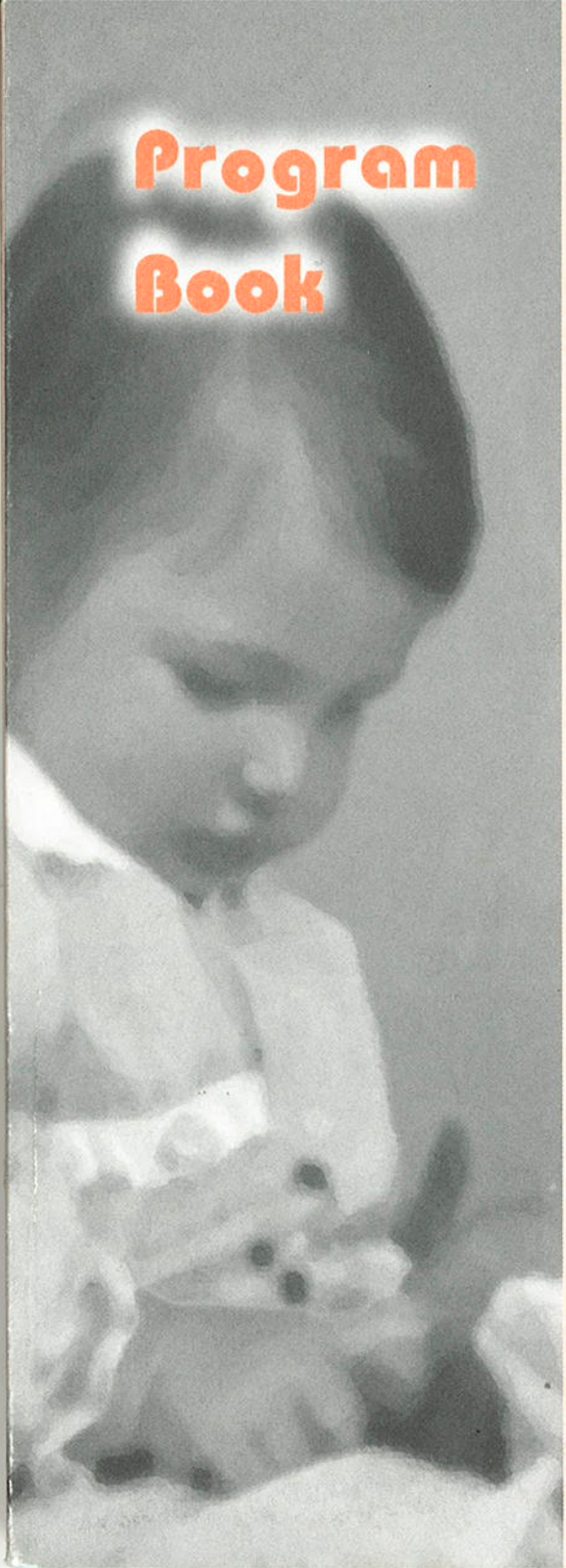


Program Book

The Section on Pediatric Neurological Surgery

of American Association of Neurological Surgeons & Congress of Neurological Surgeons

28TH Annual Meeting
December 1–4, 1999
Atlanta (Buckhead), Georgia

The AANS/CNS Section on Pediatric Neurological Surgery 28th Annual Meeting is jointly sponsored by American Association of Neurological Surgeons

The Section on Pediatric Neurological Surgery

of American Association of Neurological Surgeons &
Congress of Neurological Surgeons

28TH Annual Meeting
December 1–4, 1999
Atlanta (Buckhead), Georgia

The AANS/CNS Section on Pediatric Neurological Surgery 28th Annual Meeting is jointly sponsored by American Association of Neurological Surgeons

Table of Contents

Disclaimer 4

Continuing Medical Education Credit 4

Kenneth Shulman Award Recipients 5

Hydrocephalus Foundation Award Recipients 6

Pediatric Section Chairmen 6

Annual Winter Meeting Sites 7

Raimondi Lecturers 7

1999 Raimondi Lecturer 8

Floor Plan of the Exhibit Hall 9

Exhibitor Listing 10-11

Acknowledgements 12

About the AANS/CNS Section on Pediatric Neurological Surgery 13-14

Disclosure Information 15

Scientific Program 17-28

Poster Session 29-32

Scientific Program Oral Abstracts 33-80

Scientific Program Posters 81-103

1999 Membership Roster 105-121

Disclaimer

All drugs and medical devices used in the United States are administered in accordance with Food and Drug Administration (FDA) regulations. These regulations vary depending on the risks associated with the drug or medical devices compared to products already on the market, and the quality and scope of the clinical data available.

Some drugs and medical devices demonstrated or described on the print publications of the AANS/CNS Section on Pediatric Neurological Surgery have a FDA clearance for use for specific purposes or for use only in restricted research settings. The FDA has stated that it is the responsibility of the physician to determine the FDA status of each drug or device he or she wishes to use in compliance with applicable law.

Continuing Medical Education Credit

This activity has been planned and implemented in accordance with the Essentials and Standards of the Accreditation Council for Continuing Medical Education (ACCME) through the joint sponsorship of the American Association of Neurological Surgeons and the AANS/CNS Section on Pediatric Neurological Surgery. The Accreditation Council accredits the American Association of Neurological Surgeons to sponsor continuing medical education for physicians.

The American Association of Neurological Surgeons designates this continuing education activity for 21 credit hours (with attendance of Breakfast Seminars) in category 1 toward the AMA Physician's Recognition Award with an additional 1.5 hours available for residents/fellows attending the Lunch Seminar and an additional 5 credit hours available for those attending the Nurse Seminar. Each physician should claim only those hours that he/she actually spent in the educational activity.

Kenneth Shulman Award Recipients

1983 Kim Manwaring: Neonatal Post-Hemorrhagic Ventriculomegaly: Management with Pulsed Lumbar Cisternostomy

1984 Arno Fried: A Laboratory Model of Shunt-Dependent Hydrocephalus

1985 Anne-Christine Duhaime: The Shaken Baby Syndrome

1986 Robert E. Breeze: CSF Formation in Acute Ventriculitis

1987 Marc R. DelBigio: Shunt-Induced Reversal of Periventricular Pathology in Experimental Hydrocephalus

1988 Scott Falci: Rear Seat-Lap Belts. Are They Really "Safe" for Children?

1989 James M. Herman: Tethered Cord as a Cause of Scoliosis in Children with a Myelomeningocele

1990 Christopher D. Heffner: Basilar Pons Attracts Its Cortical Innervation by Chemotropic Induction of Collateral Branch Formation

1991 P. David Adelson: Reorganization of the Cortical-Tectal Pathway Following Neonatal Cerebral Hemispherectomy in Cats

1992 David Frim: Effects of Biologically Delivered Neurotrophins in Animal Models of Neural Degeneration

1993 Monica C. Wehby: Metabolic Demonstration of Retained CNS Function in the Rabbit Model of Infantile Hydrocephalus

1994 Ellen Shaver: Experimental Acute Subdural Hemotoma in Infant Piglets

1995 Seyed M. Emadian: Correlation of Chromosome 17p Loss with Clinical Outcome in Patients with Primitive Neuroectodermal Tumors

1996 John Park, MD, PhD: Platelet Derived Growth Factor Induces Differentiation of Neuroepithelial Stem Cells into Neurons

1997 Michael J. Drewek, MD: Quantitative Analysis of the Toxicity of Human Amniotic Fluid to Rat Fetal Spinal Cord Cultures

1998 Adriana Ranger: Implantation of Medulloblastoma Cells into Collagen Type I Gels: Invasiveness, Enzymatic Characterization, and the Impact of Surgical Excision and Radiation

Hydrocephalus Association Award Recipients

1989 Eric Altschuler: Management of Persistent Ventriculomegaly Due To Altered Brain Compliance

1990 S. D. Michowiz: High Energy Phosphate Metabolism in Neonatal Hydrocephalus

1991 Nesher G. Asner: Venous Sinus Occlusion and Ventriculomegaly in Craniectomized Rabbits

1992 Marcia DaSilva: Reversal of High Energy Phosphate Metabolism Changes in Experimental Hydrocephalus After CSF Shunting

1993 Charles Bondurant: The Epidemiology of Cerebrospinal Fluid Shunting

1994 Monica C. Wehby—Grant: The Rabbit Model for Infantile Hydrocephalus: Regional Differences in the Cortical Metabolic Response to Hydrocephalus and Shunting

1995 Richard J. Fox: Cerebrospinal Fluid Absorptive Site of the Parasagittal Dura: A Cadaveric Study

1996 Martha J. Johnson: Reactive Astrocytosis in a New Model of Obstructive Hyrocephalus

1997 No prize awarded

1998 Daniel Lieberman: In Vetro Detection of Fluid Flow in Ventriculoperitoncal Shunts (VPS) Using Contrast Enhanced Ultrasound

Pediatric Section Chairmen

1972-73	Robert L. McLaurin	1983-85	Harold J. Hoffman
1973-74	M. Peter Sayers	1985-87	William R. Cheek
1974-75	Frank Anderson	1987-89	David G. McLone
1975-76	Kenneth Shulman	1989-91	Donald H. Reigel
1976-77	E. Bruce Hendrick	1991-93	R. Michael Scott
1977-78	Frank Nulsen	1993-95	Arthur Marlin
1978-79	Luis Schut	1995-97	Harold L. Rekate
1979-81	Fred J. Epstein	1998-99	Marion L. Walker
1981-83	Joan L. Venes	1999-2000	John P. Laurent

Annual Winter Meeting Sites

1972	Cincinnati	1988	Scottsdale
1973	Columbus	1989	Washington, D.C.
1974	Los Angeles	1990	San Diego & Pebble Beach
1975	Philadelphia	1991	Boston
1976	Toronto	1992	Vancouver, BC
1977	Cleveland	1993	San Antonio
1978	Philadelphia	1994	St. Louis
1979	New York	1995	Pasadena
1980	New York	1996	Charleston
1981	Dallas	1997	New Orleans
1982	San Francisco	1998	Indianapolis
1983	Toronto	1999	Atlanta
1984	Salt Lake City	2000	San Diego
1985	Houston	2001	New York
1986	Pittsburgh	2002	Phoenix
1987	Chicago	2003	Miami

Raimondi Lecturers

1978	E. Bruce Hendrick	1989	Martin Eichelberger
1979	Paul C. Bucy	1990	George R. Leopold
1980	Floyd Gilles	1991	Judah Folkman
1981	Panel Discussion	1992	Olof Flodmark
1982	Panel Discussion	1993	Maurice Albin
1983	Derek Harwood-Nash	1994	Blaise F.D. Bourgeois
1984	Anthony E. Gallo, Jr.	1995	Robert H. Pudenz
1985	Frank Nulsen	1996	Samuel S. Flint
1986	William F. Meacham	1997	M. Michael Cohen, Jr.
1987	Dale Johnson	1998	Robert A. Zimmerman
1988	Joseph J. Volpe	1999	David B. Schurtleff

1999 Raimondi Lecturer

David Bertrand Shurtleff, MD has been chosen as the 1999 Raimodi Lecturer Recipient.

Presently, Dr. Shurtleff is on staff at the Children's Hospital and Medical Center where he has held the positions of Instructor, Assistant, Associate and Professor since 1960. Prior he was the Chief of Pediatric Section U.S. Army Station Hospital in Fort Hood, Texas from 1958- 1959.

Dr. Shurtleff graduated from Harvard College in 1951 and received his medical degree from Tufts University Medical School in 1955. Dr. Shurtleff's postgraduate training began at the Massachusetts General Hospital and Harvard Medical School from 1955-1957 and then as Chief Resident Pediatrician and Associate in Pediatrics at the University of Washington from 1957-1958.

Dr. Shurtleff is the recipient of 9 honors/awards, guest lecturer in 36 various symposiums, author of 109 scientific articles which include 20 articles on hydrocephalus and 39 articles on meningomyelocele or hydrocephalus and meningomyelocele. Dr. Shurtleff is a member of several local and international society memberships and is currently President of the Society for Research into Hydrocephalus and Spina Bifida since 1998.

Exhibit Hall Floor Plan

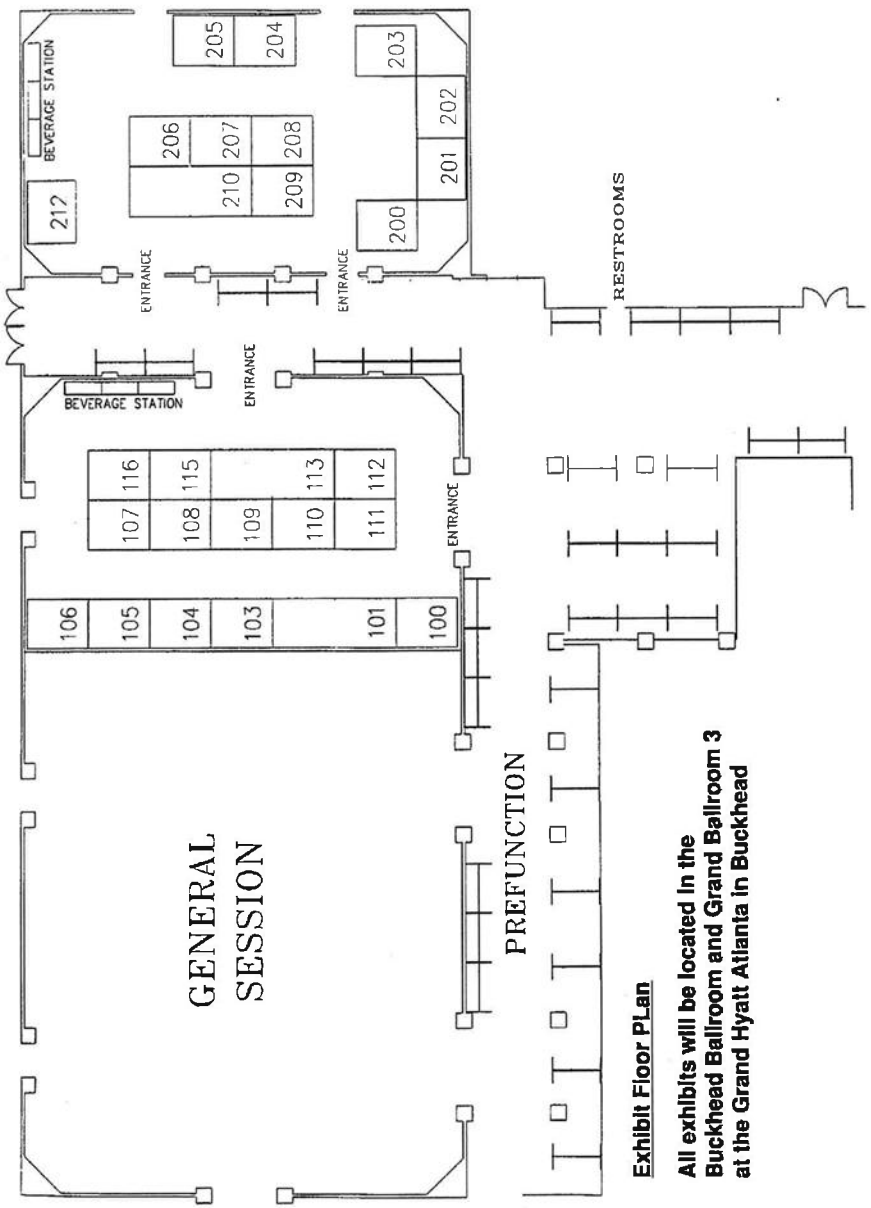


Exhibit Floor PLan

All exhibits will be located in the Buckhead Ballroom and Grand Ballroom 3 at the Grand Hyatt Atlanta in Buckhead

Exhibitor Listing

The AANS/CNS Section on Pediatric Neurological Surgery gratefully recognizes the support of the following exhibitors:

Acra-Cut, Inc. ..... Booth 112
989 Main Street
Acton, MA 01720
(978) 263-2210

BrainLAB, Inc. ..... Booth 110
100 Marine Parkway
Redwood City, CA 94065
(650) 494-7240

Carl Zeiss, Inc. ..... Booth 111
One Zeiss Drive
Thornwood, NY 10594
(800) 442-4020

Codman, a Johnson & Johnson ..... Booth 210
325 Paramount Drive
Raynham, MA 02767
(508) 828-3253

Compass International, Inc. ..... Booth 107
919 37th Avenue, NW
Rochester, MN 55901
(507) 281-2143

Haemacure Corporation ..... Booth 108
2 North Tamiami Trail, Suite 802
Sarasota, FL 34236
(941) 364-3700

Integra NeuroCare .... Booth 208
105 Morgan Lane
Plainsboro, NJ 08536
(609) 275-0500

KLS-Martin, L.P. ..... Booth 206
P.O. Box 50249
Jacksonville, FL 32250
(904) 641-7746

Leica Microsystems, Inc. ..... Booth 113
110 Commerce Drive
Allendale, NJ 07401
(800) 526-0355 ext. 5968

Lippincott William & Wilkins ..... Booth 200
1855 Bridle Ridge Trace
Roswell, GA 30075
(770) 552-4402

Medtronic Neurological ..... Booth 103
800 53rd Avenue NE
Minneapolis, MN 55112
(612) 514-5000

Medtronic PS Medical ..... Booth 101
125 Cremona
Goleta, CA 93117
(805) 968-1546

Midas Rex..... Booth 100
3000 Race Street
Ft. Worth, TX 76111
(817) 831-2604

NMT Neurosciences... Booth 115
3450 Corporate Way, Suite A
Duluth, GA 30096
(800) 423-6297 ext. 108

NS Recruitment, Incorporated..... Booth 212
P.O. Box 36096
Louisville, KY 40233-6096
(888) 673-6096

Phoenix Biomedical Corp..... Booth 202
2495 General Armistead Avenue
Norristown, PA 19403
(610) 539-9300

Radionics ..... Booth 209
22 Terry Avenue
Burlington, MA 01803
(781) 272-1233

SurgiTel/General Scientific Corporation ..... Booth 201
77 Enterprise Drive
Ann Arbor, MI 48103
(734) 996-9200

Valleylab ..... Booth 109
5920 Longbow Drive
Boulder, CO 80301
(303) 530-2300

W. B. Saunders Co. ... Booth 203
5637 Bellville Way
Norcross, GA 30092
(770) 449-9437

W. Lorenz Surgical .... Booth 104
1520 Tradeport Drive
Jacksonville, FL 32218
(904) 741-4400

Acknowledgements

The AANS/CNS Section on Pediatric Neurological Surgery wishes to thank the following companies for their educational grants and sponsorships in support of the 28th Annual Meeting.

Codman, a Johnson & Johnson Company

Integra NeuroCare

Medtronic PS Medical

NS Recruitment, Inc.

Valleylab

Thank you for your support!!

About the AANS/CNS Section on Pediatric Neurological Surgery

Officers

Chairman:	John P. (Jack) Laurent, MD (1999)
Secretary-Treasurer:	Thomas G. Luerssen, MD (1997)
Executive Committee Members at Large (two year terms):	Rich Abbott Ann Marie Flannery, MD Sarah J. Gaskill, MD Bruce Kaufman, MD (1998) C. Scott McLanahan, MD (1998)

Standing Committees

Nominating Committee:	Chairman: Marion L. Walker, MD (1999) Harold L. Rekate, MD (1997) R. Michael Scott (1995)
Rules and Regulations Committee:	Chairman: Alan R. Cohen, MD (1999)
Membership Committee:	Chairperson: Ann-Christine Duhaime, MD (1997)
Program and Continuing Education Committee:	Chairman: Frederick A. Boop MD (1997) Vice-Chairman: Jeffrey H. Wisoff, MD (1999)
Annual Meeting Chairman:	Joseph Petronio, MD
Ex Officio Members:	John P. Laurent, MD Thomas G. Luerssen, MD
Future Annual Meeting Chairmen:	2000: Hector E. James, MD 2001: I. Richmond Abbott, III, MD 2002: Harold L. Rekate, MD 2003: Glenn Morrison, MD

Ad Hoc Committees

Traveling Fellowship Committee:	Chairman: R. Michael Scott, MD
Distinguished Service Award:	Chairman: Robin P. Humphreys, MD
NEUROSURGERY://ON-CALL® Editorial Board:	Chairman: Mark S. Dias, MD Vice Chairman: I. Richmond Abbott, III, MD

About the AANS/CNS Section on Pediatric Neurological Surgery (con't)

Editorial Board Members:	Keith Aronyk, MD James Drake, MD David Jimenez, MD John Kestle, MD Michael Partington, MD James Rutka, MD Marion L. Walker, MD
Publications Committee:	Chairman: Arthur E. Marlin, MD
Co-Chairperson:	Sarah Gaskill, MD David McLone, MD, PhD Dennis Johnson, MD Rodger Hudgins, MD Paul Steinbok, MD
Liaison to the AANS Sections:	Harold L. Rekate, MD (1997)
Liaison to the American Academy of Pediatrics:	Joseph H. Piatt, Jr. MD (1997)
Liaison to the Joint Council of State Neurosurgical Societies:	C. Scott McLanahan, MD (1995)
Representatives to the Quality Assurance Committee:	Chairman: Paul A. Grabb, M.D. (1999) Sarah J. Gaskill, MD (1999) James Drake, MD (1999)
Representative to Referral Guidelines Committee:	Harold L. Rekate, MD (1997)
Representatives to Practice Guidelines Committee:	Thomas G. Luerssen, MD (1996) John Kestle, MD (1996)
Representatives to Outcomes Committee:	Bruce A. Kaufman, MD (1997) John Kestle, MD (1997)
Representative to the Washington Committee:	Marion L. Walker, MD (1999)

Disclosure Information

The AANS/CNS Section on Pediatric Neurological Surgery and the American Association of Neurological Surgeons control the content and production of this CME activity and attempt to assure the presentation of balanced, objective information. In accordance with the Standards for Commercial Support established by the Accreditation Council for Continuing Medical Education, speakers and paper presenters are asked to disclose any relationship they or their co-authors have with commercial companies which may be related to the content of their lecture.

Speakers and paper presenters/authors who have disclosed a relationship* with commercial companies whose products may have a relevance to their presentation are listed below and will be designated throughout the program book by an "†".

Ramin Abdolvahabi, MD, PhD	**Wayne State University Leede Hydrocephalus Research Fund**
Xingang Cai, MD	**Orlando Regional Healthcare System**
Susan Durham, MD	**NIH R29 NS 37132**
Paul Francel, MD, PhD	**Medtronic PS Medical**
Zhen-guo Li, MD, PhD	**Wayne State University Leede Hydrocephalus Research Fund**
Mark Luciano, MD, PhD	**NeuroCare Inc.**
Thomas Luerssen, MD	**Pfizer, Inc. NIH**
Joseph Madsen, MD	**Cyberonics Boston Life Sciences, Inc.**
Glenn Morrison, MD	**Miami Children's Hospital**
Jogi Pattisapu, MD	**Orlando Regional Healthcare System**
David Shafron, MD	**Medtronic**

*Relationship refers to receipt of royalties, consultantship, funding by research grant, receiving honoraria for educational services elsewhere, or any other relationship to a commercial company that provides sufficient reason for disclosure.

28th Annual Meeting of the AANS/CNS Section on Pediatric Neurological Surgery

Scientific Program

Wednesday, December 1, 1999

11:00 AM–12:00 NOON	Registration for Nurse's Seminar—**Ballroom Foyer**
12:00 NOON–5:00 PM	Nurses' Seminar—**Peachtree**
2:00 PM–8:00 PM	Registration—**Ballroom Foyer**
2:00 PM–8:00 PM	Speaker Preview Room—**Cassis A**
4:00 PM–6:00 PM	Poster Setup for Medical Registrants—**Ballroom Prefunction**
6:00 PM–8:00 PM	**Welcoming Reception—Grand Ballroom 1 and 2**

Thursday, December 2, 1999

7:00 AM–6:00 PM	Registration—**Ballroom Foyer**
7:00 AM–6:00 PM	Speaker Preview Room—**Cassis A**
7:00 AM–8:15 AM	Continental Breakfast with Exhibit and Poster Viewing- **Buckhead Ballroom and Grand Ballroom 3**
7:00 AM–8:15 AM	**BREAKFAST SEMINAR I: Current and Future Clinical Trials in Pediatric Neuro-oncology Through the Children's Oncology Group—Grand Ballroom 1 and 2** Moderator: *R. Alexander Sanford, MD* Faculty: *Jeffrey H. Wisoff, MD*
7:00 AM–11:30 AM & 2:00 PM–6:00 PM	**Exhibit and Poster Viewing Hours—Buckhead Ballroom, Grand Ballroom 3, Ballroom Prefunction**
8:00 AM–10:00 AM	Spouse/Guest Hospitality—**Peachtree**
8:15 AM–8:30 AM	Welcome and Opening Remarks—**Grand Ballroom 1 and 2**

8:30 AM–9:45 AM	**Scientific Session I—Grand Ballroom 1 and 2** Moderators: *R. Alexander Sanford, MD and Bruce A. Kaufman, MD*
8:30 AM–8:45 AM	**1. Natural History Study of Childhood Craniopharyngiomas Following Surgical Resection** *Michele Silva, MD, Tadanori Tomita, MD, Marianne Marymont, MD, John Kalapurakal, MD (Chicago, IL)*
8:45 AM–9:00 AM	**2. Transsphenoidal Craniotomy in the Very Young*** *Mark D. Krieger, MD, Michael L. Levy, MD, J. Gordon McComb, MD, Martin H. Weiss, MD (Los Angeles, CA)*
9:00 AM–9:15 AM	**3. The Significance of Invasion of Neural Tissue on Survival in Pediatric Patients with Craniopharyngiomas*** *Charles Y. Liu, MD, PhD, Michael L. Levy, MD, Floyd H. Gilles, MD, J. Gordon McComb, MD*
9:15 AM–9:30 AM	**4. Pediatric Spinal Axis Tumors: A Review of 15 Years Experience** *Mark A. Liker, MD, Roger Hsuing, BA, Jones George, BA, Michael L. Levy, MD, J. Gordon McComb, MD*
9:30 AM–9:45 AM	**5. Non-teratomatous Sacral Tumors in the Pediatric Population** *Cornelius H. Lam, MD, Mahmoud G. Nagib, MD (Minneapolis, MN)*
9:45AM–10:15 AM	Coffee Break with Exhibit and Poster Viewing—**Buckhead Ballroom and Grand Ballroom 3, Ballroom Prefunction**
10:15 AM–11:30 AM	**Scientific Session II—Grand Ballroom 1 and 2** Moderators: *Jeffrey H. Wisoff, MD and Liliana Goumnerova, MD*
10:15 AM–10:30 AM	**6. Hydrocephalus after Posterior Fossa Tumor Surgery: Type, Timing and Tumor Type** *Khaled B. Aly, MD*

* *Considered for Shulman Award*

10:30 AM–10:45 AM	**7. Outcome Analysis of Medulloblastoma: Correlations Between Clinical, Radiographic, Pathologic, Surgical Variables and Survival** *Alan M. Scarrow, MD, J.D. Louisa Urgo, PA, Rurik C. Johnson, BS, Ian F. Pollack, MD, Ronald L. Hamilton, MD (Pittsburgh, PA)*
10:45 AM–11:00 AM	**8. MIB-1 Staining Index of Pediatric Meningiomas** *Mark M. Souweidane, MD, David I. Sandberg, MD (New York, NY), James T. Rutka, MD, PhD (Toronto, Ontario, Canada), Lothar Resch, MD (Halifax, Nova Scotia, Canada), Mark A. Edgar, MD (New York, NY)*
11:00 AM–11:15 AM	**9. Linear Accelerator-Based Stereotactic Radiosurgery in the Pediatric Population** *Christopher D. Kager, MD, Jodie Ward, RT, Ronald E. Warnick, MD, Kerry R. Crone, MD, John C. Breneman, MD (Cincinnati, OH)*
11:15 AM–11:30 AM	**10. The Supratentorial Interhemispheric Approach to Pineal Region Tumors** *Michael L. Levy, MD, J.G. McComb, MD (Los Angeles, CA)*
11:30 AM–12:30 PM	**The 1999 Raimondi Lecture: 40 Years Experience with Myelomeningocele and Hydrocephalus—Grand Ballroom 1 and 2** *David B. Shurtleff, MD* *Professor & Chief, Birth Defects Clinic, Children's Hospital and Regional Medical Center, University of Washington*
12:30 PM–2:00 PM	**Special Luncheon Seminar for Residents and Fellows** Implications of the Managed Care Environment in Pediatric Neurological Surgery—**Peachtree** Faculty: *Peter W. Carmel, MD*
12:30 PM–2:00 PM	Lunch for all Medical Registrants—**Casis**
2:00 PM–4:00 PM	**Scientific Session III—Grand Ballroom 1 and 2** Moderators: *J. Gordon McComb, MD and Joseph H. Piatt, Jr., MD*

2:00 PM–2:15 PM
11. Effects of Shunt Treatment on the GnRH System in Congenital Hydrocephalus
Ramin M. Abdolvahabi, MD, PhD†, Jeffrey J. Cortese, CST, Fernando G. Diaz, MD, PhD, Pat McAllister, PhD (Detroit, MI)

2:15 PM–2:30 PM
12. Hydrocephalus Induces the Proliferation of Cells in the Subventricular Zone
Kimberly D. Bingaman, MD, Viorica Pencea, MD, Marla B. Luskin, PhD (Atlanta, GA)

2:30 PM–2:45 PM
13. Brain and CSF Oxygen Saturation in an Animal Model of Chronic Hydrocephalus: Response to Hyperventilation
Mark G. Luciano, MD, PhD†, Toru Fukuhara, MD, PhD, Christine L. Brant, MS, Jennifer L. Klauschie, BA, Megan E. Knoch (Cleveland, OH)

2:45 PM–3:00 PM
14. Supplemental Pharmacologic Therapy of Experimental Infantile Hydrocephalus
Eric M. Massicotte, MD, Marc R. Del Bigio, MD, PhD, FRCPC (Toronto, Canada)

3:00 PM–3:15 PM
15. Impaired Motor Learning in Children with Hydrocephalus
Yuchuan Ding, MD, PhD, Q. Lai, PhD, J.P. McAllister, PhD, S.D. Ham, MD, S.S. Sood, MD, A. I. Canady, MD (Detroit, MI)

3:15 PM–3:30 PM
16. Arachnoid Cysts: Treatment—Cyst to Peritoneal Shunt
Timothy Burson, MD, Michael S. Muhlbauer, MD, Robert A. Sanford, MD (Memphis, TN)

3:30 PM–3:45 PM
17. Minimally Invasive Craniotomy to Fenestrate Middle Fossa Arachnoid Cysts*
Michael Y. Wang, MD, Michael L. Levy, MD, J. Gordon McComb, MD

3:45 PM–4:00 PM
18. Aggressive Endoscopic Management of Primary Intracranial Cysts
Michael H. Handler, MD, FACS, FAAP

4:00 PM–4:30 PM
Beverage Break with Exhibit and Poster Viewing—**Buckhead Ballroom and Grand Ballroom 3, Ballroom Prefunction**

† Disclosure Information ** Considered for Shulman Award*

4:30 PM–6:00 PM
Scientific Session IV—Grand Ballroom 1 and 2
Moderators: *Michael R. Scott, MD and Alan R. Cohen, MD*

4:30 PM–4:45 PM
19. The Importance of Shunt Valve Position in Determining Outflow Rates
Paul C. Francel, MD, PhD†, F Alan Stevens, BS, Paul Tompkins, MS (Oklahoma City, OK)

4:45 PM–5:00 PM
20. Long-Term Outcome Analysis of Initial Neonatal Shunt Valves
Shenandoah Robinson, MD, Bruce Kaufman, MD, T.S.Park, MD (St. Louis, MO)

5:00 PM–5:15 PM
21. Direct Heart Shunt: A Viable Alternative?
Robert F. Keating, MD, Frank Midgley, MD, David W. Pincus, MD, PhD, Leon E. Moores, MD, Philip H. Cogen, MD, PhD (Washington, DC)

5:15 PM–5:30 PM
22. 45 Cases of Percutaneous Endoscopic Recanalization of Catheter (PERC Procedure)—A Long Term Study
Jogi V. Pattisapu, MD, Eric Trumble, MD, Kay Taylor, RN, Denise Howard, CST, Tina Kovach, RN, Annette Arbogast

5:30 PM–5:45 PM
23. Addition of Elemental Iodine to Surgical Irrigant for Shunt Infection Prophylaxis
SooHo Choi, MD, J. Gordon McComb, Mike L. Levy, Ignacio Gonzales, Berislav Zlokovic, Roger Bayston

5:45 PM–6:00 PM
24. Pediatric Shunt Infections: A Prospective Analysis
Elizabeth B. Claus, PhD, MD, Eileen Ogle, PA, Charles Duncan, MD (New Haven, CT)

6:30 PM–10:30 PM
Annual Reception and Banquet
Fernbank Museum of Natural History
Bus departs Grand Hyatt Atlanta at 6:30 PM from the front entrance.

