioma (grade III and IV) were treated between 1976 and 1985. Tumor sites included the cerebral hemisphere (26 patients), thalamus (6), brain stem (7), and the cerebellum (5). All patients had surgery and initial treatment with steroids. Irradiation was given in 35 patients, 3 had adjuvant chemotherapy. At the time of study, 22 were deceased. The longest period of follow-up was 65 months. Data was analyzed using the Kaplan-Meier method. The difference between mean survival times was evaluated using the Mantel-Cox Test. Survival curves were calculated starting from date of first visit to date of last evaluation or death. Mean survival times were as follows: 30 months for all patients; for cerebellar cases (14), brain stem (17), thalamic cases (26), cerebral hemisphere (33). However, the difference between mean survial times was not statistically significant. Patient's age was the single most significant factor, with those 5 to 10 years old having the worst survival curve (P=0.0036). Otherwise, there was no clinical or radiological factor which indicated a better prognosis. This was also confirmed by chi square analyses comparing 72 factors among patients with more than 24 months of survival and those with less than 24 months of survival. In particular, tumor size (greater or less than 5 cm), neurological deficits, level of consciousness, increased intracranial pressure, hydrocephalus, calcium or cyst on CT scan, and histological grading (III or IV) showed no significant correlations.

54. HEAD "LUMPS" OF INFANTS AND CHILDREN

John R. Ruge, Tadanori Tomita, Thomas P. Naidich, Yoon S. Hahn, David G. McLone (Chicago, IL)

Single, nontraumatic raised masses or "lumps" on the head may be benign, local scalp lesions or the external tips of large intracranial masses. Seventy histologically-confirmed cases were analyzed. Seventy-one percent were congenital lesions including dermoids, 13% were neoplasms and 3% were inflammatory lesions. Specifically there were 43 dermoid lesions (61%), 6 calcified cephalohematomas (9%), 5 granulomas (7%), eosinophilic meningoencephaloceles (4%), 2 each of hamartomas and capillary hemangiomas and I each of a wide variety of inflammatory and neoplastic processes. Of the 70 lesions, 11 (16%) did have significant intracranial extension. In location the lesions were predominantly parietal (33%), occipital (19%), periorbital (19%), frontal (13%), anterior fontanelle (10%) and temporal (7%). Fifty-eight percent had definite association with a suture; these were predominantly dermoids. Neuroradiologic studies localized the lesion correctly in 69 cases (99%). Neuroradiology suggested the correct histologic diagnosis in 43%, the wrong diagnosis in 11% and provided only a nonspecific list of possible differential diagnosis in 46%. These data provide a rational basis for assessing the significance of single, nontraumatic scalp lesions in the child.

55. RADIATION-INDUCED MENINGIOMAS PRESENTING IN THE PEDIATRIC PATIENT

S. David Moss, Gaylan Rockswold, Douglas Yock, Edward L. Seljeskog (Minneapolis, MN)

Radiation-induced meningiomas rarely have latency periods short enough to present in the pediatric patient. Two cases of radiation-induced intracranial meningiomas are presented. The first is a case of meningioma of the left frontal region in a 102-year-old male, six years following resection and radiation of the IVth ventricular medulloblastoma. Review of our pediatric tumors has produced a second case of left temporal fossa meningioma presenting in a 15-year-old male with a history of radiation for retinoblastoma at age 3. Three cases of meningiomas presenting in the pediatric age group following radiation therapy to the head have been reported in the literature. Our cases represent the first reported cases following radiation for medulloblastoma or retinoblastoma to present in a pediatric patient. Radiation-induced meningiomas differ between low-dose and high-dose radiation, being more aggressive and having a shorter latency in the later group. In general, post-irradiation meningiomas have a different set of distinguishing features which include latency, histologic pathology, multiplicity, location and recurrence rates. These tumors have shorter latency, have greater multiplicity, a more aggressive character and higher rate of recurrence. The literature is reviewed and our cases are presented.