† Disclosure Information

Friday December 3, 1999

7:00 AM–5:00 PM	Registration—**Ballroom Foyer**
7:00 AM–5:00 PM	Speaker Preview Room—**Cassis A**
7:00 AM–8:30 AM	Continental Breakfast with Exhibit and Poster Viewing—**Buckhead Ballroom and Grand Ballroom 3, Ballroom Prefunction**
7:00 AM–8:30 AM	**Breakfast Seminar II: Controversies in the *In Utero* Surgical Treatment of Myelomeningocele—Grand Ballroom 1 and 2** Moderator: *Bruce B. Storrs, MD* Faculty: *Leslie N. Sutton, MD and Noel Tulipan, MD*
7:00 AM–11:30 AM & 2:00 PM–6:00 PM	**Exhibit and Poster Viewing Hours Buckhead Ballroom and Grand Ballroom 3, Ballroom Prefunction**
8:00 AM–10:00 AM	Spouse/Guest Hospitality- **Piedmont**
8:30 AM–9:45 AM	**Scientific Session V—Grand Ballroom 1 and 2** Moderators: *Bruce B. Storrs, MD and Robin P. Humphreys, MD*
8:30 AM–8:45 AM	**25. Soluble Adhesion Molecules Are Elevated in the Cerebrospinal Fluid of Children with Moyamoya Syndrome** *R. Michael Scott, MD, S.G. Soriano, D.B. Cowan, M.R. Proctor (Boston, MA)*
8:45 AM–9:00 AM	**26. Multimodality Treatment in Pediatric AVMs Improves Outcome** *Brian L. Hoh, MD, Christopher S. Ogilvy, MD, William E. Butler, MD, Christopher M. Putman, MD, Jay S. Loeffler, MD, Paul H. Chapman, MD (Boston, MA)*
9:00 AM–9:15 AM	**27. Is There a Difference in Incidence and Outcome Between Children and Adults with Vasospasm*** Larry T. Khoo, MD, Michael L. Levy, MD, Steven Giannotta, MD, J. Gordon McComb, MD *(Los Angeles)*

* *Considered for Shulman Award*

Friday December 3, 1999 (con't)

9:15 AM–9:30 AM	**28. Posterior Cervical Fusion in Children and Adolescents** *William R. Boydston, MD, Roger J. Hudgins, MD*
9:30 AM–9:45 AM	**29. Spinal Arachnoid Cysts in Children*** *Hulda Magnadottir, MD, Michael L. Levy, MD, Mark Mittler, MD, J. Gordon McComb, MD*
9:45 AM–10:15 AM	Beverage Break with Exhibit and Poster Viewing—**Buckhead Ballroom and Grand Ballroom 3, Ballroom Prefunction**
10:15 AM–12:30 PM	**Scientific Session VI—Grand Ballroom 1 and 2** Moderators: *Andrew D. Parent, MD and Michael D. Partington, MD*
10:15 AM–10:30 AM	**30. Spina Bi fida Outcome: A 25 Year Prospective** *Robin M. Bowman, MD, David G. McLone, MD, PhD, John A. Grant, MD, Tadanori Tomita, MD (Chicago, IL)*
10:30 AM–10:45 AM	**31. Delayed Loss of Ambulation in Patients with Open Neural Tube Defects*** *Sooho Choi, MD, Richelle A. Lampa, Shervin Aminpour, Michael L. Levy, MD, J.G. McComb, MD (Los Angeles, CA)*
10:45 AM–11:00 AM	**32. Split-cord Malformations in Children*** Hulda Magnadottir, MD, Mark Mitler, MD, *Michael L. Levy, MD, J. Gordon McComb, MD*
11:00 AM–11:15 AM	**33. Radiographic Evaluation for Spinal Dysraphism in Long-term Follow-up of Patients with Imperforate Anus** *Tanvir F. Choudhri, MD, Ryan McTaggart, E. Sander Connolly, Jr., MD (New York, NY), Indro Chakrabarti (Los Angeles, CA), Alex Khandji, MD, Neil Feldstein, MD (New York, NY)*
11:15 AM–11:30 AM	**34. Division of the Transverse Sinus and Tentorium Cerebelli to Allow Vault Expansion and Posterior Fossa Decompression in the Treatment of Chiari II Malformation** *Timothy Burson, MD, Michael Muhlbauer, MD, Robert Sanford, MD, Raymond Fryrear, MD*

* *Considered for Shulman Award*

11:30 AM–11:45 AM	**35. Anterior Fontanelle Encephalocele** *Michael J. Burke, MD, Ken Winston, MD (Denver, CO)*
11:45 AM–12:00 PM	**36. Plagiocephaly: Orthotic and Non-Orthotic Treatment, a Three-year Follow-up** *S. David Moss, MD, Mohammed Jalaluddin, MD (Phoenix, AZ)*
12:00 PM–12:15 PM	**37. Tensile Strain Induces Increase in Intracellular Calcium Concentration in Immature Rat Cranial Sutures** *Karsten Fryburg, MD, Jack Yu, James Borke, Ann-Marie Flannery*
12:15 PM–12:30 PM	**38. Transplanted Demineralized Bone Graft in Cranial Reconstructive Surgery—Five Year Follow-up** *Mohammed Jalaluddin, MD, S. David Moss, MD (Phoenix, AZ)*
12:30 PM–2:00 PM	Lunch for all Medical Registrants—**Casis**
2:00 PM–3:30 PM	**Scientific Session VII—Grand Ballroom 1 and 2** Moderators: *Ira R. Abbott III, MD and Mark R. Lee, MD*
2:00 PM–2:15 PM	**39. Pediatric Neurosurgeons Demonstrate Increased Satisfaction with Workload and Anticipate Modest Growth in the Workforce** *Ann Marie Flannery, MD*
2:15 PM–2:30 PM	**40. Shunt Pseudotumor** *Timothy Burson, MD, Michael S. Muhlbauer, MD, Robert A. Sanford, MD (Memphis, TN)*
2:30 PM–2:45 PM	**41. Quantitative Diffusion MR Imaging of Pediatric Intracranial Abscesses** *Rimas P. Gilvydis, MD, Robert McKinstry, MD, PhD, Benjamin Lee, MD, Tae Sung Park, MD, Bruce A. Kaufman, MD (St. Louis, MO)*
2:45 PM–3:00 PM	**42. Intrathecal Baclofen for the Treatment of Dystonia** *David H. Shafron, MD†, A. Leland Albright, MD, Margaret J. Barry, MS, PT (Pittsburgh, PA)*

3:00 PM–3:15 PM	**43. Staged, Tailored Hemispherectomy in the Management of Pediatric Epilepsy: A Rational Approach for Hemispheric Malformations of Cortical Development** *Howard L. Weiner, MD, Henry Woo, MD, Mary L. Zupanc, MD, Orrin Devinsky, MD, Jeffrey H. Wisoff, MD (New York, NY)*
3:15 PM–3:30 PM	**44. Epilepsy Surgery in Early Infancy, Experience with Four Infants and Outcomes*** *Greg Olavarria, MD, Joseph A. Petronio, MD (Atlanta, GA)*
3:30 PM–5:00 PM	**Scientific Session VIII—Grand Ballroom 1 and 2** Moderators: *Frederick A. Boop, MD and Ann-Christine Duhaime, MD*
3:30 PM–3:45 PM	**45. Dysembryoplastic Neuroepithelial Tumours and Childhood Epilepsy: A Case Series** *Jeffrey D. Atkinson, MD, J.P. Farmer, MD, J.L. Montes, MD, A.G. O'Gorman, MD, S. Albrect, MD, K. Meagher-Villemure, MD (Montreal, QC)*
3:45 PM–4:00 PM	**46. The Role of the Hippocampus in Brain Tumor Associated Epilepsy: The Concept of "Dual Pathology"** *Arno H. Fried, MD, FACS, Christos Lambrakis, MD, Marcelo Lancman, MD (Hackensack, NJ)*
4:00 PM–4:15 PM	**47. Vagal Nerve Stimulation in Pediatric Patients Following Intracranial Epilepsy Surgery** *Joseph R. Madsen, MD†, Dilek Yalnizoglu, MD, Sandra Helmers, MD (Boston, MA)*
4:15 PM–4:30 PM	**48. Surgical Outcome in Children with Epilepsy Evaluated by Magnetoencephalography** *Mark R. Lee, MD, Joseph R. Smith, MD, Yong D. Park, MD, Don W. King, MD (Augusta, GA)*

Friday December 3, 1999 (con't)

4:30 PM–4:45 PM	**49. Seizure Surgery for Children with Tuberous Sclerosis Complex** *Glenn Morrison, MD†, Prasanna Jayakar, MD (Miami,FL), Susan Koh, MD (Los Angeles,CA), John Ragheb, MD, Michael Duchowny, MD, Trevor Resnick, MD, Maria Penate, RN, Patricia Dean, RN (Miami, FL)*
4:45 PM–5:00 PM	**50. Surgical Management of Gelastic Epilepsy Related To Hypothalamic Hamartoma** *J.P. Farmer, MD, S. Mittal, MD, J.L. Montes, MD, B. Rosenblatt, MD, F. Andermann, MD (Montreal, PQ)*
5:00 PM–6:00 PM	**Wine and Cheese Reception and Oral Presentation of Posters—Buckhead Ballroom and Grand Ballroom 3, Ballroom Prefunction** *Authors of posters are asked to stand next to their poster to answer questions*
6:00 PM	**Annual Business Meeting—Grand Ballroom 1 and 2**

Saturday December 4, 1999

7:00 AM–12:00 NOON	Registration—**Ballroom Foyer**
7:00 AM–12:00 NOON	Speaker Preview Room—**Cassis A**
7:00 AM–8:30 AM	Continental Breakfast with Exhibit and Poster Viewing—**Buckhead Ballroom and Grand Ballroom 3, Ballroom Prefunction**
7:00 AM–8:30 AM	**Breakfast Seminar III: Grand Ballroom 1 and 2** Surgical Management of Spasticity in the Child with Cerebral Palsy Moderator: *Ira R. Abbott III, MD*
7:00 AM–12:00 NOON	Exhibit and Poster Viewing Hours—**Buckhead Ballroom and Grand Ballroom 3, Ballroom Prefunction**
8:00 AM–10:00 AM	Spouse/Guest Hospitality—**Piedmont**
8:30 AM–9:45 AM	**Scientific Session IX—Grand Ballroom 1 and 2** Moderators: *Ira R. Abbott III, MD and Ann Marie Flannery, MD*

† Disclosure Information

Saturday December 4, 1999 (con't)

8:30 AM–8:45 AM	**51. The Contemporary Management of the Chiari I Malformation in Children*** *Mark D. Krieger, MD, Michael L. Levy, MD, J. Gordon McComb, MD (Los Angeles, CA)*
8:45 AM–9:00 AM	**52. Paroxysmal Rage as a Presenting Symptom of the Chiari 1 Malformation** *Roger J. Hudgins, MD*
9:00 AM–9:15 AM	**53. Characterization of Third Ventricular Floor Anatomy in the Hydrocephalic State Relative to 3rd Ventriculocisternotomy** *Stephanie L. Einhaus, MD, Erica Nevells, MD, Anthony Watkins, MD (Memphis, TN)*
9:15 AM–9:30 AM	**54. Cine Phase-contrast CSF Flow MRI after Third Ventriculostomy: Correlation with Endoscopic Exploration** *Toru Fukuhara, MD, PhD, Mark G. Luciano, MD, PhD, Christine L. Brant, MS (Cleveland, OH)*
9:30 AM–9:45 AM	**55. Endoscopic Fenestration of Third Ventricular Arachnoid Cysts** *Michael L. Levy, MD, Mark Liker, MD, Michael Y. Wang, MD, Hulda Magnadottir, MD, J. Gordon McComb, MD*
9:45 AM–10:15 AM	Coffee Break with Exhibit and Poster Viewing—**Buckhead Ballroom and Grand Ballroom 3, Ballroom Prefunction**
10:15 AM–12:00 NOON	**Scientific Session X—Grand Ballroom 1 and 2** Moderators: *Thomas G. Luerssen, MD† and Michael L. Levy, MD*
10:15 AM–10:30 AM	**56. A Prospective Population-based Study of Pediatric Trauma Patients with Mild Alterations in Consciousness (field Glasgow Coma Score of 13-14)*** *Michael Y. Wang, MD, Pamela Griffith, RNC, BSN, Judy Sterling, RN, J. Gordon McComb, MD, Michael L. Levy, MD*

† Disclosure Information ** Considered for Shulman Award*

10:30 AM–10:45 AM

57. Leukocyte Dependent Blood-Brain Barrier Breakdown is Attenuated by a Novel Carboxyfullerene Derivative in a Model of Newborn Hypoxic -Ischemic Encephalopathy in Piglets
Stuart S. Kaplan, MD, Jeffrey M. Gidday, PhD, Laura L. Dugan, MD, Ernesto R. Gonzales, Ronaldo S. Perez, TS Park, MD

10:45 AM–11:00 AM

58. The Role of Fast Axonal Transport Proteins in Spinal Cord Regeneration*
Brian Witwer, MD, Bermans J. Iskandar, MD, Agahan Unlu, MD, Nythia Hariharan, MD (Madison, WI), J.H. Pate Skene, PhD (Durham, NC)

11:00 AM–11:15 AM

59. The Surprisingly Sturdy Infant Brain: Why is it More Resistant to Focal Injury?*
Susan R. Durham, MD†, Susan Margulies, PhD, Ramesh Raghupathi, PhD, Jeffrey Golden, MD, Mark Helfaer, MD, Amy Brooks-Kayal, MD, Sunil Marwaha, BS, Ann-Christine Duhaime, MD (Philadelphia, PA)

11:15 AM–11:30 AM

60. Stimulation of Extensive Axon Growth in the Injured Rat Corticospinal Tract with Inosine
Joseph R. Madsen, MD†, David Goldberg, Nina Irwin, PhD, Larry I. Benowitz, PhD (Boston, MA)

11:30 AM–11:45 AM

61. Deep Venous Thrombosis in a Pediatric Neurosurgical Population*
David Hart, MD, Mark Krieger, Michael Levy, J. Gordon McComb (Los Angeles, CA)

11:45 AM–12:00 NOON

62. Cervical Spine Clearance in Major Trauma in a Pediatric Population
Leon E. Moores, MD, Jean Reardon RN, Philip H. Cogen MD, PhD, Martin Eichelberger MD, Robert F. Keating MD, David W. Pincus, MD, PhD (Washington, DC)

12:00 NOON–12:15 PM

63. Abdominal "Stealth" Incisions for VP Shunts
Michael H. Handler, MD, FACS, FAAP (Denver, CO)

12:15 PM

Adjourn

† Disclosure Information ** Considered for Shulman Award*

Poster Session

1. **Cranial Bone Fixation After Craniotomy: A New Method**
   Marjorie C. Wang, MD, Ken R. Winston, MD

2. **An Alternate Technique to Close Neurosurgical Incisions Using Octylcyanoacrylate Adhesive**
   Daniel J. Won, MD, FACS, Kuk-Wha Lee, MD, PhD, Thomas Sherwin, MD (Loma Linda, CA)

3. **Chronic Group B Streptococcal Ventriculitis—A Case Report and Review of the Literature**
   David S. Jones, MD, John D. Loyd, MD, Steven S. Glazier, MD, S. Taylor Jarrell, Michael J. Opatowsky, MD, Laurence B. Givner, MD (Winston-Salem, NC)

4. **Amoebic Meningoencephalitis Caused by Balamuthia Mandrillaris Presenting with Acute Anterograde Amnesia in a 16 Old Boy**
   Jeffrey P. Blount, MD, Jared Antevil, MD, Christopher S. Kent, MD, Michael Quigley, MD (San Diego, CA)

5. **Trampoline Injuries of the Cervical Spine**
   Peter G. Brown, MD, Mark Lee, MD (Augusta, GA)

6. **Early Management of Post-Traumatic Leptomeningeal Cysts**
   Miltos K. Sugiultzoglu, MD, Mark M. Souweidane, MD (New York, NY)

7. **FORE: Golf-Related Head Injuries at the Arnold Palmer Hospital for Children**
   Eric R. Trumble, MD, Jogi Pattisapu, MD, Kay Taylor, RN, Annette Arbogast

8. **Cerebral Perfusion and Pial Capillary Diameter Responses to Fluid Percussion Injury**
   Paul C. Framcel, MD, PhD, F. Alan Stevens, BS, Soon C. Kim, MD, Paul Tompkins, MS (Oklahoma City, OK)

9. **Shaking Impact Syndrome—A Retrospective Analysis of 35 Cases**
   Ming-Ying Liu

10. **Treatment of Pediatric Tornadic Traumatic Brain Injury**
John H. Honeycutt, MD, Sumon Bhattacharje, MD, Dave Min, James Brennan, Mary K. Gumerlock (Oklahoma City, OK)

11. **Transcranial Doppler Ultrasound Waveform Analysis for Noninvasive Intracranial Pressure Monitoring**
Jens Peter Witt, MD, Shusma Aggarwal, MD, Howard Yonas, MD (Pittsburgh,PA)

12. **Spontaneous Thrombosis of Aneurysmal Malformation of Vein of Galen**
Dimitrios C. Nikas, MD, Mark R. Proctor, MD, R. Michael Scott, MD (Boston, MA)

13. **Treatment of Moyamoya Syndrome in Children**
Ann Marie Flannery, MD, Michael Cowan, MD (Charlotte, NC), Dennis McDonnell, MD (Augusta, GA)

14. **Combined Endovascular and Microsurgical Treatment of a Bilateral Thalamic Arteriovenous Malformation Associated with Vein of Galen Dilatation**
Edward R. Flotte, MD, John A. Lancon, MD, David Scalzo, MD (Jackson, MS), J. Parker Mickle, MD (Gainesville, FL), Adam I. Lewis, MD (Jackson, MS)

15. **A Lesson to be Learned in Pediatric Subarachnoid Hemorrhage**
Michael J. Burke, MD

16. **Intractability Following Functional Hemispherectomy: Important Lessons Learned From an Unusual Case***
S. Mittal, MD, J.P. Farmer, MD, B. Rosenblatt, MD, F. Andermann, MD, J.L. Montes, MD (Montreal, PQ), J.G. Villemure, MD (Lausanne, Switzerland)

17. **Neurosurgical Management of Complex Pediatric Thoracolumbar Spinal Deformity**
Gregory C. Wiggins, MD, Christopher I. Shaffrey, MD (Seattle, WA), Henry M. Bartkowski, MD, Michael J. Rauzzino, MD (Detroit, MI)

18. **A Modified Cranial Reduction Using Circumferential Barrel Stave Wedge Osteotomies**
Jason Lifshutz, MD, Kuk-Wha Lee, MD, PhD, Ronald M. Perkin MD, Daniel J. Won, MD (Loma Linda, CA)

19. **Impact and Displacement Analysis for Pediatric Large Cranial Defect**
Hun K. Park, PhD, Manuel Dujovny, MD, Fernando G. Diaz, MD, PhD (Detroit, MI)

20. **Neuroendoscopic Third Ventriculostomy for Treatment of Chiari I Malformation with Hydrocephalus and Holocord Syringomyelia**
Jens P. Witt, MD, Michael Partington, MD (Denver, CO)

21. **Apoptosis and Dark Neurons in Hydrocephalic H-Tx Rats**
Zhen-guo Li, MD, PhD†, Hong Zhang, Pat McAllister, PhD (Detroit, MI)

22. **Hydrocephalus in the H-Tx Rat: A Monogenic Disease?**
Jogi V. Pattisapu, MD†, Xingang Cai, MD, Glenda McGraw, Laurence Von Kalm, PhD, Sue Willingham, Debra Socci, PhD, Jane Gibson, PhD (Orlando, FL)

23. **External Lumar Drainage in the Pediatrics Population**
Abdi S. Ghodsi, MD, Homan Mostafavi, BS, Farid Mousavi-Harami, BS, Arnold Menezes, MD, Vincent Traynelis, MD (Iowa City, IA)

24. **Molecular Biology and Genetics of Hydrocephalus**
Xingang Cai, MD†, Debora Bailey, MS, Jogi Pattisapu, MD (Orlando, FL)

25. **Craniopharyngiomas: Experience with Complex Approaches in Children**
Mark A. Liker, MD, Michael L. Levy MD, J. Gordon McComb, MD

26. **Histopathology of Carotid Aneurysmal Dilation after Surgery for Craniopharyngioma***
Amir Vokshoor, MD, Dan Boue', MD, Edward J. Kosnik, MD (Columbus, OH)

27. **Intraventricular Pilocytic Astrocytoma in an Infant: Case Report and Review of the Literature**
Holly S. Gilmer-Hill, MD, Kamran Sahrakar, MD, Dachling Pang, MD (Sacramento, CA)

28. **Intraspinal Hemangioendothelioma in a Premature Infant: An Epidural Hemangioma Variant**
Bohdan W. Chopko, PhD, MD, Robert Buchanan, MD, Christina Stanley, MD, Lawrence Hansen, MD (San Diego, CA)

29. **Cerebellar Mutism Associated with a Midbrain Lesion**
Marjorie C. Wang, MD, Robert E. Breeze, MD, Ken R. Winston, MD (Denver, CO)

30. **Cellular Immunotherapy of Brain Tumors Involving Intratumoral Placements of the Irradiated TALL-104 Cytotoxic T Cell Line**
Jane E. Freeman, CPNP, Ken R. Winston, MD, Nicholas Foreman, MD, David B. Paul, PhD, Carol J. Gup, BA, German Gomez, BS (Denver, CO), Sophie Visonneau, PhD, Daniela Santoli, PhD (Philadelphia, PA), Carol A. Kruse, PhD (Denver, CO)

31. **Alteration of Cell-cycle Kinetics Inhibits Growth Rate in Rat C6 Glioma Cells Transfected with Somatostatin cDNA Under Control of a MoMuLV Promoter**
Catherine A. Ruebenacker, MD, Sacchieri TA, Shao TM, Carmel PW, Siegel J.H., Feldman, SC (Newark, NJ)

32. **Intrathecal Alloreactive Cytotoxic T Lymphocytes and Interleukin-2 for Recurrent Ependymoma- A Phase 1 Study**
Ken R. Winston, MD, Jane Freeman, RN, CNPN, Nicholas Foreman, MD, Natalie Parker, PhD, Loris McGavran, PhD, Elena Savelieva, PhD, Carol A. Kruse, PhD (Denver, CO)

33. **Frameless Stereotactic Guided Resection for Thalamic/Basal Ganglia Tumors**
George I. Jallo, MD, Karl Kothbauer, MD, Jaoa Siffert MD, Fred Epstein, MD (New York, NY)

34. **Spinal Cord Tumor Presenting with Cerebral Atrophy**
Scott W. Elton, MD, Amir Vokshoor, MD, Edward Kosnik, MD (Columbus, OH)

Scientific Oral Abstracts

1. Natural History Study of Childhood Craniopharyngiomas Following Surgical Resection

Michele Silva, MD, Tadanori Tomita, MD, Marianne Marymont, MD, John Kalapurakal, MD *(Chicago, IL)*

Introduction: Despite its benign histology, childhood craniopharyngioma can recur even after total resection. In order to identify natural history, the authors analyze the relationship between the rate and time of recurrence and the extent of resection without adjuvant therapy. Patients who had incomplete resection and postoperative adjuvant therapy were excluded.

Methods: Thirty-four children with craniopharyngioma operated on from 1979 to 1996 were reviewed. All had a maximum resection without adjuvant therapy. Twenty-five had a total resection (TOTAL). Seven patients had a total resection according to surgeon's impression, but postoperative imaging showed mass or calcifications (TOTAL-equivocal). Other two had subtotal resection (SUBTOTAL).

Results: All but one are alive. Fourteen had recurrence: 13 had RT with or without reoperation. The five-year and ten-year recurrence-free survival (RFS) are 78 and 70% among TOTAL group, while the five-year RFS was 42% in TOTAL-eq. and 0% in SUBTOTAL groups (p=0.00365). Among patients with recurrence, all but one responded to RT.

Conclusion: There is a significant correlation between the RFS and the extent of resection of craniopharyngioma. For follow-up, both TOTAL and TOTAL-eq. groups are observed with surveillance imagings, while SUBTOTAL group needs early intervention with adjuvant therapy.

2. Transsphenoidal Craniotomy in the Very Young*

Mark D. Krieger, MD, Michael L. Levy, MD, J. Gordon McComb, MD, Martin H. Weiss, MD *(Los Angeles, CA)*

Transsphenoidal craniotomy has proven to be a useful surgical approach for lesions of the sella and suprasellar cistern in adults. However, the late pneumatization of the sphenoid sinus has led many surgeons to not consider

* *Considered for Shulman Award*

this approach in the very young. We report the effective use of the transsphenoidal craniotomy in 8 children who were under 3 years of age at the time of surgery. Surgery was performed for craniopharyngioma (5 cases), Rathke's cleft cyst (2 cases) and pituitary adenoma (1 case). Age ranged from 9 months to 3 years. The procedure differed from the standard transnasal transsphenoidal approach by requiring the use of the high-speed drill to remove the sphenoid, under x-ray guidance. "Early ostia" were found in all cases, facilitating the localization of the sphenoid and its removal. Operative complications in this limited series consisted of decreased monocular vision in one patient with a craniopharyngioma. Resections were complete in all patients except one of the craniopharyngiomas; this residual tumor was subsequently completely removed via a second transsphenoidal procedure. This series demonstrates the potential effectiveness of the transsphenoidal approach in the young child.

3. The Significance of Invasion of Neural Tissue on Survival in Pediatric Patients with Craniopharyngiomas*

Charles Y Liu, MD, PhD, Michael L. Levy, MD, Floyd H. Gilles, MD, J. Gordon McComb, MD

In both pediatric and adult patients, some craniopharyngiomas are known to invade surrounding neural structures. Sophisticated combined surgical approaches and microneurosurgical methods have made the safe gross total resection of even large craniopharyngiomas possible. Nevertheless, on histopathological examination, microscopic finger-like projections, islands, and perivascular growth can be still be identified. The significance this invasion is not clear. Some workers have postulated that craniopharyngiomas recur from the tumor islands and fingers, and that failure to completely resect them is largely responsible for recurrence despite gross total resection. This study examines influence on patient survival of microscopic tumor invasion in pediatric patients.

The Childhood Brain Tumor Consortium (CBTC) database included 143 cases of craniopharyngiomas. Excluding patients with survival of less than one month and those lost to follow-up, 11 out of 77 patients were found to have evidence of at least one of the three categories of microscopic invasion, that is fingers, islands, and perivascular growth. Sixty-six patients were found to had no evidence of microscopic invasion. The probability of survival at 1 year and 5 years based on Kaplan-Meier Survival Curves was 0.87 and 0.87

* *Considered for Shulman Award*

respectively. In the patients with no evidence of microscopic invasion, the survival probability at 1 year and 5 years was 0.89 and 0.74. The difference in survival probabilities between the patients with and without microscopic tumor invasion was not statistically significant ($p>0.3$).