56. SENSITIVITY OF PNET DERIVED CELL LINES TO IN VITRO CYTOLYSIS BY LYMPHOKINE-ACTIVATED KILLER CELLS

Richard E. George, Richard P. Moser (Houston, TX)

Primitive neuroectodermal tumors (medulloblastoma) of the posterior fossa (PNET-PF) comprise 20% of pediatric brain tumors. These tumors frequently metastasize via the CSF pathways to the spinal canal. Craniospinal irradiation and chemotherapeutic regimens are currently utilized to treat these metastases. Both techniques have a significant morbidity. Lymphokine-activated killer (LAK) cells have recently been generated by stimulating peripheral blood interleukin-2 (IL-2). Adoptive lymphocytes with immunotherapy with LAK cells and IL-2 has been shown to have efficacy in the treatment of metastasic solid tumors in humans. The current study was undertaken to determine whether PNET-PF derived cells would be sensitive to LAK killing. Four early passage PNET-PF derived cell lines were tested for sensitivity to allogenic LAK killing in an in vitro four hour 51Cr release assay. All PNET-PF demonstrated significant sensitivity to LAK killing (table 1). Fresh glial cells isolated from normal brain were not lysed by LAK cells. These results suggest that LAK cytolysis may be of possible therapeutic benefit. Implications for primary adjuvant therapy and treatment of leptomeningeal tumor dissemination are discussed.

Table 1. Percent specific cytolysis of PNET derived cells lines.

CELL	CONTROL LAK		EFFECTOR: TARGET CELL RATIO				
LINE	SUPERNA	ΓE	PBL.			LAK	
		1:1	1:10	1:100	1:1	1:10	1:100
RB	-3.0±3.8*	4.9±11.0	5.8±2.0	16.1± 6.7	21.2±7.7	62.6±6.4	77.5±3.6
NQ	4.1±5.3	-2.0 12.8	28.6±2.3	48.0 ± 14.8	42.7±9.2	80.7±6.7	84.2±4.8
CW	3.3±3.4	ND	ND	ND	34.l±3.6	67,7±2.8	69.9±7.8
SN	-0.5 ± 4.3	-2.7±5.0	-4.0±1.7	0.2± 5.4	2.7±7.7	20.2 \$ 8.8	38.5±5.7

PBL=peripheral blood lymphocyte; LAK=lymphokine-activated killer cell; ND=not done

^{*} percent specific cytolysis *percent standard deviation.

57. BROMODEOXYURIDINE AS A PHOTOSENSITIZER AND A RADIOSENSITIZER

Corey Raffel, Michael S.B. Edwards, Dennis F. Deen (San Francisco, CA)

Bromodeoxyuridine, a thymidine anolog incorporated into dividing cells, has recently been used in clinical trials in patients with malignant gliomas. Besides its radiosensitizing properties, the drug also sensitizes cells to ultraviolet light (UV). We have compared these two properties of BrdUrd in the rat 9L gliosarcoma model in vitro. When 9L cells are treated with 400 rads of X-rays or 18 j/m2 of UV, one log of cells are killed (isoeffective doses). When 9L cells are treated with BrdUrd prior to X-irradiation, the cell kill is increased by a factor of two: UV irradiation after treatment with BrdUrd kills 100 times more cells than UV alone. X-rays alone cause single strand breaks in the DNA of treated cells. When BrdUrd pretreatment was combined with X-irradiation, the number of breaks increased; the breaks were uniformly distributed throughout the DNA molecules. No DNA-protein crosslinks were formed. UV alone caused no single strand breaks in the DNA, but when combined with the BrdUrd pretreatment, many such breaks were formed. DNA-protein crosslinks were also detected. Repair studies indicated that the cells were much more efficient at removing the single strand breaks than the protein crosslinks from their DNA. These results suggest that BrdUrd is a far more potent photosensitizer than a radiosensitizer. If patients were treated with the drug prior to surgical resection of their tumor, and the tumor cavity treated with UV after the resection, the photosensitizing properties of BrdUrd could be exploited. Any drug remaining would still function as a radiosensitizer. In vitro experiments are in progress to determine the effectiveness of this treatment.