4. Pediatric Spinal Axis Tumors: A Review of 15 Years Experience

Mark A. Liker, MD, Roger Hsuing, BA, Jones George, BA, Michael L. Levy, MD, J. Gordon McComb, MD

Pediatric spinal axis tumors are rare entities relative to their cranial counterparts. We present one hospital's review of 15 years experience in the presentation and surgical management of these lesions. Charts were available for a total of 102 cases of both extradural and intradural lesions. Three tumors, astrocytoma, neurofibroma, and neuroblastoma constituted almost half of all lesions (N=43). Other less common tumors include the ependymoma (N=5), hemangioblastoma (N=5), sarcoma (N=5), aneurysmal bone cyst (N=4), schwannoma (N=4), and teratoma (N=4). There were 42 extradural, 37 intramedullary, and 23 intradural/extramedullary tumors. Arthrodesis was required due to instability in 21 cases. We review the respective ages of presentation, gender, spinal level of involvement, signs and symptoms, and surgical outcome.

5. Non-teratomatous Sacral Tumors in the Pediatric Population

Cornelius H. Lam, MD, Mahmoud G. Nagib, MD ***(Minneapolis, MN)***

Introduction: Pediatric sacral tumors usually occur in the newborn period with the majority being sacrococcygeal teratomas. In this paper, we exclude common benign congenital tumors such as lipomas, dermoids, and epidermoids, as well as teratomas. Non-congenital non-teratomatous sacral tumors are more common in an older infant, but nevertheless as a group are rare.

Patients: Six patients were found in a six-year period. Ages ranged from eight years to eleven years. Three were males and three females. Five of six presented with back pain, three had constipation, and two had gait difficulties. Pathologies were diverse. They include ganglioneuroma (1), myxopapillary ependymoma (2), PNET (1), aneurysmal bone cyst (1), and neuroblastoma (1).

Procedure: Four patients underwent posterior resection of their tumors. One underwent a combined anterior and posterior approach. One patient underwent a posterior resection and will undergo a second stage anterior

approach later to allow for chemotherapy to shrink the intrapelvic portion of the tumor.

Result: No progression of disease has been seen for a follow-up period of one month to six years. Radical resection did not result in instability.

Conclusion: In contradistinction to adults where chordomas and metastasis are the most common primary and secondary tumors, no predominant pathology has been identified in the pediatric group. We find tumors can attain extremely large sizes and can be very vascular. Multiple therapeutic modalities might be required including adjuvant chemotherapy and possibly embolization. Because of the wide range of pathologies, prognosis can be varied.

6. Hydrocephalus after Posterior Fossa Tumor Surgery: Type, Timing and Tumor Type

Khaled B. Aly, MD

This is a prospective study including fourty cases of posterior fossa tumors operated upon by the author in the last 18 months. An external ventricular drain was applied in every case at the beginning of surgery and left for 1-4 days post-operatively. The nature of hydrocephalus was determined, i.e. obstructive versus communicating, by performing a synchronized lumbar and ventricular manometry. We found that hydrocephalus is common, 23/40 cases (57%), mostly communicating in nature 19/23 cases (80%). Two forms of the communicating type were encountered: an immediate form (13/19) that becomes manifest imediately after surgery and a delayed one. In the delayed form (6/19), the ICP and the CSF output are initially within normal;however, these patients present 1-2 weeks later with tense pseudomeningocele not responding to repeated tapping and ending with shunt placement. Medulloblastomas have a high incidence of postoperative hydrocephalus 13/15 cases (85%), mostly communicating in nature 12/13 cases, and mostly of the imediate type 10/12 cases. On the other hand, ependymoma have a low incidence of developing hydrocephalus:1/12 cases, of the communicating delayed form. Hydrocephalus was also common with astrocytomas: 7/11 cases (63%), mostly communicating:5/7 cases, and more commonly of the delayed form: 3/5 cases. In this report, hydrocephalus was found to be commoner than in other series, and mostly communicating in nature. Thus, establishment of CSF pathways during surgery is no garantee that post-operative hydrocephalus will not develop.

We reviewed our experience with medulloblastoma to identify clinical, radiographic, pathologic, and surgical resection correlations with survival.

Thirty-three cases (15m/18f) with a median age of 6 years (range 1-17) were evaluated. Hospital charts and radiographic imaging studies were reviewed for age, sex, pre-operative hydrocephalus, duration of symptoms, Chang T/N stage, and surgical resection (total, subtotal, partial). Pathological assessment included necrosis, mitoses and immunostaining for MIB, p53, bcl, synaptophysin, and GFAP. Kaplan-Meier curves plotting these parameters against survival were analyzed with log-rank and Wilcoxon signed rank sum tests.

The average duration of symptoms was 5.7 weeks. Radiographically, 86% had evidence of hydrocephalus on presentation while 89% had evidence of a Chang T stage 3 or 4. Twenty percent had dissemination outside the tumor bed (Chang N stage 1 or greater). Gross total or subtotal resection was achieved in 94%. GFAP staining was positive in 9.1%, synaptophysin in 30.2%, bcl in 42.4%, and p53 in 48.5% (33% strongly positive). Median MIB percentage was 40.45% (range 17.78-90.94). There was no necrosis in 53% and 59% had a mitotic index of 2 or more (0-3 scale). Median survival was 49 months (range 7-142) with a 49% mortality.

None of the fourteen independent variables analyzed in this study showed a statistically significant ($p = .05$ or less) correlation with survival. Based on this data, we conclude that at our institution there is little predictive value in the clinical, radiographic, pathologic and surgical resection variables that we have analyzed.

8. MIB-1 Staining Index of Pediatric Meningiomas

Mark M. Souweidane, MD, David I. Sandberg, MD *(New York, NY),* **James T. Rutka, MD, PhD** *(Toronto, Ontario, Canada),* **Lothar Resch, MD** *(Halifax, Nova Scotia, Canada),* **Mark A. Edgar, MD** *(New York, NY)*

Introduction: In adult meningiomas, the anti-Ki-67 monoclonal antibody, MIB-1, staining index (MIB-1 SI) correlates with biological behavior and may predict tumor aggressiveness. MIB-1 SI's of pediatric meningiomas have not been previously reported.

Methods: MIB-1 SI's were performed on paraffin sections of 14 pediatric meningiomas (ages 2-17), 5 of which contained atypical or malignant features. There were 6 recurrences following gross total resections over a 36.5 month median follow-up period. All 5 of the tumors with pathologic atypia

recurred; 1 tumor without atypia recurred. MIB-1 SI's were quantified on paraffin sections with representative histology using CAS-200 image analysis software. MIB-1 SI was defined as the number of MIB-1 positive cells divided by the total number of cells in 8 high power (400X) fields.

Results: MIB-1 SI's ranged from 1.2% to 31.6% (median 9.1%). Significant differences were found between median MIB-1 SI's for tumors with atypia (median = 12.3%, range = 7.0% to 31.6%) vs. tumors without atypia (median = 7.0%, range = 1.2% to 12.6%) using Mann-Whitney non-parametric analysis ($p < 0.02$). Significant differences were also found between MIB-1 SI's for tumors which subsequently recurred (median = 12.5%, range = 7.0% to 31.6%) vs. those which did not recur (median = 6.6%, range = 1.2% to 12.2%; $p < 0.01$).

Conclusions: In this cohort of pediatric meningiomas, pathologic atypia and tendency to recur correlate with elevated MIB-1 SI. These findings suggest that MIB-1 SI may be useful in predicting aggressive clinical behavior of these tumors.

9. Linear Accelerator-Based Stereotactic Radiosurgery in the Pediatric Population

Christopher D. Kager, MD, Jodie Ward, RT, Ronald E. Warnick, MD, Kerry R. Crone, MD, John C. Breneman, MD *(Cincinnati, OH)*

Introduction: Although many reports document the use of linear accelerator (LINAC)-based stereotactic radiosurgery (SRS) for intracranial disease in adults, few describe its indications, safety, or efficacy in the pediatric population, and these few reports generally emanate from major SRS referral centers. This study reviews our experience with the use of SRS in children, and discusses technical issues particular to the performance of SRS in children, such as proper frame and pin placement and the need for anesthesia.

Methods: Between December 1990 and February 1999, 22 children (9 boys, 13 girls, all 18 years old or younger) underwent 25 LINAC-based SRS treatments. A retrospective chart and radiographic review was performed.

Results: Seven patients underwent SRS for an arteriovenous malformation (median dose 1800 cGy, median follow-up 121 weeks). Two patients received repeat SRS for persistent AVMs. Radiographically verified cure was documented in all 4 patients who had follow-up longer than 2 years; no adverse radiation effects, hemorrhages, or deaths have occurred. Fifteen

patients were treated for a broad range of neoplasms, including angiofibroma (1), choroid plexus carcinoma (1), ependymoma (2), germinoma (1), medulloblastoma (1), metastatic sarcoma (1), and glioma (8) (median dose 1800 cGy, median follow-up 70 weeks). Eight of these patients have died; however, 3 of 4 patients treated for pilocytic astrocytoma are currently alive (median follow-up 91 weeks). Adverse effects attributed to radiation occurred in 4 of the 15 (27%) patients, all of whom received radiation to the brainstem.

Conclusions: LINAC-based SRS is a safe, effective treatment for a wide range of intracranial disease in the pediatric population. Once recognized, technical issues regarding use of SRS in children can be easily managed.

10. The Supratentorial Interhemispheric Approach to Pineal Region Tumors

Michael L. Levy, MD, J.G. McComb, MD *(Los Angeles, CA)*

While various approaches to pineal region tumors have been described, many currently employ an infratentorial supracerebellar approach. In the infratentorial approach, the reach can be considerable and the venous anatomy is encountered only at the terminal stages of the operation. For lesions extending above the tentorial notch or for paramedian lesions extending into the region of the posterior third ventricle, the approach is limited.

The supratentorial interhemispheric approach was used in 43 patients (46 procedures) over 15 years. There were 28 males and 15 females (mean age = 8.4). Pure supratentorial approaches were utilized in 81% of the approaches and combined with transcallosal or modified approaches in 19%. The three-quarter lateral position was used in 82% of cases. Pathology for this series included PNET (19 cases), Grade 1 astrocytomas (2), pilocytic astrocytomas (1), anaplastic astrocytomas (5), germinoma (4), benign teratoma (2), malignant teratoma (2), mixed teratoma (5), embryonal cell carcinoma (1), and sarcoma (2).

Transient complications included hemiparesis (in 6), short-term postoperative seizures (in 7), and visual abnormalities (in 4). Permanent surgically related morbidity was present in 2 patients, major morbidity in 1, and death in 1 with a sarcoma.

This approach allows for good exposure to lesions as caudal as the vermis. Mean operative duration was 257 minutes (significantly shorter than with

infratentorial approaches in our experience,). Mean EBL was found to be at comparable values (p>0.02) to the infratentorial approach. The supratentorial approach is a safe alternative for pineal region tumors and provides ample exposure for lesions involving the pineal and posterior third ventricular region.

11. Effects of Shunt Treatment on the GnRH System in Congenital Hydrocephalus

Ramin M. Abdolvahabi, MD, PhD†, Jeffrey J. Cortese, CST, Fernando G. Diaz, MD, PhD, Pat McAllister, PhD *(Detroit, MI)*

Children treated for hydrocephalus exhibit precocious puberty and amenorrhea, but the pathogenic mechanism for these deficits is unknown. Therefore, we studied the effects of progressive hydrocephalus and its treatment on the hypothalamic gonadotropin releasing hormone (GnRH) system and reproductive function. We treated 183 H-Tx rats (established model of perinatal onset hydrocephalus) with ventricular shunts at 6 (early shunts) and 12 (late shunts) days of age. Successfully shunted animals (18 early shunts and 18 late shunts) were sacrificed at 21 or 50 days of age, and compared to age-matched control (n = 52) and untreated hydrocephalic (n = 14) littermates. In untreated animals, hypothalamic GnRH protein levels were significantly ($p<0.05$) increased at 21 days (9.17 vs 0.97 pg/ng total protein). In addition, serum LH levels in the untreated animals increased 173% at 21 days. However, GnRH immunoreactive fibers were unaffected until 50 days of age. Early and late shunting normalized hypothalamic GnRH and serum LH protein levels by day 50 but not by day 21. Functional studies revealed normal vaginal opening and estrus cycles for animals shunted successfully at day 6 (early shunts). On the other hand, the late shunted group and shunt failures exhibited delayed vaginal opening and estrus cycle irregularities. Therefore, in this model hydrocephalus affects the function of the hypothalamic GnRH system early on without compelling morphological changes. Moreover, early shunting is more effective in restoring reproductive function in the hydrocephalic animals. Damage to the hypothalamic GnRH system may be the common denominator for hydrocephalus associated reproductive abnormalities.

† Disclosure Information

12. Hydrocephalus Induces the Proliferation of Cells in the Subventricular Zone

Kimberly D. Bingaman, MD, Viorica Pencea, MD, Marla B. Luskin, PhD *(Atlanta, GA)*

The pathological changes produced by hydrocephalus are well characterized, including axonal damage and cell death. The subventricular zone (SVZ) is an area adjacent to the ependyma of the lateral ventricle and is a recognized region of neurogenesis in both young and adult rats. Recent experiments have suggested that neural stem cells are present within this region. Due to the proximity of the SVZ to the lateral ventricles, it is possible that the SVZ could be affected by hydrocephalus. The purpose of this study was to examine the proliferative response of cells within the SVZ to experimentally induced hydrocephalus in adult rats.

Hydrocephalus was induced in adult Sprague-Dawley rats by injection of a suspension of sterile kaolin into the cisterna magna. As a control, another group received cisternal injections of sterile saline. The rats were injected ip with bromodeoxyuridine (BrdU) (a marker incorporated into dividing cells) at various times prior to sacrifice at 1, 2, or 3 weeks following induction of hydrocephalus.

Although some individual variability was present, the hydrocephalic animals incorporated BrdU into a greater number of SVZ cells than the control animals. To determine the phenotype of the newly generated cells, we utilized anti-GFAP (an astrocytic marker) and TuJ1 (a neuronal marker). These results demonstrate that a substantial proportion of BrdU+ cells are neurons, suggesting that hydrocephalus induces the generation of new neurons within the SVZ. Further studies will be required to determine whether the newly-generated neurons form functional connections or are of any benefit to the hydrocephalic animal.

13. Brain and CSF Oxygen Saturation in an Animal Model of Chronic Hydrocephalus: Response to Hyperventilation

Mark G. Luciano, MD, PhD †, Toru Fukuhara, MD, PhD, Christine L. Brant, MS, Jennifer L. Klauschie, BA, Megan E. Knoch *(Cleveland, OH)*

Introduction: Decreased cerebral blood flow and hypoxia has been considered a likely mechanism in the pathophysiology of acute and chronic hydrocephalus. We sought to detect the changes in cerebral tissue and CSF oxygen saturation in chronic hydrocephalus as a possible measure of cerebral dysfunction and potential monitor of optimal drainage.

† Disclosure Information

Methods: Eleven dogs were induced with hydrocephalus via fourth ventricular cyanoacrylic occlusion. Twelve weeks after induction, cerebral tissue in the paraventricular region and ventricular CSF oxygen saturation were measured with the Licox oxygen saturation microprobe during normal ventilation and hyperventilation. Intracranial pressure was measured simultaneously. Five surgical controls were similarly studied.

Results: Intracranial pressure was higher in the hydrocephalus group (9.3 +/- 3.4) compared to the control group (5.2 +/- 0.8) and decreased significantly with hyperventilation in both groups. In the hydrocephalus group, oxygen saturation was significantly lower in tissue ($p<0.001$)and CSF ($p<0.01$) compared to control. Hyperventilation caused a change in CSF oxygen saturation (25.0 +/- 6.7 to 19.0 +/- 7.6, $p<0.001$) but not in tissue (29.7 +/- 9.3 to 30.6 +/- 9.5). In the control group, both CSF and tissue oxygen saturation significantly decreased by induced hyperventilation.

Conclusions: Tissue and CSF oxygen saturation decreased in chronic hydrocephalus. Normal response to hyperventilation could be seen in the CSF, but not in the paraventricular tissue of the hydrocephalic animals. CSF oxygen saturation may reflect global brain oxygen delivery.

14. Supplemental Pharmacologic Therapy of Experimental Infantile Hydrocephalus

Eric M. Massicotte, MD, Marc R. Del Bigio, MD, PhD, FRCPC *(Toronto, Canada)*

Introduction: Use of adjunctive drug therapy directed toward neuro-protection and regeneration has not received much attention. We studied the effects of the calcium channel blocker nimodipine, which has been shown in other situations to be neuro-protective, and a modified form of the trophic factor ciliary neurotrophic factor (CNTF) in an experimental model of infantile hydrocephalus.

Methods: Hydrocephalus was induced in 3 week-old rats by injecting kaolin into the cisterna magna. Nimodipine experiment used 1.8 mg / day for 2 weeks delivered by osmotic mini-pump subcutaneously. A total of 36 rats were studied for five weeks. CNTF experiment used 1.4 or 14.3 µg/day, which was delivered, into lateral ventricle in shunted animals (treatment groups 12 each and 12 controls). Quantifiable behavioral and histological parameters were used as outcome measures.

Results: Nimodipine-treated rats stayed on a rotating drum 15 to 32% longer ($p<0.04$), were 27 to 47% faster ($p<0.001$) swimmers. Superior memory and learning performances based on Morris water maze were also demonstrated. Histological examination showed a thicker corpus callosum in nimodipine-treated rats. The CNTF experiment suffered several complications that confounded the results and no obvious benefit was identified with the CNTF treatment.

Summary and Conclusion: Nimodipine seems to provide a certain degree of neuro-protection, which translates into a better functional outcome. Investigation with hydrocephalic patients may be the next step in light of this animal experiment and the clinical record of nimodipine. Experimental investigation using other neurotrophic agents in the post-shunt period should be considered.

15. Impaired Motor Learning in Children with Hydrocephalus

Yuchuan Ding, MD, PhD, Q. Lai, PhD, J.P. McAllister, PhD, S.D. Ham, MD, S.S. Sood, MD, A. I. Canady, MD *(Detroit, MI)*

Shunted hydrocephalic patients have various degrees of motor and cognitive deficits. To determine the motor learning and memory impairment, shunted hydrocephalic and normal children in two age groups (averaged at 10 and 14 years old, n=32) were asked to learn a predetermined timing rhythm task (including timing duration and relative timing pattern) by pressing keys on a computer keyboard. Timing pattern is believed to be essential for any sequential movement such as playing piano, handwriting, and locomotion. In acquisition phase, goal timing intervals and feedback were presented visually to participants before and after key-press, while modeled auditory information was provided prior to each trial. A retention and transfer test without the auditory information and visualized feedback was administered 1 day later. Questionnaires and neurological exams were completed prior to each experiment. All patients physically and intellectually were able to perform the programmed task. ANOVA indicated that (1) the hydrocephalic children could improve acquisition of timing duration with practice and growth like normal children, however, they produced significantly more errors on relative timing patterns compared to controls ($p<.001$). Unlike normal children, older hydrocephalic patients did not have better performance of relative timing. (2) Hydrocephalic children in the two age groups had significant difficulties on retention and information transfer tests for relative timing pattern ($p<.001$). These results suggest that during motor skill learning in shunted hydrocephalic children, the motor timing-pattern might not be consolidated into working and long-term memory, which are attributed largely to develop an accurate motor program.

16. Arachnoid Cysts: Treatment—Cyst to Peritoneal Shunt

Timothy Burson, MD, Michael S. Muhlbauer, MD,
Robert A. Sanford, MD *(Memphis, TN)*

The treatment of arachnoid cysts remains controversial with proponents advocating surgical marsupialization versus cyst-to-peritoneal shunting. At LeBonheur Children's Medical Center/University of Tennessee the authors have consistently utilized cyst-to-peritoneal shunting as the primary treatment for symptomatic arachnoid cysts. A retrospective review from 1988 to 1999 revealed 10 patients with 11 symptomatic arachnoid cysts that were treated with cyst-to-peritoneal shunt, ages ranging from 1 month to 19 years. One teenager with a large temporal lobe cyst, had surgical marsupializtion which failed and was converted to cyst-to-peritoneal shunt. All others were treated primarily with a cyst-to-peritoneal shunt. Only 2 revisions were required, one at 2 weeks, and the other at 6 months per initial shunting. There have been no shunt infections. One patient developed an extra-axial collection on the opposite hemisphere which was treated with burr hole drainage. Follow up ranges from 6 months to 10 1/2 years. All children have been followed with serial scanning, and have shown significant decrease in size or complete obliteration of their cyst.

In all surgical series, there is a significant failure rate of surgical resection aiding to cyst-to-peritoneal shunting. The authors have found that a simple cyst-to-peritoneal shunt is a primary mode of treatment and avoids complications. It is our experience that unlike ventriculoperitoneal shunts, there is a very low long-term complication rate.

17. Minimally Invasive Craniotomy to Fenestrate Middle Fossa Arachnoid Cysts*

Michael Y. Wang, MD, Michael L. Levy, MD, J. Gordon McComb, MD

A debate centers on whether to use an endoscope or microscope for fenestration. Those who champion the use of an endoscope do so as it is claimed to be less invasive than when a microscopic approach is undertaken.

We used a 2 cm or less craniotomy in 40 children (27 males:13 females with a mean age of 5 1/2 years) to fenestrate middle fossa arachnoid cysts into the cisterns Surgical time < 2 hours. Hospital stay averaged 3.6 days, with 1.1 days in the ICU (all patients were admitted post-operatively). EBL was minimal and no one required transfusion. Complications included SDH in

* *Considered for Shulman Award*

3 patients, transient 3rd nerve palsy in 2, and a bone flap infection in 1. Follow-up averaged 30 months (4 patients lost before the end of one year. 35 patients (87%) had both improvement in symptoms and a significant decrease in size of the arachnoid cyst on post-op MRI without any further surgery. Five patients with hydrocephalus pre-operatively required CSF diversion despite adequate fenestration. This may relate to increased resistance in CSF pathways rather than the mass effect from the arachnoid cyst.

Our microscopic approach is minimally invasive and roughly comparible to that of endoscopy. In addition, the use of the microscope has advantages over the endoscope in that it allows for 3D viewing, vision is not obscured by bleeding, bleeding is more readily controlled with the availability of bipolar cauterization, orientation is less problematic and it is easier to make multiple site fenestrations into the basal cisterns which, in turn, should theoretically increase the likelihood of success.

18. Aggressive Endoscopic Management of Primary Intracranial Cysts

Michael H. Handler, MD, FACS, FAAP

The endscopic approach to intracranial operations has an inherent appeal, "minimally invasive" by leaving few traces on the skin, and yet bringing instruments and a viewing portal close to the pathology. Its utility in simpifying shunt systems involving loculated collections in the ventricles has been well demonstrated. Fifteen patients presented with signs or symptoms of elevated pressure and had 7 intraventricular cycts, 3 enlarged cavum septi pellucidi or vergae, and 5 large extra-axial arachnoid cysts. 6 had associated ventriculomegaly. Each underwent an aggressive endoscopic operation to fenestrate the cyst into the ventricular system or nearby basal cisterns (as would have been done in an open microsurgical operation). Where appropriate, a third ventriculostomy was also done. 3/3 cavum fenestrations relieved symptoms and reduced the size of the collections substantially. 5/7 intraventricular cysts were successfully and permanently drained into the ventricles, while 2 did require shunting. 5/5 fenestrations of extra-axial arachnoid cysts into basal cisterns failed, with patients developing large subcutaneous fluid collections under pressure, and needed shunting. 4 of the 6 patients with hydrocephalus ultimately required shunting: 2 with posterior fossa arachnoid cysts, and 2 with intraventricular cysts. Aggressive endoscopic surgery appears promising when a collection can be widely drained to the ventricles, but very unlikely to relieve pressure from an extraaxial collection.

19. The Importance of Shunt Valve Position in Determining Outflow Rates

Paul C. Francel, MD, PhD †, F. Alan Stevens, BS, Paul Tompkins, MS
(Oklahoma City, OK)

Although many shunt valves now have an integral siphon control device, overdrainage continues to be a serious complication of shunt placement. One important, but sometimes overlooked, factor in overdrainage is the ablolute position of the valve body with respect to the reference position for measurement of intracranial pressure (ICP). Once the inlet catheter is positioned, it becomes the reference position for pressure measurements. This study was performed to underscore the importance of valve position with regard to the reference for ICP measurement (i.e. the inlet catheter tip). Valves from Medtronic PS Medical, Heyer-Schulte NeuroCare, Radionics and Elekta (OSV II) were tested on the bench to determine flow characteristics with the valve in different positions relative to the reference position. For the Medtronic PS Medical Delta, Heyer-Schulte NeuroCare, and Radionics Equi-Flow valves, flow rates at the "0" reference position were 50 ml/hr. As the valves were raised incrementally to 4cm above this position, flows decreased to 0-25ml/hr. As the valves were lowered incrementally to 8cm below the reference position, flows increased to 160-240ml/hr. The OSV II valve maintained a constant outflow of approximately 28ml/hr over the range of positions used herein. This study shows the critical importance of shunt valve position in determining outflow rates for hydrocephalic shunt valves. Extreme caution should be exercised in locating the valve assembly at positions other than horizontal with the inlet catheter tip.

20. Long-Term Outcome Analysis of Initial Neonatal Shunt Valves

Shenandoah Robinson, MD, Bruce Kaufman, MD, T.S. Park, MD
(St. Louis, MO)

Objective: Although ventricular shunts have revolutionized the treatment of hydrocephalus, a relatively high incidence of malfunction persists. We evaluated the long-term outcome of initial neonatal shunts to determine whether the initial shunt components, including the valve opening pressure, affected the shunt revision and complication rate.

Methods: Charts were retrospectively reviewed and a multivariate analysis was performed.

† Disclosure Information

Results: Two hundred consecutive infants < 1 year old underwent primary ventriculoperitoneal shunt placement. Of the 158 patients with at least 6 months documented follow-up, the mean follow-up was 40 months (range 6-99). Etiology, gestational age, catheter location, and surgeon were not independent predictors of shunt survival. The valve opening pressure significantly affected shunt survival. The four year shunt survival rate was 65% for high, and 33% for low pressure valves (p=0.0003). Patients with a low pressure valve had twice the revision rate of patients with a high pressure valve (0.52 versus 0.23 rev/pt year of follow-up). This discrepancy was more apparent after the shunt was in place for at least a year: the low pressure valve revision rate was four times higher (0.23 versus 0.06). The incidence of overdrainage problems (slit ventricles and craniosynostosis) was also significantly higher for patients with low pressure valves (p=0.0002).

Conclusions: This retrospective review of factors that predispose to shunt malfunction or overdrainage suggests that a low pressure valve results in a significantly higher rate of shunt revision and related overdrainage complications compared to a high pressure valve. A prospective study to test the validity of this conclusion is underway.

21. Direct Heart Shunt: A Viable Alternative?

Robert F. Keating, MD, Frank Midgley, MD, David W. Pincus, MD, PhD, Leon E. Moores, MD, Philip H. Cogen, MD, PhD ***(Washington, DC)***

Numerous sites for CSF diversion are available. While the ventriculoperitoneal shunt remains a mainstay, certain patients require an alternative approach. This frequently requires placement of a ventriculoatrial shunt. To investigate the viability of a direct heart shunt (DHS) as an alternative route for such patients, we reviewed our institutional experience with this approach.

A retrospective review of 52 patients (33 males, 19 females) receiving a DHS over a two year period (1976-77) at CNMC was undertaken. The average age at insertion was 3.6 yr (4m-12y) with 15/ 52 (29%) being placed initially and the remainder representing conversions from VP shunts. The etiology involved myelomeningocele (37%), congenital (35%), post-infectious (14%) orIVH (6%). Direct heart shunts were placed by a combined neurosurgical and cardiothoracic team, and frequently involved the use of a Portnoy ventricular catheter, Pudenz valve, Raimondi shunt tubing as well as a silastic pouch for redundant tubing to allow for growth. Perioperative morbidity was minimal with no deaths, rare pericardial effusions and occasional pneumonia, with an average length of stay of 6 days

(4-14d). Average long-term follow-up of 10.1 years (2-20y) demonstrated that 21/52 DHS (40%) were still functioning, with 9/52(17%) in place greater than 15 years and an average of 0.9 revisions per patient. DHS that were converted to VPS (31/52) were secondary to shunt infections. Thus, for the patient with minimal venous access in need of an alternative site for CSF diversion, the DHS may be considered an alternative.

22. 45 Cases of Percutaneous Endoscopic Recanalization of Catheter (PERC Procedure)—A Long Term Study

Jogi V. Pattisapu, MD, Eric Trumble, MD, Kay Taylor, RN, Denise Howard, CST, Tina Kovach, RN, Annette Arbogast

Introduction: Percutaneous endoscopic recanalization of catheter (PERC procedure) for certain cases of proximal shunt malfunction has been performed at our institution since 10/96. This study reviews the present outcome of these children using this technique, with an analysis of the critical factors affecting the outcome of the procedure.

Method: 38 children (mean age 45 months) underwent 45 PERC procedures for proximal shunt malfunction under general anesthesia in the operating room as per the IRB protocol (as previously described). During the study period of 33 months, 220 cases of standard open shunt revisions were performed by the authors. The children were evaluated at 2,4,8,12, and 24 weeks, and every 6 months thereafter, with clinical assessments and CT/MRI studies.

Results: There were 10 failures in this study of 45 cases (78% success rate) with a mean follow-up of 13.3 months. Most malfunctions occurred between 5-7 months, mainly in ex-premature infants, and children who suffered previous infections. 5 children underwent 7 repeat PERC procedures, and 3 of these children failed the repeat attempt, requiring an open shunt operation. There were no complications such as infections, bleeding, seizures, heat damage, etc.

Conclusion: The PERC procedure has been very effective in selected cases of proximal shunt malfunction. This long term study proves that this technique should be included in our armamentarium for shunt management.

23. Addition of Elemental Iodine to Surgical Irrigant for Shunt Infection Prophylaxis

SooHo Choi, MD, J. Gordon McComb, Mike L. Levy, Ignacio Gonzales, Berislav Zlokovic, Roger Bayston

Intoduction: Since elemental iodine (I2) can kill a broad spectrum of bacteria, fungi and viruses; we have added this compound at a concentration of 4 parts per million (ppm) to surgical irrigation during shunting procedures. Despite this, shunt infections still remain problematic. By increasing the concentration of elemental iodine, the exposure time needed to kill bacteria is reduced. Therefore, we wanted to ascertain the highest concentration of iodine that could be safely used as a surgical irrigant.