58, CEREBELLAR ASTROCYTOMA IN CHILDHOOD

Masaharu Yasue, Tadanori Tomita, David G. McLone (Chicago, IL)

Cerebellar astrocytomas are the most favorable among pediatric brain tumors. However, various problems are encountered in treatment: (1) is total resection necessary? (2) if not totally resected, will tumor recur? (3) are there any similiarities in the fashion of the tumor recurrence? (4) does hydrocephalus require a shunt procedure? This report analyzes these clinical problems according to clinical, radiological and neuropathological data.

During the past 15 years, 57 patients (29 males, 28 females) were treated for cerebellar astrocytoma. Twenty-four were located in the midline and 33 in the cerebellar hemisphere. All patients underwent a posterior fossa craniotomy and 14 patients required multiple craniotomies. Total resection was achieved in 37 patients at the primary craniotomy and in additional 9 patients, the tumor was resected at the 2nd and 3rd craniotomy. (Total resection - 46 patients.) The main reason for inability to achieve total resection was the invasion of the tumor into the cerebellar peduncles or the floor of the 4th ventricle. Pathological distribution showed 52 were grade I-II, 4 grade III and 1 gemistocytic astrocytoma, CT scans were obtained in 37 patients and were classified into 3 types: Type 1, mural nodule type: Type 2, ring enhance type: Type 3, solid type. All the cases with recurrence were type 2 and 3. Two patients died shortly after the primary craniotomy. Five patients died thereafter. The cause of death in 3 patients with benign astrocytoma was shunt malfunction, and in the other 2 patients, were recurrence of malignant astrocytoma.

In conclusion, cerebellar astrocytomas should be totally resected whenever possible. The residual tumors, particularly those of solid type astrocytoma, tend to recur despite the benign histological appearance.

59. CEREBELLAR ASTROCYTOMAS: THE SIGNIFICANCE OF THE CONTRAST ENHANCING CYST WALL

Mitchel S. Berger, Harold Hoffman, Robin Humphreys, E. Bruce Hendrick (Seattle, WA)

A series of pediatric patients diagnosed within the CT era (1975-1986) as having cerebellar astrocytomas, were retrospectively analyzed to determine what prognostic significance the contrast enchancing cyst wall has, and how it should be surgically managed.

Following intravenous contrast administration, 6 distinct CT patterns were identified in this group of 42 patients: 1) Solid enhancing tumor (19%); 2) primarily solid enhancing tumor with associated intratumoral cysts (22%); 3) irregular thick walled enhancing cyst without a mural nodule (17%); 4) enhancing thin walled cyst without a mural nodule (5%); 5) enhancing cyst wall with an enhancing mural nodule 12%); 6) non-enhancing cyst wall with an enhancing mural nodule (26%).

No patient, regardless of the CT pattern, has developed tumor recurrence, when all enhancing tissue was completely removed (29/42)p; (mean postoperative time, 52 months). Of the 13 patients who underwent a subtotal resection or biopsy, 8 developed recurrent tumor (mean time to recurrence, 19 months). Ten of 14 patients with enhancing cysts, i.e. irregular thick walled, thin walled, and those cysts associated with a mural nodule, had a gross total resection (GTR) of all enhancing tissue: no recurrences developed (mean postoperative time, 48 months). The remaining patients in this group underwent a subtotal resection or biopsy and all 4 developed recurrent tumor (mean time to recurrence, 17 months). Pathological analysis of the enhancing cyst wall demonstrated tumor in each case.

A complete resection of all CT enhancing tissue, including the cyst wall, is recommended to avoid recurrence of cerebellar astrocytomas, unless brainstem involvement is confirmed at the time of surgery. The non-CT enhancing cyst wall does not require excision and should be left intact.

60. POST-OPERATIVE MYELOGRAPHY FOR THE EVALUATION OF MALIGNANT PEDIATRIC POSTERIOR FOSSA TUMORS

Ann M. Flannery, Tadanori Tomita, Mary Ann Radkowski, David G. McLone (Chicago, IL)

The propensity of posterior fossa tumors of primitive cell origin to metastasize into the spinal subarachnoid space has been well documented. Since 1983, all children seen at the Children's Memorial Hospital for management of posterior fossa ependymomas and medulloblastomas have been evaluated with both post-operative myelography and CSF cytological studies. A CT of the spine following a myelography was used in an attempt to increase sensitivity. Nineteen patients were evaluated under this protocol, twelve with medulloblastomas (five males, seven females), seven had ependymomas (two males, five females). The patient age varied from six weeks to 15 years at the time of initial presentation. Length of follow-up ranges from six to thirty months.

In the medulloblastoma group, give (41.6%) had cytological evidence of tumor. Two of 12, or 17% had myelograms which were initially positive for metastatic disease, while three had cytology positive for malignant cells and no evidence of disease on myelogram. Initial studies were normal in the remaining 58% of medulloblastoma patients. Ependymoma patients (7) had one myelogram which was positive for both cytological and visible tumor.

In the pediatric age group, for these tumors with a known propensity to metastasize, cytology may be more sensitive than myelography. Our incidence of positive myelograms is lower than previously reported in the literature, and tumor cells are detected when the myelogram shows visible disease. We will also discuss the tendency for tumor cells to disappear with radiation and chemotherapy.

61. MEDULLOBLASTOMA IN CHILDREN: FACTORS DETERMINING SURVIVAL

John M. McGregor, Edward J. Kosnik (Columbus, OH)

One hundred thirty-three children have been diagnosed and treated for medulloblastoma at Columbus Children's Hospital. To date, there are 35 survivors with length of survival ranging from 1 month to 25 years.

We will review our series in terms of clinical features, surgical resection, pathology, postoperative therapy and survival.

In addition, we will attempt to distinguish the significant factors that have determined the survival of these 35 patients and what the long-term effects of their disease and treatment have been.

SECTION OF

PEDIATRIC NEUROLOGICAL SURGERY



MEMBERSHIP ROSTER

1986

Albright, A Leland Childrens Hosp Of Pgh Pittsburgh PA 15213 412/647-5090 Active

Alexander Jr, Eben Bowman Gray Sch Of Med Winston-Salem NC 27103 919/748-4082 Active

Altenau, Lance 4060 Fourth Avenue Ste 610 San Diego CA 92103 Active

Amacher, A Loren 85 Jefferson St Hartford CT 06106 203/246-6465 Active

Anderson, Frank M 2101 Redcliff St Los Angeles CA 90039 213/661-7845 Lifetime Active

Arkins, Thomas J Neurosurgical Associates 60 Temple Street New Haven CT 06510 203/789-2030 Active

Aronin, Patricia A 1600 Seventh Ave S Birmingham AL 35233 205/939-9653 Candidate

Arpin, Elaine M 1328 Navajo Court Louisville KY 40207 502/588-5433 Active Bailey, Walter L 280 N Smith Ave St Paul MN 55102 Active

Bartkowski, Henry M Neurological Associates 931 Chatham In Columbus OH 43221 614/457-4880 Active

Becker, Donald Paul Ucla Med Ctr D/Neurosurg Rm/74-140 Chs 405 Hilgard Los Angeles CA 90024 213/825-5111 Active

Berger, Thomas S 506 Oak St Cincinnati OH 45219 513/221-1100 Active

Bering Jr, Edgar A Creek House Oxford MD 21654 301/822-5480 Lifetime Active

Bressler, Bruce C 704 S Webster Ave Green Bay WI 54301 414/465-1900 Active

Bruce, Derek A 3115 Cornell Ave Dallas TX 75205 Active

Buchheit, William A Temple Univ Health Ctr 3401 N Broad St Philadelphia PA 19140 215/221-4068 Active Canady, Alexa Irene 4160 John R Ste 1031 Detroit MI 48201 313/833-4490 Active

Carmel, Peter W
Neurological Institute
710 W 168Th St
New York NY 10032
212/305-5208
Active

Chapman, Paul H
Massachusetts Gen Hosp
Fruit St
Boston MA 02114
617/726-3887
Active

Cheek, William R
Texas Children Hosp
Rm 0-202
HCuston TX 77030
713/790-9053
Active