Methods: Twenty -one adult male Wistar rats (200-260 grams) underwent a craniectomy. Brains were then irrigated for one hour with Ringer's lactate alone or containing various concentrations of iodine. After 72 hours of observation, the brains were examined histologically.

Results: Compared to the control group, no histologic changes were noted at 5, 10, 20 and 50 ppm. However at concentrations of 100 and 1000 ppm, severe necrosis was observed. Subsequently, time kill studies were performed with Staphylococcus aureus and epidermidis using the above concentrations of iodine as well as cefazolin (1 mg/ml) and bacitracin (50 units/ml). Even after 15 seconds of exposure, a dilute solution of iodine (20 ppm) achieved a 99.99% killing rate with an inoculum of 100 million bacteria (zero growth detected). In fact at concentrations of 20, 50, 100 and 1000 ppm; no bacterial growth was noted for all exposure times. In contrast, conventional antibiotics allowed significant growth even after 24 hours of exposure.

Conclusions: Based upon these findings, we have subsequently increased the concentration of iodine in our surgical irrigation to 40 ppm for shunt operations in hopes of maximizing the bactericidal effect and further decreasing our shunt infection rate.

24. Pediatric Shunt Infections: A Prospective Analysis

Elizabeth B. Claus, PhD, MD, Eileen Ogle, PA, Charles Duncan, MD
(New Haven, CT)

Background: Little prospective data from this decade exist for ventricular shunt infections in children.

Methods: This study uses logistic and Cox proportional hazards regression models to examine the incidence, timing, and risk factors associated with the development of pediatric shunt infection. The sample includes all individuals aged 18 years or younger undergoing surgical placement of a cerebrospinal (CSF) shunt at Yale-New Haven Hospital from January 1, 1996 to June 30, 1999.

Results: Patients (n=105) were followed for an average of 432 days. The average age was 4.9 years; the majority of patients were Caucasian (68%), female (51.5%), had congenital hydrocephalus (63.3%) and a ventriculo-peritoneal shunt (95%), and did not have a previous shunt (51%) or a myelomenigocele (85%). Thirteen of the 105 children developed a shunt infection; all but two did so within the first year after placement of the shunt. The one, six, and tweleve month infection rates are 5.0% (standard error = 2.2%), 11.0% (3.3%), and 15.5% (4.4%), respectively. By three years, approximately 20% had an infected shunt. Potential risk factors such as age, sex, and the presence of a myelomenigocele were examined for an association with the incidence and timing of shunt infection, however, no significant relationships were noted.

Conclusions: Approximately 20% of children will develop an infection after placement of a CSF shunt. The majority of these infections occur within the first year of placement.

25. Soluble Adhesion Molecules Are Elevated in the Cerebrospinal Fluid of Children with Moyamoya Syndrome

R. Michael Scott, MD, S.G. Soriano, D.B. Cowan, M.R. Proctor
(Boston, MA)

Moyamoya syndrome, a progressive cerebrovascular occlusive process presenting with recurrent strokes, may have an inflammatory component or etiology. Adhesion molecules mediate an inflammatory process during cerebral ischemia (1,2). We measured the levels of soluble isoforms of endothelial adhesion molecules, VCAM-1, ICAM-1 and E-selectin in the serum and CSF in children with Moyamoya syndrome.

Methods: Serum and CSF were obtained from children with Moyamoya syndrome (n=21) and congenital spinal deformities (n=21) undergoing neurosurgical procedures. Soluble VCAM-1, ICAM-1, and E-selectin levels were measured by enzyme-linked immunoassay. Data were compared by Pearson product-moment correlation coefficient and Mann-Whitney U test, with a $p < 0.05$ reaching statistical significance.

Results: Compared to the control group, children with Moyamoya syndrome had significantly elevated levels of sICAM-1, and sE-selectin in the CSF and sVCAM-1 in the serum ($p < 0.05$). However, there were no differences in the CSF sVCAM-1 and serum levels of sICAM-1 and E-selectin between the two groups and there was no correlation between the age and levels of both serum and CSF adhesion molecules in both groups.

Conclusions: Our study demonstrates increased CSF levels of soluble endothelial adhesion molecules, suggesting that there is an on-going inflammatory process in children with Moyamoya syndrome. These soluble adhesion molecules might serve as indicators and potential targets for pharmacological interventions. References:1. Ann.Neurol. 39:295-301, 1996 2. Stroke 30:134-139, 1999

26. Multimodality Treatment in Pediatric AVMs Improves Outcome

Brian L. Hoh, MD, Christopher S. Ogilvy, MD, William E. Butler, MD, Christopher M. Putman, MD, Jay S. Loeffler, MD, Paul H. Chapman, MD *(Boston, MA)*

Introduction: The management of pediatric AVMs has significant morbidity and mortality. In the two largest published series (Humphreys et al.,1996, Lasjaunias et al.,1995) management related morbidity was 18 to 28% and mortality 11 to 16%. We use a multidisciplinary team approach of embolization, microsurgery, and radiosurgery, either alone or in combination, to minimize complications and maximize efficacy.

Methods: 1) From 1991-1998, 37 consecutive pediatric AVMs were treated at our institution. 2) Mean followup was 42 months (range 3-111 months).

Results: Twenty patients were managed surgically (6 with preoperative embolization), 13 with radiosurgery (2 with preoperative embolization), and

4 with embolization alone. Location, Spetzler-Martin grade, and age determined treatment modality (p less than 0.05 for each).

All 20 patients treated surgically showed complete obliteration on postoperative angiogram (100%). In 5 patients, planned partial obliteration (palliative treatment) was performed (4 embolization, 1 partial radiosurgery). Overall radiographic efficacy was 78% with complete obliteration, 3% with greater than 95% obliteration, and 19% with less than 95% obliteration, with 12 patients (32%) being followed after radiosurgery and 5 patients (14%) being followed after planned partial obliteration.

Clinical outcome was associated with Spetzler-Martin grade, but not treatment modality, age, or history of hemorrhage. For the total group there was 94% excellent/good outcome (GOS 4, 5), 3% fair outcome (GOS 3), and 3% mortality. There were 2 complications (5%) and 2 recurrent hemorrhages (5%). Nine patients presented with seizures and all but one are cured (89%).

Conclusions: A multimodality approach to managing pediatric AVMs can achieve excellent results with minimal morbidity and mortality.

27. Is There a Difference in Incidence and Outcome Between Children and Adults with Vasospasm*

Larry T Khoo, MD, Michael L. Levy, MD, Steven Giannotta, MD, J. Gordon McComb, MD *(Los Angeles)*

Subarachnoid hemorrhage (SAH) secondary to ruptured cerebral aneurysms in the pediatric age group are quite uncommon. Little is known as to the incidence of vasospasm and its subsequent sequelae in this group of patients as compared with adults.

We retrospectively reviewed our series of 55 patients collected over a 15 year period with documented SAH and found that 8 (14%) had angiographic evidence of vasospasm. The mean age of these patients was 12 years with a male-female ratio of 5:3. In 4 of the 8 patients the vasospasm was documented on pre-operative angiography and occurred at a mean interval of 6.2 days following the initial SAH. With the remaining 4 patients it was only on post-operative angiography that vasospasm was noted with a mean interval of 13.5 days following the initial SAH, and 3.5 days following craniotomy to treat the aneurysm. Three of the 8 patients were clinically asymptomatic in spite of their vasospasm, while the other 5 had changes in

* *Considered for Shulman Award*

their neurologic status (progressive lethargy and/or paresis) with 3 occurring before surgical intervention and 2 following. The mean age for these 5 patients was 14 years.

Based upon one of the largest series of pediatric aneurysms to date the time-course of vasospasm following SAH is similar in children and adults as is the incidence of both angiographic (14%) and clinical (9%) vasospasm associated with childhood aneurysmal SAH. There was no significant relationship between patient age and the incidence of vasospasm.

28. Posterior Cervical Fusion in Children and Adolescents

William R. Boydston, MD, Roger J. Hudgins, MD

We present a series of 53 children who underwent posterior cervical fusion between 1989-1998. This included 35 males and 18 females with a mean age at operation of 7 years (range 0.5yrs to 19yrs). 10 Children were syndromic with diagnoses including Trisomy 21, Morquio's, chromosomal deletion and Goldenhar's. 5 Patients had a form of osteochondrodysplasia (2 Kniest's Syndrome, 2 spondyloepiphyseal dysplasias and 1 chondrodysplasia punctata) and underwent posterior cervical fusion following decompressive procedures. An additional nine children had congenital anomalies including os odontodeum, hypoplastic dens and absence of upper cervical posterior elements that predisposed them to cervical instability. The remainder of the patients required fusion secondary to traumatic fracture and/or ligamentous injury. This included 2 atlanto-occipital dislocations and 6 children with fractures below the level of C2. As expected the later group tended to occur in older age patients (range 12-19 years).

Four patients required reoperation; two sencondary to nonunion and one due to a fracturted fusion mass one year after initial operation. The remaining patient had recurrent C1-2 subluxation secondary to an inappropriate size of the original posteior autologous bone graft. 3 Children required treatment for halo pin site infections and 3 others had halo slippage necessitating readjustment. There was no increased incidence of neurologic injury following fusion.

As previously reported posterior cervical fusion tends to have a high success rate in children. We did not observe any difference in treatment success between nonsyndromic and syndromic children or those with disorders of bone and cartilege.

29. Spinal Arachnoid Cysts in Children*

Hulda Magnadottir, MD, Michael L. Levy, MD, Mark Mittler, MD, J. Gordon McComb, MD

Over a 19 year period spinal arachnoid cysts were found in 21 patients ranging in age from 5 months to 18 years (mean = 6 years) presenting to our institution. There were 5 males and 14 females in the series. 17 patients had intradural arachnoid cysts and 2 had extradural arachnoid cysts. Presenting symptoms included radiculopathy (one patient), progressive weakness (five), scoliosis (three), progressive spasticity (three), bladder abnormalities (two), progressive sensory deficits (two). Two patients were neurologically intact at presentation. The distribution of lesions included the cervicomedullary junction (one patient), cervical (one), cervicothoracic (three), thoracic (four), thoracolumbar (three), lumbar (one), lumbosacral (four), sacral (one), and thoracolumbosacral spine (one). Six of the cysts were associated with closed neural tube defects (NTD), six with open NTD, and nine were isolated.

Fourteen of the patients with intradural cysts underwent fenestration alone. One patient required a second fenestration given continued progression of deficit. One patient required a fenestration and shunt placement and two required shunt placement alone. Both extradural cysts were resected. Surgical complications included CSF leakage (one patient) and transient weakness (one). No permanent complications were noted. Postoperatively the patient with radiculopathy improved. Four patients with progressive weakness improved. One patient with scoliosis improved. No patient with progressive spasticity improved, all remaining stable. Both patients with bladder abnormalities improved as did both patients with progressive sensory deficits.

Spinal arachnoid cysts are frequently associated with closed or open NTD.. Intervention can lead to the resolution of neurologic compromise in these patients with minimal morbidity.

30. Spina Bifida Outcome: A 25 Year Prospective

Robin M. Bowman, MD, David G. McLone, MD, PhD, John A. Grant, MD, Tadanori Tomita, MD *(Chicago, IL)*

Open spina bifida is the most complex congenital abnormality compatible with long- term survival. With the conviction that the long-term outcome would be good, we have aggressively treated all children born with open spina

bifida in a non-selective manner. Our original cohort, born between 1975-1979, has now reached early adulthood. This report outlines the 20-25 year outcome for this group of patients.

Of the initial 118 children, 71 patients (38 females and 33 males) were available for our review. Nineteen patients have been lost to follow-up and 28 patients have died. All patients included in this study completed a mailed questionnaire. Of the 71 patients, 59 are actively followed in our multidisciplinary Spina Bifida clinic. Data was collected on current motor level, shunt status, education and employment, seizure history, mobility, bladder and bowel continence, history of tethered cord and/or scoliosis, latex allergy and history of posterior fossa decompression, tracheostomy and/or gastrostomy tube.

At least 75% of children born with open spina bifida can be expected to reach their early adult years. Although it is not the natural history, late deterioration in patients with myelodysplasia is common. Once deterioration is noted, a rigorous search for the cause must be undertaken. Physicians, especially neurosurgeons, must be prepared to care for this unique group of adults with chronic neurologic conditions. One of the greatest challenges in medicine today is establishing a network of care for these adult patients with spina bifida.

31. Delayed Loss of Ambulation in Patients with Open Neural Tube Defects*

Sooho Choi, MD, Richelle A. Lampa, Shervin Aminpour, Michael L. Levy, MD, J.G. McComb, MD *(Los Angeles, CA)*

Most series of patient with open neural tube defects evaluate long term follow-up in both intelligence and ambulation. Ambulation is considered regarding orthotic assistance and whether they are non-ambulators, household ambulators, or community ambulators. We attempted to analyze the frequency of those patients who initially ambulate and lose that ability over time. We retrospectively re-evaluated a series of 371 patients with open neural tube defects who were treated and followed at our institution. Of these, 30 were infants, 80 were community ambulators and 167 walked with assistive devices. 61 patients never walked. 33 remaining patients all were initial community ambulators who subsequently became unable to walk.

There were 18 females and 15 males. All 33 patients had shunts placed. Thoracic level lesions are present in 24%, thoraco-lumbar levels in 18%,

lumbar levels in 24%, lumbosacral levels in 27% and sacral lesions in 6%. Patients stopped ambulating at a mean age of 10.8 years plus or minus 2.3 years. Of these patients, 13 remain household ambulators but have had significant change in their ability to ambulate. In the initial series 13 patients were able to ambulate with AFOs, 2 with long-legged braces, 10 with HKAFOs, 7 with KAFOs and 1 with a TLSO. In conclusion, 9% of our current population have become unable to ambulate at an age of 10 years. Other large series need to be evaluated to determine if this in fact reflective of the overall population or a phenomena specific to our own institution and economic situation.

32. Split-cord Malformations in Children*

Hulda Magnadottir, MD, Mark Mitler, MD, Michael L. Levy, MD, J. Gordon McComb, MD

We reviewed our series of split cord malformations to evaluate the presentation and outcome of these more complex defects, over a nine-year period. The series included 30 patients (20 females and 10 males) with a mean age of 6.1 years (SD = 4.4). Fourteen patients had split cord malformations alone, 1 had an associated syrinx that required surgery, 15 had associated tethering, and 1 was associated with a lipomatous malformation. One was at the thoracic level, 3 at the thoracolumbar level, 11 at the lumbar level, 6 at the lumbosacral level, and 9 involved the thoracic, lumbar, and sacral spine. At presentation 1 patient had an associated open neural tube defect, Chiari II malformation and required shunting for progressive hydrocephalus.

Presenting signs/symptoms included equinovarus deformities in 4 (1 bilateral), scoliosis in 8, scoliosis with lower extremity abnormalities in 5, lower extremity abnormalities alone in 2, associated hip abnormalities in 2, and normal exams in 9. With regard to bladder function, preoperative status was unknown in 7 children. Two children had neurogenic bladders on admission and one had progressive abnormalities. The other 20 had normal exams.

Following surgery 2 patients with normal exams on admission had urologic compromise (1 transient, 1 permanent). A patient with progressive bladder abnormality had significant improvement and 2 patients with preoperative bladder abnormalities had no improvement. With regard to neurologic findings 20 patients remained at their neurologic baseline postoperatively (including 6 patients with weakness on admission had no change in exam

* *Considered for Shulman Award*

post-operatively), 7 had improvement in function, 1 had transient weakness, and 2 had permanent weakness. Surgical complications (16%) included 3 transient pseudomeningoceles (2 with leakage, 1 with subsequent infection), 1 ischemic cord injury, and 1 superficial wound infection.

Split cord malformations are frequently associated with motor and/or bladder compromise at presentation. Preoperative motor abnormalities may respond to surgical intervention whereas preoperative bladder abnormalities are less likely to do so. Associated tethering did not impact on postoperative function.

33. Radiographic Evaluation for Spinal Dysraphism in Long-term Follow-up of Patients with Imperforate Anus

Tanvir F. Choudhri, MD, Ryan McTaggart, E. Sander Connolly, Jr., MD *(New York, NY),* **Indro Chakrabarti** *(Los Angeles, CA),* **Alex Khandji, MD, Neil Feldstein, MD** *(New York, NY)*

Introduction: While the association of spinal dysraphism with anorectal malformations has been documented, the role of prophylactic surgical untethering remains controversial. Previously we presented fourteen patients who underwent surgical repair of imperforate anus (five high, four intermediate, and five low; average age 2.8 months) between 1976 and 1986, before routine screening for intraspinal pathology. Through telephone follow-up, no patient reported progressive symptoms suggestive of spinal dysraphism. In this report, we extend the study with radiographic and clinical evaluations for spinal dysraphism.

Methods: All fourteen patients received spinal MR imaging and a clinical evaluation directed towards neurological deficits, scoliosis, genitourinary dysfunction, back/leg pain, lumbosacral cutaneous findings, and lower extremity orthopedic disorders.

Results: The MR imaging revealed three patients (21%) with occult spinal dysraphism: two with a thickened/fatty filum terminale and one with a low-lying (L3) conus. With a mean 17.7 years follow-up, no patient experienced progressive signs or symptoms suggestive of a tethered spinal cord. Although some patients had bowel dysfunction (6), scoliosis (4), lower extremity orthopedic abnormalities (4), and/or incontinence (2), all these findings were long-standing and either static or improving.

Discussion/Conclusion: The incidence of radiographic spinal dysraphism in this study is in the expected range for this population. While the number of

patients are not enough to make formal conclusions, the absence of progressive symptoms in patients with spinal dysraphism with long-term follow-up suggests that in this population prophylactic surgical untethering may not be warranted.

34. Division of the Transverse Sinus and Tentorium Cerebelli to Allow Vault Expansion and Posterior Fossa Decompression in the Treatment of Chiari II Malformation

Timothy Burson, MD, Michael Muhlbauer, MD, Robert Sanford, MD, Raymond Fryrear, MD

Although somewhat controversial, a number of children with Chiari II malformation will require posterior fossa decompression for brain stem symptoms such as apnea and vocal cord paralysis. The complex anatomy in these children inhibits the amount of decompression that can be achieved and, therefore, the outcome is often poor. This report describes an alternative technique that includes dividing the transverse sinus and tentorium cerebelli. We have treated 4 children who had Chiari II malformation with ligation of the non-dominant transverse sinus and division of the tentorium. A dural patch graft was then placed to allow expansion of the vault and decompression of the posterior fossa. The age range was 4 days to 12 years (mean 3.6 years), and all children were symptomatic. Three of the 4 children treated according to this alternative protocol have done extremely well without a recurrence of crowding; one child responded well to the technique, yet expired due to complications unrelated to the procedure. The 3 survivors have had varying degrees of improvement in breath holding spells, apnea, and vocal cord dysfunction. The outcome in these patients has been favorable with the brain stem and cerebellar decompression greatly enhanced in each instance. It is our conclusion that this modified posterior fossa decompression can offer an alternative treatment for Chiari II malformation and improve the long term outcome.

35. Anterior Fontanelle Encephalocele

Michael J. Burke, MD, Ken Winston MD *(Denver, CO)*

Encephaloceles at the anterior fontanelle are rare. A literature review revealed only five cases reported to date. All were described prior to the availability of MR imaging. We report three patients (2M, 1F) with this lesion. Prenatal history and maternal health was normal. There were no chromosomal abnormalities or other system malformations found in the patients. All lesions were skin covered. Preoperative MRI revealed the defects were meningoencephalocystoceles with the body of one lateral ventricle involved in the malformation. Additional brain malformations were noted remote from the defects. All were closed using standard techniques shortly after birth. Two patients required CSF diversion at the time of repair. All three ultimately underwent ventriculoperitoneal shunting. All children have severe intellectual and developmental delay. MR images, operative photos, and literature review will be presented.

36. Plagiocephaly: Orthotic and Non-Orthotic Treatment, a Three-year Follow-up

S. David Moss, MD, Mohammed Jalaluddin, MD *(Phoenix, AZ)*

The best treatment of plagiocephaly remains controversial. Recent studies have succeeded in shifting treatment of plagiocephaly from surgical to non-surgical means. Cranial remodeling orthotic devices (headbands, helmets, etc.) have been reported to correct non-synostotic plagiocephaly. The cost of orthotic treatment has risen placing increasing burdens on third party payers and patients without insurance coverage. Effectiveness of the orthotic treatment of non-synostotic plagiocephaly has been shown to be similar to repositioning and neck stretching therapy. The first phase of a non-orthotic treatment study was initiated in June 1995 at Phoenix Children's Hospital. All new patients were categorized into two groups, those with mild-moderate asymmetry and those with moderate to severe asymmetry. Categories were determined by cephalic measurements. The patients with moderate to severe asymmetry were offered a cranial remodeling device for treatment. Those patients with mild to moderate asymmetry were observed with repeated cranial measurements. Patients treated with non-orthotic repositioning were compared to our data published in 1993 for those treated with external cranial orthosis and were found to be very similar in the amounts of improvement seen. A follow-up study of these patients at three years of age is presented showing continued similarity in the two treatments. Patients treated with orthotic devices that are now over five years old are also presented to show the outcome of long term orthotic treatment. Study data and results are presented and discussed.

37. Tensile Strain Induces Increase in Intracellular Calcium Concentration in Immature Rat Cranial Sutures

Karsten Fryburg, MD, Jack Yu, James Borke, Ann-Marie Flannery

Introduction: Calvarial morphogenesis produces a wide variety of shapes while conserving the skull patterns. The emerging concept is that cranial growth is a secondary process dictated by the growth of the brain. Since the content of the neurocranium is non-compressible, wall tension increases rapidly during cerebral development unless the cranium expands. The cranial sutures are mainly responsible for cranial bone growth and most likely to respond to increased tension.

The intracranial pressure is remarkably constant between individuals with different head shape and volumes. Alterations in CSF circulation before closure of cranial sutures leads to an increase in head circumference to accommodate the increased volume of CSF under constant ICP.
The working hypothesis as the mechanism of calvarial morphogenesis is that tensile strength will induce cellular alterations as part of a complex adaptive process. One such parameter is intracellular calcium concentration. We measured, in real-time, the intracellular calcium concentration changes as a result of wall-tension.

Methods: Immature coronal sutures were incubated in DMEM, then calcium-sensitive, membrane-permeable FURA-2/AM was added. The intracellular calcium-concentration was measured by fluorescence spectrophotometry and expressed as the ratio of Ca2+ - bound (F1) and free FURA (F2) fluorescence. The data were acquired in real-time as cells were stretched.

Results/Conclusion: Tension induces immediate, reversible increase in the intracellular Ca2+ concentration in sutural cells. This is shown by the change of F1/F2 from 1.36 to 1.53 (SD +/- 0.013, P< 0.0001 Student t-test). A significant change in an intracellular second messenger may lead to a multitude of cellular responses to adapt.

38. Transplanted Demineralized Bone Graft in Cranial Reconstructive Surgery—Five Year Follow-up

Mohammed Jalaluddin, MD, S. David Moss, MD *(Phoenix, AZ)*

Neurosurgical reconstruction of cranial deformities and synostosis is occasionally accompanied by incomplete bone growth to cover all areas of cranial vault that have been exposed in the correction. Some synostosis repairs require more bone than is available using the child's natural skull due to the restrictive nature of the synostosis birth defect. Rib and iliac crest autografts have been used with success. These grafts must be harvested from another surgical site and often result in an irregular surface that patients may find unacceptable. A split-thickness skull graft is the cranioplasty of choice but children under the age of 5 to 6 years may lack the skull thickness needed to use this technique. Transplanted demineralized bone grafts were used in 116 patients over the last nine years at Phoenix Children's Hospital for reconstruction of the cranial vault for patients with synostosis and skull defects following prior surgical repairs and following skull tumor removal. Initial success of this graft technique has been previously published. We present a five year follow-up analysis. All save two grafts have resulted in complete closure of the defect and radiographic examination indicates that natural bone has replaced the grafts. No infections or rejections were experienced. These patients are presented including the techniques of usage of transplanted demineralized bone in cranial reconstruction.

39. Pediatric Neurosurgeons Demonstrate Increased Satisfaction with Workload and Anticipate Modest Growth in the Workforce

Ann Marie Flannery, MD

Background: The need for Pediatric Neurosurgeons and the work load of currently practicing Pediatric Neurosurgery (PN) has been studied by survey for the last 3 years.

Methods: A survey was administered to attendees at the joint section on PN in 1997, 1998 and 1999. Results were tabulated and compared.

Results: The number of respondents has risen over 3 years from 54% of attendees in 1997 to 67% in 1999. The geographic and age distribution of respondents is roughly the same over all 3 years, however, members of the section are more likely to respond (74%-71 of 96) than nonmembers (51%). Respondents indicate a need for 58 Pediatric Neurosurgeons over the next 10 years. Neurosurgeons reporting insufficient work load has declined from

42% in 1998 to 31% in 1999, while the percentage reporting an appropriate work load has risen from 39% in 1998 to 54% in 1999. In 1997, 10% were too busy, 19% in 1998 and 15% in 1999. A median case number in each group has increased. Geographic distribution of work load and workforce will be discussed. In 1999, 65% of respondents had fellowships, however, about one third of those in academic practice did not have a fellowship. Of responding residents, 23 of 24 plan to practice PN.

Conclusion: This survey demonstates a potential for modest growth in the workforce over the next 5-10 years, and increased satisfaction with workload over the last 2 years.

40. Shunt Pseudotumor

Timothy Burson, MD, Michael S. Muhlbauer, MD, Robert A. Sanford, MD *(Memphis, TN)*

A retrospective review of 1,032 shunted patients at LeBonheur Children's Medical Center/University of Tennessee revealed 4 cases of pseudotumor in shunted patients The authors define shunt pseudotumor as a chronic increased intracranial pressure manifest by headaches and papilledema in a shunted hydrocephalic patient with a functioning shunt. All patients had (1) smaller than normal ventricles, (2) a functioning, non-siphoning shunt, and (3) chronic increased intracranial pressure documented by papilledema unresponsive to medical therapy (Diamox and short courses of Decadron). Intracranial pressure monitoring was performed in all patients, and documented a resting pressure inappropriate for opening pressure of the shunt valve, but plateau waves while asleep. One patient demonstrated a unilateral transverse sinus with marked increase in pressure with head turning occluding that sinus. All patients demonstrated serial head growth within the normal range for many years. Two of the children had been treated 4 and 5 years previously with an anti-siphon device for slit ventricle syndrome. All 4 children had resolution of their papilledema and headaches following cranial vault expansion. Theories as to the etiology and relationship to pseudotumor will be discussed.

41. Quantitative Diffusion MR Imaging of Pediatric Intracranial Abscesses

Rimas P. Gilvydis, MD, Robert McKinstry, MD, PhD, Benjamin Lee, MD, Tae Sung Park, MD, Bruce A. Kaufman, MD *(St. Louis, MO)*

Introduction: Diffusion MR imaging evaluates microscopic motions of water in tissues. Therefore, it may provide additional information in the evaluation and management of intracranial abscesses.

Methods: We retrospectively evaluated 5 patients (ages: 5 days to 16 years) with intracranial abscesses. Four patients had intraaxial abscesses confirmed by either surgical drainage (n=3) or lumbar puncture (n=1). The remaining patient had subdural empyemas diagnosed by lumbar puncture. Each patient was evaluated with routine and diffusion MR imaging both prior to and following therapeutic intervention. Apparent Diffusion Coefficient (ADC) images were computed and region-of-interest (ROI) analysis was performed.

Results: Ring enhancement was detected in 4 of 5 patients on pre-treatment scans. DWI images were abnormal in all patients. Quantitative evaluation showed approximately 60% reduction of abscess ADC compared with contralateral control regions.

Discussion: Pretreatment ADC images were superior to T2-weighted images for delineation of the abscess cavity from surrounding edema. In one patient, the abscess was detected by diffusion 7 days prior to contrast enhancement. The region of reduced diffusion closely paralleled the size of the contrast enhancing portion of the abscess. When fluid-debris levels were detected, the ADC values for the supernatant fluid were increased (similiar to CSF),whereas the debris ADC values remained decreased.

Conclusion: Diffusion imaging yields information unavailable with routine MR imaging of intracranial abscesses. Further study is needed to determine if diffusion can constantly detect abscess formation prior to contrast enhancement.

42. Intrathecal Baclofen for the Treatment of Dystonia

David H. Shafron, MD †, A. Leland Albright, MD, Margaret J. Barry, MS, PT *(Pittsburgh, PA)*

Introduction: The safety and efficacy of intrathecal baclofen (ITB) delivered by an implantable pump for the treatment of dystonia was studied at Children's Hospital of Pittsburgh beginning in 1995.

† Disclosure Information

Methods: Patients were typically screened with a trial of ITB delivered via an external lumbar catheter, beginning at 200 ug/day, increasing the dosage until a desired effect or intolerable side effects were seen, or to a maximum of 900 ug/day with no response. Pumps were implanted in those with a beneficial effect. Efficacy was measured utilizing a dystonia scale. Patients were evaluated for efficacy and complications at all follow-up visits (routine, for pump refills, or dosing changes).

Results: Seventy patients were evaluated. Only three patients did not have a therapeutic response to ITB, and did not receive a pump. Of the remainder, greater than 90% have achieved a long-term reduction in dystonic movements, improving quality of life and easing care of these patients. Doses required to achieve and maintain a response were considerably higher than for patients with spasticity alone, and escalating doses were often needed to achieve an optimal response. A significant rate of complications was seen, including infection requiring hospitalization and/or removal (20%), intrathecal catheter disconnection, migration, or blockage (20%), CSF leak (not due to disconnection) requiring intervention (10%), and proximal pump or catheter dysfunction (7%).

Discussion: ITB is effective for the treatment of dystonia refractory to medical therapy. There is a high risk of complications. Strategies for optimizing response and reducing complications will be discussed.

43. Staged, Tailored Hemispherectomy in the Management of Pediatric Epilepsy: A Rational Approach for Hemispheric Malformations of Cortical Development

Howard L. Weiner, MD, Henry Woo, MD, Mary L. Zupanc, MD, Orrin Devinsky, MD, Jeffrey H. Wisoff, MD *(New York, NY)*

To improve outcome following surgery for unihemispheric epilepsy, we utilized a staged approach consisting of invasive monitoring prior to resection. We hypothesized that this might enhance the non-invasive localization of both ictal onset and function, and spare functional non-epileptogenic brain in patients being considered for hemispherectomy. Ten consecutive children with medically refractory (mean age 3 years, range 6 weeks-6 years; 5 boys) seizures were managed acccordingly over a one year period. Six patients had cortical dysplasia (CD), 2 porencephalic cyst, one Sturge-Weber syndrome, and one Rasmussen's encephalitis. 6/10 children underwent at least two surgical stages, consisting of placement of subdural electrodes followed by a tailored hemispherectomy (four: 2 stages, one: 3 stages, one: 4 stages), whereas four patients (1 Rasmussen's encephalitis, 1 Sturge-Weber syndrome, 2 CD patients younger than 3 months old) underwent hemispherectomy only. In three children, functional cortex (2 visual, 1 Rolandic) was spared in order to preserve preoperative function. At a mean follow-up of one year (range 3 months-2 years), 9/10 patients are seizure-free. There was no morbidity related to invasive monitoring. One patient experienced a contralateral SDH, due to overdrainage, requiring evacuation. Two patients have subsequently required placement of VP shunts. In children with hemispheric malformations of cortical development, in particular those without disabling preoperative neurologic deficit, the staged, tailored approach appears safe, providing precise seizure-onset and functional localization. We have found this approach useful in identifying CD patients who might otherwise have been excluded from hemispherectomy. However, this approach cannot be advocated in cases of syndromic hemispheric epilepsy, in which the progressive clinical course is known. Longer follow-up is necessary to determine whether the early seizure-free outcome is durable.

44. Epilepsy Surgery in Early Infancy,Experience with Four Infants and Outcomes*

Greg Olavarria , MD, Joseph A. Petronio, MD *(Atlanta, GA)*

Intractable seizures in infancy can present a diagnostic and management dilemma for the pediatric neurologist and neurosurgeon. It has been demonstrated that intractable seizures in children can be significantly reduced or altogether eliminated by early intervention in the appropriate candidate. Few, however, have reported their results with infants. The first year of life represents a critical period for brain growth and maturation. It also represents a window for optimal recovery and regeneration. We report here our experience in the evaluation and treatment of four selected infants with intractable seizures. They represent a unique sub-set of patients in their very young age at presentation and in the severity of their seizure disorder. Two infants underwent frontal and temporal resections of seizure foci, one for cortical dysplasia and the other for tumor. The others underwent subtotal hemispherectomies. Preoperative workup involved EEG monitoring and neuro-imaging (functional PET imaging was used). Intraoperative monitoring was used in all cases as well as image-guided frameless localizing techniques to guide surgical approach. Outcomes were qualified using the Engel classification system of seizure severity. All infants were followed for at least a year with post-operative imaging and serial examinations. Two infants remain seizure free on seizure medications, while the other two remain

* *Considered for Shulman Award*

seizure free off all medications. The evaluation and treatment of these four challenging cases is discussed.

45. Dysembryoplastic Neuroepithelial Tumours and Childhood Epilepsy: A Case Series

Jeffrey D. Atkinson, MD, J.P. Farmer, MD, J.L. Montes, MD, A.G. O'Gorman, MD, S. Albrect, MD, K. Meagher-Villemure, MD
(Montreal, QC)

Objectives: Dysembryoplastic neuroepithelial tumours have been well recognized as a cause of leisonal epilepsy since the first description of the tumour in 1988. Many retrospective series have demonstrated the clinical, radiologic, and pathologic characteristics of this tumour in patients of all ages. We review our experience with DNET in an exclusively pediatric population.

Methods: A retrospective review of hospital records was performed looking for all cases of DNET which occurred at our intitution since 1988. Clinical and intra-opertive findings, radiology, EEG, pathology, and outcome were examined in these patients and compared with the existing literature.

Results: Including our first case in 1992, we identified six children with this lesion. All presented with medically intractable epilepsy and underwent lesionectomy guided by surgical navigation tools, ultrasonography, and electro-corticography resulting in Engel class I or Ib seizure outcome at a mean follow-up of 34 months. Some unique characteristics were noted in our series including the young age range of our patients (21 months to 11 years), mesial cortical lesion location, and in some cases, a significant area of vascular proliferation noted on pathological examination.

Conclusions: Early published reports of DNET included many patients in the adult age range with a long history of epilepsy. With modern imaging methods, and a more aggressive approach towards treating lesional epilepsy in children, many of these lesions can be discovered and treated successfully at a young age, and these patients may be spared the potential developmental problems associated with intractable epilepsy.

46. The Role of the Hippocampus in Brain Tumor Associated Epilepsy: The Concept of "Dual Pathology"

Arno H. Fried, MD, FACS, Christos Lambrakis, MD, Marcelo Lancman, MD *(Hackensack, NJ)*

In patients with brain lesions (tumors, AVMs) that are outside the mesial temporal lobe (neocortical), mapping of an electrical seizure focus within the hippocampus has previously been demonstrated by this group and others. The concept of dual pathology, where the hippocampus becomes epileptogenic secondary to a tumor a distance from it was studied. This study looked at hippocampal volume loss calculated in patients harboring neocortical lesions, with and without epilepsy. We compared the hippocampal volumes in patients with extra-temporal lesions with epilepsy (n=8), and without epilepsy (n=9). Cases where the mass was compressing the hippocampus were excluded. The measurement was performed using stereotactic data acquired from thin section MRIs and computer assisted imaging. The volumes were performed twice by a blinded reader. An ipsilateral to contralateral hippocampal volume ratio was determined. The mean for the normalized, ipsilateral / contralateral ratio was 1.119 (+/- 0.16) for the group without epilepsy, and 0.831 (+/- 0.17) for those with epilepsy, ($F=11.75$; $p=0.004$). This statistical difference confirms the presence of hippocampal volume loss in the group with epilepsy who had lesions outside the mesial temporal lobe. The conclusions of this study are that the group with lesion associated neocortical epilepsy, had smaller hippocampal volumes, ipsilateral to the lesion, compared to the group without epilepsy. This points to the importance of thorough evaluation of the temporal lobe in the workup and surgical planning of lesion associated epilepsy surgery and the importance of dual pathology developing within the hippocampus.

47. Vagal Nerve Stimulation in Pediatric Patients Following Intracranial Epilepsy Surgery

Joseph R. Madsen, MD †, Dilek Yalnizoglu, MD, Sandra Helmers, MD
(Boston, MA)

Rationale: Vagal nerve stimulation (VNS) has shown efficacy in seizure reduction, but has not been specifically studied in refractory pediatric epilepsy following seizure surgery. With limited treatment options available, we have was performed VNS in this patient group.

Methods: Of 49 patients who underwent implantation of a vagal nerve stimulator, 16 had previous epilepsy surgery. 14/16 patients, aged between 5-

† *Disclosure Information*

18 years, with a minimum follow up of 3 months were studied. Five patients had resective surgery, 7 patients had a corpus callosotomy (CC), 2 had resective surgery with CC. Record of seizure type was obtained during a one month baseline period. At implantation, the stimulator was activated to deliver .25mA for 30s, to repeat every 10-15 min. Medications remained the same following implantation, seizure records continued. During follow up visits stimulation parameters were changed and medications were adjusted.

Results: 9/14 patients (64%) showed >50% reduction in seizure frequency, including 2 patients with >90% reduction. Two patients (14%) showed ~30% decrease, 2 remained stable, 1 had an initial increase in seizure frequency. 2/5 resective surgery cases showed >50% decrease, 2 remained stable, 1 had an increase in seizure frequency. 6/7 patients with CC had >60% (60-99%) decrease in seizure frequency, 1 patient had a 30% reduction. Two patients with both resective surgery and CC showed decreased seizure frequency by 27% and 81%.

Conclusions: VNS is efficacious and tolerated in pediatric patients with refractory epilepsy following epilepsy surgery. Postcallosotomy patients showed the greatest improvement.

48. Surgical Outcome in Children with Epilepsy Evaluated by Magnetoencephalography

Mark R. Lee, MD, Joseph R. Smith, MD, Yong D. Park, MD, Don W. King, MD *(Augusta, GA)*

Introduction: There is little data concerning magnetoencephalograpic (MEG) abnormalities in children with intractable epilepsy. The ability of MEG to predict outcome following cortical resection has not been determined.

Methods: We reviewed interictal MEG data on 21 chldren with intractable seizures who were being evaluated for epilepsy surgery. MEG's were classified by pattern and location of primary spike focus. MEG parameters were analyzed to determine their ability to predict seizure outcome following cortical resection.

Results: Fifteen children were male and 6 were female. Age ranged from 3-16 years (mean 11 years). Location of the primary MEG spike focus was anterior temporal - 3, extra-temporal - 13, unlocalized - 5. Fourteen children underwent 15 resective operations. Twelve have had at least 1 year follow-up. Outcomes at 1 year were Class I (seizure-free)- 67%, Class II (rare seizure) - 17%, Class III (greater than 90% reduction) - 8%, Class IV (less than 90% reduction) - 8%. Of the 10 children that had complete resection of the primary MEG spike focus, 8 were Class I (seizure-free) and 2 were Class II (rare seizure). None were Class III or IV. Of the 2 children who had an incomplete resection of the primary spike focus, 1 was Class III and 1 was Class IV. None were Class I or II.

Conclusions: Children with intractable epilepsy demonstrate a variety of interictal MEG patterns similar to those of EEG. Our data suggest that resection of the primary MEG spike focus correlates strongly with excellent outcome.

49. Seizure Surgery for Children with Tuberous Sclerosis Complex

Glenn Morrison, MD †, Prasanna Jayakar, MD *(Miami,FL),* **Susan Koh, MD** *(Los Angeles,CA),* **John Ragheb, MD, Michael Duchowny, MD, Trevor Resnick, MD, Maria Penate, RN, Patricia Dean, RN** *(Miami, FL)*

Tuberous Sclerosis Complex (TSC) is a disorder wherein intractable epilepsy may represent a significant clinical problem. Although most children have multiple tubers, the seizures commonly emanate solely or primarily from one tuber. If this can be identified and removed, the intractable seizures should be eliminated.

We report on a series of 21 children with TSC who were evaluated for their intractable epilepsy. An epileptogenic focus (EF) was identified in 17 children. Interictal EEG revealed a principle spike focus corresponding to the EF in 14 patients. Ictal SPECT scans were obtained in 18 patients and in 10 there was an area of hyperperfusion that corresponded to the EF. Although all the children had multiple tubers, the EF corresponded to the largest tuber in 8 patients and in all 13 children with calcified tubers.

Thirteen children had surgery. The tuber resection was guided by electrocorticography in 7 and 6 patients had a 2 stage resection with subdural electrodes and extraoperative monitoring. At a mean follow up of 29 months, 9 of the operated children are seizure free (69%), 1 has a >75% reduction in seizures, 2 are unchanged, and 1 child has new seizures from the contralateral hemisphere.

In conclusion, children with TSC who have intractable epilepsy should be evaluated for surgery. Surface EEG and ictal SPECT scans are helpful but extraoperative monitoring may be necessary. Utilizing all data (clinical semiology, MRI, CT, SPECT, scalp/subdural electrical recordings), a relief from seizures can reasonably be expected in 69% of these children.

† Disclosure Information

50. Surgical Management of Gelastic Epilepsy Related To Hypothalamic Hamartoma

J.P. Farmer, MD, S. Mittal, MD, J.L. Montes, MD, B. Rosenblatt, MD, F. Andermann, MD *(Montreal, PQ)*

Introduction: Hypothalamic hamartomas manifest by either causing precocious puberty or gelastic epilepsy and aggressive behavior. While surgery has been shown effective for precocious puberty, hormone therapy now appears, if tolerated, to be the treatment of choice. Gelastic epilepsy and morbid behaviour difficulties remain a very difficult problem to treat. Temporal resections for what appear to be well-localized surface activity have uniformly met with failure. Despite navigational and endoscopic technical advances, direct surgical approaches to these usually sessile lesions have remained a challenge.

Method: We report the surgical management and outcome of 4 boys ranging from ages 2 to 11 years with gelastic epilepsy, hypothalamic hamartoma, and morbid behavior difficulties. One patient was studied with a depth electrode revealing that the hamartoma was pacing surface temporal activity. Three were approached via the suprasellar cistern, one via the foramen of Monroe using endoscopy.

Results: Despite incomplete, although radical resections, 3 patients have shown a dramatic improvement in behavior and seizure activity, whereas the fourth one failed but responded partly to a subsequent stereotactic lesion made above the hamartoma implantation, in the mammillothalamic tract. All 4 now live at home. None are seizure-free or off medication. None have required new endocrine medication, and psychiatric medications have been tapered.

Conclusion: Surgical resection of hypothalamic hamartomas can lead to improved psychosocial integration and seizure status in patients with gelastic epilepsy and challenging morbid aggressivity. The morbidity associated with this procedure needs to be compared with the long-term morbidity of alternative treatment methods.

51. The Contemporary Management of the Chiari I Malformation in Children*

Mark D. Krieger, MD, Michael L. Levy, MD, J. Gordon McComb, MD *(Los Angeles, CA)*

A wide variety of surgical adjuvants to the standard bony decompression have been advocated in the treatment of the Chiari I malformation, especially when the tonsillar herniation is associated with hydrosyringomyelia. Our practice has been to avoid such adjuvants as duroplasty, obex plugging, cerebellar tonsil resection, and various shunting procedures, and to perform a simple limited occipital craniectomy (less than 2.5cm in diameter), C1 laminectomy, and dural opening. To evaluate the efficacy of this more limited procedure, a retrospective review was performed of the medical records of 52 consecutive patients treated surgically over a 6-year period. This series includes long-term follow-up of a prior series (4-8 years), as well as 21 additional patients treated in a standard fashion. Included are 27 females and 25 males, ranging in age from 3 months to 18 years (median 11.5 years). Of particular interest is the large number of patients discovered during evaluation for scoliosis (38 patients-73% of the total series), which partially accounts for the large number of patients who harbored a syrinx (44-85%) in this series. All patients had at least one postoperative MRI at 6 months. Syrinx resolution or greater than 50% diminution was seen in 89%. 48 of the patients responded well; 4 patients required subsequent additional operative procedures: 2 developed progressive hydrocephalus and required ventriculoperitoneal shunting, with symptom resolution. In the other 2 patients the syrinx did not diminish; both received syringopleural shunts. Importantly, no patient who responded satisfactorily at 6 month follow-up subsequently failed either radiographically or clinically. Postoperative morbidity consisted of a 21% incidence of headaches, all of which except 2 resolved within 7 days. Nausea and vomiting occurred in 13%. Four patients had a postoperative CSF leak; all responded to bedside suturing without further sequelae. This study demonstrates the importance of addressing the Chiari I malformation, especially in the presence of scoliosis with a syrinx, and the effectiveness of a limited surgical approach.

* *Considered for Shulman Award*

52. Paroxysmal Rage as a Presenting Symptom of the Chiari 1 Malformation

Roger J. Hudgins, MD

The Chiari I malformation is characterized by herniation of the cerebellar tonsils below the foramen magnum. This malformation has been called the "adult" Chiari malformation, but with the advent of MRI as well as a better understanding of the clinical presentation, most Chiari I malformations are now being diagnosed and treated during childhood. Children with a Chiari I malformation present with a variety of symptoms including headaches, ataxia, dysphagia, crying, nonradicular shoulder or extremity pain, or loss of strength or sensation in the extremities. Not reported in the literature is a presentation with paroxysmal rage. Recently, two children who presented with intermittent violent rages were found to have Chiari I malformations. In both cases the rages ceased after Chiari decompressions. This paper discusses these cases and makes a case for MRI evaluation in children with new onset of paroxysmal behavioral disorders.

53. Characterization of Third Ventricular Floor Anatomy in the Hydrocephalic State Relative to 3rd Ventriculocisternotomy

Stephanie L. Einhaus, MD, Erica Nevells, MD, Anthony Watkins, MD *(Memphis, TN)*

The anatony of the floor of the 3rd ventricle can be variable especially in the hydrocephalic state. Particular anatomic features that make a patient a good or poor anatomic candidate for 3rd ventriculocisternotomy (TB) have not been well described. Preoperative MRI is essential in predicting anatomic candidacy.

Twenty MRIs of patients for TV were reviewed. 17 were considered acceptable anatomic candidates, and 3 unacceptable. Sixteen had the procedure successfully completed, and one patient had the procedure aborted because of a very thick floor visible on the preoperative MRI and verified endoscopically. Video tapes and operative reports were used to evaluate intraoperative anatomy and technical ease of performing the procedure.

Ideal candidates have 3 mm (median = 4 mm) of space between the mammillary bodies (MB) and the infundibulum or the dorsum sellae. The MB are located closely linear to the belly of the pons rather than pushed posteriorly. The basilar artery caput is located immediately below the MB and there is sufficient space between it and the infundibulum or dorsum sellae (3 mm). The floor should be significantly thinned. Patients with floors that are redundant and MB that are pushed downward can be technically more challenging because the prepontine cistern is smaller. If the floor is particularly redundant, it may be necessary to use a valsalva maneuver to bring the floor away from the basilar artery.

Poor candidates have a thick floor or a basilar artery that is too anteriorly located, or there is inadequate space to safely make a fenestration.

54. Cine Phase-contrast CSF Flow MRI after Third Ventriculostomy: Correlation with Endoscopic Exploration

Toru Fukuhara, MD, PhD, Mark G. Luciano, MD, PhD, Christine L. Brant, MS *(Cleveland, OH)*

Introduction: After endoscopic third ventriculostomy, cine phase-contrast CSF flow MRI is widely used as a reliable method in the detection of patency.

Methods: Records of 89 patients who had endoscopic third ventriculostomy were reviewed. In order to confirm the reliability of CSF flow studies, findings were correlated with postoperative symptoms, MRI and, in the 13 redo third ventriculostomy cases, endoscopic exploration.

Results: Ventricular size was unchanged in 75% the next day, 57.4% in 3months, 48.2% in 6 months and 41.8% in 1 year. CSF flow study revealed patent findings at ventriculostomy in all the patients with symptomatic resolutions (60 patients) except 2. In the 13 redo third ventriculostomies, 10 patients had an obstructed pattern and 3 open on CSF flow studies taken prior to re-operations. All 10 patients with obstructed pattern had significant membrane formation (8 with complete closure) but 3 with open pattern had no membrane formation at the site of ventriculostomy. In preoperative CSF flow studies taken in 51 patients, 2 patients had false-positive CSF flow at the third ventricular floor. Both cases had a vigorously pulsating membrane confirmed during third ventriculostomy.

Conclusions: Compared with ventricular size decrease, CSF flow study findings are more consistent with symptom resolution and with the result of direct exploration. False-positive preoperative CSF flow is a pitfall in postoperative CSF flow study interpretation, which can lead to false postoperative positives.

55. Endoscopic Fenestration of Third Ventricular Arachnoid Cysts

Michael L. Levy, MD, Mark Liker, MD, Michael Y. Wang, MD, Hulda Magnadottir, MD, J. Gordon McComb, MD

We retrospectively reviewed our series of 6 patients undergoing endoscopic fenestration of third ventricular arachnoid cysts. Our series included 4 females and 2 males (age = 52 + 51 months). Follow-up was a mean of 18 months. All 6 patients presented with evidence of ventriculomegaly and marked third ventricular dilatation. Pre-operative VP shunts were placed in two patients prior to presentation at our facility who were noted to have a progressive increase in ventricular size. Two patients required shunts post-operatively given a continued increase in ventricular size. A total of 3 out of 6 patients required CSF diversion in addition to endoscopic fenestration. Five patients undergoing endoscopic fenestration and were noted intra-operatively to have a slit valve mechanism at the level of the mid basilar artery in the prepontine cistern. All cysts were endoscopically fenestrated, both at the outer cystic portion and the more inferior cystic portion.

There were no complications following endoscopic fenestration. We conclude that endoscopic fenestration alone is an appropriate means of treatment for these abnormalities if both the outer and inner components of the cyst are fenestrated and may allow for the avoidance of CSF diversion in some children.

56. A Prospective Population-based Study of Pediatric Trauma Patients with Mild Alterations in Consciousness (field Glasgow Coma Score of 13-14)*

Michael Y. Wang, MD, Pamela Griffith, RNC, BSN, Judy Sterling, RN, J. Gordon McComb, MD, Michael L. Levy, MD

Context: Considerable controversy surrounds the appropriate evaluation of children with mild alterations in consciousness after trauma (Glasgow Coma Scale score of 13-14).

Objective: To determine the incidence of intracranial lesions in pediatric patients with a field Glasgow Coma Score (GCS) of 13 or 14.

Design: Population-based, multi-center prospective study of all patients transported by Emergency Medical Services over a twelve-month period.

* *Considered for Shulman Award*

Setting: Urban Los Angeles County encompassing a population of 2.4 million children, 13 designated Trauma Centers, and 94 receiving hospitals.

Results: 8,488 patients in the pediatric age group (less than 15 years old) were transported by EMS for injuries. Of these, 209 had a documented field GCS of 13 or 14. 157 were taken to Trauma Centers and 135 (86%) received head computed tomography (CT) scans. 43 (27.4%) had an abnormal CT scan, 30 (19.1%) had an intracranial hemorrhage, and five required a neurosurgical operative procedure for hematoma evacuation. The variables deteriorating mental status (0.500/0.844), loss of consciousness (0.173/0.809), skull fracture (0.483/0.875), and extracranial injury (0.205/0.814), were not found to be significantly significant as predictors of intracranial hemorrhage.

Conclusions: The incidence of intracranial injury in pediatric patients with mild alterations in consciousness in the field is significant. The great majority of these patients will not require operative intervention, but the implications of missing these hemorrhages can be severe for a subgroup of head injured patients. It is recommended that, since clinical criteria and skull x-rays are poor predictors of intracranial hemorrhage, all children with a field GCS of 13 or 14 routinely have a screening non-contrast enhanced CT scan.

57. Leukocyte Dependent Blood-Brain Barrier Breakdown is Attenuated by a Novel Carboxyfullerene Derivative in a Model of Newborn Hypoxic—Ischemic Encephalopathy in Piglets

Stuart S. Kaplan, MD, Jeffrey M. Gidday, PhD, Laura L. Dugan, MD, Ernesto R. Gonzales, Ronaldo S. Perez, TS Park, MD

Superoxide radical and its free radical products are implicated in the pathogenesis of ischemic brain injury. In peripheral vascular beds, superoxide radical mediates leukocyte-endothelial adherence and loss of vascular integrity in response to ischemia. The present study was undertaken to test the hypothesis that oxygen free radicals promote the acute inflammatory response following global cerebral ischemia in newborn piglets and that this response may be attenuated by a novel superoxide anion scavenger, C3 malonic acid carboxyfullerene. A progressive and significant ($p<0.05$) increase in adherent leukocytes was observed during the initial 2 hr of reperfusion following 9 min asphyxia (n=15) compared to nonasphyxic controls (n=14). In this model, vascular injury as assessed by significant ($p<0.05$) increases in fluorescein permeability at 2 hr reperfusion is dependent on adherent leukocytes. However, when animals (n=5) were

locally treated with the carboxyfullerene isomer (300 μM) beginning 30 min before asphyxia and continuing to 2 hr of recovery, leukocyte adherence was attenuated by 51% and 79% at 1 and 2 h of recovery, respectively, compared to untreated ischemic controls ($p<0.05$) with a reduction of blood-brain barrier disruption to nonasphyxic control levels ($p<0.05$). Local administration of SOD (60 U/ml;n=8) led to comparable reductions ($p<0.05$). These findings implicate superoxide radical as an important mediator of the postischemic acute inflammatory response in the cerebral microcirculation and demonstrate that leukocyte-mediated vascular injury can be prevented with carboxyfullerenes. These compounds may show promise in the treatment of newborn hypoxic-ischemic encephalopathy.

58. The Role of Fast Axonal Transport Proteins in Spinal Cord Regeneration*

Brian Witwer, MD, Bermans J. Iskandar, MD, Agahan Unlu, MD, Nythia Hariharan, MD *(Madison, WI),* **J.H. Pate Skene, PhD** *(Durham, NC)*

Introduction: A minority of Central Nervous System neurons can regenerate after injury, if afforded the right environment (eg, a peripheral nerve graft). For regeneration to occur, signals need to be transmitted between the cell body and the regenerating axonal tip. Such signals occur through axonal transport proteins, which are generally divided into fast and slow proteins. In this paper, we have studied the role of fast proteins in transmitting the signals that are essential in mediating axonal growth.

Methods: Sciatic nerve segments were grafted into the injured cervical spinal cord of adult male Sprague-Dawley rats. Axons are encouraged to grow into the graft because of a concomitant peripheral (sciatic nerve) injury. After a defined time period ranging from 1 to 8 weeks, the graft was backfilled with a fluorescent tracer at 1 cm from the graft site. Twenty-four hours later, the animals were perfused, the L5 dorsal root ganglia (DRG) were removed and sectioned, and the percentage of fluorescent DRG cell bodies was counted using an assumption-based method.

Results: The first evidence of regeneration was seen at 14 days after injury. No regeneration occurred at 8, 10, and 12 days, or in controls. No additional regeneration occurred at 21 days. The speed of signal transmission between the cell body and the axon tip was determined by dividing the DRG-backfill site distance (ave 8cm) by the time required for regeneration to occur. The absolute minimum rate of signal delivery between the cell body and injury site is 5.7 mm/day. The velocity of

signal transmission is even greater when one also takes into consideration the time required for initiating growth as well as the time it takes for 1 cm of regeneration to occur into the graft.

Conclusions: Fast axonal proteins play an important role in the transmission of regenerative signals in the injured rat spinal cord.

59. The Surprisingly Sturdy Infant Brain: Why is it More Resistant to Focal Injury?*

Susan R. Durham, MD †, Susan Margulies, PhD, Ramesh Raghupathi, PhD, Jeffrey Golden, MD, Mark Helfaer, MD, Amy Brooks-Kayal, MD, Sunil Marwaha, BS, Ann-Christine Duhaime, MD *(Philadelphia, PA)*

The scaled cortical contusion model was developed to deliver a proportionally identical injury to piglets at ages corresponding to human infants, toddlers and adolescents. Contrary to expectations, the percentage of injured hemisphere increases dramatically as the piglet ages, demonstrating relative resistance to focal traumatic brain injury at younger ages. Possible explanations for this age-dependent difference in vulnerability, including physiologic and cellular responses to injury, have been explored in the current work.

Regional cerebral blood flow (CBF), cortical excitability, brain/body temperature, heart rate, and mean arterial pressure (MAP) were measured before and for 3 hours following injury in 12 piglets at 3 ages. While baseline heart rate, MAP and temperatures varied with age, there were no age-specific deviations from baseline values following injury. CBF decreased following injury in the youngest animals, while the intermediate and oldest animals showed an elevated CBF following injury. EEG analysis showed increased cortical excitablity after injury in the youngest animals.

Immunohistochemical/TUNEL staining for apoptosis suggested an increase in apoptotic activity in the older animals likely related to greater expression of anti-apoptotic genes earlier in life. GFAP expression remained proportional to lesion size; there was no evidence of a more extensive area of subthreshold injury in the younger ages.

Because the physiologic differences observed between ages appear unlikely to explain relative neuroprotection, age-dependent resistance to damage from mechanical trauma appears most likely related to differences in response at the cellular level, and/or to changes in tissue mechanical properties with maturation.

60. Stimulation of Extensive Axon Growth in the Injured Rat Corticospinal Tract with Inosine

Joseph R. Madsen, MD†, David Goldberg, Nina Irwin, PhD and Larry I. Benowitz, PhD *(Boston, MA)*

The rat corticospinal tract, like that of humans, arises in the motor cortex and projects to spinal interneurons and motoneurons; the failure of this pathway to regenerate after injury results in a permanent loss of skilled movements of the distal musculature. Recovery may be enhanced in adult animals if mechanisms present in juveniles can be appropriatiely triggered. We have recently found that the purine nucleoside inosine stimulates axon outgrowth and other aspects of differentiation in some types of neurons, and enhances the effects of growth factors in others. Following a unilateral transection of the rat corticospinal tract in the ventral medulla, we applied inosine with a minipump for two weeks to stimulate axon sprouting in uninjured pyramidal cells of the contralateral motor cortex. Using biotinylated dextran amine to trace axon trajectories, we found that this treatment stimulated the growth of hundreds of axon collaterals that descended for at least 1 cm into the denervated corticospinal tract; some newly grown fibers were also traced laterally into the denervated spinal gray matter. The growth of new axon collaterals was confirmed with the use of antibodies to the growth associated protein, GAP-43. Thus, inosine, a naturally occurring metabolite without known adverse side effects, might be useful in promoting functional recovery after CNS injury. Support: National Institutes of Health (NIH EY05690), and Boston Life Sciences, Inc

61. Deep Venous Thrombosis in a Pediatric Neurosurgical Population*

David Hart, MD, Mark Krieger, Michael Levy, J. Gordon McComb *(Los Angeles, CA)*

We reviewed 32 cases of deep venous thrombosis (DVT) in pediatric patients presenting over 10 years. Ages ranged 1.5 to 20 years (mean=13.9). There were 17 females and 15 males. There were 29 DVTs in the lower extremity (91%), two in the inferior vena cava (6%), and one in the upper extremity (3%). Presenting symptoms included swelling of the affected extremity (59%), pain (56%), respiratory arrest (6%), and one patient each with erythema, chest pain, hickman catheter failure, cardiac arrest, or no symptoms. Fourteen of the 32 patients had active neurosurgical problems including hydrocephalus, brain tumor, cerebral infarction, head trauma,

brain abscess, and sciatic nerve palsy. There was no difference in age, presenting symptom of DVT, modality of diagnosis, location of DVT, or treatment in this subset of patients. All patients had comorbidities including sepsis, multiple fractures, orthopedic procedures, dehydration, renal disease, pneumonia, pre-existing coagulation disorders (n=5), rhematologic disease (n=4) and tumors.