Coulon Jr, Richard A 311 Midway Dr River Ridge LA 70123 504/834-7070 Active

Crosby, Robert M N 1205 York Rd Lutherville MD 21093 301/823-1300 Active

Davidson, Robin Ian Univ Of Mass Med Ctr 55 Lake AVe N Worcester MA 01605 617/856-3079 Active

Dorsen, Michael 711 W 38Th St Building B, Ste B4-A Austin TX 78705 Active

Alphabetical by Name

Duncan, Charles Cecil Yale Univ Sch Of Med Neurosurgery Section New Haven CT 06511 203/785-2805 Active

Edwards, Michael Univ Of California Dept Of Neurosurgery/M787 San Francisco CA 94143 415/666-1087 Active

Eisenberg, Howard M Univ Of Texas Med Branch Div Of Neurosurgery Calveston TX 77550 409/761~1500 Active

Eisenbrey, Arthur B 4160 John Rd Ste 1031 Detroit MI 48201 313/833-4490 Active

Epstein, Mel H Rhode Island Hosp/Neuros 110 Lockwood Stret Providence RI 02903 Active

Fell, David A 6767 S Yale Tulsa OK 74136 918/492-7587 Active

Fischer, Edwin G Childrens Hosp Med Ctr Boston MA 02115 617/735-6009 Active

Foltz, Eldon L U C I Medical Center 101 City Dr S Orange CA 92668 714/634-5775 Active French, Barry Norman 2801 K Street #300 Sacramento CA 95816 916/452-4811 Active

Gahm, Norman Henry 100 Retreat Ave Ste 705 Hartford CT 06106 203/278-0070 Active

Gainsburg, Duane B Dept Of Neurosurgery Soroka Medical Center Beersheva ISRAEL Active Foreign

Galicich, Joseph H Memorial Hosp 1275 York Ave New York NY 10021 212/988-1629 Active

Gallo Jr, Anthony E 3131 Sw Sam Jackson Pk Rd Portland OR 97201 503/225-7736 Active

Gamache Jr, Francis W 525 E 68 St Rm K-6 New York NY 10021 212/472-4909 Active

Glasauer, Franz E Suny At Buffalo Clin Ctr Dept Of Neurosurgery Buffalo NY 14215 716/894-2065 Active

Guido, Laurance Jacobius 340 Capitol Ave Bridgeport CT 06606 Active Gutierrez, Francisco A 707 N Fairbanks Ct Ste 914 Chicago IL 60611 312/256-5039 Active

Hahn, Joseph F Cleveland Clinic 9500 Euclid Ave Cleveland OH 44106 216/444-5753 Active

Haines, Stephen J Box 96 Mayo 420 Delaware St S E Minneapolis MN 55455 612/373-8785 Active

Hammargren, Lonnie 3196 S Maryland Pkwy Las Vegas NV 89109 702/735-7272 Active

Hammock, Mary Kathryn 111 Michigan N W Washington DC 20010 202/745-3020 Active

Hanigan, William C 214 N E Glen Oak Ste 500 Peoria IL 61603 309/676~0766 Active

Harwood-Nash, Derek C Hosp For Sick Children Neuro Padiologist Toronto ON 00000 416/598-6026 Associate

Hawk, Thomas J 3545 Clentangy River Rd Columbus OH 43214 614/268-5655 Active

Hawkins III, John C 1871 Montgomery Place Jacksonville FL 32205 904/388-6516 Active

Hemmy, David C 8700 W Wisconsin Ave Milwaukee WI 53226 414/257-4846 Active

Hendee Jr, Robert W 1010 E 19Th Ave Ste 605 Tammen Denver CO 80218 303/861-4985 Active

Hendrick, E Bruce
Hosp For Sick Children
555 Univ Ave/Ste 1502 A
Toronto ON 00000
416/597-0808
Active

Hoffman, Harold J Hosp For Sick Children 555 University Ave Toronto ON 00000 416/598-1210 Active

Hollenberg, Robert D Mc Master Univ Medical Ct Dept Of Surgery / Rm 4U4 Hamilton ON 00000 416/521-2100 Active