Diagnostic studies included ultrasonography in 56%, venogram in 44%, chest CT in 13%, V/Q scan or tagged RBC scan in 3% each. Twenty-four patients were treated with IV heparin followed by PO warfarin. One had an IVC filter placed. Four were treated with warfarin alone. One was treated with urokinase, IV heparin, then SC heparin. Two died while hospitalized.

Five suffered recurrences during followup (mean 20 months). Of these, two had 6 recurrences each, and one other patient had 4 recurrences. None of the recurrences occurred in patients with underlying coagulation disorders.

Further research into the risks, diagnosis, and treatment of DVT in children is required to develop a better understanding and treatment of this potentially fatal disease process.

62. Cervical Spine Clearance in Major Trauma in a Pediatric Population

Leon E. Moores, MD, Jean Reardon, RN, Philip H. Cogen, MD, PhD, Martin Eichelberger, MD, Robert F. Keating, MD, David W. Pincus, MD, PhD *(Washington, DC)*

Objectives: Cervical spine injuries are rare in pediatric trauma. We review an algorithm for clearance of the cervical spine in a pediatric population.

Methods: The Children's National Medical Center trauma database from 1JAN96 to 31DEC98 was screened. 970 patients evaluated as major trauma were included in the study. Patients were followed through hospitalization and clinic follow up.

Results: 165 (17%) awake, cooperative patients without neurologic deficit, neck pain, or tenderness on passive range of motion were cleared without radiographs. 593 (61%) patients were cleared with clinical examination in combination with lateral radiographs, and/or CT scans, and/or flexion/extension radiographs. 11 (1%) patients expired with associated cervical spine injuries. An additional 12 (1%) patients had cervical spine injuries diagnosed with lateral radiographs. Passive flexion/extension radiographs

were obtained on 84 patients who had no injuries on static imaging and could not cooperate with examination 24 hours after injury. No additional injuries were noted on the dynamic films.

Conclusion: In a pediatric population, cervical spine injuries in major trauma were associated with 48% mortality. Effective clearance of cervical spine injuries was accomplished with lateral radiographs or CT scan. No additional injuries were seen on passive flexion/extension films.

63. Abdominal "Stealth" Incisions for VP Shunts

Michael H. Handler, MD, FACS, FAAP (Denver, CO)

Historically, neurosurgeons have not paid much attention to the cosmetic impact of the abdominal incisions, which record the placement of VP shunts; it is not uncommon to see a belly criss-crossed with multiple old scars. Our general surgical brethren now routinely do major abdominal procedures through two or three stab incisions of 5 mm each. 14 patients had attempted placement of distal tubing using a small umbilical incision, abdominal insufflation with a Varess needle, and laparoscopy. One patient unexpectedly had unacceptable adhesions and had placement of a pleural shunt. The rest had perforation of the abdominal wall through a 3-mm skin nick, using a disposable trocar to pass the catheter under direct visualization to a point away from any adhesions. Usually, an old scar could be re-used. The cosmetic advantage is obvious, and the technique prevents many potential complications of the abdominal trocar, especially in larger patients. The technique is easily learned, and the technology widely available in any hospital where pediatric neurosurgery is performed.

Scientific Posters

1. Cranial Bone Fixation After Craniotomy: A New Method

Marjorie C. Wang, MD, Ken R. Winston, MD

Methods for cranial bone fixation have been of great concern to surgeons since the beginning of modern neurosurgery. Techniques for fixing the bone flap at the completion of an intracranial operation vary widely in clinical practice but are necessarily influenced by the integrity of the removed bone. We describe a new method for fixating cranial bone flaps with absorbable suture and bone chips. The results of use of this technique in 121 pediatric patients is reviewed. This technique is simple and cost efficient, and reliably achieves stability and excellent cosmesis in closing craniotomies. It can be used for both pediatric and adult populations. The kerf created by the craniotome is bridged by bone chips taken from the inner edge of the bone flap. Good bony fusion is evident after several weeks by histopathological analysis. The contour of the bone flap and skull with this technique is cosmetically preferable in areas such as the forehead where wires or titanium plates would be visible or palpable, or where postoperative depression of the bone flap would be obvious. While miniplates and screws provide an alternate technique, the use of readily available absorbable suture and the preexisting bone flap for bone chips avoids the use of permanent foreign bodies and provides a simpler method with excellent results in this series.

2. An Alternate Technique to Close Neurosurgical Incisions Using Octylcyanoacrylate Adhesive

Daniel J. Won, MD, FACS, Kuk-Wha Lee, MD, PhD, Thomas Sherwin, MD *(Loma Linda, CA)*

We report a series of 125 cases using the recently FDA approved tissue adhesive octylcyanoacrylate in the final layer closure of routine pediatric neurosurgical cases. The deep layers are closed in standard manner and adhesive is painted on the skin. Wound dressings and suture removal are eliminated.

Follow-up of six to twelve month revealed: (1) Two cases of wide (>5mm) scar (2) Three cases of slightly wide (3-5mm) scar (3) Two cases of wound infection (4) One case of deep suture extruding through the incision.

We conclude that octylcyanoacrylate is an acceptable alternative to nylon, staple, vicryl, or steristrip closure of the neurosurgical incision in children.

3. Chronic Group B Streptococcal Ventriculitis—A Case Report and Review of the Literature

David S. Jones, MD, John D. Loyd, MD, Steven S. Glazier, MD, S. Taylor Jarrell, Michael J. Opatowsky, MD, Laurence B. Givner, MD *(Winston-Salem, NC)*

In the early 1970's, Group B Streptococcus (GBS) emerged as the leading cause of meningitis and sepsis in newborns. Approximately 25-30% of infants with GBS infection have meningitis. Though the mortality rate has declined with improvements in early recognition and aggressive treatment, the infection results in permanent neurological sequelae in up to 50% of children. Two distinct clinical syndromes related to age at diagnosis have been described. Early onset disease, occurring in neonates less than 7 days old, is a more severe illness with nonspecific signs and symptoms. Late onset disease, presenting beyond the first week of life, is characterized by a less severe illness with typical signs and symptoms of meningitis. While the onset of symptoms and the severity of illness are clearly variable, the presenting symptoms typically occur within hours prior to diagnosis. We describe a 26-day-old infant with insidious onset of disease. The child presented with a two-week history of vomiting and failure to thrive. Subsequently, she was found to have obstructive hydrocephalus secondary to chronic GBS meningitis/ventriculitis. She was treated with intravenous antibiotics and continuous external ventricular drainage for 4 weeks, followed by ventriculoperitoneal shunting. To our knowledge, there is only one other case of chronic GBS meningitis in the literature. Because of the highly varied and often nonspecific signs of GBS meningitis, one must maintain a high index of suspicion and promptly institute appropriate management to limit the serious consequences of the disease.

4. Amoebic Meningoencephalitis Caused by Balamuthia Mandrillaris Presenting with Acute Anterograde Amnesia in a 16 Old Boy

Jeffrey P. Blount, MD, Jared Antevil, MD, Christopher S. Kent, MD, Michael Quigley, MD *(San Diego, CA)*

Objective and Importance: Meningoencephalitis caused by free living amoeba is rare. Balamuthia mandrillaris is one of four species of the genus

Acanthomeba and accounts for 12% of cases of human CNS amoebic infection. Fewer than 70 cases have ever been reported in the world literature and there has never been a case recorded that presented with acute anterograde amnesia.

Clinical Presentation: A previously healthy 16 year old boy presented with mild bifrontal headache, intermittent blurring of vision and pronounced acute anterograde amnesia. Mild fever (101 degrees) was noted. General physical exam revealed only a single oral aphthous ulcer. Neurologic exam revealed only a dramatic deficit in short term memory (recall < one minute). Comprehensive serum lab values were normal. LP revealed normal opening pressure and a lymphocytic pleocytosis (343 WBC - 57% lymphocytes). UA and CXR were normal. CT revealed right frontal and left parietal areas of low attenuation.

Intervention: Broad spectrum antibiotics and acyclovir was administered. MRI revealed multiple additional areas of abnormal intensity throughout the cerebral cortex (primarily gray-white junction). Negative titers for Lyme, Toxplasma, Coccidiomyces, Cryptococcus, and RPR were obtained from serum and CSF specimens. Four vessel cerebral angiography was negative for vasculitis. PCR for HSV, CMV and enteroviruses were negative from CSF samples.

Significant neurologic decline was noted with progressive somnolence, fever to 102 degrees and complete loss of orientation. High dose steroids were administered without change in course. Repeat PCR for HSV, TB, arbovirus(Western/Eastern Equine Encephalitis, California Encephalitis, St. Louis Encephalitis) were negative. A low opening pressure was encountered when a right frontal ventriculostomy was placed. Open right frontal brain biopsy was performed. The patient progressed to deep coma with elevated ICP refractile to all means of control. Brain stem reflexes were lost and he was declared brain dead. Post mortem examination of the brain revealed cyst and trophozoite forms of an amoebic parasitic organism. Immunoflourescence studies performed at the Center for Disease Control confirmed the diagnosis of Balmuthia mandrillaris.

Conclusion: 1.) Amoebic meningoencephalitis must be considered in the differential diagnosis of patients presenting with clinical and radiographic evidence of meningoencephalitis. 2.) Despite an aggressive course including open brain biopsy the course of amoebic encephalitis was relentless and resulted in patient mortality. All cases thus reported of Balamuthia encephalitis have proven universally fatal.

5. Trampoline Injuries of the Cervical Spine

Peter G. Brown, MD, Mark Lee, MD *(Augusta, GA)*

Trampolines were responsible for over 6,500 pediatric cervical spine injuries in 1998, a five-fold increase in just ten years. Paraplegia, quadriplegia, and death have all been reported. We present a trampoline-related cervical spine fracture, and review the relevant literature. Additionally, we examine the efforts made to reduce the incidence of trampoline injures, and discuss why these have failed. We conclude that safety guidelines and warnings are inadequate, and support recommendations for a ban on pediatric use of trampolines.

6. Early Management of Post-Traumatic Leptomeningeal Cysts

Miltos K. Sugiultzoglu, MD, Mark M. Souweidane, MD *(New York, NY)*

Introduction: Leptomeningeal cysts, or growing skull fractures, are rare complications of pediatric head trauma. They are typically diagnosed several weeks to months following the injury when an enlarging scalp mass is recognized. Seizures or motor weakness can accompany this process and imaging studies at diagnosis are notable for an underlying porencephalic cyst and a large calvarial defect. There is currently no evidence to suggest a spontaneous resolution of this lesion.

Methods: Two infants with parietal skull fractures are used to illustrate our approach toward early management of post-traumatic leptomeningeal cysts. Both were diagnosed within a period of seven days, initially by physical examination, and definitively by MRI. The exposed area was debrieded, the dura repaired using autologus (pericranial) graft, and an autologus cranioplasty was performed in both cases. Results: An excellent cosmetic result was achieved in both children, which had an uneventful postoperative course and remain free of seizures, neurologic deficits, development of porencephalic cysts, or other complications after two and sixteen months respectively.

Discussion/Conclusion: With the recent recognition that MRI is highly predictive of which children are likely to develop a leptomeningeal cyst, we have adopted a policy of early management in an effort to avoid the aforementioned problems with delayed intervention. Our approach illustrates the point that the complication of a leptomeningeal cyst represents an entity in which a high index of suspicion can lead to the early and definitive management of this rare but potentially serious sequale of infantile skull fractures.

7. FORE: Golf-Related Head Injuries at the Arnold Palmer Hospital for Children

Eric R. Trumble, MD, Jogi Pattisapu, MD, Kay Taylor, RN, Annette Arbogast

Introduction: Sports-related injuries are a common cause of morbidity in children. Head Injuries are the most common cause of death by trauma. Previous studies of sports-related pediatric head injuries have found football to be a common cause of pediatric head injuries.

Results: In Orlando, at the Arnold Palmer Hospital for Children, we have found golf to be the most common sports-related cause of head injuries requiring neurosurgical evaluation. In the past 2 years, we have treated 16 sports-related head injuries, 7 of which were golf-related. The mean age of the patients injured was 11.1 years. Of these 7 children, 3 required surgery, all for repair of a depressed skull fracture with dural injury. Admission Glasgow Coma Scale was 14-15 in all patients and outcome was uniformily excellent. The other sports-related injuries were 2 jet skiing, 2 softball, 2 football, and 3 baseball.

Conclusion: Children must be closely monitored while engaging in golf-related activities in order to prevent injury. Should an injury occur, prompt neurosurgical treatment is associated with an excellent outcome.

8. Cerebral Perfusion and Pial Capillary Diameter Responses to Fluid Percussion Injury

Paul C. Framcel, MD, PhD, F. Alan Stevens, BS, Soon C. Kim, MD, Paul Tompkins, MS *(Oklahoma City, OK)*

Traumatic brain injury results in a variety of brain abnormalities, both physiological and biochemical. There is evidence that the outcome of closed head injury may be dependent on early events such as reduced brain blood flow and vascular injury. Due to the time required to transport patients to the emergency room, little information is currently available from the clinical setting on these early vascular events. Current treatment centers on returning brain blood flow to normal levels once it has been altered. While these attempts are sometimes successful, a lack of information on early chemical/vascular events following head injury impedes successful treatment of many patients in this category. Using a closed cranial window model in the rat, we measured pial artery vessel diameter and laser Doppler cerebral perfusion (CP) in response to a moderate fluid-percussion (FP) cerebral

injury (1.5 atm). The control broup showed no change in either vessel diameter orlaser Doppler perfusion over a two hour period. FP results in a 50% decrease in CP within 5 minutes of injury, and at two hours post injury SP is still reduced by 30% from pre-injury levels. No significant changes in pial artery diameter or mean arterial blood pressure were detected over this time frame. Moderate cerebral trauma gives rise to prolonged alterations in CP that may contribute to long term vascular pathology. This decrease in CP does not appear to be the consequence of arterial vasoconstriction (20-100um diameter).

9. Shaking Impact Syndrome—A Retrospective Analysis of 35 Cases

Ming-Ying Liu

From July, 1997, to June,1999, the author has treated 35 patients with shaking impact syndrome. There were 19 male and 16 female. The age was from 1 month to 2 years with median 6 months. The clinical manifestations were: poor feeding in 25 patients, lethergy in 23, unresponsiveness in 30, and seizure in 18. Retinal hemorrhage was present in 31 patients, and and bruise at parieto-occipital regions in 23. Special interest was noted that there were 17 patients showing thumb pressing marks at bilateral anterior chest and fingers pressing marks at bilateral upper back due to firm holding of the babies while doing the shaking. CT scan revealed subdural hematoma (19), subarachnoid hemorrhage (13), and intracerebral hematoma (3). Twenty-one caretakers were not the baby's parents. Only 18 caretakers admitted violent shaking to the babies. The patients were reported to the Children Protection Bureau for continuous follow up.

10. Treatment of Pediatric Tornadic Traumatic Brain Injury

John H. Honeycutt, MD, Sumon Bhattacharje, MD, Dave Min, James Brennan, Mary K. Gumerlock *(Oklahoma City, OK)*

On May 3rd, 1999, an F5 tornado struck Oklahoma City and the surrounding area with resulting mass destruction and numerous casualties and injuries. At Oklahoma Children's Hospital, we treated 6 children (age 19 months to 10 years/old) with traumatic brain injury, with 4 of them being categorized as severe. Two patients were considered to have mild traumatic brain injury and were observed overnight. However, three of the patients had severe traumatic brain injury that required intubation and intracranial pressure monitoring. The other child had an open depressed skull fracture

that required operative debridement and closure. This 19 m/o male had debris driven into his wound that required vigorous debridement to clean. His wound cultures were polymicrobic, including Argobacterium radiobacter and an unidentifiable fungus. The severely head injured patients required intracranial pressure monitoring with some of them requiring ventricular drainage and mannitol to control intracranial pressure. All of the children had good outcomes with all of them eventually returning home. Tornadic traumatic brain injury is not uncommon and is a frequent cause of death in tornado injuries. Aggressive management of intracranial pressure should be undertaken. Any open wounds should be aggressively debrided due to the unique type of abrasive trauma from high wind speeds. The wounds are usually contaminated with polymicrobial flora and require broad spectrum antibiotics. With prompt appropriate care, late morbidity and mortality can be minimalized

11. Transcranial Doppler Ultrasound Waveform Analysis for Noninvasive Intracranial Pressure Monitoring

Jens Peter Witt, MD, Shusma Aggarwal, MD, Howard Yonas, MD *(Pittsburgh,PA)*

Introduction: In Search for a noninvasive technique to monitor intracranial pressure (ICP)we evaluated ICP and blood flow velocity (TCD-Outline) in the middle cerebral artery (MCA) simultaneously.

Methods: In 12 patients with fulminant hepatic failure an average of 14 recordings (Range: 4-35) were done in each patient resulting in 166 TCD studies and 110 TCD and ICP stuides overall. The studies were performed in regular intervals or whenever a significant change in the patients clinical status or the monitored ICP occurred. The waveform of the transcranial Doppler ultrasound outline recording was analyzed off-line and described with 23 parameters. The recorded ICP values were arbitrarely grouped into 3 clincal valuable categories: Group I (ICP less than 20 mmHg), Group II (ICP = 20 - 38 mmHg), Group III (ICP greater than 38 mmHg).

Results: Nine waveform parameters out of 23 allowed to distinguish significantly (p is less than 0.05) between these 3 ICP categories. These nine parameters were put into a multiregression analysis with stepwise variable selection in order to determine the particular waveform parameters that contribute significantly to the correlation between the ICP value and the TCD waveform. Three waveform parameters showing the highest partial correlation were chosen for linear multiregression : the adjusted systolic peak

velocity, the diastolic upstroke time, and the ratio between the absolute increase in systolic velocity and the mean blood flow velocity (PI).

Discussion: Transcranial Doppler Ultrasound waveform analysis provides us with a good estimate of intracranial pressure in patients with diagnoses that need noninvasive ICP monitoring, i.e. fulminant hepatic failure, pediatric patients.

12. Spontaneous Thrombosis of Aneurysmal Malformation of Vein of Galen

Dimitrios C. Nikas, MD, Mark R. Proctor, MD, R. Michael Scott, MD *(Boston, MA)*

Introduction: Two cases of aneurysmal malformations of the vein of Galen (AVG) with spontaneous thrombosis with excellent clinical outcomes are reported.

Materials/Methods: Both of them had no high-flow symptoms or parenchymal cerebral abnormalities, with the exception of one patient's hydrocephalus. Angiogram and MRI demonstrated AVGs with slow arteriovenous shunts and associated stagnation of contrast in the venous sac secondary to severe outflow restriction. Based on these findings, one patient was managed conservatively, and the other underwent placement of a ventriculoperitoneal shunt.

Results: Surveillance of the lesions with subsequent MRIs revealed spontaneous thrombosis of the AVGs with excellent clinical outcomes.

Discussion/Conclusions: We recommend therapeutic intervention based on the patient's presentation and course. The degree of shunting remains the main factor determining the overall prognosis and management. Proposed mechanisms of spontaneous thrombosis include slow flow shunts, obstruction of the venous outflow, or of the feeding artery. A low flow pattern - due to stagnation of blood in the sac - demonstrated by color Doppler, MR techniques or conventional angiography appears to be a favorable condition for thrombosis. Additionally, detection of regressive changes in the vessel wall or the presence of small clots in the AVG or venous system might lead to thrombosis. Patients who may go on to complete aneurysmal thrombosis do not present with congestive heart failure, but rather are discovered incidentally or present with macrocephaly and hydrocephalus. An asymptomatic infant with the type of AVG described above could be treated expectantly and followed with MRI/MR angiography and serial examinations.

13. Treatment of Moyamoya Syndrome in Children

Ann Marie Flannery, MD, Michael Cowan, MD *(Charlotte, NC),* **Dennis McDonnell, MD** *(Augusta, GA)*

Background: Moyamoya disease causes vascular compromise which may result in infarcts in children and hemorrhage in young adults. The best available treatments are surgical supplying additional blood flow to the brain. A new technique for synangiosis has been sucessful in our patients.

Materials and Methods: Since 1994, a total of 5 patients have undergone 9 encephaloduralsynangiosis (EDAS) to supply additional blood flow to the brain. All treated individuals had angiographic evidence of moyamoya disease. One patient had sickle cell disease, one patient had sickle trait and one patient had Down Syndrome. In the remaining two patients no contributory disease was found. The patients ranged in age from 2-21 years and included three females and two males. Patients have been followed from 6 months to 5 years.

Results: Four of five patients had no new neurological events since their encephaloduralsynangiosis (EDAS). The patient with sickle cell disease has experienced transit neurological deficits related to high concentration of hemoglobin S. The encephaloduralsynangiosis (EDSA) performed was a modification of the procedure utilizing a dural graft to allow the donor vessel to be apposed to the pial surface without tension, to allow for a more water tight closure of the dura to prevent later wound complications.

Conclusion: A modification of EDAS technique has provided symptomatic relief, improved blood flow is documented by MRA and/or increase in caliber of basal ganglia vessels in patients with moyamoya disease. The technical aspects of this modified procedure will be discussed.

14. Combined Endovascular and Microsurgical Treatment of a Bilateral Thalamic Arteriovenous Malformation Associated with Vein of Galen Dilatation

Edward R. Flotte, MD, John A. Lancon, MD, David Scalzo, MD *(Jackson, MS),* **J. Parker Mickle, MD** *(Gainesville, FL),* **Adam I. Lewis, MD** *(Jackson, MS)*

The authors present the case of an 18-year-old male who was diagnosed shortly after birth with bilateral thalamic arteriovenous malformations (AVMs) associated with vein of Galen dilatation (VOGD), also known as a type IV vein

of Galen malformation. At eight years of age he underwent staged transtorcular embolization resulting in partial occlusion and reduction of flow through the lesion. At seventeen years of age, he developed progressive dilatation of the facial veins, retro-orbital pain, and cognitive decline. Transarterial embolization of the AVM was performed using N-butyl cyanoacrylate with a 60% reduction in flow. Two days later, a right posterior interhemispheric approach was performed to obliterate the remaining feeding arteries.

Angiographic obliteration of the malformation was demonstrated one month postoperatively, and the symptoms of venous hypertension were improved. One year following surgery, the patient lives independently and maintains gainful employment. Although thalamic AVMs with VOGD have traditionally been considered to be an inoperable subtype of vein of Galen malformation, combined microsurgical and endovascular treatment may be successful in reducing symptoms and risk of hemorrhage.

15. A Lesson to be Learned in Pediatric Subarachnoid Hemorrhage

Michael J. Burke, MD

A unique case report of the diagnosis and treatment of a ruptured cerebral aneurysm in a five-year-old male is presented. The child presented with subarachnoid hemorrhage and initial CT imaging was concerning for a right posterior communicating artery aneurysm. Cerebral angiography, MR/MRA imaging was normal. Repeat cerebral angiogram one month later was also normal. Sixteen months later an angiogram was performed to evaluate a subtle carotid bruit found on exam. A carotid bifurcation aneurysm pointing inferior and posterior was found. Craniotomy to clip the aneurysm was performed and marked adherent gliosis around the lesion was found supporting the fact that this was the causal lesion of his bleed in the past. Albeit a single case report, it underscores the need for very long term follow-up/evaluation of idiopathic pediatric subarachnoid hemorrhage.

16. Intractability Following Functional Hemispherectomy: Important Lessons Learned From an Unusual Case*

S. Mittal, MD, J.P. Farmer, MD, B. Rosenblatt, MD, F. Andermann, MD, J.L. Montes, MD *(Montreal, PQ)*, J.G. Villemure, MD *(Lausanne, Switzerland)*

Introduction: Surgical failures following functional hemispherectomy occur in about 15% of individuals with catastrophic epilepsy. These are traditionally attributed to incomplete disconnection, incorrect localization of epileptiform activity, or persistent focus in the ipsilateral insular cortex.

Clinical Presentation: We report the case of a 7-year-old boy with an intractable seizure disorder of classical "frontal adversive flavor" related to extensive unilateral left hemispheric cortical dysplasia. Intervention: Guided by intraoperative electrocorticography, extensive removal of epileptic frontal and precentral dysplastic tissue and transcortical subpial resection of dysplastic motor strip were done. Subsequently, a standard functional hemispherectomy with insular cortex resection was performed for persistent seizures. He was seizure-free for 6 months, then developed a new pattern consisting of an aura of fear with dystonic posturing of the right arm and unusual appetite post-ictally coupled to a "diencephalic-like" syndrome interictally. EEG and ictal/interictal SPECT localized the focus to the ipsilateral basal ganglia. MRI suggested a standard disconnection pattern. However, the presence of residual dysplastic tissue in the frontobasal area, normally left in place, was noted. Therefore, a final volumetric resection of the epileptogenic frontobasal tissue up to the anterior commissure was completed. The patient remains seizure-free for over 9 months.

Conclusion: Standard hemispherectomy techniques, although providing extensive disconnection, leave behind some frontobasal tissue, which rarely, especially if dysplastic, can represent a source of refractory seizures. Cortical dysplasia patients with ongoing post-hemispherectomy seizures are often thought to have microscopic contralateral dysplasia. It remains important to investigate them fully before considering them refractory to surgery.

17. Neurosurgical Management of Complex Pediatric Thoracolumbar Spinal Deformity

Gregory C. Wiggins, MD, Christopher I. Shaffrey, MD (Seattle, WA), Henry M. Bartkowski, MD, Michael J. Rauzzino, MD *(Detroit, MI)*

Surgical management of complex pediatric spinal deformity is increasingly performed by neurosurgeons. We present our experience in scoliosis (18-idiopathic, 4-neuromuscular, 1-neurofibromatosis) and severe kyphoscoliosis (2- congenital, 1-segmental spinal dysgenesis, 1-post laminectomy) and 1 grade IV spondylolisthesis. We prospectively evaluated the preoperative percent curve correction, operative time, blood loss, transfusion requirements, and complications. Follow-up averaged 11.9 months (4.5-21.9). All patients had their deformity corrected with anterior (Kaneda scoliosis system or Isola) or posterior (C-D Horizon) instrumentation with arthrodesis. Patients only received directed donation or autogenic blood

* *Considered for Shulman Award*

products. Deformity correction averaged 57 percent in patients with idiopathic scoliosis, 58 percent in non-idiopathic scoliosis, and 70 percent in kyphoscoliosis. The patient with grade IV spondylolisthesis had an 87 percent kyphotic angle correction.Complications in this series include one patient with inferior hook displacement requiring revision. Another patient with King II thoracolumbar scoliosis developed progression of her lumbar curve after T1-T12 instrumentation. Her construct was extended to L4 and since has done well. One patient developed acute hydrocephalus after his shunt became disconnected due to traction placed on it after curve correction. No case was complicated by neurological deterioration, loss of SSEP's, cardio-pulmonary disease, infection, donor site complication or wound breakdown. A detailed analysis of postoperative outcomes reveals significant improvement in cosmesis and no patient with continued postoperative pain. All patients have been able to return to their preoperative activities. Compared with other major operations, segmental instrumentation for pediatric deformity is a safe procedure with minimal morbidity and low risk of allogeneic blood products.

18. A Modified Cranial Reduction using Circumferential Barrel Stave Wedge Osteotomies

Jason Lifshutz, MD, Kuk-Wha Lee, MD, PhD, Ronald M. Perkin MD, Daniel J. Won, MD *(Loma Linda, CA)*

Cranial Reduction is a procedure associated with significant morbidity and seldom indicated as a treatment for macrocephaly secondary to congenital hydrocephalus. We report a combination of previously described techniques used in a successful one-stage reduction of a 7-month-old infant skull. Cranial Reduction was considered to prevent recurrent erosion at the shunt site and to decrease head mass. The technique involved: (1) initial removal of entire calvarium, (2) circumferential barrel stave wedge osteotomies, (3) plicating dura over convexities and sagittal sinus, (4) controlled incremental CSF drainage, (5) constricting the in-fractured, barrel staved and wedge resected skull base, and (6) recontouring the cranial vault. Post surgical outcome was satisfactory and cosmetic and functional improvement is documented in six-month followup.

19. Impact and Displacement Analysis for Pediatric Large Cranial Defect

Hun K. Park, PhD, Manuel Dujovny, MD, Fernando G. Diaz, MD, PhD *(Detroit, MI)*

Cranial vault defects in children can be induced by the congenital, traumatic, infectious or surgical event. The cranioplasty can restore cerebral protection, cosmetic aspect and neuronal function. It also reconstitutes the natural intracranial physiologic milieu. In children, acquired skull defects often close spontaneously. However, the difficulties arise when neurosurgeon has attempted on the larger defect greater than 10 cm2 in size. Such defect may not provide proper cerebral protection from post surgical injuries. We evaluated the cranioplasty using four popular materials with and without impact releasing hole by using finite element modeling.

A patch (area 46.7 cm2) from the cranial defect was designed by rapid prototyping process from 14 years old male patient. The properties of four implant materials (compact bone, spongy bone, PMMA and hydroxyapatite HA) were applied to calculate biomechanical parameters at the conjunction point with titanium plate.

The stress was mostly located at the impact site in all four materials. The stress was higher in PMMA (35.6 MPa) and lower in HA (33.4 MPa). Maximum displacement by the impact was 36 times higher in PMMA than in HA. Result showed that HA patch has lower strain (46) upon impact compare to PMMA (1140). Subsequently, the impact releasing holes in the patch increased stress (45.7±0.8 MPa) and maximum displacement (0.85±1.3 mm) on impact site. However, the strain (657% less) and stress (573% less) at the conjunction point has decreased significantly. Thus the hole initiated wide spread of regional impact to the general area of the patch. In this study, HA is appeared as a good candidate for large cranial defect and the reducing effect of marginal stress and strain was boosted by the presence of impact releasing hole.

20. Neuroendoscopic Third Ventriculostomy for Treatment of Chiari I Malformation with Hydrocephalus and Holocord Syringomyelia

Jens P. Witt, MD, Michael Partington, MD *(Denver, CO)*

Introduction: Neuroendoscopic Third Ventriculostomy as first line treatment for Chiari I related obstructive hydrocephalus with holocord syringomyelia. Methods: A 22 year old male presented to the Emergency

room with acute onset of severe headaches, neck pain and inability to stand or to walk. MRI of brain and spine revealed Chiari I malformation with massive Hydrocephalus and a holocord syringomyelia.