Hudson, Alan Roy Division Of Neurosurgery 38 Shuter St Toronto CN 00000 416/864-5588 Active

Humphreys, Robin P Hosp For Sick Children 555 Univ Ave/Ste 1504 Toronto CN 00000 416/598-6427 Active James, Hector E 225 Dickinson St H-893 San Diego CA 92103 619/294-5540 Active

Jerva, Michael Joseph 55 E Washington St Ste 3005 Chicago IL 60602 312/236-0668 Active

Johnson, Dennis I. Childrens Hosp Ntl M C 111 Michigan Avenue Washington DC 20010 Active

Johnson, Martin 2800 N Vancouver Ste 106 Portland OR 97227 503/287-2646 Active

Johnson, Mary M 5675 Peachtree Dunwcody Ste 307B Atlanta GA 30342 503/256-4510 Candidate

Joseph, Allen S 2237 S Acadian Ste 400 Baton Rouge LA 70808 504/928-5972 Active

Kasoff, Samuel S New York Med Col/Neuros Muger Pavillion Valhalla NY 10595 Active

Keener, Ellis B 434 Academy St N E Gainesville GA 30501 504/292-4612 Active Kelly Jr, David L Bowman Gray Sch Of Med Section Of Neurosurgery Winston-Salem NC 27103 919/748-4049 Active

Klein, David Mendel Dept Of Neurosurgery 219 Bryant St Buffalo NY 14222 716/878-7386 Active

Kosnik, Edward J Chatham Vlg Prof Bldg 931 Chatham In Columbus OH 43221 614/457-4880 Active

Kramer, Paul William 4640 N Federal Hwy Ft Lauderdale FL 33308 305/772-0100 Active

Laurent, John P Texas Childrens Hosp Ste 0-202 Houston TX 77030 713/790-9053 Active

Laws, Edward R Mayo Clinic Dept Of Neurosurgery Rochester MN 55901 507/284-2611 Active

Loeser, John D Univ Of Washington Dept Of Neuro R1-20 Seattle WA 98195 206/543-3574 Active

Iongo-Cordero, Rafael Ashford Medical Bidg 604 Santurce PR 00907 809/723-9075 Active

Alphabetical by Name

Magid, Gail A 1661 Soquel Dr Santa Cruz CA 95065 408/476-8900 Active

Marlin, Arthur E 343 W Houston San Antonio TX 78205 512/224-1631 Active

Mawk, John Robert Univ Of Neb Med Ctr Forty-Second And Dewey Omaha NE 68105 402/559-4301 Active

Mc callum, Jack E 1522 Cooper St Fort Worth TX 76104 Active

Mc comb, J Gordon 1300 N Vermont Ave Ste 903 Los Angeles CA 90027 213/663-8128 Active

Mc cullough, David C 111 Michigan Ave N W Washington DC 20010 202/745-3020 Active

Mc lanahan, C Scott Charlotte Neuros Assoc 1010 Edgehill Road N Charlotte NC 28207 704/376-1605 Active

Mc laurin, Robert L 1111 Wellington Pl Cincinnati OH 45219 513/381-6111 Active Mc lone, David Gordon 2300 Childrens Plaza Chicago IL 60614 312/649-4373 Active

Meacham, William F Vanderbilt Univ Hosp Dept Of Neurol Surgery Nashville TN 37232 615/322-3343 Senior

Mealey Jr, John Indiana Univ Med Ctr 545 Barnhill/Emerson 139 Indianapolis IN 46223 317/264-8549 Active

Menezes, Arnold H University Hospitals Dept Of Neurosurgery Icwa City IA 52242 319/356-2768 Active

Meyer, Glenn A 8700 W Wisconsin Milwaukee WI 53226 414/257-6465 Active

Michelsen, Jost J Box 148 Back Bay Annex Boston MA 02117 617/536-0028 Senior

Michelsen, W Jost 161 Fort Washington Ave New York NY 10032 212/305-5451 Active

Milhorat, Thomas H Suny Downstate Med Ctr Neurosurgery, Box 1189 Brocklyn NY 11203 212/270-2111 Active Miller, Clinton F Seaccast Neuroscience Grp 745 Central Ave Dover NH 03824 504/891-2663 Active