Results: After Neuroendoscopic Third Ventriculostomy his clinical improvement was rapid and dramatic. Postoperative magnetic resonance imaging several months after discharge demonstrated an almost completely vanished syringomyelia and dramatic shrinkage of all four ventricles.

Discussion: We reviewed all reports of treatment of syringomyelia or Chiari I malformations with Neuroendoscopic procedures and compared this technique with other surgical treatment options.

Conclusion: This simple and safe Neuroendoscopic Third Ventriculostomy should be considered as treatment of choice for patients with Chiari I malformation accompanied by syringomyelia and hydrocephalus.

21. Apoptosis and Dark Neurons in Hydrocephalic H-Tx Rats

Zhen-guo Li, MD, PhD†, Hong Zhang, Pat McAllister, PhD *(Detroit, MI)*

We have previously characterized dark and shrunken neurons in a kitten model of mechanically induced hydrocephalus. The objective of this study is to investigate if dark neurons occur in H-Tx rats with congenital hydrocephalus, and to explore the relationship between apoptotic neurons and dark neurons. Fixed brains were taken from H-Tx rats at 6, 12, and 21 days of age. Apoptotic neurons were identified by TUNEL assay and dark neurons were identified with Nissl staining. The number of dark cells was counted in 20 randomly picked areas (1 sq.mm each). At 21 days, when ventriculomegaly was most severe, the density of dark cells in hydrocephalics was significantly higher (28.0 +/- 2.6 %) than that of age-matched controls (13.0 +/- 5.0%, $p < 0.05$) and hydrocephalics at 12 days (18.9 +/- 3.5 %, $p < 0.05$) and 6 days ages (12.0 +/- 0.8 %, $p < 0.01$). Apoptotic cells were evident in the hippocampus, choroid plexus, and the ependyma of the lateral ventricle. TUNEL positive neurons in the hippocampus were found primarily in the same location as the dark cells, whereas TUNEL positive neurons accounted for only a small proportion of the dark cells in neocortex. Our results confirm the appearance of dark cells in the brains of H-Tx rats, and suggest that a portion of dark cells are undergoing apoptosis. Since the majority of dark cells may represent surviving neurons, therapies aimed at improving function of those neurons may contribute to improved management of hydrocephalus.

† Disclosure Information

22. Hydrocephalus in the H-Tx Rat: A Monogenic Disease?

Jogi V. Pattisapu, MD†, Xingang Cai, MD, Glenda McGraw, Laurence Von Kalm, PhD, Sue Willingham, Debra Socci, PhD, Jane Gibson, PhD *(Orlando, FL)*

The H-Tx rat is a model of hydrocephalus with a poorly understood mechanism of inheritance. We have analyzed data from eight generations of H-Tx rats and four generations of cross-matings of H-Tx rats and Sprague Dawley (SD) rats. We observed the hydrocephalus phenotype in 113 of 129 (87.60%) random brother-sister matings, with males and females equally affected. In the first generation of the cross-matings there was no hydrocephalic rats in 124 pups (F1). However, subsequent brother-sister matings of F1 animals yielded a lower incidence of the disease in their pups (F2) (4.67% in hydrocephalic HTx rat/SD rat matings and 5.11% in normal HTx rat/SD rat matings, respectively) than the overall incidence observed for the H-Tx rat colony (30.35%). The back-cross matings between the F2 generations and the H-Tx rats yielded the hydrocephalus with a higher incidence than the cross matings but still lower than the H-Tx rat colony. The data from random matings, cross-matings and backcross matings strongly suggest that the H-Tx rat is a homozygous carrier of an autosomal recessive hydrocephalus gene. The invariable incidence in the H-Tx rats suggests that the gene is incompletely penetrated. The lower incidences in the cross-matings and in the back-cross matings may be attributed at least in part to the modified effect of the SD rat genome. Furthermore, the data clearly rule out sex-linked and polygenic modes of inheritance and provide further insight with respect to genetic inheritance of hydrocephalus.

23. External Lumar Drainage in the Pediatrics Population

Abdi S. Ghodsi, MD, Homan Mostafavi, BS, Farid Mousavi-Harami, BS, Arnold Menezes, MD, Vincent Traynelis, MD *(Iowa City, IA)*

Lumbar drainage in the pediatrics population is an uncommon neurosurgical procedure. Studies to evaluate indications, techniques, effectiveness and complications of these catheters are rare. We retrospectively reviewed our experience with the use of external lumbar subarachnoid drainage from 1988 to 1999 at the University of Iowa Hospitals & Clinics. A total of 25 lumbar drains were placed in 21 children ranging in age from 2-16 years old (Avr.10.5). Most (n=15) were used intraoperatively for assistance with brain relaxation and exposure. These ranged from tumor or AVM resection to cranioplasty. In 12 cases the drain was used successfully to

† Disclosure Information

achieve the goal. In 3 cases the drains were not needed. CSF drainage in the former ranged from 50 to 300 cc (mean 145 cc) of spinal fluid. The remainder of the drains (n=10) were placed for CSF leakage or transient hydrocephalus. These drains were maintained from 2-10 days with average length of 6.2 days. Of the 10 drains, 8 were considered successful but 2 failed in resolving CSF leaks requiring operative intervention. In our series, there was 1 case of E. Coli meningitis attributed to the drain, which was successfully treated with antibiotics. There were also 2 cases of persistent spinal fluid leakage from the catheter entry site with one requiring a blood patch for resolution. Our data shows that external lumbar drainage is an effective and safe tool for both intraoperative use and for assistance with CSF leakage/communicating hydrocephalus in various pathological conditions.

24. Molecular Biology and Genetics of Hydrocephalus

Xingang Cai, MD †, Debora Bailey, MS, Jogi Pattisapu, MD *(Orlando, FL)*

Hydrocephalus is an etiologically heterogeneous disease. Its causes include trauma, subarachnoid hemorrhage, infection (meningitis), developmental abnormalities and genetic changes. The pathogenesis of hydrocephalus, however, is still unknown. We have reviewed the recent advances of the molecular biology of hydrocephalus, focusing on the most widely studied or newly identified genes. 1.) Cell adhesion molecule L1 (L1CAM). It is the only gene recognized to cause human hydrocephalus. It is a single transmembrane molecule involved in neural cell adhesion, neurite outgrowth and pathfinding, neuronal migration and myelination, and memory and learning. Mutations in the L1CAM gene are highly variable occurring throughout the molecule and cause hydrocephalus and other neurological abnormalities. 2.) Transforming growth factor ß1 (TGF-ß1). TGF-ß1 is a multifunctional cytokine. Overexpression of TGF-ß1 in transgenic mice brain caused severe hydrocephalus. The mechanism is proposed to relate with increased production of extracellular matrix components. Also TGF-ß1 is increased in some other neurological diseases. 3.) Forkhead/winged helix. The forkhead/winged helix is a member of a large family of evolutionarily conserved DNA-binding proteins. It was recently reported that disruption of this gene caused severe hydrocephalus in mouse. The mechanism is unknown. 4.) Otx2. Otx2 functions as a head organizer. It plays an important role in head morphogenesis. The heterozygotes mice of mutant Otx2 gene showed eminent dilation of lateral ventricles and a ballooned cerebrum. Histological analysis shows edematous change of the periventricular white matter. The mechanism is unknown.

† Disclosure Information

25. Craniopharyngiomas: Experience with Complex Approaches in Children

Mark A. Liker, MD, Michael L. Levy MD, J. Gordon McComb, MD

Craniopharyngiomas are benign intracranial tumors that can present complicated management problems due to the invasive nature of the lesions and the poor response to adjuvant therapy. Controversy exists relating to the correct combination of surgery and adjuvant radiation or chemotherapy. Given that optimal cure rates are related to the completeness of resection, efficacy of surgical approach is a significant determinant of potential outcomes. We evaluated our series of nineteen patients since 1996 with craniopharyngiomas. Twenty distinct operative procedures were performed. Approaches depended on the location and expanse of the tumor. Overall, six transphenoidals, eleven anterior or anterolateral skull-base approaches (subfrontal, subfrontal/pterional, subfrontal/orbitozygomatic, orbitozygomatic) and three combined procedures (interhemispheric/ orbitozygomatic, transbasal/interhemispheric) were performed. Patient ages ranged from two years and seven months at the date of surgery to 17 years two months with an average of eight years two months (+/- four years six months). Seven of the patients were female and twelve male. Average follow-up was fifteen months with a range of two months to 32 months. We will discuss the indications for each approach and the related complications including hemianopsia, ophthalmoplegia, diabetes insipidus, panhypopituitarism, coma, and recurrence. The attendant limitations of each approach will be discussed with regard to tumor extension and injury to eloquent structures.

26. Histopathology of Carotid Aneurysmal Dilation after Surgery for Craniopharyngioma*

Amir Vokshoor, MD, Dan Boue', MD, Edward J. Kosnik, MD *(Columbus, OH)*

Introduction: Aneurysmal dilatation of carotid artery has been reported in association with surgical resection of craniopharyngioma The type of aneurysms in these cases have mostly been fusiform, although their histopathology to date has not been reported. We report on a child who was found to have a post-operative fusiform dilatation one year after surgery and lived 5 years without any symptoms related to the aneurysm, before dying of an unrelated cause.

** Considered for Shulman Award*

Methods: During autopsy the aneurysmal dilatation was found in the suparclinoid ICA just above the M-1, A-1 branch points. The aneurysm was serially sectioned to reveal irregular fibrous thickening of the wall. Microscopic sectioning of the aneurysm with special attention to the internal elastic lamina was performed.

Results: Light Microscopy revealed severe degeneration of the internal elastic lamina. There was no medial defect and no evidence of intimal stretch injury. The adventitia was scarred but not violated. There was a rim of hyalinizing fibrous obliteration of the lumen with signs of chronic inflammation indicative of low flow state.

Conclusion: This fusiform aneurysm has a definite association with surgical manipulation, but one has to consider alterations in the hemodynamics causing the critical amount of injury to the internal elastic lamina. The natural history illustrated by these lesions demonstrate a low rate of hemorrhage and is further confirmed by our findings of chronic inflammation and low flow state.

27. Intraventricular Pilocytic Astrocytoma in an Infant: Case Report and Review of the Literature

Holly S. Gilmer-Hill, MD, Kamran Sahrakar, MD, Dachling Pang, MD
(Sacramento, CA)

Pilocytic astrocytoma occurs most frequently in children and young adults. It occurs only rarely in the supratentorial compartment in infants, and, to our knowledge, has never been reported arising intraventricularly in an infant. We present a four-month-old infant who presented with right gaze deviation, irritability, frequent vomiting, and rapidly-increasing head circumference. Imaging studies showed a large left-sided, minimally-enhancing intraventricular mass extending laterally and superiorly into the frontal, parietal, and temporal regions. Gross total resection of the mass was performed via left frontotemporoparietal craniotomy. Pathologic examination revealed benign juvenile pilocytic astrocytoma. The epidemiology, presentation, and management of these neoplasms is reviewed.

28. Intraspinal Hemangioendothelioma in a Premature Infant: An Epidural Hemangioma Variant

Bohdan W. Chopko, PhD, MD, Robert Buchanan, MD,
Christina Stanley, MD, Lawrence Hansen, MD *(San Diego, CA)*

Purpose: The diagnosis of intraspinal neoplastic mass lesions during infancy is rare, with the cervical spine the least likely affected site. We present a case of intraspinal hemangioendothelioma that progressed to severe cervical cord compression prior to recognition.

Methods: A 6 month old male, born premature at 26 weeks gestation, presented with 2 months of progressive upper extremity weakness. On exam, he had near complete flaccid paralysis of the upper extremities, increased tone in the lower extremities, and a right supraclavicular soft tissue mass. MR demonstrated a fusiform, enhancing mass within the spinal canal, extending from C2 to T2, leading to severe compression and displacement. The mass was contiguous via the right C6 neural foramen with the right paravertebral mass.

Results: At surgical resection, a tan, well-vascularized extradural mass was encountered, which was densely adherent to dura and spinal roots. Pathology revealed a highly cellular and vascular tumor composed of rounded endothelial cells forming back-to-back small lumina or sinusoidal vascular channels. The tumor cells showed minimal pleiomorphism, bland nuclei, virtually no mitotic activity, and no necrosis. These feaures were consistent with hemangioendothelioma, or benign hemangioma of infancy. Postoperatively, the patient achieved significant motor recovery, and is free of recurrence at 2 year follow-up.

Conclusion: This case illustrates the difficulty of early recognition of infantile cord compression, and is the youngest reported case of intraspinal hemangioendothelioma. Additional unusual features include the epidural configuration of the mass, and occurrence at the cervical level.

29. Cerebellar Mutism Associated with a Midbrain Lesion

Marjorie C. Wang, MD, Robert E. Breeze, MD, Ken R. Winston, MD
(Denver, CO)

There have been many reports of cerebellar mutism since its initial description in the 1980's; however, the pathophysiology and the anatomic basis of this disorder have remained unclear. The clinical picture involves

mutism beginning several hours or days after surgery, followed by a period of dysarthria encompassing weeks to months but eventually resolving. It usually occurs in the pediatric population. Various theories have been proposed to explain this phenomenom. The dentate nucleus has been implicated in some reports, but other authors target the left cerebellar hemisphere. Most cases have been associated with posterior fossa tumors. Bilateral lesions of the thalamus have been shown to cause a similar clinical picture. There is one report of cerebellar mutism arising from a lesion in the pons that was removed via a supratentorial approach.

We describe a fifteen year old boy who hemorrhaged from a cavernous angioma in the left mesencephalic tectum. He presented with an acute left third nerve palsy, right hemiparesis and mild dysarthria. Following resection of the lesion via a posterior subtemporal route, the patient displayed a clinical picture consistent with "cerebellar" mutism. We hypothesize that involvement of the dentato-thalamic fibers unilaterally at the level of the mesencephalon was responsible for this child's transient speech impairment, thereby providing support for the theory that the dentate nucleus plays a key role in cerebellar mutism.

30. Cellular Immunotherapyof Brain Tumors Involving Intratumoral Placements of the Irradiated TALL-104 Cytotoxic T Cell Line

Jane E. Freeman, CPNP, Ken R. Winston, MD, Nicholas Foreman, MD, David B. Paul, PhD, Carol J. Gup, BA, German Gomez, BS *(Denver, CO)*, Sophie Visonneau, PhD, Daniela Santoli, PhD *(Philadelphia, PA)*, Carol A. Kruse, PhD *(Denver, CO)*

Pediatric and adult recurrent malignant brain tumor patients are being accrued in a Phase I immunotherapy trial. The study is designed to establish safety and the maximum tolerated dose of irradiated TALL-104 cells given intratumorally. TALL-104 cells are capable of non-MHC restricted lysis of brain tumors. Patients can receive up to 5 treatment cycles at 8-week intervals. One cycle consists of 3 infusions of TALL-104 cells over a 5-10 day period. One pediatric patient (13 yo male) initially diagnosed with a malignant astrocytoma, and now ependymoma, of the cerebellum has been entered onto protocol. During his clinical course he has undergone a series of aggressive therapies always followed by tumor recurrences. The therapies have included multiple surgeries, radiation therapy (5400cGy), chemotherapy (BCNU/cisplatin), high dose chemotherapy (VP16/thiotepa/carboplatin) with stem cell rescue, and oral VP16 and Tamoxifen. In April, 1999 the boy underwent surgical debulking of his tumor followed by 3 infusions of 10e7 TALL-104 cells

over 9 days. At surgery a catheter was directed into the tumor bed to facilitate subsequent infusions. Eight weeks later, a second cycle of TALL-104 cells, consisting of 3 infusions at 10e7, were administered on days 1, 3 and 5. At this time his tumor is stable by MRI. The patient tolerated the immunotherapy well. He exhibited little to no reaction: low-grade fever at treatment cycles 1 (post-operative) and 2, and minor headache at treatment cycle 2. This patient was the first to receive TALL-104 cells intracranially and where delivery was directly into a tumor bed.

31. Alteration of Cell-cycle Kinetics Inhibits Growth Rate in Rat C6 Glioma Cells Transfected with Somatostatin cDNA Under Control of a MoMuLV Promoter

Catherine A. Ruebenacker, MD, Sacchieri TA, Shao TM, Carmel PW, Siegel JH, Feldman, SC *(Newark, NJ)*

Somatostatin (SRIF) alters cellular growth and division through autocrine, paracrine and endocrine mechanisms. SRIF binds to specific receptors that affect cell-cycle kinetics. C6 glioma cells transfected with SRIF cDNA, under the control of a MoMuLV promoter demonstrate growth inhibition in vitro and in vivo.

C6 glioma cells transfected with a plasmid carrying SRIF cDNA in sense orientation (C6-pSS) exhibit a significant reduction in growth rate without increase in apoptosis. Anti-sense transfectants (C6-pAS) grow at rates similar to wild-type glioma cells.

Transcription of SRIF cDNA was verified with RT-PCR. Wild-type and C6-pAS transfectants do not generate SRIF mRNA, whereas high titer transcription of SRIF was appreciated in sense transfectants. Antibodies against SRIF were used to confirm translation and expression of somatostatin.

Stable transfection and constituitive expression of SRIF alters cell-cycle kinetics dramatically. A significant change occurs in G2-M and S phases. S phase population is 27% in wild-type glioma cells. The C6-pSS transfectants show reduced entry into S phase with only 12% of the population dispersed in S phase at any cell density.

Animal studies demonstrate decreased malignancy of the SRIF sense transfectants, as compared to wild-type.

The MoMuLV promoter generates high titer somatostatin transcripts in the sense transfected C6-pSS cell line. The "constituitive" expression of SRIF inhibits growth rate through alteration of cell-cycle kinetics. A block in the cell-cycle prohibits progression through mitosis. Decreased tumorigenicity results without apoptosis. The inhibition of growth rate through SRIF gene transfer and forced expression has interesting therapeutic potential.

32. Intrathecal Alloreactive Cytotoxic T Lymphocytes and Interleukin-2 for Recurrent Ependymoma—A Phase 1 Study

Ken R. Winston, MD, Jane Freeman, RN, CNPN, Nicholas Foreman, MD, Natalie Parker, PhD, Loris McGavran, PhD, Elena Savelieva, PhD, Carol A. Kruse, PhD *(Denver, CO)*

A 7-year-old girl with recurrent ependymoma with a CSF shunt, having failed prior aggressive treatment consisting of surgery, chemotherapy, and radiation therapy, was treated with intrathecal (IT) administrations of alloreactive cytotoxic T lymphocytes (aCTL) and Interleukin-2 (IL-2). Three treatment cycles consisted of 8 IT infusions and totaled 30.9 x 10e8 aCTL. The aCTL, sensitized to patient histocompatibility locus antigens (HLA), were generated in CELLMAX® artificial capillary systems. In one-way mixed lymphocyte reactions, responder lymphocytes from HLA-disparate donors, differing at 3 HLA-AB loci to the patient, were incubated with irradiated sensitizing lymphocytes from the patient. aCTL developed over 2-3 weeks in IL-2-containing medium. Two weeks following radiosurgery to reduce the bulk of the enhancing lesion, 3 IT administrations of aCTL and IL-2 (60,000 IU/infusate) were given over 9 days. Second and third treatment cycles, spaced 8 weeks apart, consisted of 2 and 3 IT aCTL infusions, respectively. aCTL for each cycle were from different male donors. For the third cycle the ventricular shunt was disconnected and aCTL infusions were through a catheter directed toward one of 2 enhancing lesions. The patient mounted an endogenous immune reaction evidenced by cell counts and differentials of cerebrospinal fluid cells and by FISH analyses using X (patient) and Y (donor) chromosome probes. The immunotherapy was tolerated well. Transient toxicity (fever, headache and nausea) at grades 1-2 (NCI Common Toxicity Scale criteria) was observed. This is the first instance of IT delivery of aCTL in a child.

33. Frameless Stereotactic Guided Resection for Thalamic/Basal Ganglia Tumors

George I. Jallo, MD, Karl Kothbauer, MD, Jaoa Siffert, MD, Fred Epstein, MD *(New York, NY)*

Objective: Deep midline tumors (thalamic, basal ganglia, third ventricle) are difficult tumors to remove in children. Although the majority of these tumors are low grade astrocytomas, the surgical resection is hazardous. We describe our results for 8 tumors which were resected with stereotactic guidance.

Methods: During a two year period, 8 children (age, 2-15 years) with deep seated tumors underwent stereotactic craniotomies. All patients had preoperative MRI and registration to the frameless stereotactic system (ISG wand). All tumors were approached via a transcallosal route.

Results: Gross total resection was possible in 7 children and radical but subtotal removal (>80%) was performed in 1 child. Transient worsening of the hemiparesis was seen in 20% of the patients, but all improved in the follow-up period.

Conclusion: Frameless stereotaxy allows for the safe and guided removal of deep seated neoplasms with a minimally invasive approach.

34. Spinal Cord Tumor Presenting with Cerebral Atrophy

Scott W. Elton, MD, Amir Vokshoor, MD, Edward Kosnik, MD *(Columbus, OH)*

Intramedullary spinal cord tumors in children are unusual. We present a case report of a child who was diagnosed with developmental regression. During the patient's workup, significant cerebral atrophy was found. Subsequently, an intramedullary spinal cord tumor was discovered and underwent biopsy. Pathological examination revealed a glioblastoma multiforme. Cerebrospinal carcinomatosis was not seen, despite extensive evaluation of the spinal fluid. This is the first case of a spinal cord tumor presenting with cerebral atrophy and developmental regression. We discuss the salient features of this case and suggest a possible mechanism linking the cerebral atrophy with the spinal cord tumor.

American Association of Neurological Surgeons
and
Congress of Neurological Surgeons

Section on Pediatric Neurological Surgery

1999 Membership Roster

Munir H. Abbasy, MD
80 Congress St.
Springfield, MA 01104-3427

Rick Abbott, III, MD
Beth Israel Med. Ctr.
170 E. End Ave./Neuro.
New York NY 10128

P. David Adelson, MD
Children's Hosp. of Pittsburgh
3705 Fifth Ave./Neurosurgery
Pittsburgh PA 15213-2583

Nejat Akalan, MD
Dept. of Neurosurgery Hacettepe
Univ. Sch. of Med.
Ankara 6100 Turkey

A. Leland Albright, MD
Children's Hosp. Pittsburgh
3705 5th Ave./Neurosurgery
Pittsburgh, PA 15213-2524

Lance Altenau, MD
501 Washington, Suite 700
San Diego, CA 92103-2231

A. Loren Amacher, MD
Dept. of Neurosurgery Geisinger
Clinic Danville, PA 17822-0001

Luis V. Amador MD
1440 Veteran Ave., #336
Los Angeles, CA 90024-4832

Ahmed S. Ammar, MD, PhD
KFHU/Neuro. PO Box 40040
Al-Khobar 31952 Saudi Arabia

Jim D. Anderson, MD
PO Box 658
San Carlos, CA 94070

Brian T. Andrews, MD,
UCSF/Dept. of Neurosurgery
2100 Webster St., Suite 521
San Francisco, CA 94115-2382

Thomas J. Arkins, MD
Connecticut Neurosurgery, PC
60 Temple St., 5B
New Haven, CT 6510

Patricia A. Aronin, MD
4314 Medical Pkwy., Suite 201
Austin, TX 78756

Elaine J. Arpin, MD
S.W. Florida Neuro. Assoc.
413 Del Prado Blvd., Suite 102
Cape Coral, FL 33990-5710

Wilson T. Asfora, MD
800 Watercress Cir.
Sioux Falls, SD 57108-2816

Saleh S. A. Baeesa, MD
King Abdulaziz Univ. Hosp.
PO Box 6615
Jeddah 21452 Saudi Arabia

Walter L. Bailey, MD
Doctors' Professional Bldg.
280 N. Smith Ave., Suite #234
St. Paul, MN 55102-2420

Gene A. Balis, MD
Suite 340 3000 E. Fletcher Ave.
Tampa, FL 33613-4645

Steven J. Barrer, MD
1584 Old York Rd.
Abington, PA 19001-1709

Henry M. Bartkowski, MD, PhD
Henry Ford/Wm. Beaumont Hosp.
2799 W. Grand Blvd., K11,W1141
Detroit, MI 48202

James E. Baumgartner, MD
6410 Fannin, Suite 1400
Houston, TX 77030

Robert M. Beatty, MD
8919 Parallel Pkwy., Suite 455
Kansas City, KS 66112-1655

William O. Bell, MD
Carolina Neuro. Assoc., PA
2810 Maplewood Ave.
Winston-Salem, NC 27103-4138

Edward C. Benzel, MD
Cleveland Clinic Fdtn.
9500 Euclid Ave., Desk S80
Cleveland, OH 44195

Mitchel S. Berger, MD
UCSF/Neurosurgery
505 Parnassus, Room M-787
San Francisco, CA 94143-0112

Thomas S. Berger, MD
3300 E. Galbraith Rd.
Cincinnati, OH 45236

Karin S. Bierbrauer, MD
St. Christopher's Hosp.
Erie & Front St./Ped. Neuro.
Philadelphia, PA 19134

Peter McL. Black, MD, PhD
Children's Hosp./Neuro.
300 Longwood Ave.
Boston, MA 2115

Jeffrey P. Blount, MD
Apt. 2505 77 Gergaro
St. Toronto, ON M5G 2A1 Canada

John Scott Boggs, MD
1820 Barrs St., Suite 104
Jacksonville, FL 32204-4742

Frederick A. Boop, MD
9601 Lile Dr., Suite 750
Little Rock, AR 72205

William R. Boydston, MD
5455 Meridian Mark Rd.
Suite #540
Atlanta, GA 30342-1640

Bruce C. Bressler, MD
720 S. Van Buren
Green Bay, WI 54301-3538

Douglas L. Brockmeyer, MD
Primary Children's Med. Ctr.
100 N. Medical Dr., Suite 2400
Salt Lake City, UT 84113-1100

Jeffrey Alan Brown, MD
Med. Coll. of Ohio/Neurosurg.
3065 Arlington Ave.
Toledo, OH 43614

Derek A. Bruce, MD
Neurosurgeons for Children
7777 Forest Ln., B308
Dallas, TX 75230

William A. Buchheit, MD
6014 Cricket Rd.
Flourtown, PA 19031

Michael James Burke, MD
Neurological Clinic
3006 S. Alameda St.
Corpus Christi, TX 78404-2601

Leslie D. Cahan, MD
Neurosurgery
1505 N. Edgemont St., Rm.4141
Los Angeles, CA 90027-5209

Alexa Irene Canady, MD
Children's Hosp. of Michigan
3901 Beaubien, 2nd Fl.
Detroit, MI 48201-2119

Carolyn Marie Carey, MD
Primary Children's Med. Ctr.
100 N. Medical Dr.
Salt Lake City, UT 84113-1100

Peter W. Carmel, MD
New Jersey Med. Sch./Neuro.
90 Bergen St., Suite 7300
Newark, NJ 07103-2499

Benjamin Carson, MD
Johns Hopkins Hosp.
600 N. Wolfe St., Harvey 811
Baltimore, MD 21287-8811

Michael J. Chaparro, MD
Neuro. Inst. of Palm Beach
5507 S. Congress Ave., #150
Atlantis, FL 33462

Paul H. Chapman, MD
Massachusetts Gen. Hosp.
Fruit St., GR8502"
Boston, MA 2114

William R. Cheek, MD
3009 Robinhood
Houston, TX 77005-2343

Maurice Choux, MD
Hopital Des Enfants
De La Timone, Cedex 5
Marseilles 13385 France

Samuel F. Ciricillo, Jr., MD
5238 Fair Oaks Blvd.
Carmichael, CA 95608

Philip Harry Cogen, MD, PhD
Children's National Med. Ctr.
111 Michigan Ave., N.W./Neuro.
Washington, DC 20010-2916

Alan R. Cohen, MD
Case Western Reserve Univ.
11100 Euclid Ave./Neuro.
Cleveland, OH 44106-1736

Shlomi Constantini, MD
Dana Children Hosp.
6 Weizman St./Ped. Neurosurg.
Tel Aviv 64239 Israel

Richard A. Coulon, Jr. MD
Ochsner Clinic/Neuro.
1514 Jefferson Hwy.
New Orleans, LA 70121-2429

Jeffrey W. Cozzens, MD
Div. of Neurosurgery
2650 Ridge Ave.
Evanston, IL 60201-1718

Kerry R. Crone, MD
Children's Hosp. Med. Ctr.
3333 Burnet Ave.
Cincinnati, OH 45229-3039

T. Forcht Dagi, MD
Suite 475
2500 Northwinds Pkwy.
Alpharetta, GA 30004

Robin I. Davidson, MD
Univ. Massachusetts Med. Ctr.
55 Lake Ave., N./Neuro.
Worcester, MA 01655-0002

Richard A. A. Day, MD
601 W. Spruce St., Suite E
Missoula, MT 59802-4047

Concezio Di Rocco, MD
Univ. Cattolica/Neurochirurgia
Largo Gemelli 8 Rome 168 Italy

Mark S. Dias, MD
Children's Hosp. of Buffalo
219 Bryant St.
Buffalo, NY 14222-2006

Michael Dorsen, MD
Suite 210
3615 NW Samaritan Dr.
Corvallis, OR 97330

James R. Doty, MD
570 Del Rey Ave.
Sunnyvale, CA 94086-3528

James M. Drake, MD
Hosp. for Sick Children
555 University Ave., #1504-D
Toronto ON M5G 1X8 Canada

Ann-Christine Duhaime, MD
Child. Hosp. of Philadelphia 34th
& Civic Ctr. Blvd./Neuro.
Philadelphia, PA 19104

Charles Cecil Duncan, MD
333 Cedar St., TMP 416
PO Box 208082
New Haven, CT 06520-8082

John A. Duncan, III, MD
Suite 530 2 Dudley
St. Providence, RI 02905-3236

Mary E. Dunn, MD
280 N. Smith Ave., #234
St. Paul, MN 55102-2420

Michael S. B. Edwards, MD
2800 L St., #340
Sacramento, CA 95816-5616

Michael R. Egnor, MD
NY Spine & Brain Surgery PC
Dept. of Neurosurgery/HSC T12
Stony Brook, NY 11794-8122