Minella, Philip Arthur 146 Wyoming St Dayton OH 45409 513/228-5261 Active

Moiel, Richard H 7400 Fannin St 1160 One Fannin Bldg Houston TX 77054 713/797-1160 Active

Morrison, Glenn 4685 Fonce De Leon Blvd Coral Gables FL 33146 305/661-5318 Active

Moyes, Peter D 5326 Angus Dr Vancouver BC 00000 604/266-4881 Lifetime Active

Murtagh, Frederick Univ Of Pennsylvania Hosp 3400 Spruce St Philadelphia PA 19104 215/662-3487 Active

Nadell, Joseph M Dept Of Neurosurgery 1430 Tulane Ave New Orleans LA 70112 504/588-5565 Active

Nijensohn, Daniel E 340 Capitol Ave Bridgeport CT 06606 203/367-4433 Active

Nishioka, Hiro 704 S Webster Ave Green Bay WI 54301 414/465-1900 Active

Nugent, G Robert W Virginia Univ Med Ctr Div Of Neurosurgery Morgantown WV 26506 304/293-5041 Active

Nulsen, Frank E 32 Tenth Ave South Naples FL 33940 216/844-3004 Lifetime Active

O'Brien, Mark Stephen Emory Univ Clinic 1365 Clifton Rd N E Atlanta GA 30322 216/321-0111 Active

Oakes, W Jerry Box 3272 Duke Univ Med Ctr Durham NC 27710 919/684-5013 Active

Page, Larry Keith 1501 N W Ninth Ave Miami FL 33136 305/547-6946 Active

Pang, Dachling Child Hosp Of Pittsburgh 125 Desoto St Pittsburgh PA 15213 412/647-5090 Active

Parent, Andrew D 1468 Mossline Dr Jackson MS 39211 601/987-5654 Active Parkinson, Dwight 5111-750 Bannatyne Ave Winnipeg MB 00000 204/943-1627 Lifetime Active

Penix, Jerry O'Don 607 Medical Tower Norfolk VA 23507 804/622-5325 Active

Pevehouse, Byron C 2351 Clay St Ste 314 San Francisco CA 94115 415/923-3616 Active

Pittman, Hal Watson Barrow Neuro Inst 2910 N 3Rd Ave Phcenix AZ 85013 602/285-3468 Active

Pitts, Frederick W 1245 Wilshire Blvd Ste 305 Los Angeles CA 90017 213/977-1102 Active

Portnoy, Harold D 1431 Woodward Ave Bloomfield H1 MI 48013 313/334-2568 Active

Pudenz, Robert H 574 Garfield Ave So Pasadena CA 91030 Senior

Raimondi, Anthony P O Box 14081 Chicago IL 60614 312/472-7355 Active Ranschoff, Joseph New York Univ Med Ctr 550 First Ave New York NY 10016 212/340-6414 Active

Reigel, Donald H 4815 Liberty Ave Ste 158 Pittsburgh PA 15224 412/682-0400 Active

Rekate, Harold Louis 2910 N Third Ave Phoenix AZ 85013 602/285-3632 Active

Reynolds Jr, Arden F Magan Medical Clinic 420 West Rowland St Covina CA 91723 Active

Rhoton Jr, Albert L Univ Of Florida H1th Ctr Dept Of Neuros / Box J265 Cainesville FL 32610 904/392-4331 Active

Robinson, Walker L 1205 York Rd Lutherville MD 21093 301/823-1300 Active

Salmon, James Henry Med Arts Bldg 225 W 25Th St / Ste 303 Erie PA 16502 814/453-5380 Active

Sanders, Morris 5959 Harry Hines Blvd Ste 620 Dallas TX 75235 214/637-0420 Active

Alphabetical by Name

Sanford, Robert Alexander Semmes-Murphy Clinic 920 Madison Ave / Ste 201 Memphis TN 38103 Active

Sato, Osamu

Dept Of Neurosurgery
Bo Sei D Ai Isehara
Kanagawa JAPAN
046/393-1121
Corresponding

Sayers, Martin P 2860 Canterbury Rd Columbus OH 43221 614/457-4880 Lifetime Active

Scarff, Timothy B 6584 Professional P1 Ste B Riverdale GA 30274 614/991-0150 Active

Schut, Luis Child Hosp Of Phila 34Th & Civic Ctr Blvd Philadelphia PA 19104 215/596-9368 Active

Scott, R Michael New England Med Ctr Boston MA 02111 617/956-5860 Active

Seljeskog, Edward L Univ Of Minn Hosp Dept Of Neuro Minneapolis MN 55455 612/373-8785 Active

Selker, Robert G Montefiore Hospital 3459 Fifth Ave Pittsburgh PA 15213 412/648-6753 Active Shallat, Ronald F 3000 Colby Berkeley CA 94705 408/843-0261 Active

Shapiro, Kenneth N Montefiore Medical Center 111 E 210 St Bronx NY 10467 212/920-4324 Active

Shillito, John 300 Longwood Ave Boston MA 02115 617/735-6012 Active

Simmons, James C H 920 Madison Ave Ste 201 Memphis TN 38103 901/525-8431 Active

Sklar, Frederick H 8226 Douglas Ave Douglas Plaza / Ste 627 Dallas TX 75225 214/361-2233 Active

Smith, Frank P 880 Cass St Ste 101 Monterey CA 93940 408/373-3762 Active

Stein, Sherman Charles 80 Congress St Springfield MA 01104 403/733-4153 Active

Steinbok, Paul G620 Shaughnessy Hospital 4500 Oak St Vancouver BC 00000 604/875-2094 Active Storrs, Bruce B Childrens Memorial Hosp. 2300 Childrens Plaza Chicago II 606.14 312/880-4373 Active

Sukoff, Michael 801 N Tustin Ste 406 Santa Ana CA 92705 714/834-1303 Active

Susen, Anthony F 3600 Forbes Ave Pittsburgh PA 15213 412/682-5900 Active

Sutton, Leslie N Childrens Hosp Of Phila Dept Of Neurosurgery Philadelphia PA 19104 Active

Taekman, Michael S 3000 Colby Berkeley CA 94705 415/843-0261 Active

Tew Jr, John M 506 Oak St Cincinnati OH 45219 513/221-1100 Active

Tomita, Tadanori Childrens Memorial Hosp 2300 N Childrens Plaza Chicago IL 60614 312/880-4373 Active

Venes, Joan U Of Mi Med Ctr/Neurosrgy C-5070 Outpatient Bldg Ann Arbor MI 48109 313/763~3524 Active

Ventureyra, Enr Cls Garcia 401 Smyth Rd Ottawa CN 00000 613/737-2316 Active

Vries, John Kenric Child Hosp Of Pittsburgh 125 Desoto St Pittsburgh PA 15213 412/647-5090 Active

Wald, Steven L University Health Ctr One S Prospect Burlington VT 05401 Active

Waldman, John B Albany Medical College Div Of Neurosurgery Albany NY 12208 Active

Walker, Marion I. 320 12Th Ave Primary Childrens Salt Lake Cty UT 84103 801/521-1209 Active

Walsh, John Willson Univ Of Kentucky Med Ctr Div Of Neurosurgery Lexington KY 40506 606/233-5862 Active

Waltz, Thomas A Scripps Clinic 10666 N Torrey Pines La Jolla CA 92037 Active

Ward, John D 1504 Harborough Rd Richmond VA 23233 513/257-4507 Active Weiss, Martin H U S C Medical Ctr Box 786 1200 N State St Los Angeles CA 90033 213/226 Active

Welch, W Keasley Childrens Hosp Med Ctr 300 Longwood Ave Boston MA 02115 617/735-6008 Active

White, Robert Joseph Metropolitan Gen Hosp 3395 Scranton Rd Cleveland OH 44109 216/459-4383 Active

Winston, Ken Rose 300 Longwood Ave Boston MA 02115 617/735-6011 Active

Yamada, Shokei Loma Linda U Schl Of Med Section Of Neurosurgery Loma Linda CA 92350 714/824-4417 Active