Stephanie L. Einhaus, MD
Semmes-Murphy Clinic
930 Madison Ave., Suite 600
Memphis, TN 38103-2152

Howard M. Eisenberg, MD
Div. of Neurosurgery
22 S. Greene St., #S10B11
Baltimore, MD 21201-1734

Ibrahim Muftah El Nihum, MD
Scott & White Hosp.
2401 S. 31st
St. Temple, TX 76508-0001

Richard G. Ellenbogen, MD
Children's Hosp. & Med. Ctr.
4800 Sand Point Way, N.E.
Seattle, WA 98105-0371

Seyed M. Emadian, MD
Mountain Neuro. Inst.
7 McDowell St.
Asheville, NC 28801

Fred J. Epstein, MD
Beth Israel Med. Ctr., N. Div.
170 E. End Ave.
New York, NY 10128-7603

Mark D. Erasmus, MD
522 Lomas Blvd., N.E.
Albuquerque, NM 87102-2454

Walter J. Faillace, MD
Univ. of Florida, Jacksonville
653 W. Eighth St./Neurosurgery
Jacksonville. FL 32209

Neil Arthur Feldstein, MD
Neurological Inst.
710 W. 168th St., Room #414
New York, NY 10032

David A. Fell, MD
Neurosurgery Spec.
6767-A S. Yale
Tulsa, OK 74136-3303

Edwin G. Fischer, MD
110 Francis St., Suite 3B
Boston, MA 02215-5501

Ann Marie Flannery, MD
Med. Coll. of Georgia
1120 15th St./Neurosurgery
Augusta, GA 30912-4010

Eldon L. Foltz, MD
UCI Med. Ctr./Neurosurgery
PO Box 14091
Orange, CA 92651

Paul C. Francel, MD, PhD
Univ. of Oklahoma HSC/Neuros.
711 Stanton L. Young Blvd.,206
Oklahoma, City OK 73104

Kathleen B. French, MD
3020 Hamaker Ct., Suite B104
Fairfax, VA 22031-2220

Arno H. Fried, MD
Hackensack Univ. Med. Ctr.
30 Prospect Ave./WFAN Peds Ctr
Hackensack, NJ 7601

David M. Frim, MD
Univ. of Chicago MC/Neuro.
5841 S. Maryland Ave., MC-4066
Chicago, IL 60637-1463

Herbert E. Fuchs, MD, PhD
Duke Med. Ctr./Neurosurgery
Box 3272
Durham, NC 27710-0001

Norman H. Gahm, MD
Suite 705
100 Retreat Ave.
Hartford, CT 06106-2528

Joseph H. Galicich, MD
PO Box 276
Alpine, NJ 07620-0276

Francis W. Gamache, Jr., MD
Neuroscience Inst.
523 E. 72nd St., 7th Fl.
New York, NY 10021-4099

Sarah J. Gaskill, MD
4499 Medical Dr., Suite 397
San Antonio, TX 78229-3735

Rosemaria Gennuso, MD
1661 Soquel Dr., Suite F
Santa Cruz, CA 95065

Richard E. George, Jr., MD
3506 21st St., #400
Lubbock, TX 79410-1200

Timothy M. George, MD
Duke Univ. Med. Ctr. Box 3272/
Div. of Neurosurgery
Durham, NC 27710-0001

P. Langham Gleason, MD
2736 La Bajada
Santa Fe, NM 87505

John C. Godersky, MD
2841 De Barr Rd., Suite 34
Anchorage, AK 99508-2956

James T. Goodrich, MD, PhD
Dept. of Neuro./Montefiore
111 E. 210th
St. Bronx, NY 10467-2401

Liliana C. Goumnerova, MD, FRCSC, FACS
Children's Hosp., Bader 3
300 Longwood Ave.
Boston, MA 02115-5724

Paul A. Grabb, MD
Children's Hosp.
1600 7th Ave., S., ACC 400
Birmingham, AL 35233-1711

Clarence S. Greene, Jr., MD
2650 Elm Ave., #218
Long Beach, CA 90802

Laurance J. Guido, MD
30 Sutton Pl., #15A
New York , NY 10022-2365

Francisco A. Gutierrez, MD
201 E. Huron, #9-160
Chicago, IL 60611

Jens Haase, MD
Aalborg Univ. Hosp./Neurosurg.
Aalborg 9100 Denmark

Yoon Sun Hahn, MD
Hope Children's Hosp.
4440 W. 95th St./Ped. Neuros.
Oak Lawn, IL 60453-2600

Stephen J. Haines, MD
MUSC/Dept. of Neurosurgery
171 Ashley Ave.
Charleston, SC 29425-2272

Sten E. Hakanson, MD
J O Wallins Vag 7
Solna S-17164 Sweden

Mark G. Hamilton, MD
Alberta Children's Hosp.
1820 Richmond Rd., S.W.
Calgary AB T2T 5C7 Canada

Mary Kathryn Hammock, MD
8620 Rolling Rd.
Manassas, VA 20110-3828

Michael H. Handler, MD
Suite 605, Tammen Hall
1010 E. 19th St.
Denver, CO 80218-1808

William C. Hanigan, MD, PhD
Suite 500
214 N.E. Glen Oak Ave.
Peoria, IL 61603

Michael D. Heafner, MD
Carolina Neurosurgery & Spine
1010 Edgehill Rd., N.
Charlotte, NC 28207-1885

Michael A. Healy, MD
2727 Westchester
Toledo, OH 43615-2243

Dan S. Heffez, MD
2515 N. Clark St., Suite 800
Chicago, IL 60614-2720

Leslie C. Hellbusch, MD
111 N. 84th St.
Omaha, NE 68114-4101

David C. Hemmy, MD
20611 Watertown Rd., Suite J
Waukesha, WI 53186-1871

Robert W. Hendee, Jr., MD
4710 Lookout Mountain Cv.
Austin, TX 78731-3654

E. Bruce Hendrick, MD
63 Leggett Ave.
Etobicoke ON M9P 1X3 Canada

Tenoch Herrada-Pineda, MD
Apartado Postal 101-87
Mexico City 4530 Mexico

Harold J. Hoffman, MD
110 Bloor St., W., Apt. 607
Toronto ON M5S 2W7 Canada

Thomas J. Holbrook, Jr., MD
3 Medical Park Rd., Suite 310
Columbia, SC 29203-6873

Robert D. Hollenberg, MD
McMaster Univ./Surgery Dept.
1200 Main St., W., Room #4E8
Hamilton ON L8N 3Z5 Canada

Gregory W. Hornig, MD
4320 Wornall Rd., Suite 714
Kansas City, MO 64111-3210

Roger Hudgins, MD
Suite 540
5455 Meridan Mark Rd.
Atlanta, GA 30342

Stephen L. Huhn, MD
Stanford Univ.
300 Pasteur Dr., R161
Palo Alto, CA 94305-5327

Robin P. Humphreys, MD
Hosp. for Sick Children
555 University Ave., #1504
Toronto ON M5G 1X8 Canada

Bermans J. Iskandar, MD
17 St. Lawrence Cir.
Madison, WI 53717-1827

Hector E. James, MD
7930 Frost St., Suite 304
San Diego, CA 92123-2737

John A. Jane MD, PhD
Univ. of Virginia/Neurosurgery
Hosp. Box 212
Charlottesville, VA 22908

David F. Jimenez, MD
Univ. of Missouri Hosp. & Clin
One Hospital Dr., N521
Columbia, MO 65212-0001

Dennis L. Johnson, MD
Milton Hershey Med. Ctr.
PO Box 850/Div. of Neuro.
Hershey, PA 17033-0850

John K. Johnson, MD
20 Medical Ridge Dr.
Greenville, SC 29605

Martin Johnson, MD
1818 N.E. Irving St.
Portland, OR 97232-2298

Mary M. Johnson, MD
3223 Chatham Rd.
Atlanta, GA 30305-1101

Robert F. C. Jones, MD
21 Norfolk St.
Paddington NSW 2021 Australia

Allen S. Joseph, MD
Suite 10000
7777 Hennessy Blvd.
Baton Rouge, LA 70808-4300

John E. Kalsbeck, MD
Riley Children's Hosp.
702 Barnhill Dr.
Indianapolis, IN 46202-5200

Paul M. Kanev, MD
Hershey Med. Ctr./Neurosurgery
PO Box 850
Hershey, PA 17033-0850

Samuel S. Kasoff, MD
3141 Rte. 9W, Suite 2
New Windsor, NY 12553

Bruce A. Kaufman, MD
St. Louis Children's Hosp.
1 Children's Pl./Neuro.
St. Louis, MO 63110

Robert F. Keating, MD
Children's National Med. Ctr.
111 Michigan Ave., N.W./Neuro.
Washington, DC 20010-2401

David L. Kelly, Jr., MD
Wake Forest Sch. of Med.
Medical Center Blvd./Neuro.
Winston-Salem, NC 27157-1029

John R. W. Kestle, MD
Ped. Neurosurgery
100 N. Medical Dr., Suite 2400
Salt Lake City, UT 84113-1100

David M. Klein, MD
690 Fearrington Post
Pittsboro, NC 27312-8507

Laurence I. Kleiner, MD
5304 Indian Grave Rd., Suite A
Roanoke, VA 24014-6608

David S. Knierim, MD
GED 4
9300 Valley Childrens
Pl. Madera, CA 93638

Edward J. Kosnik, MD
Chatham Village Prof. Bldg.
931 Chatham Ln.
Columbus, OH 43221-2417

Cornelius H. Lam, MD
Box 96 Mayo, D-429 Mayo
420 Delaware St., S.E./Neuros.
Minneapolis, MN 55455

John P. Laurent, MD
Texas Childrens Hosp.
6621 Fannin St., MC 3-3435
Houston, TX 77030-2399

Edward R. Laws, Jr., MD
Univ. of Virginia/Neuro.
Health Sciences Ctr., Box 212
Charlottesville, VA 22908-0001

Mark Robert Lee, MD
Med. Coll. of Georgia
1120 15th St./Neuros. BIW-348
Augusta, GA 30912-4010

Michael Lee Levy, MD
1300 N. Vermont St., Suite 906"
Los Angeles, CA 90027

Veetai Li, MD
Dept. of Neurosurgery
219 Bryant St.
Buffalo, NY 14222-2006

Kenneth I. Lipow, MD
267 Grant St.
Bridgeport, CT 06610-2805

John D. Loeser, MD
Univ. of Washington
Box 356470/Neurosurgery
Seattle, WA 98195

Morris D. Loffman, MD
Suite 1205
16311 Ventura Blvd.
Encino, CA 91436-2152

Rafael Longo-Cordero, MD
Univ. Gardens Calle Rochester 911
San Juan, PR 00927-4812

Ralph C. Loomis, MD
7 McDowell St.
Asheville, NC 28801-4103

Kenneth M. Louis, MD
Suite 340
3000 E. Fletcher Ave.
Tampa, FL 33613-4645

Mark G. Luciano, MD, PhD
Cleveland Clinic Foundation
9500 Euclid Ave., S-80
Cleveland, OH 44195-1004

Thomas G. Luerssen, MD
Riley Hosp. for Children
One Children's Sq., #2510
Indianapolis, IN 46202-5200

Joseph R. Madsen, MD
Children's Hosp./Neurosurgery
300 Longwood Ave., Room 312
Boston, MA 02115-5724

Gail A. Magid, MD
1661 Soquel Dr., F
Santa Cruz, CA 95065-1709

Gary Magram, MD
1 Gristmill Ct., Apt. 203
Baltimore, MD 21208

Kim Herbert Manwaring, MD
Phoenix Children's Hosp.
909 E. Brill St./Neurosurgery
Phoenix, AZ 85006-2513

Timothy B. Mapstone, MD
Childrens Hosp. of Alabama
1600 Seventh Ave., S.
Birmingham, AL 35233

Arthur E. Marlin, MD
Methodist Plaza
4499 Medical Dr., Suite 397
San Antonio, TX 78229-3713

John R. Mawk, MD
1601 N.W. 114th St., Suite 134
Des Moines, IA 50325-7007

James P. McAllister, II, PhD
Wayne State Univ./Neurosurgery
4201 St. Antoine, #6E
Detroit, MI 48201

Jack E. McCallum, MD
800 8th Ave., Suite 220
Fort Worth, TX 76104-2619

J. Gordon McComb, MD
Suite 906
1300 N. Vermont Ave.
Los Angeles, CA 90027-6005

C. Scott McLanahan, MD
Carolina Neuro. & Spine Assoc.
1010 Edgehill Rd., N.
Charlotte, NC 28207-1885

Robert L. McLaurin, MD
2412 Ingleside Ave., Apt. 5C
Cincinnati, OH 45206-2185

David Gordon McLone, MD, PhD
Children's Mem. Hosp.
2300 Children's Plaza, Box 28
Chicago, IL 60614-3318

John Mealey, Jr., MD
Indiana Univ. Med. Ctr.
545 Barnhill, Emerson 139
Indianapolis, IN 46202-5112

Arnold H. Menezes, MD
Univ. of Iowa Hosp. & Clinics
200 Hawkins Dr.
Iowa City, IA 52242-1009

Glenn A. Meyer, MD
Med. Coll. of Wisconsin
9200 W. Wisconsin Ave.
Milwaukee, WI 53226-3522

W. Jost Michelsen, MD
Harbour Pl. #15
135 Bow St.
Portsmouth, NH 3801

Thomas H. Milhorat, MD
State Univ. of NY/HSC-Brooklyn
Box 1189, 450 Clarkson Ave.
Brooklyn, NY 11203-2098

Clinton F. Miller, MD
Coastal New Hampshire Neuro.
330 Borthwick Ave., Suite 108
Portsmouth, NH 03801-7110

John I. Miller, MD
St. John's Queens Hosp./Neuro.
95-25 Queens Blvd., 2nd Fl.
Rego Park, NY 11374

Richard H. Moiel, MD
3656 Ella Lee Ln.
Houston, TX 77027-4105

Mark S. Monasky, MD
900 E. Broadway
PO Box 1114
Bismarck, ND 58502-1114

Jose L. Montes, MD
The Montreal Children's Hosp.
2300 Tupper St., Room C811
Montreal PQ H3H 1P3 Canada

German Montoya, MD
1801 Cook Ave.
Orlando, FL 32806-2913

Mahmood Moradi, MD
920 Frostwood, Suite 640
Houston, TX 77024

Thomas M. Moriarty, MD, PhD
210 E. Gray St., Suite 1102
Louisville, KY 40202

William J. Morris, MD
1112 6th Ave., #302
Tacoma, WA 98405-4048

Glenn Morrison, MD
Miami Children's Hosp.
3200 S.W. 60 Ct., Suite 301
Miami, FL 33155-4071

S. David Moss, MD
Phoenix Children's Hosp.
909 E. Brill St.
Phoenix, AZ 85006-2513

Kamel F. Muakkassa, MD
130 W. Exchange St.
Akron, OH 44302-1701

Michael S. Muhlbauer, MD
930 Madison Ave., Suite 600
Memphis, TN 38103-3401

Michael G. Muhonen, MD
455 S. Main
Orange, CA 92868

Cheryl A. Muszynski, MD
BIMC-North Div.
170 E. End Ave. at 87th St.
New York, NY 10128-7603

S. Terence Myles, MD
1820 Richmond Rd., S.W.
Calgary AB T2T 5C7 Canada

John S. Myseros, MD
Children's Hospital Med. Ctr.
3333 Burnet Ave., Ped. Neuros.
Cincinnati, OH 45229-3039

Joseph M. Nadell, MD
Children's Hosp.
200 Henry Clay Ave.
New Orleans, LA 70118-5720

Mahmoud G. Nagib, MD
305 Piper Bldg.
800 E. 28th St.
Minneapolis, MN 55407-3799

Richard C. Nagle, MD
3000 Colby, Suite 101
Berkeley, CA 94705-2058

Gregory Nazar, MD
5008 Long Knife Run
Louisville, KY 40207-1175

Mark Stephen O'Brien, MD
1900 Century Blvd., Suite 4
Atlanta, GA 30345-3307

W. Jerry Oakes, MD
Childrens Hosp. of Alabama
1600 7th Ave., S., Suite 400
Birmingham, AL 35233

Larry Keith Page, MD
13845 S.W. 73rd Ct.
Miami, FL 33158-1213

Dachling Pang, MD
Kaiser Permanete Hosp.
2025 Morse Ave.
Sacramento, CA 95825

Andrew D. Parent, MD
Univ. of Mississippi MC
2500 N. State
St. Jackson, MS 39216

Tae Sung Park, MD
St. Louis Children's Hosp.
1 Children's Pl./Neurosurgery
St. Louis, MO 63110

Michael David Partington, MD
Neurosurgery Assoc.
280 N. Smith Ave., #234
St. Paul, MN 55102

Jogi V. Pattisapu, MD
Suite 204
22 W. Lake Beauty Dr.
Orlando, FL 32806-2037

Jerry O. Penix, MD
607 Medical Tower
Norfolk, VA 23507

Joseph A. Petronio, MD
Emory Univ. Sch. of Med./Neuro
1365 Clifton Rd., Suite B-6400
Atlanta, GA 30322

Joseph H. Piatt, Jr., MD
Div. of Neurosurgery, L472
3181 S.W. Sam Jackson Park Rd.
Portland, OR 97201-3098

Hal Watson Pittman, MD
44 E. Palm Ln.
Phoenix, AZ 85004

Thomas Pittman, MD
St. Louis Univ. Hosp.
1465 S. Grand
St. Louis, MO 63110-1003

Ian F. Pollack, MD
Children's Hosp. of Pittsburgh
3705 Fifth Ave./Neurosurgery
Pittsburgh, PA 15213-2524

Harold D. Portnoy, MD
888 Woodward Ave., Suite 506
Pontiac, MI 48341-2982

Antonio R. Prats, MD
PO Box 440757
Miami, FL 33144-0757

Mark R. Proctor, MD
300 Longwood Ave., Bader 3
Boston, MA 2115

Corey Raffel, MD, PhD
Mayo Clinic/Neurosurgery
200 First St., S.W.
Rochester, MN 55905-0001

John Ragheb, MD
Univ. of Miami/Neurosurg.
D4-6 1501 N.W. 9th Ave.
Miami, FL 33136-1407

Anthony J. Raimondi, MD
Villa Monteleone
37020 Gargagnago
Verona 37100 Italy

Donald H. Reigel, MD
134 Shenot Rd.
Wexford, PA 15090

Harold Louis Rekate, MD
Barrow Neurosurg. Assoc. Ltd.
2910 N. 3rd Ave.
Phoenix, AZ 85013-4434

Arden F. Reynolds, Jr., MD
Quincy Med. Group
1025 Maine St.
Quincy, IL 62301

Theodore S. Roberts, MD
Childrens Hospital Med. Ctr. (CN-50) 4820 Sand Point Ray NE
Seattle, WA 98105

Walker L. Robinson, MD
New Jersey Neurosci. Inst.
65 James St./Neurosurgery
Edison, NJ 8818

Luis A. Rodriguez, MD
1150 N. 35th Ave., Suite 300
Hollywood, FL 33021-5424

Bruce R. Rosenblum, MD
Riverview Med. Ctr.
160 Ave. at The Commons
Shrewsbury, NJ 7702

Alan Rosenthal, MD
Long Island Neuro. Assoc.
410 Lakeville Rd.
New Hyde Park, NY 11042-1101

Allen S. Rothman, MD
1160 Fifth Ave., Suite 106
New York, NY 10029-6928

John R. Ruge, MD
630 S. Oak St.
Hinsdale, IL 60521-4634

James T. Rutka, MD
Hosp. for Sick Children
555 University Ave./Neuro.
Toronto ON M5G 1X8 Canada

Petr O. Ruzicka, MD
741 Northfield Ave.
West Orange, NJ 07052-1104

Robert A. Sanford, MD
Semmes-Murphey Clinic
930 Madison Ave., Suite 600
Memphis, TN 38103-3401

Osamu Sato, MD
1-5-40, Tamagawa Gakuen
Machida Tokyo 194-0041 Japan

Guy M. Sava, MD
Dhahran Hlth. Ctr./Surg. Svc.
Room A-422, Box 76
Dhahran 31311 Saudi Arabia

Timothy B. Scarff, MD
Suite 25
33 S.W. Upper Riverdale Rd.
Riverdale, GA 30274-2626

Steven J. Schiff, MD
Krasnow Inst.
George Mason Univ., MS 2A1
Fairfax, VA 22030-4444

Steven J. Schneider, MD
Long Island Neuro. Assoc.
410 Lakeville Rd.
New Hyde Park, NY 11042-1101

Luis Schut, MD
Childrens Hosp.
Philadelphia 34th and Civic Center Blvd.
Philadelphia, PA 19104

R. Michael Scott, MD
The Children's Hosp.
300 Longwood Ave., Bader 319
Boston, MA 02115-5724

Edward L. Seljeskog, MD
Suite 110
2805 Fifth St., S.
Rapid City SD 57701-7306

Robert G. Selker, MD
Western Pennsylvania Hosp.
4800 Friendship Ave./Neuro.
Pittsburgh, PA 15224-1722

Wan Tew Seow, MD
94 Greenwood Ave.
Singapore 289300 Singapore

Ronald F. Shallat, MD
3000 Colby St., Suite 101
Berkeley, CA 94705-2058

Kenneth N. Shapiro, MD
1935 Motor St.
Dallas, TX 75235-7794

John Shillito, MD
1109 Fearrington Post 6 Caswell Sq.
Pittsboro, NC 27312-5014

James C. Simmons, MD
190 Grove Park Rd.
Memphis, TN 38117

Gary Robert Simonds, MD
237 Sunbury Rd.
PO Box 14
Riverside, PA 17868-0014

Frederick H. Sklar, MD
1935 Motor St.
Dallas, TX 75235-7794

Harold P. Smith, MD
300 20th Ave., N., #506
Nashville, TN 37203-2131

Lenwood P. Smith, Jr., MD
3 Medical Park, Suite 310
Columbia, SC 29203

Mark M. Souweidane, MD
New York Hosp./Star Pav.
520 E. 70th St., Room 651
New York, NY 10021

Phillip G. St. Louis, MD
Florida Med. Plaza, Suite 540N
2501 N. Orange Ave.
Orlando, FL 32804

Sherman Charles Stein, MD
310 Spruce St.
Philadelphia, PA 19106-4201

Paul Steinbok, MD
B.C. Children's Hosp.
4480 Oak St., Room A325
Vancouver BC V6H 3V4 Canada

Bruce B. Storrs, MD
Univ. of New Mexico/Neurosurg.
2211 Lomas, N.E., 2ACC
Albuquerque, NM 87131

Douglas L. Stringer, MD
2011 Harrison Ave.
Panama City, FL 32405-4545

Merle Preston Stringer, MD
2011 N. Harrison Ave.
Panama City, FL 32405-4545

Michael H. Sukoff, MD
17602 17th St., #102-118
Tustin, CA 92680-1961

Anthony F. Susen, MD
193 Old Glebe Point Rd.
Burgess, VA 22432

Leslie N. Sutton, MD
Child. Hosp. of Philadelphia 34th & Civic Center Blvd.
Philadelphia, PA 19104

Michael S. Taekman, MD
15 Oakmont Ct.
San Rafael, CA 94901-2058

John M. Tew, Jr., MD
Mayfield Clinic
506 Oak St.
Cincinnati, OH 45219

Tadanori Tomita, MD
Children's Mem. Hosp./Neuros.
2300 Children's Plaza, Box 28
Chicago, IL 60614-3318

Noel Tulipan, MD
8533 McCrory Ln.
Nashville, TN 37221-5905

Michael S. Turner, MD
Suite 535 1801 N. Senate Blvd.
Indianapolis, IN 46202-1228

David D. Udehn, MD
4350 Seventh
St. Moline, IL 61265-6870

Ronald H. Uscinski, MD
3301 Woodburn Rd., #209
Annandale, VA 22003-1229

Michael Vassilyadi, MD
Childrens Hosp. Medical Center
333 Burnet, Div. of Neurosurg.
Cincinnati, OH 45229-3039

Joan L. Venes, MD
123 Waldon St.
Brentwood, CA 94513-6212

Enrique C. Ventureyra, MD
401 Smyth Rd. Ottawa
ON K1H 8L1 Canada

John Kenric Vries, MD
Univ. of Pittsburgh
217 Victoria Bldg.
Pittsburgh, PA 15261-0001

Steven L. Wald, MD
10 Grove Ln.
Shelburne, VT 05482-7232

John B. Waldman, MD
Albany Med. Coll.
Div. of Neurosurgery/MC-61-NE
Albany, NY 12208

Marion L. Walker, MD
Primary Children's Med. Ctr.
100 N. Medical Dr./Ped. Neuro.
Salt Lake City, UT 84113-1100

John Willson Walsh, MD
Tulane Univ. MC/Neurosurgery
1430 Tulane Ave., SL-47
New Orleans, LA 70122

John D. Ward, MD
Virginia Commonwealth Univ.
Box 980631
Richmond, VA 23298-0631

Benjamin C. Warf, MD
Univ. of Kentucky Med. Ctr.
800 Rose St./Neurosurgery
Lexington, KY 40536-0084

Martin H. Weiss, MD
USC Med. Ctr., Box 786
1200 N. State St., Room 5046
Los Angele, CA 90033

Robert Joseph White, MD, PhD
MetroHealth Med. Ctr.
2500 MetroHealth Dr.
Cleveland, OH 44109-1998

Jean K. Wickersham, MD
3030 Children's Way, Suite 402
San Diego, CA 92123

Philip J. A. Willman, MD
3006 S. Alameda St.
Corpus Christi, TX 78404-2601

Ronald J. Wilson, MD
Suite 210
901 S. Mopac Expy., Bldg. V
Austin, TX 78746

Joel W. Winer, MD
York Neurosurgical Assoc., PC
2319 S. George
St. York, PA 17403-5009

Jeffrey A. Winfield, MD, PhD
Hill Med. Ctr.
1000 E. Genesse St., Ste. 202
Syracuse, NY 13210-1853

Ken R. Winston, MD
1950 Ogden, B467
Denver, CO 80218-1022

Jeffrey H.Wisoff, MD
NYU Med. Ctr./Ped. Neurosurg.
550 1st Ave.
New York, NY 10016

Daniel Won, MD
11780 Pecan Way
Loma Linda, CA 92354-3452

Meredith V. Woodward, MD
4208 W. Alluvial
Fresno, CA 93722

Shokei Yamada, MD
Room 25628/Neurosurgery
Loma Linda Univ. Sch. of Med.
Loma Linda, CA 92350-0001

Karol Zakalik, MD
William Beaumont Hosp.
3535 W. 13 Mile Rd., Suite 504
Royal Oak, MI 48073-6704

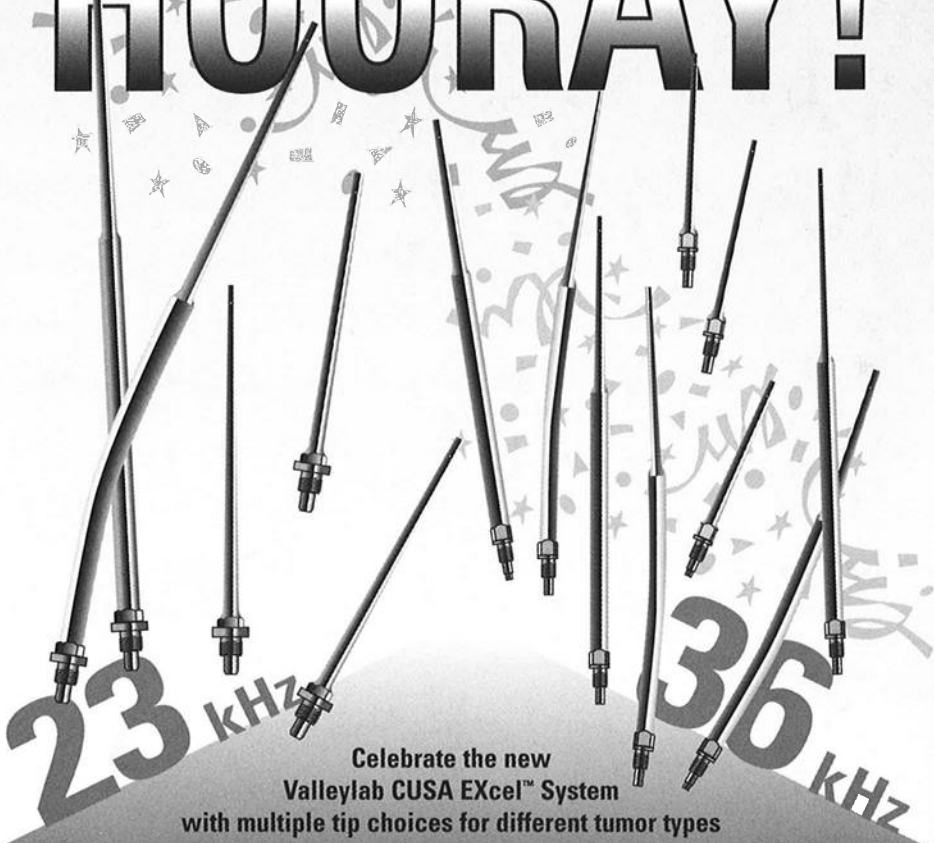
Ahmad Zakeri, MD
4235 Secor Rd.
Toledo, OH 43623-4231

Edward J. Zampella, MD
10 Parrott Mill Rd.
PO Box 808
Chatham, NJ 07928-2744

L. Manuel Zavala, MD
2400 Country Dr., #100
Fremont, CA 94536

John G. Zovickian, MD
3000 Colby St., Suite 101
Berkeley, CA 94705-2058

TIP, TIP HOORAY!



Celebrate the new Valleylab CUSA EXcel™ System with multiple tip choices for different tumor types

Now you can select the tip you need before a surgical procedure AND change your tip choice intra-operatively

Experience "EXcel"lence in ultrasonic aspiration systems with the new Valleylab CUSA EXcel™ System

CUSA EXcel™

Valleylab™
Innovations That Work

Valleylab • Boulder, CO 80301-3299 • 800-255-8522 • www.valleylab.com